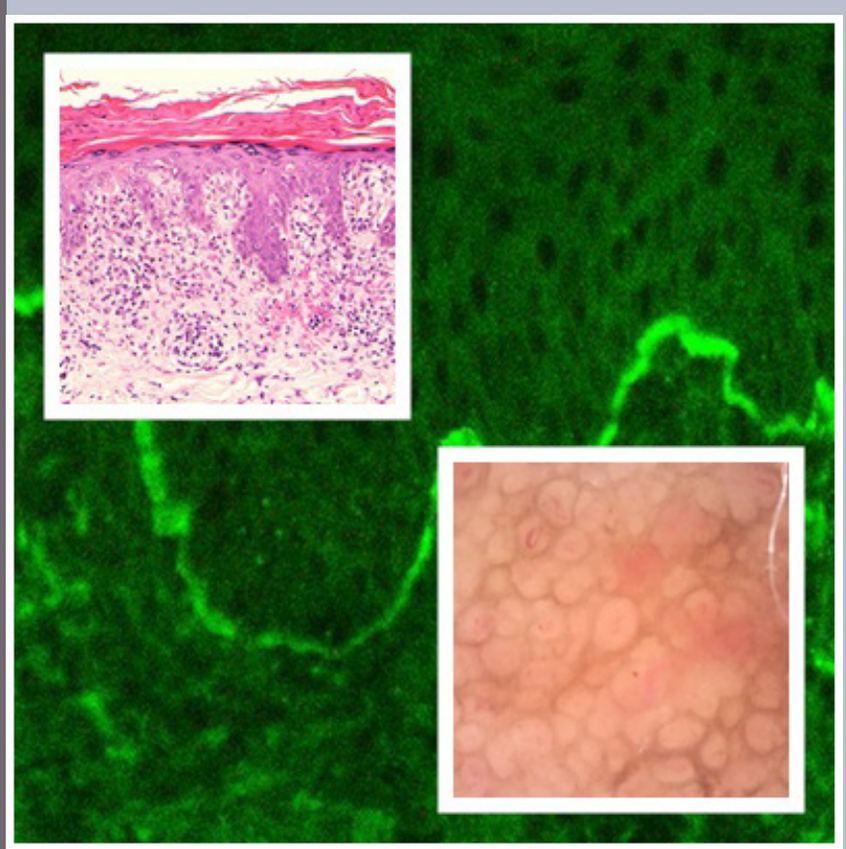
Volume 17, Number 2 April 2026
p. 146-282
Issue online since Wednesday April 01 2026

ISSN: 2956-7904
DOI: 10.7241/ourd

Our Dermatology Online

www.odermatol.cowm



- A study of clinical and sociodemographic features of trichomoniasis in symptomatic and asymptomatic female patients attending the STD clinic using wet mount and culture as diagnostic tools;
- Epidemiological, clinical, and evolutionary profile of patients treated with cryotherapy at the Bamako Dermatology Hospital (HDB) from January 2021 to December 2023;
- Nevi in children: Epidemiological, clinical, and dermoscopic profiles and epidemiological-clinical-dermoscopic correlations;
- Lupus pernio as an important feature of different cutaneous diseases in a case series of 57 cases ;
- Micropigmentation in dermatological rehabilitation: Scar camouflage through permanent makeup;
- Mucin-secreting cutaneous diseases: Clinical and histopathological study in a series of 84 cases;
- Follicular mucinosis: A retrospective study;
- Prognosis of skin cancers in the Conakry Cancer Department, Guinea;
- Chronic loose scaly cheilitis: Often-Overlooked variant;
- Are eponyms easy to remember?

Issue 2.2026

Editorial Pages

e-ISSN: 2956-7904
DOI: 10.7241/ourd

Quarterly
Our Dermatol Online

published since 01/06/2010 years

www.odermatol.com

Indexed in:

Universal Impact Factor for year 2012 is = 0.7319
system of opinion of scientific periodicals INDEX COPERNICUS (100.00)
(Academic Search) EBSCO
(Academic Search Premier) EBSCO
MNiSW-Ministerstwo Nauki i Szkolnictwa Wyższego (20.00)
DOAJ (Directory of Open Acces Journals)
Geneva Foundation for Medical Education and Research (GFMER), Google Scholar, Open J-Gate, NewJour,
International Committee of Medical Journal Editors (ICMJE), Genamics JournalSeek, Hinari,
Bielefeld Academic Search Engine (BASE), WorldCat, e -journal, WorldWideScience.org, National Science Library,
LibSearch, Sciencegate, Virtual Science Library (VSL), Wanfang Data, COnnecting REpositories (CORE),
CAB Abstracts, Global Health, Journal Indexed in Directory of Research Journals Indexing,
OAIster: The Open Access Initiative, OAJSE - Open Access Journals Search Engine, Scirus

Previous website:	issue 1.2010	www.ndermatol.like.pl
	since issue 2.2010 to issue 3.2011	www.odermatol.like.pl
	since issue 4.2011	www.odermatol.com
Previous shortcut:	since issue 1.2010 to issue 3.2011	N Dermatol Online
	since issue 4.2011	Our Dermatol Online
Previous ISSN:	2081-7904	

Editorial Board

Contents

Contents

Contents

Our Dermatology Online

Original Article

A study of clinical and sociodemographic features of trichomoniasis in symptomatic and asymptomatic female patients attending the STD clinic using wet mount and culture as diagnostic tools

Anupama Manohar Prasad[1], Aneeha Ramesh Babu[2], Praveen Kumar Arinaganahalli Subbanna[3]

[1]Professor, Department of Dermatology, The Oxford medical college hospital and research centre, Yadavanahalli, Attibele, Bengaluru, India, [2]Consultant Dermatologist, Citizen Skin Clinic, Bengaluru, India, [3]Consultant cardiologist, Fortis Hospital, Bengaluru, India

Corresponding author: Anupama Manohar Prasad, E-mail: dranupama.mp@gmail.com

ABSTRACT

Background: Trichomoniasis is a sexually transmitted infection caused by the protozoan *Trichomonas vaginalis*, which accounts for more than half of all curable STIs (sexually transmitted infections) worldwide. **Objectives:** The study was performed to determine the disease characteristics and prevalence of trichomoniasis in asymptomatic and symptomatic female patients using wet mount and culture as the diagnostic methods. **Materials and Methods:** A cross-sectional study was conducted at the OPD of the Institute of Dermatology and Venereology at a tertiary-care center in South India for a period of nine months. 500 female patients aged > 18 years and < 55 years were enrolled in the study. 250 symptomatic and 250 asymptomatic patients were selected randomly. **Results:** A total of 8% of the women had trichomoniasis; 31 (12.4%) were in the symptomatic group and 9 (3.6%) were in the asymptomatic group. The risk factors associated with trichomoniasis in our study were women from urban areas with low socioeconomic status, who were single and separated, women with multiple sexual partners, and women whose husbands had extramarital contacts. **Conclusion:** Trichomoniasis is a marker for high-risk sexual behavior and the relatively high prevalence rate in the asymptomatic group indicates the need for routine screening of women in their reproductive age group to reduce the risks of acquiring other STIs and HIV infection.

Key words: Diamond's medium, *Trichomonas vaginalis*, wet mount, culture

INTRODUCTION

Trichomoniasis is caused by the pathogenic protozoan *Trichomonas vaginalis*, and it is one of the commonest non-viral sexually transmitted diseases (STDs) [1]. Trichomonads are flagellated eukaryotic microbes that belong to the protozoan order Trichomonadida. Typically Trichomonads are pyriform shaped, 7-32 micrometer long, 5-12 micrometer wide and roughly the size of leukocyte [2]. It causes approximately more than 180 million infections worldwide annually. Many infected individuals remain mostly asymptomatic; when symptomatic, they present with vaginal discharge, cervicitis, pelvic inflammatory disease, and infertility in women and non-gonococcal urethritis in men [3]. A higher prevalence of trichomoniasis has been seen in women who are multiparous, married at a very early age, and pregnant women [4]. Other risk factors associated are multiple sexual contacts, poor personal hygiene, and low socioeconomic status [5]. Coinfections with other sexually transmitted disease caused by pathogens such as *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, *Treponema*

How to cite this article: Manohar Prasad A, Ramesh Babu A, Arinaganahalli Subbanna PK. A study of clinical and sociodemographic features of trichomoniasis in symptomatic and asymptomatic female patients attending the STD clinic using wet mount and culture as diagnostic tools. Our Dermatol Online. 2026;17(2):146-153.

Submission: 03.06.2025; **Acceptance:** 11.11.2025

DOI: 10.7241/ourd.20262.1

pallidum, human papillomavirus, or herpes simplex virus types 1 and 2 [6] are common in women with trichomoniasis. Coinfection of trichomoniasis and HIV infection may facilitate viral transmission and acquisition by eliciting an inflammatory response and the recruitment of CD4 cells in the vaginal epithelium; also, there is an increased risk of HIV transmission in *Trichomonas vaginalis*-infected patients [7,8] by 2- to 3-fold, and the prevalence rate of *T. vaginalis* infection in HIV-affected patients ranges from 9% to 30% [9]. Trichomoniasis as such is highly prevalent in sexually active women, varying from 5% to 74% and 5% to 29% in men. About 10% to 50% of patients harbor trichomonads without developing any symptoms, which serve as a major reservoir of infection [10]. In this background, we planned to conduct a study to determine the sociodemographic, and clinical characteristics, and to diagnose as well as compare *Trichomonas* infection in asymptomatic and symptomatic female patients attending the STD OPD, using wet mount and culture as the diagnostic methods, which are the gold standard [6].

MATERIALS AND METHODS

A cross-sectional study was conducted at a tertiary-care hospital at the Institute of Dermatology and Venereology in South India for the period of nine months. 500 female patients aged > 18 years and < 55 years were enrolled in the study. Female patients with complaints of vaginal discharge, pruritus, dysuria, dyspareunia, and lower abdomen pain were included in the symptomatic group, and patients attending the STD OPD for routine checkups and screening were included in the asymptomatic group. Patients on any antibiotics, including metronidazole, taken two to three weeks before the study enrollment and those with severe medical comorbidities were excluded from the study. A detailed history was obtained on the following parameters: age, occupation, socioeconomic status, educational status, marital history, sexual, contraceptive, obstetric, past, personal, recent treatment history, history suggestive of systemic ailments, and symptoms related to STIs. Detailed genital examination was done using Cusco's self-retaining bivalve speculum, and the abnormalities in the vulva, vagina, and cervix were noted. The amount, odor, color, and consistency of vaginal discharge were noted. Four vaginal swabs were taken from the posterior fornix or collected directly from the discharge. One swab was used for the wet mount preparation, another was inoculated directly into the Diamonds TYIS-33 medium, and the other two swabs were used for Gram's staining and KOH preparation, respectively. Cervical smear was also taken from all patients. The institutional ethical committee clearance was taken, and written informed consent was obtained from all recruited women. All participants underwent culture for *Gonococcus*, blood VDRL, and HIV.

Statistical analysis: The data was collected and tabulated in a Microsoft Excel worksheet. Computer-based analysis was performed using SPSS 13.0 software (SPSS, Chicago, IL, USA). The categorical variables were summarized as proportions and percentages. The continuous variables were summarized as means and standard deviations. For comparison of means, the unpaired *t*-test and one-way ANOVA were used for two and more than two groups, respectively. For the comparison of proportions, the chi-squared test was employed. If the cell values were less than five, Fisher's exact test was used. For comparison within the group, the chi-squared goodness-of-fit test was used.

The diagnosis of trichomoniasis was established on the basis of the following tests.

Wet mount: A drop of normal saline was put over a clean, grease-free microscopic slide. To this, a drop of vaginal fluid was added and mixed well. A coverslip was put over the mixture to allow a uniform spread without air bubbles. The slide was observed under 40x magnification.

Reading: Pear-shaped flagellated organisms approximately the size of a lymphocyte (10–20 μm) or that of a small neutrophil with the characteristic jerky motility were noted.

Culture: The wet mounts were prepared from the drop of Diamond's culture media and examined for the presence of motile trichomonads after 48 hours of incubation and on the 3rd, 5th, and 7th day. The Diamond's culture media tubes with 5 mL of the broth were incubated in an anaerobic atmosphere at 35°C.

Mucopurulent cervicitis: Diagnosed in those patients with Gram stain of cervical smear, showing polymorphonuclear leukocytes more than 30 per oil immersion field in the cervical mucus.

Pelvic inflammatory disease (PID): The diagnosis of PID was made if, in addition to the presenting symptoms of abnormal vaginal discharge, lower

abdominal pain, adnexal structures involvement elicited on clinical examination.

Ethics Statement

Ethical clearance was obtained from the ethical committee of the institution prior to the study.

No: 05012012

RESULTS

The mean age of the patients in the symptomatic group was 33.85 ± 9.65 years, and in the asymptomatic group, it was 34.25 ± 9.36, (Fig. 1). In our study, 8% of women had trichomoniasis out of the 500 women examined. 31 out of the 250 women (12.4%) in the symptomatic group had an infection with *T. vaginalis* by culture and/or wet mount, and 9 out of 250 (3.6%) asymptomatic women had trichomoniasis by culture and/or wet mount. The wet mount test was positive in 67.74% of the symptomatic participants and 55.5% of the asymptomatic (Figs. 2a and 2b). The wet mount preparation using normal saline had low sensitivity and high specificity. Culture done using Diamond's liquid media had sensitivity and specificity of 100%, PPV 100%, and NPV 100% when observed within three days of inoculation (Fig. 3). The culture examined on the 7th day of inoculation had a sensitivity of 77.50%, a specificity of 100%, a PPV of 100%, and an NPV of 98.5%. The comparison between wet mount and culture is shown in Table 1. The mean age group of *Trichomonas*-positive patients in the symptomatic group was 32 ± 7 years, whereas in the asymptomatic group, the mean age was 34 ± 8 years. 74.2% and 62.50% in the symptomatic and asymptomatic groups were married, and 25% were single among both the symptomatic and asymptomatic patients. 21 patients (67.7%) in the symptomatic group and 6 patients (67%) in the asymptomatic group lived in urban areas. 54.80% and 75% of the patients in the symptomatic and asymptomatic groups, respectively, belonged to the lower class of socioeconomic status, followed by 32.3% of the symptomatic patients and 25% of the asymptomatic patients belonging to the upper lower class. Most of the patients in both groups (45.20%) had an education level till high school, and 12% in the symptomatic group were illiterate. 14 (45.2%) patients in the symptomatic group and 4 (40%) patients in the asymptomatic group had a history of pre/extramarital contact. 22.6% of the partners of the symptomatic group

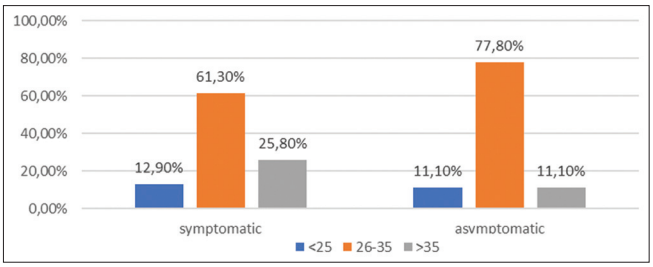


Figure 1: Age distribution of *Trichomonas*-positive cases.

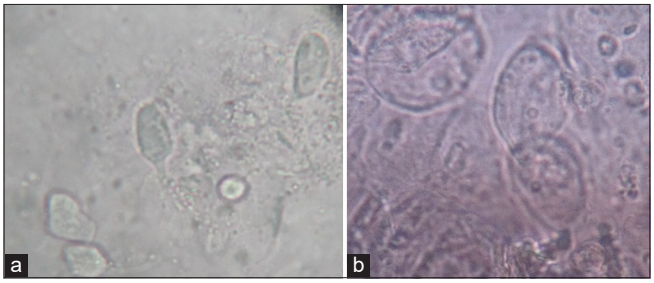


Figure 2: (a and b) Image showing *Trichomonas vaginalis*: Wet mount in low and high power.

Table 1: TV-positive cases among both the symptomatic and asymptomatic patients

Wet-Mount	Culture in Symptomatic Group		Culture in Asymptomatic Group	
	Positive	Negative	Positive	Negative
Wet-mount positive	21	0	5	0
Wet-mount negative	10	219	4	241
Total	31	219	9	241

were promiscuous. 7.7% of the partners of positive cases had a history of urethritis, dysuria, and pruritus. The disease characteristics of *Trichomonas*-positive cases in our study are as follows. The mean age of symptom onset in patients with trichomoniasis was 30 years of age, and the mean duration of vaginal discharge in patients with trichomoniasis was 4.8 months; 77.4% of women had vaginal discharge for less than a year, and 22.6% had discharge for more than a year. Associated vulval itching was found in 29% and lower abdominal pain in 9.7%. About 90.3% of women in the symptomatic group had moderate to profuse vaginal discharge, and 64.5% had foul-smelling discharge (Fig. 4) (Table 2). Out of nine positive cases in the asymptomatic group, one patient had moderate vaginal discharge on examination. 12.9% of the patients had a history of abortion when compared with negative cases, which was statistically significant. 65.5% of women had increased symptoms during menstruation. Only 12.9% of the patients had erythema on the vaginal wall. None of the patients in our study showed a strawberry cervix (colposcopy not done). A genital ulcer was noted in only

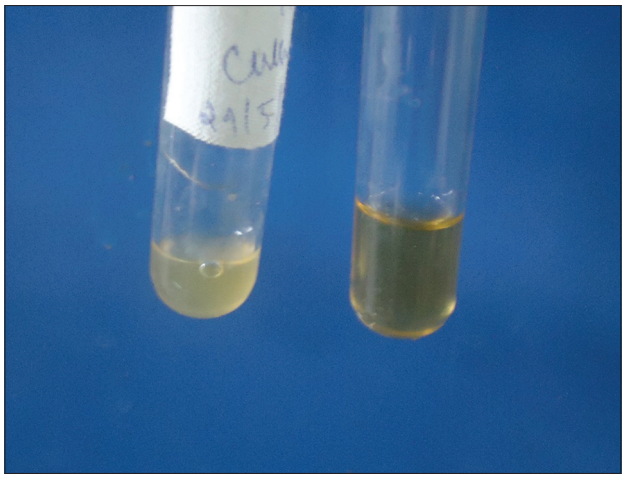


Figure 3: Image indicating the growth of Trichomonas vaginalis in modified Diamond's medium as a turbid media.

Figure 4: Image of a patient with trichomoniasis: profuse, frothy vaginal discharge.

one patient. Bacterial vaginosis was the commonest coinfection noted followed by mucopurulent cervicitis and candidiasis. Three patients in symptomatic and six patients in the asymptomatic group were positive for HIV. Table 3 lists the various diagnoses of the patients enrolled in our study. All trichomoniasis patients were treated with tablet metronidazole 400 mg twice daily for seven days, and the symptomatic partners were treated with tablet secnidazole 2 gm stat dose.

DISCUSSION

Trichomoniasis is a common non-viral sexually transmitted infection. The prevalence rate of *Trichomonas vaginalis* infection largely varies among the female population all over the world, depending on the group of population studied and the method of investigation employed in the diagnosis, ranging from 5% to 74%. The highest rate of prevalence was reported

Table 2: Vaginal discharge characteristics of the TV-positive cases

Vaginal Discharge		Patients			
		Symptomatic Group		Asymptomatic Group	
		Trichomoniasis		Trichomoniasis	
		Positive		Positive	
		N	%	N	%
Discharge amount	Scanty	3	9.7	8	88.8
	Moderate	15	48.4	1	11.1
	Profuse	13	41.9	0	0
Discharge odor	Odorless	9	29.0	6	66.7
	Foul-smelling	20	64.5	2	22.2
	Fishy	2	6.5	1	11.1
Discharge consistency	Curdy	0	0.0	0	0.0
	Flocculent	1	3.2	1	11.1
	Thick, homogeneous	1	3.2	0	0.0
	Frothy	18	58.1	2	22.2
	Watery	5	16.1	2	22.2
	Mucoid	6	19.4	4	44.4
Discharge color	White	18	58.1	6	66.7
	Greyish	1	3.2	0	0.0
	Purulent	12	38.7	0.0	0.0
	Serosanguinous/ white	0	0.0	3	33.3
	Total	31	100.0	9	100.0

Table 3: Diagnosis of the study groups

Disease	Symptomatic Group	Asymptomatic Group
1. Trichomonas vaginalis	31 (12.4%)	9 (3.6%)
2. Bacterial vaginosis	54 (21.6%)	27 (10.8%)
3. Candidiasis	32 (12.8%)	29 (11.6%)
4. Mucopurulent cervicitis	40 (16%)	26 (10.4%)
5. Physiological/non venereal	69 (27.6%)	145 (58%)
6. Herpes simplex	6 (2.4%)	0
7. HIV	3 (1.2%)	6 (2.4%)
8. Wart	2 (0.8%)	0
9. Genital scabies	1 (0.4%)	0
10. Cervical cancer	5 (2%)	0
11. Cervical growth	1 (0.4%)	0
12. Others	6 (2.4%)	8 (3.2%)

among STI clinic attending patients and among high-risk populations [10,11]. In our study, a total of 8% (40 out of 500) of women had trichomoniasis, 31 (12.4%) women in the symptomatic group and 9 (3.6%) women in the asymptomatic group diagnosed by culture. The prevalence of trichomoniasis observed in our study was comparable to the study done by Mahmoud et al., which was a hospital-based study done in Egypt with a sample size of 450, among which 290 were symptomatic and 160 were asymptomatic. The prevalence of trichomoniasis in their study was 7.7%, among which 30 (10.34%) were symptomatic cases and 3.1% were asymptomatic [12]. In a study by Cevahir et al, which included 310 symptomatic patients, the prevalence rate of trichomoniasis was

found to be 12.9%. This is comparable to our study (12.4%) in the symptomatic group [13]. Another study was done by Chakaraborthy et al. [14] on the symptomatic and asymptomatic patients from rural and urban areas (102 cases) in Surat, India, showing a total prevalence of 34.4%, which was significantly higher when compared to our study (8%). The prevalence rate of *Trichomonas vaginalis* infection increases with age, unlike in *Chlamydia* and gonorrhea [10]. The increased prevalence of *Trichomonas* infection in older women suggests a longer duration of the infection and its predominantly asymptomatic nature [15], which was evident in our study, as the mean age at presentation for women with *T. vaginalis* infection was 32 ± 7 years in the symptomatic and 34 ± 9 years in the asymptomatic group. This was comparable with a population-based study from Vanuatu, Australia, by Fotinatos et al. [16], and a study done by Haythan et al., who reported a mean age of 36.6 years. However, a study by Leon et al. [17] reported a lower mean age at presentation, i.e., 20 to 25 years. This probably reflects the early onset of sexual activity in the high-risk population. About 74.2% of women in the symptomatic group and 62.5% of women in the asymptomatic group were married, and 25% of women were single among both the symptomatic and asymptomatic patients. The risk of infection is more likely in women living single (16.1%) as compared to *Trichomonas*-negative single women (5.5%). Klinger et al. [18] observed that the risk of trichomoniasis was significantly increased among women who were separated and was comparable to our study. Although trichomoniasis was observed in the majority of married women, the prevalence in separated women was found to be higher in their study. About 54.8% of women with trichomoniasis in our study belonged to lower (class V) socioeconomic status when compared to the upper lower (class IV) in *Trichomonas*-negative patients. The majority of women with trichomoniasis in our study had low education status till primary standard. About 48.7% of women studied till 5th standard, 35.9% were educated till 6–12th standard, whereas most of the *Trichomonas*-negative cases had (52.2%) education till 6–12th standard. 12% were illiterate in the symptomatic group. However, in a study conducted by Haythm et al., illiterates formed the majority (61.8%) [19]. Most of the *Trichomonas*-positive women and *Trichomonas*-negative women in our study were tubectomized. However, 10.3% of *Trichomonas*-positive women gave a history of at least one episode of abortion when compared to only 2% of *Trichomonas*-negative women with a history of abortion, which is significant. Six women in the symptomatic group and two women in the asymptomatic group gave a history of previous infection in the form of vaginal discharge, and two patients in the symptomatic group had a history of PID, which was not significant when compared to *Trichomonas*-negative cases. Only two women among the *Trichomonas*-positive cases in the asymptomatic group had a history of alcohol abuse, which was not significant, and no woman gave a history of smoking in our study. About 45.2% of *Trichomonas*-positive cases in the symptomatic group and 50% of women in the asymptomatic group gave a history of premarital and extramarital contact, when compared with only 8.5% of women in the *Trichomonas*-negative group had a history of pre- and extramarital contact, which was statistically significant. Seven women among the *Trichomonas*-positive cases were indulged in prostitution as compared to four women among the *Trichomonas*-negative cases. About 22.6% of trichomoniasis-positive women in the symptomatic group gave a history of their husband having extramarital contact when compared to only 1.25% of women among the negative cases, which was statistically significant. A history suggestive of symptoms of urethritis and balanitis was noted in 7.7%, and 5.1% of men had a history of dysuria and pruritus. The risk factors associated with trichomoniasis infection in our study were women with low socioeconomic status, with a history of pre- or extramarital sexual contact, their husband having extramarital contact, a history of a symptomatic partner with, for instance, dysuria, and urethral discharge, which was suggestive of trichomoniasis in men. Kaur et al. [20], in a study done in North India to assess the prevalence rate of *Trichomonas vaginalis* infection in the symptomatic women and in women diagnosed with carcinoma cervix and HIV, observed that women who were housewives, from low to middle socioeconomic status and non-users of contraception were significantly associated with trichomoniasis. Klinger et al. [18] in their study to determine the predictors and risk factors associated with *Trichomonas* infection in women in Moshi, Tanzania, observed that having a partner with infection was the strongest risk factor in women. Other risk factors observed in their study were daily alcohol consumption, being separated, and having a partner with extramarital contact. Sutton et al. [21] observed that factors associated with an increased risk of *T. vaginalis* infection in women in the U.S. were belonging to a non-Hispanic black race, having more sex partners, being elderly, having low education level, and belonging to a low socioeconomic status.

Trichomonas vaginalis is known to cause persistently untreated cases [22]. In this study, the mean duration of the onset of symptoms in patients diagnosed with trichomoniasis was thirty years, and the mean duration of vaginal discharge in positive patients was 4.8 months (149 days). Signs of infection in symptomatic women include vaginal discharge (92.5%), frothy, foul-smelling discharge (50%), and vaginal wall erythema (10%) [23]. The characteristic features of the infection are present in only about 40% to 50% of patients [24]. Women with *T. vaginalis* may have abdominal pain due to salpingitis or endometritis and postcoital bleeding due to cervicitis [19]. The clinical features associated with trichomoniasis infection have a relatively low positive predictive value because of the frequent occurrence of similar signs and symptoms among women with other STI infections [25]. Fouts et al. [26] in their study conducted on women attending the STI clinic in Georgia found that, if only the clinical features alone were used to diagnose trichomoniasis, about 88% of the infected women would be missed, and 29% women would be falsely diagnosed as having an infection. In our study, vaginal discharge was the most frequently reported symptom in trichomoniasis-positive cases (sensitivity: 81.5 %), followed by vulval itching, lower abdominal pain, and dysuria. However, these symptoms had low positive predictive values for trichomoniasis. Women with concurrent trichomoniasis and other infections were more symptomatic with a higher frequency of reported itching and lower abdominal pain as compared to those with trichomoniasis alone, although this was not statistically significant. Among the clinical signs documented, women presenting with profuse, malodorous, frothy vaginal discharge, and mucopurulent discharge were significantly associated with trichomoniasis. Frothy vaginal discharge was observed in 58% of women in our study and was the most specific sign with a positive predictive value of 100%. The mucopurulent discharge was observed among 38.7% of women. Colpitis macularis, which is a specific sign of *Trichomonas* infection, is detected reliably only by colposcopy and rarely by routine examination [24, 25]. The colpitis macularis sign was not seen in any of the women by naked eye examination in our study, as the colposcopy was not done. Wolner-Hanssen et al. [27] in their study on clinical manifestations of trichomoniasis done on women attending an STI clinic observed that frothy discharge was found in only 8% of women with trichomoniasis and had a specificity of 99% and a PPV of 62%. Colpitis macularis was a highly specific sign (99%) and had a high PPV (90%), but was seen without a colposcope in only 2 of the 52 women who had the finding on colposcopy. Purulent vaginal discharge in their study had a specificity of 76% and a PPV of only 30%.

Investigation

Wet mount: Wet mount using normal saline for the demonstration of trichomoniasis had low sensitivity (65%) and high specificity (100%), PPV 86.7%, NPV 97%, and LR 74.75%. The wet mount was positive in 21 patients and negative in 10 patients in the symptomatic group, positive in 5 patients and negative in 4 patients in the asymptomatic group. All wet-mount negative patients were positive by culture P-value < 0.05 and were significant among both the symptomatic and asymptomatic patients.

Culture of Diamond's medium: Culture proved diagnosis in all 40 cases of trichomoniasis with 100% sensitivity and specificity. The culture had 100% sensitivity and specificity when examined within three days of inoculation. On the 5th day and 7th days, when the sensitivity levels decreased to 97.5% and 77.5%, respectively, the specificity remained at 100%. In a study done by Mohmoud et al. [12] on 450 cases, culture identified 35 (100%) cases whereas wet-mount and PAP smear diagnosed 34.2% and 60% of cases, respectively, which is similar to our study. Another study, done by Cevahir et al. [13], showed that culture was positive in all 40 cases of trichomoniasis (100%), whereas wet mount was positive in only 20 (50%) cases.

COINFECTION

The majority of *Trichomonas*-positive patients in our study were coinfected with bacterial vaginosis (64.3%) in the symptomatic group and about 35.7% in the asymptomatic group with a p-value of 0.285, which was not significant. Mucopurulent cervicitis ($p = 0.821$) and candidiasis ($p = 0.999$) were not statistically significant in our study.

CONCLUSION

The risk factors associated with trichomoniasis in our study were women belonging to low socioeconomic status, who were single, separated from their husbands, having multiple sexual partners, and women whose husbands had extramarital contact. Diagnosis by the culture method is the gold standard investigation for trichomoniasis, with a 100% sensitivity and specificity. The wet mount test had low sensitivity and high

specificity. Also, wet mount failed to identify 14 cases that were positive by culture ($p < 0.05$). Routine use of wet mounts solely for the diagnosis of trichomoniasis may lead to false-negative results, hence culture must be routinely done whenever wet mount is negative, trichomoniasis is strongly suspected, and in all high-risk groups. The clinical characteristics of symptomatic patients are moderate to profuse foul-smelling frothy, mucopurulent vaginal discharge when compared to scanty to moderate odorless mucoid discharge of asymptomatic patients. The relatively high prevalence in the asymptomatic group also indicates the need for routine screening of women in their reproductive age group and include counseling and behavioral changes to reduce the risks of acquiring other STIs.

ACKNOWLEDGMENTS

I would like to acknowledge Prof. Dr. V. Sudha, M.D, D.V, D.D (former Director and Professor, Institute of Venereology MMC) for her constant guidance throughout the study and Prof. Dr. Mangala Adisesh, M.D, (Professor and Head of the Department of Serology MMC) Institute of Venereology for guidance and assistance in Diamond's media preparation.

Study Limitations

The limitation of this study was the lack of the use of molecular diagnostic methods such as PCR and NAAT as diagnostic methods, which have higher sensitivity and specificity.

ABBREVIATIONS

OPD: Outpatient department
STIs: Sexually transmitted infections
PID: Pelvic inflammatory disease
STD: Sexually transmitted disease
VDRL: Venereal Disease Research Laboratory
PPV: Positive predictive value
NPV: Negative predictive value
LR: Likelihood-ratio test
PCR: Polymerase chain reaction
NAAT: Nucleic Acid Amplification Test
TV: Trichomonas vaginalis
BV: Bacterial vaginosis

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the committee responsible on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. World Health Organization. Global Prevalence and Incidence of Selected Curable Sexually Transmitted Infections. Geneva: WHO, 2001.
2. Petrin D, Delgaty K, Bhatt R, Garber G. Clinical and microbiological aspects of Trichomonas vaginalis. Clin Microbiol Rev. 1998;11:300-17.
3. Herath S, Balendran T, Herath A, Iddawela D, Wickramasinghe S. Comparison of diagnostic methods and analysis of socio-demographic factors associated with Trichomonas vaginalis infection in Sri Lanka. PLoS One. 2021;16:e0258556.
4. Cotch MF, Pastorek JG 2nd, Nugent RP, Hillier SL, Gibbs RS, Martin DH, et al. Trichomonas vaginalis associated with low birth weight and preterm delivery. The Vaginal Infections and Prematurity Study Group. Sex Transm Dis. 1997;24:353-60.
5. Quinn TC. Trichomoniasis. In: Tropical and Geographic medicine (warren KS, et al, Eds). NewYork, McGraw Hill,1900:358.
6. Ibáñez-Escribano A, Nogal-Ruiz JJ. The Past, present, and future in the diagnosis of a neglected sexually transmitted infection: Trichomoniasis. Pathogens. 2024;13:126.
7. Ghys PD, Diallo MO, Ettiègne-Traoré V, Yeboué KM, Gnaoré E, Lorougnon F, et al. Genital ulcers associated with human immunodeficiency virus-related immunosuppression in female sex workers in Abidjan, Ivory Coast. J Infect Dis. 1995;172:1371-4.
8. Ter Meulen J, Mgaya HN, Chang-Claude J, Luande J, Mtiro H, Mhina M, et al. Risk factors for HIV infection in gynaecological inpatients in Dar es Salaam, Tanzania, 1988-1990. East Afr Med J. 1992;69:688-92.
9. Johnston VJ, Mabey D. Global epidemiology and control of *Trichomonas vaginalis*. Curr Opin Infect Dis. 2008;21:56-64.
10. Neinstein L, Goldenring J, Carpenter S. Nonsexual transmission of sexually transmitted diseases: An infrequent occurrence. Pediatrics. 1984;74:67-76.
11. Plourd DM. Practical guide to diagnosing and treating vaginitis. Medscape Women's Health. 1997;2:2.
12. Mahmoud MS, Abdel-Aziz SS, El-Sherif EA, Swidan KH. Diagnosis of symptomatic and asymptomatic Trichomonas vaginalis infection by applying one tube nested PCR to vaginal discharge. Department of Parasitology, Faculty of Medicine, Ain Shams University, Cairo 11566. Egypt. J Egypt Soc Parasitol. 1999;29:1031-46.
13. Cevahir N, Kaleli I, Kaleli B. Evaluation of direct microscopic examination, acridine orange staining and culture methods for studies of Trichomonas vaginalis in vaginal discharge specimens. Mikrobiyol Bul. 2002;36:329-35.
14. Chakraborty T, Mulla SA, Kosambiya JK, Desai VK. Prevalence of Trichomonas vaginalis infection in and around Surat. Indian J Pathol Microbiol. 2005;48:542:5.
15. Verteramo R, Calzolari E, Degener AM, Masciangelo R, Patella A. Trichomonas vaginalis infection: Risk indicators among women attending for routine gynecologic examination. J Obstet Gynaecol Res. 2008;34:233-37.
16. Fotinatos N, Warmington A, Walker T, Pilbeam M. Trichomonas vaginalis in Vanuatu. Aust J Rural Health. 2008;16:23-7.
17. Leon S, Konda K, Bernstein K, Pajuelo J, Rosasco A, Caceres CF, et al. Trichomonas vaginalis infection and associated risk factors in a socially-marginalized female population in Coastal Peru. Infect

Dis Obstet Gynecol. 2009;2009:752437.
18. Klinger EV, Kapiga SH, Sam NE, Aboud S, Chen CY, Ballard RC, Larsen U. A community-based study of risk factors for Trichomonas vaginalis infection among women and their male partners in Moshi urban district, Northern Tanzania. Sex Transm Dis. 2006;33:712-8.
19. Haytham, M.A, Nawfal,Y.A. and Ghada, A.A. The prevalence of Trichomonas vaginalis in association with other microorganism among women with vaginal discharge in Mosul. Ann Coll Med Mosul. 2005;31:37-44.
20. Kaur S, Khurana S, Bagga R, Wanchu A, Nancy M. Trichomoniasis among women in North India: A hospital-based study. Ind J Sex Transm Dis AIDS. 2008;29:76-81.
21. Sutton M, Sternberg M, Koumans EH, McQuillan G, Berman S, Markowitz L. The prevalence of Trichomonas vaginalis infection among reproductive-age women in the United States, 2001-2004. Clin Infect Dis. 2007;45:1319-26.
22. Jerajani HR, Melkote S. Sexually transmitted diseases in women and reproductive health. In: Sharma VK, ed. Sexually Transmitted Diseases and HIV/AIDS.2ndedn. India: Viva Books Pvt. Ltd. 2009. p. 56-68.
23. Al-Salihi FL, Curran JP, Wang JS. Neonatal trichomonas vaginalis: Report of three cases and review of the literature. Pediatrics. 1974;53:196-200.
24. Schwebke J, Burgess D. Trichomoniasis. Clin Microbiol Rev. 2004;17:794-803.
25. Domeika M, Zhurauskaya L, Savicheva A, Frigo N, Sokolovskiy E, Hallen A, et al. Guidelines for the laboratory diagnosis of trichomoniasis in East European countries. J Eur Acad Dermatol Venereol. 2010;24:1125-34.
26. Fouts AC, Kraus SJ. Trichomonas vaginalis: Reevaluation of its clinical presentation and laboratory diagnosis. J Infect Dis. 1980;141:137-43.
27. Wølner-Hanssen P, Krieger JN, Stevens CE, Kiviat NB, Koutsky L, Critchlow C, et al. Clinical manifestations of vaginal trichomoniasis. JAMA. 1989;261:571-6.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Original Article

Epidemiological, clinical, and evolutionary profile of patients treated with cryotherapy at the Bamako Dermatology Hospital (HDB) from January 2021 to December 2023

Yamoussa Karabinta[1,2], Kouressi Tall[1], Alfousseyni Niamazié Dissa[1], Ousmane Sylla[1], Mamadou Gassama[1,2], Bakary N'tio Coulibaly[1], Sidy Touré[1], Amadou Dicko[1], Ténin Karambé[3], Chata Traoré[1], Chaka Fomba[4], Chaka Koné[1], Somita Keita[1,2]

[1]Dermatology Teaching Hospital, Bamako, Mali, [2]Faculty of Medicine and Dentistry, Bamako, Mali, [3]Gabriel Toure Teaching Hospital, Bamako, Mali, [4]Referral Health Center, Commune IV, Bamako District, Mali

Corresponding author: Yamoussa Karabinta, MD, E-mail: ykarabinta@yahoo.com

ABSTRACT

Background: Cryotherapy is a widely used dermatological treatment for various benign and premalignant skin conditions. However, data on its application, indications, and outcomes in African settings remain limited. This study aimed to describe the epidemiological, clinical, and therapeutic profile of patients treated with cryotherapy at the Bamako Dermatology Hospital (HDB) from January 2021 to December 2023. **Methods:** A retrospective, descriptive, cross-sectional study was conducted on all patients treated with liquid nitrogen cryotherapy during the study period. Data was collected from consultation and treatment records, covering sociodemographic variables, types of lesions, diagnoses, lesion topography, number of cryotherapy sessions, and treatment outcomes. Data was analyzed using SPSS, version 21. **Results:** Out of 43,380 patients consulted, 676 underwent cryotherapy, giving a hospital frequency of 1.6%. Persons with albinism represented 18% of the sample. The mean age of patients was 15.23 years, with a male predominance (54.7%, sex ratio of 1.3). A steady increase in cryotherapy cases was noted, from 150 patients in 2021 to 234 in 2023. The majority of the patients (96.9%) consulted directly at the hospital. The most common elementary lesion was papular (60.9%), and diffuse lesions accounted for 20.1%. The main indications for cryotherapy were molluscum contagiosum (46%), HPV infections such as cutaneous warts (14.5%), Heck's disease (10.9%), and condyloma (7.1%). In 29.7% of the cases, patients received two cryotherapy sessions, with most molluscum and warts resolving within two sessions, whereas Heck's disease and condyloma required up to six. **Conclusion:** Cryotherapy is effective and well-tolerated in the treatment of various benign dermatoses in our context. However, the lack of long-term follow-up and the absence of certain cryotherapy-responsive conditions highlight the need for prospective studies to better assess recurrence rates and optimize treatment protocols in resource-limited settings.

Key words: Cryotherapy, Epidemiology, Clinical Profile, Dermatology, Bamako

INTRODUCTION

Cryotherapy, or cold therapy, is a non-invasive technique that uses cryogenic agents, such as liquid nitrogen, to freeze tissues at extremely low temperatures, leading to the destruction of targeted cells. Liquid nitrogen cryotherapy is a widely adopted therapeutic option in dermatology for the management of various skin conditions, including precancerous lesions, warts, actinic keratoses, molluscum contagiosum, and other dermatological disorders [1,2]. This method offers several advantages, notably the simplicity and speed

How to cite this article: Karabinta Y, Tall K, Dissa AN, Sylla O, Gassama M, Coulibaly BN, Touré S, Dicko A, Karambé T, Traoré C, Fomba C, Koné C, Keita S. Epidemiological, clinical, and evolutionary profile of patients treated with cryotherapy at the Bamako Dermatology Hospital (HDB) from January 2021 to December 2023. Our Dermatol Online. 2026;17(2):154-159.

Submission: 18.07.2025; **Acceptance:** 11.10.2025

DOI: 10.7241/ourd.20262.2

of the procedure, the absence of mandatory local anesthesia, minimal side effects, low cost, and good patient tolerance [2].

In France, in 2018, 53% of actinic keratosis cases were managed solely by physical treatment—predominantly cryotherapy [3]. Similarly, a survey conducted by the American Society for Dermatologic Surgery and published in 1990 indicated that 87% of participating dermatologists regularly used cryotherapy in their practice [4]. In the United States, Damstra and Van Vloten [5] reported a 92% cure rate among 58 patients with condyloma acuminatum after three months of cryotherapy, while Kuflik [6,7] documented a 97.4% cure rate in eighty patients treated for common warts.

Although cryotherapy has become increasingly popular worldwide due to its effectiveness and ease of use, there remains a significant paucity of studies focusing on its long-term outcomes, especially in resource-limited settings such as those found in Africa. Moreover, data on the specific indications, protocols, patient experiences, and recurrence rates within African populations is scarce.

MATERIALS AND METHODS

Study Setting and Location

The study was conducted at the Bamako Dermatology Hospital (HDB), located in the Djicoroni Para neighborhood. The hospital was established under the 2016-2020 National Hospital Map, by Ordinance No. 2019-010/P-RM dated March 27, 2019, ratified by Law No. 2019-022 of July 3, 2019. It resulted from recent reforms by the Ministry of Health, which led to the division of the National Center for Disease Control (CNAM) and the redistribution of its services between two new structures: one with a public health focus, the National Institute of Public Health (INSP), and the other with a hospital focus, the Bamako Dermatology Hospital (HDB).

The Bamako Dermatology Hospital comprises several clinical departments, the main ones being: the dermatology department, medical imaging department, surgery department (Onco-Surgery and Plastic Surgery), physiotherapy and rehabilitation unit, leprology department, and anesthesiology and intensive care unit. The dermatology department is equipped with ten (10) consultation boxes, one (01) minor surgery room, one (01) cryotherapy room, one (01) treatment room, and two (02) hospitalization wards (one for women, one for men). The hospital has a total of seventeen (17) dermatologists and ten (10) nurses.

Type of Study

This was a descriptive, cross-sectional study conducted over a period of three (3) years, from January 2021 to December 2023.

Study Population

The study included all patients treated with liquid nitrogen cryotherapy at the Bamako Dermatology Hospital (HDB) during the study period.

Case Definition

Cases included all patients who received liquid nitrogen cryotherapy during the study period.

Inclusion Criteria

All patients meeting the case definition were included in the study.

Non-Inclusion Criteria

Patients with incomplete medical records were excluded.

This study aims to evaluate the efficacy of cryotherapy in the treatment of dermatological conditions in our context, while also documenting the practical challenges encountered in its application. The findings of this research will contribute to enriching the knowledge base and potentially guiding clinical practice and policy-making in dermatology across African healthcare settings.

Data Collection

Data was collected from the consultation and treatment registers. A data collection questionnaire was designed, which included information on age, sex, marital status, occupation, place of origin, ethnicity, mode of admission, type of elementary lesion, lesion topography, diagnosis, investigations performed, treatment administered, and clinical outcome.

Data Analysis

Data was entered using Microsoft Word and Excel, version 2016, and analyzed with SPSS, version 21.

Ethical Considerations

This was a retrospective study based on the analysis of routinely collected health data. The data collected was anonymized and did not allow for the identification of patients. No blood samples, other biological specimens, or health products were collected or administered. Overall, the study posed no risk to the patients who had already been seen in consultation at the Bamako Dermatology Hospital.

RESULTS

From January 2021 to December 2023, out of 43,380 patients seen in consultation, we collected 676 patient records, among which 124 were persons with albinism, representing a hospital frequency of 1.6%.

Sociodemographic Data of Patients Without Albinism

During the study period, we observed a progressive increase in the number of patients treated with liquid nitrogen, rising from 150 to 234 (Table 1). Children and students accounted for 65.9% of the patients seen. Male patients accounted for 54.7%, which gives a sex ratio of 1.3.

96.9% of the patients consulted directly at the Bamako Dermatology Hospital (Table 2).

During the study period, we observed a progressive increase in the number of patients treated with liquid nitrogen, rising from 150 to 234. The Dermatology Hospital of Bamako has liquid nitrogen containers (Figs. 1 and 2) for the management of certain dermatological conditions that specifically require this therapeutic approach.

Table 1: Distribution of the sample according to year of consultation.

Year	Number of Patients	Percentage (%)
2021	150	27.2
2022	168	30.4
2023	234	42.4
Total	552	100.0

Table 2: Distribution of the sample according to mode of admission.

Mode of Admission	Number of Patients	Percentage (%)
Came on their own	17	3.1
Referred from a consultation box at the hospital	535	96.9
Total	552	100.00

96.9% of the patients consulted directly at the Bamako Dermatology Hospital.

Clinical Data of Patients

The main conditions that motivated cryotherapy were molluscum contagiosum (46%) and HPV infections, including cutaneous warts (14.5%), Heck's disease (10.9%), and condyloma (7.1%) (Table 3).

Among our cases, two sessions of liquid nitrogen application were reported in 29.7% of the patients (Table 4).

DISCUSSION

We conducted a retrospective study on the epidemiological and clinical profile of patients treated with cryotherapy from January 2021 to December 2023 at the Bamako Dermatology Hospital, aiming to evaluate the effectiveness of cryotherapy in managing dermatoses. A total of 676 patients were selected for the study out of 43,380 patients seen in consultation, representing a hospital frequency of 1.6%. Persons with albinism accounted for 124 cases, representing 18% of the sample. The majority of the patients were male (54.7%), with a mean age of 15.23 years. The year 2023 had the highest patient volume, contributing 42.4% of the study sample. Over 93% of the sample came from the city of Bamako. Students and children were the most represented group. The majority of the patients were referred from consultation boxes within the hospital. Papular lesions were the most common elementary

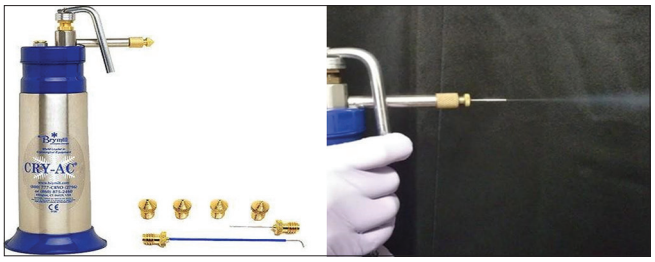


Figure 1: Image of a Cryo Spray, self-pressurized container for liquid nitrogen storage (photo taken at HDB).

Figure 2: The liquid nitrogen containers.

Table 3: Distribution of the sample according to etiology.

Type of Dermatosis	Number of Patients	Percentage (%)
Nuchal cheloid acne	11	2
Other	1	0.2
Condyloma	39	7.1
Callus	60	10.9
Dermatosis papulosa nigra (DPN)	13	2.4
Corn	1	0.2
Epidermodysplasia verruciformis (EDV)	6	1.1
Verrucous hamartoma	7	1.3
Actinic keratosis	4	0.7
Lichenification	13	2.3
Heck's disease	60	10.9
Molluscum contagiosum	254	46.0
Molluscum pendulum	1	0.2
Inflammatory linear epidermal verrucous nevus (NEVIL)	2	0.4
Skin wart	80	14.5
Total	552	100.0

Table 4: Distribution of the sample according to the number of sessions.

Number of Sessions	Number of Patients	Percentage (%)
1.0	82	14.9
2.0	164	29.7
3.0	66	12.0
4.0	74	13.4
5.0	75	13.6
6.0	58	10.5
7.0	27	4.9
8.0	5	0.9
11.0	1	0.2
Total	552	100.0

lesion. Molluscum contagiosum represented 46% of treated lesions, followed by HPV infections, including cutaneous warts (14.5%), Heck's disease (10.9%), and condyloma (7.1%). The number of cryotherapy sessions was typically two (29.7% of cases), with two sessions for molluscum contagiosum and cutaneous warts, and six sessions for Heck's disease and condyloma.

Persons with albinism were mainly treated for ephelides (96%), with an undetermined number of sessions in the majority of cases (38.7%). The study also highlighted a relationship between the type of dermatosis treated and the number of cryotherapy sessions, as well as the relationship between the type of dermatosis and the age of the treated patients.

Limitations of the Study

This study was conducted using patient records, which presents a primary selection bias as some data were missing or lacked follow-up information on the patients' outcomes.

Nonetheless, this study provided valuable insights into the effectiveness of cryotherapy in treating dermatoses.

Epidemiological Aspects

Hospital frequency

During our study period, 43,380 patients were recorded, of which 676 were included, yielding a prevalence of 1.6%, with an average of 255.33 patients seen per year.

Sociodemographic Data

Sex

In our study, 54.7% of the patients were male, giving a sex ratio of 1.3. Our results were similar to those of Helina Fikre [8] in Ethiopia, who reported a predominance of males (71.4%). This higher frequency in males could be random since the indication for cryotherapy is similar across both sexes.

Age

Children under ten years of age were a majority (44.7%), with a mean age of 15.23 years. In the Ethiopian study, Helina et al. [8] reported a mean age of 23 years. The predominance of young patients could be explained by the viral nature of the conditions treated with liquid nitrogen in our context, such as molluscum contagiosum and HPV infections, which are highly common in children and are not immunizing diseases [9].

Origin

Patients from Bamako represented 93.5% of the cases. This result can be explained by the fact that the Bamako Dermatology Hospital is located in the city, making it more accessible to local populations.

Profession

Students and children who are not yet of school age were the most represented groups, accounting for 36.8% and 28% of the cases, respectively. The marked disparity in schooling between children living in urban and rural areas is well documented: for example, in Mali, urban children are significantly more likely to be registered for school and to complete primary education compared to their rural counterparts [10].

Clinical Aspects

Mode of admission

In our study, almost all patients came on their own, representing 96.9% of the cases. This could be

explained by the benign nature of most conditions treated with liquid nitrogen.

Sometimes, transfer cases are overlooked due to the absence of transfer forms.

Type of lesion

Papular lesions were the most common elementary lesions, accounting for 60.9% of the cases (Table 5), followed by verrucous keratotic lesions (24.1%) and keratotic lesions (13.8%). This clinical situation is consistent with the conditions encountered, including molluscum contagiosum and HPV infections.

Lesion localization

All parts of the body were affected by lesions (Table 6). The most common locations were diffuse, facial, and acral areas. According to the literature, there is no absolute contraindication regarding the site for liquid nitrogen application. It is considered a good indication for the excision of certain tumors, such as those in the centrofacial area and at the tip of the nose [11,12].

Type of dermatosis

Cryotherapy is indicated for benign and premalignant lesions. In our series, the main indications were infectious lesions, such as molluscum contagiosum (46%), HPV infections, including cutaneous warts (14.5%), Heck's disease (10.9%), and condyloma (7.1%) (Table 6). This result differs from Fraissenet M. [3] in France, where warts were the main indication for cryotherapy in 99.4% of cases.

Table 5: Distribution of the sample according to the type of lesion.

Type of Lesion	Number of Patients	Percentage (%)
Plaque	1	0.2
Keratotic	76	13.8
Macular	2	0.4
Associated lesion	1	0.2
Nodular	3	0.5
Papular	336	60.9
Verrucous	133	24.1
Total	552	100.00

Papular lesions accounted for 60.9% of our sample

Table 6: Distribution of the sample according to lesion topography.

Lesion Topography	Number of Patients	Percentage (%)
Anal	4	0.7
Oral (buccal)	62	11.2
Scalp	12	2.2
Back	1	0.2
Genital	30	5.4
Lower limbs	90	16.3
Upper limbs	52	9.4
Diffuse	111	20.1
Trunk	84	15.2
Face	106	19.2
Total	552	100.0

Diffuse lesions accounted for 20.1% of our sample

The large difference may be explained by the patient recruitment method.

Calluses and Heck's disease each represented 10%. The high demand for cryotherapy can be attributed to the accessibility of the product and ease of use.

However, our study did not include several pathologies such as leishmaniasis, Kaposi's disease, and small cancerous lesions, which are also indications for cryotherapy. In 2017, Sehdev et al. in India reported that the effectiveness of cryotherapy is comparable to electrocoagulation in treating plantar warts, with a short healing time for cryotherapy [13]. In 2016, a meta-analysis showed that cryotherapy is effective in treating leishmaniasis, with similar efficacy to meglumine antimoniate [14]. For cancerous lesions, such as squamous cell carcinoma or basal cell carcinoma, small lesions under 2 cm are considered optimal indications for cryotherapy according to various authors [15].

CONCLUSION

This study has demonstrated the effectiveness and safety of cryotherapy in treating numerous benign dermatoses, such as molluscum contagiosum and HPV infections, including cutaneous warts, Heck's disease, and condyloma. Molluscum contagiosum and cutaneous warts were cured after two sessions of application, while Heck's disease and condyloma generally required six sessions for treatment. However, the lack of long-term follow-up for our patients and the absence of certain pathologies responsive to cryotherapy in our study should be considered for future research.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. Andrews MD. Cryosurgery for common skin conditions. Am Fam Physician. 2004;69:2365-72.
2. Kuflik EG. Cryosurgery updated. J Am Acad Dermatol. 1994;31:925-44.
3. Uhlenhake EE, Weeks PM, Jacobs MA, Hairston JG, Stoff BJ, Stulberg DL. Epidemiology and management of actinic keratosis in France: General population survey (REAKT). J Eur Acad Dermatol Venereol. 2009;23:163-71.
4. Hanke CW, Bailin PL. Tendances actuelles dans la pratique de la chirurgie dermatologique. J Dermatol Surg Oncol. 1990;16:130-1.
5. Damstra RJ, van Vloten WA. Cryothérapie dans le traitement du condylomata acuminata: une étude contrôlée de 64 patients. J Dermatol Surg Oncol. 1991;17:273-6.
6. Kuflik EG. Indications spécifiques pour la cryochirurgie de l'unité unguéale: kystes myxoïdes et verrues péri-unguéales. J Dermatol Surg Oncal. 1992;18:702-6.
7. Kuflik EG. Traitement cryochirurgical des périonguliers. J Dermatol Surg Oncol. 1984;10:673-6.
8. Fikre H, Mohammed R, Atinafu S, van Griensven J, Diro E. Clinical features and treatment response of cutaneous leishmaniasis in North-West Ethiopia. Trop Med Int Health. 2017;22:1293-301.
9. Paudel V, Parajuli S, Chudal D. Cutaneous leishmaniasis in Nepal: An emerging public health concern. Our Dermatol Online. 2020;11:76-8.
10. République du Mali, Gouvernement du Mali. Stratégie de Réduction de la Pauvreté et de Croissance (GPRSP), 2009–2011*:* Rapport de progrès 2011. Bamako: Présidence du Conseil des Ministres/ Ministère de l'Économie et des Finances; 2011. §258.
11. Biro L, Coll J. Cryosurgery in dermatology. J Am Acad Dermatol. 1982;6:10429.
12. Lair G, Marie I, Cailleux N, Blot E, Boullié MC, Courville P, et al. [Langerhans histiocytosis in adults: Cutaneous and mucous lesion regression after treatment with thalidomide]. Rev Med Interne. 1998;19:196-8.
13. Singh S, Neema S. Comparison of electrosurgery by electrodessication versus cryotherapy by liquid nitrogen spray technique in the treatment of plantar warts. Med J Armed Forces India. 2020;76:156-60.
14. López-Carvajal L, Cardona-Arias JA, Zapata-Cardona MI, Sánchez-Giraldo V, Vélez ID. Efficacy of cryotherapy for the treatment of cutaneous leishmaniasis: Meta-analyses of clinical trials. BMC Infect Dis. 2016;16:360.
15. Saurat J-H, Lachapelle J-M, Lipsker D, Thomas L, Borradori L. Dermatologie et infections sexuellement transmissibles. 6e éd. Paris : Elsevier Masson; 2017. ISBN : 978-2-294-74649-9.

Source of Support: This article has no funding source. Yes (This article has no funding source.)

Conflict of Interest: The authors have no conflict of interest to declare. Yes (The authors have no conflict of interest to declare.)

Our Dermatology Online

Original Article

Nevi in children: Epidemiological, clinical, and dermoscopic profiles and epidemiological-clinical-dermoscopic correlations

Oumaima Bouraqqadi[1], Meryem Soughi[1,2], Sokaina Chhiti[1,2], Zakia Douhi[1,2], Sara Elloudi[1,2], Hanane Baybay[1,2], Fatima Zahra Mernissi[1,2]

[1]Dermatology Department, Hassan II University Hospital, Fez, Morocco, [2]Dermatology Department, University Hospital Hassan II, URL CNRST N15, Human Pathology, Biomedicine, And Environment Laboratory. Faculty of Medicine, Pharmacy, And Dental of Fez, Sidi Mohamed ben Abdellah University, Fez, Morocco

Corresponding author: Oumaima Bouraqqadi, MD, E-mail: Bouraqqadi.oumi@gmail.com

ABSTRACT

Background: Melanocytic nevi are frequently encountered benign cutaneous lesions that develop when altered melanocytes cluster together. Their prevalence, morphology, and dermoscopic patterns vary according to age, phototype, and anatomical location. While extensively studied in Caucasian populations, data on nevi in non-Caucasian pediatric populations remains limited. **Objective:** The objective was to determine the epidemiological, clinical, and dermoscopic characteristics of melanocytic nevi in Moroccan children and establish epidemiological–clinical–dermoscopic correlations. **Methods:** This was a retro-prospective, cross-sectional, analytical investigation of 850 melanocytic nevi collected from 264 pediatric patients aged 0–16 years. Epidemiological, clinical, and dermoscopic data was collected. Nevi were classified into congenital and acquired types and further categorized based on histological and dermoscopic patterns. All statistical tests were conducted using SPSS, version 23, with a significance threshold of $p < 0.05$. **Results:** More than half of the patients (56.4%) were between 11 and 16 years, with a predominance of phototype IV skin (61.3%). A family history of nevi was reported in 55.8% of the cases, and sun exposure in 64.7%. Nevi were most frequently located on the head and neck (34%) and trunk (32.8%). Acquired nevi were predominantly junctional (76.78% on the head and neck, 53.4% on the limbs), while dermal and compound nevi were more common on the trunk. Dermoscopic analysis showed a predominance of the reticular pattern in junctional nevi (82.6%) and the globular pattern in dermal nevi (71.1%). Congenital nevi displayed diverse features, including hair (76.1%), perifollicular white spots (67.4%), and a cobblestone pattern (13%). Statistical analyses showed significant associations between nevus type, anatomical location, and dermoscopic patterns ($p < 0.05$). **Conclusion:** Our study provides a comprehensive overview of melanocytic nevi in Moroccan children, highlighting their epidemiological, clinical, and dermoscopic characteristics. The findings emphasize the influence of phototype and anatomical location on nevus presentation and dermoscopic patterns. These findings contribute to our knowledge of nevi in North African populations and may help refine diagnostic approaches and surveillance protocols for pediatric patients.

Key words: Child, Dermoscopy, Nevus, Pigmented, Skin Neoplasms

INTRODUCTION

Nevi, previously known as pigmented or melanocytic nevi, are benign tumors formed by the clustering of altered melanocytes (known as "nevocytes") at the dermo-epidermal junction [1]. Such clusters, known as nests, differentiate nevi from normal melanocytes. Common in children, nevi are a frequent reason for

How to cite this article: Bouraqqadi O, Soughi M, Chhiti S, Douhi Z, Elloudi S, Baybay H, Mernissi FZ. Nevi in children: Epidemiological, clinical, and dermoscopic profiles and epidemiological-clinical-dermoscopic correlations. Our Dermatol Online. 2026;17(2):160-166.

Submission: 08.02.2025; **Acceptance:** 26.11.2025

DOI: 10.7241/ourd.20262.3

dermatological consultations. They may be congenital, appearing at birth, or acquired, developing later in life. Clinically, nevi appear as macules, papules, or pigmented plaques with symmetrical borders and a homogeneous color, ranging from pinkish orange in lighter skin tones to brown or black in darker phototypes [2,3].

Melanocytic nevi are especially common in children, particularly among Caucasians who typically present with 15 to 30 nevi, while individuals of African, Asian, or Native American heritage tend to exhibit fewer, averaging 5 to 10 nevi by the end of their first decade [4] Several epidemiological factors influence the development of nevi [5]. Age plays a key role, as dermoscopic patterns shift over time: Globular patterns dominate in pre-pubertal years, while reticular-homogeneous patterns are more common during puberty and adulthood [6]. Race also impacts prevalence, with nevi being more frequent in Caucasians and rarer in sub-Saharan African and Asian populations [4,5]. Additionally, phototype influences nevus characteristics, with lighter-skinned individuals typically developing lighter brown, hypopigmented nevi, and darker-skinned individuals exhibiting darker, hyperpigmented, reticular patterns [4,7]. Other factors, including sun exposure, immunosuppression, and family history, may affect nevi characteristics, size, and color [6].

The diagnosis of nevi is primarily clinical, although some lesions may closely resemble melanomas. Clinically, nevi are classified as congenital or acquired, and histologically, they are subdivided into junctional, compound, and dermal nevi based on where the melanocytic clusters are in the skin [2,3]. Dermoscopy has revolutionized the assessment of melanocytic proliferations by enabling high-resolution, non-invasive visualization of the skin's surface. It provides detailed insights into the structures, colors, and patterns of nevi, facilitating their dermoscopic classification. This technique aids in the precise differentiation of nevus types by identifying characteristic features, such as the reticular, globular, or homogeneous patterns, as well as specific features of nevi in special sites [8]. Dermoscopy not only confirms the benign nature of many nevi yet also helps to detect suspicious changes early, guiding clinicians in deciding when further evaluation, such as a biopsy, may be necessary [9,10].

OBJECTIVES

This study aimed to determine the epidemiological, clinical, and dermoscopic profiles of nevi in children in our context and to establish an epidemic-clinical-dermoscopic correlation of nevi in children.

MATERIALS AND METHODS

Study Design and Patients

We conducted a retro-prospective, cross-sectional, and analytical study involving 850 nevi from 264 patients diagnosed at our department. Epidemiological, clinical, and dermoscopic data was collected, covering data such as demographics and family history of nevi, nevus evolution, functional symptoms, sun exposure, and skin reactions to sunlight.

The participants included in the study were individuals aged 0–16 years, regardless of the reason for consultation, who presented with at least one lesion of any size identified as a melanocytic nevus. The nevus had to be confirmed both clinically and dermoscopically, regardless of its location or whether it was acquired or congenital.

Patients were excluded if they had lesions deemed suspicious and required a biopsy to exclude a malignancy, as well as special types of nevi such as Spitz nevus, nevus spilus, Sutton nevus, and blue nevus.

Diagnosis

The diagnosis was based on clinical and dermoscopic evaluation, with images captured by a single examiner using a DermLite 4 paired with a smartphone in both non-polarized and polarized light modes, with and without immersion. For the purpose of the study, the participants were categorized into two age groups: 0–10 years and 11–16 years. Two examiners analyzed the images independently, and any discrepancies were resolved by consensus. Dermoscopic patterns were classified into four primary groups: globular (Figs. 1a and 1b), reticular (Fig. 2), homogeneous (Fig. 3), and compound (Figs. 4a and 4b), with the latter comprising combinations of the other patterns, such as globular-reticular, globular-homogeneous, and reticular-homogeneous. Nevi located on the scalp, mucosa, nails, and palmoplantar areas were counted in the overall tally of nevi yet evaluated separately for

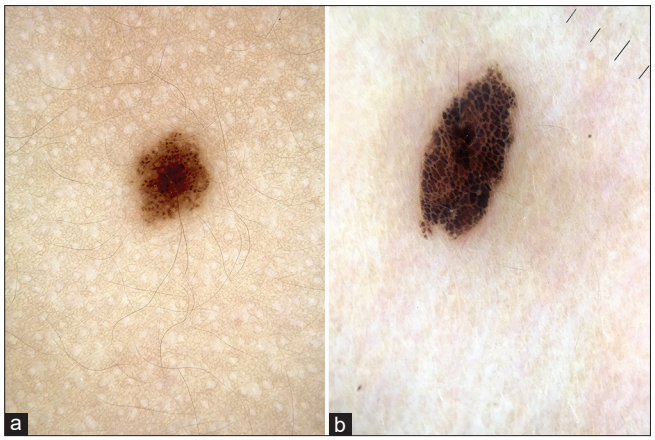


Figure 1: (a) Globular pattern nevi in a 7-year-old child. (b) Cobblestone pattern in an adolescent.

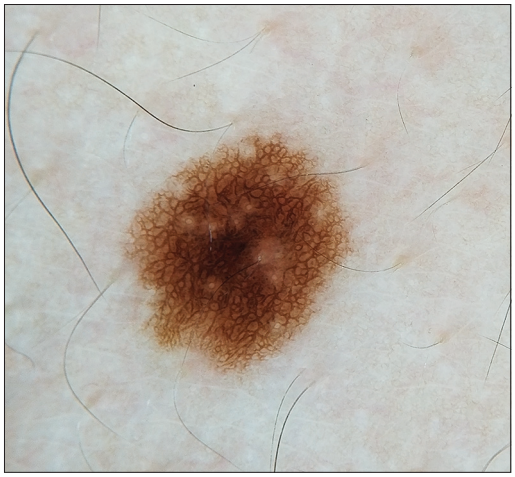


Figure 2: Reticular pattern in a 16-y.o. adolescent.

dermoscopic characteristics when determining the predominant pattern.

Statistical Analysis

Percentage comparison tests served to determine epidemiological, clinical, and dermoscopic characteristics associated with nevi in children. Data was analyzed with SPSS Statistics software, version 23. Correlation analyses between variables—such as age and nevus type, nevus topography and type, and dermoscopic features and nevus type—were conducted using chi-squared and Fisher tests. A p-value below 0.05 was deemed statistically significant, highlighting a meaningful correlation between the variables.

RESULTS

We collected 850 nevi from 264 children, distributed across three age groups: 56.4% were aged 11–16 years,

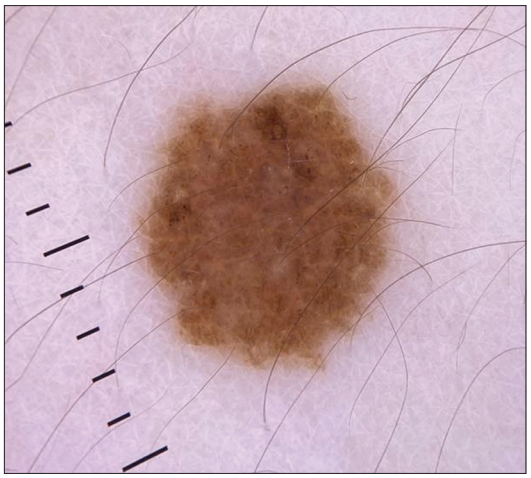


Figure 3: Homogeneous pattern in a 14-y.o. adolescent.

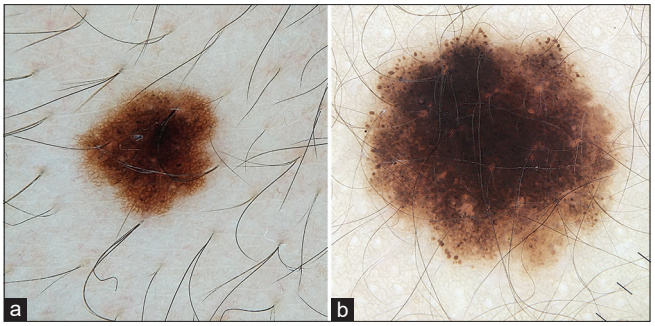


Figure 4: (a) Compound pattern: reticular-globular. (b) Compound pattern: homogeneous center with peripheral globules indicating growth.

28.8% were aged 6–10 years, and 15.2% were aged 0–5 years. A family history of nevi was present in 55.8% of the patients, of which 64% were acquired. Sun exposure was noted in 64.7% of the cases. Recent changes were observed in 31.1% of all nevi, all of which were regular and symmetrical. In terms of lesion distribution, the most frequently affected areas were the head and neck (34%), followed by the trunk (32.8%) and limbs (19.6%). In terms of special site nevi, palmar-plantar nevi were the most frequently observed (45%), followed by scalp nevi (30%), nail nevi (13.5%), and mucosal nevi (11.5%). Most participants had phototype IV skin (61.3%).

The majority of nevi (89%) were smaller than 1.5 cm in diameter, while 9.3% measured between 1.5 cm and 19.5 cm, and 1.5% exceeded 20 cm. Congenital nevi, which constituted 20% of all cases, were primarily located on the trunk (50%), followed by the head and neck (30%) and the limbs (20%). For acquired nevi, the ones located in the head and neck region were predominantly junctional (76.78%), followed by compound nevi (13.67%) and dermal nevi (9.55%).

On the trunk, compound nevi were the most common (45%), with dermal nevi accounting for 33.4% and junctional nevi comprising 21.5%. Acquired nevi on the limbs included 53.4% junctional nevi, 24.4% dermal nevi, and 22.1% compound nevi.

The dermoscopic patterns associated with junctional nevi were predominantly reticular (82.6%), followed by central hyperpigmentation with a peripheral network (16.3%). Among the dermal nevi, 71.1% displayed a globular pattern, 68% a homogeneous pattern, and 21% contained hairs. Mixed or compound nevi commonly exhibited a peripheral reticular network with central globules (62.5%), a multicomponent pattern (27.5%), or a homogeneous pattern combined with a reticular network (5%). Congenital nevi showed various features, with 76.1% presenting hair, 67.4% showing perifollicular white spots, 39.1% having dots, 30.4% exhibiting a globular pattern, 50% displaying a reticular pattern, and 34.8% showing a homogeneous pattern. Additional findings in congenital nevi included a cobblestone pattern in 13%, pseudocysts in 6.5%, and one nevus displaying a blue-white veil.

Palmar-plantar nevi most frequently demonstrated a parallel-furrow pattern (61.1%), followed by a homogeneous pattern (16.4%), a fibrillar pattern (15%), and a lattice-like pattern (10%). Nail nevi consistently exhibited a regular pattern with pseudo-Hutchinson's sign. Four cases involved longitudinal melanonychia covering more than two-thirds of the nail.

Statistical analyses revealed significant age-related and anatomical associations. Junctional nevi were most common in the 11–16 age group, while congenital nevi were more frequent in the 0–10 age group ($p = 0.014$). Junctional nevi were predominantly located on the head and neck (65.1%; $p < 0.0001$). Dermal nevi were primarily found on the trunk (54.1%; $p = 0.032$), as were compound or compound nevi (53.8%; $p = 0.04$) and congenital nevi (52.2%; $p = 0.04$) (Table 1). Dermoscopic correlations for junctional nevi included the reticular pattern (82.6%) and central hyperpigmentation with a peripheral network (14%), both statistically significant. For dermal nevi, significant associations were found with the globular pattern (71.1%), homogeneous pattern (68.4%), and the presence of hair (23.7%) ($p < 0.0001$). Compound nevi significantly correlated with a peripheral reticular network with central globules (62.5%), a multicomponent pattern (27.5%), and a globular peripheral pattern with a central network (5%).

Table 1: Distribution of nevus types by anatomical location.

Nevus Type	Scalp n (%)	Head and neck n (%)	Trunk n (%)	Limbs n (%)	*p* value
Junctional nevi	7 (2.3%)	190 (65.1%)	37 (12.8%)	58 (19.8%)	< 0.0001
Dermal nevi	3 (2,7%)	24 (18.9%)	68 (54.1%)	31 (24.3%)	0.032
Compound nevi	7 (5.3%)	27 (20.4%)	71 (53.8%)	27 (20.4%)	0.04
Congenital nevi	7 (4.3%)	34 (21.7%)	82 (52.2%)	34 (21.7%)	0.04

DISCUSSION

Melanocytic nevi, benign proliferations of melanocytes, are a cornerstone of pediatric dermatology. These lesions are not only highly prevalent yet also hold clinical significance as potential markers of melanoma risk in adulthood [11]. Their occurrence is shaped by a complex interplay of genetic and environmental factors, varying widely across different ages, anatomical locations, and racial backgrounds. Despite their ubiquity, data on their epidemiological and dermoscopic characteristics in non-Caucasian pediatric populations remain limited. Our study bridges this knowledge gap by providing a comprehensive analysis of nevi in a cohort of Moroccan children, offering valuable insights into the interplay between age, phototype, and anatomical location in shaping nevus characteristics.

The age of onset for nevi revealed that 60.8% developed during the first years of life (6 months to 10 years), while 20% developed before 6 months, and 19.2% appeared between the ages of 11 and 16. This finding is consistent with studies showing that nevi counts increase toward the end of the first decade, with a mean count of 15 to 30 in Caucasian children and 5 to 10 in non-Caucasian populations [5,12,13]. Furthermore, 20% of nevi in our cohort appeared before 6 months of age, suggesting that these may be congenital, reinforcing the literature's assertion that early-onset nevi are often congenital in nature [14].

Hereditary factors were significant in our study, with 55.8% of the patients reporting a history of nevi in the family. This supports findings in other studies, which highlight the role of genetics in the development of congenital and acquired nevi [1]. Sun exposure, particularly intense and intermittent exposure, was another commonly reported factor among most of our patients. This may have influenced the number and type of nevi observed, which is in line with studies showing

the impact of sun exposure on nevi development in children [4,15,16]. However, it is worth noting that Yarak et al.'s study of Brazilian schoolchildren did not find a significant correlation between sun exposure and the number of nevi, which may point to environmental or genetic variations across different populations [17].

The clinical presentation of nevi may change dynamically over time, particularly during childhood and adolescence. In our study, 30.1% of nevi were symptomatic, with symmetrical changes, increased palpability, and regression being common features. Studies suggest that such changes in pediatric nevi are part of their natural evolution and do not necessarily indicate a malignancy [4,18,19]. This finding is supported by a Spanish study that reported frequent clinical and dermoscopic changes in melanocytic nevi in children, including the development of new nevi and regression of existing ones over time [20,21].

Regarding phototype, 61.3% of our patients had phototype IV, which reflects Morocco's predominant skin type. Research suggests that children with darker skin, such as those with phototypes III and IV, generally have fewer nevi than those with lighter skin tones [1,22,23]. Additionally, Zalaudek et al. and others have shown that reticular dermoscopic patterns are more frequently observed in individuals with darker skin, whereas globular and homogeneous patterns are more typical in those with lighter skin [21,24,25]. Our results are consistent with these findings, as the majority of nevi in our population exhibited reticular patterns, likely due to increased melanocyte activity and melanin transfer in darker skin types.

Dermoscopic analysis revealed that junctional nevi were the most frequent type of nevi in our sample, accounting for 36.4% of the cases. A significant association was observed between age and nevus type, with junctional nevi more commonly seen in older children (11–16 years) while dermal nevi tended to develop later in life. This pattern was noted in previous studies [24,26]. Additionally, Zalaudek et al. noted that reticular patterns are more frequent in younger children, whereas globular patterns become increasingly prominent with age [25]. Genetic and phenotypic variations may explain these differences in nevus types and patterns across populations, as our study predominantly involved patients with darker skin types.

Topographically, nevi were most frequently observed in the head and neck (34%) and the trunk (32.8%), similarly to findings from studies conducted in Brazil and Spain [21,24]. Junctional nevi were more commonly found on the head and neck, which is consistent with reports from multiple studies [24,27]. In contrast, dermal nevi were predominantly located on the trunk, which is consistent with research indicating that this nevus type is more commonly found in this area [28]. Congenital nevi in our sample were most commonly found on the trunk, further supporting Stefanaki et al.'s findings that the trunk and extremities are the most frequent sites for congenital nevi [29].

Our dermoscopic findings confirmed well-established associations between certain nevus types and dermoscopic patterns. Junctional nevi were primarily linked to the reticular pattern (82.6%), which has been well documented in the literature as a hallmark of junctional melanocytic nevi [6,30]. Dermal nevi were most commonly correlated with the globular pattern (63.4%), a dermoscopic feature reflecting dermal melanocytic activity [24,26]. Compound nevi, characterized by both junctional and dermal components, predominantly exhibited both reticular and globular patterns, reflecting their mixed histopathological structure [6].

In our study, congenital nevi displayed a reticular pattern in 60% of the cases and a globular pattern in 30%, which aligns with previous research identifying these patterns as typical for congenital nevi, especially smaller and medium-sized lesions [25,31]. Additionally, palmoplantar nevi were most often linked to the parallel furrow pattern (68%), a characteristic dermoscopic feature for nevi located on acral areas such as the palms and soles [32].

Our study found a significant correlation between skin phototypes and dermoscopic patterns. Patients with darker phototypes predominantly displayed reticular patterns, aligning with findings from other studies that attribute this to increased melanin activity in darker skin [27]. Symptomatic nevi in our cohort were mostly correlated to the globular pattern, a relationship supported by the literature on symptomatic nevi [29].

This study offers a detailed analysis of melanocytic nevi in a North African pediatric population, offering valuable insights into the epidemiological, clinical, and dermoscopic characteristics of these lesions. Our findings underscore the significant influence of regional and racial factors on nevus characteristics, as evidenced by the high prevalence of phototype IV in our cohort

and its association with darker, hyperpigmented nevi. This observation highlights the need to consider demographic and phenotypic diversity in studies of melanocytic nevi, as such factors influence both the clinical presentation and dermoscopic patterns of these lesions. However, our single-center design suggests that future multicentric studies are warranted to validate these observations and further elucidate the genetic and environmental determinants of nevus characteristics in diverse populations.

CONCLUSION

Our study provides important insights into the epidemiological, clinical, and dermoscopic characteristics of melanocytic nevi in a Moroccan pediatric population. The findings highlight the influence of age, phototype, and sun exposure on nevus development and appearance. The significant correlations between these factors and specific dermoscopic patterns enhance our understanding of nevus management in children. Future research should prioritize longitudinal studies to deepen the understanding of these relationships, particularly for the early detection and treatment of atypical nevi and melanoma in pediatric populations.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. Levy R, Lara-Corrales I. Melanocytic nevi in children: A review. Pediatr Ann. 2016;45:e293-8.
2. Tan J-M, Tom LN, Soyer HP, Stark MS. Defining the molecular genetics of dermoscopic naevus patterns. Dermatology. 2019;235:19-34.
3. Zalaudek I, Longo C, Ricci C, Albertini G, Argenziano G. Classifying Melanocytic Nevi. Nevogenesis, Berlin, Heidelberg: Springer Berlin Heidelberg; 2012, p. 25-41.
4. Schaffer J V. Update on melanocytic nevi in children. Clin Dermatol. 2015;33:368-86.
5. Zhang Y, Ostrowski SM, Fisher DE. Nevi and melanoma. Hematol Oncol Clin North Am. 2024;38:939-52.
6. Zalaudek I, Manzo M, Savarese I, Docimo G, Ferrara G, Argenziano G. The morphologic universe of melanocytic nevi. Semin Cutan Med Surg. 2009;28:149-56.
7. Zalaudek I, Argenziano G, Mordente I, Moscarella E, Corona R, Sera F, et al. Nevus type in dermoscopy is related to skin type in white persons. Arch Dermatol. 2007;143:351-6.
8. Kaushik A, Natsis N, Gordon SC, Seiverling EV. A practical review of dermoscopy for pediatric dermatology part I: Melanocytic growths. Pediatr Dermatol. 2020;37:789-97.
9. Scope A, Marchetti MA, Marghoob AA, Dusza SW, Geller AC, Satagopan JM, et al. The study of nevi in children: Principles learned and implications for melanoma diagnosis. J Am Acad Dermatol. 2016;75:813-23.
10. Shah SA, Jahnke MN. Nevi: When to refer. Curr Opin Pediatr. 2024;36:411-7.
11. Keskinkaya Z, Kaya Ö, Işık Mermutlu S, Garipcan Karaemir H, Oğuz Kılıç S. Clinical and dermoscopic patterns of acquired melanocytic nevi in children and adolescents: A cross-sectional study from Turkey. An Bras Dermatol. 2025;100:22-30.
12. Crane LA, Mokrohisky ST, Dellavalle RP, Asdigian NL, Aalborg J, Byers TE, et al. Melanocytic nevus development in Colorado children born in 1998. Arch Dermatol. 2009;145:148-56.
13. Buendía-Eisman A, Paláu-Lázaro MC, Arias-Santiago S, Cabrera-León A, Serrano-Ortega S. Prevalence of melanocytic nevi in 8- to 10-year-old children in Southern Spain and analysis of associated factors. J Eur Acad Dermatol Venereol. 2012;26:1558-64.
14. Marghoob AA. Congenital melanocytic nevi. Dermatol Clin. 2002;20:607-16.
15. Oliveria SA. Sun exposure and risk of melanoma. Arch Dis Child. 2005;91:131-8.
16. Lee TK, Rivers JK, Gallagher RP. Site-specific protective effect of broad-spectrum sunscreen on nevus development among white schoolchildren in a randomized trial. J Am Acad Dermatol. 2005;52:786-92.
17. Yarak S, Ogawa MM, Hirata S, De Almeida FA. Prevalence of acquired melanocytic naevi in Brazilian schoolchildren. Clin Exp Dermatol. 2009;35:581-7.
18. Cohen B. To biopsy or not to biopsy changing moles in children and adolescents: Are we removing too many pigmented nevi in this age group? Arch Dermatol. 2011;147:659.
19. Siskind V, Darlington S, Green L, Green A. Evolution of melanocytic nevi on the faces and necks of adolescents: A 4-year longitudinal study. J Invest Dermatol. 2002;118:500-4.
20. Scope A, Dusza SW, Marghoob AA, Satagopan JM, Braga Casagrande Tavoloni J, Psaty EL, et al. Clinical and dermoscopic stability and volatility of melanocytic nevi in a population-based cohort of children in Framingham school system. J Invest Dermatol. 2011;131:1615-21.
21. Sosa-Seda IM, Valentín-Nogueras S, Figueroa LD, Sánchez JL, Mercado R. Clinical and dermoscopic patterns of melanocytic nevi in Hispanic adolescents: A descriptive study. Int J Dermatol. 2014;53:280-7.
22. Dulon M, Weichenthal M, Blettner M, Breitbart M, Hetzer M, Greinert R, et al. Sun exposure and number of nevi in 5- to 6-year-old European children. J Clin Epidemiol. 2002;55:1075-81.
23. Synnerstad I, Nilsson L, Fredrikson M, Rosdahl I. Frequency and distribution pattern of melanocytic naevi in Swedish 8–9-year-old children. Acta Derm Venereol. 2004;84:271-6.
24. Piazza CD, Yamada S, Marcassi AP, Maciel MG, Seize MP, Cestari SCP. Dermoscopic patterns of melanocytic nevi in children and adolescents: A cross-sectional study. An Bras Dermatol. 2017;92:340-4.
25. Zalaudek I, Grinschgl S, Argenziano G, Marghoob AA, Blum A, Richtig E, et al. Age-related prevalence of dermoscopy patterns in acquired melanocytic naevi. Br J Dermatol. 2006;154:299-304.
26. Zalaudek I, Schmid K, Marghoob AA, Scope A, Manzo M, Moscarella E, et al. Frequency of dermoscopic nevus subtypes by age and body site. Arch Dermatol. 2011;147:663.
27. Krengel S. Nevogenesis: New thoughts regarding a classical problem. Am J Dermatopathol. 2005;27:456-65.

28. Hofmann-Wellenhof R, Blum A, Wolf IH, Piccolo D, Kerl H, Garbe C, et al. Dermoscopic classification of atypical melanocytic nevi (Clark nevi). Arch Dermatol. 2001;137:1575-80.
29. Stefanaki C, Soura E, Stergiopoulou A, Kontochristopoulos G, Katsarou A, Potouridou I, et al. Clinical and dermoscopic characteristics of congenital melanocytic naevi. J Eur Acad Dermatol Venereol. 2018;32:1674-80.
30. Scope A, Marghoob AA, Dusza SW, Satagopan JM, Agero ALC, Benvenuto-Andrade C, et al. Dermoscopic patterns of naevi in fifth-grade children of the Framingham school system. Br J Dermatol. 2008;158:1041-9.
31. Martin L. Naevus: physiopathologie et conseils pratiques. Réalités Pédiatriques - Mises au point interactives – Dermatol Pédiatr. 2018;221:16-8.
32. Savas Erdogan S, Falay Gur T, Turgut Erdemir AV, Dogan B. Dermoscopic characteristics of acral melanocytic nevi in children and adolescents. Pediatr Dermatol. 2020;37:597-603.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Original Article

Lupus pernio as an important feature of different cutaneous diseases in a case series of 57 cases

Khalifa E. Sharquie, Raed I. Jabbar

[1]Department of Dermatology, College of Medicine, University of Baghdad. Medical City Teaching Hospital, [2]Department of Dermatology, Fallujah Teaching Hospital, Al-Anbar Health Directorate, Anbar, Iraq

Corresponding author: Prof. Khalifa E. Sharquie, MD PhD, E-mail: ksharquieprof@yahoo.com

ABSTRACT

Background: Lupus pernio has traditionally been associated with sarcoidosis, presenting as purple nodules or plaques on the cheeks, ears, and nose. Now, it has been identified alongside different diseases, such as cutaneous tuberculosis, cutaneous leishmaniasis, rosacea, and discoid lupus erythematosus. **Objective:** The objective was to record all dermatological conditions possessing the same clinical features as lupus pernio. **Patients and Methods:** This is a case series, descriptive study conducted from 2014 to 2024 where all patients with different skin diseases associated with lupus pernio were included. This study was mainly concerned with nasal involvement having features of lupus pernio. The diagnosis of each disease was based on clinical features supported by histopathological examination and confirmed by specific investigations. **Results:** Fifty-seven patients with typical clinical features of lupus pernio were included in this study, with their ages ranging from 2 to 60 years, with a mean of 33 years, with 30 (52.63%) females and 27 (47.36%) males. The patients had different skin diseases with typical features of lupus pernio in the form of bulky swelling of the nose with associated dusky erythematous rash on the cheeks together with cutaneous leishmaniasis in 23 (40.35%) patients, rosacea in 16 (28.07%) cases, cutaneous tuberculosis in 7 (12.28%), sarcoidosis in 6 (10.52%), and discoid lupus erythematosus in 5 (8.77%). **Conclusion:** This was the first study recording lupus pernio in association with different skin diseases. All patients presented with a dusky red, bulky nose with nasal mucosa invasion in many cases but, in particular, patients with cutaneous leishmaniasis and tuberculosis. This was in contrast with the dermatological literature in which only sarcoidosis was well defined to be associated with lupus pernio.

Key words: Lupus pernio, Sarcoidosis, Cutaneous leishmaniasis, Histopathology, Cutaneous tuberculosis

INTRODUCTION

Sarcoidosis is a multi-system disorder of unknown etiology that may affect nearly any part of the body [1]. The skin is the most common part of extra-thoracic involvement. Cutaneous sarcoidosis (CS) is present in up to 30% of sarcoidosis cases, while it might be pure CS in 25% [2]. Sarcoid-specific lesions typically exist histologically with a granulomatous alignment in the tissue [3-5].

Lupus pernio is one of the skin manifestations of sarcoidosis [6]. It was first described by Besnier in 1889 who suggested the term *lupus pernio* to describe a patient with multiple purple nodules or plaques of a chronic nature usually affecting the cheeks, nose, ears, and extremities [7].

Lupus pernio tends to be associated particularly with other forms of chronic fibrotic sarcoidosis, including upper respiratory tract sarcoidosis, lacrimal gland, bone cysts, and renal sarcoidosis, and with hypercalcemia and hyperglobulinemia. Lupus pernio tends to persist as lesions for more than two years, seldom to resolve [8]. The facial disfigurement may cause emotional scarring, which may justify aggressive lines of treatment, including plastic surgery [9].

Despite intensive investigation, the cause of lupus pernio and other forms of CS is unknown. Sarcoidosis is

How to cite this article: Sharquie KE, Jabbar RI. Lupus pernio as an important feature of different cutaneous diseases in a case series of 57 cases. Our Dermatol Online. 2026;17(2):167-171.

Submission: 13.05.2025; **Acceptance:** 15.08.2025

DOI: 10.7241/ourd.20262.4

identified as a chronic cell-mediated immune response to an unknown antigen, in which CD4 T-lymphocytes and activated macrophages release cytokines that trigger the formation of granulomas. At least some cases of CS may be due to an unusual host reaction to one or more infective agents, such as *Mycobacterium tuberculosis*, histoplasmosis, and other fungi. Yet, lupus pernio itself is not infectious [10-12].

There are many skin diseases sharing the same clinical features with lupus pernio of sarcoidosis, such as cutaneous tuberculosis (CTB), rosacea, cutaneous leishmaniasis, discoid lupus erythematosus (DLE), granulomatous secondary syphilis, and others, which is poorly documented in the medical literature [3,10,13-15]. Hence, reaching a definite diagnosis can be difficult, and the presence of specific histological features and other laboratory tests of each disease may be the ideal method to reach a definite diagnosis.

As lupus pernio remains not well determined and only well documented in patients with sarcoidosis, the aim of this study was to record all dermatological conditions that possess the same clinical features as lupus pernio.

PATIENTS AND METHODS

This was a case series, descriptive study conducted from 2014 to 2024 in which all patients with different skin diseases associated with lupus pernio were included. Full demographic and clinical features were thoroughly described and analyzed. The study was mainly concerned with nasal involvement having features of lupus pernio. The study was conducted in accordance with principles of the Declaration of Helsinki, and informed consent was taken from each participant or his/her guardian. A thorough history was taken from each patient regarding their name, age, sex, address, disease onset, associated symptoms, and medical and drug history.

A close physical examination was done, including the site of involvement, color, associated signs, and size of the lesion.

The diagnosis of each disease was based on clinical features supported by histopathological examination and confirmed by specific investigations as well as other techniques to detect any forms of systemic involvement, especially in sarcoidosis patients.

RESULTS

Fifty-seven patients with typical clinical features of lupus pernio were included in the study, with their ages ranging from 2 to 60 years, with a mean of 33 years, with 30 (52.63%) females and 27 (47.36%) males. The patients had different skin diseases with typical features of lupus pernio in the form of bulky swelling of the nose with associated dusky, erythematous rash on the cheeks together with the following:

Cutaneous leishmaniasis included 23 (40.35%) patients, 14 (60,87%) males and 9 (39,13%) females; their ages ranged from 2 to 60 years, with a mean of 30 years. 12 (52.17%) cases presented with bilateral, indurated, erythematous cheeks in addition to nasal involvement, while 11 (47.82%) cases presented with an erythematous, bulky nose only. In 9 (39.13%) cases, there was the invasion of the nasal mucosa (Figs. 1a and 1b).

Rosacea was recorded in 16 (28.07%) cases, with 13 (81.25%) females and 3 (18.75%) males; their ages ranged from 30 to 63 years, with a mean of 45 years. All patients presented with multiple papules and pustules distributed on persistent erythema of the central face (Figs. 2a and 2b). Bulky nasal involvement was seen in all cases, but there was no nasal mucosal invasion.

Cutaneous tuberculosis was observed in 7 (12.28%) patients, with their ages ranging from 12 to 60 years, with a mean of 37 years, with 5 (71.42%) males and 2 (28.57%) females. In all cases, the nose was affected, including the nasal mucosa of the nostril presenting with erythematous, papulonodular lesions (Figs. 3a and 3b), apart from one case presenting with

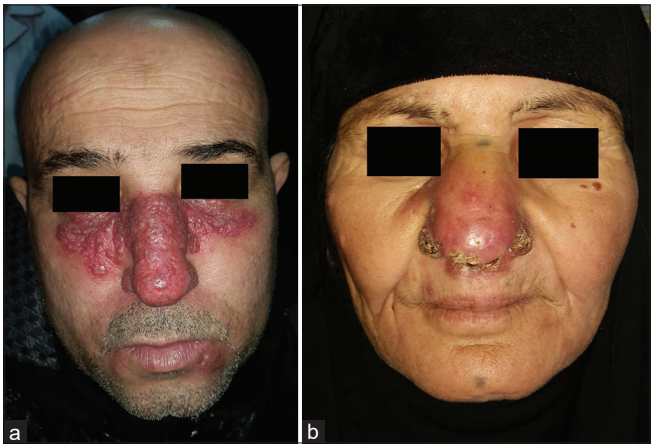


Figure 1: a) Cutaneous leishmaniasis with lupus pernio in a male patient. b) Bulky nose with some invasion of the nasal mucosa in a female patient.

Figure 2: Rosacea with lupus pernio in a) a male patient and b) a female patient.

ulcerative lesions. The histopathological features of CTB ranged from diffuse lymphocytic infiltrate throughout the entire dermis without granuloma formation to well-developed granuloma with central necrosis surrounded by lymphocytes (Fig. 3c).

Sarcoidosis was detected in 6 (10.52%) patients, 4 (66.66%) females and 2 (33.33%) males; their ages ranged from 25 to 60 years, with a mean of 41 years. The lesions presented as erythematous to violaceous papules, nodules, or plaques involving the cheeks, nose, and eyelids. In 2 (33.33%) cases, the lesions extended to involve the extremities and trunk. (Figs. 4a and 4b). All patients demonstrated nasal skin involvement, with 3 (50%) cases exhibiting the invasion of the nasal mucosa. The histopathological assessment of sarcoid lesions showed a marked granulomatous reaction consisting of multiple non-caseating granulomas with sparse lymphocytic infiltrate at the border of the granulomas (so-called naked granuloma). These granulomas were loaded with foamy cells, and there were different types of giant cells (Fig. 4c).

Discoid lupus erythematosus was detected in 5 (8.77%) cases, with 3 (60%) males and 2 (40%) females; their ages ranged from 20 to 45 years, with a mean of 38 years. The skin lesions were characterized by multiple, erythematous, scaly plaques with bulky nasal involvement but without nasal mucosal invasion (Figs. 5a and 5b).

In all patients, physical examination, in addition to appropriate investigations, showed no obvious systemic involvement at diagnosis.

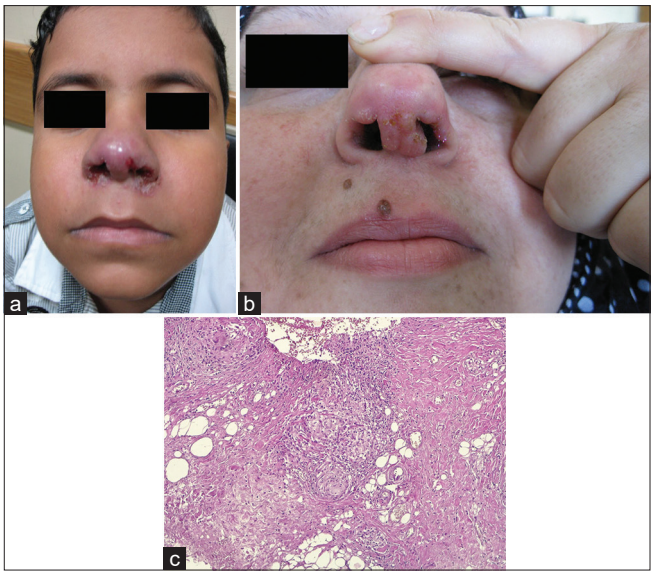


Figure 3: (a and b) Cutaneous tuberculosis with lupus pernio involving the nose with nasal mucosal invasion in both patients. c) H&E-stained section from the patient in Fig. b showing diffuse lymphocytic infiltration with poorly developed granuloma with multiple giant cells (10x).

DISCUSSION

Lupus pernio has traditionally been associated with sarcoidosis, presenting as purple nodules or plaques on the cheeks, ears, and nose [16]. However, in the current study, it was identified alongside diseases such as CTB, cutaneous leishmaniasis, rosacea, and DLE. This raises important questions about the interaction of lupus pernio with other skin diseases.

Lupus pernio in sarcoidosis was detected in 10.52% of the patients and found to produce isolated skin lesions with some invasion of the nostril mucosa in 50% of the cases, but without systemic involvement. These results were in line with another Iraqi study, in which systemic involvement was not an important feature [10], while they differed from previous studies that recorded 40–62% of patients with CS developing systemic involvement [17,18]. The exact etiology beyond this discrepancy could not be well explained. Still, it could be racial, or the early presentation with rapid diagnosis before systemic involvement could be the most important cause of this difference.

Still, cutaneous leishmaniasis is the most common cause (40.35%) of lupus pernio, as reported in this study. This is because cutaneous leishmaniasis is endemic in Iraq and involves mostly the exposed parts of the body, including the face [19,20].

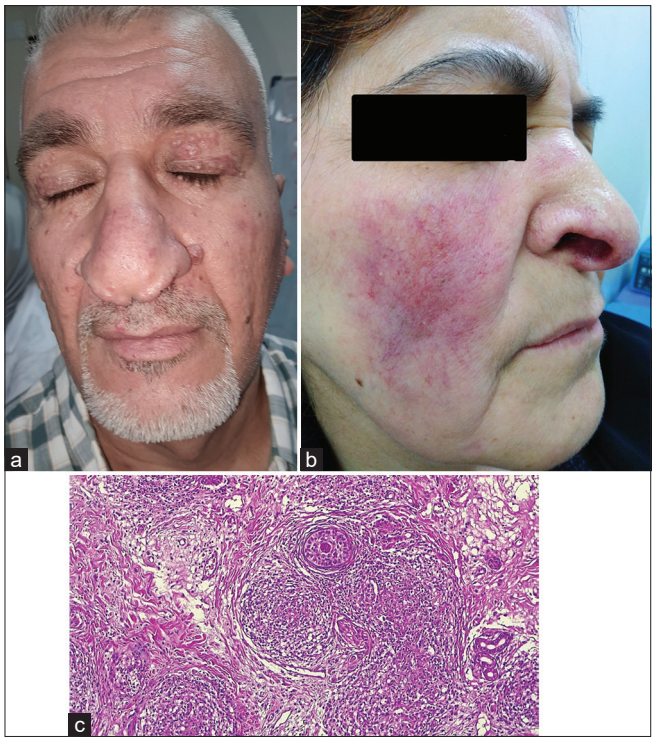


Figure 4: Sarcoidosis with lupus pernio involving the eyelids and nose a) and cheeks and nose b) with nostril mucosal invasion in both cases. c) H&E-stained section from the patient in Fig. a showing multiple non-caseating granulomas (naked granuloma) loaded with foamy cells (10x).

A striking observation in this study was the presence of a lupus pernio lesion with nasal mucosal involvement in patients with cutaneous leishmaniasis and CTB. Nasal mucosal invasion was recorded in 39.13% of cases with cutaneous leishmaniasis and in 100% of cases with CTB, which was in contrast to DLE and rosacea, where such involvement was absent. This suggests that granulomatous infections such as TB and leishmaniasis may share some pathogenic mechanisms with sarcoidosis in eliciting lupus pernio lesions as some cases of sarcoidosis had a previous history of tuberculosis in the same patient [10].

Rosacea was another important condition that was associated with a lupus pernio lesion in the present study. Although rosacea typically manifests as centrofacial erythema and phymatous changes, its ability to present with a lupus pernio lesion is well-documented [3,10]. These findings propose that chronic inflammatory changes in rosacea may induce a tissue reaction resembling lupus pernio of sarcoidosis.

Similarly, DLE was found to exhibit a lupus pernio lesion involving the face and nose resembling lupus pernio of sarcoidosis. Both DLE and sarcoidosis can present with chronic erythematous plaques, and the overlap of skin findings in both conditions was reported in this study.

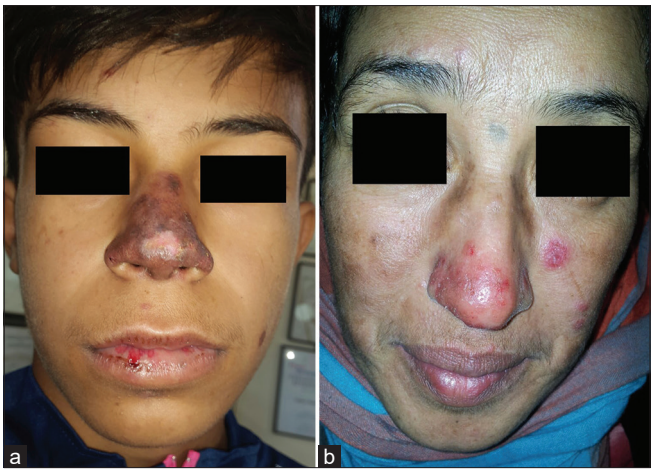


Figure 5: (a and b) Discoid lupus erythematosus with lupus pernio.

In both DLE and rosacea, no nasal mucosal involvement was recorded for unknown reasons, but the most acceptable explanation is that nasal mucosal invasion appears in granulomatous skin diseases such as TB, leishmaniasis, and sarcoidosis rather than in other inflammatory conditions such as rosacea and DLE.

One of the most important implications of our results is the need for pathological confirmation in cases of suspected sarcoidosis. Given that cutaneous diseases like CTB, DLE, rosacea, and leishmaniasis can present with the same cutaneous features, especially when involving the face, a misdiagnosis can lead to mismanagement. This highlights the necessity of taking a biopsy, and microbiological examinations, particularly in endemic areas for TB and leishmaniasis.

CONCLUSION

This was the first study that recorded lupus pernio in association with different skin diseases. All patients presented with a dusky red, bulky nose with nasal mucosa invasion in many cases, but in particular, patients with cutaneous leishmaniasis, tuberculosis, and sarcoidosis. This is in contrast with the dermatological literature, in which only sarcoidosis was reported to be associated with lupus pernio. This study focused on the clinical and histopathological features of lupus pernio, a hallmark feature of sarcoidosis, and its relation to different skin diseases other than sarcoidosis. The results are fascinating and provide insight into the diversity of lupus pernio presentations and clinical

features that are shared with other dermatological conditions. Accordingly, lupus pernio is not a specific feature of sarcoidosis but can be more observed in diverse cutaneous diseases.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. Drent M, Crouser ED, Grunewald J. Challenges of sarcoidosis and its management. New Eng J Med. 2021;385:1018-32.
2. Boch K, Langan EA, Zillikens D, Ludwig RJ, Kridin K. Evaluation of clinical and laboratory characteristics of patients with cutaneous sarcoidosis: A single-center retrospective cohort study. Front Med. 2022;9:980507.
3. Haimovic A, Sanchez M, Judson MA, Prystowsky S. Sarcoidosis: A comprehensive review and update for the dermatologist: part I. Cutaneous disease. J Am Acad Dermatol. 2012;66:e691-9.
4. Grunewald J, Grutters JC, Arkema EV, Saketkoo LA, Moller DR, Müller-Quernheim J. Publisher correction: Sarcoidosis. Nat Rev Dis Primers. 2019;5:45.
5. Ezeh N, Caplan A, Rosenbach M, Imadojemu S. Cutaneous sarcoidosis. Dermatol Clin. 2023;41:455-70.
6. Kerdel FA, Moschella SL. Sarcoidosis: An updated review. Jam Acad Dermatol. 1984;11:1-19.
7. Besnier E. Lupus pernio de la face: synovites fongueses symertriques des extremities superieures. Ann Dermatol Syphilol. 1889;10:333-6.
8. DJ. Sarcoidosis. In: Burns T, Breathnach S, Cox N, Griffiths C, editors. Rooks Textbook of Dermatology. Vol. 3. Massachusetts: Blackwell Science Ltd.; 2010. p. 61.1-21.
9. Yanardag H, Pamuk ON, Pamuk GE. Lupus pernio in sarcoidosis: Clinical features and treatment outcomes of 14 patients. J Clin Rheumatol. 2003;9:72-6
10. Sharquie K, Kubaisi TA, Sharquie IK. Cutaneous sarcoidosis as upsurging mimicking granulomatous diseases. J Pak Assoc Dermatol. 2024:34:698-70.
11. Loke WSJ, Herbert C, Thomas PS. Sarcoidosis: Immunopathogenesis and immunological markers. Int J Chron Dis. 2013;2013:928601.
12. Facco M, Cabrelle A, Teramo A, Olivieri V, Gnoato M, Teolato S, et al. Sarcoidosis is a Th1/Th17 multisystem disorder. Thorax. 2011;66:144-50.
13. Ishak R, Kurban M, Kibbi AG, Abbas O. Cutaneous sarcoidosis: Clinicopathologic study of 76 patients from Lebanon. Int J Dermatol. 2015;54:33-41.
14. Noe MH, Rosenbach M. Cutaneous sarcoidosis. Curr Opin Pulm Med. 2017;23:482-6.
15. Bimbi C. Sarcoidosis mimicking seborrheic dermatitis: Another case of Sherlockian Dermatology. Our Dermatol Online. 2014;5:423-5.
16. Howard A. Non-infectious granulomas. In:. Bolognia J L, Sccaffer J V, Ceroni L. Text book of dermatology, 5th edition. China, Elsevir. 2024;93:1660-67.
17. Paolino A, Galloway J, Birring S, Brex P, Larkin G, Patel A, et al. Clinical phenotypes and therapeutic responses in cutaneous-predominant sarcoidosis: 6-year experience in a tertiary referral service. Clin Exp Dermatol. 2021;46:1038-45.
18. Byrne B, Goh A, Izham NF, Porter E, Field S. Systemic evaluation of cutaneous sarcoidosis: 15-year dermatology experience at University Hospital Limerick. Clin Exp Dermatol. 2022;47:850-7.
19. Sharquie KE, Jabbar RI. Triple therapy for acute and chronic cutaneous leishmaniasis using oral zinc sulfate, oral ketoconazole and topical podophyllin. J Pak Assoc Dermatol. 2022;32:502-9.
20. Sharquie KE, Jabbar RI. Leishmania recidivans could be induced by intralesional infiltration of cutaneous leishmaniasis by sodium stibogluconate, with new therapeutic trial. J Clin Exp Invest. 2022;13:em00788.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Original Article

Micropigmentation in dermatological rehabilitation: Scar camouflage through permanent makeup

Olga V. Yakovleva

Praxis Olga V. Yakovleva, 102 Nasirdin Isanov Street, Bishkek, 720021, Kyrgyzstan

Corresponding author: Olga V. Yakovleva, MD, E-mail: yakovlevs.pro@gmail.com

ABSTRACT

Background: Micropigmentation (permanent makeup) is increasingly recognized not only as a decorative technique but also as a method of dermatological rehabilitation. Pigment implantation into the superficial dermis enables effective camouflage of scars, reducing psycho-emotional stress and improving patients' quality of life. **Objective:** The objective was t o explore the potential applications of micropigmentation for scar correction, to define the indications and limitations of the method, and to evaluate its impact on the psycho-emotional state of patients. **Methods:** This work provides definitions of "micropigmentation" and "scar," reviews the classification of scar types, and describes clinical indications and procedural algorithms. An analysis of current studies and systematic reviews was conducted to assess the efficacy and safety of the method. **Results:** Micropigmentation demonstrated the most significant positive effects in cases of normotrophic and atrophic scars. The patients reported a marked improvement in appearance, enhanced self-esteem, and greater social confidence. Keloid and severe hypertrophic scars require caution and prior dermatological consultation. Successful practice demands specific knowledge, skills in color theory, and precise pigment distribution techniques. Clinical evidence confirms reductions in anxiety and depressive symptoms following the procedure. **Discussion:** Micropigmentation is a minimally invasive yet clinically significant method of dermatological rehabilitation. It serves both aesthetic and psychosocial functions. The absence of standardized training protocols and long-term studies underscores the need for further research. **Conclusion:** Scar micropigmentation deserves recognition as an essential element of comprehensive aesthetic and medical rehabilitation strategies. With interdisciplinary collaboration and high professional competence, the method may substantially improve the accessibility and quality of care for patients with scar tissue alterations.

Key words: Micropigmentation, Permanent makeup, Scars, Camouflage, Aesthetic rehabilitation, Quality of life

INTRODUCTION

Modern aesthetic medicine increasingly faces the challenge not only of improving appearance but also of restoring lost confidence in patients with pronounced skin defects. Among these, scar formations occupy a special place—visible marks on the skin resulting from trauma, surgical interventions, or inflammatory processes. Their presence may become a source of psychological distress, particularly when located in exposed areas of the body.

In recent decades, micropigmentation, the technique of implanting pigment into the superficial layers of the skin, has established itself not only as a tool for long-lasting aesthetic makeup but also as an effective method for visual scar camouflage [1]. The evolution of techniques, the development of specialized pigments, and the growing expertise of practitioners have allowed this approach to be integrated into the system of restorative and dermato-aesthetic care. Medical micropigmentation has been widely adopted in reconstructive practice, particularly after oncological surgery, demonstrating high patient satisfaction and a psychosocial benefit [2].

Despite the variety of therapeutic methods for scar correction, micropigmentation provides a gentle,

How to cite this article: Yakovleva OV. Micropigmentation in dermatological rehabilitation: Scar camouflage through permanent makeup. Our Dermatol Online. 2026;17(2):172-177.

Submission: 26.12.2025; **Acceptance:** 11.02.2025

DOI: 10.7241/ourd.20262.5

non-invasive way to improve the appearance of the affected area by bringing it closer to the natural skin tone. This is especially important for patients unwilling or unable to undergo surgical interventions, or those with contraindications to other corrective methods [3].

The present study aims to explore the potential applications of micropigmentation in treating various types of scars, to define the scope of competence for specialists in this field, and to assess the impact of the procedure on patients' psycho-emotional well-being. The discussion addresses indications, ethical considerations, and the potential for interdisciplinary collaboration among professionals in cosmetology, dermatology, and psychology.

Concept of Micropigmentation

Micropigmentation, also known as permanent makeup, is a technique of implanting pigments (dyes) into the superficial layers of the dermis (papillary layer) (Fig. 1) using specialized equipment, with the aim of correcting or visually improving the appearance of the skin [4,5]. It is applied both for decorative purposes (classic use in the eyebrow, lip, and eyelid areas) and for medical-aesthetic purposes, including areola reconstruction after mastectomy, scar camouflage, and correction of vitiligo [3].

Definition and Classification of Scars

The wound healing process is a complex sequence of well-organized biochemical and cellular events aimed at restoring skin integrity. Any disturbance or imbalance in these processes, often influenced by unfavorable conditions or an excessive connective tissue response, leads to abnormal healing [6]. In other words, a scar is a connective tissue formation that develops at the site of skin injury during the healing process.

In dermatological practice, scars are classified according to the amount of newly formed connective scar tissue (Fig. 2):

1. Normotrophic scars — located at the level of the surrounding skin, soft and minimally noticeable. They typically appear as thin, linear, whitish lines that do not differ significantly in color from healthy skin. In most cases, they result from surgical interventions.
2. Atrophic scars — located within the wound area, below the level of healthy tissue, creating a "tissue deficit" effect. They usually develop after acne or chickenpox, as well as in the form of striae (stretch marks).
3. Hypertrophic scars — thickened, smooth, dense, and elevated above the skin surface, but not extending beyond the wound margins. They form due to excessive collagen accumulation, creating a "tissue surplus" effect.
4. Keloid scars — considered an unfavorable outcome of scar formation. These are irregularly shaped, nodular, vascular-rich, and often painful lesions. They extend beyond the initial wound area, protrude above the skin surface, and are prone to recurrence [5].

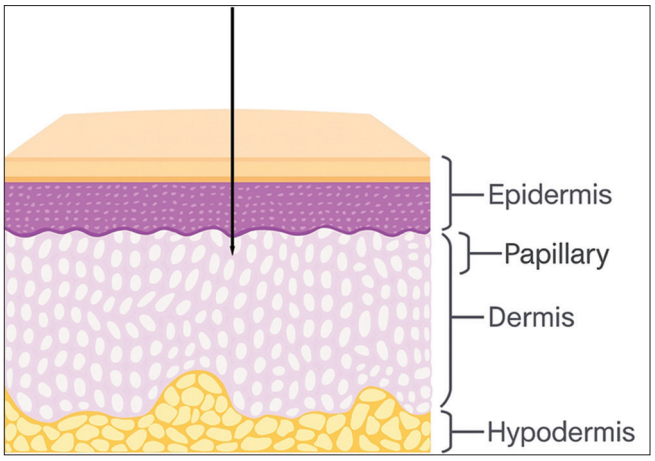


Figure 1: Structure of the skin.

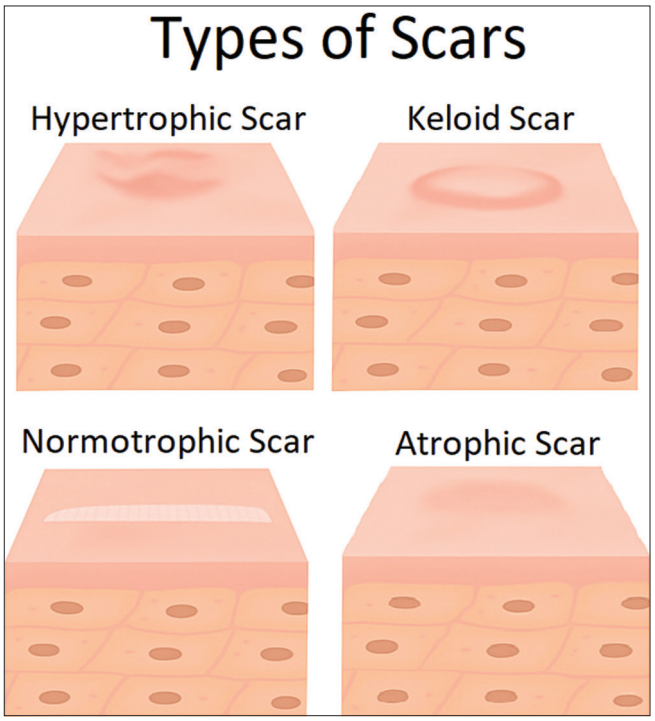


Figure 2: Classification of scars.

Clinical Indications and Limitations

According to current data [1,7], permanent makeup practitioners may safely and effectively work with:

- normotrophic scars,
- atrophic scars,
- hypopigmented areas of scar tissue.

Keloid and pronounced hypertrophic scars are contraindications for the procedure without prior consultation with a dermatologist.

Procedure Technology and Work Specifics

Performing micropigmentation on scar tissue requires a high level of professional qualification, a deep understanding of skin anatomy, and knowledge of the principles of connective tissue healing. Unlike standard permanent makeup zones, a scar represents a morphologically altered skin area with impaired vascularization, reduced elasticity, and often distorted surface relief. This necessitates an individualized approach, adaptation of technique, and careful adjustment of equipment parameters (Fig. 3). In some cases, a multi-stage strategy is advisable: initial preparatory work with the tissue (massage, topical creams), followed by 2–3 sessions with intervals to ensure stable healing and color fixation [5,7].

Figure 3: Professional equipment.

A specialist focusing on scar camouflage must possess the following professional competencies:

- Basic medical knowledge — understanding of skin physiology, scar formation stages, scar types, and risk factors for complications [3];
- Knowledge of dermatological contraindications — the ability to distinguish keloid and active hypertrophic scars, in which the procedure may be unsafe [1];
- Color theory and camouflage skills — accurate pigment selection considering skin phototype, scar depth, and its current coloration. Errors in pigment choice may worsen the visual defect;
- Mastery of shading and pixel pigment distribution techniques — ensuring a natural color transition and avoiding sharp demarcation between the scar and healthy skin.

Protocol for Micropigmentation Procedure in Scar Camouflage

Prior to performing micropigmentation, a mandatory stage is the preliminary patient assessment, aimed at identifying contraindications, reducing the risk of complications, and selecting an individualized technique.

1. Medical History Collection
   - Examination and evaluation of the skin condition in the target area, including the identification of the type of scar. Scar tissue undergoes a maturation process that may take up to 1.5 years.
   - At this stage, the practitioner conducts a detailed assessment of the treatment area, explains the possibilities and limitations of the procedure.
   - Obtaining informed consent from the patient, with a detailed explanation of all procedural stages and expected outcomes.
2. Preparatory Stage
   - The practitioner cleanses the skin and applies an antiseptic.
   - Application of a topical anesthetic cream.
3. Equipment Preparation
   - All working surfaces and containers are covered with protective film to prevent cross-contamination.
   - The professional tattoo device is sealed with a barrier protection and secured in the grip area with a bandage wrap.

- A single-use cartridge (a plastic module containing one or multiple needles with an elastic membrane to prevent pigment or bodily fluids from entering the machine body and motor, thereby eliminating the risk of cross-contamination) is opened in front of the patient. For scar camouflage, needle groupings of 3 to 7–9 in different configurations are used (Round Liner – RL, Flat, Magnum) (Fig. 4).
- Skin-tone pigments are dispensed into single-use caps.

4. Pigment Implantation Procedure

- After a 10–15 minute anesthetic exposure, the practitioner begins pigment implantation using professional equipment. The pigment is deposited into the upper dermal layers with shading or layering movements to cover the required area. Work is performed carefully, continuously monitoring the scar's response to microtrauma. Pigment shades chosen are as close as possible to the patient's natural skin tone.
- Various machine settings can be used, with needle stroke ranging from short (2.8 mm) to long (4.0 mm). The practitioner's skill and preferences play a significant role.
- Three types of pigments may be used: mineral (inorganic, based on metal oxides), hybrid (a mixture of organic and inorganic components), and organic (synthetic, hydrocarbon-based dyes characterized by bright saturation, ease of implantation, and high color density). Hybrid pigments are generally preferred, as they combine the benefits of both types—color stability, ease of implantation, a wide shade range, and longer-lasting results.

5. Post-Procedure Care
   - After the procedure, the practitioner provides the patient with detailed instructions for skin care during the healing period and recommendations to prolong the effect of the treatment.

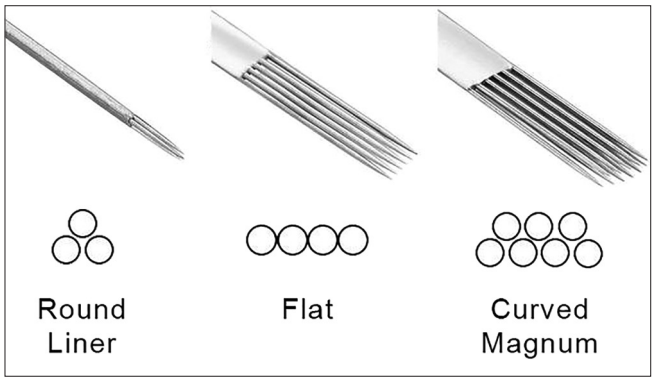


Figure 4: Types of needles used in scar camouflage micropigmentation.

6. Touch-Up After 1–1.5 Months
   - Within 1–2 months, depending on age, the skin fully heals, allowing evaluation of pigment retention and identification of any uneven areas. A correction session one month after the initial procedure is recommended to consolidate results, eliminate irregularities, and achieve the desired color intensity and blending. In some cases, more than one touch-up may be required to obtain the optimal outcome.

MICROPIGMENTATION (PERMANENT MAKEUP) AS A TOOL FOR RESTORING THE PATIENT'S QUALITY OF LIFE

The modern understanding of health encompasses not only physical well-being but also psychological, emotional, and social comfort. In this context, permanent makeup applied for medical and aesthetic purposes extends beyond a simple cosmetic procedure and attains the status of a full-fledged tool for restoring patients' quality of life.

This procedure becomes particularly significant in cases of pronounced aesthetic defects caused by trauma, surgical interventions, burns, dermatological conditions (including vitiligo, acne, and post-acne), as well as in post-oncological rehabilitation (for example, after mastectomy with areola reconstruction). In such clinical scenarios, micropigmentation functions as a form of "social camouflage," allowing patients to reintegrate into society without constant reminders of their past medical conditions.

According to a systematic review by Becker and Cassisi (2021) [3], more than 80% of patients who underwent medical micropigmentation (including scar correction) reported improvements in quality of life, including reduced social anxiety, enhanced self-esteem, and restored confidence in social interactions. These effects were particularly evident among women after breast surgery, patients with facial defects, and young individuals with post-acne scars.

Additional evidence of the psychosocial benefits of permanent makeup is provided by studies [1] demonstrating statistically significant reductions in anxiety and depressive symptoms following aesthetic scar correction. Another striking example is the multinational mixed-method study conducted by Dr. Jerry Tan and colleagues, *"Assessment of*

Psychological Well-being and Social Consequences of Atrophic Acne Scarring," which highlighted the profound impact of scarring on individuals' lives. The study included discussions with 30 adults and a survey of 723 adults with atrophic scars. Results revealed that 31.6%, 49.6%, and 18.8% of respondents reported mild, moderate, and severe/very severe scarring, respectively. Approximately 25.7% of the participants felt less attractive due to their scars, 27.5% expressed embarrassment or shyness, 8.3% reported experiencing verbal or physical abuse related to their scars, and 15.9% believed they had been unfairly dismissed from employment. Alarmingly, 37.5% felt that scars affected how others perceived them, 35.5% avoided public speaking, and 43.2% believed their scars negatively influenced their romantic relationships [8].

Practitioners performing micropigmentation for rehabilitative purposes serve not only as technical specialists but also as mediators between the physical condition of the skin and the patient's internal state. This work requires a respectful, ethical, and individualized approach, reinforcing the interdisciplinary significance of this procedure—at the intersection of aesthetic medicine, psychology, and dermatology.

Thus, in its medical application, permanent makeup is not merely a method of improving appearance but also an effective means of reducing psychosocial burden, supporting patients' return to an active and fulfilling life.

DISCUSSION

The findings, along with a review of current studies, indicate that micropigmentation represents an important component of restorative dermatology and aesthetic medicine. Although the method initially developed within the framework of cosmetic correction, it is increasingly being applied in a clinical context—including in the management of post-traumatic and postoperative scars [9-11].

The effectiveness of the procedure is largely determined by the type of scar, its depth, pigmentation characteristics, and the condition of the surrounding skin. Particularly favorable outcomes are observed when treating normotrophic and atrophic scars, while hypertrophic and keloid scars require heightened caution or complete exclusion from treatment. This highlights the importance of a multidisciplinary approach, in which the permanent makeup practitioner collaborates with dermatologists, surgeons, or rehabilitation specialists [12,13].

Additionally, it should be emphasized that micropigmentation provides not only aesthetic but also psycho-emotional benefits. It helps reduce social anxiety, eliminate visual "reminders" of traumatic experiences, and promotes better social adaptation for patients. This makes the method relevant not only in private practice but also within state-supported medical and social rehabilitation programs. Numerous studies demonstrate that visible scars significantly impair quality of life, leading to social withdrawal, anxiety, and reduced self-esteem [14,15].

Nevertheless, unresolved issues remain, including the need for standardized training protocols, unified safety guidelines, and the development of an evidence base supported by a larger number of clinical cases. Further research is also required to evaluate long-term outcomes, pigment safety, and the interactions of micropigmentation with dermatological and cosmetic procedures.

CONCLUSION

Scar micropigmentation is a modern and clinically significant technique situated at the intersection of aesthetic and restorative medicine. It improves the appearance of the skin, enhances patients' quality of life, and supports their reintegration into society without embarrassment or psychological discomfort.

Scar correction through permanent makeup requires high professional training, ethical practice, and individualized treatment planning. The method demonstrates particular effectiveness in cases of atrophic and normotrophic scars, as well as in hypopigmented areas.

Thus, micropigmentation deserves recognition as an integral part of comprehensive rehabilitation strategies for patients with skin defects, especially when more invasive treatment options are unavailable or undesirable. Its inclusion in clinical protocols and educational programs could significantly improve the accessibility and quality of aesthetic and medical care.

ACKNOWLEDGMENTS

The author expresses gratitude to colleagues and patients who participated in the clinical practice.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

REFERENCES

1. Kerure AS, Kerure Amit S, Marwah M, Wagh Nitika D, Udare S. Micropigmentation in dermatological conditions. Indian Dermatol J. 2023;14:605-10.
2. van der Velden EM, Meesters AA, van der Hulst RRWJ. Medical tattooing after breast reconstruction: A systematic review. J Plast Reconstr Aesthet Surg. 2019;72:458-66.
3. Becker SJ, Cassisi JE. Applications of medical tattooing: A systematic review. Otolaryngol Head Neck Surg Open J. 2021;3:ojab015.
4. Garg G, Thami GP. Micropigmentation: Tattooing for medical purposes. Dermatol Surg. 2005;31(8 Pt 1):928-31.
5. Andreou E, Hatziantoniou S, Rallis E, Kefala V. Why permanent makeup (PMU) is not a lifetime application. Cosmetics (MDPI). 2024;11:160.
6. Prokhorov DV, Shcherbeneva AA, Ngema MV, Ispiryan MB, Kuznetsova MYu. Skin scars: Current concepts of etiopathogenesis, clinical presentation, and diagnosis. Crim Ther J. 2021;2:19-20.
7. Garina AG. Technique of applying permanent makeup for mature skin: Materials and specific features of their use. Int J Human National Scien. 2025;2:101.
8. Tan J, Beissert S, Cook-Bolden F, Chavda R, Harper J, Hebert A, et al. Assessment of psychological well-being and social consequences of atrophic acne scarring: A multinational mixed-methods study. JAAD Int. 2022;6:43-50.
9. De Cuyper C. Permanent makeup: Indications and complications. Clin Dermatol. 2007;25:367-74.
10. Curyło W, Chwaleba K, Klak I, Szerocki J, Milewska-Plis K, Mazur K, et al. LED photobiomodulation: From dermatological care to neuroprotection and pain management – review. Our Dermatol Online. 2025;16:e32.
11. Grudina A, Karalus M, Herian M, Klak I, Bartnik K, Chwaleba K, et al. Skin microbiome disruption and dermatological risk in athletes. Our Dermatol Online. 2025;16:e26.
12. Talwar A, Puri N. A study on scar revision. Our Dermatol Online. 2016;7:155-9.
13. Kouotou EA, Nguena Feungue U, Tounouga DN, Ngoune Madjoukeng AS, Nkoro GA, Sigha BO, et al. Severity of acne and quality of life of patients treated at Cameroonian hospitals (sub-Saharan Africa). Our Dermatol Online. 2023;14:16-22.
14. Serup J, Kluger N, Bäumler W. Tattooed skin and health. Berlin: Springer. 2015.
15. Brown BC, McKenna SP, Siddhi K, McGrouther DA, Bayat A. The hidden cost of skin scars: Quality of life after skin scarring. J Plast Reconstr Aesthet Surg. 2008;61:1049-58.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Original Article

Mucin-secreting cutaneous diseases: Clinical and histopathological study in a series of 84 cases

Khalifa E Sharquie[1], Waqas S. Abdulwahhab[2]

[1]Department of Dermatology, College of Medicine, University of Baghdad. Dermatology Center, Medical City Teaching Hospital, Iraq, [2]Department of Dermatology, Al-Qassimi Hospital, Sharjah, UAE. College of Medicine, University of Sharjah, Sharjah, UAE

Corresponding author: Prof. Khalifa E Sharquie, MD PhD, E-mail: ksharquieprof@yahoo.com

ABSTRACT

Background: Cutaneous mucinoses are a diverse group of skin disorders that involve the accumulation of an abnormal amount of mucin in the skin. The mucin is a jelly-like complex carbohydrate substance, called hyaluronic acid, that occurs normally in minor amounts in the dermis or mid-layer of the skin. This abnormal deposition can be localized or widespread. **Objective:** The objective was to collect the different skin diseases that introduce mucin to the skin during the course of the disease. **Patients and Methods:** This was a case series, descriptive study that was conducted during the period from 2014 to 2024 where all patients with cutaneous mucinosis were collected. Full demographic and clinical evaluation was performed. Biopsies for histopathological assessment were taken. **Results:** Eighty-four cases with cutaneous mucinosis were analyzed, with their ages ranging from 17 to 60 years, with 50 (59.5%) females and 34 (40.5%) males. The following diseases were evaluated: Granuloma annulare was observed in 50 (59.5%) cases, with ages ranging from 17 to 62 with a mean of 34 years, 42 (84%) females and 8 (16%) males; papular mucinoses in 25 (28.8%) cases, with ages ranging from 22 to 60 years, with a mean of 22 years, 19 (76%) males and 6 (24%) females; pretibial myxedema in 4 (4.8%) male patients, with ages ranging from 35 to 61, with a mean of 50 years; scleredema in 3 (3.6%) patients, with ages ranging from 36 to 45 years, with a mean of 40 years, 2 (66.66%) males and one (33.33%) female; follicular mucinosis (alopecia mucinosa) in 2 (2.3%) cases, 27-year-old female and 48-year-old male. A histopathological study of the biopsies showed obvious dermal mucin deposition using H&E stain, apart from the specific histopathology of each disease. **Conclusion:** This study showed different cutaneous diseases with mucin deposition. Some are common, such as granuloma annulare and papular mucinosis, while others are rare, like pretibial myxedema, scleroderma, and follicular mucinosis. They have diverse clinical pictures, but all create a dermal mucin deposition, with the prognosis being variable among patients.

Key words: Mucinosis, Granuloma annulare, Papular mucinosis, Scleromyxedema, Scleredema, Pretibial myxedema

INTRODUCTION

Skin mucinoses are a cluster of disorders in which abnormal amounts of mucin accumulate on the skin in a diffuse or focal pattern [1].

The fibroblasts produce mucin in small quantities as part of the dermal extracellular matrix. It is an amorphous, jelly-like compound made from acid glycosaminoglycans [2]. There are two types of acid glycosaminoglycans: those that are interconnected, as in dermatan sulfate and chondroitin sulfate, and those that are free, as in hyaluronic acid, which is an important component of dermal mucin [3].

The water and salt equilibrium and in the dermis is maintained by mucin, which can hold water up to 1000 times its weight [4]. The presence of either a blue-staining material between collagen bundles or empty spaces within the dermis is an important indicator of

How to cite this article: Sharquie KE, Abdulwahhab WS. Mucin-secreting cutaneous diseases: Clinical and histopathological study in a series of 84 cases. Our Dermatol Online. 2026;17(2):178-186.

Submission: 01.06.2025; **Acceptance:** 14.08.2025

DOI: 10.7241/ourd.20262.6

mucin deposition in routinely stained sections [5]. Stains such as Alcian blue, toluidine blue, and colloidal iron can be used for verification [2].

Pathogenesis

Mucinosis has an unknown pathogenesis. In general, circulating cytokines, such as tumor necrosis factor-alpha, transforming growth factor beta, and interleukin-1, increase fibroblast reproduction and glycosaminoglycan production. Fibroblasts can produce more DNA when exposed to serum from patients with scleromyxedema *in vitro* [3].

Classification

There are two types of cutaneous mucinosis: primary, where mucin deposition is the major histological feature that leads to clinically unique lesions, and secondary, where mucin constitutes a secondary finding. There are two types of primary mucinoses: degenerative-inflammatory and hamartomatous-neoplastic. Degenerative-inflammatory forms are further divided into dermal and follicular forms [4].

Classification of Primary Mucinoses

A. Degenerative-inflammatory mucinoses:
   1. Dermal
      a. Scleromyxedema
      b. Localized variants of lichen myxedematosus (LM):
         - Discrete papular mucinosis
         - Acral persistent papular mucinosis
         - Cutaneous mucinosis of infancy (CMI)
         - Nodular mucinosis
      c. Self-healing cutaneous mucinosis
         - Juvenile type
         - Adult type
      d. Scleredema
         - Non-diabetic (types I and II)
         - Diabetic (type III)
      e. Mucinoses associated with altered thyroid function
         - Localized (pretibial) myxedema
         - Generalized myxedema
      f. Reticular erythematous mucinosis
      g. Papulonodular mucinosis associated with autoimmune connective tissue disease
      h. Digital mucous cyst (myxoid cyst)
      i. Cutaneous focal mucinosis
      j. Miscellaneous mucinoses
   2. Follicular
      - Follicular mucinosis
      - Urticaria-like follicular mucinosis

B. Hamartomatous-neoplastic mucinoses:
   - (Angio)myxoma
   - Mucinous nevus

Classification of Secondary Mucinoses (Associated with Histological Deposition of Mucin)

1. Epithelial mucinosis
   - Basal cell carcinoma
   - *Rare:* Squamous cell carcinoma, verruca, seborrheic keratosis, keratoacanthoma, mycosis fungoides.
2. Dermal mucinosis
   - Granuloma annulare
   - Lupus erythematosus, scleroderma, and dermatomyositis
   - Epithelial tumors (e.g., basal cell carcinoma, eccrine carcinoma)
   - Mesenchymal tumors (e.g., myxosarcoma, myxoid lipoblastoma)
   - Neural tumors (e.g., neurofibroma, lobular neuro myxoma)
   - Other tumors (e.g., cutaneous metastases, mucinous carcinoma of the eyelid)
   - Hypertrophic scarring
   - Obesity-associated lymphedema
   - *Rare:* chronic graft-versus-host disease, cutaneous reactions to interferon, herpes zoster, venous insufficiency.
3. Follicular mucinosis
   - Eczematous dermatoses
   - Mycosis fungoides
   - *Rare:* lupus erythematosus, insect bites, familial reticuloendotheliosis, a side effect of imatinib.

Granuloma Annulare (GA)

An annular pattern of multiple papules characterizes this benign granulomatous skin disorder [5]. Ranges from skin color to red in most cases. Several variants of GA occur, including localized (subdivided into generalized annular plaques and disseminated papular) that affect children, perforating, and subcutaneous [5,6]. The incidence and prevalence of GA have not been assessed in large cohort studies [7]. As of yet, the etiology and pathogenesis of GA are unknown. Histologically, a delayed-type hypersensitivity reaction is most likely [8]. According to studies, interferon-gamma-producing T

helper 1 lymphocytes promote inflammation and tissue destruction by activating macrophages [6].

Although generalized GA may occur as an isolated cutaneous-limited disorder, numerous potential systemic conditions have been described in association, including diabetes mellitus, cancer, thyroid disease, rheumatoid arthritis, lipid abnormalities, HIV, hepatitis B, hepatitis C, BCG vaccination, and COVID-19. GA was recently reported following the COVID-19 vaccine, and it may also be triggered by insect bites, trauma, and herpes zoster [9].

In order to get an accurate diagnosis of GA, a skin biopsy is recommended for clinicopathological correlation. Among the differential diagnoses for GA are nummular eczema, psoriasis, sarcoidosis, necrobiosis lipoidica, tinea, lupus, eruptive xanthomas, leprosy, verruca vulgaris, and granulomatous mycosis fungoides [10,11]. Based on the clinical background, subcutaneous lesions should be distinguished from panniculitis, sarcoidosis, rheumatoid nodules, and infection.

In order to diagnose GA, histopathology presents distinctive features including dermal palisading granulomas with collagen degeneration in the center, mucin, and lymphohistiocytic infiltration [12]. There are four different patterns of histiocytes in GA: interstitial, palisading granulomas, nodule formation similar to sarcoidosis, and a combination of these [11]. In contrast to generalized GA, localized GA usually exhibits more prominent collagen necrosis [12]. A perforating GA differs from other variants in that collagen is expelled through the epidermis [13].

Generally, granuloma annulare does not require treatment because the patches disappear on their own within several months [10]. Sometimes, however, they persist for a long time [11]. An individual lesion may benefit from treatment, but the treatment is not curative [12]. An effective treatment includes topical corticosteroid ointment under occlusion, intralesional steroid injections, cryotherapy or laser ablation, imiquimod cream, and topical calcineurin inhibitors (tacrolimus and pimecrolimus) [13]. In widespread granulomas annulare, systemic steroids, isotretinoin, methotrexate, potassium iodide, dapsone, hydroxychloroquine, pentoxifylline, and phototherapy may be considered [12].

Lichen Myxedematosus (Lichen Myxedematosus)

This is also known as *papular mucinosis*, a rare skin disorder characterized by mucin deposits in the skin [14]. Lichen myxedematosus has localized and generalized forms [1]. Localized scleromyxedema is more favorable than generalized scleromyxedema, which can involve other organs and sometimes be fatal [3].

Lichen myxedematosus and scleromyxedema are often used interchangeably, but scleromyxedema refers to the generalized form [4].

Lichen myxedematosus has no known cause [14]. Monoclonal gammopathy, in which blood levels of an immunoglobulin called paraprotein are abnormally high, is almost always associated with scleromyxedema [15]. IgG-lambda light chain molecules are usually involved [4]. There may be a small increase in plasma cells in the bone marrow [3]. Other cases of scleromyxedema have been linked to cancers of the bone marrow, such as myeloma, lymphoma, and leukemia [1]. There have been several cases of localized lichen myxedematosus associated with HIV infection, hepatitis C infection, toxic oil exposure, and contaminated L-tryptophan [16].

Adults between the ages of 30 and 50 are often affected by scleromyxedema [1]. Both men and women are affected equally [4]. Scleromyxedema is characterized by skin-colored papules on the face, trunk, and extremities [14]. The mucous membranes and scalp are not affected [15]. The papules are 2–3 mm in diameter, waxy, and closely arranged [4]. Their color may change to red-brown over time. The brow may develop deep furrows as the condition progresses [1]. Skin stiffening, fingers (sclerodactyly), and reduced mobility of the mouth can occur, similarly to systemic sclerosis scleroderma, without telangiectasia or calcinosis [15].

Small, firm, waxy papules are confined to a few sites in the localized forms of lichen myxedematosus [15]. The distribution and course of each localized form subtype differ [14]. There is no hardening of the skin, and the disease is usually stable. In the blood, there are no abnormal levels of protein [16].

The main diagnostic test for suspected lichen myxedematosus is a skin biopsy, which shows characteristic pathological signs. Among other

tests, serum and urine protein electrophoresis for paraproteins, thyroid function, and auto-antibodies, including antinuclear factor, may also be performed [3].

It is possible to refer the patient to a hematologist and undergo a bone marrow biopsy if paraprotein is present. In patients with widespread skin involvement, a general physician may assess the involvement of internal organs [14].

There are difficulties and disappointments associated with treating scleromyxedema. Treatments have included isotretinoin, corticosteroids, methotrexate, UVA1 phototherapy, PUVA, intravenous immunoglobulin, plasmapheresis, electron beam radiation, and dermabrasion [17].

Treatments that interfere with the development of precursor cells of the bone marrow have been tried, but they are usually reserved for patients with severe and rapidly progressive diseases [17]. The most common of these therapies are: cyclophosphamide, melphalan, and chlorambucil. As a result of its ability to modulate the immune system, thalidomide has emerged as a potential treatment in recent years. Skin induration (hardening) may be reduced with topical corticosteroid creams and oral isotretinoin [18].

Pretibial Myxedema

This is a form of diffuse mucinosis characterized by the accumulation of glycosaminoglycans in the dermis and subcutis [19]. Glycosaminoglycans, also known as mucopolysaccharides, are complex carbohydrates that maintain tissue hydration and lubrication [3]. A major glycosaminoglycan in pretibial myxedema is hyaluronic acid, which is created by fibroblasts [19].

Pretibial myxedema is also known as localized myxedema, thyroid dermopathy, and infiltrative dermopathy [4]. Lower leg swelling and lumpiness are most commonly seen on the shins (pretibial areas) [20].

Up to 13% of people with severe eye disease suffer from tibial myxedema, which affects 0.5–4.3% of patients with Graves' disease. Also seen in patients with Hashimoto thyroiditis, primary hypothyroidism (underactive thyroid), and euthyroidism (normal thyroid function) [3]. TSH-R antibodies are present in high concentrations in the serum. The most common age group for this condition is between 40 and 60 years of age. It more commonly affects females, with a female-to-male ratio of 3.5:1 [21].

Scleredema

An unknown cause of cutaneous mucinosis. There is a difference between scleredema and scleroderma, in which the skin is fibrotic (morphea and systemic sclerosis).

There is an adult population affected by scleredema [22]. Many people with scleredema have underlying systemic diseases. Diabetes mellitus, hyperparathyroidism, Sjögren's syndrome, rheumatoid arthritis, multiple myeloma, malignant insulinoma, and HIV infection are among them [23].

During a skin biopsy, mucin deposits are found between collagen bundles in the dermis, confirming the diagnosis [23]. Since scleredema is rare, there is no established treatment. PUVA, cyclophosphamide, oral corticosteroids, ciclosporin, UVA1 phototherapy, and electron beam radiation have all shown some benefits [22-24].

Alopecia Mucinosis (follicular mucinosis)

Under a microscope, follicular mucinosis is the appearance of mucin around the hair follicles [25]. Bald patches of skin with prominent hair follicles are characteristic of the condition [1]. In the dermis, mucin looks like stringy, clear, or whitish goo largely composed of hyaluronic acid. A number of other types of mucinosis are described and classified: a primary and acute condition that occurs in children and adolescents (Pinkus type), a primary and chronic condition that affects people over 40 years old, a secondary condition associated with benign or malignant skin conditions, and a rare condition called urticaria-like follicular mucinosis [26].

In the case of alopecia mucinosis, there is no known cause, but it is believed that mucinous material deposits and accumulates in hair follicles and sebaceous glands, resulting in an inflammatory condition that subsequently destroys hair follicle function [27].

Most commonly, alopecia mucinosis affects the face, neck, and scalp, but it can affect any part of the body [25]. It appears as grouped follicular papules within reddened, dry patches or plaques. The diameter of patches or plaques is usually 2–5 cm, but they can

be larger [3]. There may be one lesion at the onset, or multiple lesions may develop over time. The early stages of hair loss are non-scarring and reversible, but in more advanced stages, the hair follicles are destroyed, resulting in scarring [27].

On biopsy, alopecia mucinosa is diagnosed by its clinical appearance and histopathological findings: accumulation of mucin in sebaceous glands and pilosebaceous follicles, keratinous debris within cystic cavities, inflammation, and degeneration of follicular structures [28].

Currently, there is no proven treatment for mucinous alopecia [27]. Primary and acute alopecia mucinosis in children usually resolves spontaneously [26]. In other forms of the disease, spontaneous resolution is rare, making it difficult to assess the effect of treatment [25]. In addition to topical, intralesional, and systemic corticosteroids, oral antibiotics such as minocycline, dapsone, indomethacin, interferons, topical and systemic, photochemotherapy (PUVA), topical nitrogen mustard, radiation, UVA1 phototherapy, and topical bexarotene 1% gel have been tried with limited success [28,29]. If the underlying skin disease is cutaneous T-cell lymphoma, secondary alopecia mucinosis should be treated appropriately [29].

The aim of the present study is to examine the different skin diseases containing mucin in the skin over a ten-year period, trying to find the frequency of these diseases.

PATIENTS AND METHODS

Eighty-four patients complaining of cutaneous mucinosis gathered during the period from June 2014 to 2024 years at the outpatient dermatology unit of Baghdad Teaching Hospital, and a private clinic (KES), in Baghdad, Iraq, were involved in this case-series, descriptive, observational, clinical-histopathological study. The study was conducted following the Declaration of Helsinki. After discussing the nature of the study with all patients, informed consent forms were obtained from all. The close-up picture of the object was taken at the same place with a fixed distance and lighting in the same direction. Also, all patients included in the study accepted the idea of sharing their photos. A full epidemiological and demographic profile was recorded during the study. In order to establish the right clinical diagnosis, a detailed history of the patient was taken along with a thorough physical examination, in addition to their name, age, sex, residence, occupation, duration of the disease, site of involvement, distribution, or number of lesions. Medical and drug history, investigations for underlying diseases such as diabetes mellitus, thyroid disease, malignancy, lipid abnormalities, associated viral infection (HIV, hepatitis B &C), and rheumatoid arthritis were done when suspected. Biopsies for histopathological assessment were taken from the patients.

RESULTS

Eighty-four patients complaining of cutaneous mucinosis were considered in the present work, with their ages ranging from 17 to 60 years, with a mean of 38 years, with 50 (59.5%) females and 34 (40.5%) males. The following diseases were evaluated: Granuloma annulare was observed in 50 (59.5%) cases, with their ages ranging from 17 to 62, with a mean of 34 years, 42 (84%) females, and 8 (16%) males. Papular mucinoses in 25 (29.8%) cases, with ages ranging from 22 to 60 years, with a mean of 22 years, 19 (76%) males and 6 (24%) females. Pretibial myxedema in 4 (4.8%) male patients, with ages ranging from 35 to 61, with a mean of 50 years. Scleredema in 3 (3.6%) patients, with ages ranging from 36 to 45 years, with a mean of 40 years, 2 (66.66%) males and one (33.33%) female. Follicular mucinosis (alopecia mucinosis) in 2 (2.3%) cases, a 27-year-old female and a 48-year-old male. A histopathological study of biopsies showed obvious dermal mucin deposition using H&E stain, apart from the specific histopathology of each disease (Tables 1 – 3) (Figs. 1 – 7).

DISCUSSION

The term *cutaneous mucinosis* refers to a group of uncommon skin disorders where mucin accumulates abnormally in the skin in all of these conditions [1]. Normally, hyaluronic acid occurs as a jelly-like substance in the dermis or mid-layer of the skin as part of the connective tissue [2]. As a result of mucinosis, abnormal deposits may appear locally or all over the body [3]. In terms of severity, they can range from minor cosmetic inconveniences to potentially severe conditions that affect the internal organs. There is no clear understanding of the underlying causes of this group of disorders [4].

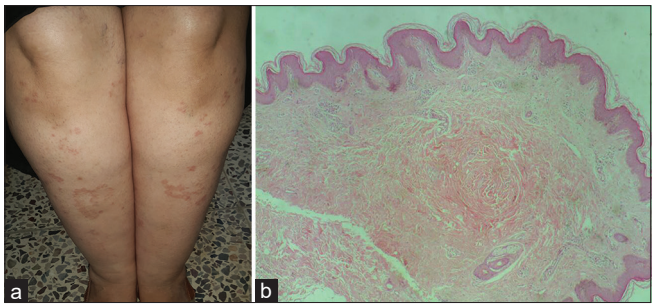


Figure 1: a) Case of granuloma annulare in a 45-year-old female. b) Case of granuloma annulare in a 25-year-old male; mainly mucin deposition and degenerative necrosis of collagen (H&E; 10x).

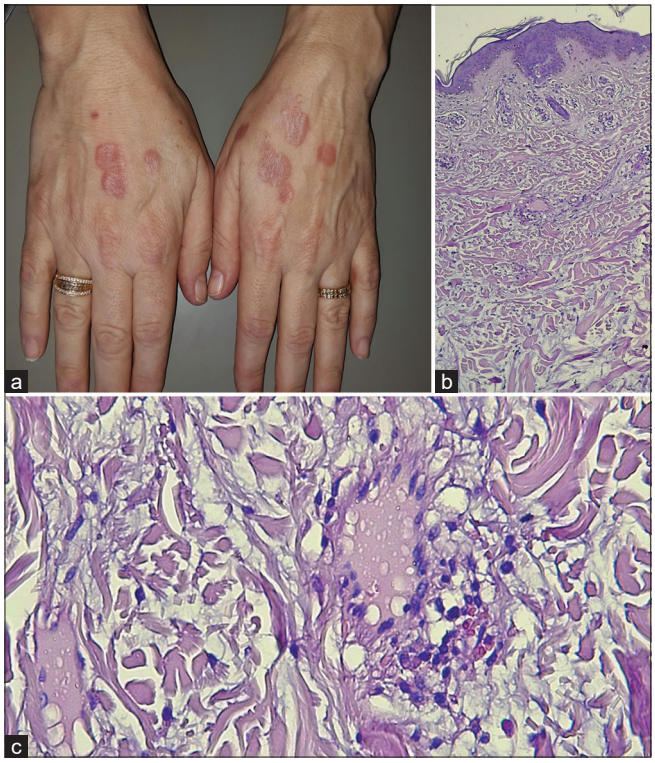


Figure 2: a) 36-year-old female with granuloma annulare. (b and c) Same patient with granuloma annulare showing a dermal interstitial granulomatous reaction with mucin deposition in addition to giant cells (H&E; 10x, 40x).

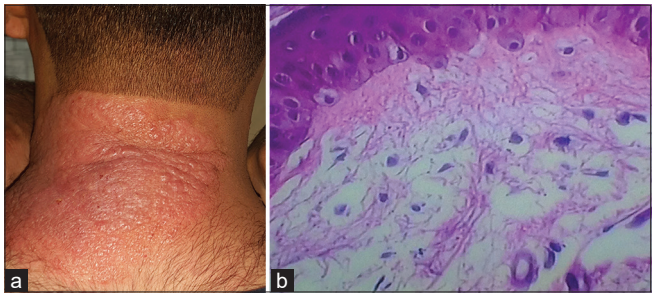


Figure 3: a) Papular mucinosis of the neck with a shiny pea-de-orange appearance. b) Papular mucinosis of the neck showing massive deposition of dermal mucin (H&E; 10x).

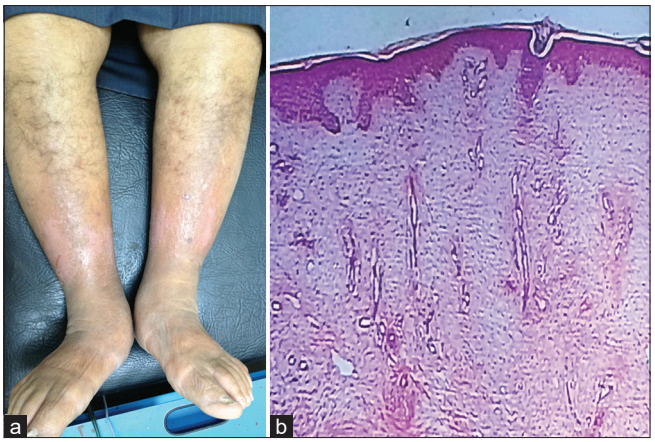


Figure 4: a) Case of pretibial myxedema on both shins. b) Histopathology of pretibial myxedema showing dermal mucinosis (H&E; 10x).

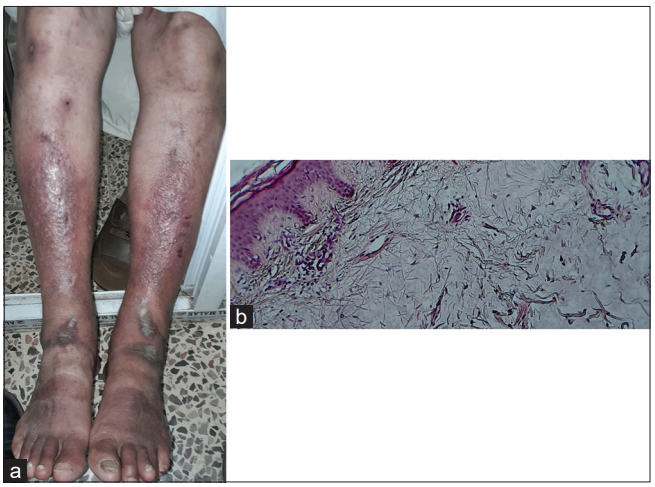


Figure 5: a) 60-year-old male patient with pretibial myxedema of the legs and feet. b) Histopathology of pretibial myxedema showing massive dermal mucin deposition (H&E; 10x).

Table 1: Sociodemographic characteristics of the study sample ($n = 84$)

Characteristic	*n*	(%)
Age (yrs.):		
- Min.	22	
- Max.	60	
Sex:		
- Male	50	(59.5)
- Female	34	(40.5)

The present study reported 84 patients complaining of cutaneous mucinosis, ranging in age from 17 to 60 years, with a mean of 38 years, 59.5% females and 40.5% males. 59.5% of the patients exhibited granuloma annulare; the average age of the patients was 34 years, 84% were females, and 16% were males. Meanwhile, 29.8% of the patients had papular mucinosis, with a mean age of 22 years, 76% were male

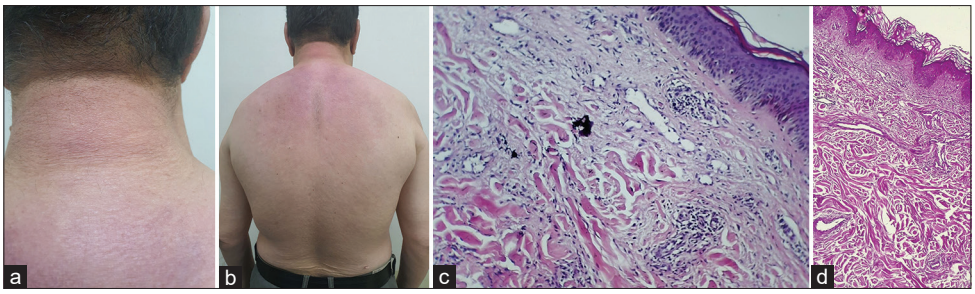


Figure 6: a) Case of scleredema of the upper back. b) Case of scleredema affecting the upper back. c) Histopathology of scleredema showing dermal mucin deposition with dermal lymphocytic infiltrate (H&E; 10x). d) Histopathology of scleredema consisting of mucin deposition with separation of collagen bundles (H&E; 10x).

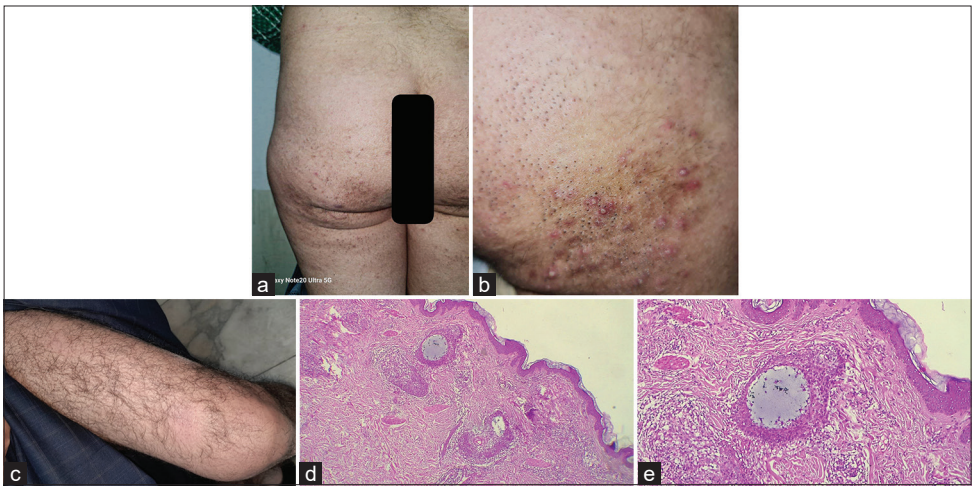


Figure 7: a) Patient with follicular mucinosis. b) Patient with follicular mucinosis. c) Patient with follicular mucinosis. d) Histopathology of follicular mucinosis showing the accumulation of mucin in hair follicles (H&E; 10x). e) Histopathology of follicular mucinosis under higher magnification (H&E; 40x).

Table 2: Disease description of the study sample (n = 84)

Characteristic	*n*	(%)
Disease description:		
- Granuloma annulare	50	(59.5)
- Papular mucinoses	25	(29.8)
- Pretibial myxedema	4	(4.8)
- Scleredema	3	(3.6)
- Follicular mucinosis	2	(2.3)

Table 3: Distribution of the study sample based on sociodemographic characteristics and disease description (n = 84)

Characteristic	Age (yrs.)			Sex	
	Min.	Max.	Mean	Male	Female
Disease description:					
- Granuloma annulare (n = 50)	17	62	34	8 (16%)	42 (84%)
- Papular mucinoses (n = 25)	22	60	22	19 (76%)	6 (24%)
- Pretibial myxedema (n = 4)	35	61	50	4 (100%)	0
- Scleredema (n = 3)	36	45	40	2 (66.6%)	1 (33.3%)
- Follicular mucinosis (alopecia mucinosis) (n = 2)	48	27	37.5	1 (50%)	1 (50%)

and 24% were female. An estimated 4.8% of the male patients, aged 35–61 with an average age of 50, had pretibial myxedema. Scleredema was noticed in 3.6% of the patients, whose age ranged from 36 to 45 years, with a mean age of 40, 66.66% males and 33.33% females. There were two cases of follicular mucinosis (alopecia mucinosis) in 2.3% of the cases, a 27-year-old female and a 48-year-old male. Apart from the specific histopathology of each disease, H&E stain analysis of the biopsies showed obvious dermal mucin deposition.

In a study performed by Sharquie et al., they found that annular granuloma annulare usually presented with typical annular beaded lesions and was classified as generalized (87.23%), localized (8.51%), or profundus (4.25%). Meanwhile, some patients had only one site affected, such as the scalp and penis. The histopathological picture showed palisading granulomas. In both clinical and histopathological cases, it can mimic numerous granulomatous diseases, but sarcoidosis is the most significant mimic.

In another study conducted by Sharquie et al. [29], a total of nine patients with papular mucinosis were

reported, ranging in age from 20 to 56 years, with a mean age of 35 years, and only one 4-year-old child. There was a female predominance (70% females). The face was the most common site of involvement, but the upper arms and necks were also affected. As a result of the rash, skin-colored or red fleshy papules and plaques were observed, as well as diffuse erythematous orange peel-like forms. In most cases, the rash was asymptomatic. The pathology of the disease revealed diffuse mucin deposition in the dermis [29].

Currently, this study is considered to be the first work that examined the frequency of important cutaneous mucinoses in one collective study.

CONCLUSIONS

This study showed different cutaneous diseases with mucin deposition. Some are common, like granuloma annulare and papular mucinosis, while others are rare, like pretibial myxedema, scleroderma, and follicular mucinosis. They have diverse clinical pictures but all share one pathological feature: dermal mucin deposition. Prognosis is variable among patients depending on the variety of mucinosis.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. Rongioletti F. Mucinoses. In: Rongioletti F, Smoller BR, editors. Clinical and pathological aspects of skin diseases in endocrine, metabolic, nutritional and deposition disease. New York: Springer; 2010. p. 146-7.
2. Warner TF, Wrone DA, Williams EC, Cripps DJ, Mundhenke C, Friedl A. Heparan sulphate proteoglycan in scleromyxedema promotes FGF-2 activity. Pathol Res Pract. 2002;198:701-7.
3. Rongioletti F, Rebora A. Mucinoses. In: Bolognia JL, Jorizzo JJ, Schaffer JV, editors. Dermatology. 3rd ed. Philadelphia: Elsevier Saunders; 2012. p. 687-98.
4. Rongioletti F. Mucinosis. In: Bolognia JL, Schaffer JV, Cerroni L, editors. Textbook of Dermatology. 4th ed. China: Elsevier; 2018. p. 742-52.
5. Thornsberry LA, English JC. Etiology, diagnosis, and therapeutic management of granuloma annulare: An update. Am J Clin Dermatol. 2013;14:279-90.
6. Piette EW, Rosenbach M. Granuloma annulare: pathogenesis, disease associations and triggers, and therapeutic options. J Am Acad Dermatol. 2016;75:467-79.
7. Rubin CB, Rosenbach M. Granuloma annulare: A retrospective series of 133 patients. Cutis. 2019;103:102-6.
8. Fayyazi A, Schweyer S, Eichmeyer B, Herms J, Schwarz S, Schulz A, et al. Expression of IFNγ, coexpression of TNFα and matrix metalloproteinases, and apoptosis of T lymphocytes and macrophages in granuloma annulare. Arch Dermatol Res. 2000;292:384-90.
9. Andreu-Barasoain M, Gómez de la Fuente E, Pinedo F, Díaz-Pérez JL, Martínez-Cuesta A, Aramburu JA, et al. Long-lasting interstitial generalized granuloma annulare on sun-exposed areas. Photodermatol Photoimmunol Photomed. 2012;28:216-8.
10. Toro JR, Chu P, Yen TS, LeBoit PE. Granuloma annulare and human immunodeficiency virus infection. Arch Dermatol. 1999;135:1341-6.
11. Kakurai M, Kiyosawa T, Ohtsuki M, Kawashima M. Multiple lesions of granuloma annulare following BCG vaccination: Case report and review of the literature. Int J Dermatol. 2001;40:579-81.
12. Houcke-Bruge C, Delaporte E, Catteau B, Piette F. Granuloma annulare following BCG vaccination. Ann Dermatol Venereol. 2001;128:541-4.
13. Rebellato PR, Carbonar MB, Tabuti NI, Rastelli GJ. Case for diagnosis. Lichen myxedematosus. An Bras Dermatol. 2016;91:842-3.
14. Hadj I, Gallouj S, Meziane M, Mernissi FZ. Discrete papular lichen myxedematosus: A rare entity or an under-diagnosed disease? Pan Afr Med J. 2014;19:180.
15. Baybay H, Lamouaffaq A, Bennani M, Senhaji G, Douhi Z, Elloud S, Rimani M, Mernissi FZ. Atypical scleromyxedema improved by immunoglobulins intravenous. Our Dermatol Online. 2019;10:352-4.
16. Luchsinger I, Coulombe J, Rongioletti F, Haspeslagh M, Dompmartin A, Melki I, et al. Self-healing juvenile cutaneous mucinosis: Clinical and histopathologic findings of 9 patients: The relevance of long-term follow-up. J Am Acad Dermatol. 2018; 78:1164-70.
17. Graves MS, Lloyd AA, Ross EV. Treatment of acral persistent papular mucinosis using an Erbium-YAG laser. Lasers Surg Med. 2015; 47:467-8.
18. Fatourechi V. Pretibial myxedema: Pathophysiology and treatment options. Am J Clin Dermatol. 2005;6:295-309.
19. Shroff A, Simpson G. Cutaneous manifestations of thyroid disease: A case of thyroid-induced myxedema. Case Rep Dermatol Med. 2011;2011:1-2.
20. Doshi DN, Blyumin ML, Kimball AB. Cutaneous manifestations of thyroid disease. Clin Dermatol. 2008;26:283-7.
21. Sreela L, Sharma R, Sudheesh M. An unusual presentation of a rare disorder. Scleredema of Buschke. J Adv Med Dent Sci Res. 2015;3:74-7.
22. Pereira M, Pinheiro RR, Lencastre A, Bártolo E. Scleredema diabeticorum. Dermatol Reports. 2022;14:9477.
23. Bowen AR, Smith L, Zone JJ. Scleredema adultorum of Buschke treated with radiation. Arch Dermatol. 2003;139:780-4.
24. Brown HA, Gibson LE, Pujol RM, Lust JA, Pittelkow MR, Muller SA. Primary follicular mucinosis: Long-term follow-up of patients younger than 40 years with and without clonal T-cell receptor gene rearrangement. J Am Acad Dermatol. 2002;47:856-62.
25. Cerroni L, Fink-Puches R, Bäck B, Kerl H. Follicular mucinosis: A critical reappraisal of clinicopathologic features and association with mycosis fungoides and Sezary syndrome. Arch Dermatol. 2002;138:182-9.
26. Zvulunov A, Shkalim V, Ben-Amitai D, Feinmesser M. Clinical

and histopathologic spectrum of alopecia mucinosa/follicular mucinosis and its natural history in children. J Am Acad Dermatol. 2012;67:1174-81.

27. Rongioletti F, De Lucchi S, Meyes D, Mora M, Gianotti R, Metze D, et al. Follicular mucinosis: A clinicopathologic, histochemical, immunohistochemical and molecular study comparing the primary benign form and the mycosis fungoides-associated follicular mucinosis. J Cutan Pathol. 2010;37:15-9.
28. Geller S. Follicular mucinosis in hematologic malignancies: A review of clinical and pathological features. J Forensic Res. 2024;15:632.
29. Sharquie KE, Jabbar RI. Papular mucinosis (papular lichen myxedematosus): Clinical and histopathological evaluation. Our Dermatol Online. 2021;12:113-9.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Original Article

Follicular mucinosis: A retrospective study

Alexandra Victoria Medina Garduño[1], Mary Jose Santiago Benitez[2], María Elisa Vega Memíje[3], Miren Lorea Cárdenas Hernández[3]

[1]Social Service Medical Intern, General Hospital "Dr. Manuel Gea González," Mexico City, Mexico, [2]Fourth-Year Resident in Internal Medicine, Médica Sur Hospital, Mexico City, Mexico, [3]Dermatologist, General Hospital "Dr. Manuel Gea González," Mexico City, Mexico

Corresponding author: Alexandra Victoria Medina Garduño, MD, E-mail: alee.medinag@gmail.com

ABSTRACT

Background: Follicular mucinosis (FM) is a rare condition characterized by the deposition of mucin in the follicular epithelium. It may be associated with benign or malignant processes (cutaneous T-cell lymphomas). Its recognition is important due to its clinical and histopathological confusion with other dermatoses. Reports in the literature are limited, most corresponding to small series or isolated cases. This study seeks to provide institutional evidence on the clinical and epidemiological presentation of follicular mucinosis in a cohort of patients treated at a referral hospital in Mexico. **Objective:** The objective was to describe the demographic and clinical characteristics of cases of follicular mucinosis treated at the Dr. Manuel Gea González General Hospital between October 1996 and June 2025. **Materials and Methods:** An observational, descriptive, retrospective, cross-sectional study was conducted using a database from the Dermatopathology Department of the Dr. Manuel Gea González General Hospital (Mexico City) for the period between October 1996 and June 2025. **Results:** Nineteen cases of patients diagnosed with follicular mucinosis were identified. Of these, 73.7% (n = 14) were female, and 26.3% (n = 5) were male. The average age of the patients was 31 years (SD: 18.64; range: 5–68 years). The median age was 31 years. The duration of clinical evolution ranged from 15 days to 10 years, with a median of 5 months. Most patients consulted after 1–2 months of evolution (31.6%), followed by 3–5 months (21.1%), while a significant group (47.4%) presented a prolonged evolution (> 6 months) before diagnosis. **Conclusion:** There was a predominance of females and earlier ages of onset than described in international series, with predominant involvement of the face and trunk.

Key words: Follicular mucinosis, Mucinous alopecia, Mycosis fungoides

INTRODUCTION

Follicular mucinosis (FM) is a rare inflammatory dermatosis characterized by the accumulation of mucin in the follicular epithelium and sebaceous glands, leading to degeneration of the outer root sheath and partial or total follicular loss [1]. Pinkus and Braun-Falco first described it in 1957 as mucinous alopecia, although it was later recognized that not all cases present with alopecia [2].

Clinically, it manifests as papules or follicular erythematous infiltrated plaques, which may resemble acneiform, urticarial, or lichenoid conditions, depending on the depth and extent of the infiltrate. The predominant locations include the face, trunk, and extremities [3]. Its course is variable and depends on whether it is primary or secondary. The primary form is generally localized, associated with inflammatory processes, and is typically found in children and young adults, often limited to the head and neck, with a tendency toward spontaneous resolution (Fig. 1).

The secondary form occurs more frequently in adults and is associated with diseases such as systemic lupus erythematosus, insect bites, eczema, trauma, herpes virus infection, alopecia areata, hypertrophic lichen planus, radiotherapy, mycosis fungoides, Sézary

How to cite this article: Medina Garduño AV, Santiago Benitez MJ, Vega Memíje ME, Cárdenas Hernández ML. Follicular mucinosis: A retrospective study. Our Dermatol Online. 2026;17(2):187-191.

Submission: 11.12.2025; **Acceptance:** 20.02.2026

DOI: 10.7241/ourd.20262.7

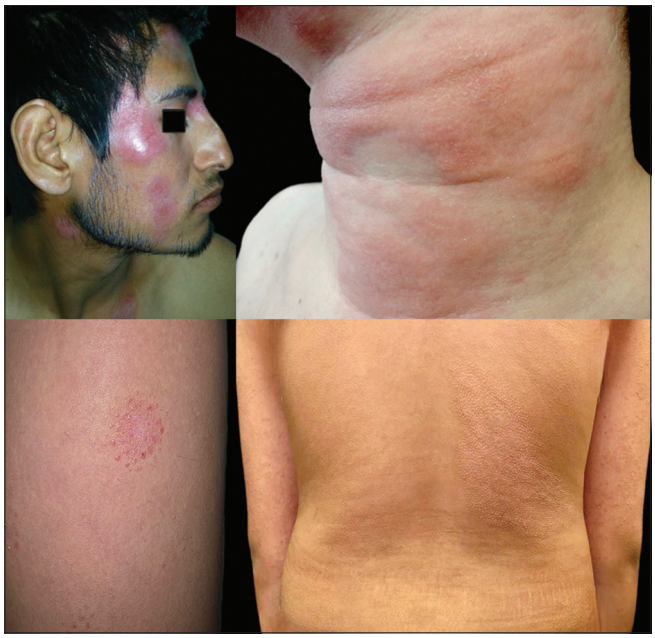


Figure 1: Clinical images of the different morphological aspects that may occur in follicular mucinosis.

syndrome, cutaneous leukemia, cutaneous B-cell lymphoma, and Hodgkin's disease, with a more chronic course and therapeutic resistance [4,5].

Histopathology is essential to confirm the diagnosis and differentiate MF from morphologically similar conditions. Histology reveals a mixed perifollicular inflammatory infiltrate and mucin deposits in the follicular epithelium and sebaceous glands, most visible with stain (Fig. 2) [6,7]. It has been proposed that mucin results from the stimulation of follicular keratinocytes by cytokines released by perifollicular T lymphocytes, which explains the association with the possible progression to cutaneous lymphomas. Immunohistochemical studies have shown the expression of clonal T cells in many cases of primary mucinosis [3]. The clinical evolution of MF shows contrasting behaviors: primary forms tend to remit spontaneously, while secondary forms may persist or progress to cutaneous lymphomas, hence prolonged follow-up and periodic histopathological evaluation are recommended [8].

Multiple therapeutic options have been described, including the use of topical, intralesional, or systemic corticosteroids; calcineurin inhibitors; dapsone; antimalarials; isotretinoin; antibiotics such as minocycline, doxycycline, or erythromycin; non-steroidal anti-inflammatory drugs; as well as imiquimod, interferon alpha, photodynamic therapy, photopheresis, heliotherapy, UVA-1 or NB-UVB phototherapy, pulsed laser therapy, and surgery. Therapeutic results have been variable; to date, no modality has demonstrated complete efficacy, nor has a first-line treatment been established by consensus. Likewise, cases of chronic and disseminated primary MF have been documented in the Mexican population with a persistent course and limited therapeutic response, reinforcing the clinical of this entity [9-12].

Given the scarcity of Latin American reports and the variability observed between international and local series, it is essential to document the characteristics of follicular mucinosis in different populations. Therefore, the present study aims to describe the clinical, demographic, and histopathological behavior of cases of follicular mucinosis treated at the Dr. Manuel Gea González General Hospital (1996–2025), providing evidence on its epidemiological profile in Mexico.

MATERIALS AND METHODS

An observational, descriptive, retrospective, cross-sectional study was conducted using a database from the Department of Dermatopathology at the Dr. Manuel Gea González General Hospital in Mexico City, covering the period from October 1996 to June 2025.

Non-probabilistic convenience sampling was used, based on the availability of cases in the institutional database. The inclusion criteria were patients with a histopathological diagnosis of follicular mucinosis and with complete demographic and clinical data available, including duration of evolution, topography of the lesions, morphology, and symptoms; and patients of any age and sex. Exclusion criteria were inconclusive or questionable histopathological diagnoses and incomplete clinical records. A descriptive statistical analysis was performed using Microsoft Excel 2019®, calculating the mean, median, and mode for continuous variables, and frequencies and percentages for categorical variables.

RESULTS

Nineteen patients diagnosed with follicular mucinosis were identified. Seventy-three point seven percent (n = 14) were women, and 26.3% (n = 5) were men (Fig. 3). The average age was 31 years (SD 18.64; range 5–68 years), with a median age of 31 years.

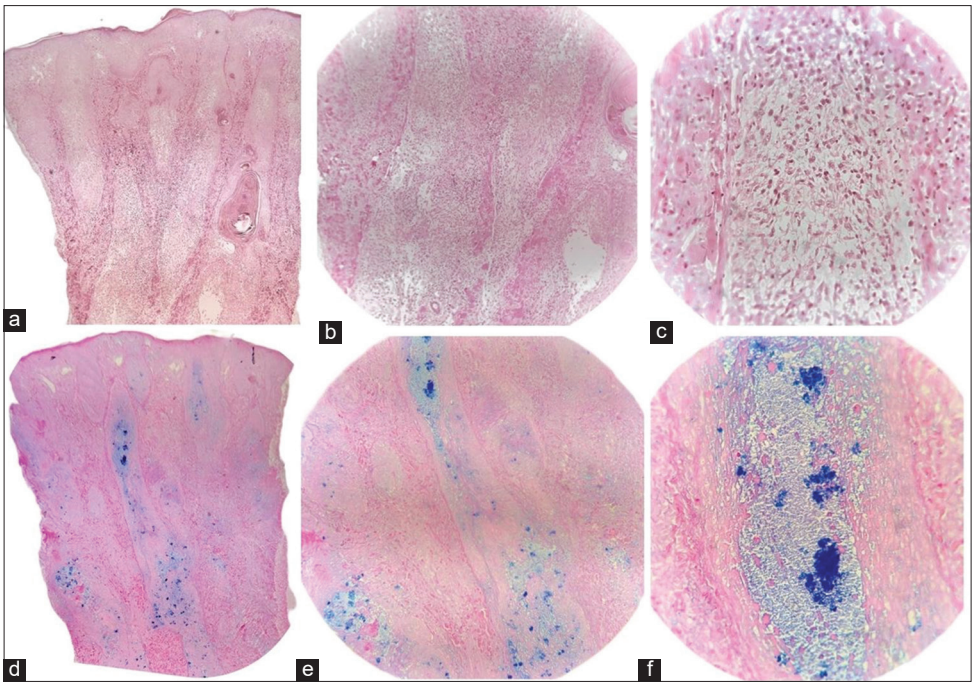


Figure 2: Histological findings of follicular mucinosis. (a-c) Histological appearance of follicular mucinosis (H&E; 10x, 40x, 60x). Hair follicles with separation of keratinocytes due to the deposited material are observed. (d-f) Alcian blue staining (10x, 40x, 60x) showing mucin deposits between keratinocytes.

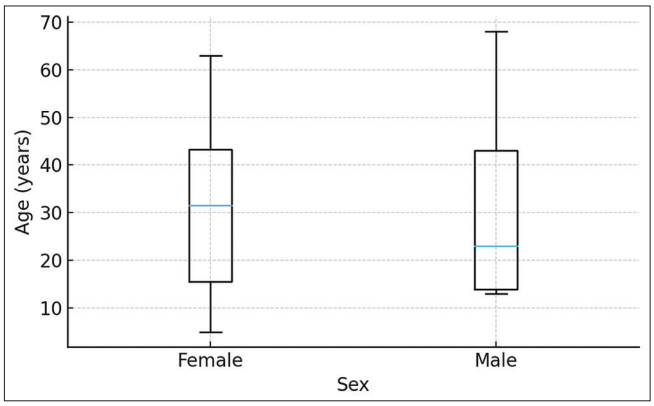


Figure 3: Age distribution according to sex.

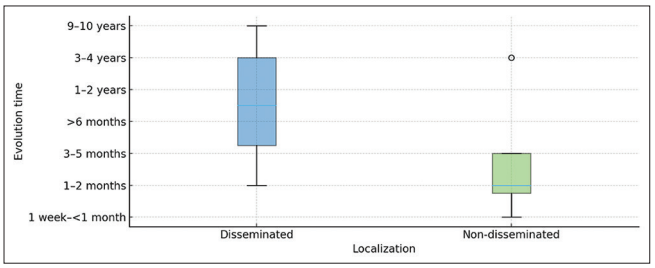


Figure 4: Comparison of disease evolution time between the disseminated and non-disseminated cases.

Table 1: Distribution of disease evolution time

Evolution Time	n (%)
< 1 month	2 (10.5%)
1–2 months	4 (21.1%)
3–5 months	4 (21.1%)
> 6 months	2 (10.5%)
1–2 years	2 (10.5%)
3–4 years	3 (15.8%)
9–10 years	2 (10.5%)
Total	19 (100%)

The duration of clinical evolution ranged from 15 days to 10 years, with a median of 5 months. Most patients consulted after 1–2 months (31.6%), followed by 3–5 months (21.1%); 47.4% had a prolonged evolution (> 6 months – 10 years) before diagnosis (Table 1).

A comparison of the time of evolution according to the extent of the disease showed that disseminated cases had longer times of evolution and greater variability, while non-disseminated cases had a more recent onset and a shorter evolution (Fig. 4).

Regarding anatomical location, 55.6% presented disseminated involvement, 33.3% had lesions on the face or head, 5.6% on the trunk, and 5.6% in other areas (extremities or mixed locations) (Fig. 5).

Among the total number of patients, 11 (57.9%) had confirmed diagnoses, 2 (10.5%) had confirmed diagnoses with differential entities, and 3 (15.8%) were probable cases. Likewise, in five patients (26.3%), an association with mycosis fungoides was

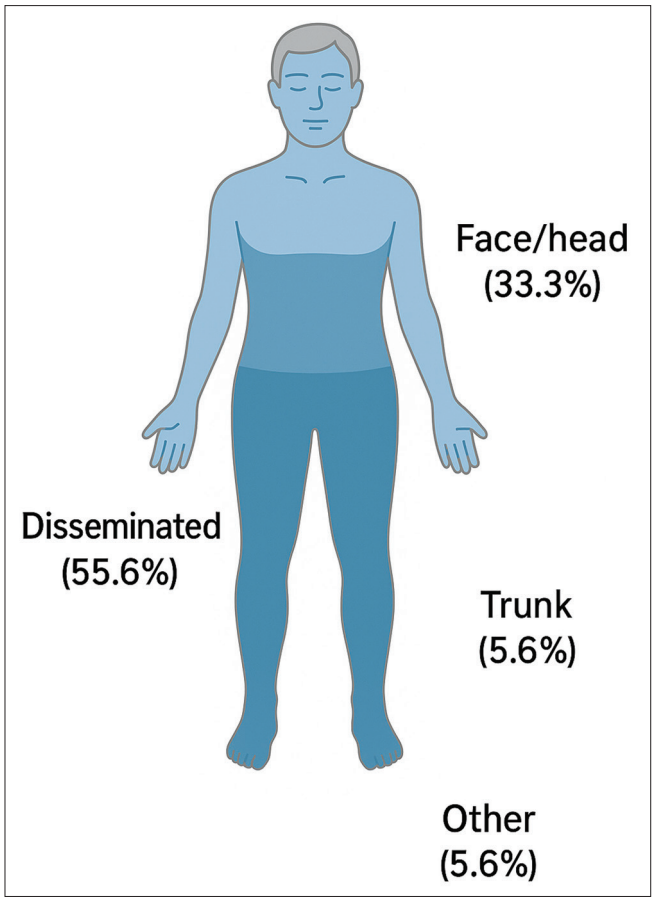


Figure 5: Anatomical distribution of lesions in patients with follicular mucinosis.

documented, ranging from early stages to tumor stages.

DISCUSSION

Our cohort showed a clear female predominance (73.7%), whereas in other reported series, the majority of cases showed a male predominance of up to 69.5–70% [13,14]. This striking contrast could reflect differences in population characteristics, a true geographical or genetic variation in the presentation of the disease. In terms of age, our cohort had a mean age of 31 years, indicating a presentation in younger patients compared to that described in other published series. In a series of 23 patients in Valencia, Spain [13], an average age of 48 years (range 11–78) was reported, while in a series of 33 patients reported by the Mayo Clinic [14], averages between 27 and 37 years were observed in primary variants and 54 years in cases secondary to lymphoma. These differences suggest that, in our population, follicular mucinosis tended to manifest earlier, which was probably related to a higher proportion of primary and disseminated forms included in our cohort, while in the classic series, secondary forms associated with lymphoproliferative processes predominated. The main locations were the face and trunk (33.3% and 5.6%, respectively), in accordance with the literature, although cases were also documented in the extremities. The main challenge in diagnosis lies in its clinical similarity to other inflammatory dermatoses, particularly cutaneous lymphomas, mainly mycosis fungoides [15], which reinforces the importance of histopathological study. In our cohort, 26.3% (n = 5) of the patients had a confirmed diagnosis of mycosis fungoides with follicular involvement, ranging from early stages to the tumor stage. This proportion is highly similar to those described in other series [13-15], with reports of 27-32% associated with cutaneous T-cell lymphomas. However, relevant differences were observed in demographic characteristics: in the North American cohort [14], males predominated (70%), with a mean age of 48 years, whereas in the Spanish series [13], all secondary cases occurred in males, with a mean age of 54 years. In the Italian series [16], primary cases predominated in young women with solitary lesions located in the head and neck, while secondary cases occurred in older men with multiple lesions and extracranial localization.

In contrast, in our cohort, although females and younger age also predominated, cases associated with lymphoma were not limited to males or elderly patients. These findings reinforce the idea that, although the association frequency with lymphoma is consistent across populations, patients' clinical and demographic characteristics may vary, underscoring the need for a comprehensive diagnostic approach that includes clinical, histological, and immunogenetic criteria. Studies with larger, multicenter series are needed to determine whether these differences reflect regional variation or factors specific to each cohort.

CONCLUSION

In this series, MF showed a predominance in females and an earlier age of onset than that described in international series, with predominant involvement of the face and trunk; its diagnosis requires high clinical suspicion and histological confirmation. The contribution of this series lies in expanding knowledge of local epidemiology in Mexico and highlighting the importance of continuing multicenter studies to more accurately define the clinical behavior and prognosis

of follicular mucinosis in our population, despite its low frequency of presentation. Additional studies with a larger number of cases will allow for a better understanding of its epidemiology and association with other entities.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. Cameras-Melchor SE, Aquino F, Vega-Memije E, Arenas-Guzmán R. Mucinosis folicular: reporte de caso. Dermatol Cosmét Méd Quirúrg (DCMQ). 2016;14:118-22.
2. Pinkus H. Alopecia mucinosa. Arch Dermatol. 1983;119:690.
3. Passaro EMC, Silveira MT, Valente NYS. Acneiform follicular mucinosis. Clin Exp Dermatol. 2004;29:396-8.
4. Losoya-Jaquez MR, Salazar Torres FJ, Rojas Castañeda RG, Lopez Yañez Blanco A, Hernández Torres M de las M. Mucinosis folicular primaria variante acneiforme: reporte de un caso pediátrico. Piel (Barc, Internet). 2024;39:476-8.
5. Pérez-Rojas DO, Guevara-Sanginés E, Vega-Memije ME. Mucinosis folicular primaria crónica diseminada: reporte de caso y revisión de la literatura. Dermatología Revista Mexicana. 2021;65:55-9.
6. Esteves M, Nogueira A, Azevedo F, Mota A. Pediatric follicular mucinosis. Indian Dermatol Online J. 2021;12:172-3.
7. Gibson LE, Muller SA, Leiferman KM, Peters MS. Follicular mucinosis: Clinical and histopathologic study. J Am Acad Dermatol. 1989;20:441-6.
8. Sharquie K, Jabbar RI. Papular mucinosis (papular lichen myxedematosus): Clinical and histopathological evaluation. Our Dermatol Online. 2021;12:113-9.
9. Rodríguez-Rangel X, Santiago-Benítez MJ, Vega-Memije ME. Mucinosis folicular: revisión de la literatura. Dermatología Cosmética, Médica y Quirúrgica (DCMQ). 2025;23:245-53.
10. Schneider SW, Metze D, Bonsmann G. Treatment of so-called idiopathic follicular mucinosis with hydroxychloroquine. Br J Dermatol. 2010;163:420-3.
11. Kluk J, Krassilnik N, McBride SR. Follicular mucinosis treated with topical 0.1% tacrolimus ointment. Clin Exp Dermatol. 2014;39:227-8.
12. Harthi FA, Kudwah A, Ajlan A, Nuaim A, Shehri F. Urticaria-like follicular mucinosis responding to dapsone. Acta Derm Venereol. 2003;83:389-90.
13. Jiménez OS, Botella Estrada R, Guillén Barona C, Rodríguez Serna M, Aliaga Boniche A. Mucinosis folicular: nuevas entidades clínico-patológicas. Estudio clínico, histológico, inmunohistoquímico e inmunogenotípico de 23 pacientes. Actas Dermosifiliogr. 2002;93:102-13.
14. Mehregan DA, Gibson LE, Muller SA. Follicular mucinosis: Histopathologic review of 33 cases. Mayo Clin Proc. 1991;66:387-90.
15. Stewart FA, Andea A, Hughey LC. Mycosis fungoides-associated follicular mucinosis after bone-marrow transplantation. J Am Acad Dermatol. 2012;66:e240-1.
16. Rongioletti F, De Lucchi S, Meyes D, Mora M, Rebora A, Zupo S, et al. Follicular mucinosis: A clinicopathologic, histochemical, immunohistochemical and molecular study comparing the primary benign form and the mycosis fungoides-associated follicular mucinosis. J Cutan Pathol. 2010;37:15-9.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Brief Report

Prognosis of skin cancers in the Conakry Cancer Department, Guinea

Malick Bah, Alhassane Ismael Touré, Kalil Cisse, Mamadou Bobo Souare, Mamady Keita, Bangaly Traore

Department of Oncology, National Donka Hospital, University Hospital of Conakry, Guinea

Corresponding author: Malick Bah, MD, E-mail: lickmadem@yahoo.fr

ABSTRACT

Objective: The objective was to study the prognosis of skin cancers in the oncology department of Conakry. **Materials and Methods:** This was a 14-year retrospective, descriptive, and analytical study conducted from April 11, 2007, to January 1, 2021, including patients with histologically confirmed cancer. **Results:** We collected 227 histologically confirmed cases. The mean age was 45.8 ± 20.8 years. Males represented 51.5% of the cases. The consultation delay was greater than six months (65.6%). The clinical aspect was mainly ulcerative-proliferative (50.7%). The lesions were located in the head/neck region (46.2%) and lower limbs (37.9%). The cancers included non-melanoma epithelial carcinomas (70.9%) and melanoma epithelial carcinomas (21.6%). The mean lesion size was 11.02 ± 9 cm. Clinical stages were locally advanced (79.7%) and metastatic (11.9%). Treatment consisted of surgery (68.3%), chemotherapy (30.9%), and radiotherapy (0.7%). After an average follow-up of 8.43 ± 10.3 months, 32 cases (23.0%) relapsed. Recurrence was local (53.1%) or metastatic (40.6%). Overall survival at 12 months was 23%. Good prognostic factors were statistically significant for iterative resection (0.01) and initiation of treatment (0.01). **Conclusion:** Skin cancer is a common pathology in our context. Diagnosis at an advanced stage and limited access to treatment make the prognosis poor for these otherwise curable cancers. This study showed that early diagnosis and appropriate management could reverse this prognosis.

Key words: Prognosis, Skin cancer, Guinea

INTRODUCTION

Skin cancers are malignant tumors that develop from one of the skin components, which may be epidermal, dermal, hypodermal, melanocytic, or adnexal in origin. These cancers are characterized by an uneven distribution worldwide [1]. Depending on the affected tissue, they may be carcinomas, melanomas, Kaposi's sarcoma, or other types (sarcoma, lymphoma), differing in clinical, evolutionary, and histological expression [2]. According to the WHO, skin carcinomas are 15 to 20 times more frequent than melanomas. Between two and three million non-melanoma skin cancers are recorded worldwide [3]. In Africa, all histological types of skin cancer are observed, but with different proportions [4]. Their incidence has increased significantly in recent decades, although still relatively rare in the African literature [5]. The most frequent risk factors are exposure to UV rays, albinism, chronic ulcers, and burn scars. A biopsy with histopathological confirmation is necessary for diagnosis, evaluation, management, and prognosis [6]. In Guinea, Traoré B. et al. [2], in 2017, reported that the overall survival of patients with skin cancer after surgical treatment was 65.2%, with prognostic factors including iterative tumor resection, surgical margins, ulceration, and recurrence [7]. In general, cancer management is multidisciplinary. In our context, several difficulties are encountered, including late diagnosis, the high cost of chemotherapy, and the absence of radiotherapy. In the oncology department of Donka National Hospital, surgery has been the standard treatment

How to cite this article: Bah M, Touré AI, Cissé K, Souaré MB, Keita M, Traoré B. Prognosis of skin cancers in the Conakry Cancer Department, Guinea. Our Dermatol Online. 2026;17(2):192-195.

Submission: 24.09.2025; **Acceptance:** 23.11.2025

DOI: 10.7241/ourd.20262.8

since 2007. The global incidence of these cancers, their prevalence in Guinea, and the need to improve patient management and prognosis motivated this study, which aimed to assess the prognosis of skin cancers in the oncology department of Conakry.

MATERIALS AND METHODS

This was a retrospective cohort study conducted over 14 years, from April 11, 2007, to January 1, 2021. It included all histologically confirmed cases of skin cancer during the study period. Epidemiological aspects, characteristics of skin cancers, therapeutic approaches, and patient follow-up were described. Data was analyzed using SPSS 21.0 software. Categorical variables were expressed as frequencies or percentages, and quantitative variables as means (± standard deviations) or medians with interquartile ranges (IQR). Survival was calculated using the Kaplan–Meier method, and factors associated with survival were analyzed using the Cox model. A test was considered significant when $p < 0.05$.

RESULTS

We recorded 227 cases of skin cancer (Fig. 1), representing 1.3% of all cancers during the study period. The mean patient age was 45.8 ± 20.8 years. Males accounted for 51.5%, with a sex ratio of 1.02. Most patients resided in rural areas (51.5%), were married (65.2%), illiterate (73.1%), and had low socioeconomic status (81.5%). Hypertension (16.7%) and HIV (5.7%) were the main comorbidities. Risk factors included sun exposure (81.5%), albinism (10.1%), burn scars (8.4%), smoking (24.2%), and combined tobacco-alcohol use (15.0%). Patients consulted for swelling (70%), nodules (16.3%), or ulceration (13.6%), with a consultation delay greater than 6 months (65.6%). Lesions were ulcerative-proliferative (50.7%) and ulcerated (21.1%), infiltrative (60.8%), and unique (90.7%). The most common locations were the head and neck (46.2%) and lower limbs (37.9%). Histologically, non-melanoma epithelial carcinoma (70.9%) and melanoma (21.6%) were the most frequent. Tumors were mainly T4 (59.5%), with inguinal lymphadenopathy (39.2%) and stages III (79.7%) and IV (11.9%). Only 139 cases (61.2%) received specific treatment (Table 1): surgery (41.8%), chemotherapy (18.9%), and radiotherapy (1 case). Follow-up was less than 3 months in 63.3% of cases and more than 6 months in 25.9% (Table 2). Recurrence was observed in 23.0% of cases: local (53.1%), locoregional (6.3%), and metastatic (40.6%). At the last follow-up, 47.1% of patients were alive, 30.8% had died, and 22% were lost to follow-up. Overall 12-month survival was 23% (Fig. 2). Iterative surgery and initiation of treatment (surgery or chemotherapy) were statistically significant good prognostic factors (Table 3).

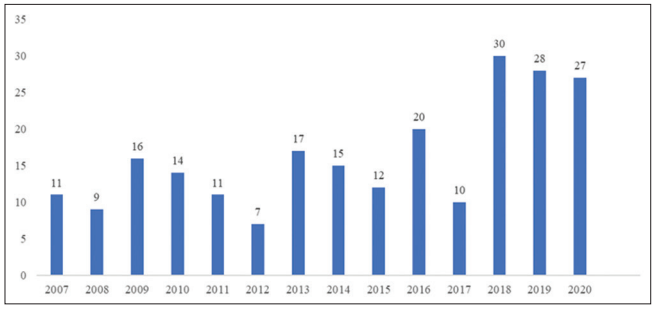


Figure 1: Distribution of selected patients by year.

DISCUSSION

Over 14 years, we recorded a notable frequency of skin tumors (1.3%). This proportion is lower than that reported by Traoré B. et al. in the Journal of Cancer Therapy (7.8%) [7] and much lower than that of Diallo et al. in Senegal (16%) [8], but higher than that of Andrianarison M. et al. in Madagascar (0.5%) [1]. This difference may be

Table 1: Distribution of the patients according to specific treatments.

Characteristic	Number (n=139)	Percentage (%)
Surgery	95	68.3
Chemotherapy	43	30.9
Radiotherapy	1	0.7

Table 2: Distribution of the patients according to progression (recurrence) after treatment.

Characteristic	Number (n=139)	Percentage (%)
Follow-up		
< 3 months	88	63.3
3–6 months	15	10.8
> 6 months	36	25.9
Progression		
Recurrence	32	23.02
Not specified	107	76.9
Recurrence (n=32)		
Local	17	53.1
Locoregional	2	6.3
Metastatic	13	40.6
Status at last update		
Alive	107	47.1
Deceased	70	30.8
Lost to follow-up	50	22.0

Table 3: Distribution of the prognostic factors according to follow-up.

Factor	Alive		O.R	P-value	C.I
	Yes	No			
Age (yrs.)					
<40	75	17	1.06	0.51	0.88–1.27
≥40	83	52			
Sex					
Female	80	32	0.87	0.15	0.73–1.04
Male	78	37			
Time to consultation					
≤3 months	30	10	0.79	0.6	0.62–1.00
>3 months	128	59			
Lymph node involvement					
N0	113	41	0.90	0.31	0.74–1.09
N+	45	28			
Tumor size (cm)					
≤5	19	6	1.05	0.73	0.77–1.42
>5	58	40			
Stage					
Stages I and II	21	13	0.96	0.80	0.74–1.24
Stage III	108	61			
Stage IV	9	15			
Repeat resection					
Yes	12	6	1.49	0.01	1.07–2.08
No	146	63			
Treatment					
No	114	49	1.64	<0.01	1.34–2.01
Yes	44	20			
Surgery					
Yes	34	13	1.55	<0.01	1.24–1.93
No	124	56			
Chemotherapy					
Yes	12	8	1.41	0.03	1.03–1.92
No	147	60			
Recurrence					
Local	12	5	1.07	0.19	0.96–1.19
Locoregional	2	0			
Metastatic	0	1			

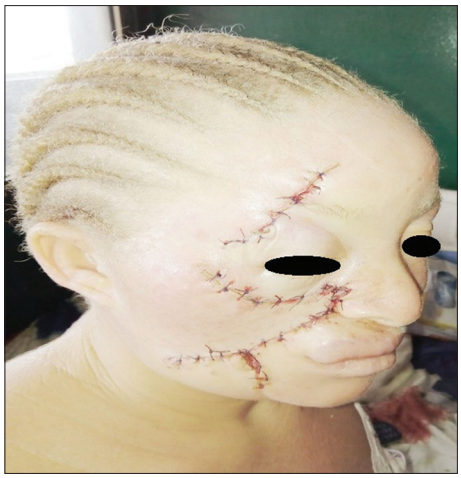


Figure 2: Overall survival curve for the patients with skin cancer.

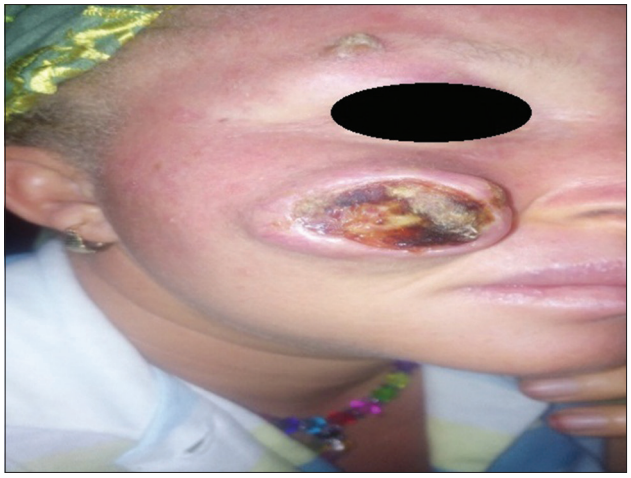


Figure 3: Direct suture after a wide excision of a squamous cell carcinoma of the cheek in a 34-year-old female patient on postoperative day 1.

explained by limited diagnostic and therapeutic facilities in Guinea and patient neglect. Our series was marked by a peak in 2018, possibly due to cancer screening campaigns. The mean age of patients was 45.8 years, lower than that reported in Burkina Faso (48.5 years) [5] and Morocco (57.7 years) [9]. This suggests skin cancer often affects older individuals with greater UV exposure. We observed a slight male predominance (sex ratio 1.02), similar to studies in the French Antilles [10] and other African reports [11,12], though Ouédraogo et al. [5] found female predominance in Burkina Faso. Tobacco use was significant (55%), and albinism was present in 10.1% of cases, higher than Mali (2.3%) [13,14]. The long consultation delay (average 21.4 months) contributed to advanced disease stages, worsened by traditional medicine and self-medication. Clinically, most tumors were ulcerative-proliferative, consistent with squamous cell carcinoma. Lesions were mainly located in the head/neck (46.2%) (Fig. 3) and lower limbs (37.9%), matching literature findings [15-17]. Histologically, squamous cell carcinoma predominated, while basal cell carcinoma was rare, as reported in African studies [2,5,7,8,18]. Advanced stage tumors (mostly T4) reflected delayed diagnosis. Surgery was the primary treatment (68.3%) (Figs 3 and 4), though access to radiotherapy remains absent in Guinea. Recurrence was observed in 23%, lower than Traoré et al. (28.8%) [7]. Overall 12-month survival was only 23%, compared to 75% at 5 years in India [19,20]. Poor outcomes reflect advanced presentation and limited treatment access. Prognostic factors included iterative resection and initiation of treatment, consistent with previous Guinea findings [7].

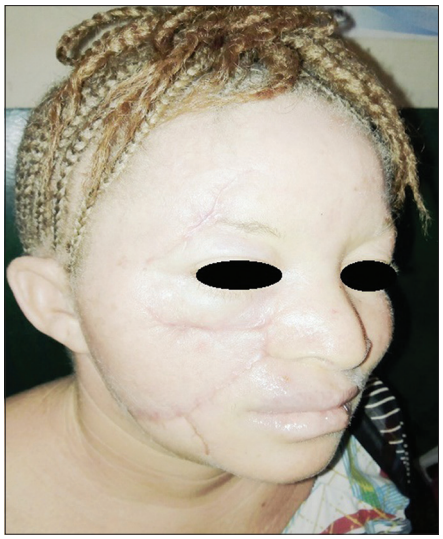


Figure 4: Scar from a wide excision of a squamous cell carcinoma of the cheek in a 34-year-old female patient.

CONCLUSION

Skin cancer is a frequent pathology in our context. Advanced-stage diagnosis and limited access to treatment lead to poor prognosis for these otherwise curable cancers. Early diagnosis and appropriate management could significantly improve patient outcomes.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. Malalaniaina A, Lovasoa T, Mamisoa RI, Malala R, Soavina RL, Florine R, et al. [Skin cancers in Madagascar: Where do we stand?]. Pan Afr Med J. 2019;34:167.
2. Traore B, Keita M, Condé M, Keita M, Diane S, Cissé M, et al. Caractéristiques anatomo-cliniques des cancers cutanés à l'unité de chirurgie oncologique du CHU de Conakry. Rev CAMES SANTE. 2016;4:78-82.
3. Organisation Mondiale de la Santé «Cancer de la peau non mélanome ou carcinome cutané et facteurs environnementaux» santé.fr 2018
4. Didier B, Francès C, Guillot B, Guilhou JJ. Manifestations dermatologiques des maladies du système hématopoïétique et oncologie dermatologique. Paris: Springer Verlag; 2009. XIV, 325
5. Ouédraogo MS, Zongo N, Ouedraogo NA, Tapsoba GP, Korsaga/Somé NN, Nyamba YB, et al. Cancers cutanés sur peau noire en milieu tropical africain. Ann Dermatol Venereol 2015;142;389-90.
6. Linares MA, Zakaria A, Nizran P. Skin cancer. Primary care: Clinics in office practice. 2015;42:645-59.
7. Traore B, Lamah L. Outcomes of surgical treatment of skin cancer at Surgical Oncology Unit of Donka, Conakry University Hospital. JCT. 2017;08:1086-94.
8. Diallo M, Dieng MT. Profils épidemiologiques et anatomo-cliniques des cancers cutanés au CHU Aristide Le Dantec de Dakar sur une période de 10 ans. Ann Dermatol Vénéréole. 2016;143:36.
9. Bolac C, Cordel N, Deschamps L, Renier M, Quist D, Derancourt C, et al. [Diagnosis of skin cancer by dermatologists in the French West Indies: A prospective study]. Ann Dermatol Venereol. 2011;138:11-6.
10. Bergeret B, Stoebner PE, Jardot M. Prévalence des cancers cutanés dans une cohorte rétrospective de patients sans abris. Ann Dermatol Venerol. 2022;2:A169.
11. Kadakia S, Chan D, Mourad M, Ducic Y. The prognostic value of age, sex and subsite in cutaneous head and neck melanoma: A clinical review of recent literature. Iran J Cancer Prev. 2016;9;5079-82.
12. Saka B, Souley Z, Kombaté K, Mouhari-Toure A, Akakpo S, Napo-Koura G, Tchangaï-Walla K, Pitché P. [Skin cancers in Togo: A 223-case series]. Med Trop (Mars). 2010;70:169-71.
13. Epidémiologie des cancers. Institut National du Cancer 2017 www.e-cancer.fr. Derrière mise à jour 19/03/2017.
14. Almamy D, Lenga Loumingou IA, Mienwoley Armel O, Mutiyu Akanbi S, Irené G, Anderson Stephen KA, et al. Squamous cell carcinoma on a Buruli ulcer graft scar: Ivory Coast. Our Dermatol Online. 2023;14:194-6.
15. Samison LH, Randrianasolo P, Ravalisoa A, Ratrimoarivony C, Rapelanoro Rabenja F. Apport de Chirurgie sur les tumeurs basocellulaires à Madagascar. 1er Congrès de Dermatologie Vénéréologie de l'Océan indien, 2004.
16. Fontaine J, Mielczarek S, Meaume S, Senet P. [Incidence of undiagnosed skin cancers in a geriatric hospital]. Ann Dermatol Venereol. 2008;135:651-5.
17. Razafindrakoto R, Randrianandraina M, Rakotoarisoa A, Razanakoto G, Rakoto F. Les carcinomes basocellulaires de la face: à propos de cinq cas malgaches. Rev Med Madagd. 2015;5:493-6.
18. Asuquo M, Ebughe G. Cancers cutanés fréquemment rencontrés à l'hôpital universitaire de Calabar, à Calabar, dans le sud du Nigéria. Int J Dermatol. 2012;51:36-40.
19. No authors. Prise en charge diagnostique et thérapeutique du carcinome basocellulaire de l'adulte. Ann Pathol. 2004;24:460-72.
20. Deo SV, Hazarika S, Shukla NK, Cancersy T. Prise en charge chirurgicale de la peau Experience d'un centre regional de cancerologie du nord de l'inde. J Indien Cancer. 2005;42:145-50.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Brief Report

Chronic loose scaly cheilitis: An often-overlooked variant

Khalifa E. Sharquie, Robert A. Schwartz, Adil A. Noaimi, Inas K. Sharquie, Sara A Ali

[1]Department of Dermatology, College of Medicine, University of Baghdad. Iraqi and Arab Board for Dermatology and Venereology, Baghdad Teaching Hospital, Medical City, College of Medicine, Baghdad, Iraq, [2]Dermatology, Rutgers New Jersey Medical School, 185 South Orange Avenue, 07103. Newark, NJ, USA, [3]Department of Microbiology & Immunology, College of Medicine, University of Baghdad, Baghdad, Iraq, [4]Department of Dermatology, Baghdad Teaching Hospital; Medical City, Baghdad, Iraq

Corresponding author: Prof. Khalifa E. Sharquie MD, PhD, FRCP Edin, FRCP Lon, E-mail: *ksharquieprof@yahoo.com*

ABSTRACT

Background: Chronic cheilitis is a common dermatological problem where it is caused by many diseases, such as atopic dermatitis, licking dermatitis, and cheilitis simplex. Exfoliative cheilitis is a well described entity where its etiopathogenesis is not well elucidated. **Objective:** The objective was to report a new variant of exfoliative cheilitis named chronic loose scaly cheilitis that has new distinctive and descriptive clinical features. **Patients and Methods:** This cross-sectional, descriptive study was conducted during the period from 2014 to 2021, in which all patients with chronic loose scaly cheilitis were collected. Full demographic and clinical evaluation was performed. Patients were advised not to use any lipstick, toothpaste drugs, or chemicals on their lips during the course of the study. **Results:** Ten patients were fully analyzed. Their ages ranged from 25 to 45 years, with a mean of 32 years, with 6 females and 4 males. The duration of the disease ranged from 3 to 5 years. No history of lips licking or sucking was present, and no drugs, lipsticks, or chemicals were applied to the lips. Also, no history of excessive sun exposure was mentioned. The patients gave a highly characteristic picture as they presented with loose, easily detachable, thick scale crust sheets affecting both lips, but when removed, leaving oozing fleshy red lips with this scaly crust being reformed again shortly. Surprisingly, the patients had no desire to remove it. On examination, all patients showed thick, scaly crusts covering both lips, very loose and easily removed, leaving a bleeding red surface. The oral cavity was normal. The patients' psychology apparently was normal as no features of anxiety, depression, or psychosis were detected. No personal or family history of atopy or psoriasis was recorded. All types of therapy, including topical emollients, steroid ointment, and tranquilizer, were tried but gave mild relief. **Conclusion:** This is a new variant of cheilitis that has a highly distinctive chronic course and a characteristic clinical picture, refractory to therapy, that bears the name *chronic loose scaly cheilitis*.

Key words: Chronic cheilitis; Exfoliative cheilitis; Chronic loose scaly cheilitis

INTRODUCTION

Cheilitis is an acute or chronic inflammation of the lips. The need to delineate a group of cheilitis was recognized by Sharquie [1], who noted the morphology as a chronic loose scaling cheilitis and suggested a reemphasis based upon the clinical appearance. Usually, it involves the lip vermilion and the vermilion border, but it may affect the surrounding skin, and the oral mucosa may also be affected by inflammatory reactions [2].

Exfoliative cheilitis (EC) is an inflammatory disease affecting the lips and is characterized by the production of a thick keratin scale that is painful and crusted [3]. There is continuous production of desquamated thick, brown scales and crusts of keratin [4,5]. The keratin layer of the epidermis of the

How to cite this article: Sharquie KE, Schwartz RA, Noaimi AA, Sharquie IK, Ali SA. Chronic loose scaly cheilitis: An often-overlooked variant. Our Dermatol Online. 2026;17(2):196-200.

Submission: 07.11.2025; **Acceptance:** 03.01.2026

DOI: 10.7241/ourd.20262.9

lips experiences an accelerated growth and death rate than normal and desquamates [6]. When these scales-crusts are removed, an apparently normal appearing lip is revealed beneath [5]. Although there may be associated erythema and edema, this problem has no well-defined particular cause [3].

In some patients, there is an association with anxiety, depression, stress or personality disorder [3,4]. In one report, 87% of patients had some form of psychiatric disturbances, and 47% had dysfunction of the thyroid, which in turn may induce psychiatric problems such as depression [7]. Some cases, exfoliative cheilitis is thought to represent factitious damage, termed *factitious cheilitis* or *artifactual or cheilitis artefacta* [4,5,7,8]. This could be related to repetitive lip licking or pricking habits [3]. This is seen as crusting and ulceration caused by repetitive sucking and chewing of the lips [8]. Some consider habitual lip picking or licking to be a form of a nervous tic [9]. This habit is sometimes termed *perleche* [4]. Factitious cheilitis is significantly more commonly seen among young females [7,8]. Exfoliative cheilitis has also been linked to HIV/AIDS [7]. Treatment consists commonly of keeping the lips moist and the application of topical corticosteroids ranging from hydrocortisone to clobetasol. There have also been reports of using topical tacrolimus [10]. This may be helpful in some cases, but others require psychotherapy such as tranquillizers or antidepressants [11,12]. In one study, patch testing was conducted and was negative for the common allergens.

It is a general term as there are many well-differentiated types and with variable etiological factors. Cheilitis simplex: also called *chapped lips* [13,14] or common [15]. Are featured by drying, peeling and fissuring of the lips, and are one of the most common variants of cheilitis [13,16]. The lower lip is commonly involved but it may also affect the upper lip [16]. There may also be the formation of large, painful cracks when these lips are stretched. Lip biting, licking, or rubbing habits are frequently involved in the pathogenesis of this type cheilitis. Paradoxicalbe theinuous licking of the lips induces drying and irritation, and finally, the mucosa splits or cracks and this act, over time, becomes a habit. The lips have a greater tendency to dry out in cold, dry weather. The digestive enzyme present in saliva may also irritate the lips, and evaporation of water in saliva saps moisture from them [17]. Some children have the habit of chewing and sucking on the lower lip, producing a combination of cheilitis and sharply demarcated perioral erythema [18]. Therapy could be successful with barrier lubricants such Vaseline [13]. Medical-grade (USP) lanolin accelerates the repair of the lips [19] and is used in some lip repair products. Some complementary and alternative medicine sources claim that nasal sebum may be a beneficial remedy [14].

Eczematous cheilitis (also termed *lip dermatitis* [20]): Lip cheilitis is a diverse group of disorders that often have an unknown cause [2]. Chronic eczematous reactions account for most cases of chronic cheilitis. It is divided into endogenous due to an inherent characteristic of the individual, and exogenous where it is caused by an external cause. The main cause of endogenous eczematous cheilitis is atopic cheilitis (atopic dermatitis), and the main causes of exogenous eczematous cheilitis are irritant contact agents inducing allergic contact cheilitis [21]. The latter is characterized by dryness, cracking, edema, and crusting [10]. It affects females more commonly than males, in a ratio of about 9:1 [22]. The most common causes of allergic contact cheilitis is lip cosmetics, including lipsticks and lip balm, followed by toothpaste [22]. An allergy to balsam of Peru can also induce as chronic cheilitis [23]. Allergies to metal, wood, or other component may cause cheilitis reactions in musicians, especially players of wood wind and brass instruments [24], that is, the so called *clarinetists cheilitis* [25]. Therapy employs an emollient and topical corticosteroids.

Differential Diagnosis

Actinic cheilitis

Actinic cheilitis, also termed *solar cheilosis*, is a disease that is the result of chronic overexposure to UV light in sunlight radiation. It usually involves the lower lip, causing dry, scaling, and a wrinkled, gray white appearance [22]. It is especially common in people with light skin types who live in sunny climates, and in persons who spend outdoor activities for long periods of time. There is a small risk of this condition going into squamous carcinoma in the long term, yet lip cancer is usually noticed early and, hence, has a good prognosis compared to oral cancer generally.

Angular cheilitis

Angular cheilitis is inflammation of one or both angles of the mouth [9]. It is a fairly common condition, and often affects elderly people associated with infection.

There are numerous possible causes, including nutritional deficiencies such as of iron, zinc, B vitamin, folate, contact allergy, infections such as *Candida albicans*, *Staphylococcus aureus* or B-hemolytic streptococci.

Infectious cheilitis

Infectious cheilitis refers to cheilitis caused by an infectious disease [8]. The names *bacterial cheilitis* [5] and *candidal cheilitis* [26] are sometimes used to refer to the involvement of *Candida* organisms and bacterial species, respectively. Herpes labialis is a common cause of infectious cheilitis [8,27,28]. Recurrence of latent herpes simplex infection may cause lesions in the mouth corners and be confused with other causes of angular cheilitis. In fact, this is herpes labialis and is sometimes called *angular cheilitis*.

Granulomatous cheilitis

Orofacial granulomatosis is the enlargement of both lips due to the formation of a non-caseating granulomatous reaction, which obstructs lymphatic drainage of orofacial soft tissue, causing lymphedema. A related condition is Melkersson–Rosenthal syndrome, a triad of facial palsy, chronic lip edema, and fissured tongue [28]. Meischer's cheilitis [29], and *granulomatous macrocheilitis* [30] are synonyms of granulomatous cheilitis.

Drug-induced cheilitis

Common causes of drug-related cheilitis include etretinate, protease inhibitors, indinavir, and isotretinoin [9,31]. Uncommon causes include atorvastatin, clofazimine, busulphan, cyancobalamin, clomipramine, gold methyl dopa, psoralen, streptomycin, and tetracycline [9]. A condition known as "drug-induced ulcer of the lip" is described as ulcerations of the lip without induration [10]. It is induced by the oral administration of drugs, and the condition stops when these drugs are withdrawn [32].

Cheilitis glandularis

Cheilitis glandularis is a rare inflammatory reaction of the minor salivary glands, usually in the lower lip, which appears swollen and everted [10]. There may also be crusting ulceration, abscess, and sinus tracts. It is an acquired disease, yet the cause is not well known [33]. Suspected causes include sunlight, tobacco, poor oral hygiene, syphilis, and genetic factors [7]. The opening of the minor salivary gland ducts becomes inflamed and dilated, and there might be muco-purulent discharge from these ducts. Cheilitis glandularis usually affects middle-aged and elderly males and has the risk of malignant transformation to squamous cell carcinoma (18% to 35%) [7]. Preventive management such as vermilionectomy is, therefore, the therapy of choice [7].

Plasma cell cheilitis

Plasma cell cheilitis is a very rare condition that occurs more on the gingiva and sometimes on the tongue [34]. Plasma cell cheilitis appears as a well-defined, infiltrated, dark red plaque with superficial lacquer-like glazing [10]. Plasma cell cheilitis usually affects the lower lip [34]. The lip looks dry, atrophic, and fissured [7].

Other causes

1. Lupus erythematosus [35], sometimes termed *lupus cheilitis* [7].
2. Crohn's disease (angular cheilitis) [3].
3. Nutritional cheilitis [27], e.g., iron, zinc, pyridoxine (vitamin B6) deficiency [7].
4. Lichen planus actinicus [27].
5. Pemphigoid [27].
6. Xerostomia [14].

Thus, the aim of the present work is to assess the new picture of cheilitis that has a distinctive, characteristic clinical course, which makes it different from other types of cheilitis.

PATIENTS AND METHODS

This cross-sectional, descriptive study was conducted during the period from 2014 to 2021 during which all patients with chronic loose scaly cheilitis were collected. Full demographic and clinical evaluation was performed. Psychological assessment was done. All other causes of the cheilitis or cheilitis-like picture were excluded. Also, any history of drug intake related to cheilitis such as retinoids was confirmed. Patients were advised not to use any lipstick, toothpaste drugs, or chemicals on their lips during treatment and follow-up.

RESULTS

Ten cases were seen, 6 females and 4 males, with a ratio of 1.5. Their ages ranged from 25 to 45 years, with a mean of 32 years. They presented with chronic cheilitis of many years duration, which ranged from 3 to 5 years. No history of lip licking or sucking was present, and no drugs, lipsticks, or chemicals were applied to the lips. Also, no history of excessive sun exposure was mentioned. The patients gave a highly characteristic picture that, when seen once, could not be missed again. The patients presented with loose, easily detachable, thick scale crust sheets affecting both lips, which when removed, left oozing fleshy red lips, with this scaly crust being reformed again shortly.

Surprisingly, the patients had no desire to remove it, so sometimes, it was suspected and confused with dermatitis neglecta. On examination, all patients showed thick, scaly, wet crusts covering both lips, very loose and easily removed, leaving a bleeding red oozing surface (Figs. 1a – 1d). Full examination of the oral cavity revealed no abnormalities. The patients' psychology apparently was normal as no features of anxiety, depression, or psychosis were detected. No personal or family history of atopy or psoriasis was recorded.

All types of therapy, including topical emollients, steroid ointment, and tranquilizer were tried but all gave temporary and mild relief followed by recurrence.

DISCUSSION

This chronic, loose, scaly cheilitis has some features similar to exfoliative cheilitis but we are not sure that both conditions are the same, although this new variant has a more distinctive and characteristic clinical course that makes it different from other types of cheilitis. Still, the name *chronic eczematoid cheilitis* could be applied to this variant of cheilitis [6,21].

No features of other skin diseases such as atopic dermatitis, solar cheilitis, lichen planus actinicus, psoriasis were detected in ten cases after a long follow-up. Thus, these patients are unique in their characteristic descriptive features, which deserve the term *chronic loose scaly cheilitis*.

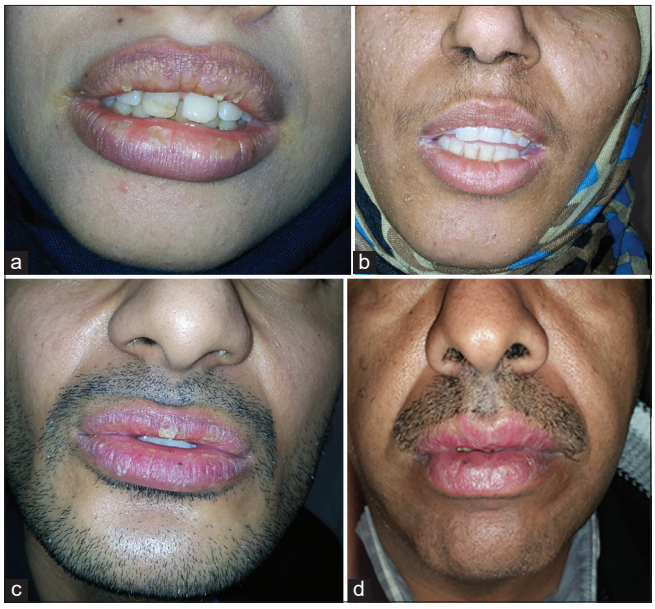


Figure 1: (a) A 27-year-old woman with chronic loose scaly cheilitis. (b) A 35-year-old woman with chronic loose scaly cheilitis. (c) A 28-year-old male showing chronic loose scaly cheilitis. (d) A 40-year-old male with chronic loose scaly cheilitis after removing the crust.

This condition is usually chronic, and its course takes many years and is refractory to all types of therapy. Although patients are worried about their condition, they have no apparent morbid psychology. The patients have no desire to remove the thick scale crusts on their lips. So, dermatitis neglecta [36] is suspected in some patients. These patients might have some psychological disturbances in the form of depression or anxiety, yet these emotional problems usually appear as a consequence of this chronic, refractory, and annoying condition.

In conclusion, chronic loose scaly cheilitis has highly characteristic descriptive features that deserve to be considered a specific variant of exfoliative cheilitis that needs more attention and research to reach the actual etiopathogenesis.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. Sharquie K.E. Chronic loose scaly cheilitis. Difficult cheilitis. VGRD Blog. http://www.vgrd.blogspot.com 2015
2. Saha A, Seth J, Bindal A, Samanta AB, Gorai S, Sharma A. Dermatosis neglecta: An increasingly recognized entity with review of literature. Indian J Dermatol. 2016;61:450-2.
3. Bruch JM, Treister NS. Clinical oral medicine and pathology. 1 ed: Humana Totowa, NJ. 2010;47;8,144 p.
4. Park KK, Brodell RT, Helms SE. Angular cheilitis, part 1: Local etiologies. Cutis. 2011;87:289-95.
5. Daley TD, Gupta AK. Exfoliative cheilitis. J Oral Pathol Med. 1995;24:177-9.
6. Mani SA, Shareef BT. Exfoliative cheilitis: Report of a case. J Can Dent Assoc. 2007;73:629-32.
7. Neville BW, Damm DD, Allen CM, Chi AC. Oral and maxillofacial pathology: Elsevier Health Sciences. 2015.
8. Aydin E, Gokoglu O, Ozcurumez G, Aydin H. Factitious cheilitis: A case report. J Med Case Rep. 2008;2:1752-947.
9. Romita P, Barlusconi C, Mercurio CS, Hansel K, Stingeni L, Foti C. Photoallergic contact cheilitis from benzydamine hydrochloride contained in a mouthwash. Contact Dermatitis. 2020;83:130-2.
10. Schena D, Fantuzzi F, Girolomoni G. Contact allergy in chronic eczematous lip dermatitis. Eur J Dermatol. 2008;18:688-92.
11. Ophaswongse S, Maibach HI. Allergic contact cheilitis. Contact

Dermatitis. 1995;33:365-70.
12. Leão JC, Hodgson T, Scully C, Porter S. Review article: Orofacial granulomatosis. Aliment Pharmacol Ther. 2004;20:1019-27.
13. Sun K, Liu L, Chen X, Jiang W. Treatment of exfoliative cheilitis with Traditional Chinese Medicine: A systematic review. J Tradit Chin Med. 2017;37:147-58.
14. Jadotte YT, Schwartz RA. Solar cheilosis: An ominous precursor: part I. Diagnostic insights. J Am Acad Dermatol. 2012;66:173-84.
15. Thongprasom K. Glycerin borax treatment of exfoliative cheilitis induced by sodium lauryl sulfate: A case report. Acta Stomatol Croat. 2016;50:158-61.
16. Almazrooa SA, Woo SB, Mawardi H, Treister N. Characterization and management of exfoliative cheilitis: A single-center experience. Oral Surg Oral Med Oral Pathol Oral Radiol. 2013;116:016.
17. Gronostay A, Hellmich L, Rasokat H, Hunzelmann N. Coincidence of cheilitis granulomatosa and chronic spontaneous angioedema: Successful treatment with dapsone and omalizumab. J Dtsch Dermatol Ges. 2020;18:611-3.
18. Zhang QQ, Xu P, Sun C, Liu LJ, Jiang WW. Topical tacrolimus with different frequency for exfoliative cheilitis: A pilot study. J Dermatolog Treat. 2022;33:550-4.
19. Abroug N, Lahdhiri M, Kechida M, Abdeljlil A, Khochtali S, Khairallah M. Panuveitis revealing Melkersson Rosenthal syndrome: A case report. Tunis Med. 2020;98:90-2.
20. João AL, Brasileiro A, Neves JM, Lencastre A, Pinheiro RR. Discoid lupus erythematosus of the lip: A case of refractory cheilitis: Lupus. 2020;29:804-5.
21. O'Gorman SM, Torgerson RR. Contact allergy in cheilitis. Int J Dermatol. 2016;55:6.
22. Lim SW, Goh CL. Epidemiology of eczematous cheilitis at a tertiary dermatological referral centre in Singapore. Contact Dermatitis. 2000;43:322-6.
23. Nanda A, Wasan A. Allergic contact dermatitis to balsam of Peru. Ann Allergy Asthma Immunol. 2016;117:208-9.
24. Mariano M, Patruno C, Lembo S, Balato N. Contact cheilitis in a saxophonist. Dermatitis. 2010;21:119-20.
25. Ruiz-Hornillos FJ, Alonso E, Zapatero L, Pérez C, Martínez-Molero I. Clarinetist's cheilitis caused by immediate-type allergy to cane reed. Contact Dermatitis. 2007;56:243-5.
26. Acton QA. Stomatitis: New Insights for the Healthcare Professional: 2013 Edition: ScholarlyBrief: ScholarlyEditions; 2013.
27. Brasch J. Recent literature: L. Kanerva, P. Elsner, JE. Wahlberg, HI. Maibach (Eds): Condensed handbook of occupational dermatology. Arch Dermatol Res. 2005;296:588-9.
28. Rose AE, Leger M, Chu J, Meehan S. Cheilitis granulomatosa. Dermatol Online J. 2011;17:15.
29. Oudrhiri L, Chiheb S, Marnissi F, Zamiati S, Benchikhi H. Successful treatment of Miescher's cheilitis in Melkersson-Rosenthal syndrome with betamethasone injections and doxycycline. The Pan African Medical Journal. 2012;13.
30. Khadir K, Hali F, Tirhazouine I, Zamiati S, Laraqui A, Lakhdar H, et al. [Granulomatous macrocheilitis: 19 cases]: Ann Dermatol Venereol. 2013;140:56-8.
31. Amichai B, Shemer A, Grunwald MH. Low-dose isotretinoin in the treatment of acne vulgaris. J Am Acad Dermatol. 2006;54:644-6.
32. Mackie BS. Drug-induced ulcer of the lip. Br J Dermatol. 1967;79:106-10.
33. Carrington PR, Horn TD. Cheilitis glandularis: A clinical marker for both malignancy and/or severe inflammatory disease of the oral cavity. J Am Acad Dermatol. 2006;54:336-7.
34. Janam P, Nayar BR, Mohan R, Suchitra A. Plasma cell gingivitis associated with cheilitis: A diagnostic dilemma! J Indian Soc Periodontol. 2012;16:115-9.
35. Chi AC, Neville BW, Krayer JW, Gonsalves WC. Oral manifestations of systemic disease. Am Fam Physician. 2010;82:1381-8.
36. Saha A, Seth J, Sharma A, Biswas D. Dermatitis neglecta: A dirty dermatosis: Report of three cases. Indian J Dermatol. 2015;60:185-7.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Brief Report

Are eponyms easy to remember?

Salkin Cinki Ayse Irem[1], Aksungur Varol Lutfu[2]

[1]Edirne Sultan 1. Murat State Hospital, Department of Dermatology, Edirne Merkez/Edirne, Turkey, [2]Cukurova University, Department of Dermatology, Adana, Turkey

Corresponding author: Salkin Cinki Ayse Irem, MD, E-mail: driremsalkin@gmail.com

ABSTRACT

Background: There is a current interest in eponymous dermatological signs. This report aims to investigate their memorability. **Material and Methods:** Twenty-five residents in dermatology were trained with a slide show on 51 eponymous signs. Because of familiarity and ease of answering, seven of them were excluded. For the remaining 44 signs, both eponym-focused and description-focused questions were prepared. Two weeks after the presentation, the residents had an exam composed of randomly selected 12 eponym-focused and 12 description-focused questions. **Results:** The median number of correct answers was 3 (interquartile range: 2–4) for the eponym-focused questions, whereas this figure was 9 (interquartile range: 7–10) for the description-focused questions. The difference was statistically significant ($p < 0.001$). Success rates were higher in the description-focused questions for all signs except two. **Conclusion:** These results suggest that eponyms are less memorable than descriptions. Since there is an ongoing controversy on the elimination of eponyms from the medical literature, more studies should be done on the cognitive aspects of eponymy.

Key words: Eponyms, Dermatology, Cognition

INTRODUCTION

For most scientific findings, it is not widely known who first described them. In other words, they are anonymous. For the rest of scientific findings, it is known who first reported them. However, only a small part of them is named after their discoverers. In other words, they are eponymous.

Recently, numerous reviews on dermatological signs have been published [1-12]. They give quite long lists of signs. Eponymous signs have an important place within these lists. Despite this interest, in the case where discoverers of signs are known, there is a lack of standardization in labeling them with descriptive names or with eponyms. Whatever the reasons for this deficiency, it is worth examining the place of eponyms in learning dermatological signs.

For this purpose, residents in dermatology were trained about dermatological eponymous signs, their descriptions, and their causes. Then, they had an exam composed of different types of questions. Thereafter, it was evaluated whether eponyms or descriptions were more memorable.

MATERIALS AND METHODS

Dermatological eponymous signs were compiled from recently published articles [1-14]. Widely known signs, such as the Auspitz, Darier, and Nikolsky signs, were excluded. Also, signs that were seen in dermatological diseases but were related to organs other than the skin, such as Hutchinson's teeth sign, were not included. As a result, 51 eponymous signs were used in the present study. They were the Barnett, Battle, Berliner, Besnier, Biederman, Borsieri, Brenner, Buschke-Ollendorff, Casal, Crowe, Cullen, Filipovitch, Fox, Frank, Giovannini, Greenhow, Grey Turner, Guérin, Hildreth, Hoagland, Ingram, Jacquet, Joffroy, Kaposi-Stemmer, Kerr, Krisovski, Liddle, Love, Maroni, Meffert, Milian, Mizutani, Nagayama, Nazzaro, Orentreich, Patrick Yesudian, Pemberton, Pittaluga, Premalatha, Punshi,

How to cite this article: Irem SCA, Lutfu AV. Are eponyms easy to remember?. Our Dermatol Online. 2026;17(2):201-205.

Submission: 21.12.2025; **Acceptance:** 06.02.2026

DOI: 10.7241/ourd.20262.10

Russell, Samitz, Shelly, Shuster, Silex, Sister Mary Joseph, Tasleem, Trousseau, Vieira, Walzel, and Zirelí signs.

A slide show was prepared to train residents in dermatology. In this show, each eponymous sign had two slides. In the first slide, only the eponym was written, and it was voiced in its original language. In the second slide, there were a representative image of the sign, its description, its causes, and the full name of the author. Whenever available, the author's country, specialty, and dates of birth and death were added.

Twenty-five residents receiving specialized training in our department for periods ranging between one month and four years participated in the study. Before the slide show, they were asked to mark the signs known to them from the sign list. Then, the slide show was presented. After the presentation, the residents confirmed their marks. The presentation was given to the residents as a video file. They were asked to watch it for the next two weeks, in order to learn thoroughly the content of the video.

Multiple-choice questions were prepared for each eponymous sign. However, five of them, namely the Cullen, Grey Turner, Pemberton, Sister Mary Joseph, and Trousseau signs, were excluded, since they were familiar before the slide show to a majority of the residents. The questions were of four types. In the first type, the question stem was focused on the eponym, as in the following example, "which disease causes the Brenner sign?." Its choices were diseases. In the second type, the stem was focused on the disease, as in the following example, "which sign is caused by melanoma?." Its choices were eponyms. In the third type, the stem was focused on the description of the finding, as in the following example, "which disease causes an ill-defined erythema adjacent to a hyperpigmented lesion?." Its choices were diseases. In the fourth type, the stem was the same as that of the second type. However, its choices were descriptions of the findings.

For the Kerr and Punshi signs, descriptions could easily recall their causes. Therefore, they were also excluded. Thus, 176 questions related to the 44 eponymous signs remained. It was planned to use 6 questions for each type, giving a total of 24 questions. It was aimed that each of these 24 questions was related to a different sign. In order to use questions related to all 44 signs, it was preferred to use, for each resident, a different set of questions, which were randomly selected from the 176 questions. In order to lower the effects of anxiety about scoring low on their performance, the residents were asked not to write their names on the exam papers. Thus, it was given up to evaluate the effects of the individual characteristics of the residents on the results.

The main analysis was to compare success rates in questions of the first two types to those in questions of the last two types. Since the questions of the first two types mentioned eponyms either in question stems or in choices, whereas the questions of the last two types used descriptions instead of eponyms, such a comparison gave an opportunity to determine which were more memorable, eponyms or descriptions. Comparisons were also done separately for each eponym, with questions restricted to each. The Wilcoxon signed-rank test was used for paired difference test, and the Spearman test was used for the correlation test. R software and its library "PairedData" were used in statistical analysis and plotting [15,16].

The study was approved by the Ethics Committee of Çukurova University, and written informed consent was obtained from all participants prior to enrollment.

RESULTS

The number of correct answers ranged from 3 to 22 in the exam composed of 24 questions. The median was 11 (interquartile range: 9–13). If only twelve questions mentioning eponyms in their stems or choices, in other words eponym-focused questions, were taken into consideration, the range of the number of correct answers was 0 to 10. The median was 3 (interquartile range: 2–4). If only twelve questions mentioning descriptions in their stems or choices, in other words description-focused questions, were taken into consideration, the range of the number of correct answers was 2 to 12. The median was 9 (interquartile range: 7–10).

The difference between the eponym-focused questions and the description-focused questions in terms of the number of correct answers was statistically significant according to the Wilcoxon signed-rank test ($p < 0.001$). The number of correct answers to the eponym-focused questions and to the description-focused ones was plotted for each participant in Figure 1. The number of correct answers to the description-focused questions was higher in all participants except one. Increments in the number of correct answers ranged from 1 to 11. The most frequent increment was 7, which was seen in 8 participants. The only participant showing decrement

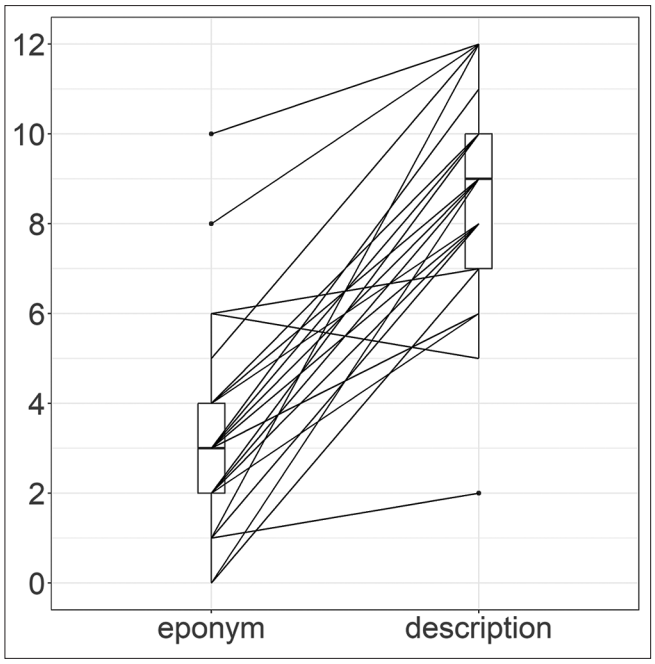


Figure 1: The number of correct answers to the eponym-focused questions and to the description-focused questions for each participant.

gave correct answers to 6 eponym-focused questions, and this figure lowered to 5 in the description-focused questions.

Since there were 25 participants and 24 questions for each participant, the total number of questions was 600. The distribution of these 600 questions according to their related signs was given in Table 1. The success rates in eponym-focused questions and in description-focused questions were equal for a sign, namely the Milian sign. The success rate in eponym-focused questions was higher for a sign, namely the Liddle sign. For the remaining 42 signs, higher success rates were observed in description-focused questions. For example, 8 eponym-focused questions and 8 description-focused questions were related to the Guérin sign. Success rates in those were 50% and 75%, respectively. Similarly, 8 eponym-focused questions and 8 description-focused questions were related to the Filipovitch sign. Success rates in those were 12% and 62%, respectively. According to the Spearman test, there was no correlation between eponym-focused questions and description-focused questions in terms of success rates ($r = 0.18$, $p > 0.05$).

DISCUSSION

The results of this study showed that eponyms were less memorable than descriptions, since participants, namely residents in dermatology, gave fewer correct answers in number to eponym-focused questions than description-focused questions. Moreover, when questions related to each sign were evaluated separately, the rate of correct answers to eponym-focused questions was lower for almost all signs. Our findings support the hypothesis that there is difficulty in the retrieval of proper names [17].

Recently, numerous reviews compiling eponyms used in dermatology have been published. A striking example is an article series written by Piotr Brzeziński and his colleagues. This series is in a dictionary format composed of over thirty articles beginning to be published in 2010 and continuing until 2020 [18,19]. In a recent study, trends in dermatology eponyms were examined in a PubMed-based search [20]. It was found that citations of dermatology-focused eponyms increased in a parallel course to that of all citations from 1880 to 2020. In a recent review, arguments for both abandoning and maintaining eponyms were mentioned while discussing the value of eponyms in dermato-trichological nomenclature [21].

Not only for dermatology but also for all medicine, it is a current topic to discuss whether or not eponyms should be maintained [22-29]. The main reason that some advocate for maintaining the use of eponyms is to honor the discoverers. They also assume eponyms are more practical, since they are usually shorter than descriptive terms. However, there is a law about eponymy named after Stigler [30]. He said, "no scientific discovery is named after its original discoverer." He gave his eponym as an example of this law, since others have also reached this conclusion previously.

An example of Stigler's law in dermatology is a well-known sign, namely the Auspitz sign. Kaposi stated in his textbook that easy bleeding in psoriatic lesions had been known since Willan, who was the founder of modern dermatology [31]. He added that bleeding of psoriatic lesions after removing scales was mentioned by Hebra, who was the mentor of both Auspitz and himself. On the other hand, acantholytic cells are not known by the name of Auspitz, despite the fact that he was the first user and describer of this histopathological term [32].

Although defenders of eponymy suppose that proper names are more useful in practice, the present study showed that they are less memorable. Besides difficulty in learning and remembering proper names, there may be a competition in their retrieval [17]. A well-known

Table 1: The distribution of correctly answered questions and all questions according to their related signs

Sign	Number of questions correct answers/total (%)				Sign	Number of questions correct answers/total (%)			
	Eponym- focused		Description- focused			Eponym- focused		Description- focused	
Barnett	4/10	(40)	3/3	(100)	Krisovski	0/9	(0)	5/8	(62)
Battle	5/8	(62)	3/3	(100)	Liddle	2/4	(50)	4/9	(44)
Berliner	2/6	(33)	8/10	(80)	Love	2/4	(50)	6/7	(86)
Besnier	1/5	(20)	7/7	(100)	Maroni	2/11	(18)	4/5	(80)
Biederman	2/8	(25)	1/1	(100)	Meffert	0/9	(0)	4/6	(67)
Borsieri	0/9	(0)	5/6	(83)	Milian	3/8	(38)	3/8	(38)
Brenner	1/8	(12)	4/5	(80)	Mizutani	3/10	(30)	3/4	(75)
Buschke-O.*	2/8	(25)	2/6	(33)	Nagayama	2/8	(25)	3/5	(60)
Casal	2/4	(50)	9/10	(90)	Nazzaro	1/7	(14)	3/5	(60)
Crowe	1/7	(14)	5/5	(100)	Orentreich	4/10	(40)	3/3	(100)
Filipovitch	1/8	(12)	5/8	(62)	Patrick Y.‡	2/4	(50)	6/10	(60)
Fox	5/9	(56)	6/7	(86)	Pittaluga	1/4	(25)	6/9	(67)
Frank	2/6	(33)	3/4	(75)	Premalatha	3/7	(43)	4/5	(80)
Giovannini	3/9	(33)	1/2	(50)	Russell	2/9	(22)	3/7	(43)
Greenhow	0/5	(0)	6/6	(100)	Samitz	0/4	(0)	4/10	(40)
Guérin	4/8	(50)	6/8	(75)	Shelly	0/5	(0)	6/10	(60)
Hildreth	2/5	(40)	8/9	(89)	Shuster	1/8	(12)	4/6	(67)
Hoagland	2/6	(33)	2/2	(100)	Silex	2/6	(33)	8/9	(89)
Ingram	2/5	(40)	11/12	(92)	Tasleem	0/3	(0)	8/9	(89)
Jacquet	0/4	(0)	8/9	(89)	Vieira	1/5	(20)	3/11	(27)
Joffroy	1/6	(17)	5/10	(50)	Walzel	1/7	(14)	3/5	(60)
Kaposi-S.†	0/5	(0)	10/12	(83)	Zirelí	3/9	(33)	4/4	(100)

*, Buschke–Ollendorff; †, Kaposi–Stemmer; ‡, Patrick Yesudian

example is "Moses illusion." A substantial proportion of people who are asked, "how many animals of each kind did Moses take on the Ark?" respond with "two" rather than "none," since they fail to distinguish that the subject of the question should have been Noah. Such a replacement may be a result of mislearning. In a survey, 93 consultants or registrars in orthopedic surgery were questioned about Finkelstein's test [33]. Among them, 84 reported that they used it in their practice. However, 83 participants were unable to recognize the correct method between three descriptive pictures. The misrecognition was found to be due to misinformation in the literature, because Leao quoted Eichhoff's manoeuvre as Finkelstein's test in 1958 [34].

In the present study, the design of the exam consisting of 24 multiple-choice questions and using a different set of questions for each participant may be a limitation. In further studies, some changes in the design may be tried, such as increasing the number of questions, preferring open-ended questions instead of multiple-choice questions, and using only a single set of questions for all participants. All that aside, the main limitation of the present study was its short duration. A longitudinal study spanning the whole duration of the training will provide more precise results.

CONCLUSION

As arguments for abandoning eponyms, it is undoubtedly important that some eponyms are connected to Nazi medicine, most eponyms celebrate European or North American physicians, and women account only for a minority of eponyms [22,29]. However, the destiny of eponyms should be determined according to the results of new studies investigating the cognitive aspects of eponymy, as attempted in the present study.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. Freiman A, Kalia S, O'Brien EA. Dermatologic signs. J Cutan Med Surg. 2006;10:175-82.
2. Patel LM, Lambert PJ, Gagna CE, Maghari A, Lambert WC. Cutaneous signs of systemic disease. Clin Dermatol. 2011;29:511-22.
3. Madke B, Nayak C. Eponymous signs in dermatology. Indian Dermatol Online J. 2012;3:159-65.
4. Pradhan S, Madke B, Singh AL, Kabra P. Less-known clinical signs in dermatology. Indian Dermatol Online J. 2016;7:421-23.
5. Rastogi V, Singh D, Tekiner H, Ye F, Mazza JJ, Yale SH. Abdominal physical signs of inspection and medical eponyms. Clin Med Res. 2019;17:115-26.
6. Kulkarni AS, Birangane RS, Parkarwar PC, Kazi AZ. Clinical and radiological signs of importance for the oral physician and oral surgeon. J Indian Acad Oral Med Radiol. 2019;31:257-62.
7. Das A, Toshniwal A, Madke B. Newer signs in dermatology [2016-

2020]. Indian Dermatol Online J. 2020;12:342-45.
8. Sharma S, Khaitan BK, Kumarasinghe SP. Cutaneous signs in dermatological diseases: An overview. Indian J Dermatol. 2021;66:530-39.
9. Ramesh H, Somashekar S. The unsung signs of dermatology. Indian J Dermatol. 2021;66:705-10.
10. Amrani A, Sil A, Das A. Cutaneous signs in infectious diseases. Indian J Dermatol Venereol Leprol. 2022;88:569-75.
11. Mancy A. Handbook of signs in clinical dermatology. 2nd ed. Haditha (Iraq): Al-Yusr Press; 2023.
12. Polra RV, Tandel JJ, Shah MM, Nair PA. Signs in dermatology: Clinical, dermoscopic, and histopathological. Clin Dermatol Rev. 2024;8:273-89.
13. Kassam K, Kumar M. The importance of oral examination facial trauma: Recognition of the Guerin sign in Le Fort 1 injuries. Clin Case Rep. 2014;2:112.
14. Veasey JV, de Macedo PM, Amorim JR, Orofino-Costa R. The correct nomenclature of Zirelí sign in the propaedeutics of pityriasis versicolor (in memoriam). An Bras Dermatol. 2021;96:591-94.
15. R Core Team (2024). R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria. Available from: https://www.R-project.org/. Accessed Jan. 12, 2025.
16. Champely S (2018). PairedData: Paired Data Analysis. R package version 1.1.1. Available from: https://CRAN.R-project.org/package=PairedData. Accessed Jan. 12, 2025.
17. Abrams L, Davis DK. Competitors or teammates: How proper names influence each other. Curr Dir Psychol Sci. 2017;26:87-93.
18. Brzeziński P. Dermatology Eponyms – Phenomenon/sign – Dictionary (A). N Dermatol Online. 2010;1:34-5.
19. Brzeziński P. Martini L, Sood S. Dermatology Eponyms – sign – Lexicon (Y). Our Dermatol Online. 2020;11:e181.1-e181.2.
20. He L, Cornish TC, Kricka LJ, Vandergriff TW, Yancey K, Nguyen K, et al. Trends in dermatology eponyms. JAAD Int. 2022;7:137-43.
21. Trüeb RM. Value of eponyms in dermato-trichological nomenclature. Skin Appendage Disord. 2018;4:71-77.
22. Woywodt A, Matteson E. Should eponyms be abandoned? Yes. BMJ. 2007;335:424.
23. Whitworth JA. Should eponyms be abandoned? No. BMJ. 2007;335:425.
24. Collier R. Eponymy: Make that Hippocrates-Janin-Neumann-Reis-Bluthe-. -Behcet's disease. CMAJ. 2012;184:1878-80.
25. Ferguson RP, Thomas D. Medical eponyms. J Community Hosp Intern Med Perspect. 2014;4.
26. Chaudhary H, Silaj K. What's in an eponym? The ethics and efficacy of eponym use in education. UCLA Psychology [serial online]. 2021 [cited 2025 Jan 1]. Available from: https://sites.lifesci.ucla.edu/psych-pia/2021-10-7-whats-in-an-eponym-the-ethics-and-efficacy-of-eponym-use-in-education
27. Barthakur R, Dawka S. Eponyms in medicine: A Socratic dialogue. Glob J Med Pharm Biomed Update. 2022;17.
28. Barr J, Mangold A. Call it as it is: Why medical education should reconsider disease eponyms. Med Teach. 2024;46:1108-10.
29. Nedumannil L, Lewis D. Should medical eponyms continue to be used in everyday practice? Med J Aust. 2024;221:297-98.
30. Stigler, SM. Stigler's law of eponymy. Trans N Y Acad Sci. 1980;39:147-57.
31. Kaposi M. Pathologie et traitement des maladies de la peau. Tome 1. 2nd ed. Paris: G. Masson; 1891:542-43.
32. Shareef S, Shareef F, Tongdee E, Florez-White M. Acantholysis or the Auspitz sign? A revelation of the life of Carl Heinrich Auspitz. Indian J Dermatol Venereol Leprol. 2017;83:512.
33. Waseem M, Khan M, Hussain N, Giannoudis PV, Fischer J, Smith RM. Eponyms: Errors in clinical practice and scientific writing. Acta Orthop Belg. 2005;71:1-8.
34. Leao L. De Quervain's disease: A clinical and anatomical study. J Bone Joint Surg Am. 1958;40-A:1063-70.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Brief Report

Unlawful practice of aesthetic medicine, patient attractiveness, and consequences

Kaoutar Benchekroun, Maryam Ghaleb, Salim Gallouj, Ouiame El Jouari

Department of Dermatology, Mohamed VI University Hospital Center of Tangier, Morocco

Corresponding author: Kaoutar Benchekroun, MD, E-mail: kaoutar.benchekroun@usmba.ac.ma

ABSTRACT

Background: The global expansion of aesthetic medicine has been paralleled by the rise of illegal procedures performed outside medical settings, exposing patients to avoidable complications. **Objective:** The objective was to assess the attractiveness of illegal aesthetic practices, reported complications, and risk perception in a northern Moroccan population. **Methods:** We conducted a prospective, analytical, cross-sectional survey between February and July 2023 at a tertiary dermatology center in Northern Morocco. An online questionnaire (Google Forms) disseminated via social networks collected sociodemographic data, aesthetic practices, adverse effects, and risk perception. Data was analyzed using SPSS v.2021 (chi-squared; $p < 0.05$). **Results:** A total of 234 respondents participated; most were women (61.9%), aged twenty-one to thirty years (58.9%), with a university education (73.2%). Prior attendance at beauty centers was common (62.1%), and 56.4% desired future procedures, strongly influenced by celebrities and social media (78.7%). Illegal procedures were reported by 26.7%, mainly laser hair removal and hydrafacial (42.7%). Among injected participants, 86.2% received no information on pre- or post-procedure precautions. Post-procedure events included redness (83.1%), irritation (70.9%), asymmetry (32.7%), abscesses (12.3%), and granulomas (7.2%); prolonged sequelae occurred in 23.5%. Dissatisfaction after care in nonmedicalized settings was high (63.3%). Cost (70.3%) and social media advertising (47.8%) were the main reasons for choosing such venues. **Conclusion:** Illegal aesthetic practices are highly attractive to young women and generate frequent, sometimes severe, complications. Regulatory reinforcement, product traceability, and targeted education—especially through social media—with dermatologists as key stakeholders are essential.

Key words: Illegal aesthetic medicine, Fillers, complications, Social media, Regulation

INTRODUCTION

Aesthetic medicine has expanded rapidly worldwide, reflecting the growing importance of physical appearance [1]. While procedures performed by trained physicians are generally safe, a parallel market has emerged where interventions are offered in beauty salons or spas by unqualified providers using uncertified products [2]. These practices expose patients to both immediate complications (erythema, infection) and severe adverse events such as vascular occlusion, skin necrosis, or blindness after filler injections [3]. Delayed complications, including nodules and granulomas, often linked to biofilms, have also been reported [4]. Social and cultural factors, particularly the influence of celebrities and social media, play a major role in normalizing such procedures, especially among young women [5]. This study aimed to evaluate the extent of illegal aesthetic practices in northern Morocco, document their complications, and assess patient risk perception.

MATERIALS AND METHODS

We conducted a prospective, analytical, cross-sectional study at the Department of Dermatology and Venereology, Mohammed VI University Hospital Center of Tangier, between February and July 2023. The participants were adults residing in northern Morocco. Data was collected using a structured questionnaire specifically designed

How to cite this article: Benchekroun K, Ghaleb M, El Ali FZ, Gallouj S, El Jouari O. Unlawful practice of aesthetic medicine, patient attractiveness, and consequences. Our Dermatol Online. 2026;17(2):206-209.

Submission: 06.09.2025; **Acceptance:** 16.11.2025

DOI: 10.7241/ourd.20262.11

for this study and disseminated via Facebook, WhatsApp, and Instagram. The questionnaire explored sociodemographic characteristics, motivations for aesthetic procedures, types and sites of procedures performed, complications experienced, risk perception, and preferred practitioners. Responses were collected anonymously through Google Forms and analyzed with SPSS v.2021. Qualitative variables were expressed as frequencies and percentages, and comparisons were made using the χ^2 test, with significance at $p < 0.05$. Participation was voluntary and preceded by electronic informed consent. Anonymity and confidentiality were guaranteed. The study was conducted in accordance with the Declaration of Helsinki (2013 revision).

Ethics Statement

The study was conducted in accordance with the Declaration of Helsinki (2013 revision). All participants provided electronic informed consent. Personal data confidentiality was ensured.

RESULTS

A total of 234 participants completed the survey. Most were women (61.9%), predominantly aged twenty-one to thirty years (58.9%), and university educated (73.2%). Prior attendance at beauty centers for aesthetic care was reported by 62.1%, and 19.3% attended regularly. Future interest in procedures was expressed by 56.4%, and 78.7% acknowledged influence from celebrities and social media when considering interventions (Table 1). Illegal procedures performed in salons or spas were reported by 26.7%, with laser hair removal and hydrafacial being the most common (42.7%). Among participants who had received injections, 86.2% had not been informed about pre- and post-procedure precautions. Adverse events included redness (83.1%), irritation (70.9%), and persistent skin changes (63.3%). More severe complications were also noted, including asymmetry (32.7%), abscesses (12.3%), and granulomas (7.2%) (Fig. 1); prolonged sequelae were reported by 23.5%. Overall dissatisfaction after care received in nonmedicalized settings was high (63.3%). Cost (70.3%) and social media advertising (47.8%) were the main reasons for choosing such venues (Fig. 2). Regarding risk perception, 62% considered these practices hazardous, mainly due to infection (82%) and unsatisfactory or asymmetric outcomes (71%), whereas fewer than twenty percent identified blindness or stroke as major risks. Most participants considered dermatologists as authorized professionals (79.2%), followed by general practitioners (50.9%). Conversely, 43.5% believed beauticians were authorized, and 11.6% thought anyone could perform these procedures (Fig. 3). Finally, 68.3% favored regulatory reinforcement to curb illegal practice.

Table 1: Previous and future use of aesthetic care.

Variable	Category	Percentage (%)
Previous use	Visited beauty centers	62.1
Previous use	Regular use	19.3
Future interest	Interested in procedures	56.4
Influence	Social media/celebrities	78.7

DISCUSSION

Our findings highlight the significant attractiveness of illegal aesthetic practices, particularly among young, educated women strongly influenced by social media. This aligns with international reports showing that exposure to online beauty standards increases body dissatisfaction and the likelihood of seeking aesthetic procedures, even outside medical contexts [1,5]. The complications observed in our cohort are consistent with global literature, which describes a wide spectrum

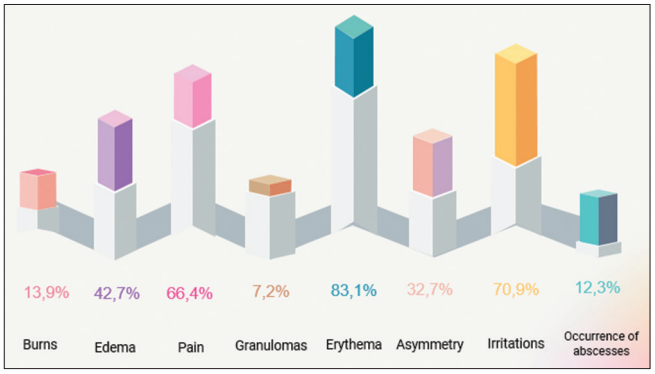


Figure 1: Observed effects after interventions in non-medical centers.

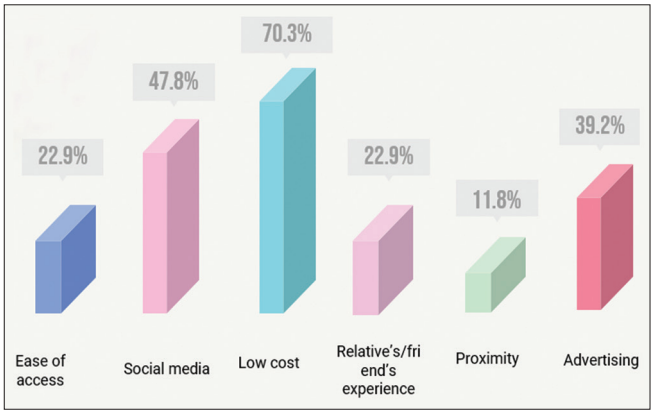


Figure 2: Criteria for choosing beauty centers.

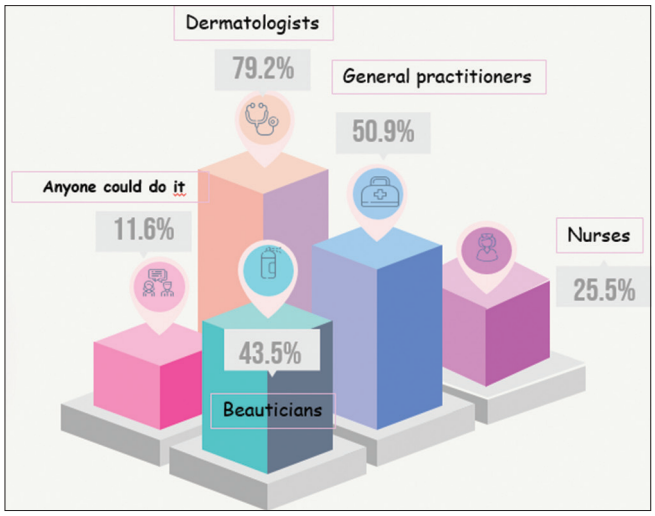


Figure 3: Professionals authorized for this practice according to the participants.

of adverse effects ranging from mild local reactions to severe events such as vascular occlusion and blindness [2,3]. Although rare, vascular complications are devastating, and their prevention relies on anatomical expertise and proper injection techniques—skills absent in non-medical settings. The management of vascular occlusion requires prompt recognition and immediate administration of high-dose hyaluronidase with adjunctive measures, as recommended in clinical guidelines [6]. Such resources are generally unavailable outside healthcare environments, increasing the risk of irreversible sequelae. Delayed reactions such as nodules and granulomas, reported in 7.2% of our respondents, are often related to biofilm formation and chronic immune-inflammatory responses [4,7]. These complications are challenging to treat, often requiring combined therapies with hyaluronidase, corticosteroids, and antibiotics targeting biofilms, or surgical excision in refractory cases. Although less frequent, systemic complications have also been described, including non-thrombotic pulmonary embolism and fatal outcomes following illegal filler injections [8,9]. Such cases underscore that aesthetic procedures are true medical interventions requiring qualified practitioners, certified products, and safe environments. Even when performed by experienced physicians, fillers are not entirely free of complications [10]. This reality further strengthens the argument for strict prohibition of non-medical practices and the implementation of coordinated strategies, including regulatory enforcement, product traceability, public education via social media, and the central involvement of dermatologists in patient safety and advocacy.

CONCLUSION

Illegal aesthetic practices are particularly attractive to a young, socially networked population and are associated with frequent and sometimes severe complications. Prevention requires stricter regulation, product control, enhanced surveillance, and targeted education, with dermatologists and health authorities working jointly to curtail this growing public health concern.

ACKNOWLEDGMENTS

The authors thank all participants for their time and responses.

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all participants.

REFERENCES

1. Rahman E, Webb WR, Rao P, Yu N, Garcia PE, Ioannidis S, et al. A systematic review on the reinforcement loop in aesthetic medicine and surgery: The interplay of social media, self-perception, and repeat procedures. Aesthetic Plast Surg. 2024;48:3475-87.
2. Chayangsu O, Wanitphakdeedecha R, Pattanaprichakul P, Hidajat IJ, Evangelista KER, Manuskiatti W. Legal vs. illegal injectable fillers: The adverse effects comparison study. J Cosmet Dermatol. 2020;19:1580-6.
3. Sito G, Manzoni V, Sommariva R. Vascular complications after facial filler injection: A literature review and meta-analysis. J Clin Aesthet Dermatol. 2019;12:65-72.
4. Artzi O, Cohen JL, Dover JS, Suwanchinda A, Pavicic T, Landau M, et al. Delayed inflammatory reactions to hyaluronic acid fillers: A literature review and proposed treatment algorithm. Clin Cosmet Investig Dermatol. 2020;13:371-8.
5. Mironica A, Popescu CA, George D, Tegzeşiu AM, Gherman CD. Social media influence on body image and cosmetic surgery considerations: A systematic review. Cureus. 2024;16:e65626.
6. Murray G, Convery C, Walker L, Davies E. Guideline for the management of hyaluronic acid filler-induced vascular occlusion. J Clin Aesthet Dermatol. 2021;14:61-9.
7. Funt DK. Treatment of delayed-onset inflammatory reactions to hyaluronic acid filler: An algorithmic approach. Plast Reconstr Surg Glob Open. 2022;10:e4362.
8. Jang JG, Hong KS, Choi EY. A case of nonthrombotic pulmonary embolism after facial injection of hyaluronic acid in an illegal cosmetic procedure. Tuberc Respir Dis (Seoul). 2014;77:90-3.

9. Yang Y, Sheng H, Gu Q, Su L, Tong H, Chen J, et al. Death caused by vaginal injection of hyaluronic acid and collagen: A case report. Aesthet Surg J. 2020;40:263-8.
10. Nishikawa A, Aikawa Y, Kono T. Current status of early complications caused by hyaluronic acid fillers: Insights from a descriptive, observational study of 41,775 cases. Aesthet Surg J. 2023;43:893-904.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Report

Mucocutaneous leishmaniasis: A clinical case report and literature review of health implications for human migration

Alejandra García, Mariana Vélez Pintado, Ana Paula Landeta Sa, Karen Marañón, Ingeborg Becker, Carla Román, Griselda Montes de Oca Sánchez, M. Fernanda González Lara, Alfredo Ponce de León, Alexandro Bonifaz

Instituto Nacional de Ciencias Médicas y Nutrición "Salvador Zubirán", Hospital General de México "Dr. Eduardo Liceaga", Centro Médico ABC, México

Corresponding author: José Alexandro Bonifaz, MD PhD, E-mail: a_bonifaz@yahoo.com.mx

ABSTRACT

Mucocutaneous leishmaniasis (MCL) is a severe form of leishmaniasis. Migration poses a challenge in the diagnosis and treatment of this disease, as patients may present with species not typically recognized as endemic. *Leishmania panamensis* is the predominant species in Panamá, and it is associated with clinical progression. There are no previous reports of this species in Mexico. Herein, we present a 41-year-old woman who presented to the emergency department with skin ulcers on her forehead, right arm, nasal mucosa, and abdomen while traveling on foot from Colombia to Mexico in a migrant caravan. She underwent several medical evaluations and received multiple treatments without improvement. The diagnosis of mucocutaneous leishmaniasis by *L. panamensis* was established three months later. She was treated with liposomal amphotericin B (LAmB). After four months of follow-up, the patient remained asymptomatic, with no lesions. This highlights delays that may occur in non-endemic settings and their impact on migrant populations.

Key words: Migrant caravan, Mosquito, Mucocutaneous, Leishmaniasis, *Leishmania panamensis*, Liposomal amphotericin B

INTRODUCTION

Migration through Central America has reached unprecedented levels, with most individuals arriving in Mexico and waiting for an opportunity to enter the United States. This significant movement comes with changing epidemiology patterns and diagnostic challenges [1].

Leishmaniasis is a chronic infection caused by a flagellated protozoan belonging to the genus *Leishmania*. It is an intracellular parasite transmitted by the bite of female sandflies, specifically *Lutzomyia* in the Americas. Reports of *L. panamensis* are not common in Mexico; it is primarily reported in Central America, especially in Colombia, Costa Rica, and Panamá; a high suspicion is required in migrant populations because of its classification as complex leishmaniasis due to the potential of affecting the mucosa. The clinical manifestations of leishmaniasis vary according to the species involved and the host's immune response. The *L. panamensis* reservoir are sloths, and it is especially associated with the sandfly *Lutzomia trapidoi*. Its infection may be severe, leading to disfigurement or mucosal necrosis and functional impairment. This type of leishmaniasis does not cure spontaneously. This species has lymphatic dissemination; the mucosal membranes more frequently affected are the nose and pharynx. The use of LAmB must be individualized, given limited access to miltefosine and the antimonial's toxicity [2-4].

How to cite this article: García A, Vélez Pintado M, Landeta Sa AP, Marañón K, Becker I, Román C, Montes de Oca SG, González Lara MF, Ponce de León A, Bonifaz A. Mucocutaneous leishmaniasis: A clinical case report and literature review of health implications for human migration.. Our Dermatol Online. 2026;17(2):210-214.

Submission: 24.11.2025; **Acceptance:** 23.01.2026

DOI: 10.7241/ourd.20262.12

CASE REPORT

Herein, we present a 41-year-old woman with no significant medical history. She was born in Barinas, Venezuela, and lived in an urban area in Cali, Colombia, from 2022 until September 2023. That month, she decided to migrate to the United States. She joined a migrant caravan traveling on foot through Panamá, Costa Rica, Nicaragua, El Salvador, and Guatemala, arriving in Mexico City in October 2023. During her journey, she slept on roads, in shelters, in hostels, and on city streets. She crossed rivers, lakes, forests, fields, and walked through the Darien Gap at the Colombia-Panama border and the Lacandon jungle in the state of Chiapas, Mexico. She reported frequent exposure to rats, cockroaches, mosquitoes, and ticks. While traveling, she developed a dermatosis on her forehead, right arm, and abdomen after being bitten by a mosquito during her foot journey from Colombia to Mexico. The lesions began as papules, progressed to nodules, and slowly ulcerated over thirty days. She did not have access to health services until she arrived in Mexico City. After her arrival, she underwent several medical evaluations and received multiple treatments, including acyclovir, dicloxacillin, itraconazole, and amoxicillin/clavulanate, without improvement.

In January 2024, she presented at the emergency department of a general hospital with skin ulcers. She denied medication use, chronic diseases, and other systemic symptoms. On physical examination, the patient appeared well. Vital signs were blood pressure 100/70 mm Hg, pulse 68 beats per minute, temperature 36.2°C, respirations 16 breaths per minute, and oxygen saturation 95% on room air. Dermatologic evaluation revealed a disseminated dermatosis affecting the right arm (Fig. 1), forehead (Figs. 2a and 2b), and abdomen (Figs. 3k and 3l), consisting of three ulcers, some with a warty appearance, others with an erythematous base containing granulation tissue, and with indurated, raised, erythematous-violet borders. The smallest was 1 x 1 cm, and the largest was 7 x 5 cm. The rest of the physical examination and the routine laboratory tests were normal.

A skin biopsy was taken, and a direct skin imprint (microscopic analysis with Giemsa stain) revealed amastigotes. A positive ELISA test (serum antibodies) was also obtained, and a skin biopsy culture yielded promastigotes. The histopathological report of the skin biopsy concluded a granulomatous inflammatory infiltrate from the papillary dermis to the middle

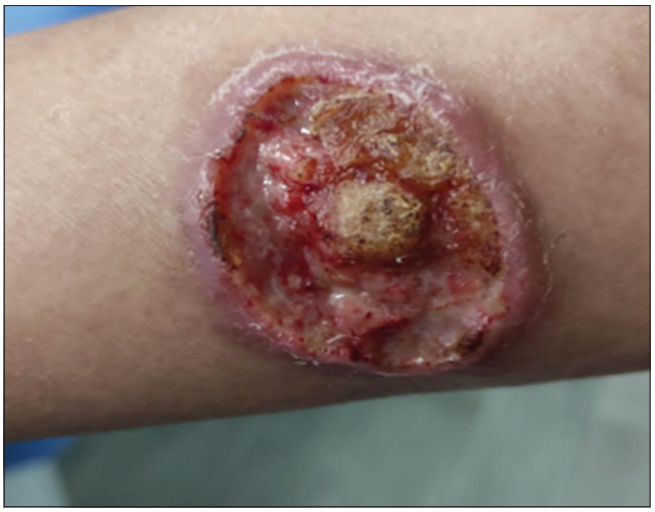


Figure 1: Right forearm skin lesion: Ulcer with an irregular, erythematous base and areas of hyperkeratosis; indurated and elevated borders with a reddish-violet color.

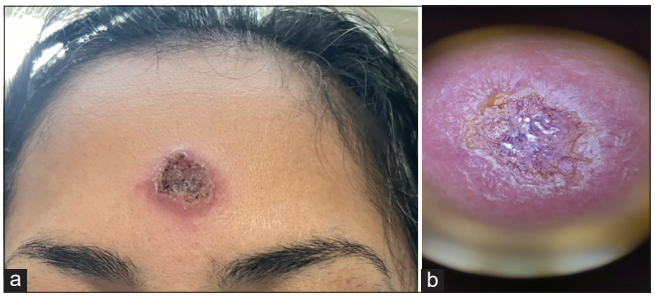
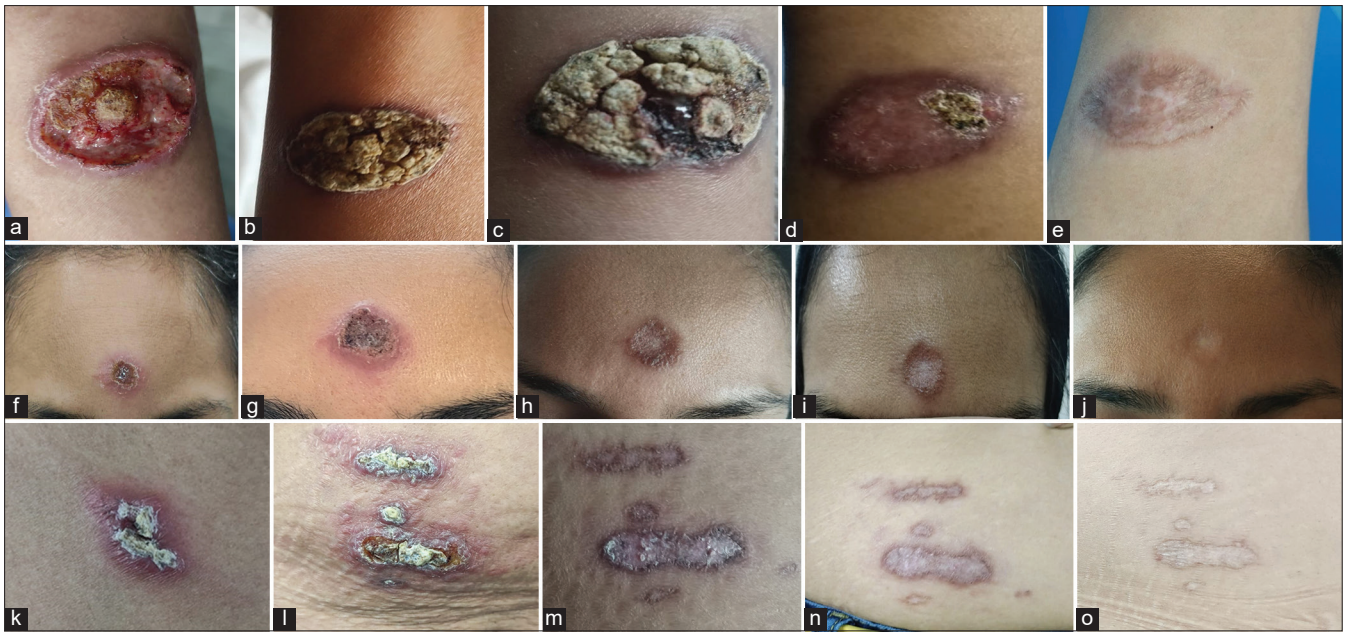


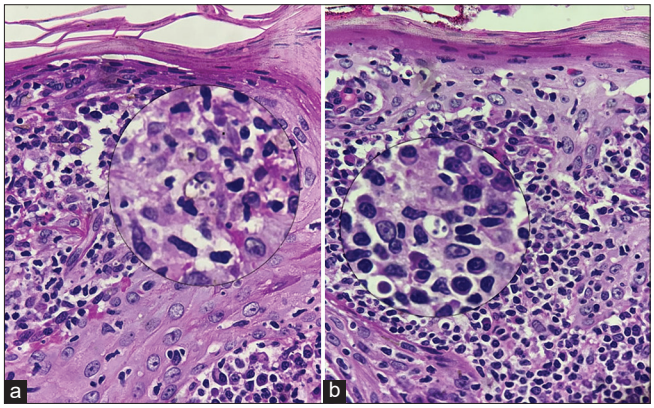
Figure 2: (a and b) Forehead skin lesion and its dermatoscopy: Round ulcer with an erythematous base partially covered by a bloody and meliceric crust; elevated and erythematous borders.

reticular dermis and abundant mixed inflammatory infiltrate with neutrophils, plasma cells, and vacuolated histiocytes, some of which presented rounded intracytoplasmic structures in the periphery, with positive PAS and Grocott staining compatible with amastigotes (Figs. 4a and 4b). Finally, the immunoparasitology laboratory (Faculty of Medicine, Universidad Nacional Autónoma de México) took a skin biopsy, which was positive for *Leishmania sp.* (ITS1)/*Leishmania viannia* (LM9/LV2)/*Leishmania panamensis* (L13). Due to the limited availability of pentavalent antimonials, the patient was referred to a tertiary care center.

Upon arrival, she reported nasal pain, and a nasal ulcer was found afterward. Otorhinolaryngology performed a nasal endoscopy, reporting a pale mucosa with a crusty lesion in the left nasal vestibule extending to the inferior turbinate head with a friable and erythematous mucosa. A nasal mucosa biopsy showed ulcerated mucosa with lymphoplasmacytic inflammation,



Figures 3: (a-e) Evolution of the skin lesions on the right arm. (f-j) Evolution of the skin lesions on the forehead. (k-o) Evolution of the skin lesions on the abdomen.



Figures 4: (a and b) Histopathological sample of an ulcer biopsy: Granulomatous inflammatory infiltrate made up of abundant mixed inflammatory infiltrate with neutrophils, plasma cells, and vacuolated histiocytes, some of which present rounded intracytoplasmic structures aligned to the positive periphery to PAS and Grocott staining compatible with amastigotes.

without evidence of microorganisms (negative Giemsa, PAS, gram, and Grocott stains). Other physical findings and routine laboratory tests were normal. A diagnosis of mucocutaneous leishmaniasis secondary to *Leishmania panamensis* was established.

Given the urgency of treatment due to the disease presentation, Liposomal amphotericin B (LAmB) at the dose of 3 mg/kg was initiated in February 2024. After twelve days of treatment, she developed acute kidney injury (serum creatinine: 5.7 mg/dL), and since the cumulative target dose of 40 mg/kg had been reached, treatment was discontinued. She remained hospitalized for seven days, during which her renal function was monitored, and she received intravenous hydration. After a total hospital stay of twenty days, the skin lesions on her forehead and abdomen had shown slight improvement. As she no longer met the criteria for hospitalization, she was discharged for outpatient follow-up. Thirty days later, she returned to the outpatient clinic for a check-up, showing visible improvement. After a four-month follow-up, the patient remained asymptomatic, with no new skin lesions and no recurrence of the previous lesions (Figs. 3a – 3o).

DISCUSSION

The WHO recognizes leishmaniasis as an emerging, undercontrolled, and neglected infection affecting millions each year. Increased conflict and forced displacement in endemic areas of cutaneous leishmaniasis have led to a surge in cases, both in endemic countries and in clinics worldwide, with imported cases being reported in non-endemic countries [5,6]. One case was reported in the United States in an immunocompromised immigrant, where diagnosis was made thanks to the high index of suspicion of the healthcare personnel [7]. In Turkey, Şakru et al. published a systematic review on leishmaniasis among migrants and refugees, with the majority of cases reported in Syrian people [8]. Another case involved a pediatric patient from Venezuela

diagnosed in the north of Mexico, a region historically free of leishmaniasis [9]. Lemieux et al. described a ten-year case series in Canada in which the rise in cases was attributed to increased travel and migration. In this series, the median time for diagnosis was 89 days, and LAmB was the most commonly used treatment [10].

These cases highlight how a disease once confined to specific regions has become an emerging global health concern. This infection spreads rapidly in overcrowded camps, which provide ideal conditions for sandfly breeding. Migrants often experience delayed diagnosis and treatment due to reduced healthcare access. Furthermore, in many areas, even those bordering endemic countries, healthcare providers may lack the awareness and experience needed to diagnose and treat leishmaniasis effectively [11].

The epidemiology of cutaneous leishmaniasis in the Americas is complex, characterized by multiple circulating *Leishmania* species, several reservoir hosts and sandfly vectors, and variable clinical manifestations and therapy response. *L. panamensis* strains may exhibit different levels of virulence [12]. While cutaneous leishmaniasis is generally considered mild, 1–10% of patients infected with a strain from the *Viannia* subgenus subsequently develop mucocutaneous leishmaniasis, which can be life-threatening and highly disfiguring [13]. *L. panamensis* is also associated with diffuse cutaneous leishmaniasis, but this is more frequently seen in immunocompromised patients [14]. The lesional parasites in MCL are rarely present, but there have been reports of a good treatment response, especially with pentamidine for *L. panamensis* [15].

Clinical suspicion is essential, although some differentials can be taken into account, depending on the context of the person's case. These differentials include cutaneous cryptococcus, cutaneous histoplasma, coccidioidomycosis, paracoccidioidomycosis, sporotrichosis, chromoblastomycosis, yaws, mycobacterial infections, cutaneous neoplasms, *Ballamuthia mandrilaris* infection, and partially healed bacterial skin infections [6].

Although no single reference test exists, the observation of amastigotes in a clinical specimen confirms the diagnosis [16]. Given the limited sensitivity of tissue-sampling approaches, a combination of methods is recommended, especially molecular techniques that amplify nuclear or kinetoplast DNA. Recently developed real-time kDNA PCR assays have demonstrated high accuracy in detecting and quantifying *Viannia* species in lesion biopsies [17]. Treatment decisions are driven by the need to accelerate cure, reduce scarring, and prevent progression to mucocutaneous leishmaniasis. This may be supported by diagnosis and species identification.

Historically, pentavalent antimonials have been considered the first-line treatment, but they are associated with toxicity and risk of resistance. Amphotericin B (including lipid formulations) emerged as second-line therapy [18]. Due to the limited availability of pentavalent antimonials, liposomal amphotericin B is often used for the urgent management of these conditions. Observational studies have suggested pan-species cure rates with liposomal amphotericin B of 80% to 90%, but no controlled trials have been conducted [4]. Only one case of *L. panamensis* has been described, associated with a Mexican hospital in a returning traveler [19].

CONCLUSION

Global migration has contributed to the rising burden of leishmaniasis. At the same time, management remains challenging due to low index of suspicion, limited sensitivity of diagnostic tests, and limited availability of molecular methods. Access to pentavalent antimonials is limited, and their toxicity is high; the need for other treatments or first-line agents is essential, considering the difficulty of follow-up in migrant populations. Further research is needed to address evidence gaps, improve therapeutic options, understand pathogenesis, mitigate adverse drug effects and resistance.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. CSIS. Mind the Darién Gap, Migration Bottleneck of the Americas. Washington (DC): Center for Strategic and International Studies; 2024 May 16. Available from: https://www.csis.org/analysis/mind-darien-gap-migration-bottleneck-americas. Note: Not a MEDLINE-indexed journal article.

2. Abadías-Granado I, Diago A, Cerro PA, Palma-Ruiz AM, Gilaberte Y. Cutaneous and mucocutaneous leishmaniasis. Actas Dermosifiliogr (Engl Ed). 2021;S0001-7310:00108-3.
3. Hernández-Rivera MP, Hernández-Montes O, Chiñas-Pérez A, Batiza-Avelar JM, Sánchez-Tejeda G, Wong-Ramírez C, et al. Study of cutaneous leishmaniasis in the state of Campeche (Yucatan Peninsula), Mexico, over a period of two years. Salud Publica Mex. 2015;57:58-66.
4. Aronson N, Herwaldt BL, Libman M, Pearson R, Lopez-Velez R, Weina P, et al. Diagnosis and treatment of leishmaniasis: Clinical practice guidelines by the Infectious Diseases Society of America (IDSA) and the American Society of Tropical Medicine and Hygiene (ASTMH). Clin Infect Dis. 2016;63:1539-57.
5. De Vries HJC, Schallig HD. Cutaneous leishmaniasis: A 2022 updated narrative review into diagnosis and management developments. Am J Clin Dermatol. 2022;23:823-40.
6. Burza S, Croft SL, Boelaert M. Leishmaniasis. Lancet. 2018;392:951-70.
7. De La Hoz A, Gadi N, Lopez CG, Barrera-Godinez A, Miller NS, Bourque DL, et al. Immune reconstitution inflammatory syndrome in a patient with cutaneous leishmaniasis and HIV: A diagnostic challenge for clinicians caring for a migrant population in the United States. Open Forum Infect Dis. 2024;11:ofae587.
8. Şakru N, Özbel Y, Töz S. Refugees/immigrants and leishmaniasis in the world's largest hosting country, Türkiye: A systematic review. PLoS Negl Trop Dis. 2025;19:e0012947.
9. Gómez-Ponce CA, Pérez-Barragán E, Méndez-Palacios DM, Ramírez-Romero KO, Pérez-Cavazos S. Emerging infectious diseases and migration: A case of leishmaniasis in northern Mexico. Lancet Infect Dis. 2023;23:648-50.
10. Lemieux A, Lagacé F, Billick K, Ndao M, Yansouni CP, Semret M, et al. Cutaneous leishmaniasis in travellers and migrants: A 10-year case series in a Canadian reference centre for tropical diseases. CMAJ Open. 2022;10:E546-53.
11. Kim J, Zieneldien T, Ma S, Cohen BA. Cutaneous leishmaniasis in the context of global travel, migration, refugee populations, and humanitarian crises. Clin Pract. 2025;15:77.
12. Urrea DA, Duitama J, Imamura H, Álzate JF, Gil J, Muñoz N, et al. Genomic analysis of Colombian Leishmania panamensis strains with different level of virulence. Sci Rep. 2018;8:17336.
13. Marsden PD. Mucosal leishmaniasis ("espundia" Escomel, 1911). Trans R Soc Trop Med Hyg. 1986;80:859-76.
14. Mann S, Frasca K, Scherrer S, Henao-Martínez AF, Newman S, Ramanan P, et al. A review of leishmaniasis: Current knowledge and future directions. Curr Trop Med Rep. 2021;8:121-32.
15. Olivo Freites C, Gundacker ND, Pascale JM, Saldaña A, Diaz-Suarez R, Jimenez G, et al. First case of diffuse leishmaniasis associated with Leishmania panamensis. Open Forum Infect Dis. 2018;5:ofy281.
16. Vega-López F. Diagnosis of cutaneous leishmaniasis. Curr Opin Infect Dis. 2003;16:97-101.
17. Jara M, Adaui V, Valencia BM, Martinez D, Alba M, Castrillon C, et al. Real-time PCR assay for detection and quantification of Leishmania (Viannia) organisms in skin and mucosal lesions: Exploratory study of parasite load and clinical parameters. J Clin Microbiol. 2013;51:1826-33.
18. Pradhan S, Schwartz RA, Patil A, Grabbe S, Goldust M. Treatment options for leishmaniasis. Clin Exp Dermatol. 2022;47:516-21.
19. Mann S, Phupitakphol T, Davis B, Newman S, Suarez JA, Henao-Martínez A, et al. Case report: Cutaneous leishmaniasis due to Leishmania (Viannia) panamensis in two travelers successfully treated with miltefosine. Am J Trop Med Hyg. 2020;103:1081-4.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Report

Efficacy of JAK inhibitors in the treatment of alopecia: A case report of 10 patients

Asmaa Lahrougui, Mariem Aboudourib, Layla Bendaoud, Ouafa Hocar, Said Amal

Department of Dermatology and Venereology, Mohammed VI university hospital-Marrakech-Morocco.

Corresponding author: Asmaa Lahrougui, MD, E-mail: asmaa.lahrougui@gmail.com

ABSTRACT

The efficacy of JAK inhibitors in the treatment of alopecia has been proven by numerous studies, yet the short- and long-term evolution remains unpredictable. We conducted a prospective study of 10 patients with different forms of alopecia, with two common denominators: failure to respond to any of the topical and systemic treatments for alopecia, treated with tofacitinib 11 mg/day for an average of thirteen months with complete regrowth in 80% of the patients, with only one case of no response to treatment. Long-term evolution showed three cases of relapse after three months of treatment discontinuation, with no side effects reported in any of the patients. Although JAK inhibitors have been shown to be effective in treating alopecia, their suspensory effect on the discontinuation of treatment is significant, hence the importance of informing the patient before initiating treatment.

Key words: Treatment efficacy, JAK inhibitors, Alopecia, Therapeutic response

INTRODUCTION

The increasing incidence of alopecia in its various clinical forms and its impact on the patient's quality of life have highlighted the need for an effective and safer therapeutic option.

Particular interest has been shown in molecules that inhibit the JAK-STAT signaling pathway: JAK inhibitors. Their efficacy has been proven by numerous studies, yet the therapeutic response is person-dependent and, above all, unpredictable, with a significant suspensory effect.

Moreover, the use of JAK inhibitors is not without risk and may be subject to numerous side effects, hence the need to respect both the indications and contraindications of these molecules, as well as to perform rigorous clinical and biological monitoring.

CASE REPORT

We conducted a prospective study on 10 patients with different forms of alopecia treated with tofacitinib 11 mg/day for an average of thirteen months.

The average age was 31.5, ranging from 10 to 55 years, with a female predominance (60%, n = 6).

Herein, we describe ten cases of alopecia, including 5 universal, 4 decalvating, and 1 plaque, with two common denominators: failure to respond to any of the topical and systemic treatments for alopecia, notably inflitrations, bolus and minipulses of corticosteroid therapy, weekly treatment with methotrexate, and the significant psychological impact.

Dermoscopy of all patients revealed black and yellow spots, as well as several downy hairs in places. The therapeutic decision was to treat the patients with tofacitinib 11 mg/day after a pre-therapeutic workup, which proved normal in all patients.

Clinical evolution was marked by partial hair regrowth at 6 months in 90% of the patients and complete regrowth between 12 and 13 months in 80%, with only one case of no response to treatment (Figs. 1 – 3).

How to cite this article: Lahrougui A, Aboudourib M, Bendaoud L, Hocar O, Amal S. Efficacy of JAK inhibitors in the treatment of alopecia: A case report of 10 patients. Our Dermatol Online. 2026;17(2):215-218.

Submission: 30.01.2025; **Acceptance:** 04.04.2025

DOI: 10.7241/ourd.20262.13

Figure 1: Initial appearance of patient 1 (a), partial regrowth after 6 months of treatment (b), total regrowth at 13 months (c).

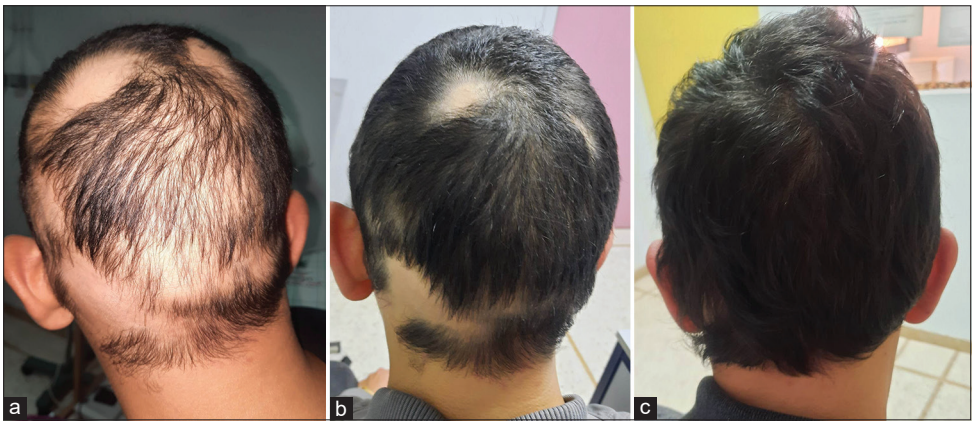


Figure 2: Initial appearance of patient 2 (a), partial regrowth after 6 months of treatment (b), full regrowth at 13 months (c).

Figure 3: Initial appearance of patient 3 (ab), full regrowth at 13 months (cd).

Long-term evolution showed three cases of relapse after three months of treatment discontinuation, with no side effects reported in any of the patients.

DISCUSSION

Alopecia is an autoimmune disease resulting from an attack on the hair follicle by autoreactive CD8 T cells [1]. Interleukin 15 (IL-15), which is secreted by hair follicles, activates CD8 T lymphocytes via JAK1 and 3, which promote the secretion of interferon-gamma, which by binding with hair follicles via JAK-1 and JAK-2, generates greater secretion of IL-15 responsible for a vicious circle.

It would, therefore, seem that JAK inhibitors agents could interrupt the pathogenesis of alopecia by acting

on the hair follicle via JAK-1 and JAK-2, or by acting on T lymphocytes via JAK-1 and JAK-3 [2,3].

Among the JAK inhibitors most frequently used in the treatment of alopecia, that is, tofacitinib (JAK 1-2-3), baricitinib (JAK 1-2), and ruxolitinib (JAKS 1-3), topical forms are also available, notably, ruxolitinib cream 1.5% and tofacitinib cream 2% [4-6].

Treatment duration generally depends on the therapeutic response and individual tolerance, dosing varies according to the molecule. For ruxolitinib, the dose is 5–25 mg twice daily; for tofacitinib, the dose is 11 mg as a single dose or 5 mg twice a day; and for baricitinib, the dose is 4 mg/day [1,7].

Like all drugs, JAK inhibitors are not innocuous treatments, so it is essential to ask for a pre-treatment check-up to detect any contraindications, including a blood count, renal, liver, and lipid profiles, and serological tests.

JAK inhibitors are blamed for a multitude of side effects, including a higher incidence of common infections, notably upper respiratory tract infections, urinary tract infections, as well as an increased incidence of tuberculosis, shingles, and other opportunistic infections [8-10].

The risk of developing neoplasia, notably lymphoma and melanoma, has been incriminated by certain cohorts [11-13], there is also the possibility of developing toxidermia [1] or cardiovascular and thromboembolic diseases [1,14,15], as well as biological disturbances [1], hence the importance of conducting a check-up during the first month of treatment and then quarterly, including a CBC and liver, kidney, and lipid tests.

As with all immunosuppressive drugs, live attenuated vaccines are contraindicated; if they are necessary, they must be administered at least three weeks before the beginning of treatment, whereas all inactivated vaccines may be administered [1,16].

Although JAK inhibitors have been shown to be effective in treating alopecia [17], their suspensory effect on the discontinuation of treatment is significant, hence the importance of informing the patient before initiating treatment.

CONCLUSION

The efficacy of anti-JAKs in the treatment of alopecia has been proven by numerous studies, yet the short- and long-term evolution remains unpredictable and, above all, person-dependent.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Damsky W, King BA. JAK inhibitors in dermatology: The promise of a new drug class. J Am Acad Dermatol. 2017;76:736-44.
2. Xing L, Dai Z, Jabbari A, Cerise JE, Higgins CA, Gong W, et al. Alopecia areata is driven by cytotoxic T lymphocytes and is reversed by JAK inhibition. Nat Med. 2014;20:1043-9.
3. Ghoreschi K, Jesson MI, Li X, Lee JL, Ghosh S, Alsup JW, et al. Modulation of innate and adaptive immune responses by tofacitinib (CP-690,550). J Immunol. 2011;186:4234-43.
4. Craiglow BG, Tavares D, King BA. Topical ruxolitinib for the treatment of alopecia universalis. JAMA Dermatol 2016;152:490-1.
5. Putterman E, Castelo-Soccio L. Topical 2% tofacitinib for children with alopecia areata, alopecia totalis, and alopecia universalis. J Am Acad Dermatol. 2018;78:1207-9.
6. Bokhari L, Sinclair R. Treatment of alopecia universalis with topical Janus kinase inhibitors: A double-blind, placebo, and active controlled pilot study. Int J Dermatol. 2018;57:1464-70.
7. Mackay-Wiggan J, Jabbari A, Nguyen N, Cerise JE, Clark C, Ulerio G, et al. Oral ruxolitinib induces hair regrowth in patients with moderate-to-severe alopecia areata. JCI Insight. 2016;1:e89790.
8. Winthrop KL, Curtis JR, Lindsey S, Tanaka Y, Yamaoka K, Valdez H, et al. Herpes zoster and tofacitinib: Clinical outcomes and the risk of concomitant therapy. Arthritis Rheumatol. 2017;69:1960-8.
9. Winthrop KL, Park SH, Gul A, Cardiel MH, Gomez-Reino JJ, Tanaka Y, et al. Tuberculosis and other opportunistic infections in tofacitinib-treated patients with rheumatoid arthritis. Ann Rheum Dis. 2016;75:1133-8.
10. Adas MA, Alveyn E, Cook E, Dey M, Galloway JB, Bechman K. The infection risks of JAK inhibition. Expert Rev Clin Immunol. 2022;18:253-61.
11. Maneiro JR, Souto A, Gomez-Reino JJ. Risks of malignancies related to tofacitinib and biological drugs in rheumatoid arthritis: Systematic review, meta-analysis, and network meta-analysis. Semin Arthritis Rheum 2017;47:149–56.
12. Russell MD, Stovin C, Alveyn E, Adeyemi O, Chan CKD, Patel V, et al. JAK inhibitors and the risk of malignancy: A meta-analysis across disease indications. Ann Rheum Dis. 2023;82:1059-67.
13. Furqan M, Mukhi N, Lee B, Liu D. Dysregulation of JAK-STAT pathway in hematological malignancies and JAK inhibitors for clinical application. Biomark Res. 2013;1:5.
14. Xie W, Huang Y, Xiao S, Sun X, Fan Y, Zhang Z. Impact of Janus kinase inhibitors on risk of cardiovascular events in patients with rheumatoid arthritis: Systematic review and meta-analysis of

randomised controlled trials. Ann Rheum Dis. 2019;78:1048-54.
15. Cohen SB, Tanaka Y, Mariette X, Curtis JR, Lee EB, Nash P, et al. Long-term safety of tofacitinib for the treatment of rheumatoid arthritis up to 8.5 years: Integrated analysis of data from the global clinical trials. Ann Rheum Dis. 2017;76:1253-62.
16. Winthrop KL, Silverfield J, Racewicz A, Neal J, Lee EB, Hrycaj P, et al. The effect of tofacitinib on pneumococcal and influenza vaccine responses in rheumatoid arthritis. Ann Rheum Dis. 2016;75:687-95.
17. Yan D, Fan H, Chen M, Xia L, Wang S, Dong W, et al. The efficacy and safety of JAK inhibitors for alopecia areata: A systematic review and meta-analysis of prospective studies. Front Pharmacol. 24;13:950450.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Report

Inoculation canker sores during genital transmission of mpox: A report of two cases

Ida Lenga Loumingou[1], Stéphanie Ntsame Ngoua[2], Ousmane Faye[3], Raphaël Taty Taty[4]

[1]Department of Dermatology, University of Brazzaville Medical Center, Brazzaville, Republic of the Congo, [2]Department of Dermatology, University Hospital Center of Libreville, Gabon, [3]Director of the Bamako Dermatology Hospital, University Hospital Center specialized in Dermatology in Mali, [4]Infectious Disease Specialist in Pointe-Noire, Republic of Congo

Corresponding author: Ida Lenga Loumingou, MD, E-mail: idalengaloumingou@gmail.com

ABSTRACT

Mpox was recently classified as an emerging communicable disease. Ubiquitous, mpox is more common in sub-Saharan Africa. The morbidity and mortality caused by mpox are concerning, and its etiopathogenesis is debated. It is transmitted through direct contact. Cases of sexual transmission are increasingly being reported. Two adolescents, aged 20 and 22, were referred by the emergency department for a rash. Clinical examination revealed disseminated umbilicated papules and a chancre of the balanoprenoptic fold in one and a pubic fold in the other, with bilateral inguinal lymphadenopathy. Treponemal and chlamydial serologies were negative. There were no treponemes or hemophilus on direct examination after analysis of the chancroids' serosity; PCR performed on one papule was positive for mpox. The etiopathogenesis of mpox remains debated. The presence of a genital chancre is a strong argument for diagnosis.

Key word: MPOX; sexual contamination; chancre

INTRODUCTION

Mpox is a ubiquitous zoonosis transmitted by contact, declared a public health emergency of international concern on August 14, 2024 [1,2]. Central Africa is the most affected region [2]. During the disease, the symptomatology is dominated by cutaneous involvement. This localization helps guide the diagnosis [1]. In 2023, cases of sexual contamination of mpox were reported in West Africa [3,4] without specifying the mechanism. Herein, we report two transmissions of mpox with inoculation chancres.

Case 1

A twenty-year-old adolescent boy presented with a pubic chancre two days after sexual contact (Fig. 1). A disseminated papular rash with progressive onset appeared one week after the chancre. The papules were umbilicated and non-pruritic. The patient had a fever of 38°C, without chills. He had inflammatory inguinal lymphadenopathy. Serological tests for syphilis, HIV, and chlamydia were negative. A sample of serum from the chancre revealed no bacteria. PCR performed on a papule was positive for mpox.

Case 2

This case involved a 22-year-old man who presented to the emergency department with disseminated papular lesions predominantly affecting the pelvic limbs and abdomen. These lesions had been progressing for one week. Clinical examination revealed a linear, oozing ulcerated lesion in the balano-preputial sulcus (Fig. 2). This painless chancre preceded the eruption by five days, and bilateral, tender, and firm lymphadenopathy was noted. Syphilis serology was negative. There were no treponemes or Donovan bodies on direct examination of the chancre. PCR of one papule was positive for mpox.

How to cite this article: Lenga Loumingou I, Ntsame Ngoua S, Faye O, Taty Taty R. Inoculation canker sores during genital transmission of mpox: A report of two cases. Our Dermatol Online. 2026;17(2):219-221.

Submission: 21.12.2025; **Acceptance:** 18.02.2026

DOI: 10.7241/ourd.20262.14

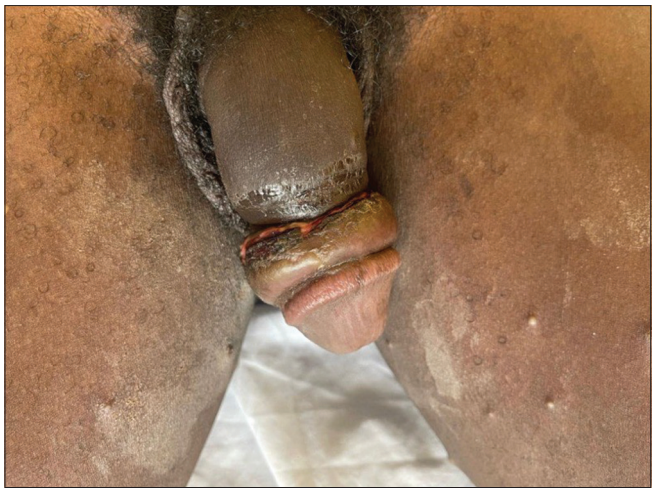


Figure 1: Penile chancre and ambilicated pubic papules.

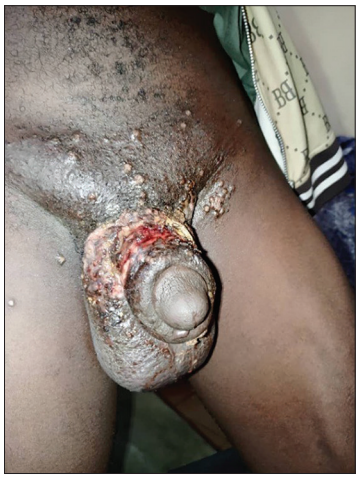


Figure 2: Chancre of the balano-preputial groove and ambilicate papules on the thighs.

DISCUSSION

These were both patients with mpox with typical skin lesions preceded by chancres in the genital area several days after sexual contact. The notion of a sexually transmitted infection has been raised for mpox [4,5]. Mpox typically presents with an incubation period of twelve days on average, followed by an infectious syndrome and a maculopapular eruptive phase of fisiculopustules and diffuse crusts, followed by polyadenopathy [1]. The rash appears to predominate in the anogenital regions. Inoculation chancre is not classic [6]; its presence could mislead the diagnosis and suggest pathologies such as herpes, chancroid, or primary syphilis. The chancre in MPOX does not appear to have a typical characteristic. In both of our cases, it has irregular shapes, detached edges, and an oozing base. Harnessed lymphadenopathy is present in both cases. These bilateral, inflammatory inguinal lymphadenopathies are firm and of variable size. Chancres should be systematically sought in other mucous membranes. It would be wise to routinely perform PCR for mpox on a chancre in endemic areas.

There is, therefore, a variability of clinical forms, ranging from simple forms to severe forms [7]. Diffuse skin forms are the most described [8], but mucosal locations, especially genital ones, represent a particular epidemiological and diagnostic interest [9,10].

CONCLUSION

The etiopathogenesis of mpox infection still raises questions. This pathology may be considered an STI for which an inoculation chancre should be systematically sought.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Zinsou A, Clause O, Thonneau P. Cartographie des cas de Monkey en Afrique quemiologie et santé publique. 2023;71:101998.
2. Lemaille C, Halbrook M, Merritt S, Anta Y, Lunyanga L, Mukadi PK, et al. Assessing mpox knowledge and sexual behaviours within high-risk populations in the Democratic Republic of the Congo. medRxiv. 2025:2025.04.20.25326123.
3. Low N, Bachmann LH, Ogoina D, McDonald R, Ipekci AM, Quilter LAS, et al. Mpox virus and transmission through sexual contact: Defining the research agenda. PLoS Med. 2023;20:e1004163.
4. Yadaw R, Chaudary AA, Srolavasva U. MPOX 2022 to 2025 update: A comprehension review on its compilations, transmission, diagnostics, treatment. Viruses. 2025;17:753.
5. Ebede SO, Orabueze IN, Maduakor UC, Nwafia IN, Ohanu ME. Recurrent Mpox: Divergent virulent clades and the urgent need for strategic measures including novel vaccine development to sustain global health security. BMC Infect Dis. 2025;25:536.
6. Zhu WF, Song SJ, Wei LW, Qiao JJ. Monkeypox presenting as a chancre-like rash: A case report. World J Clin Cases. 2023;11:6025-30.
7. Corona-Herrera JM, González-Torres JA, Bermúdez-Rodríguez SP, Méndez Flores S, Domínguez-Cherit JG. Monkeypox fulminante: une partie du spectre clinique du syndrome inflammatoire de reconstitution immunitaire ? Our Dermatol Online. 2023;14:393-5.
8. Sigha OB, Medi SC, Nkoro GA, Ndom MS, Mbenoun ML, Kamdem J, Essomba NE, Kouotou EAS. Défis dans la prise en

charge du Monkeypox dans un pays en développement: cas du Cameroun. Our Dermatol Online. 2024;15(Supplément 1):24-9.
9. Nasaha TK, Nagesh M, Riyaz N, Fathima S. Mpox: A recent addition to the differential diagnosis of genital ulcer disease. CosmoDerma. 2025;5:68.
10. Sasidharanpillai S. Monkeypox: An update. J Skin Sex Transm Dis. 2022;4:149.

Source of Support: This article has no funding source.
Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Report

Kindler syndrome with typical clinical manifestations: A case report from Syria

Lina Al Soufi[1], Razan Younis[1], Heba Fawal[1], Zuheir Al-Shehabi[2]

[1]Department of Dermatology, National Hospital, Lattakia, Syria, [2]Department of Pathology, Faculty of Medicine, Tishreen University, Lattakia, Syria

Corresponding author: Razan Younis, MD, E-mail: razanmallayounis@gmail.com

ABSTRACT

Kindler's syndrome is a highly rare hereditary disorder, a form of epidermolysis bullosa, caused by a mutation in the KIND1 gene, which is characterized clinically by photosensitivity, poikiloderma, trauma-induced blistering that begins at birth, skin atrophy, mucosal involvement, and a risk of malignancy. Herein, we present a case of Kindler's syndrome and highlight the classical manifestations and associated features of this rare syndrome and diagnosis methods, differential diagnosis, and the necessity of a multidisciplinary team to control the symptoms and improve the patient's quality of life.

Key words: Kindler's syndrome, Photosensitivity, Blistering, Fragility, Poikiloderma, Onycholysis

INTRODUCTION

Kindler's syndrome (KS) is a rare autosomal recessive condition. These genodermatosis, which was first described by Theresa Kindler in the 1950s and classified as a rare form of epidermolysis bullosa, is the outcome of a mutation in the KIND1 or FERMT1 genes. In turn, this mutation makes the skin susceptible to increased photosensitivity, blistering, and fragility [1]. Throughout the world, there are only about 250 cases reported [2,3]. This syndrome is considered to be a combination of manifestations of congenital poikiloderma and inherited blistering skin disorders [3,4].

KS may affect persons of any race and may affect both men and women with no sex predilection [5].

The disease's severity may range from mild symptoms such as blisters and skin ulcers, to more severe symptoms with mucosal involvement, severe esophageal stenosis, anemia, and rarely, colitis [6].

Herein, we report the case of a 39-year-old woman with KS and no familial history.

CASE REPORT

A 39-year-old unmarried woman presented to the dermatology department with complaints of photosensitivity and poikiloderma on sun-exposed areas (face, neck, and V area of the upper chest) (Figs. 1a and 1b). Throughout her life, she suffered from recurrent blistering after minor trauma beginning after birth. The changes were more prominent on the extremities and tended to disappear after the age of 13 years. Subsequently, discoloration and atrophy of the skin developed. Also, she had palmoplantar hyperkeratosis (Fig. 1c), in addition to swallowing difficulties with solid foods and oral medications because of esophageal strictures. Cutaneous examination revealed atrophic, dry skin with cigarette paper-like wrinkling, diffuse poikiloderma (atrophy, telangiectasia, and reticular pigmentation) mainly on the sun-exposed areas (Figs. 1a – 1d). There was also eye redness, ectropion, actinic cheilitis with reduced ability to open the mouth, gingivitis, periodontitis (Fig. 1e) and nail changes, including subungual hyperkeratosis, onycholysis, and transverse and longitudinal ridges (Fig. 1c).

How to cite this article: Lina Al Soufi, Razan Younis, Heba Fawal, Zuheir Al-Shehabi. Kindler syndrome with typical clinical manifestations: A case report from Syria. Our Dermatol Online. 2026;17(2):222-225.

Submission: 30.10.2025; **Acceptance:** 08.12.2025

DOI: 10.7241/ourd.20262.15

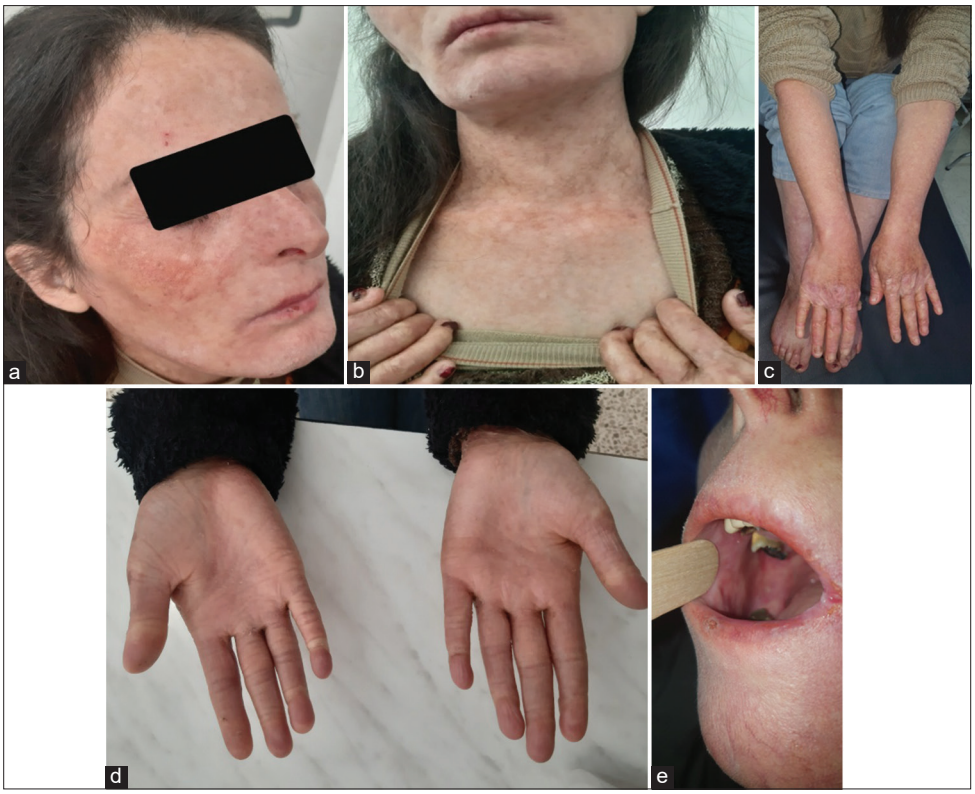


Figure 1: (a and b) Hypo- and hyperpigmented macules with telangiectasia and photosensitivity on the patient's face, neck, and V area of the upper chest. (c) Atrophic skin with cigarette paper-like wrinkling of the skin. Nail changes, including subungual hyperkeratosis, and onycholysis. (d) Absence of dermatoglyphics. (e) Erosions in the buccal mucosa, angular cheilitis with poor oral hygiene and periodontitis.

Two skin punch biopsies were taken from the forearm and lower leg. Histopathology of these biopsies showed epidermal atrophy with hyperkeratosis, focal liquefaction degeneration of the basal layer with pigmentary incontinence (Fig. 2a). There was dermal edema with dilated blood vessels and a patchy perivascular mononuclear inflammatory cell infiltrate (Fig. 2b).

Based on the patient's history, clinical features, and histopathological findings, Kindler syndrome was diagnosed. For confirmation, genetic testing was advised, but because of the financial limitations, the patient could not afford it.

DISCUSSION

Kindler syndrome is a subtype of epidermolysis bullosa that results from a mutation in the FERMT1 gene that encodes kindlin-1 protein [7]. Intermittent cases are not uncommon [4].

KS has been mainly described in Arab origin, as from Pakistan, Iran, Turkey, and India. It has also been reported in European individuals, as well as Caucasian, Albanian, Italian, and Serbian [3].

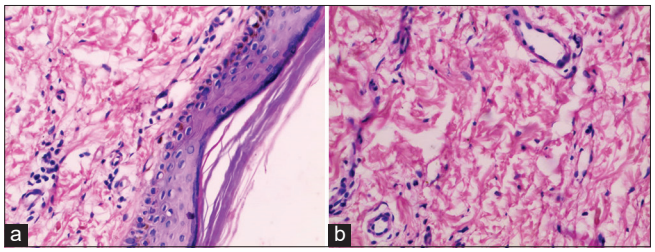


Figure 2: a) Histopathology of the biopsies showing epidermal atrophy, focal vacuolar degeneration of the basal layer with subepidermal cleft (H&E, 100x). b) Dilated blood vessels in the upper dermis (H&E, 200x).

Clinically, KS is characterized by minor trauma-induced blisters at birth or during the first days of child life, and these blisters regress with age. Other features, including photosensitivity, skin fragility, poikiloderma, diffuse skin atrophy, particularly in sun-exposed areas, tend to happen in infancy or early childhood. While photosensitivity becomes better with age, atrophy and poikiloderma worsen over time [3].

KS also has mucosal involvement, which usually begins during adolescence. The most commonly affected mucosa is the oral mucosa, leading to hemorrhagic gingivitis, premature tooth loss,

periodontal disease [2], xerostomia, oral ulcerations, and microstomia [5].

After the age of 10, ocular, esophageal, anal, and urogenital mucosa involvement is more frequent [2].

The ocular features of this disorder include kerato-conjunctivitis, cicatricial ectropion, blepharitis, recurrent corneal erosions, corneal ectasia, pigment deposits over the lens capsule, symblepharon, segmental chorioretinal atrophy, and conjunctival scarring [7].

The patients with KS have an increased risk of developing squamous cell carcinoma of the skin after the age of 45 [2].

The histopathology of Kindler syndrome shows poikilodermatous changes [4].

Numerous diseases that may result in cutaneous atrophy, blistering, and/or poikiloderma should be differentiated from KS, [4] including Weary syndrome, Bloom syndrome, Rothmund–Thompson syndrome, Cockayne syndrome [8], X-linked recessive epidermolysis bullosa simplex, dyskeratosis congenita, Xeroderma pigmentosum, and dermatopathia pigmentosa reticularis [9].

It has also been proposed that KS and Weary's HAP are variants of one condition themselves [4]. However, the pattern of inheritance of Weary is autosomal dominant, while it is autosomal recessive in KS [9,10]. Also, the occurrence of blistering, photosensitivity, and the presence of eczema is not the same in these two syndromes [10]. In Kindler syndrome, photosensitivity is evident while, in Weary syndrome, there is no photosensitivity. Also, blisters begin arising within the first six months of life in Weary syndrome, unlike KS, where blisters begin at birth, in addition to widespread dermatitis in Weary syndrome, which is absent in KS [9,10].

It might be difficult to distinguish KS from variants of epidermolysis bullosa in newborns. However, in KS, there is the progressive improvement of photosensitivity, blistering, poikiloderma, and cutaneous atrophy over time, unlike epidermolysis bullosa [4].

The associated manifestations in Rothmund–Thomson syndrome, an autosomal recessive disease, such as hypogonadism, sparse fine scalp hair, alopecia, microdontia, and rarely, mental retardation, are different from KS [9,10].

There is no true poikiloderma in Bloom syndrome, contrary to KS. In Cockayne's syndrome, associated features, including dwarfism, progressive pigmentary retinopathy, cachexia, deafness, and birdlike faces, help to distinguish this syndrome from KS. In dyskeratosis congenita, unlike KS, the pigmentary alterations are not truly poikilodermatous, and bullae are not an essential feature of this uncommon genodermatosis [4].

In 2004, Angelova-Fischer et al. presented clinical criteria to facilitate the accurate diagnosis of this syndrome (Table 1).

Our patient had met all major criteria, one of the minor criteria, and six of the associated findings.

According to the suggested criteria, diagnosis is confirmed by the presence of four major criteria, diagnosis is probable by the presence of three major and two minor criteria, and diagnosis is likely when two major and two minor/additional features are present [10].

The diagnosis of KS may also be established by immunofluorescence mapping and/or detecting FERMT1 gene mutations [3].

Actually, there is no confirmed treatment for KS, and the management is mainly symptomatic and preventive [4,8].

The patient should avoid sun and heat exposure, apply sunscreens and moisturizers, observe skin malignancies,

Table 1: Diagnostic criteria for Kindler syndrome [8]

Major Criteria
Acral blistering in infancy and childhood
Progressive poikiloderma
Skin atrophy
Abnormal photosensitivity
Gingival fragility and/or swelling
Minor Criteria
Syndactyly
Mucosal involvement (urethral, anal, esophageal, laryngeal stenosis)
Associated Findings
Nail dystrophy
Ectropion of the lower lid
Palmoplantar keratoderma
Pseudoainhum
Leukokeratosis of the lips
Squamous cell carcinoma
Anhidrosis/hypohidrosis
Skeletal abnormalities
Poor dentition/dental caries
Periodontitis

and receive nutritional and psychosocial support [3,8]. The patient should also avoid skin trauma, take care of wounds, and treat infected bullous lesions and ulcerations with topical and systemic antibiotics to minimize the morbidity, which results from secondary infections of blisters, mucosal involvement and leads to anal, urethral, and esophageal stenosis, ocular complications, and aggressive periodontitis [3,4].

The life expectancy of patients with KS is usually normal, but they have a significant risk of developing malignancies, including squamous cell carcinoma on the lip, hard palate, and acral skin, in addition to transitional cell carcinoma of the bladder, which runs a severe course [3].

Patients with Kindler syndrome should be treated with a multidisciplinary team of dermatologists, ophthalmologists, urologists, dentists, gastroenterologists, pediatricians, dieticians, nurse specialists, psychologists, and geneticists [8].

CONCLUSION

Kindler syndrome is an extremely rare disorder. We presented a case with specific features of KS, its associated manifestations, histopathological findings, differential diagnosis and management and aimed to increase understanding of the classical clinical manifestations of KS to facilitate the diagnosis and minimize the need for invasive investigations, as well as ensure that treatment is made by a multi-specialization team.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Saaqib Y, Shahzadi N, Tahir Z, Azfar NA. Clinical diagnosis of Kindler syndrome: A report of two cases from a district hospital in Gujranwala, Pakistan. J Pak Assoc Dermatol. 2023;33:1106-11.
2. Torres-Iberico R, Condori-Fernández Y, Apagüeño-Ruiz C, Andia-Ticona M, Pomar-Morante R. Kindler syndrome: A multidisciplinary management approach. Actas Dermosifiliogr. 2020;111:775-80.
3. Gkaitatzi M, Kalloniati E, Has C, Kiritsi D, Spiliopoulos T, Georgiou S. Kindler syndrome: A rare case report from Greece. Oxford Med Case Rep. 2019;2019:omz003.
4. Suman N, Kaur S, Kaur S, Sarangal V. Kindler's syndrome: A rare case report. Contemp Clin Dent. 2014;5:217-20.
5. Blanchet I, Tardieu C, Casazza E. Oral care in Kindler syndrome: 7-year follow-up of 2 brothers. J Clin Pediatr Dent. 2021;45:41-7.
6. Idkaidak S, Albandak M, Alqarajeh F, Dukmak ON, Imhaimeed J, CN Khalil N. Kindler syndrome presenting as colitis in an infant. Cureus. 2023;15:e43928.
7. Maharana PK, Sahay P, Mandal S, Nagpal R, Sharma N. Ocular manifestations in Kindler syndrome. Indian J Ophthalmol. 2022;70:2585-7.
8. Ghorai R, Singh G, Mittal A, Panwar VK, Talwar H. Urological manifestations of Kindler syndrome: A case report. Cureus. 2022;14:e24758.
9. Fischer IA, Kazandjieva J, Vassileva S, Dourmishev A. Kindler syndrome: A case report and proposal for clinical diagnostic criteria. Acta Dermatovenerol Alp Pannonica Adriat. 2005;14:61-7.
10. Angelova-Fischer I, Kazandjieva J, Vassileva S, Dourmishev A. Kindler syndrome: A case report and proposal for clinical diagnostic criteria. Acta Derm Venereol Alp Pannonica Adriat. 2005;14:61-7.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Report

Wells' syndrome mimicking bullous infectious cellulitis in a 3-year-old child

Soukaina Lazouzi, Fatima-Zahra El Fatoiki, Fouzia Hali, Soumia Chiheb

Dermatology Department, Ibn Rochd Hospital Center, University Hassan II, Casablanca, Morocco

Corresponding author: Soukaina Lazouzi, MD, E-mail: souki08@hotmail.com

ABSTRACT

Wells' syndrome, or eosinophilic cellulitis, is a rare inflammatory dermatosis frequently associated with recurrence. Acute in onset and often presenting as an inflammatory or urticarial patch, it often leads to confusion with infectious cellulitis and, thus, treatment with antibiotics without resolution of symptoms. Histology is suggestive, showing an eosinophilic infiltrate with flame-like formations, and treatment relies primarily on topical or oral steroids. We, herein, report the case of a three-year-old girl who presented with an erysipelas-like eruption, which turned out to be Wells' cellulitis.

Key words: Wells' syndrome, Eosinophilic cellulitis, Cellulitis, Bullous cellulitis with eosinophilia, Case report

INTRODUCTION

Wells' syndrome, also named "eosinophilic cellulitis," is a rare self-limiting but often recurring inflammatory dermatosis [1]. It is uncommon amongst children and is clinically variable but most often manifests as pruritic urticarial plaques [2]. Herein, we report the case of a three-year-old child who presented with the bullous eosinophilic cellulitis variant mimicking infectious cellulitis.

CASE REPORT

A three-year-old girl, with no pathological history, presented with a tender plaque of the left lower limb that had been evolving for four days, with a history of an insect bite prior to symptom onset. Examination found a febrile child at 38.9°C, edema of the left leg extending beyond the knee, with erythema, bullae, and crusty lesions in places (Fig. 1a), and a similar plaque on the anterolateral side of the right thigh (Fig. 1b). There were no lymphadenopathies or other signs reported or found. A complete blood count and CRP revealed an elevated white blood cell count of 12,890/mm^3 (range = 4000–10,000) with an elevated eosinophil count of 3003/mm^3 (range = 100–500), a normal neutrophil count of 4924/mm^3, and a CRP of 10.98 mg/L (range < 6). The patient was initially initiated on oral antibiotics, as erysipelas was suspected; however, due to symptom persistence, treatment was switched to topical corticosteroids and oral antihistamines, leading to a favorable outcome. Histological analysis was not performed due to the rapid clinical improvement observed following treatment initiation, which made invasive diagnostic procedures unnecessary.

DISCUSSION

Wells' syndrome, also named Wells' cellulitis or eosinophilic cellulitis, is a rare and frequently recurring inflammatory dermatosis of unknown etiology, but potential etiological factors such as infections (e.g., varicella, and parvovirus), insect bites, medications (e.g., penicillin or infliximab), hematologic disorders and malignancies, radiotherapy, immunomodulators, vaccines, and contact dermatitis have been reported [1–3].

How to cite this article: Lazouzi S, El Fatoiki F-Z, Hali F, Chiheb S. Wells' syndrome mimicking bullous infectious cellulitis in a 3-year-old child. Our Dermatol Online. 2026;17(2):226-227.

Submission: 30.07.2025; **Acceptance:** 31.08.2025

DOI: 10.7241/ourd.20262.16

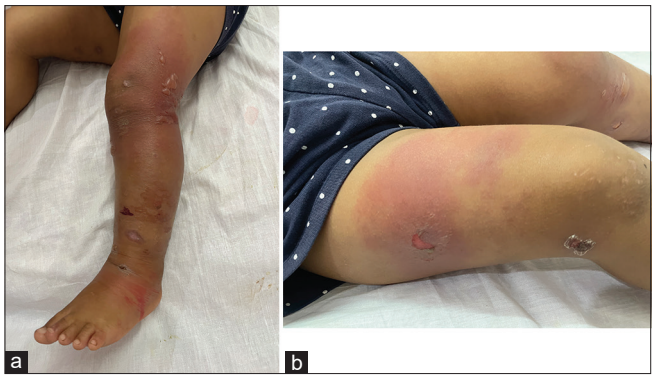


Figure 1: (a) Edema with erythema, bullae, and crusty lesions on the left leg of the 3-year-old child. (b) Erythematous plaque with bullae and post-bullous erosions on the right thigh of the 3-year-old child.

Authors describe a wide range of clinical presentations, including urticarial, vesiculobullous, nodular, papulonodular, and annular forms, preceded by itching or tenderness, often leading to confusion with other etiologies [3,4]. The main differential diagnosis, thus, remains infectious cellulitis, further supported by the acute onset and frequent association with fever, as was the case in our patient, in whom erysipelas was the initial diagnosis [5].

Paraclinical elements can help in the diagnosis and usually note hyperleukocytosis with peripheral hypereosinophilia and elevated CRP levels, although cases with normal CRP levels have been described, and our observation fits into this category [3].

The histopathological changes progress through three stages: an early phase with dermal edema and eosinophilic infiltration, a subacute phase marked by histiocyte infiltrates and flame figures, and a late phase with fewer eosinophils, histiocytes, and residual flame figures [6].

Although the disease usually resolves spontaneously within weeks, its recurring nature often calls for treatment, but no standard guidelines are available. Many patients are initially misdiagnosed and mistakenly treated with antibiotics, leading to no resolution of symptoms, further complicating the diagnostic process. Based on case reports and small series, treatment relies on local and oral steroids, with dapsone or cyclosporine for resistant cases, and in some instances, TNF-α inhibitors, omalizumab (anti-IgE), mepolizumab (interleukin-5 inhibitor) and benralizumab (interleukin-5 receptor inhibitor) have been proven to be successful [4].

CONCLUSION

Wells' syndrome remains a rare and frequently misdiagnosed condition, particularly in pediatric patients. This case highlights the importance of increasing awareness among clinicians to facilitate accurate diagnosis and effective management. Given its favorable prognosis and usually spontaneous resolution, systemic treatment should be reserved for cases unresponsive to local therapy or with extensive lesions.

ACKNOWLEDGMENTS

An abstract based on this case was accepted as a poster presentation at the EADV Symposium 2025, held in Prague, Czech Republic, in May 2025.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Abbasi MS, Ouellette S, Rao BK. A case report on eosinophilic cellulitis. J Community Hosp Intern Med Perspect. 2023;13:46-8.
2. Baabdullah AM, Hawsawi KAA, Alhubayshi BS, Gammash MR. Eosinophilic cellulitis (Wells syndrome): A case report. Int J Res Dermatol. 2021;7:450-3.
3. Deniz M, Demir-Önder K, Özkaraman Y, Adıgüzel Z, Balaban K. Clinical entity mimicking infectious cellulitis: Eosinophilic cellulitis (Wells' syndrome). Infect Dis Clin Microbiol. 2023;5:376-9.
4. Blomberg M, Winther C, Høyrup S, Skov L. Treatment of widespread eosinophilic cellulitis (Wells' syndrome) with benralizumab. Acta Derm Venereol. 2020;100:5943.
5. Gowda SK, Garg S, Behera B, Thakur V, Sahu DK. Mimickers of erysipelas and cellulitis: A narrative review. Indian J Dermatol Venereol Leprol. 2025;91:346-55.
6. Heelan K, Ryan JF, Shear NH, Egan CA. Wells syndrome (eosinophilic cellulitis): Proposed diagnostic criteria and a literature review of the drug-induced variant. J Dermatol Case Rep. 2013;7:113.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Report

Complications of great and small saphenous vein sclerotherapy: Case analysis and clinical recommendations

Mirela Vasileva[1,2], Dimitar Spoa[3]

[1]Faculty of Medical Sciences, Goce Delcev University, Shtip, Republic of North Macedonia, [2]Clinical Hospital-Stip, Shtip, Republic of North Macedonia, [3]Remedika General Hospital Skopje, Republic of North Macedonia

Corresponding author: Mirela Vasileva, MD, E-mail: mirelanaceva@yahoo.com

ABSTRACT

Sclerotherapy is a widely accepted treatment for reticular veins and telangiectasias, but its use in major truncal veins such as the great saphenous vein (VSM) and small saphenous vein (VSP) remains controversial. This paper presents several clinical cases of patients who developed serious complications, including non-healing ulcers and chronic limb edema, following truncal vein sclerotherapy. We discuss the contraindications for sclerotherapy in these large superficial veins, the pathophysiological reasons for complications, and review current best practices for the management of varicose veins, including endovenous thermal ablation and surgical options. Sclerotherapy in major truncal veins remains controversial due to frequent complications and the short-term effect.

Key words: Sclerotherapy, Varicose veins, Complications, Ulcers

INTRODUCTION

Varicose veins represent one of the most common manifestations of chronic venous disease, affecting up to 25–30% of the adult population. To improve the therapeutic possibilities, we must always begin from the base, that is, the etiology and pathophysiology of the disease [1].

Chronic venous insufficiency (CVI) is a condition where the flow in the veins is impaired and venous hypertension occurs. In CVI, several changes occur in the lower extremities, such as edema, trophic changes in the skin, and a feeling of heaviness in the legs. The diagnosis of CVI is based on clinical features according to the Clinical, Etiological, Anatomical, and Pathophysiological (CEAP) classification to improve consistency in the reporting, diagnosis, and management of CVI.

Most systemic diseases impact the process of vascularization, granulation, and finally epithelization of the ulcer tissue [1]. The primary underlying mechanism for CVI is valvular reflux, other etiologies involved in the disease process are venous stasis, arteriovenous malformation and failure of the calf muscle pump, lifestyle, lower abdominal surgery, pregnancy, obesity, lower extremity injury. There may also be a hereditary component, i.e., genetic disorders such as Klippel–Trénaunay and Parks Weber causing CVI. These etiological factors may lead to chronic endothelial inflammation and other pathophysiological changes [2].

The approach to CVI management involves several different strategies, including conservative therapies (e.g., compression, elevation, and exercise), pharmacologic treatments, and more invasive procedures (sclerotherapy, endovenous laser or radiofrequency ablation, surgical ligation) [2].

Sclerotherapy

While sclerotherapy is a simple and minimally invasive procedure widely used for treating varicose,

How to cite this article: Vasileva M, Spoa D. Complications of great and small saphenous vein sclerotherapy: Case analysis and clinical recommendations. Our Dermatol Online. 2026;17(2):228-233.

Submission: 24.10.2025; **Acceptance:** 01.12.2025

DOI: 10.7241/ourd.20262.17

telangiectasias, reticular, spider veins, and some hemorrhoids, its application in the great saphenous vein (VSM) and small saphenous vein (VSP) remains controversial.

Injection sclerotherapy induces vascular fibrosis, improves cosmesis, relieves venous insufficiency symptoms, and improves overall vascular health but in small venous blood vessels.

The great saphenous vein and small saphenous vein play a central role in venous return from the lower extremities, and obliteration with a sclerosant carries a risk of significant complications, including recanalization, thrombosis, pigmentation, tissue necrosis, and chronic edema.

The sclerosants commonly employed in current clinical practice may be classified as irritants, osmotic agents, or detergents [3].

Sclerosants damage endothelial cells of the intima and tunica. Irritants and osmotic sclerosants are directly cytotoxic; detergents are less cytotoxic yet disrupt intercellular junctions [3].

In this paper, we present several cases of severe complications following truncal vein sclerotherapy and discuss why this approach should be avoided in favor of evidence-based alternatives.

Contraindications and Complications in Sclerotherapy

There are absolute and relative contraindications to sclerotherapy for varicose veins. Absolute contraindications to sclerotherapy include patients with hypersensitivity to sclerosing agents, acute systemic infection, or acute localized infection at the site of treatment, deep vein thrombosis, peripheral arterial disease, systemic autoimmune connective tissue diseases.

Relative contraindications to sclerotherapy include pregnancy, lactation, impaired mobility, asthma, deep venous insufficiency, and thrombophilia.

Large varicose veins are also a contraindication to sclerotherapy due to the increased risk of recanalization, complications, and treatment failure.

In patients in whom sclerotherapy is contraindicated, other treatment options should be considered.

Complications of sclerotherapy can be systemic and local. Local complications occur at the injection site and include pain, erythema, edema, ulceration, pruritus, and telangiectatic changes. Approximately 30% of patients may experience skin hyperpigmentation or discoloration from hemosiderin deposition 6 to 8 weeks after sclerotherapy [3].

Systemic side effects of sclerotherapy include chest tightness, transient ischemic attacks, dizziness, visual problems, and headaches. These side effects are more common in patients who have had a problem with injecting the sclerosant into the wrong vein. A systematic review found no significant differences in complication rates or optimal outcomes between foam and liquid sclerotherapy [3].

The patient must always be advised that there is a risk of skin necrosis or cosmetic complications such as pigmentation and telangiectasias, all patients who are on oral contraceptives and other exogenous estrogens, tetracyclines as well as those receiving medications for psychiatric diagnoses may increase the risk of side effects or compromise optimal treatment outcomes [4].

Sclerotherapy of VSM/VSP is a poor choice because of the high recurrence rate. A foam or liquid sclerosant often fails to permanently occlude long truncal veins. Also, there is a risk of deep vein extension due to saphenofemoral or saphenopopliteal junction reflux. The sclerosant may enter the deep system. Tissue necrosis and ulceration are also important complications due to accidental extravasation or reflux into tributaries causing skin breakdown. A very common complication is chronic limb edema because of loss of major superficial venous drainage that may worsen venous hypertension if collaterals are inadequate.

Because VSP is close to the sural nerve, it can be damaged. Cosmetic complications are the most common but less dangerous; the legs are with pigmentation and matting.

CASE REPORT 1

A 68-year-old female patient came for an examination due to an ulcer on the right foot after sclerosis eight months previously (Fig. 1). She had been treated several times with bandages, antibiotics, but without improvement. She gave information that pronounced swelling, redness and wounds on the right lower leg occurred after intervention st. post sclerosatio VSM

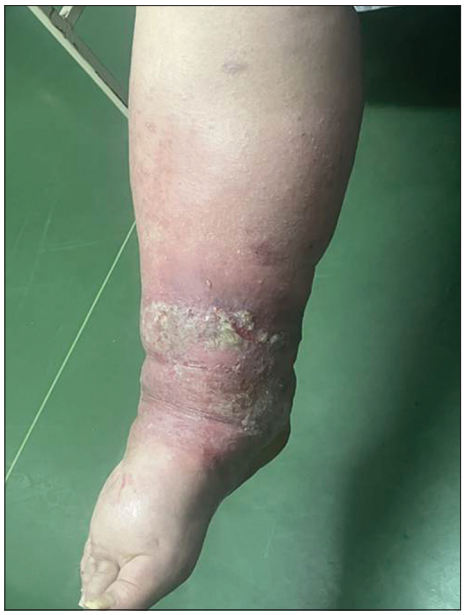


Figure 1: Lower leg eroded with ulceration and impetiginization.

lat. dex, st. post sclerosatio v. Parva lat. dex 8 months ago. The patient is DM type 2 on insulin, HBB, CVI ischemicum regio parietalis lat. syn., Dihemiparesis pp lat.dex., HTA. She was regularly followed by the endocrinologist. Pseudomonas aeruginosa was isolated from a wound swab. On clinical examination, the patient was hardly mobile, with pronounced swelling of the right lower leg, erythema. On the heel, there was ulceration with a granulated bottom and scars. Above the malleolus, the entire lower third of the lower leg was eroded.

A Doppler echo was performed, which registered the presence of atherosclerotic plaques in all arteries and deep venous insufficiency in the right leg. Laboratory analyses showed leukocytosis, elevated D dimers, and high CRP.

The patient had pronounced lymphedema and compression therapy was completely contraindicated, we hospitalized her and she was placed on a diuretic, a broad-spectrum antibiotic and regular hydrocolloid dressings, and advised to elevate her leg. Unfortunately, the patient suffered another ischemic stroke, after which she fell into a coma for several days and died.

CASE REPORT 2

A 68-year-old male patient presented to the doctor due to the recurrence of a venous ulcer on the right foot (Fig. 2). He reported that he had had recurrent venous ulcers and lymphedema for the past three years. The first swelling and appearance of the ulcer occurred

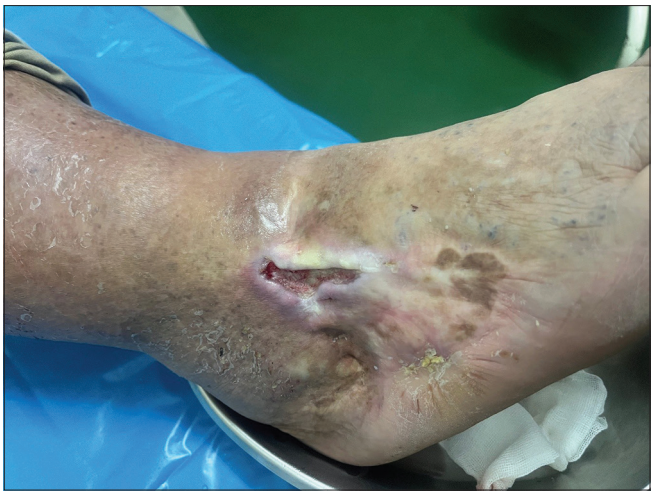


Figure 2: Stasis dermatitis, ulceration, and scars from past ulcerations.

shortly after sclerosing the right great saphenous vein three years previously. On clinical examination, varicose veins were visible on the left limb. On the right limb, the middle and lower third of the lower leg was stasis dermatitis and, in places, the beginning of lipodermatosclerosis and ulceration.

Echo Doppler of the lower extremities:

Arterial flow - regular Doppler signal at all levels of AFS, AP, ATP, ATA

Venous flow - left - deep veins flowing and compressible, insufficient especially v. popliteal

Left - SFU - not visible, VSM was not visible to the upper third of the thigh, in the middle third of the thigh, a perforator was present. VSM was dilated and branched, especially around the knee joint with numerous varicosities. No acute thrombotic process was observed. Organized lymph nodes were observed on the left inguinal.

Right arterial triphasic regular Doppler signal at the level of AFS, AP. Due to pronounced swelling of the lower leg and foot, ATP and ATA signals cannot be obtained. Deep veins on the right were compressible and insufficient. Superficial veins - VSM was observed in the upper third of the upper leg, then it was occluded, but numerous insufficient varicose veins observed in the upper leg much more pronounced at the level of the knee joint and lower leg where several perforators and several varicosities that were non-compressible as part of the PTS. VSM appeared in the lower third of the lower leg, dilated and insufficient. At the level of the entire lower leg and ankle, pronounced edema

was observed. VSP dilated with numerous varicosities and a perforator in the middle third. In the right inguinal region, an enormously enlarged lymph node was present. The patient was placed on hydrocolloid dressings and compression therapy was given: oral anticoagulant therapy rivaroxaban 20 mg continuously for three months and troxerutin 300 mg twice daily for three months. After complete healing of the ulcer, which healed with the formation of a scar, the patient was referred to regular lymphatic drainage.

A recommendation was given after a three-month break to take troxerutin 300 mg twice a day for another three months, vitamin C 1000 mg continuously, in combination with lymphatic massage and compression therapy. In this way, the patient has not opened a new venous ulcer for a year, although the problem with lymphedema remains.

CASE REPORT 3

A 74-year-old man presented for examination due to a persistent ulcer for more than four months after sclerosing of the saphenous vein magna and saphenous vein parva on the right leg (Fig. 3). After the treatment, the condition worsened with swelling, hyperpigmentation and the beginning of ulceration. The patient was admitted to the same institution for several months for glutathione infusion therapy and surgery without success. At the time of the examination, extensive ulcerations were present on the right lower leg with dimensions of 20 x 7 cm, larger and the size of a child's hand, two smaller posteriorly. There were ulcers with zones of necrosis, fibrin plaques, in places deeper and with an unpleasant odor. An echo Doppler examination of the right extremity was performed. Arterial flow - AFS with two-phase extended Doppler signal with discrete atherosclerotic changes at the beginning and with larger occlusive changes in the middle third of the thigh. ATP so monophazen continuous Doppler signal. Venous flow - superficial veins were completely non-compressible. Dlaboki vein insufficiency. There was an expressed island along the entire length. The entire right foot was swollen and fluctuant, during which an incision was made, from which purulent contents were drained. During the drainage, a rubber drain came out from the inside that was placed three months ago during a bandage and never removed. An X-ray of the foot was done.

Figure 3: Ulcer with dimensions of 20 x 7 cm, zones of necrosis, fibrin plaques.

The X-ray of the right foot showed osteopenia of the depicted skeleton of the right foot with advanced arthritic changes with asymmetric and reduced distal and proximal interphalangeal joint spaces. The was subchondral sclerosis of the joint surfaces and luxation of the proximal phalanx of the V finger.

The patient had myasthenia gravis and was on chronic therapy with Decortin 25 mg, tbl. Ciclosporin 50 mg 2x1, tbl. pyridostigmine 60 mg. The patient was admitted for a swab from the wound. *Staphylococcus aureus* was isolated and antibiotic therapy was prescribed according to the antibiogram ceftriaxone. Wound debridement was performed and daily dressings with a spray containing stable ozonides with vitamin E acetate, tea tree leaf oil Melaleuca alternifolia, thioctic acid were started. After a month and a half, we began doing PRF once a week on the patient, and the results were visible after the first week. The patient was not a candidate for wound closure with his own skin due to the dimensions of the wound and the peripheral arterial occlusive disease. The patient also received vitamin C 1000 mg, vitamin E 400 IU, cilostazol 100 mg 2x1, and acetylsalicylic acid 100 mg 1x1. The patient is still using these medications, took trypsin, chimotripsin, and serrapeptase for a month. The patient showed significant improvement, although the healing process was long. After only a month there, there was a significant reduction in the dimensions of the wounds. After two months, the wound was already at skin level. The swelling and pain in the foot resolved in two weeks.

DISCUSSION

Sclerosis of the great saphenous vein and the lesser saphenous vein carries greater risks than benefits.

Given the duration of the effect of sclerosing, and the magnitude of the side effects and complications, according to our experience and practice, the treatment of saphenous veins with sclerosing is risky. In order to perform sclerosing of the saphenous veins, a good assessment is required in order to minimize the risk of complications that the patient then faces for a long period of time and some, unfortunately, for life. According to numerous studies, foam sclerotherapy has a low complication rate, low cost, and acceptable total occlusion rate and reproducibility [5]. However, our practice has shown that complications are quite common, although all of them are present in patients who are probably not followed up to the end or are not well investigated for existing comorbidities. According to research, thrombophilia may also be considered a contraindication for sclerotherapy, after a large group of patients who were studied retrogradely and faced thromboembolism shortly after the performed sclerosing of the saphenous veins; however, most had thrombophilia diagnosed after the incident [6].

Skin necrosis as a complication is described both after the perivascular injection of high-percentage sclerosant and, in rare cases, after correct intravascular injection of the sclerosant at low concentrations [7].

To reduce the risk of skin necrosis, the injection of large volumes at any injection point should be avoided. The sclerosant should be injected at the lowest possible pressure [6].

Extensive necrosis may occur following inadvertent intra-arterial injection; the risk of this complication may be reduced by the use of ultrasound to identify blood vessels. If severe pain occurs during the injection of the sclerosant, the procedure should be stopped immediately. If intra-arterial injection is suspected, anticoagulant therapy should be administered. Prompt administration of systemic corticosteroids may help reduce the inflammatory reaction.

Sometimes, there are malformations of the blood vessels, and then complications are almost inevitable, which is why a thorough duplex ultrasound is recommended, and sometimes additional examinations are needed to clarify the anatomical and hemodynamic condition.

Other transient general or local reactions that may occur after sclerotherapy include chest tightness, vasovagal syncope, metallic taste in the mouth or nausea, thrombus, hematoma, ecchymosis or pain at the injection site, and the appearance of local swelling, induration and erythema [7].

Deep venous insufficiency undiagnosed before sclerotherapy will lead to the appearance of lymphedema, a condition that is permanent, and the patient will face swelling in the extremities for life, all of which will lead to secondary wound opening, impetigo of cracked skin, immobility, and reduced quality of life.

It is important that, before sclerotherapy, a diagnosis shall be obtained, including medical history and clinical and duplex ultrasound examinations, and that a good assessment be made.

CONCLUSION

Before sclerotherapy, patients should be informed about possible complications and side effects. Each patient should be aware of alternative treatment methods with their advantages and disadvantages, as well as details about the entire sclerotherapy procedure. Each possible complication should be explained, as well as common side effects.

Regarding the expected outcome of sclerotherapy, patients should be informed that more frequent controls or re-treatment may be necessary.

Foam sclerotherapy is more effective than liquid sclerotherapy for subcutaneous varicose veins.

Ultrasound-guided foam sclerotherapy may avoid the need for intra-arterial injection.

Sclerotherapy of the great and small saphenous veins carries an unacceptably high risk of complications compared to modern alternatives. Based on clinical evidence and the cases presented, truncal vein sclerotherapy should not be considered a primary treatment option. Endovenous ablation techniques remain the gold standard, with proven efficacy, durability, and safety.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The

patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Vasileva M, Brishkoska Boshkovski V, Drakalska Sersemova E. Comorbidities as prognostic factors in the healing of venous ulcers. Our Dermatol Online. 2023;14:177-79.
2. Patel SK, Surowiec SM. Venous Insufficiency. [Updated 2024 Feb 14]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK430975
3. Beneat A, Oropallo A. Sclerotherapy. [Updated 2024 Jan 11]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK599526
4. Wong M, Parsi K, Myers K, De Maeseneer M, Caprini J, Cavezzi A, et al. Sclerotherapy of lower limb veins: Indications, contraindications and treatment strategies to prevent complications: A consensus document of the International Union of Phlebology-2023. Phlebology. 2023;38:205-58.
5. Karacalilar M, Kutas HB. Approach to tortuous vena saphena magna with insufficiency: Foam scleroterapy. Vascular. 2023;31:1003-10.
6. Rabe E, Breu FX, Flessenkämper I, Gerlach H, Guggenbichler S, Kahle B, et al. Sclerotherapy in the treatment of varicose veins: S2k guideline of the Deutsche Gesellschaft für Phlebologie (DGP) in cooperation with the following societies: DDG, DGA, DGG, BVP. Hautarzt. 2021;72(Suppl 2):23-36.
7. Goldman MP, Sadick NS, Weiss RA. Cutaneous necrosis, telangiectatic matting and hyperpigmentation following Sclerotherapy. Dermatol Surg. 1995;21:19-29.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Report

A Merkel cell carcinoma case with high but preventable morbidity

Glenn Kolansky[1], Zach Kolansky[2]

[1]Medical Director of Advanced Dermatology Surgery and Laser Center & Assistant Clinical Professor of Hackensack Meridian School of Medicine, Nutley, NJ, USA. & Double Board Certified in Dermatology and Mohs Micrographic Surgery, [2]Graduate of Tulane University School of Medicine, New Orleans, LA, USA

Corresponding author: Zach Kolansky, MD, E-mail: zacharykolansky@gmail.com

ABSTRACT

Merkel cell carcinoma (MCC) is a rare and aggressive neuroendocrine tumor that grows rapidly and metastasizes early. Typically, MCC presents as an erythroscaly papule but may vary in morphology, possessing etiologies consistent with UV exposure and Merkel cell polyomavirus (MCPyV) infection. Biopsy-driven histopathologic and immunohistochemical analysis are necessary for diagnosis. We present an original case of MCC overlying a recent arthroplasty and morbidities from combined factors. A 75-year-old patient with a recent total arthroplasty presented with an erythematous and hyperkeratotic nodule which was asymptomatic and previously diagnosed by an orthopedist as a Baker's cyst. Biopsy results showed MCC, yielding treatment modality including but not limited to surgery and radiation therapy. The patient experienced a chronically-open wound and radiation contracture, ultimately leading to above-the-knee amputation. This case emphasizes the importance of early evaluation of suspicious lesions by a dermatologist and the necessity of clinical considerations when MCC arises near prosthetic joints.

Key words: Merkel cell carcinoma, Total arthroplasty, Radiation contracture, Baker's cyst, Surgical site infection

INTRODUCTION

Merkel cell carcinoma (MCC) is a rare and aggressive neuroendocrine tumor that commonly originates in the stratum basale. Depending on the source, MCC has a 5-year survival rate that ranges between 36 to 65% in men and 47-84% in women [1]. MCC lesions often present as an erythroscaly papule [2]. However, MCC can present as a papule or nodule of any morphology. Etiology of MCC is related to cumulative DNA damage from UV radiation. In some MCC's, the Merkel cell polyomavirus (MCPyV) has integrated into damaged Merkel cells, and the virus has an oncogenic role in MCC proliferation. The MCPyV is a ubiquitous virus that does not typically integrate into the host genome and instead replicates in the nucleus [2]. Risk factors for MCC include age (70+), significant UV exposure, and immunocompromised status [1].

Histopathology and immunohistochemistry are central to the diagnosis of MCC. Specimens of MCC contain a blue basaloid dermal proliferation arranged in nodules, or sheets between reticular dermal collagen bundles. At low-power, MCC does not have peripheral palisading cells, a useful characteristic to distinguish it from Basal cell carcinoma (BCC). At high power, MCC consists of uniform round blue cells with minimal cytoplasm, numerous mitoses, and a stippled ("salt and pepper") pattern in the nuclei characteristic of other neuroendocrine tumors. Merkel cell carcinoma is almost always positive for CK20 and lymphovascular invasion (LVI), whereas BCC is CK20 negative without LVI. Finally, MCC is often positive for synaptophysin, chromogranin, and neurofilament protein [3].

We present a case of complications with a recent total arthroplasty from coincident treatment of Merkel cell carcinoma via radiation therapy.

How to cite this article: Kolansky G, Kolansky Z. A Merkel cell carcinoma case with high but preventable morbidity. Our Dermatol Online. 2026;17(2):234-237.

Submission: 10.06.2025; **Acceptance:** 13.11.2025

DOI: 10.7241/ourd.20262.18

CASE REPORT

On September 30, 2020, a 75-year-old woman visited the dermatologist with a complaint that a growth on her left knee was enlarging for 3 months. The patient was previously reassured by her orthopedist that the growth of concern appeared to be a Baker's cyst, and there was no need to be concerned. Clinical examination showed it to be an erythematous and hyperkeratotic nodule. On July 17, 2020, 2 months prior to this visit, she underwent a total left knee arthroplasty. At the time, medical history was significant for DM (HgA1c-7), hypertension, ½ a pack of cigarettes a day for 53+ years, hyperlipidemia, and degenerative joint disease. Since the surgery, she could not ambulate because she could not straighten her leg and had 10/10 pain despite use of 5mg oxycodone 2-3 times per day. She underwent a biopsy for the lesion, measuring 6.5 cm x 5.5 cm (Fig. 1).

The biopsy results (Figs. 2a - 2c) showed Merkel cell carcinoma (MCC) that was positive for pan-cytokeratin, CK20, synaptophysin, and chromogranin. Immunohistology analysis showed characteristic blue sheets of cells with aggregates that intercalate between reticular collagen bundles (Fig. 2a). Histopathology showed that the neoplastic cells broadly extended to the base of the specimen with a Breslow thickness greater than 3mm (Fig. 2b). Further characterization of the cells as having small and rounded size and shape, granular and scant cytoplasm, and high rate of mitosis further corroborated the diagnosis of MCC (Fig. 2c) [4].

On October 13, 2020, a PET scan showed an avid soft tissue mass overlying the left lateral tibia and fibula without bony destruction, consistent with primary malignancy. Additionally, the scan demonstrated no avid nodal metastasis, and a right parotid mass. The parotid mass was worked up and was later diagnosed as Warthin's parotid tumor, unrelated to MCC. The patient consulted with oncology and radiation oncology in the following weeks and experienced multiple falls in part from her leg disability with nuances of surgery, radiation, and immunotherapy discussed. Ultimately, surgical options were ruled out by the patients due to significant morbidity risk and radiation therapy was opted for; the patient never followed through with discussion of immunotherapy. The physician team noted among themselves that radiation would be

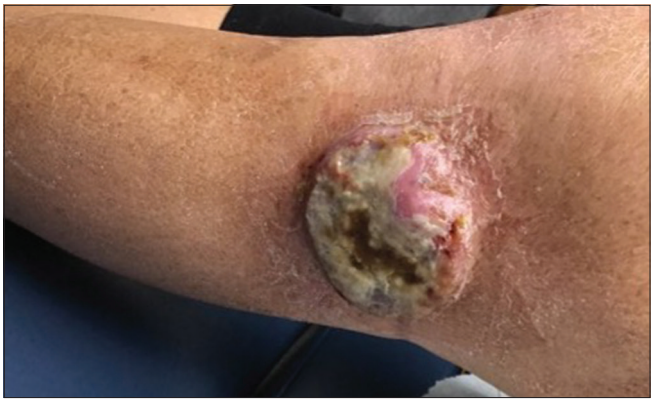


Figure 1: Pre-biopsy photo-documentation of the erythroscaly ulcerated nodular papule lesion of concern, measured 6.5 cm x 5.5cm. Taken on September 30th 2020.

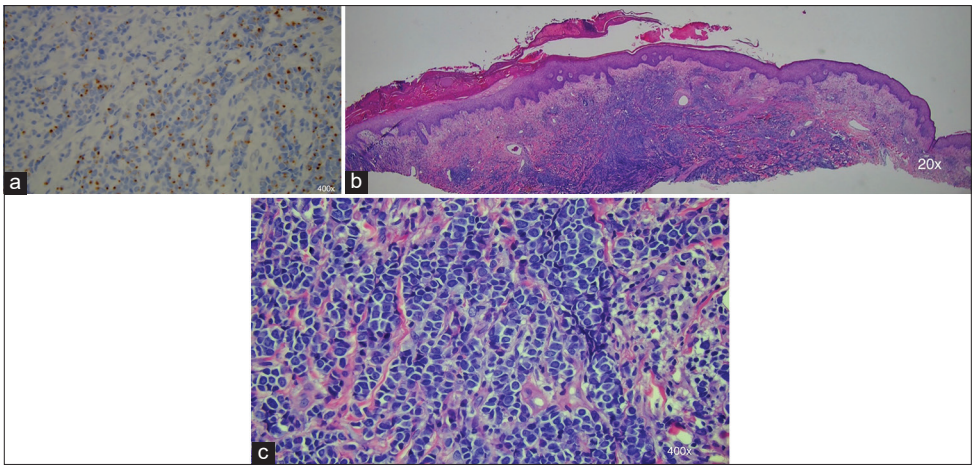


Figure 2: (a) Immunohistochemical analysis via CK-20 Staining at 400x magnification of a sample of the lesion shows characterization of neoplastic growth and Breslow thickness greater than 3.0mm. (b) Histopathological analysis of sample of the lesion via H&E staining at 20x magnification shows absence of peripheral palisading formations. (c) Histopathological analysis of sample of the lesion via H&E staining at 400x magnification shows aggregations of neoplastic cells with large round nuclei and granular cytoplasm. Mitosis and necrotic areas are present.

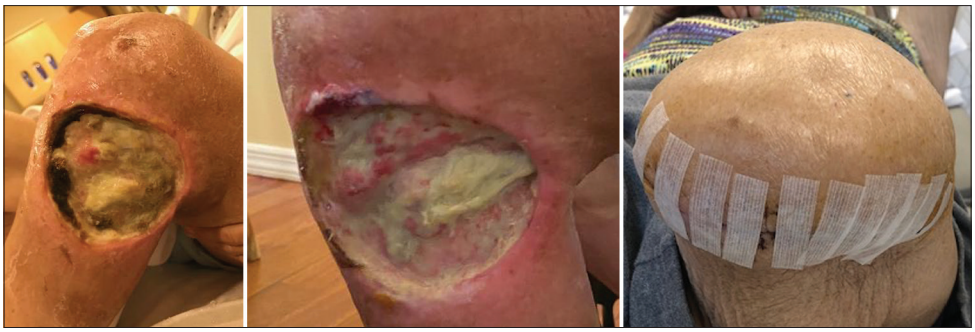


Figure 3: Photo-documentation of state of merkel cell carcinoma radiation site at different dates. Left: March 2021, Pre-amputation. Middle: April 2021, Pre-amputation. Right: June 2021, Post-amputation.

associated with long term morbidity to the knee joint. It is unclear whether the details of long-term joint morbidity from radiation were adequately understood by the patient. The radiation oncology note explains the risks of radiation included fatigue, skin breakdown, poor wound healing, joint stiffness, lymphedema, and scarring.

Radiation was administered to the left knee and groin between November 2020 and January 2021, with a dose of 2139 mGy-cm by the end of treatment (Sentinel lymph node biopsy of groin was negative). Post radiation, she developed a painful, chronically open wound, had major difficulty with pain control, and had 13 documented falls. Failed pain management included tramadol, medical marijuana, cyclobenzaprine, and oxycodone. By May 2021, she developed a 90-degree flexion contracture of the left knee along with the continued open wound. The wound became infected, requiring intense antibiotic therapy, and was debrided once. She was not a candidate for flap coverage due to significant vascular disease, smoking, diabetes. The patient was assessed for options to straighten the knee; it was explained that soft tissue releases would be insufficient to straighten the knee, and an external fixator would be necessary. Discussion conveyed that this is a very painful process with the possibility of cancer recurrence complicating treatment. Moreover, for her to keep the leg, the chronic wound would have to heal. With the goals of pain relief and improved mobilization, she agreed with an above the knee amputation which occurred in late May 2021 (Fig. 3).

DISCUSSION

MCC has multiple mechanisms to escape destruction by the immune system such as inducing PD-1 expression, down regulation of both MHC class 1 and MICA and MICB, down regulation tumor vascular E-selectin, and suppression of the antitumor cGas-STING pathway. MICA and MICB are proteins that stimulate natural killer cells, and E-selectin is necessary for cytotoxic T-cells to invade the tumor [1].

Therapy options include biologics, radiation, chemotherapy, and surgery.

The forefront MCC management includes PD-1/PD-L1 checkpoint inhibitors, CTLA-4 related therapy, adoptive T-cell therapy, neoadjuvant immunotherapy, and adjuvant immunotherapy. In Jurgen's 2024 comprehensive MCC overview, he describes the response rate of metastatic MCC to PD-1/PD-L1 inhibitors [1]. Specifically in his Table 2, he describes the objective response rate varied from 33% to 64% in 5 clinical trials, with a median patient age ranging from 66-74. Three of the five trials had been completed before 2020, so there was documented evidence in the therapeutic value of targeting this pathway at the time of the patient's diagnosis. Radiation therapy has associated risks regardless of the presence of an orthopedic prosthesis. Given the discussion of complications from radiation, we believe it would have been prudent to have pushed for immunotherapy prior to attempting radiation.

Due to diminished quality of soft tissue surrounding the joint due to radiation therapy, there is an increased risk for a periprosthetic joint infection in locations of treatment that are coincident with the radiation site [5]. It should be further evaluated the time necessary to minimize infection risk due to new arthroplasty in the treatment area of any dermatological procedure. These complications are especially important in patients with extensive medical history, such as smoking history and diabetes as shown in this case.

CONCLUSIONS

While there is currently no clinical consensus, skin procedures should be avoided for at least 3 to 7 months after any colocalized arthroplasty [5,6]. In clinical practice, it is often recommended not to do any surgery in the vicinity of an upcoming knee arthroplasty. Risk of infection is always a concern – so much so that there are even guidelines that recommend not to shave 2 days prior to a knee replacement [5]. Even though the standard procedure maintains that no intraarticular injections are given prior to arthroplasty, this is extrapolated to avoid skin surgery prior to knee replacement [6,7].

When there is a potentially cancerous growth, it is our recommendation that it should be evaluated by a dermatologist. If the growth is uncertain or questionable, it should be biopsied prior to surgery, even if it means delaying the surgical procedure.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Becker JC, Stang A, Schrama D, Ugurel S. Merkel Cell Carcinoma: Integrating Epidemiology, Immunology, and Therapeutic Updates. Am J Clin Dermatol. 2024;25:541-57.
2. Coggshall K, Tello TL, North JP, Yu SS. Merkel cell carcinoma: An update and review: Pathogenesis, diagnosis, and staging. J Am Acad Dermatol. 2018;78:433-42.
3. Durst CR, Rezzadeh KT, Than JP, Rajaee SS, Spitzer AI. Intra-Articular corticosteroid injections into a preexisting total knee arthroplasty are associated with increased risk of periprosthetic joint infection and revision. Arthroplast Today. 2023;24:101237.
4. Gardner JM. Survival Guide to Dermatopathology. EA. Montgomery, Ed., Innovative Pathology Press, 2020. 47-52. Isbn:978-1-933477-49-7.
5. Zuidhof R-JWJ, Lowik CAM, Ploegmakers JJW, Dijkstra SPD, Wouthuyzen-Baker M, Jutte PC. Periprosthetic joint infection in orthopaedic surgical oncology. Ann Joint. 2019;4:26.
6. Yang X, Li L, Ren X, Niew L. Do preoperative intra-articular injections of corticosteroids or hyaluronic acid increase the risk of infection after total knee arthroplasty? A meta-analysis. Bon Joint Res. 2022:11:171-9.
7. Kim YM, Joo YB, Song JH. Preoperative intra-articular steroid injections within 3 months increase the risk of periprosthetic joint infection in total knee arthroplasty: a systematic review and meta-analysis. J Orthop Surg Res. 2023;18:148.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Report

Systemic lupus mimicking Stevens-Johnson syndrome: About one case

Zineb Bennouna, Zakia Douhi, Meryem Soughi, Sara Elloudi, Hanane Baybay, Fatimazahra Mernissi

Dermatology Department, Hassan II University hospital, Fez, Morocco

Corresponding author: Zineb Bennouna, MD, E-mail: zineb.bennouna@usmba.ac.ma

ABSTRACT

The Stevens–Johnson Syndrome (SJS) is a severe cutaneous adverse reaction with high morbidity and mortality, often drug-induced. In rare cases, other factors such as infections and vaccinations have been implicated. This report describes the case of a young adolescent who presented with cutaneous involvement characterized by target-like lesions and erosions covered with hemorrhagic crusts, predominantly acral in distribution. There was erosive involvement of the oral and nasal mucosa, as well as hemorrhagic cheilitis, progressing over one month before the consultation. No medication was found. The evolution was marked by the appearance of a lupus mask on the face. Immunological assessment revealed positive homogeneous antinuclear antibodies and anti-native DNA antibodies associated with hypocomplementemia and bicytopenia, leading to the diagnosis of systemic lupus. This case highlights the importance of considering SLE in the differential diagnosis of severe dermatopathies, especially in atypical clinical contexts.

Key words: Stevens–johnson Syndrome, Systemic Lupus, Chronic Progression, Photodistribution

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease affecting multiple organs and is characterized by the aberrant production of autoantibodies. Among its numerous clinical manifestations, dermatological involvement is common and varied. However, atypical presentations, such as skin lesions mimicking Stevens–Johnson syndrome (SJS), are rarely reported. SJS is a severe mucocutaneous distress syndrome, often triggered by medications or infections, and is associated with a high mortality rate. The clinical resemblance between SLE and SJS may complicate the diagnosis, leading to therapeutic challenges. This article examines a case of systemic lupus mimicking SJS, highlighting the underlying mechanisms, diagnostic approach, and therapeutic implications.

CASE REPORT

Herein, we report the case of an eighteen-year-old patient with no prior medical history, who presented with painful acral skin lesions and erosive involvement of the oral and nasal mucosae, evolving for one month. Upon admission, clinical examination revealed a cachectic patient in poor general condition, febrile at 40°C. Cutaneous findings included target lesions on the extremities, erosive pulpitis, diffuse paronychia, a hemorrhagic crusted plaque on the nose, and bilateral auricular impetiginization. Mucosal examination showed hemorrhagic cheilitis and erosions of the oral and nasal mucosae (Figs. 1a – 1c).

Initial diagnoses of major erythema multiforme and Stevens–Johnson syndrome were considered; however, no history of drug intake was identified. The patient reported no systemic symptoms such as arthralgia, photosensitivity, sicca syndrome, or Raynaud's phenomenon. Three days post-admission, the patient developed an erythematous, butterfly-shaped plaque with hemorrhagic crusts on the face (Fig. 2). A skin biopsy was taken but was nonspecific. Immunological workup revealed positive antinuclear antibodies (ANA) with a homogeneous pattern and positive anti-DNA

How to cite this article: Bennouna Z, Douhi Z, Soughi M, Elloudi S, Baybay H, Mernissi F. Systemic lupus mimicking Stevens-Johnson syndrome: About one case. Our Dermatol Online. 2026;17(2):238-240.

Submission: 18.11.2024; **Acceptance:** 18.01.2025

DOI: 10.7241/ourd.20262.19

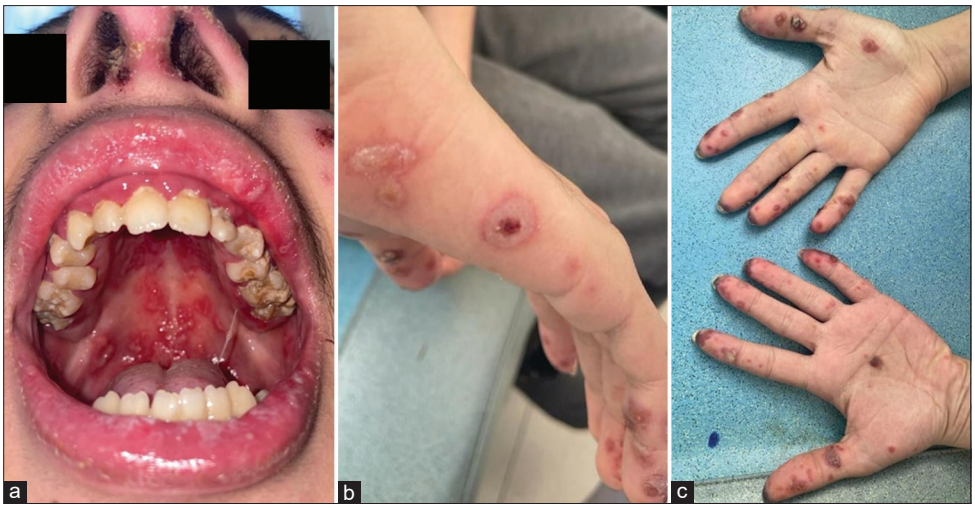


Figure 1: (a-c) Clinical images showing oral and nasal erosions, target lesions, and diffuse erosive pulpitis.

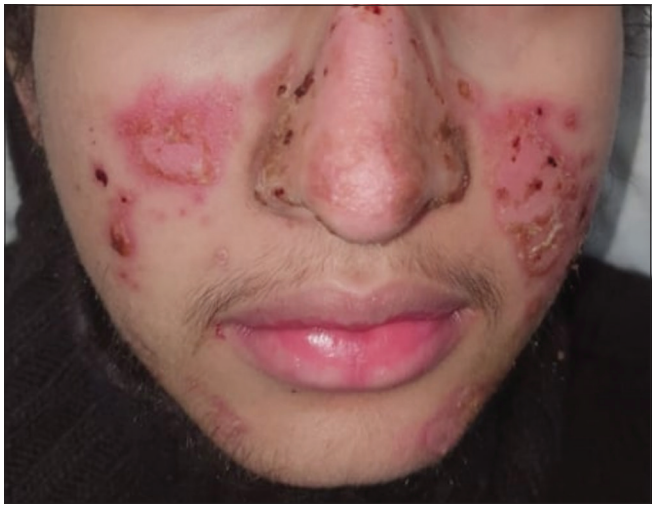


Figure 2: Erythematous butterfly-shaped plaque topped with hemorrhagic crusts.

antibodies. Systemic evaluation showed bicytopenia, altered liver function tests, and hypocomplementemia. The diagnosis of lupus erythematosus mimicking Stevens–Johnson syndrome was retained. The patient was prescribed synthetic antimalarials and oral corticosteroids, leading to significant improvement. After eighteen months of follow-up, the patient remained in good health.

DISCUSSION

Stevens–Johnson syndrome (SJS) is a rare yet serious condition characterized by severe cutaneous and mucosal involvement. While historically associated with drug reactions, it may also occur in response to infections or idiopathically. Systemic lupus erythematosus (SLE), on the other hand, is a multisystem autoimmune disease with varied clinical manifestations, including dermatological ones. SLE may mimic SJS due to the presence of severe skin lesions and mucosal involvement. This clinical similarity results from generalized inflammation and epidermal apoptosis, which are also characteristic of SJS. However, there are fundamental differences in the pathogenesis [1,2]:

- In SLE, skin lesions are mediated by immune complexes, antibody deposits, and complement activation, leading to exaggerated keratinocyte apoptosis.
- In SJS, lesions are primarily related to a type-IV hypersensitivity reaction, triggered by drugs or infections.

There are diagnostic criteria that help to distinguish the two entities. SLE is clinically characterized by an insidious onset and a prolonged evolution, significantly longer than drug-induced SJS, which progresses more rapidly, often with signs of epidermal detachment within hours to days [1]. While our patient presented with buccal ulcers and hemorrhagic cheilitis, there was no genital or perineal involvement. This absence has also been noted in previous studies in this form of SLE [2,3]. Moreover, the initially affected and most severely impacted areas in our patient were the photodistributed regions, suggesting UV radiation as a triggering or aggravating factor [4].

In the literature, there are cases of SJS where no drug was clearly responsible for the symptoms [5,6]. In such idiopathic cases, ANA screening is recommended

to exclude SLE, as it could represent the initial manifestation, as seen in our patient.

The presence of target lesions and positive ANA could suggest Rowell syndrome. However, the homogeneous ANA pattern, negative anti-SSA and anti-SSB antibodies, and normal rheumatoid factor failed to meet the necessary criteria for this diagnosis [7].

The therapeutic management of these two conditions differs significantly, highlighting the necessity of an accurate diagnosis. In SJS, the immediate discontinuation of the suspected drug is crucial, followed by symptomatic management, including hydration, the prevention of secondary infections, and the treatment of ulcers. In severe cases, immunomodulators such as intravenous immunoglobulins (IVIg) or TNF-α inhibitors may be considered [1]. Conversely, SLE often requires systemic corticosteroids to control acute flares, combined with synthetic antimalarials such as hydroxychloroquine for long-term management. In severe forms, immunosuppressants such as mycophenolate mofetil or azathioprine are indicated [4,8].

Thus, in cases where SLE mimics SJS, a delayed diagnosis may lead to rapid disease progression with multiorgan involvement. A multidisciplinary approach involving dermatologists, internists, and immunologists is often required.

CONCLUSION

Systemic lupus erythematosus may exceptionally mimic Stevens–Johnson syndrome, complicating diagnosis and management. Early recognition and appropriate treatment are crucial to improving prognosis. This case highlights the importance of considering SLE in the differential diagnosis of severe dermatopathies, especially in atypical clinical contexts.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Ingen-Housz-Oro, S. Nécrolyse épidermique (syndromes de Stevens-Johnson et de Lyell): manifestations cliniques et prise en charge. Bull Acad Natl Méd. 2023;207:917-23.
2. Ting W, Stone MS, Racila D, Scofield RH, Sontheimer RD. Toxic epidermal necrolysis-like acute cutaneous lupus erythematosus and the spectrum of the acute syndrome of apoptotic pan-epidermolysis (ASAP): A case report, concept review and proposal for new classification of lupus erythematosus vesiculobullous skin lesions. Lupus. 2004;13:941-50.
3. Paradela S, Martínez-Gómez W, Fernández-Jorge B, Castiñeiras I, Yebra-Pimentel T, Llinares P, Fonseca Capdevila E. Toxic epidermal necrolysis-like acute cutaneous lupus erythematosus. Lupus. 2007;16:741-5.
4. Lee HY, Tey HL, Pang SM, Thirumoorthy T. Systemic lupus erythematosus presenting as Stevens-Johnson syndrome and toxic epidermal necrolysis: A report of three cases. Lupus. 2011;20:647-52.
5. Guillaume JC, Roujeau JC, Revuz J, Penso D, Touraine R. The culprit drugs in 87 cases of toxic epidermal necrolysis (Lyell's syndrome). Arch Dermatol. 1987;123:1166-70.
6. Sassolas B, Haddad C, Mockenhaupt M, Dunant A, Liss Y, Bork K, et al. ALDEN, an algorithm for assessment of drug causality in Stevens-Johnson Syndrome and toxic epidermal necrolysis: Comparison with case-control analysis. Clin Pharmacol Ther. 2010;88:60-8.
7. Jaramillo C, Andrea P, Ovalle G, Andrea J, Molina C, David, et al. Rowell syndrome: Case report and review of the literature. Rev Colomb Reumatol (Eng Ed). 2023;4:332-6.
8. Bellakhal S, Ben Kaab B, Teyeb Z, Souissi A, Derbel F, Douggui MH. [Systemic lupus erythematosus presenting as Stevens-Johnson syndrome]. Pathol Biol (Paris). 2015;63:222-3.

Source of Support: This article has no funding source,
Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Report

Disabling pansclerotic morphea of childhood with skin ulceration and tendon retraction: A case report

Meriem El Haddad, Ouiame El Jouari, Salim Gallouj

Dermatology and Venerology Department, Mohammed VI University Hospital, Tangier, Morocco

Corresponding author: Meriem El Haddad, MD, E-mail: hb.meriem94@gmail.com

ABSTRACT

Disabling pansclerotic morphea of childhood (DPMC) is a rare, severe form of generalized morphea with significant diagnostic and therapeutic challenges. It is characterized by deep tissue involvement, extending to the muscles, tendons, and bones, leading to complications such as joint stiffness, deformities, and ulcerations. Herein, we report the case of a six-year-old girl with a five-month history of widespread sclerosis involving the lower limbs, acral areas, severe ulcerations, and tendon retractions. Histopathological evaluation confirmed DPMC, revealing a thickened dermis with eosinophilic collagen bundles and atrophic adnexal structures, without dermal homogenization. Treatment included monthly corticosteroid pulses, methotrexate, and physiotherapy. While partial improvement was observed, with reduced joint stiffness and improved mobility, significant sclerosis and depigmentation persisted, underscoring the disease's refractory nature. This case highlights the variability of DPMC, the importance of early recognition, and the need for aggressive multidisciplinary care. Further research is essential to optimize outcomes and establish standardized protocols for this condition.

Key words: Disabling pansclerotic morphea, Childhood, Ulceration, Tendon retraction, Multidisciplinary treatment

INTRODUCTION

Disabling pansclerotic morphea of childhood (DPMC) is a rare and severe form of localized scleroderma, primarily affecting children under the age of 14, with a prevalence of less than 1 per 10,000,000 [1]. It is characterized by systemic inflammation, extensive skin and soft tissues, and occasionally extends to the underlying muscles and bones.

The Juvenile Scleroderma Working Group of the Pediatric Rheumatology European Society identified linear morphea as the most common type (65%), followed by plaque-type morphea (26%), generalized morphea (7%), and deep morphea (2%) [2]. DPMC is recognized as a rare subtype of deep morphea [3].

DPMC may lead to complications such as muscle and skeletal atrophies, joint contractures, ankylosis, trophic skin ulcers, nerve compressions, and an increased risk of skin cancer [4-6]. Although its etiology remains unclear, mechanisms such as endothelial cell injury, immune activation, and fibroblast hyperactivity, resulting in excessive collagen synthesis, are proposed. Genetic predisposition and environmental factors may also play a role.

Herein, we report the case of a six-year-old child with DPMC presenting with atrophic sclerosis over five months and significant cutaneous and articular complications. This case highlights the rarity and rapid progression of DPMC, contributing to the limited literature on this condition.

CASE REPORT

A six-year-old girl with no prior medical history presented with irregular, infiltrated plaques, beginning

How to cite this article: El Haddad M, El Jouari O, Gallouj S. Disabling pansclerotic morphea of childhood with skin ulceration and tendon retraction: A case report. Our Dermatol Online. 2026;17(2):241-244.

Submission: 04.02.2025; **Acceptance:** 06.06.2025

DOI: 10.7241/ourd.20262.20

at the root of the left thigh and extending over the entire lower limb for five months. Additionally, the patient reported widespread joint pain, morning stiffness, fatigue, and weight loss. Developmental milestones were normal, and she was up-to-date with vaccinations. The family history was unremarkable.

On examination, skin sclerosis extended from the pubic region to the dorsum of the left foot, with sclerotic lesions noted over the toes of the right foot and along the lateral aspect of the right thigh. Furthermore, the patient exhibited tight, shiny skin on the proximal interphalangeal (PIP) and metatarsophalangeal (MTP) joints. Mild desquamation, hypopigmentation, and hyperpigmentation patches were noted (Figs. 1a – 1d). Two ulcerations were identified: one on the external right thigh (4 cm) and another on the internal left thigh, (7 cm), with irregular edges, fibrinous bases, and inflammatory halos on a scleroatrophic base (Fig. 2).

Functional assessment revealed a reduced range of motion in the left knee and ankle, attributed to hamstring tendon retraction and cutaneous sclerosis, causing walking difficulty.

Table 1 summarizes the initial laboratory investigations. X-rays of the left knee and ankle showed preserved joint spaces without structural abnormalities. Histopathological examination (Figs. 3a and 3b) revealed a thickened mid-dermis with eosinophilic collagen bundles surrounding the adnexal structures. There was no evidence of dermal tissue homogenization. The adnexal structures were atrophic, consistent with the features of DPMC. Lung function tests, capillaroscopy, as well as cardiac and ophthalmologic evaluations, revealed no abnormalities.

Treatment included monthly pulses of glucocorticosteroids (methylprednisolone 15 mg/kg/day for three days) for six months and methotrexate 15 mg/m^2/week and physiotherapy. Ulcer care involved local wound management and debridement with antimicrobial dressings.

After three months, the patient showed partial improvement. Joint stiffness improved, and mobility increased, yet skin sclerosis and depigmentation persisted. This partial response highlighted the complexity of managing DPMC and the need for ongoing care.

DISCUSSION

This case highlighted the rarity and complexity of DPMC, a severe and progressive form of generalized morphea classified as an autoimmune disease. It deeply affects the subcutaneous tissue, muscles, tendons, and bones, leading to complications such as joint stiffness, deformity, ulcerations, and calcifications. Lesions typically involve the extensor surfaces of the limbs and trunk [7], sparing acral areas; however, in our case, acral areas were notably affected. Additionally, ulcerations and tendon retractions, rarely reported in pediatric cases, were prominent features.

Diagnosing DPMC is particularly challenging in its early stages, as it mimics other localized scleroderma subtypes such as plaque-type morphea or linear morphea [7]. Definitive diagnosis is often delayed until complications such as ankylosis or muscle atrophy become evident. Extensive clinical and histopathological evaluations were crucial in differentiating this case from other connective tissue diseases.

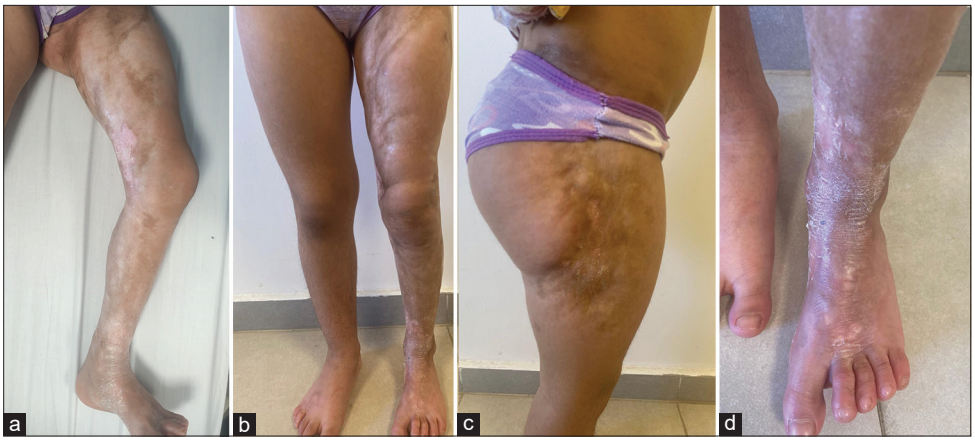


Figure 1: (a-d) Clinical photos showing extensive sclerotic lesions with areas of hypo- and hyperpigmentation.

Table 1: Baseline laboratory findings in a patient diagnosed with disabling pansclerotic morphea of childhood

Category	Parameter	Results	Units	Reference Range
1. Inflammatory markers	C-reactive protein (CRP)	10	mg/L	<8
	Erythrocyte sedimentation rate (ESR)	12	mm/h	0–6
2. Immunity and autoimmunity	Antinuclear antibodies (ANA)	1:160 (speckled pattern)	-	<1:80
	Specific autoantibodies (SSA, SSB, etc.)	Negative	-	-
3. Eosinophilia	Eosinophils	11.8	%	<5
4. Coagulation and inflammation	Platelets (thrombocytosis)	593	$\times 10^9$/L	120–340
5. Muscle enzymes	Creatine kinase (CK)	141	IU/L	40–240
	Lactate dehydrogenase (LDH)	517	U/L	313–618

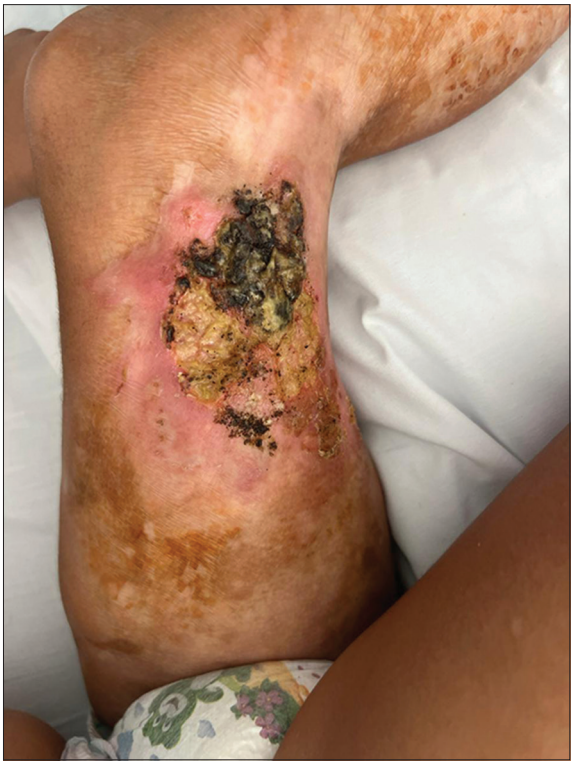


Figure 2: Clinical photo showing ulceration with irregular edges and erythematous inflammatory halos resting on a scleroatrophic base.

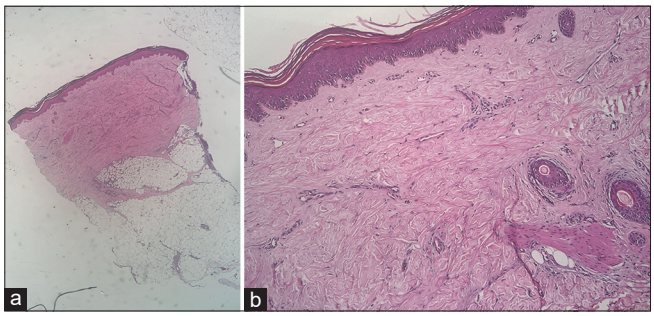


Figure 3: (a) Histopathologic examination at low magnification (x10) showing a compact and thickened dermal layer with dense collagen bundles extending into the deeper layers of the skin. (b) Histopathologic examination showing thickened collagen bundles in the mid-dermis, without collagen homogenization, and a sparse perivascular lymphohistiocytic infiltrate (H&E stain; x100).

To date, only 39 cases of DPMC have been reported in the literature, with even fewer involving ulcerations or squamous cell carcinoma (6.7% incidence), as noted by Wollina et al. [8].

Managing DPMC remains challenging due to limited evidence. In a cross-sectional study [9], methotrexate (MTX) has been identified as the second most commonly initiated treatment after systemic corticosteroids. MTX disrupts inflammatory cascades mediated by cytokines such as IL-1, IL-2, IL-4, IL-6, and TNF, which are implicated in the disease. It has been used in combination with corticosteroids (both intravenous and oral) with varying degrees of success.

Emerging therapies, including mycophenolate mofetil, biologics, tyrosine kinase inhibitors (TKIs), and Janus kinase inhibitors (JAKIs), have shown promise in conditions such as systemic sclerosis, supporting their trial use in DPMC. Alternative treatments, including sildenafil for ulcers, colchicine, cyclosporine, and tacrolimus, have shown benefits in individual cases.

Most children with DPMC require high-dose immunosuppressive therapy, with MTX and corticosteroids forming the cornerstone of care. The growing number of case reports and advances in biologics offer hope for improved treatments. Collaborative research and clinical studies are essential to optimize outcomes and standardize protocols.

CONCLUSION

DPMC is a rare and severe form of generalized morphea with significant diagnostic and therapeutic challenges. This case highlighted the disease's variability, including unique acral involvement, ulcerations, and tendon retractions. Early diagnosis, multidisciplinary care, and ongoing research are essential for improving outcomes.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Fett N, Werth VP. Update on morphea: Part I. Epidemiology, clinical presentation, and pathogenesis. J Am Acad Dermatol. 2011;64:217-28.
2. Zulina F, Athreya BH, Laxer R, et al. Juvenile localized scleroderma: clinical and epidemiological features in 750 children. An international study. Rheumatology. 2006;45:614-20.
3. Chung L, Lin J, Furst DE, Fiorentino D. Systemic and localized scleroderma. Clin Dermatol. 2006;24:374-92.
4. Forsea AM, Cretu AN, Ionescu R, Giurcaneanu C. Disabling pansclerotic morphea of childhood: Unusual case and management challenges. J Med Life; 2008;1:348-54.
5. Kura M, Jindal S. Disabling pansclerotic morphea of childhood with extracutaneous manifestations. Indian J Dermatol. 2013;58:159.
6. Doede T, Wollina U, Hindermann W, Schier F, Bondartschuk M. Pansclerotic morphea in childhood: A case report. Pediatr Surg Int. 2003;19:406-8.
7. Diaz-Perez JL, Connolly SM, Winkelmann RK. Disabling pansclerotic morphea of children. Arch Dermatol. 1980;116:169-73.
8. Wollina U, Buslau M, Heinig B, et al. Disabling pansclerotic morphea of childhood poses a high risk of chronic ulceration of the skin and squamous cell carcinoma. Int J Low Extrem Wounds. 2007;6:291-8.
9. Kim A, Marinkovich N, Vasquez R, Jacobe HT. Clinical features of patients with morphea and the pansclerotic subtype: A cross-sectional study from the morphea in adults and children cohort. J Rheumatol. 2004;41:106-12.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Narrative Review

Regenerative biologics for aging skin: A narrative review comparing exosomes, mesenchymal stem cell secretome, and platelet-rich plasma

Theressia Handayani[1,2], Ketut Kwartantaya Winaya[1,3], I Gusti Nyoman Darmaputra[1,3]

[1]Faculty of Medicine, Universitas Udayana, Denpasar, Indonesia, [2]Bethesda Hospital, Yogyakarta, Indonesia, [3]Prof. I.G.N.G. Ngoerah General Hospital, Denpasar, Indonesia

Corresponding author: Theressia Handayani, E-mail: theressdr@gmail.com

ABSTRACT

Skin aging reflects a gradual decline in extracellular matrix integrity, cellular senescence, and chronic inflammation. Regenerative biologics such as exosomes, mesenchymal stem cell (MSC) secretome, and platelet-rich plasma (PRP) offer biologically driven approaches aimed at restoring dermal homeostasis. Exosomes and MSC secretome demonstrate antioxidant, immunomodulatory, and collagen-stimulatory effects in multiple preclinical models. Early human data reports improvements in texture, fine lines, erythema, and procedural recovery, especially when used with microneedling or laser resurfacing. PRP remains the most clinically validated therapy, supported by randomized trials demonstrating enhanced periorbital rejuvenation, dermal remodeling, and global photoaging improvement. While exosomes and secretome show strong biological promise, current limitations include product heterogeneity, regulatory constraints, and limited long-term data. PRP offers the most favorable balance of evidence, safety, and accessibility. Further comparative studies are needed to clarify optimal clinical use.

Key words: Skin aging, Photoaging, Exosomes, Extracellular vesicles, Mesenchymal stem cell secretome, Platelet-rich plasma, Regenerative dermatology

INTRODUCTION

Skin aging is driven by intrinsic factors—such as mitochondrial decline, collagen fragmentation, and telomere attrition—and extrinsic insults, such as UV radiation and pollution. These changes trigger fibroblast senescence, extracellular matrix (ECM) degradation, and chronic inflammation [1-3]. Clinically, this manifests as wrinkles, laxity, dyschromia, and impaired barrier recovery.

A shift toward regenerative dermatology has introduced biologic therapies targeting cellular communication and tissue restoration rather than solely corrective interventions. Exosomes, MSC secretome, and PRP represent three leading biologic platforms with converging but distinct mechanisms.

Exosomes are nanoscale vesicles rich in proteins, lipids, and microRNAs that regulate cellular signaling [2,4,5]. MSC secretome contains soluble trophic factors and extracellular vesicles with strong reparative and immunomodulatory effects [6-9]. PRP offers an autologous source of platelet-derived growth factors integral to wound healing [10-12]. This review compares these modalities with emphasis on mechanisms, evidence, and translational considerations.

MATERIALS AND METHODS

This work is a narrative review based on a structured search of PubMed, Scopus, Web of Science, and Google Scholar for articles published between 2015 and 2025. The keywords included *exosomes*, *mesenchymal stem cell*

How to cite this article: Handayani T, Winaya KKW, Darmaputra IGN. Regenerative biologics for aging skin: A narrative review comparing exosomes, mesenchymal stem cell secretome, and platelet-rich plasma. Our Dermatol Online. 2026;17(2):245-249.

Submission: 30.11.2025; **Acceptance:** 04.02.2025

DOI: 10.7241/ourd.20262.21

secretome, *conditioned medium*, *platelet-rich plasma*, *skin aging*, and *rejuvenation*. Only peer-reviewed, English-language studies relevant to biologic mechanisms, clinical applications, and safety were included. Non-scientific sources and unreviewed commercial materials were excluded. Due to the heterogeneity of study designs, findings were synthesized qualitatively without meta-analysis.

Ethics Statement

This article is a narrative review and does not involve human subjects, patient data, or clinical interventions. Therefore, ethical approval and informed consent were not required.

RESULTS

Exosomes in Skin Rejuvenation

Biological rationale

Exosomes derived from MSCs, dermal fibroblasts, and platelets deliver bioactive molecules capable of stimulating fibroblast proliferation, enhancing collagen I/III synthesis, reducing MMP expression, and attenuating oxidative stress [1-5]. Their microRNA cargo (such as miR-21, miR-29, and miR-146a) modulates pathways central to skin aging [2,3].

Clinical evidence

Clinical use remains emergent but promising. Prospective studies and split-face trials show improvements in fine wrinkles, skin smoothness, erythema, and accelerated healing when exosomes are applied after microneedling, Q-switched toning, or fractional laser resurfacing [4-7]. Most evidence highlights exosomes' synergistic role alongside controlled dermal injury.

Limitations

Challenges include heterogeneity in sources (MSC, fibroblast, platelet), differences in isolation techniques, batch variability, and undefined potency metrics [5-7]. Regulatory classification as biologics limits routine injectable use. Long-term safety data is not yet available.

Mesenchymal Stem Cell Secretome/Conditioned Medium

Biological rationale

MSC secretome includes cytokines (e.g., VEGF, HGF), matrix-modulating proteins, antioxidants, and extracellular vesicles produced during MSC culture [6-9]. Secretome promotes fibroblast proliferation, collagen/elastin synthesis, angiogenesis, and reduced oxidative stress. Umbilical cord MSC secretome demonstrates notable wound-healing and anti-inflammatory potential [7].

Clinical Evidence

Early clinical studies show improvements in hydration, elasticity, radiance, and superficial wrinkles following topical, microneedling-assisted, or intradermal secretome application [6,8]. Secretome enhances re-epithelialization and reduces erythema post-laser resurfacing [7,9].

Limitations

As an allogeneic biologic, secretome varies by donor characteristics and culture conditions. Regulatory classification as an advanced biologic increases oversight [13]. Evidence remains promising but less mature than that for PRP.

Platelet-Rich Plasma (PRP)

Biological rationale

PRP contains concentrated platelets that release PDGF, TGF-β, VEGF, EGF, and IGF-1 upon activation—promoting fibroblast proliferation, angiogenesis, ECM synthesis, and tissue repair [10-12]. Its autologous nature ensures minimal immunogenicity.

Clinical evidence

PRP is the most extensively studied regenerative biologic. Randomized trials and histologic studies show improvements in fine wrinkles, periorbital pigmentation, dermal thickness, and overall photoaging [11,12,14,15]. Combination treatments—microneedling + PRP or fractional laser + PRP—yield consistently superior outcomes [11,14].

Limitations

Variability exists between preparation systems, platelet concentrations, leukocyte content, and injection techniques. Nevertheless, PRP remains a clinically reliable and safe modality.

Comparative Synthesis

Strength of evidence

PRP exhibits the strongest clinical evidence and longest track record [10-12,14-16]. Exosomes show robust mechanistic rationale with emerging clinical

support [1-5]. Secretome presents excellent preclinical data with early but limited human studies [6-9].

Safety

PRP has a well-established safety profile [10-12,14]. Exosomes and secretome appear safe but require better-defined manufacturing standards and long-term data [5,13].

Regulatory considerations

PRP is widely permitted as a minimally manipulated autologous biologic. Exosomes and secretome face stricter biologic regulations [5,13].

To support comparative interpretation, two summary tables were constructed. Table 1 outlines the biological characteristics, mechanisms, evidence strength, and limitations of PRP, MSC secretome, and exosomes. The table clearly demonstrates that PRP currently has the strongest clinical and histologic evidence, whereas secretome offers broad reparative signaling, and exosomes provide advanced molecular precision but remain the least standardized. Table 2 synthesizes practical recommendations for clinical integration across various procedural contexts, including microneedling, fractional resurfacing, and recovery optimization.

DISCUSSION

Regenerative biologics represent a pivotal evolution in dermatologic anti-aging interventions, emphasizing cellular communication and intrinsic repair mechanisms rather than externally imposed structural correction. Although exosomes, MSC secretome, and PRP share overlapping pathways—collagen induction, fibroblast activation, angiogenesis, and inflammatory modulation—their biological precision, clinical maturity, and translational readiness differ significantly.

Exosomes: Mechanistic Sophistication, Early-Stage Translation

Exosomes embody the most advanced biological design, functioning as naturally targeted delivery systems for microRNAs, proteins, and lipids. Their ability to modulate oxidative stress pathways, downregulate MMPs, suppress NF-κB, and enhance type I collagen synthesis positions them as elegant molecular regulators of skin aging [1-4]. However, clinical application remains at an early phase, with current evidence largely limited to small studies and adjunctive procedural use [4,5]. Major translational barriers include variability in manufacturing, the absence of standardized potency assays, and stringent regulatory oversight. Without GMP-certified exosomal platforms and long-term safety data, their role remains primarily experimental.

MSC Secretome: Broad, Physiologic Repair Signaling

Secretome contains a diverse array of soluble factors and extracellular vesicles that more closely mimic the native paracrine activity of MSCs. This breadth of signaling—spanning angiogenesis, antioxidant pathways, ECM remodeling, and immunomodulation—offers a physiologic and multi-modal regenerative approach [6-9]. Early clinical results show meaningful

Table 1: Comparative summary of PRP, MSC secretome, and exosomes in skin rejuvenation

Parameter	PRP	MSC secretome	Exosomes
Biological source	Autologous platelets	Allogeneic MSC-derived factors	MSC/fibroblast/platelet EVs
Key mechanisms	Growth factor–mediated fibroblast activation	Broad reparative signaling & immunomodulation	miRNA-driven targeted molecular regulation
Evidence strength	Strong (multiple RCTs)	Moderate (early clinical data)	Emerging (small early trials)
Clinical benefits	Texture, fine lines, periorbital aging	Hydration, elasticity, recovery	Redness reduction, post-procedure healing
Limitations	Preparation variability	Donor variability	Lack of standardization & regulatory constraints
Safety	Excellent	Generally safe; allogeneic	Appears safe; long-term data limited
Best use	Microneedling combination	Adjunct to resurfacing	Post-laser healing+procedural booster

Table 2: Clinical integration and practical recommendations

Clinical scenario	Most suitable biologic	Rationale
Collagen induction/dermal remodeling	PRP	Reliable growth-factor–driven fibroblast activation; strongest evidence
Post-laser recovery (ablative/non-ablative)	Secretome or exosomes	Superior anti-inflammatory, healing acceleration, barrier recovery
Patients needing rapid downtime reduction	Exosomes	Best redness reduction and post-procedure comfort
Patients preferring autologous therapy	PRP	Excellent safety and regulatory simplicity
Enhancing hydration and elasticity	Secretome	Broad trophic signaling and ECM support
Maximal synergistic rejuvenation	PRP+Secretome/Exosomes	Complementary mechanisms (stimulation+modulation)

improvements in elasticity, hydration, and recovery post-resurfacing. Secretome has advantages over exosomes in manufacturing scalability and biological robustness, yet it remains limited by donor variability, regulatory complexities, and non-standardized assays.

PRP: Clinically Established and Translationally Mature

PRP remains the benchmark among regenerative biologics. Its mechanisms—rooted in platelet-derived growth factor release—are highly aligned with phases of wound healing and dermal repair [10-12]. Randomized controlled trials consistently demonstrate improvements in fine lines, periorbital changes, and overall texture [10-12,14,15], while histologic evidence confirms enhanced collagen density and dermal architecture. Unlike exosomes and secretome, PRP benefits from strong regulatory acceptance, minimal immunogenicity, affordability, and widespread accessibility. Its limitations, centered on preparation variability, are comparatively minor and manageable.

Current evidence establishes a clear functional hierarchy among regenerative biologics for aging skin. PRP remains the most reliable and clinically validated modality, supported by controlled trials, histologic data, and predictable safety. MSC secretome represents an intermediate option with broad reparative signaling and promising early clinical results, yet still limited by variability and the need for stronger standardization. Exosomes, while mechanistically the most advanced, remain the least standardized and least supported by high-quality clinical data, placing them at the earliest stage of translational readiness.

In practice, the most consistent outcomes arise from procedure-integrated applications rather than standalone biologics. PRP combined with microneedling reliably enhances collagen induction. Secretome or exosomes applied after laser resurfacing accelerate recovery and improve dermal remodeling. Increasingly, clinicians use multi-modal, tailored combinations to achieve synergistic benefits aligned with individual patient needs and aging profiles.

The comparative tables reinforce the therapeutic hierarchy observed in current literature. As illustrated in **Table 1**, PRP remains the most evidence-supported biologic, driven by reproducible growth-factor stimulation and strong safety. Secretome occupies an intermediate position with wide-ranging reparative signals, while exosomes—although mechanistically sophisticated—are constrained by standardization and regulatory gaps. **Table 2** highlights that optimal outcomes arise from procedure-integrated approaches. PRP reliably enhances collagen induction with microneedling, whereas secretome and exosomes are superior as post-resurfacing modulators, accelerating healing and improving overall rejuvenation. These tables emphasize that biologics should be selected based on therapeutic precision, patient needs, and procedural context.

Remaining Gaps and Future Directions

Despite promising developments, regenerative biologics cannot advance without resolving several critical gaps. The field urgently requires standardized potency assays, direct comparative trials, and robust long-term safety data, particularly for allogeneic products. Clear dose–response parameters and protocol standardization are also needed to minimize clinical variability. Future studies must incorporate biomarker-based, imaging, and histologic endpoints to objectively quantify outcomes. Only through these scientific refinements can regenerative therapies transition from emerging innovations to reliable, evidence-driven dermatologic interventions. Advancements in GMP manufacturing, machine-learning–assisted phenotyping, and personalized biologic formulations may further refine regenerative dermatology.

CONCLUSION

Exosomes, MSC secretome, and PRP each offer unique regenerative advantages for aging skin. At present, PRP remains the most validated option for routine clinical use. Exosomes and secretome demonstrate compelling biological potential and are likely to become central tools in dermatologic rejuvenation as manufacturing quality and evidence advance.

ACKNOWLEDGMENTS

The author thanks the Faculty of Medicine, Universitas Udayana, for academic support in anti-aging and regenerative medicine.

REFERENCES

1. Domaszewska-Szostek A, Wojas-Pelc A, Cichy J, Skopiński P. Extracellular vesicles in skin rejuvenation: Mechanisms and applications. Stem Cell Res Ther. 2025;16:112.

2. Sreeraj H, Nath AK, Thappa DM. Exosomes in dermatology: Advances and emerging applications. Dermatol Ther (Heidelb). 2024;14:345-60.
3. Liang C, Xiong Y, Huang Y, et al. Exosome-mediated regulation of oxidative stress and extracellular matrix remodeling in skin aging. Stem Cell Res Ther. 2025;16:210.
4. Lee YS, Kim JH, Park JH, Lee SY. Clinical effects of mesenchymal stem cell-derived exosomes in skin rejuvenation: A split-face study. Dermatol Ther (Heidelb). 2025;14:789-804.
5. Rodriguez C, Sainz-Arnal P, Nistal JF. Exosomes and cell culture–conditioned media in aesthetic medicine: Current status and regulatory perspectives. Cosmetics. 2024;11:154.
6. Putri WE, Sunarto W, Kurniawan A. Mesenchymal stem cell-conditioned medium in skin aging and rejuvenation: A systematic review. Dermatol Rep. 2021;13:9236.
7. Li X, Zhang Y, Wang Y, et al. Umbilical cord mesenchymal stem cell secretome accelerates skin healing and rejuvenation: Mechanistic and translational insights. Cell Prolif. 2024;57:e13421.
8. Zare S, Hosseini SM, Kalantar R, et al. Clinical evaluation of adipose-derived mesenchymal stem cell secretome for facial skin rejuvenation. Clin Cosmet Investig Dermatol. 2025;18:55-67.
9. Wong RSY, Cheong SK. Mesenchymal stem cell-based therapeutics in dermatology: Mechanisms, applications, and challenges. Curr Dermatol Rep. 2025;14:22-34.
10. White C, Stewart DA, Mraz S. Platelet-rich plasma in dermatologic surgery and facial rejuvenation: A comprehensive review. Dermatol Surg. 2021;47:1052-62.
11. Banihashemi M, Zare S, Yazdanpanah M. Platelet-rich plasma for facial rejuvenation: Results of a clinical trial and literature review. Acta Biomed. 2021;92:e2021129.
12. Xiao H, Zhang X, Liang J, et al. Platelet-rich plasma in photoaged skin: clinical and histologic improvements in facial rejuvenation. Clin Cosmet Investig Dermatol. 2021;14:521-30.
13. Maguire G. The safety and efficacy of secretome-based therapy in dermatology. J Clin Aesthet Dermatol. 2019;12:38-44.
14. Schoenberg E, Kim A, Paul R. Early clinical experience with platelet-rich plasma for periorbital rejuvenation. J Cosmet Laser Ther. 2020;22:345-52.
15. Koshkinbayeva A, Ualiyeva A, Zhumanova S. Autologous platelet-rich plasma for facial aging: Clinical outcomes and patient satisfaction. Cosmetics. 2024;11:175.
16. Arshdeep, Kumaran MS. Platelet-rich plasma in dermatology: Current evidence and future trends. Cureus. 2024;16:e45512.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Review Article

Topical corticosteroids versus topical calcineurin inhibitors in atopic dermatitis: A comparative review of efficacy and safety

Weronika Durańska[1], Anna Maria Modzelewska[2], Wiktor Modzelewski[1], Michał Szczupak[2], Hubert Chmielewski[1], Julia Komar[1], Maciej Antoni Kasner[1], Martyna Tyszko[1], Patrycja Siebiatyńska[1], Patryk Łaźny[1]

[1]Medical University of Bialystok, Jana Kilińskiego 1, 15-089 Białystok, Poland, [2]University Clinical Hospital in Bialystok ul. M. C. Skłodowskiej 24a, 15-276 Białystok, Poland

Corresponding author: Weronika Durańska, MD, E-mail: weronikaduranska121001@gmail.com

ABSTRACT

Introduction: Atopic dermatitis (AD) is a chronic, relapsing inflammatory dermatosis with complex pathogenesis and remains a major challenge in dermatology. Topical therapy constitutes the foundation of treatment across all disease severities. **Objectives:** To compare the clinical efficacy, mechanisms of action, and safety profiles of topical glucocorticosteroids (TCS) and topical calcineurin inhibitors (TCI). **Methods:** A narrative review of the literature, case reports, and international therapeutic guidelines published between 2021 and 2025 concerning the use of TCS and TCI in the treatment of AD was conducted. **Discussion:** TCS are the first-line agents for acute flares but are limited by the risk of skin atrophy with prolonged use. TCIs provide an effective steroid-sparing alternative without inducing epidermal thinning, making them suitable for sensitive areas and long-term therapy. **Conclusions:** TCS and TCI remain central to topical management of AD. Optimal disease control requires proactive, individualized treatment tailored to patient age and lesion localization.

Key words: Atopic Dermatitis, Topical Glucocorticosteroids, Calcineurin Inhibitors, Tacrolimus, Proactive Therapy

INTRODUCTION

Atopic dermatitis (AD), also referred to as atopic eczema, is a chronic, relapsing inflammatory dermatosis characterized by persistent pruritus, xerosis, and eczematous lesions with a typical distribution. It constitutes a central component of the so-called atopic triad and is often the first manifestation of the "atopic march," preceding the development of other allergic conditions such as allergic rhinitis, food allergy, bronchial asthma or tropical endemic limbo-conjunctivitis [1-3]. Owing to its complex pathogenesis and chronic course, AD represents one of the major challenges in contemporary dermatology and allergology.

Epidemiological data indicate that AD is highly prevalent worldwide, with a marked increasing trend in industrialized countries. The disease affects approximately 10–20% of the pediatric population, with the majority of cases manifesting in early childhood [2,4]. Although historically considered a childhood disease, current evidence demonstrates a substantial prevalence among adults, with reported rates ranging from 2% to as high as 10% [1,4,5]. The clinical significance of AD extends far beyond cutaneous manifestations. According to global burden of disease analyses, AD ranks 15th among all noncommunicable diseases and first among skin diseases in terms of disability-adjusted life years (DALYs) [2]. Severe pruritus, the predominant symptom, leads to sleep disturbances,

How to cite this article: Durańska W, Modzelewska AM, Modzelewski W, Szczupak M, Chmielewski H, Komar J, Kasner MA, Tyszko M, Siebiatyńska P, Łaźny P. Topical corticosteroids versus topical calcineurin inhibitors in atopic dermatitis: A comparative review of efficacy and safety. Our Dermatol Online. 2026;17(2):250-257.

Submission: 30.12.2025; **Acceptance:** 10.02.2026

DOI: 10.7241/ourd.20262.22

anxiety, depression, and reduced productivity at work and school, resulting in considerable socioeconomic costs and a profound deterioration in patients' quality of life [1,5].

The etiopathogenesis of AD is multifactorial and involves complex interactions between genetic predisposition, environmental factors, epidermal barrier dysfunction, and immune dysregulation [2,6,7]. A pivotal role in disease pathophysiology is played by defects of the epidermal barrier, frequently resulting from loss-of-function mutations in the filaggrin (FLG) gene. Filaggrin is essential for proper keratinocyte differentiation and for the formation of the natural moisturizing factor (NMF). Its deficiency leads to increased transepidermal water loss (TEWL), elevated skin pH, and enhanced permeability to allergens and pathogens, consistent with the "outside-to-inside" hypothesis [1,2,6].

Concomitantly with barrier impairment, patients with AD exhibit exaggerated activation of the Th2-dependent immune pathway. Key cytokines, including interleukin-4 (IL-4) and interleukin-13 (IL-13), play a central role in driving inflammation [5,6]. These cytokines not only stimulate IgE production but also downregulate the expression of barrier proteins, such as filaggrin and loricrin, as well as antimicrobial peptides, thereby creating a self-perpetuating pathogenic loop [4,5]. In addition, cytokines such as IL-31 are directly responsible for the induction of pruritus through activation of sensory neurons [5].

An important component of the clinical phenotype is cutaneous microbiome dysbiosis. Patients with AD demonstrate a marked reduction in microbial diversity accompanied by dominance of *Staphylococcus aureus*. Colonization with *S. aureus* occurs in 30% to 100% of patients and correlates with disease severity [1]. Through the production of toxins, superantigens, and biofilm formation, *S. aureus* amplifies type 2 inflammatory responses and further disrupts the epidermal barrier, exacerbating the clinical course of the dermatosis [2,4]. Given this complex pathophysiology, topical corticosteroids and topical calcineurin inhibitors remain the cornerstone of anti-inflammatory therapy, and their comparison in terms of efficacy and safety is essential for the optimization of treatment strategies.

Significance of Topical Therapy

Topical therapy constitutes the cornerstone of management in atopic dermatitis (AD), playing a pivotal role in both the treatment of disease flares and long-term maintenance, irrespective of disease severity [8-11]. The primary therapeutic objectives are restoration of the epidermal barrier and reduction of inflammation and pruritus, achieved through regular use of emollients and topical anti-inflammatory medication [10-12]. Despite the emergence of novel topical treatments, such as Janus kinase (JAK) inhibitors and phosphodiesterase-4 (PDE-4) inhibitors, topical corticosteroids (TCS) and topical calcineurin inhibitors (TCI) have remained the two principal pillars of topical pharmacotherapy for decades [11,13-15]. Current clinical guidelines, including European, Canadian, and Indian consensus statements, consistently identify these drug classes as the standard of care [11,16-18].

Topical Corticosteroids (TCS)

Topical corticosteroids are first-line agents for the treatment of active cutaneous inflammation in AD [8,10,12].

- Mechanism of action: TCS exert potent anti-inflammatory, antiproliferative, and vasoconstrictive effects. At the molecular level, they bind intracellular glucocorticoid receptors, leading to suppression of proinflammatory cytokine gene transcription and inhibition of phospholipase A2, thereby reducing the synthesis of inflammatory mediators [10,12,15].
- Potency classification: TCS are categorized according to their potency. Different classification systems are used globally, for example a seven-class system in the United States [9,10]. Selection of an appropriate potency depends on patient age, anatomical site (e.g., face versus hands), and disease severity [8,10,11].
- Limitations: Despite high efficacy, prolonged use of TCS is associated with adverse effects, most notably cutaneous atrophy, telangiectasia, striae, and steroid-induced acne [8,16,19]. Additional concerns include tachyphylaxis (decrease of treatment effectiveness in time) and rebound flares following abrupt discontinuation. These risks contribute to so-called steroid phobia, which represents a significant barrier to effective disease control [8,10,19].

Topical Calcineurin Inhibitors (TCI)

Topical calcineurin inhibitors, tacrolimus and pimecrolimus, represent an important steroid-sparing alternative to TCS [15,19].

- Mechanism of action: TCIs bind intracellular immunophilins (tacrolimus to FKBP-12),

forming a complex that inhibits calcineurin phosphatase activity [19]. This blockade prevents dephosphorylation of nuclear factor of activated T cells (NFAT), thereby inhibiting its nuclear translocation and suppressing transcription of key proinflammatory cytokines, including IL-2, IL-4, IL-5, IFN-γ, and TNF-α [15,19].

- Agents: Tacrolimus is available as 0.03% and 0.1% ointment, while pimecrolimus is formulated as a 1% cream. Tacrolimus demonstrates efficacy comparable to mid-potency topical corticosteroids, whereas pimecrolimus is recommended primarily for milder course of disease [10,19].
- Long-term safety: A major advantage of TCIs is the absence of skin atrophy, rendering them suitable for long-term use on sensitive areas such as the face and intertriginous regions [10,19]. Long-term safety has been confirmed in multiple studies. They are recommended not only for adults but also for children. Long time using of tacrolimus is proven to be safe for kids [14,20]. Expert groups, including the Indian STAND AD group, also emphasize the favorable safety profile of TCIs and recommend their use, including off-label application in children under 2 years of age, proving the lack of systemic adverse effects when used appropriately [11].

Other Topical Treatment Modalities

PDE4 Inhibitors

Advances in the understanding of atopic dermatitis (AD) pathophysiology have led to the development of novel anti-inflammatory molecules targeting specific molecular pathways. An important class comprises phosphodiesterase-4 (PDE4) inhibitors, which target an enzyme responsible for the degradation of cyclic adenosine monophosphate (cAMP). Increased PDE4 activity in inflammatory cells results in excessive production of proinflammatory cytokines. [9,13]. Crisaborole 2% ointment is a nonsteroidal PDE4 inhibitor approved for the treatment of mild to moderate AD in patients from 3 months of age [6,9]. Clinical trials have demonstrated its efficacy in reducing skin lesions and pruritus, although the therapeutic effect is modest compared with drug vehicle [1,9]. The most common adverse event is application-site pain (burning or stinging), reported in approximately 4–32% of patients [12,13]. Other PDE4 inhibitors under investigation include difamilast, approved in Japan, and roflumilast, which has demonstrated anti-inflammatory efficacy in phase III trials [15,21].

JAK Inhibitors

Another major therapeutic advance is represented by topical Janus kinase (JAK) inhibitors. The JAK–STAT pathway mediates signaling of multiple cytokines central to AD pathogenesis, including IL-4, IL-13, and IL-31, the latter being directly implicated in pruritus [13,21]. Ruxolitinib 1.5% cream, a JAK1/JAK2 inhibitor, demonstrated rapid pruritus reduction (within 12 hours) and significant improvement in skin lesions in adolescents and adults in phase III TRuE-AD trials [13,15,22]. Delgocitinib, a pan-JAK inhibitor approved in Japan for use in both children and adults, has also shown efficacy in chronic hand eczema [13,21]. Despite their high efficacy, JAK inhibitors carry warnings regarding potential systemic adverse effects; however, the risk appears low with topical administration [15,17,22].

Emerging Directions: AhR Modulators and the Skin Microbiome

Tapinarof 1% cream is a novel aryl hydrocarbon receptor (AhR) modulator that inhibits STAT6 activation and enhances the expression of epidermal barrier proteins, including filaggrin and loricrin [13,21]. Clinical studies have demonstrated its efficacy in moderate to severe AD in both adults and children. The most frequent side effect event was folliculitis being [13]. In parallel, therapies aimed at modulating the skin microbiome are under investigation, with the goal of reducing *Staphylococcus aureus* colonization and restoring bacterial homeostasis, using strains such as *Roseomonas mucosa* or *Staphylococcus hominis* A9. To date, however, clinical trial results have been inconsistent [9,13].

Systemic and Adjunctive Therapies

In severe AD refractory to topical treatment, initiation of systemic therapy is required. Conventional immunosuppressive agents, including cyclosporine, methotrexate, and azathioprine, are effective but associated with significant risks during long-term use, such as nephrotoxicity and hypertension [10,12,23]. A major therapeutic breakthrough has been the introduction of biologic agents, notably dupilumab, a monoclonal antibody targeting the IL-4/IL-13 receptor pathway, which demonstrates a favorable efficacy and safety profile, and tralokinumab, an anti–IL-13 antibody [9,12,24]. Oral JAK inhibitors, such as upadacitinib and abrocitinib, represent additional options, offering rapid pruritus relief but requiring laboratory monitoring. Adjunctive treatments

include phototherapy (narrowband UVB), wet-wrap therapy during disease flares, and sodium hypochlorite baths to reduce bacterial superinfection, although their superiority over water alone remains a matter of debate [9,12,22,23].

MATERIAL AND METHODS

The literature review was conducted using the PubMed, MEDLINE, Scopus, Web of Science, and Google Scholar databases. The search included publications from 2021 to 2025, with particular emphasis on recent clinical studies, systematic reviews, expert guidelines, and consensus documents concerning the use of TCS and TCI in the treatment of atopic dermatitis.

DISCUSSION

Atopic dermatitis (AD) is one of the most prevalent chronic inflammatory skin diseases, affecting a substantial proportion of both the pediatric and adult populations worldwide [6,10,18]. The complex pathophysiology of AD, involving epidermal barrier dysfunction, immune dysregulation, and environmental and genetic factors, necessitates multidirectional therapeutic strategies [4,23,25]. Despite the emergence of novel targeted therapies, including Janus kinase (JAK) inhibitors and biologic agents such as dupilumab, topical therapy remains the cornerstone of management in mild to moderate disease [12,26,27]. This discussion compares the two principal classes of topical anti-inflammatory agents-topical corticosteroids (TCS) and topical calcineurin inhibitors (TCI)-with respect to their mechanisms of action, clinical efficacy, safety profiles, and practical implications in contemporary dermatology.

Comparison of the Mechanisms of Action of Topical Corticosteroids and Calcineurin Inhibitors

The fundamental distinction between TCS and TCI lies in their different molecular targets within the inflammatory cascade, which determines both their therapeutic effects and adverse event profiles. Topical corticosteroids, long regarded as the gold standard for the treatment of AD flares, exert broad anti-inflammatory, antiproliferative, and immunosuppressive effects [27,28]. Their mechanism of action involves inhibition of antigen processing and suppression of pro-inflammatory cytokine release through effects on multiple immune cell types, including T lymphocytes, monocytes, macrophages, and dendritic cells [21,27]. In addition, TCS influence lipid and protein synthesis in the skin, which with prolonged use may result in structural alterations such as epidermal and dermal atrophy [29,30]. Studies by Aschoff et al., using optical coherence tomography (OCT), demonstrated that potent corticosteroids such as betamethasone valerate induce significant epidermal thinning, whereas this effect is not observed with weaker agents used intermittently [29].

Calcineurin inhibitors, including tacrolimus and pimecrolimus, act in a more selective manner. These agents bind to cytosolic immunophilins (FKBP12), forming a complex that inhibits calcineurin phosphatase activity [19]. Inhibition of calcineurin prevents dephosphorylation of the nuclear factor of activated T cells (NFAT) and its translocation to the nucleus, thereby suppressing transcription of genes encoding key pro-inflammatory cytokines, including IL-2, IL-3, IL-4, IL-5, and TNF-α [19,31,32]. Unlike TCS, TCIs do not interfere with collagen synthesis, eliminating the risk of skin atrophy and making them suitable for use in sensitive areas such as the face, neck, and intertriginous regions [27,30,33]. Additional evidence suggests that TCIs may modulate mediator release from mast cells and basophils and reduce FcεRI expression on Langerhans cells, further attenuating allergic inflammation [19,32].

Epidermal barrier impairment and pruritus signaling play a central role in AD pathogenesis. Both TCS and TCI may contribute to normalization of filaggrin and loricrin expression, supporting barrier repair; however, TCIs do not adversely affect epidermal lipid synthesis, which is critical for long-term skin integrity [5,30].

Efficacy of Topical Corticosteroids Versus Calcineurin Inhibitors

Clinical evidence indicates that the efficacy of both drug classes depends on potency and patient population. Comparative studies have shown that tacrolimus 0.1% provides efficacy comparable to mid-potency corticosteroids and superior to weak corticosteroids and pimecrolimus 1% [17,19,30]. A systematic review by Peña et al. confirmed that tacrolimus achieved significantly greater reductions in disease severity, measured by the modified Eczema Area and Severity Index (mEASI), compared with weak TCS in most analyses [30].

In a pediatric study by Mohamed et al., tacrolimus 0.03% ointment was compared with hydrocortisone 1% cream. Although both treatments achieved similar reductions in clinical disease severity, tacrolimus resulted in a significantly greater decrease in serum inflammatory markers, including IL-10, IL-17, and IL-23 [32]. These findings suggest a stronger effect of TCIs on subclinical inflammation, which may be relevant for long-term disease control.

Long-term observational data also support the effectiveness of both approaches. A 3-year study by Perälä et al. in children with moderate to severe AD demonstrated comparable reductions in disease activity with TCS and tacrolimus, with no significant differences in body surface area involvement or EASI scores at study completion [20]. However, direct comparisons between pimecrolimus and tacrolimus consistently show faster onset of action and greater efficacy with tacrolimus, positioning it as the preferred option in more severe disease, while pimecrolimus is generally reserved for milder forms [18,19].

Proactive therapy represents an important strategy in long-term management. Intermittent application of TCS or TCI, typically twice weekly to previously affected areas, effectively reduces relapse rates. Tacrolimus is approved for this indication in many regions, although TCS are commonly used in a similar manner [8,34,35]. Handa et al. demonstrated comparable efficacy of fluticasone and tacrolimus in preventing disease flares, supporting the use of either strategy in maintenance therapy [34].

Adverse Effects

Safety considerations are central to therapeutic decision-making, particularly in pediatric patients. Despite their high efficacy, TCS are associated with a risk of adverse effects, especially with prolonged use of high-potency formulations. Common local adverse effects include skin atrophy, telangiectasia, striae, and steroid-induced acne [21,27,32]. Using high-frequency ultrasound and OCT, Aschoff et al. confirmed that treatment with betamethasone valerate (a mid-potency corticosteroid) resulted in measurable epidermal thinning within weeks, whereas pimecrolimus did not induce such changes, underscoring the favorable atrophy-related safety profile of TCIs [29].

Concerns also exist regarding systemic effects of TCS absorption, including suppression of the hypothalamic–pituitary–adrenal axis and potential effects on bone and glucose metabolism. In a randomized study, Gether et al. demonstrated that intensive application of betamethasone ointment over large body surface areas resulted in detectable systemic drug levels and a reduction in markers of bone formation (P1NP), suggesting a potential impact on bone homeostasis, although no short-term effect on insulin sensitivity was observed [36]. Conversely, a systematic review by Harvey et al. suggested that intermittent TCS use for up to five years is unlikely to be associated with significant risks of growth impairment or adrenal suppression in children, which is reassuring for clinicians [37].

For TCIs, the most commonly reported adverse effect is a burning or stinging sensation at the application site, particularly during the initial phase of treatment. These symptoms are usually transient and diminish as the skin heals [16,19,32,38,39]. Importantly, TCIs do not cause skin atrophy, making them the preferred option for lesions on the face, eyelids, and skin folds [27,33]. Although the U.S. Food and Drug Administration issued a black box warning regarding a potential risk of malignancy associated with TCI use, extensive long-term studies and meta-analyses have not demonstrated a causal association between topical TCI use and increased risk of lymphoma or skin cancer in humans [16,19,21]. Both European and American dermatologic societies consider TCIs safe when used appropriately [18].

Long-term Safety

Long-term safety is particularly relevant in AD due to its chronic course. In the 36-month study by Perälä et al., no significant differences in safety outcomes were observed between patients treated with TCS and those treated with tacrolimus, including rates of cutaneous infections and other adverse events [20]. Similarly, the systematic review by Harvey et al. found no increased risk of lymphoma or other malignancies in studies of up to five years' duration in children treated with either TCS or TCIs [37].

Concerns regarding long-term TCS-induced skin thinning can be mitigated by intermittent treatment regimens and avoidance of potent corticosteroids in sensitive areas. Evidence suggests that corticosteroid-induced epidermal atrophy is often reversible upon treatment discontinuation. TCIs, owing to their non-atrophogenic profile, can be safely used on a long-term

basis, as supported by observational studies extending up to 10 years for tacrolimus [28,29].

Nevertheless, vigilance is warranted regarding rare but potential systemic effects. Cohort studies suggest a possible, albeit weak, association between prolonged TCS use and an increased risk of type 2 diabetes or osteoporosis in adults, particularly in those applying potent agents to large body surface areas over extended periods [36,37]. For TCIs, despite the absence of evidence for carcinogenicity in humans, sun protection during treatment is generally recommended [35].

Practical Implications

Based on the available evidence, the choice between TCS and TCI should be individualized, taking into account patient age, lesion location, disease severity, and patient and caregiver preferences.

According to international guidelines, including Canadian and European recommendations, TCS remain first-line therapy for the management of AD flares due to their rapid onset of action and high efficacy. For lesions on the face, neck, genital area, and skin folds, TCIs (pimecrolimus or tacrolimus) are preferred as first-line agents or as alternatives following a short course of low-potency corticosteroids to minimize the risk of atrophy. These considerations are particularly important in pediatric patients, in whom a higher body surface area–to–weight ratio increases the risk of systemic absorption [11,17,18].

A major challenge in clinical practice is "steroid phobia," affecting up to 80% of patients and caregivers, leading to poor adherence and suboptimal disease control [28,30]. Patient education regarding the safety of TCS, explanation of fingertip unit (FTU) dosing for accurate application, and reassurance about the low systemic risk with appropriate use are essential for improving adherence [28,35,40]. TCIs provide a valuable alternative for patients reluctant to use corticosteroids, although higher costs may limit access in some healthcare systems [28,30].

Proactive therapy, consisting of twice-weekly application of an anti-inflammatory agent (TCS or TCI) to previously affected skin, is recommended for patients with frequent relapses, as it prolongs remission and reduces overall medication use [11,18,35]. Treatment algorithms proposed by European and Canadian experts emphasize a flexible approach that combines intensive flare management with long-term maintenance therapy, alongside continuous use of emollients as the foundation of care [11,17,18].

In summary, both TCS and TCI occupy well-established positions in the management of AD. Successful therapy relies on balancing efficacy and safety, tailoring treatment to individual patient characteristics, and providing education to build confidence in the proposed treatment plan. The introduction of newer agents, such as PDE-4 inhibitors and JAK inhibitors, further expands therapeutic options, addressing unmet needs in mild to moderate AD, particularly in cases where conventional therapies are ineffective or poorly tolerated [8,26].

CONCLUSIONS

Analysis of the available literature allows for clear conclusions regarding the role of topical corticosteroids (TCS) and topical calcineurin inhibitors (TCI) in the management of atopic dermatitis (AD). Both drug classes constitute the foundation of topical anti-inflammatory therapy; however, they differ in safety profile, mechanism of action, and preferred sites of application, which determines their use in clinical practice.

Topical corticosteroids remain the first-line treatment for the control of acute disease flares due to their rapid onset of action and high anti-inflammatory and antipruritic efficacy [27]. They are particularly recommended for lesions located on the trunk and extremities, where the skin is thicker. It should be emphasized, however, that prolonged use, especially of high-potency preparations is associated with a risk of clinically significant adverse effects, including skin atrophy, telangiectasia, striae, and, less frequently, systemic suppression of the hypothalamic-pituitary-adrenal axis [10]. Therefore, TCS therapy should be limited to short treatment courses or applied using intermittent regimens [17].

Calcineurin inhibitors (tacrolimus and pimecrolimus) represent a valuable alternative, particularly as steroid-sparing agents. Their key advantage is the absence of atrophogenic potential, which makes them the treatment of choice for lesions located in sensitive areas such as the face, neck, eyelids, and intertriginous regions. Clinical studies confirm that tacrolimus, particularly at a concentration of 0.1%,

demonstrates efficacy comparable to mid-potency topical corticosteroids and superior to pimecrolimus, which is generally recommended for milder forms of the disease [10]. Long-term observational data and meta-analyses have not substantiated earlier concerns regarding an increased risk of malignancy, including lymphoma or skin cancer, associated with TCI use, supporting their safety in chronic therapy [18]. The most commonly reported adverse effect of TCIs is a transient burning sensation at the site of application [10].

Current therapeutic strategies in AD favor a proactive approach, involving long-term intermittent application of anti-inflammatory agents (TCS or TCI), for example twice weekly, to previously affected but clinically healed skin in order to prevent relapses. This strategy has been shown to be more effective than the use of emollients alone. The choice between TCS and TCI should be individualized, taking into account patient age, lesion location, disease severity, and patient preferences, including the frequently encountered phenomenon of steroid phobia, which may adversely affect treatment adherence [10]. Despite the emergence of new therapeutic options, such as phosphodiesterase-4 inhibitors (crisaborole) and Janus kinase inhibitors, TCS and TCI continue to represent the gold standard in the topical treatment of atopic dermatitis [17,22].

REFERENCES

1. Abdel-Mageed HM. Atopic dermatitis: a comprehensive updated review of this intriguing disease with futuristic insights. Inflammopharmacology. 2025;33:1161–87.
2. Afshari M, Kolackova M, Rosecka M, Čelakovská J, Krejsek J. Unraveling the skin; a comprehensive review of atopic dermatitis, current understanding, and approaches. Front Immunol. 2024;15:1361005.
3. Diatta BA, Ben Amara C, Ndiaye JMM, Mendy P, Nibirantije P, Fall N, et al. Atopic dermatitis associated with tropical endemic limbo-conjunctivitis: Epidemiology, clinical phenotype, and allergological investigations. Our Dermatol Online. 2023;14:367-71.
4. Schuler CF, Billi AC, Maverakis E, Tsoi LC, Gudjonsson JE. Novel insights into atopic dermatitis. J Allergy Clin Immunol. 2022;151:1145–54.
5. Kwatra SG, Misery L, Clibborn C, Steinhoff M. Molecular and cellular mechanisms of itch and pain in atopic dermatitis and implications for novel therapeutics. Clin Transl Immunology. 2022;1;11.
6. Tokura Y, Hayano S. Subtypes of atopic dermatitis: From phenotype to endotype. Allergol Int. 2021;71:14–24.
7. Atadokpede F, DégboéB, Adégbitè NE, Akpadjan F, Hounnou d'Almeida M, Alao J, et al. Environmental factors associated with the severity of atopic dermatitis in Cotonou and Parakou, Benin, in 2020. Our Dermatol Online. 2024;15:365-9.
8. Eichenfield LF, Gold LFS, Hebert AA, Guenther L, Valdman-Grinshpoun Y, Ben-Amitai D, et al. Management of Mild-to-Moderate Atopic Dermatitis with Topical Treatments by Dermatologists: A Questionnaire-Based Study. JEADV Clin Pract. 2025;4:471–81.
9. Schoch JJ, Anderson KR, Jones AE, Tollefson MM, Wright T, Hunt R, et al. Atopic Dermatitis: Update on Skin-Directed Management: Clinical Report. Pediatrics. 2025;19;155.
10. Ko HC, Woo YR, Ko JY, Kim HO, Na CH, Bae Y, et al. Consensus-Based Guidelines for the Treatment of Atopic Dermatitis in Korea (Part I): Basic therapy, Topical therapy, and Conventional Systemic Therapy. Ann Dermatol. 2025;37:201.
11. Sarkar R, Dogra S. Standardising the diagnosis and management of atopic dermatitis in India: A consensus statement by the modified Delphi method by IADVL Special Interest Group of Pediatric Dermatology (STAND AD). Indian J Dermatol Venereol Leprol. 2025;0:1–11.
12. Ross G. Treatments for atopic dermatitis. Aust Prescr. 2023;46:9–12.
13. Sideris N, Paschou E, Bakirtzi K, Kiritsi D, Papadimitriou I, Tsentemeidou A, et al. New and Upcoming Topical Treatments for Atopic Dermatitis: A Review of the Literature. J Clin Med. 2022;11:4974.
14. Lazar M, Zhang AD, Vashi NA. Topical Treatments in Atopic Dermatitis: An Expansive review. J Clin Med. 2024;13:2185.
15. Pinto LM, Chiricozzi A, Calabrese L, Mannino M, Peris K. Novel therapeutic strategies in the topical treatment of atopic dermatitis. Pharmaceutics. 2022;14:2767.
16. Zhao S, Hwang A, Miller C, Lio P. Safety of topical medications in the management of paediatric atopic dermatitis: An updated systematic review. Br J Clin Pharmacol. 2023;89:2039–65.
17. Gooderham MJ, Hong HCH, Lynde C, Papp KA, Yeung J, Lui H, et al. Canadian Consensus Guidelines for the Management of Atopic Dermatitis with Topical Therapies. Dermatol Ther. 2025;15:1467–85.
18. Augustin M, Luger T, Pincelli C, Reich A, Baselga E, Wahn U. A practical algorithm for the treatment of mild-to-moderate atopic dermatitis (AD) in pediatric patients in Europe: expert recommendations. J Dermatol Treat. 2025;36:2503281.
19. Al-issa A. Beyond the skin: Unraveling the connection between atopic dermatitis and obsessive-compulsive disorder. Our Dermatol Online. 2025;16:48-51.
20. Perälä M, Salava A, Malmberg P, Pelkonen AS, Mäkelä MJ, Remitz A. Topical tacrolimus versus corticosteroids in childhood moderate-to-severe atopic dermatitis and the impact on airway inflammation: a long-term randomized open-label study. Clin Exp Dermatol. 2023;48:660–6.
21. Kleinman E, Laborada J, Metterle L, Eichenfield LF. What's new in topicals for atopic dermatitis? Am J Clin Dermatol. 2022;23:595–603.
22. Karalus M, Grudina A, Grodzicka J, Biesaga P, Sanko O, Fortuniak W, et al. JAK inhibitors in dermatology: Expanding indications and safety concerns. Our Dermatol Online. 2025;16:e30.
23. Idris FS. Treatment of atopic dermatitis in children. Cureus. 2024;16:e69416.
24. Kreouzi M, Theodorakis N, Prokopiou E, Thomaidou E. Efficacy and safety of dupilumab in adult moderate-to-severe atopic dermatitis: An update narrative literature review. Our Dermatol Online. 2022;13:6-15.
25. Elfatoiki FZ, Mansouri M, Dahbi Skali H, Hali F, Chiheb S. Association atopic dermatitis and psoriasis in Moroccan children. Our Dermatol Online. 2020;11:201-3.
26. Hernandez TD, Aleman SJ, Bao-Loc-Trung M, Forte MV, Brandt W, Armstrong C, et al. Advancing Treatment in Atopic Dermatitis: A comprehensive review of clinical efficacy, safety, and comparative insights into corticosteroids, calcineurin inhibitors, and phosphodiesterase-4 inhibitors as topical therapies. Cureus. 2024;16:e55393.
27. Calabrese G, Licata G, Gambardella A, De Rosa A, Alfano R, Argenziano G. Topical and conventional systemic treatments in atopic dermatitis: Have they gone out of fashion? Dermatol Pract Concept. 2022;12:e2022155.

28. Halubiec P, Jaworek AK, Wojas-Pelc A. Clinical aspects of the treatment of atopic dermatitis with topical glucocorticoids and calcineurin inhibitors — a pilot questionnaire study. Folia Med Cracov. 2023;63:97–108.
29. Aschoff R, Lang A, Koch E. Effects of Intermittent Treatment with Topical Corticosteroids and Calcineurin Inhibitors on Epidermal and Dermal Thickness Using Optical Coherence Tomography and Ultrasound. Skin Pharmacol Physiol. 2021;35:41–50.
30. Pena J, Zameza PA, Pixley JN, Remitz A, Feldman SR. A comparison of topical corticosteroids and topical calcineurin inhibitors for the treatment of atopic dermatitis. J Allergy Clin Immunol Pract. 2023;11:1347–59.
31. Wilson MM. Effectiveness of Topical Treatment of Steroid and Calcineurin Inhibitor Combination Therapy for Atopic Dermatitis in Children. Serb J Dermatol Venereol. 2024;2:51-6.
32. Mohamed AA, Borolossy RE, Salah EM, Hussein MS, Muharram NM, Elsalawy N, et al. A comparative randomized clinical trial evaluating the efficacy and safety of tacrolimus versus hydrocortisone as a topical treatment of atopic dermatitis in children. Front Pharmacol. 2023;14:1202325.
33. Bratu D, Boda D, Caruntu C. Reflectance confocal microscopy in monitoring atopic dermatitis treated with topical calcineurin inhibitors. Healthcare. 2023;11:152.
34. Handa S, Chandrasegaran A, Kanwar A, Mahajan R. Comparing the effectiveness of topical fluticasone 0.05% cream versus topical tacrolimus 0.1% ointment in pediatric atopic dermatitis: A randomized controlled trial. Indian J Paediatr Dermatol. 2022;23:111.
35. Wollenberg A, Werfel T, Ring J, Ott H, Gieler U, Weidinger S. Atopic dermatitis in children and adults. Dtsch Arztebl Int. 2023;120:224–34.
36. Gether L, Storgaard H, Kezic S, Jakasa I, Hartmann B, Skov-Jeppesen K, et al. Effects of topical corticosteroid versus tacrolimus on insulin sensitivity and bone homeostasis in adults with atopic dermatitis—A randomized controlled study. Allergy. 2023;78:1964–79.
37. Harvey J, Lax SJ, Lowe A, Santer M, Lawton S, Langan SM, et al. The Long-Term Safety of Topical corticosteroids in atopic dermatitis: a Systematic review. Skin Health Dis. 2023;3:e268.
38. Umar BU, Rahman S, Dutta S, Islam T, Nusrat N, Chowdhury K, et al. Management of atopic dermatitis: The role of tacrolimus. Cureus. 2022;14:e28130.
39. Dhar S, De A, Saha A, Chitnis KR, Mane A, Dhoot D, et al. Intermittent or sequential topical tacrolimus in atopic dermatitis: systematic review and meta-analysis. Cureus 2023;15:e50640.
40. Mikkelsen CS, Poulsen CB, Bjerring P, Hove LS. Skin diseases in the world`s indigenous peoples – with special focus on Greenland's Inuit`s population. Our Dermatol Online. 2024;15:179-87.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

miniReview Article

Can environmental changes modify the manifestations of cutaneous diseases and via what mechanism?

Khalifa E Sharquie[1], Thamir A Kubaisi[2]

[1]Department of Dermatology, College of Medicine, University of Baghdad, Iraqi and Arab Board of Dermatology and Venereology, Center of Dermatology and Venereology, Baghdad Teaching Hospital, Medical City, Medical Collection Office, Baghdad, Iraq, [2]Department of Internal Medicine/Dermatology, College of Medicine, University of Anbar; Head of Ramadi Center of Iraqi Board of Dermatology and Venereology, Ramadi Teaching Hospital; Ramadi, Anbar, Iraq

Corresponding author: Prof. Khalifa E Sharquie, MD PhD, E-mail: ksharquieprof@yahoo.com

ABSTRACT

The environment exerts a profound influence on both skin health and overall human well-being. As the body's primary protective barrier, the skin safeguards against harmful chemicals, ultraviolet radiation, and repeated exposure to water. Understanding how environmental factors interact with cutaneous biology is essential for effective prevention, diagnosis, and management of skin disorders. The skin also acts as a visible indicator of systemic and external changes, reflecting alterations that may arise through epigenetic mechanisms. These mechanisms can modify gene expression, trigger new dermatologic conditions, impair immune responses, and contribute to resistance to treatment. Hence, dermatologists must remain attentive to environmental shifts, recognizing their impact on skin function and disease patterns to promote comprehensive patient care.

Key words: Environment, Skin health, Epigenetic mechanisms, Vaccine

It has long been proven that the surrounding environment plays an important role in the appearance of the skin and general health. The main function of the skin is to preserve the human body and protect from harmful substances, whether they are noxious chemicals, ultraviolet light, or continued/frequent exposure to water [1]. Understanding the interaction between the environment and the skin and its diseases is vital to reach effective prevention and management. In order to encourage skin health within these environmental stresses, medical doctors must keep in mind the following 16 ideal issues to support human well-being (Fig. 1):

- Pathogens: In a special community media, friendly kissing is considered to have an important role in molluscum contagiosum skin infection [2]. During the COVID-19 pandemic, numerous skin manifestations were reported, such as hair loss, diffuse petechiae, chilblain-like lesions, acneiform rash, pyoderma gangrenosum, and unusual skin hypersensitivity to drugs [3,4].
- Vaccine: Vaccination strategies are linked to adverse effects, including a variety of skin lesions ranging from pruritus, tenderness, erythema, and edema, to erosions, papules, and pigmentation [5]. All over the world, several autoimmunological skin reactions may happen after COVID-19 vaccination. For example, urticaria and angioedema represent type-I hypersensitivity responses, whereas erythema multiforme-like rashes, leukocytoclastic vasculitis, systemic lupus erythematosus, and thrombocytopenia represent type-IV hypersensitivity responses [6].
- Insect bite: Mites such as scabies and head lice are the main infestations in children. Bite reactions caused by fleas and bed bugs are common sources that present with chronic and recurrent papular urticaria [7].
- Pollution (industrial): Similar to ultraviolet radiation, airborne pollutants cause skin pigmentation. Eumelanin has an antioxidative effect, and it is

How to cite this article: Sharquie KE, Kubaisi TA. Can environmental changes modify the manifestations of cutaneous diseases and via what mechanism? Our Dermatol Online. 2026;17(2):258-261.

Submission: 02.06.2025; **Acceptance:** 08.09.2025

DOI: 10.7241/ourd.20262.23

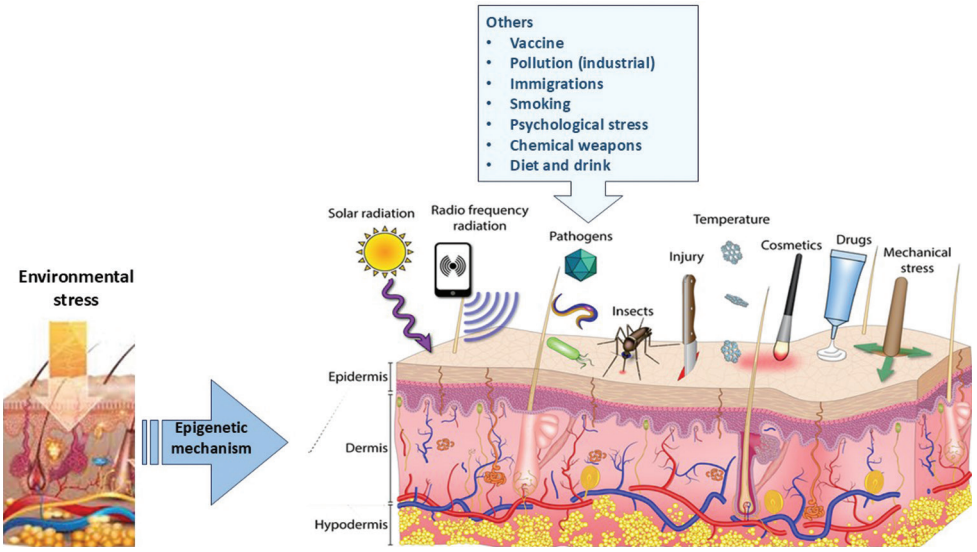


Figure 1: The environmental factors that might harm the human skin (modified; medical video).

anticipated that this response acts as a protective strategy against oxidative stress caused by air pollution [8]. The study revealed an epidemic of acne vulgaris caused by air pollutants, such as sulfur dioxide and nitric dioxide, which may increase the concentration of cutaneous oxidative stress, which leads to acne flares. Furthermore, the raised humidity contributing to keratinocyte swelling and hair follicle obstruction can, thus, intensify the growth of *Cutibacterium acnes* [9]. On the other hand, current epidemiological studies show that the black carbon elements and polycyclic aromatic hydrocarbons increase the incidence of cutaneous cancer [9].

- Solar radiation: Sun exposure and the increased risk of skin tumors, particularly for fair-skinned people, remains a matter of debate. Additional cutaneous disorders associated with ultraviolet radiation are sunburn, phototoxic and photoallergic reactions, solar UV damage, long-lasting pigmentation, melasma, and others [10].
- Extreme temperature changes: The important role of the skin is thermoregulation or homeostasis. Frostbite might happen in a very cold situation. Moderate degrees of cold may produce cold urticaria, chilblains, acrocyanosis, cold panniculitis, Raynaud's syndrome, and cryoglobulinemia [11]. Prolonged exposure to an elevated temperature and humidity increases the prevalence of miliaria, types of physical urticarias, and erythema ab igne. Moreover, it can disturb sebum production and skin hydration. This can encourage the growth of *Cutibacterium acnes* and be responsible for the epidemic of acne vulgaris in hot climate countries [9,11]. An association between irritant skin changes and low temperatures or humidity has been well-known. Dry and cold weather upsurges the risk of flares of atopic dermatitis [12].
- Diet: In recent years, the role of nutritional alterations as a means to treat or prevent dermatological diseases has received special consideration. Eating habits, chiefly the eating of dairy products and highly glycemic foods, have been associated with the severity of acne, through the exacerbation of sebum production and local irritation [9]. Numerous dietary issues such as spicy foods, hot beverages, caffeine, vanilla, alcohol, and cinnamon have been considered triggers for seborrheic dermatitis or rosacea [13]. Some possible mechanisms of acne involve spicy foods. The idea that this might trigger the disease is a matter of controversy, and there are limited studies. The probiotics showed excessive potential in stopping and managing cutaneous disorders, including acne, atopic dermatitis, urticaria, solar skin damage, wound protection, and cosmetic substances [14]. Moreover, the ketogenic diet is used in treating skin diseases, such as psoriasis, acne, and hidradenitis suppurativa. Conversely, this regimen could produce prurigo pigmentosa and nutritional deficiencies as an abnormal cutaneous reaction [15].
- Chemical weapons: Previous studies from Iraq anticipated the dramatic upsurge in the frequency and severity of mycosis fungoides, Kaposi sarcoma, Paget's disease, squamous cell carcinoma, and dermatofibrosarcoma protuberans that were

significantly influenced by war crimes [16]. Skin disorders have been an imperative health alarm in military employees throughout times past. The spectrum of cutaneous diseases in the Gulf War's military was eczema (seborrheic dermatitis), and infections (bacterial, viral, or fungal), which accounted significantly for the majority of referrals. During World War II, up to 75% of all clinical presentations were skin complaints. These were attributed principally to the different types of used weapons [17].

- Immigration: Immigrants showed an increased hazard for many skin diseases, especially for scabies and bacterial infections. Recently, the incidence of resistant fungal infections of the skin has increased. The exact causes behind this are still obscure, but it is generally prevalent among immigrants [18,19].
- Radiofrequency: Ablative and non-ablative radiofrequency was used to improve and rejuvenate damaged skin. The described drawback reactions include swelling, edema, erythema, crusting, hyperpigmentation, and acne [20].
- Injury, detergents, chemical and mechanical stress: Daily use of detergents by housewives, as well as mechanical stress by farmers, is frequently associated with irritant hand dermatitis. Likewise, the Pacha boil (Orf) affected the injured hands of housewives [21].
- Smoking: Cigarette smoking leads to increased skin changes, including cancer, and accelerates the natural course of skin aging [22].
- Hair dyes and cosmetics: Black color hair dyeing may be associated with several dermatological diseases, ranging from allergic contact dermatitis to facial melanosis and erythema multiforme-like eruption [23]. The common adverse skin reactions to facial cosmetics or perfumes are erythema, xerosis, and pruritus. Cosmetic acne is a known induced disease variant [9].
- Psychological stress: Psychoneuroimmunology provides locally expressed complex stress-induced reactions that have been established as active in many cutaneous diseases such as chronic urticaria, psoriasis, atopic dermatitis, warts, hair loss, and acne. Psychological stress might induce numerous cutaneous diseases such as dermatitis and hair loss. A previous study proposed a significant association between psychological stress and the severity of acne. Stress leads to hormonal variations that raise sebum production, which has been linked to the development of acne vulgaris [9].
- Drugs and herbal effects: There were various reported cutaneous side effects caused by many injectable, oral, or topical drugs, as well as an excessive intake of antibiotics, tonics, and herbals for certain reasons, whether by doctors or non-doctors. In certain circumstances, this might influence the behavior of numerous skin diseases by affecting gene functions or expression (epigenetic mechanism). In addition, this impact on the host immunological influence leads to the reappearance of many autoimmune or genetic diseases such as benign bullous disease of childhood and lipoid proteinosis, as it happened in Iraq since these diseases were common diseases before the 1990s, then completely disappeared, and now, they are returning [24].
- During outbreaks of infections and epidemics: There will be changes in the cutaneous manifestations of these diseases as they become more widespread, with new cutaneous features and more resistance to therapies as observed during outbreaks of scabies, cutaneous tuberculosis, and dermatophytosis. The frequency of autoimmune diseases such as vitiligo and alopecia areata increased during the COVID-19 epidemic [4,25].

In conclusion, the skin may be considered a mirror that reflects what is happening inside or outside of the body. Accordingly, any environmental change that affects the body will be reflected in the function and behavior of the skin. This could be explained by the epigenetic mechanism through which there are changes in the expression and functions of genes, hence the emergence of new cutaneous manifestations, new skin diseases, and impaired immunological reactions with the appearance of resistance to drug therapy. Therefore, dermatologists should be aware of what happens in their surrounding environment and what will be reflected in the human skin function and behavior.

REFERENCES

1. English J, Dawe R, Ferguson J. Environmental effects and skin disease. Br Med Bull. 2003;68:129-42.
2. Kubaisi TA. Molluscum contagiosum of the face: Kissing as an impact cause of viral transmission. J Pak Assoc Dermatol. 2023;33:1004-8.
3. Farhood IG, AbdulHasan AT, Lafta SF, Mamoori A, Al-Hasani F. Dermatological manifestations of patients with COVID-19: A cross-sectional study. Al-Anbar Medical Journal. 2022;18:43-7.
4. Sharquie KE, Sharquie IK. The frequency of autoimmune diseases among patients with COVID-19 infection. J Pak Assoc Dermatol. 2022;32:532-8.
5. Gutiérrez RL, Porter CK, Jarell A, Alcala A, Riddle MS,

Turiansky GW. A grading system for local skin reactions developed for clinical trials of an intradermal and transcutaneous ETEC vaccine. Vaccine. 2020;38:3773-9.
6. Gambichler T, Boms S, Susok L, Dickel H, Finis C, Abu Rached N, et al. Cutaneous findings following COVID-19 vaccination: Review of world literature and own experience. J Eur Acad Dermatol Venereol. 2022;36:172-80.
7. Kamath S, Kenner-Bell B. Infestations, bites, and insect repellents. Pediatra Ann. 2020;49:e124-e31.
8. Grether-Beck S, Felsner I, Brenden H, Marini A, Jaenicke T, Aue N, et al. Air pollution-induced tanning of human skin. Br J Dermatol. 2021;185:1026-34.
9. Kim HJ, Kim YH. Exploring acne treatments: From pathophysiological mechanisms to emerging therapies. Int J Mol Sci. 2024;25:5302.
10. Merin K, Shaji M, Kameswaran R. A review on sun exposure and skin diseases. Indian J Dermat. 2022;67:625.
11. Page EH, Shear NH. Temperature-dependent skin disorders. J Am Acad Dermatol. 1988;18:1003-19.
12. Engebretsen K, Johansen JD, Kezic S, Linneberg A, Thyssen J. The effect of environmental humidity and temperature on skin barrier function and dermatitis. J Eur Acad Dermatol Venereol. 2016;30:223-49.
13. Alia E, Feng H. Rosacea pathogenesis, common triggers, and dietary role: The cause, the trigger, and the positive effects of different foods. Clinic Dermatol. 2022;40:122-7.
14. Roudsari MR, Karimi R, Sohrabvandi S, Mortazavian A. Health effects of probiotics on the skin. Crit Rev Food Sci Nutr. 2015;55:1219-40.
15. Harb D, Abdullah L, Kurban M, Abbas O. Ketogenic diet and the skin. Skinmed. 2023;21:315-20.
16. Sharquie KE, Kubaisi TA. Mycosis fungoides: Frightening up-surging cases as a new challenge for dermatologists and as a witness of the crime of frequent wars. J Pak Assoc Dermatol. 2023;33:1225-7.
17. Higgins E, Ismail K, Kant K, Harman K, Mellerio J, Du Vivier A, et al. Skin disease in Gulf war veterans. QJM. 2002;95:671-6.
18. Sirka CS, Behera B, Pradhan S, Rout AN, Sahu K, Dash G, et al. Diagnosis of a recurred lesion in dermatophytosis patients after 2 weeks of antifungal therapy: A prospective observational study. J Family Med Prim Care. 2024;13:465-70.
19. Kubaisi TA. Resistant dermatophytosis: A stubborn habit and major challenge in Iraq. Al-Anbar Medical Journal. 2023;19:78-80.
20. Vassão PG, Balão AB, Credidio BM, Do Vale GCA, Assis Garcia L, Martignago CCS, et al. Radiofrequency and skin rejuvenation: A systematic review. J Cosmet Laser Ther. 2022;24:9-21.
21. Kubaisi TA. Does Orf (Pacha boil) affect housewives? Biomedicine. 2022;42:297-303.
22. Urbańska M, Nowak G, Florek E. Cigarette smoking and its influence on skin aging. Przegl Lek. 2012;69:1111-4.
23. Sharquie KE, Abdulwahhab WS, Sharquie IK. Black hair dye dermatitis is a major cause of concealed facial melanosis in patients with dark complexion. J Pak Assoc Dermatol. 2024;34:474-9.
24. Sharquie IK, Sharquie KE. The role of epigenetics in immunosuppression in patients with infectious diseases as a fascinating applied observation. J Pak Assoc Dermatol. 2023;33:275-82.
25. Sharquie KE, Jabbar RI. New multiple therapy of resistant and relapsing dermatophyte infections during epidemic status. J Pak Assoc Dermatol. 2022;32:464-71.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Clinical Image

Idiopathic scrotal calcinosis: The extensive case of a young adult

Rihame Al Heyasat[1], Sara Elloudi[1], Laila Tahiri Ousrouti[2], Houda Elabbade[1], Zakia Douhi[1], Meryem Soughi[1], Hanane Baybay[1], Fatima Zahra Mernissi[1]

[1]Department of Dermatology, University Hospital Hassan II, Fes, Morocco, [2]Department of Anatomopathology, University Hospital Hassan II, Fes, Morocco

Corresponding author: Rihame Al Heyasat, MD, E-mail: rihame.alheyasat@usmba.ac.ma

Idiopathic scrotal calcinosis (ISC) is a rare benign condition, first described by Lewinski in 1883 [1]. Its etiopathogenesis remains controversial and has not yet been fully elucidated. ISC is characterized by the presence of multiple calcified nodules within the scrotal wall. The condition predominantly affects young adults, most commonly between 20 and 40 years of age, although the majority of reported cases involve patients aged 20 to 50 years [2,3]. Due to the benign nature of the symptoms and the slow progression, the interval between the onset of the first lesions and therapeutic intervention is often prolonged.

Clinically, the nodules gradually increase in size, eventually forming tumor-like masses that may ulcerate and discharge a whitish, chalky material. No extra-scrotal involvement is observed. Laboratory investigations, including serum and urinary calcium-phosphate levels, uric acid, alkaline phosphatase, parathyroid hormone, calcitonin, and vitamin D, are typically within normal ranges. Histological examination confirms the diagnosis by demonstrating intradermal calcium deposits. Surgical excision is the treatment of choice and is curative in most cases, with recurrence being uncommon.

Herein, we report the case of a 39-year-old patient, followed in psychiatry for schizophrenia under treatment, who presented with painless scrotal nodules that had been evolving for several years. The lesions had progressively increased in size and occasionally ruptured, discharging a chalky white material. Clinical examination revealed multiple yellowish, calcified, hard, stone-like nodules, sometimes clustered into tumor-like masses occupying nearly the entire scrotum (Fig. 1). The nodules were firm but mobile relative to the underlying planes. The testes and epididymides were clinically normal. Laboratory tests were normal. Histopathological analysis of a skin biopsy showed a regular epidermis overlying dermal calcium nodules, with a focal foreign body granulomatous reaction, confirming the diagnosis of ISC (Fig. 2). Complete surgical excision was performed, and no recurrence was observed after twelve months of follow-up.

Figure 1: Multiple yellowish, confluent scrotal nodules, forming true tumor-like masses.

How to cite this article: Al Heyasat R, Elloudi S, Ousrouti LT, Elabbade H, Douhi Z, Soughi M, Baybay H, Mernissi FZ. Idiopathic scrotal calcinosis: The extensive case of a young adult. Our Dermatol Online. 2026;17(2):262-263.

Submission: 23.08.2025; **Acceptance:** 09.12.2025

DOI: 10.7241/ourd.20262.24

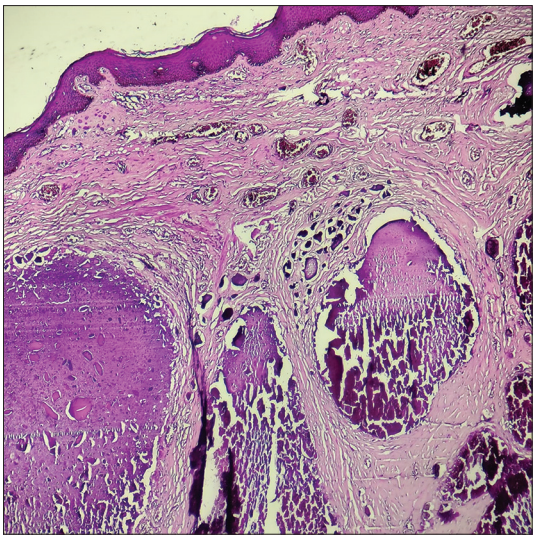


Figure 2: Histopathological examination revealing a normal epidermis with dermal calcium deposits and a focal foreign body granulomatous reaction.

Consent

The examination of the patient was conducted according to the Declaration of Helsinki principles.

REFERENCES

1. Lewinski HM. Lymphangiome der Haut mit Verkalkleminhalt. Arch Pathol Anat. 1883;91:371-7.
2. Ito A, Sakamoto F, Ito M. Dystrophic scrotal calcinosis originating from benign eccrine epithelial cysts. Br J Dermatol. 2001;144:146-50.
3. Ruiz Genao DP, Rios Buceta L, Herrero L, Fraga J, Aragues M, Garcia Diez A. Massive scrotal calcinosis. Dermatol Surg. 2002;28:745-7.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Letter

Neurofibromatosis type 1 (NF1): Experience of the Dermatology Department at Mohammed VI University Hospital in Oujda, Morocco

Rania Bouabdallaoui[1], Nada Tahri[1], Nassiba Zerrouki[2], Nada Zizi[1,2]

[1]Department of Dermatology, Venereology and Allergology, Mohammed VI University Hospital, Oujda, Morocco, [2]Laboratory of Epidemiology, Scientific Research and Public Health, Faculty of Medicine and Pharmacy, Mohammed First University, Oujda, Morocco

Corresponding author: Rania Bouabdallaoui, Dr, E-mail: raniabouabed123@gmail.com

Sir,

Neurofibromatosis type 1 (NF1) is an autosomal dominant disorder caused by mutations in the NF1 gene located on chromosome 17, which encodes neurofibromin, a protein regulating cell growth [1,2].

This multisystem condition is characterized by cutaneous abnormalities (café-au-lait spots, neurofibromas), bone disorders (dysplasias, scoliosis), and neurological involvement (optic pathway gliomas, cognitive impairments) [3,4]. Diagnosis is based on clinical criteria, with skin manifestations often serving as the earliest indicators [3].

Dermatologists play a key role in the early detection and management of systemic complications [2,3,6].

The objective of this study was to evaluate the prevalence of complications associated with NF1 and to describe the epidemiological, clinical, and paraclinical characteristics of affected patients with the aim of improving multidisciplinary management and follow-up of this complex condition.

We conducted a retrospective, descriptive study over a ten-year period (June 2014–June 2024) at the Mohammed VI University Hospital in Oujda, including all patients diagnosed and followed for NF1 at our department.

A total of 28 patients were included, with a mean age of 14 years (range: 5–53 years). Among them, 17 were female (60.71%) and 11 were male (39.29%). 46.4% had a first-degree family history of NF1, while 4.14% had second-degree relatives with the condition.

Regarding medical history, 21.4% of the patients had learning difficulties and a history of academic failure. Neurological manifestations were reported in 4% of the patients, with headaches being the most common symptom.

The main reason for consultation was the presence of hyperpigmented lesions, observed in 82.14% of the patients. Café-au-lait spots and lentigines were present in all cases. Crowe's sign was positive in 35% of the patients, and cutaneous or subcutaneous neurofibromas were observed in 67.8%. Plexiform neurofibromas were identified in 6 patients (21.4%).

As for paraclinical investigations, Lisch nodules were detected during the ophthalmologic examination in 9 patients (32.14%). Bone involvement was noted in 3 patients (10.71%), and 7 patients (25%) had lymphadenopathy on ultrasound.

All patients underwent brain MRI. Nine (32.14%) showed unidentified bright objects (UBOs), and 3 had intracranial tumors, one of which resulted in bulbo-medullary compression. These patients were referred to neurosurgery for further management.

Neurofibromatosis type 1 (NF1) is an autosomal dominant genetic disorder with an estimated prevalence of 1 in 2,500 to 3,000 births [2]. Clinical manifestations

How to cite this article: Bouabdallaoui R, Tahri N, Zerrouki N, Zizi N. Neurofibromatosis type 1 (NF1): Experience of the Dermatology Department at Mohammed VI University Hospital in Oujda, Morocco. Our Dermatol Online. 2026;17(2):264-265.

Submission: 02.06.2025; **Acceptance:** 07.09.2025

DOI: 10.7241/ourd.20262.25

are diverse, involving the skin, nervous system, and bones, consistent with our observations. Café-au-lait spots, present in all patients, as well as lentigines and Crowe's sign (35%), align with existing literature on the cutaneous manifestations of NF1 [1].

Cutaneous and subcutaneous neurofibromas were seen in 67.8% of the cases, which was in line with reported data [3]. Plexiform neurofibromas were present in 21.4% of the cases, which is lower than rates described in the literature (up to 50%) [2]. Neurologically, our findings supported the high prevalence of low-grade gliomas among young adults with NF1 [2].

The presence of Lisch nodules (32.14%) and bone abnormalities (10.71%) in our cohort was consistent with literature-reported rates, highlighting the importance of ophthalmologic screening and radiological evaluation of skeletal anomalies in these patients [3,5].

The identification of space-occupying lesions requiring neurosurgical intervention emphasizes the critical need for multidisciplinary surveillance [2,5].

Despite the limitation of a small sample size, our findings underscored the importance of close follow-up for the early detection of tumor and skeletal complications and to optimize patient outcomes [6].

The dermatologist plays a crucial role in the early detection of NF1 manifestations and in identifying patients at risk of developing severe tumor or skeletal complications. Multidisciplinary monitoring remains essential to ensure optimal management of this complex disease [1,2].

Statement of Human and Animal Rights

All the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the 2008 revision of the Declaration of Helsinki of 1975.

Statement of Informed Consent

Informed consent for participation in this study was obtained from all patients.

REFERENCES

1. Association Neurofibromatose Québec. Recommandations pour la prise en charge adulte des patients atteints de neurofibromatose de type 1. ANFQ. 2022.
2. Bertrand N, Valeyrie-Allanore L, Wolkenstein P, Vidaud D, Pinson S, Layet V, et al. Neurofibromatosis 1 French national guidelines based on an extensive literature review since 1966. Orphanet J Rare Dis. 2020;15:25.
3. Pérez-Atamoros F, Zavala-Cerna MG, García-Cruz D, Vega-Memije ME. Dermatologic manifestations of neurofibromatosis type 1 and their impact on quality of life in adult patients. Orphanet J Rare Dis. 2023;18:215.
4. Kuorilehto T, Nissinen M, Koivunen J, Väänänen K, Peltonen J. Musculoskeletal manifestations of neurofibromatosis type 1. AJR Am J Roentgenol. 2012;199:E102-9.
5. Li Z, Bhavana R, Surendra S, Xiuli L, Jingjun Z. Advances in targeted strategies for managing neurofibromatosis type 1 related tumors. Our Dermatol Online. 2023;14:311-8.
6. Hadjkacem F, Gargouri I, Kalthoum M, Elleuch M, Charfi N, Abid M. A pheochromocytoma in a patient with neurofibromatosis type 1: A case report. Our Dermatol Online. 2020;11:e174.1-

Source of Support: Nil, **Conflict of Interest:** None declared.

Our Dermatology Online

Case Letter

Combined vacuum therapy and superficial micropuncture in the treatment of white striae: A case series

Airton Barbalho Rodrigues

AB Clínica de Medicina Estética, Castellón de la Plana, Spain

Corresponding author: Airton Barbalho Rodrigues, E-mail: airtonb.contato@gmail.com

Sir,

Striae distensae are common dermal lesions resulting from mechanical stretching, hormonal influences, and connective tissue alterations [1-3]. White striae (striae distensae alba) represent a chronic stage characterized by dermal atrophy, disruption of collagen and elastic fibers, hypopigmentation, and reduced local vascularization [2,3].

Several therapeutic modalities have been proposed to stimulate dermal remodeling in striae, including lasers, radiofrequency, and microneedling [4-6]. However, outcomes remain variable, especially in long-standing white striae [1,4].

Vacuum therapy is applied using low to moderate negative pressure, adjusted according to the tissue response and patient tolerance, promoting transient tissue deformation and increased microcirculation [7].

Following vacuum stimulation, superficial micropuncture is performed at the epidermal–dermal interface using a dermograph equipped with a single-tip needle [5,6].

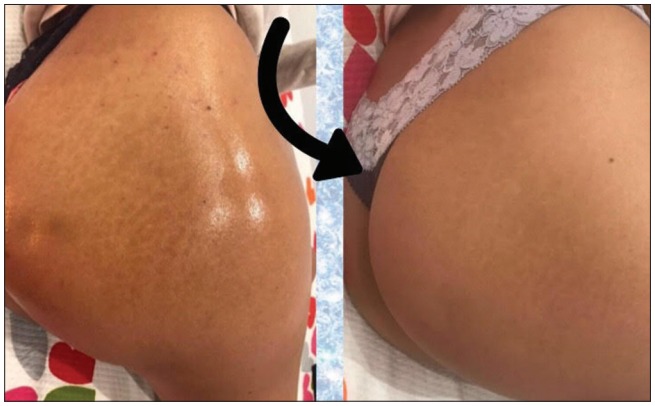


Figure 1: Immediate localized inflammatory response observed after the application of the combined vacuum therapy and superficial micropuncture technique in the gluteal region, characterized by transient hyperemia and capillary micro-extravasation.

An immediate localized inflammatory response following the combined procedure, characterized by transient hyperemia, is illustrated in Fig. 1 [7].

Mechanical negative pressure has been associated with increased cutaneous microcirculation and stimulation of fibroblast activity, contributing to collagen synthesis and dermal remodeling [7] (Figs. 2 – 4).

Superficial micropuncture induces controlled micro-injuries that activate a wound-healing cascade [5,6].

How to cite this article: Barbalho Rodrigues A. Combined vacuum therapy and superficial micropuncture in the treatment of white striae: A case series. Our Dermatol Online. 2026;17(2):266-267.

Submission: 14.12.2025; **Acceptance:** 01.02.2026

DOI: 10.7241/ourd.20262.26

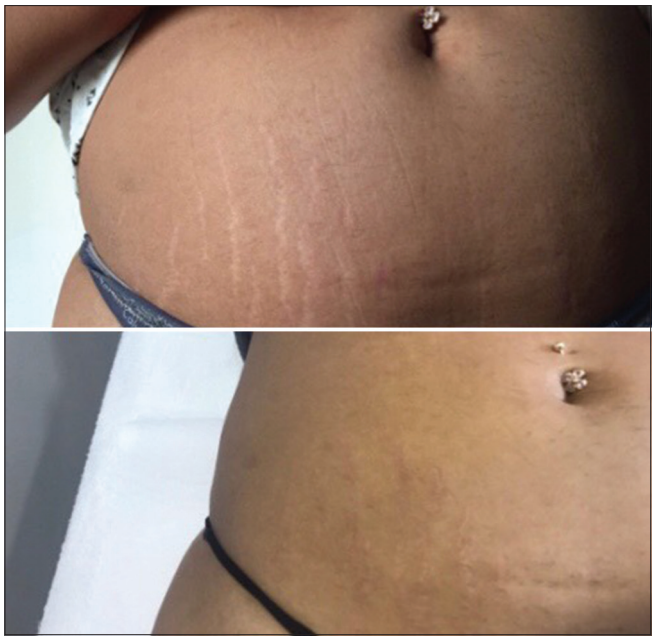


Figure 2: Female patient with hypopigmented atrophic striae in the gluteal region before treatment and after the completion of the combined vacuum therapy and superficial micropuncture protocol, showing improved texture uniformity and partial repigmentation.

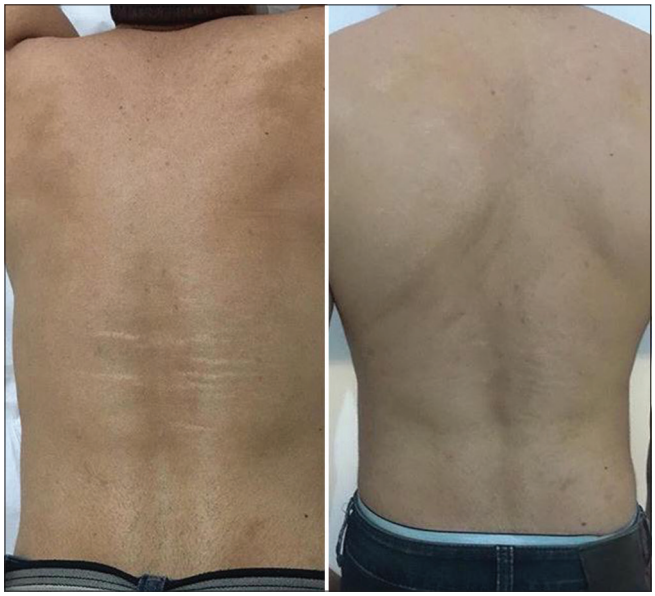


Figure 3: Female patient with post-pregnancy white atrophic striae in the abdominal region before treatment and after the completion of the combined vacuum therapy and superficial micropuncture protocol, demonstrating improved tone uniformity and dermal texture.

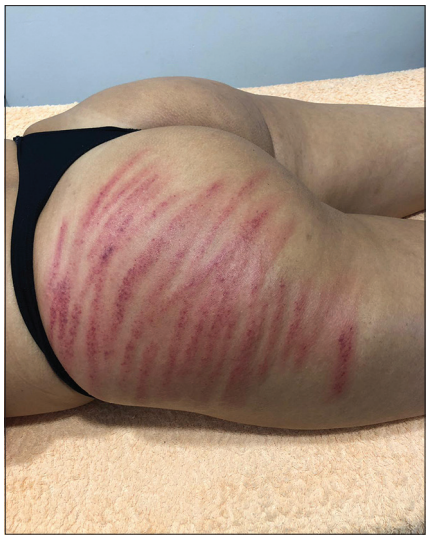


Figure 4: Male patient with horizontal white striae developed during puberty in the dorsal region before treatment and after the completion of the combined vacuum therapy and superficial micropuncture protocol, showing reduced visibility of the striae and improved surface homogeneity.

REFERENCES

1. Elsaie ML. Striae distensae: A review and evidence-based evaluation of treatment options. J Cosmet Dermatol. 2009;8: 293-303.
2. Watson RE, Parry EJ, Humphries JD, Jones CJ, Polson DW, Kielty CM, et al. Fibrillin microfibrils are reduced in skin exhibiting striae distensae. Br J Dermatol. 1998;138:931-7.
3. Ud-Din S, Bayat A. New insights on striae distensae. J Eur Acad Dermatol Venereol. 2013;27:679-89.
4. Alexiades-Armenakas M. Laser and light-based treatments of striae distensae. J Drugs Dermatol. 2009;8:1023-5.
5. Fernandes D. Minimally invasive percutaneous collagen induction. Oral Maxillofac Surg Clin North Am. 2005;17:51-63.
6. Lima EV, Lima MM, Takano D. Microneedling in facial recalcitrant melasma. An Bras Dermatol. 2015;90:919-21.
7. Atiyeh BS, El Khatib AM, Dibo SA. Pressure and vacuum-assisted therapies in dermatology and wound healing. Ann Plast Surg. 2013;70:241-7.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Case Letter

A case of lupus erythematosus profundus unresponsive to hydroxychloroquine but successfully treated with belimumab

Natsuko Matsumura, Tatsuhiko Mori, Toshiyuki Yamamoto

Department of Dermatology, Fukushima Medical University, Fukushima, Japan

Corresponding author: Prof. Toshiyuki Yamamoto, MD PhD, E-mail: toyamade@fmu.ac.jp

Sir,

A 42-year-old female was diagnosed with systemic lupus erythematosus (SLE) based on the findings of malar rash, chilblain lupus in the digits, Raynaud phenomenon, joint pain in the extremities, positive serum anti-nuclear antibody (1:320, homogeneous and speckled), anti-Sm antibody, and hypocomplementemia, at the age of 16 years. She was under follow-up with oral prednisolone (10 mg/day) at our hospital. She presented to our department, complaining of tenderness of the scalp, which began one year previously during the course of prednisolone tapering. Physical examination revealed painful, indurated plaques and subcutaneous nodules in the postauricular and occipital regions with hair loss (Fig. 1). A biopsy revealed lobular panniculitis with focal dermal infiltration of inflammatory cells (Fig. 2a). Higher magnification revealed fat cell degeneration, lipogranuloma with foam cells, lymphohistiocytic and neutrophil infiltration in the subcutis, hyaline fat degeneration, and fibrosis of the subcutaneous tissues (Fig. 2b). Direct immunofluorescence examination showed linear deposition of IgM in the epidermal basement membrane (Fig. 2c). Immunohistochemistry revealed that mononuclear cells were positive for CD3, CD4, CD8, CD20, CD27, and CD79a (Figs. 3a – 3d). The addition of dapsone (75 mg/day), methotrexate, and hydroxychloroquine (HCQ) (200 mg/400 mg on alternate days) resulted in little effect. In the second biopsy, which was taken twelve months after the initial biopsy, CD3-positive cells were markedly reduced in the dermis but still observed in the subcutaneous tissues, whereas CD20-positive cells were unchanged in the dermis (Figs. 3e and 3f). Belimumab (200 mg) was then subcutaneously administered every week, and six months later, the induration and pain disappeared, and her alopecia recovered.

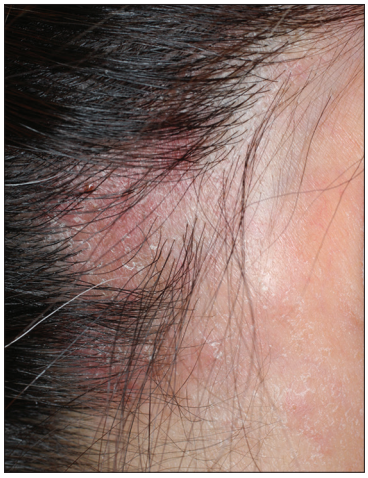


Figure 1: Tender subcutaneous nodules with superficial scaly erythema on the right postauricular area.

B-cells have been implicated in the pathogenesis of SLE due to production of autoantibodies and cytokines [1,2]. B-cells are reported to infiltrate the lesional skin of discoid LE [3,4], and high expression of B-cell activating factor (BAFF) is observed in cutaneous LE (CLE), including LE profundus (LEP). Interferon-α (IFN-α) is a key cytokine in the pathogenesis of SLE, and has multiple effects, one of which is the upregulation of BAFF [5]. Recent studies have shown that patients with chronic CLE share B-cell abnormalities and expansion of effector B-cell subsets, suggesting that chronic CLE may benefit from B-cell

How to cite this article: Natsuko Matsumura, Tatsuhiko Mori, Toshiyuki Yamamoto. A case of lupus erythematosus profundus unresponsive to hydroxychloroquine but successfully treated with belimumab. Our Dermatol Online. 2026;17(2):268-269.

Submission: 12.08.2025; **Acceptance:** 14.10.2025

DOI: 10.7241/ourd.20262.27

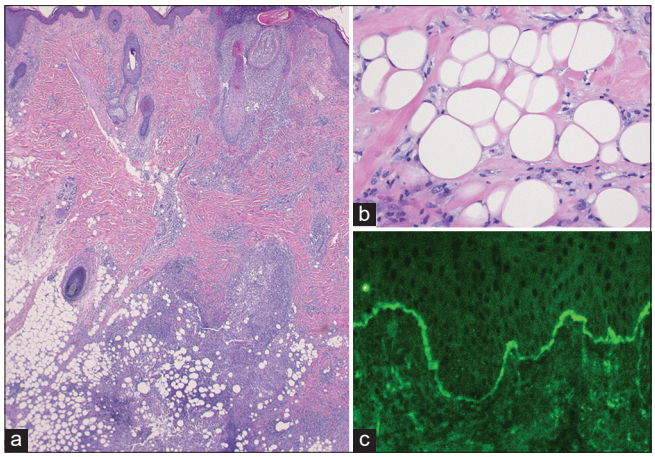


Figure 2: a) Histopathological features showing lobular panniculitis with focal dermal infiltration of mononuclear cells. b) Higher magnification showing fat cell degeneration and sclerotic stroma. c) DIF showing IgM deposition in the basement membrane.

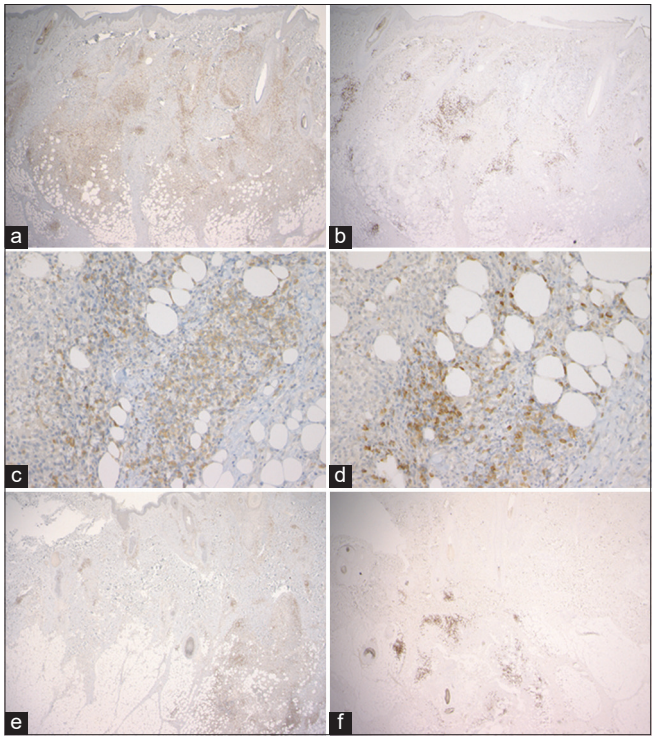


Figure 3: Immunohistochemistry showing positive expression of a) CD3, b) CD20, c) CD27, d) and CD79a. Expression of e) CD3 and f) CD20 in the scalp lesion of the second biopsy (original magnification: b×20, c×400, d×200, e×20, f×20, g×200, h×200, i×20, j×20).

targeting therapies [6]. In the present case, CD20-, CD27 (memory B-cells)-, and CD79a-positive B-cells were observed in the subcutis, suggesting the role of B-cells in LEP.

Belimumab is a human monoclonal antibody that specifically binds to soluble B-lymphocyte stimulator (BLyS), and has been used for SLE. Belimumab showed significant improvements in maculopapular eruption, alopecia, and DLE in patients with SLE [7]; however, reports on the clinical response of CLE to belimumab are scarce at present. LEP is sometimes resistant to various therapies, and the response rate of LEP to HCQ is lower among CLE subtypes. Our patient with SLE suffered from tenderness and alopecia, which were unresponsive to previous therapies but successfully treated with belimumab. Our case may suggest that B-cells play an important role in the pathogenesis of LEP, and belimumab is a promising new therapy for refractory LEP.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Dörner T, Lipsky PE. The essential roles of memory B cells in the pathogenesis of systemic lupus erythematosus. Nat Rev Rheumatol. 2024;20:770-82.
2. Stockfelt M, Teng YKO, Vital EM. Opportunities and limitations of B cell depletion approaches in SLE. Nat Rev Rheumatol. 2025;21:111-26.
3. O'Brien JC, Hosler GA, Chong BF. Changes in T cell and B cell composition in discoid lupus erythematosus skin at different stages. J Dermatol Sci. 2017;85:247-9.
4. Wenzel J, Landmann A, Vorwerk G, Kuhn A. High expression of B lymphocyte stimulator in lesional keratinocytes of patients with cutaneous lupus erythematosus. Exp Dermatol. 2018;27:95-7.
5. Tsokos G, Lo MS, Reis PC, Sullivan KE. New insights into the immunopathogenesis of systemic lupus erythematosus. Nat Rev Rheumatol. 2016;12:716-30.
6. Jenks SA, Wei C, Bugrovsky R, Hill A, Wang X, Rossi FM, et al. B cell subset composition segments clinically and serologically distinct groups in chronic cutaneous lupus erythematosus. Ann Rheum Dis. 2021;80:1190-200.
7. Manzi S, Sánchez-Guerrero J, Merrill JT, Furie R, Gladman D, Navarra SV, et al. Effects of belimumab, a B lymphocyte stimulator-specific inhibitor, on disease activity across multiple organ domains in patients with systemic lupus erythematosus: combined results from two phase III trials. Ann Rheum Dis. 2012;71:1833-8.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Letter

Pemphigus presenting with prominent neutrophilic pustules mimicking two diseases

Pallavi Hegde[1], Meghana Ravikumar[1], Raghavendra Rao[2]

[1]Department of Dermatology Venereology and Leprosy, Karnataka Medical College and Research Institute, Hubli, Karnataka, India, [2]Department of Dermatology, Venereology and Leprosy, Kasturba Medical College, Manipal, Karnataka, India

Corresponding author: Pallavi Hegde, MD, E-mail: meghanaravigh@gmail.com

Sir,

Pemphigus is a group of immunobullous disorders with intraepidermal blisters that may mimic some other conditions clinically and histologically. Hereby, we report a case of pemphigus masquerading as subcorneal pustular dermatoses (SCPD) and IgA pemphigus confirmed by immunofluorescence.

A 68-year-old female presented with a sudden onset of itchy, pus-filled lesions and redness over the face, neck, both upper limbs, lower limbs, and trunk for three days. On examination, multiple discrete to grouped pustules, several erosions, and crusting on a background of diffuse erythema over the face, neck, bilateral upper limbs, lower limbs, and trunk were noted. (Figs. 1a and 1b). The mucosa and scalp were unaffected. No history of drug intake prior to the onset of the lesion was present. The patient gave a history of itchy, fluid-filled lesions affecting similar areas four years previously. Histopathological (HPE) examination of the vesicle showed features of bullous pemphigoid, and the patient was initiated on dapsone, later switched to dexamethsone cyclophosphamide pulse (DCP) therapy i/v/o dapsone hypersensitivity reaction. She was lost to follow-up after eight cycles but continued taking oral cyclophosphamide 50 mg per day on her own for 2.5 years; meanwhile, the patient had two episodes of exacerbation and was treated with steroids.

Based on the current clinical presentation, the possibility of SCPD and IgA pemphigus was considered and investigated accordingly. Tzanck smear showed only neutrophils. HPE of the pustule showed a subcorneal split, neutrophils, and some acantholytic cells (Fig. 2a). Direct immunofluorescence (DIF) examination of perilesional skin showed intercellular staining of epidermis with IgG and C3 (Fig. 2b).

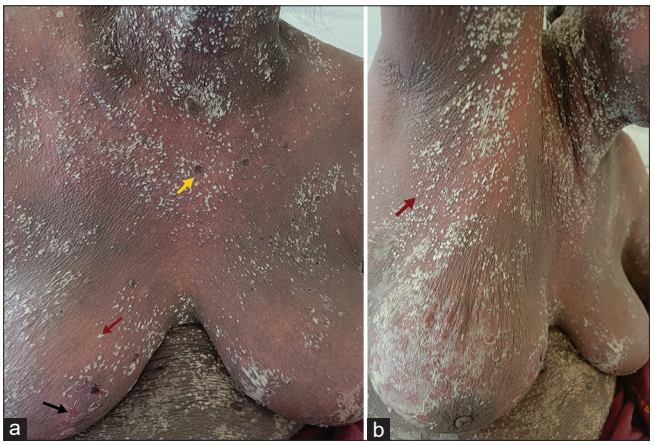


Figure 1: (a and b) Multiple discrete to grouped pustules on a background of erythema (red arrow) present on the face, neck, bilateral upper limbs, lower limbs, and trunk. Some erythematous erosions (black arrow) and yellowish to brown crusts (yellow arrow) present on the trunk.

A typical case of pemphigus presents with widespread flaccid blisters, painful erosions, and prominent mucosal involvement. However, atypical presentations are not uncommon. A similar case of a young male patient, showing features of SCPD and IgA pemphigus clinically, was reported, showing DIF features of pemphigus. The patient showed a dramatic improvement with oral corticosteroids [1]. Similarly, a case of an elderly female who presented with features of sweet syndrome initially later developed a crusting similar to pemphigus foliaceus (PF). This

How to cite this article: Hegde P, Raviumar M, Rao R. Pemphigus presenting with prominent neutrophilic pustules mimicking two diseases. Our Dermatol Online. 2026;17(2):270-271.

Submission: 08.08.2025; **Acceptance:** 09.10.2025

DOI: 10.7241/ourd.20262.28

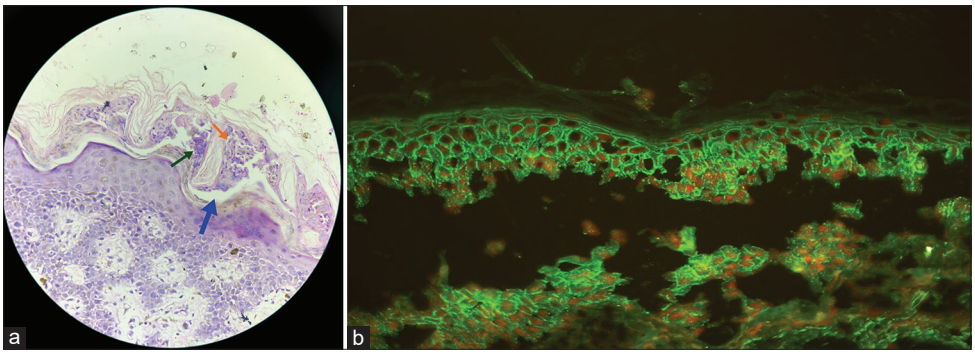


Figure 2: (a) Epidermis showing a subcorneal split (blue arrow) containing neutrophils (green arrow) and some acantholytic cells in the blister cavity (orange arrow) (H&E; 40x). (b) Intercellular staining of the epidermis with IgG and C3 with a fish net pattern (DIF; 10x).

was confirmed on DIF, and the patient responded well to DCP therapy [2]. Another case, of a young boy presenting with typical features of PF with prominent pustules progressing to erythroderma, is mentioned in the literature. The patient showed a dramatic improvement with colchicine after failing to respond to conventional treatment [3]. A middle-aged man with pre-existing psoriasis vulgaris lasting for 35 years presented with a new onset of erythematous, scaly lesions with prominent pustules. Interestingly, the patient did not respond to conventional psoriasis therapy but showed features of PF on DIF [4]. A case of PF in a young female presenting with predominant neutrophilic pustules confirmed on DIF was reported [5]. In the majority of such cases of an atypical presentation of pemphigus, DIF is of great importance, as in our case, to identify the correct diagnosis, as pemphigus requires to be treated with specific immunosuppressive therapy.

REFERENCES

1. Kabra V, Pai K, Pai B S, Shenoi S, Rao R. Pemphigus vulgaris masquerading as subcorneal pustular dermatoses: A case report. Our Dermatol Online. 2014;5:157-9.
2. Kandathil TS, Maneek KF, Samson JF, Yesudasan RD. From sweet syndrome to pemphigus foliaceus: An unusual transition. J Skin Sex Trans Dise. 2025:1-3.
3. Mathachan SR, Arora P, Sardana K, Paliwal P. Pemphigus foliaceous with prominent neutrophilic pustules: A rare variant responsive to colchicine. Indian J Dermatol. 2022;67:481.
4. Sousa VB, Santana CN, Pereira DD, Gripp AC. Pemphigus foliaceus with pustular presentation in a patient with psoriasis. An Bras Dermatol. 2017;92:115-7.
5. Mendez-Flores S, Dominguez-Cherit J, Gracia-Hidalgo L, Saeb Lima M. A rare presentation of pemphigus foliaceus with neutrophilic pustules. J Am Acad Dermatol. 2012;66(4,Suppl 1):AB102.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Case Letter

Erythema ab igne at an unusual location. Triggering factors revisited

Eleni Klimi

Department Dermatology Thriassio General Hospital Magula Athens, Greece

Corresponding author: Eleni Klimi, MD, PhD, E-mail: eklimi2018@gmail.com

Sir,

A 21-year-old woman with an unremarkable medical history, 1.72 m, 87 kg, consulted for lesions that appeared a week prior to consultation. Clinical examination revealed symmetrical, reticulated, pigmented rash below both breasts (Figs. 1a and 1b). The clinical diagnosis was compatible with erythema ab igne. No additional laboratory examinations were conducted as the etiology of the rash was evident, and the clinical condition of the patient was excellent. The patient, while the external temperature exceeded 40°C, had been working on two computers simultaneously, therefore, excessive heat is considered to be linked with the development of erythema ab igne.

This dermatological condition manifests with the appearance of reticulated pigmented rash in parts of the body following exposure to excessive heat [1,2]. Predilection sites include the back, where heaters are usually applied, the abdomen, the inner surface of thighs, and the upper arms and lower legs. Breasts are an unusual site. Erythema ab igne has been reported in patients using laptops in contact with the affected areas [3]. Although affection is benign, associations with autoimmunity as well as malignant transformation to squamous cell carcinoma have been described in the literature [4,5].

While not mentioned, a thorough review of the literature revealed that most patients presenting with erythema ab igne are females. This might be explained by hormonal changes that provoke alterations in thermoregulation in females. Obesity observed in the reported patient, 1.72 m, 87 kg, might have contributed to the development of erythema ab igne, as excess fat leads to diminished heat elimination. Another publication reported three cases of erythema igne occurring in three girls with anorexia nervosa [6]. It is known that anorexia nervosa is associated with weight loss, intolerance to cold vasoconstriction, and increased need for heat. The assumption that both an excess in body weight or emaciation may be associated with the development of erythema ab igne should be taken into account. This case is reported because of the unusual location of erythema ab igne (no case on and below the breasts was found in the literature) and to increase awareness of physicians about the possible associations of the affection with either excessive or diminished body weight, especially in female patients.

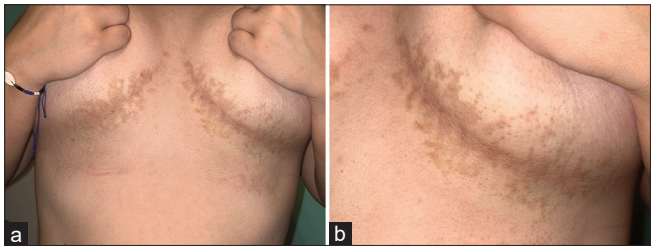


Figure 1: a) Erythema ab igne on and below both breasts. b) Erythema ab igne on and below the left breast.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

How to cite this article: Klimi E. Erythema ab igne at an unusual location. Triggering factors revisited. Our Dermatol Online. 2026;17(2):272-273.

Submission: 27.07.2025; **Acceptance:** 11.09.2025

DOI: 10.7241/ourd.20262.29

REFERENCES

1. Kettelhut EA, Traylor J, Sathe NC, Roach JP. Erythema ab igne. 2022 Dec 24. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–.
2. Phadnis P, Rathoriya SG, Singhal R, Choudhary V. Descriptive assay of the clinico-morphological characteristics of dermatoses presenting with reticulate pigmentation. Our Dermatol Online. 2023;14:372-9.
3. Bimbi C, Brzezinski P. Laptop-induced erythema ab igne: A case report. Our Dermatol Online. 2014;5:430-1.
4. van Rensburg R, Reuter H. Abdominal erythema ab igne: Beyond the rash. Clin Rheumatol. 2024;43:2351-2.
5. Wipf AJ, Brown MR. Malignant transformation of erythema ab igne. JAAD Case Rep. 2022;26:85-7.
6. Dessinioti C, Katsambas A, Tzavela E, Karountzos V, Tsitsika AK. Erythema ab igne in three girls with anorexia nervosa. Pediatr Dermatol. 2016;33:e149-50.

Source of Support: Nil, **Conflict of Interest:** None declared.

Our Dermatology Online

Case Letter

Inflammatory tinea corporis

Patricia Chang[1], Engracia Estefania Quijada Ucelo[2], Roberto Orozco[3]

[1]Dermatologist at Paseo Plaza Clinic Center, Guatemala City, Guatemala, [2]General Practitioner, DermaQ Specialized Dermatology Center, Sixtino II, Guatemala City, Guatemala, [3]Pathologist at Private Practice, Guatemala City, Guatemala

Corresponding author: Patricia Chang, MD, E-mail: pchang2622@gmail.com

Sir,

Tinea corporis is a dermatophytosis that typically presents as annular, scaly plaques with central clearing and an active, erythematous, raised border. However, when modified by immunosuppressive topical agents such as corticosteroids and calcineurin inhibitors, its appearance may change substantially, leading to diagnostic confusion [1].

A 67-year-old female patient presented with a six-month history of a pruritic lesion located on the extensor surface of her left elbow. The lesion had initially been diagnosed as psoriasis and treated with topical clobetasol dipropionate and tacrolimus. However, the patient showed no clinical improvement. The lesion gradually enlarged and became pruriginous. She had no relevant personal or family history. On physical examination, a well-demarcated erythematous-squamous plaque with reddish nodules on the left elbow was observed (Figs. 1a – 1b). No other cutaneous or systemic findings were seen.

A skin biopsy was taken, considering granuloma annulare vs. inflammatory tinea corporis as the differential diagnoses. Histopathology revealed fungal hyphae and spores between the stratum corneum and stratum granulosum, as highlighted by Periodic Acid–Schiff (PAS) staining (Figs. 2a – 2c). These findings were consistent with inflammatory tinea corporis, hence oral treatment was initiated with terbinafine 250 mg daily for four weeks, leading to complete clinical resolution (Figs. 3a and 3b).

This case highlights how topical immunosuppressants can alter the clinical appearance of dermatophytosis. The absence of the typical annular border and the presence of nodular lesions and a chronic plaque that showed no response to corticosteroids or tacrolimus raised uncertainty about the diagnosis. Inflammatory tinea may mimic other inflammatory dermatoses, such as psoriasis, eczema, granuloma annulare, or even cutaneous neoplasms [1].

A review published by Belmokhtar et al. described a variety of atypical superficial mycoses enhanced or modified by prior use of corticosteroids or immunosuppressants, noticing the appearance of infiltrated, nodular, or pseudo-tumoral lesions in patients, often mistaken for inflammatory dermatoses [1]. Our patient aligned closely with these clinical features, particularly the persistence of a non-annular, pruritic plaque with nodular components, despite immunomodulators.

A study published by Zacharopoulou et al. described an increase in frequency of non-classical tinea presentations, particularly in patients exposed to inappropriate therapies [2]. They recommended an early histopathology examination and the use of fungal stains in steroid-resistant dermatoses—a recommendation that was fundamental in reaching the correct diagnosis in this case.

Although the clinical suspicion was initially directed to granuloma annulare due to the nodular aspect or deep fungal infection, histological analysis showed fungal structures between the stratum corneum and granulosum, excluding deeper dermatophytosis. This confirmed inflammatory tinea corporis. In reports by Saito et al. and Mehta et al., PAS stain was crucial to identify fungal structures in lesions with atypical morphology or those altered by immunosuppressive therapy [3,4].

Our findings are similar to other authors' reports of atypical forms of dermatophytosis. A case of atypically inflammatory tinea caused by *Microsporum gypseum*

How to cite this article: Chang P, Quijada Ucelo EE, Orozco R. Inflammatory tinea corporis. Our Dermatol Online. 2026;17(2):274-276.

Submission: 03.08.2025; **Acceptance:** 02.10.2025

DOI: 10.7241/ourd.20262.30

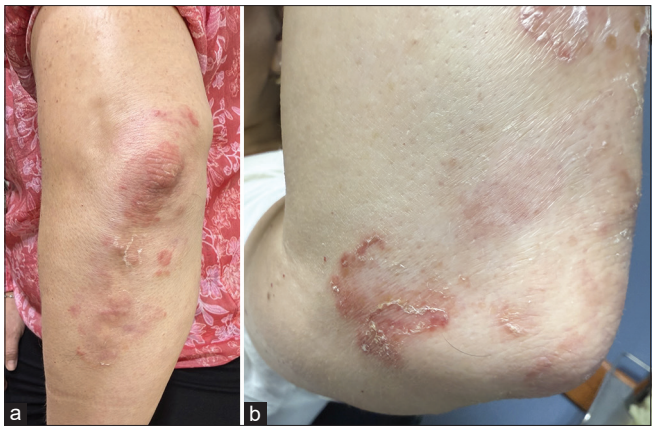


Figure 1: (a and b) Erythematous-squamous plaque with nodular lesions on the left elbow.

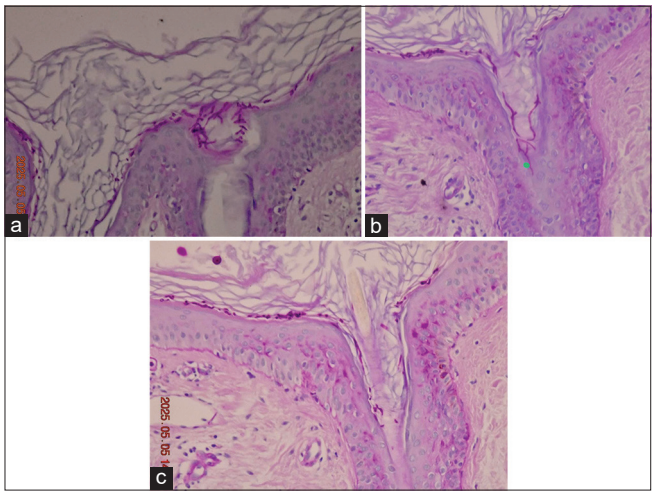


Figure 2: (a and b) Skin biopsy: Histopathological examination with PAS stain showing fungal hyphae and spores between the stratum corneum and stratum granulosum. (c) Histopathological examination with PAS stain showing fungal hyphae and spores around the entrance of the hairy infundibulum.

was reported by Torres-Guerrero et al., underscoring the necessity of mycological confirmation for effective treatment and the importance of considering geophilic agents as causative microorganisms of uncommon presentations of tinea corporis [5]. In order to broaden the range of atypical presentations, Hashas et al. described the dermatophytid phenomenon, in which hypersensitivity reactions occur at distant skin sites from the primary lesion, without active fungal elements [6]. Additionally, Karimi et al. reported tinea in a psoriatic patient, initially mistaken as a psoriasis flare, demonstrating how underlying chronic dermatoses and topical corticosteroid use may mask fungal infections and delay diagnosis [7].

Although the clinical features of the lesion partially resembled *tinea incognita*, histology confirmed the

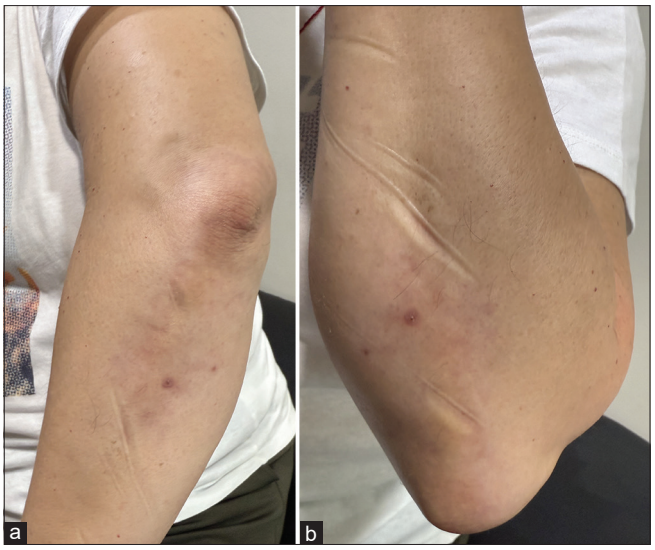


Figure 3: (a and b) Complete clinical resolution after four weeks of oral terbinafine treatment.

diagnosis of inflammatory tinea corporis limited to the epidermis. This case underscored the importance of considering dermatophytosis in chronic, treatment-resistant plaques and supported the approach of taking early biopsies and the use of fungal stains in unclear cases to ensure diagnostic accuracy and guide effective therapy.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Belmokhtar Z, Djaroud S, Matmour D, Merad Y. Atypical and unpredictable superficial mycosis presentations: A narrative review. J Fungi. 2024;10:295.
2. Zacharopoulou A, Tsiogka A, Tsimpidakis A, Lamia A, Koumaki D, Gregoriou S. Tinea incognito: Challenges in diagnosis and management. J of Clin Med. 2024;13:3267.
3. Saito R, Sawada M, Ishizaki S, Harada T. A case of inflammatory tinea corporis by Epidermophyton floccosum. Nihon Ishinkin Gakkai Zasshi. 2008;49:211-5.
4. Mehta N, Choudhary R, Bhari N, Baskaran N. Tinea corporis masquerading as inflammatory papulo-squamous disease. Indian Dermatol Online J. 2023;14:719-20.
5. Torres – Guerrero E, Espinoza – Hernández CJ, Arroyo – Camarena S, Atoche – Diéguez CE. Tinea caused by Microsporum gypseum. Our Dermatol Online. 2018;9:380-5.
6. Hashas FZ, Douhi Z, Mejjati K, Soughi M, Elloudi S, Baybay H,

et al. Dermatophytid in tinea capitis: A phenomenon to keep in mind. Our Dermatol Online. 2023;14:333-4.
7. Karimi S, Aboudourib M, Hocar O, Amal S. Adult tinea in a psoriatic patient. Our Dermatol Online. 2024;15:100-1.

Source of Support: This article has no funding source.
Conflict of Interest: The authors have no conflict of interest to declare.

Case Letter

A case of bullous lichen planus preceding Hodgkin's lymphoma

Maki Takada, Aki Honda, Toshiyuki Yamamoto

Department of Dermatology, Fukushima Medical University, Fukushima, Japan.

Corresponding author: Maki Takada, MD, E-mail: takadamk@fmu.ac.jp

Sir,

A 70-year-old male was referred to our department due to a progressive rash that first appeared seven months previously. He had a medical history of hypertension and hyperlipidemia, but without hepatitis B or C. A physical examination revealed scaly erythema on the upper extremities, erythema and blisters on the dorsa of the hands (Fig. 1), and hemorrhagic blisters on the lower extremities. A biopsy specimen taken from the erythema on the elbow showed irregular extensions of epidermal protrusions, lymphocytic infiltration under the epidermis, liquefaction degeneration of the basal layer, and individual cell keratinization in the epidermis (Fig. 2a). Another biopsy specimen taken from a blister on the dorsum of the hand revealed lymphocytic infiltration under the epidermis, forming subepidermal blisters (Fig. 2b). No deposition of immunoglobulins was observed in the basement membrane zone by direct immunofluorescence. Amlodipine and atorvastatin, which the patient had been receiving for hypertension and hypercholesterolemia, were discontinued, and dental metal removal was performed; however, no improvement was observed. Both topical corticosteroids and phototherapy were ineffective, and treatment with etretinate was discontinued due to liver dysfunction after only two weeks of intake. Two years after the diagnosis of bullous lichen planus (LP), he complained of general fatigue, and blood tests showed elevated C-reactive protein levels. Whole-body CT revealed multiple lymphadenopathies. A lymph node biopsy from the neck led to a diagnosis of Hodgkin's lymphoma (Fig. 3). The patient was admitted to another hospital for further treatment.

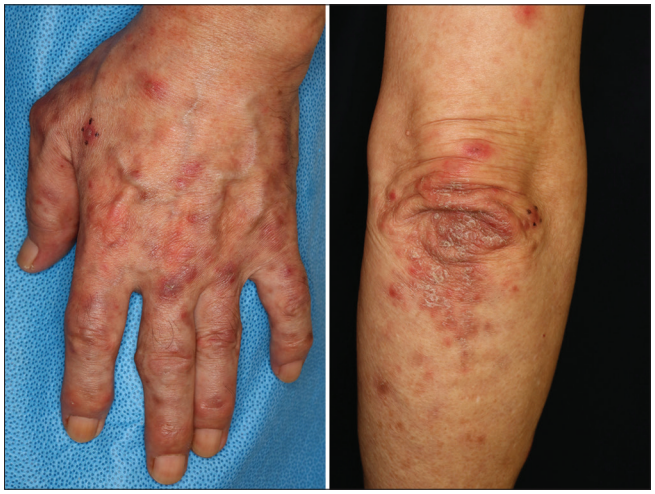


Figure 1: Clinical features of the elbow and the dorsum of the left hand.

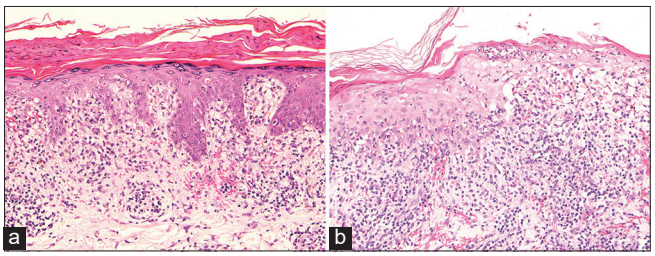


Figure 2: (a) Histopathology of the erythema on the elbow showing subepidermal lymphocytic infiltration, a poorly defined epidermal basal layer, and necrosis of epidermal cells (H&E, 200×). (b) Histopathology of a blister on the dorsum of the hand showing subepidermal lymphocytic infiltration and blister formation (H&E, 200×).

Bullous LP generally tends to appear on the oral mucosa and lower extremities, with blisters appearing near or over existing LP lesions [1]. In the present case, blisters were seen mainly on the lower extremities. In addition, the biopsy revealed subepidermal blisters, and direct immunofluorescence revealed no deposition in the basement membrane zone. The patient was diagnosed with Hodgkin's lymphoma about two years after the diagnosis of bullous

How to cite this article: Takada M, Honda A, Yamamoto T. A case of bullous lichen planus preceding Hodgkin's lymphoma. Our Dermatol Online. 2026;17(2):277-278.

Submission: 14.08.2025; **Acceptance:** 13.11.2025

DOI: 10.7241/ourd.20262.31

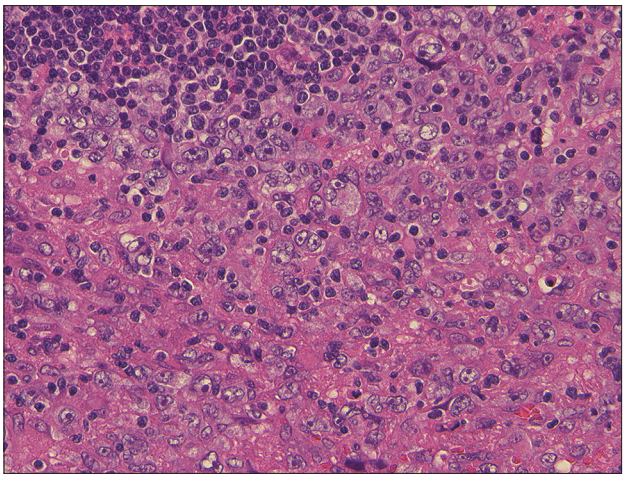


Figure 3: Atypical lymphocytes increased and Reed–Sternberg cells observed (H&E, 400×).

LP. Concurrent occurrence of LP and malignant lymphoma has been reported in only three cases in the English literature, including the present case (Table 1) [2,3]. The patients were two males and two females, and the malignant lymphomas included non-Hodgkin's lymphomas (n = 2) and Hodgkin's lymphoma (n = 1) (one was unknown). Except for the present case, malignant lymphoma preceded the occurrence of LP, and the time from the diagnosis of malignant lymphoma to LP onset ranged from 1 month to 6 years. By contrast, in the present case, LP preceded the development of the lymphoma by two years.

Various co-morbidities have been reported for LP, including hepatitis C virus infection, autoimmune diseases, internal malignancies, dyslipidemia, and viral infections [4]. Among over 13,000 reported female LP patients in Finland, 31 patients died of malignant lymphoma, among which 27 were non-Hodgkin's lymphomas and 4 Hodgkin's lymphomas [5], both showing increased standardized mortality ratios compared to the general population. Although the co-existence may be fortuitous in the present case, further accumulation of similar cases is necessary to determine the relationship between bullous LP and a hematologic malignancy.

Table 1: Report of malignant lymphoma combined with LP.

Case	Age	Sex	Types of Lymphoma	Treatment of LP	Time from Diagnosis of ML to Diagnosis of LP
1	47	F	Unknown	PSL 60 mg	6 years
2	63	F	Non-Hodgkin's lymphoma	Etretinate (1 mg/kg/day), CyA mouthwash	1 month
3	58	M	Non-Hodgkin's lymphoma	PSL 150 mg, AZP 50–100 mg, CyA 40 mg	(unknown)
Our case	70	M	Hodgkin's lymphoma	Topical steroid, oral etretinate, phototherapy	-

Abbreviations: Cya, cyclosporine; AZA, azathioprine; ML, malignant lymphoma

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Liakopoulou A, Rallis E. Bullous lichen planus: A review. J Dermatol Case Rep. 2017;11:1-4.
2. Feuerman EJ, Sandbank M. Lichen planus pemphigoides with extensive melanosis: Occurrence in a patient with malignant lymphoma. Arch Dermatol. 1971;104:61-7.
3. Helm TN, Camisa C, Liu AY, Valenzuela R, Bergfeld WF. Lichen planus associated with neoplasia: A cell-mediated immune response to tumor antigens? J Am Acad Dermatol. 1994;30:219-24.
4. Gorouhi F, Davari P, Fazel N. Cutaneous and mucosal lichen planus: A comprehensive review of clinical subtypes, risk factors, diagnosis, and prognosis. Sci World J. 2014;2014:742826.
5. Halonen P, Jakobsson M, Heikinheimo O, Gissler M, Pukkala E. Incidence of lichen planus and subsequent mortality in Finnish women. Acta Derm Venereol. 2020;100:adv00303.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Our Dermatology Online

Case Letter

Dermoscopy of Schamberg's disease: Identification of a novel fried-egg appearance

Pihu Sethi[1], Kriti Maheshwari[2]

[1]Government Institute of Medical Sciences, Greater Noida, Uttar Pradesh, India, [2]Yashoda Medicity, Ghaziabad, Uttar Pradesh, India

Corresponding author: Kriti Maheshwari, MD, E-mail: Maheshwari.kriti@gmail.com

Sir,

Pigmented purpuric dermatoses (PPD) represent a group of chronic cutaneous disorders that present with petechial hemorrhage occurring due to capillaritis. Extravasated erythrocytes result in purpura, and hemosiderin-laden macrophages give a red-brown appearance to older lesions. It is characterized by symmetrical petechial and pigmented macules, often confined to the lower limbs [1]. Although adults are more frequently affected, there are numerous reports of childhood PPD as well. Clinically, PPDs are categorized into five primary types: Schamberg disease (which is the commonest variant), pigmented purpuric lichenoid dermatosis of Gougerot and Blum, purpura annularis telangiectodes or Majocchi's disease, lichen aureus and eczematid-like purpura of Doucas and Kapetanakis [2].

A 45-year-old female presented to the dermatology outpatient department with complaints of a reddish-brown rash on both lower limbs lasting for the previous one year. The rash was slowly progressive and initially asymptomatic but, one month previously, developed mild itching. There were no other significant findings in her family or personal history. Cutaneous examination revealed multiple reddish to brown macules along with several lesions showing central stippled puncta on the anteromedial aspect of both legs (Fig. 1). Dermoscopy revealed a reddish-brown background, brown globules with central, red dots giving a fried-egg appearance and scattered, coiled or punctuate vessels. Dermoscopic examination of two more patients with Schamberg's disease was performed and showed a similar fried-egg appearance (Figs. 2a and 2b).

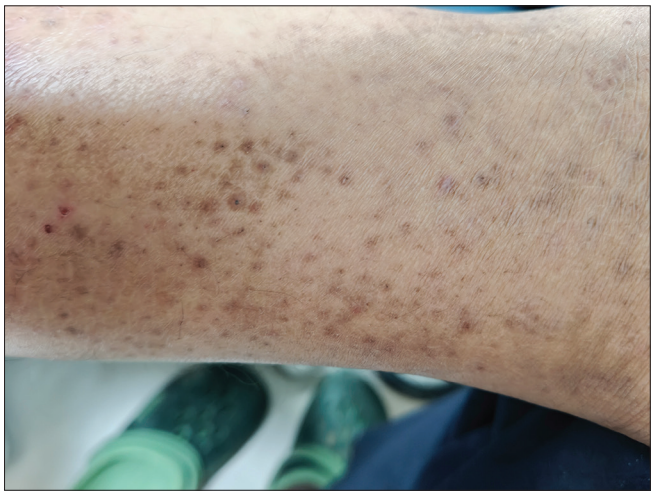


Figure 1: Discrete to confluent reddish-brown "Cayenne pepper"-like patches on the left leg.

The differential diagnosis of Schamberg's disease includes leukocytoclastic vasculitis, contact dermatitis, early cutaneous T-cell lymphoma, hypergammaglobulinemic purpura of Waldenström, bleeding diathesis, stasis pigmentation, scurvy, and drug-hypersensitivity reactions [1]. A summary of clinical and dermoscopic features of differential diagnosis of Schamberg's disease can be seen in Table 1 [2-7].

Dermoscopy epiluminescence microscopy is a great non-invasive tool for evaluating pigmentary lesions as it often reduces the need for invasive tests such as a biopsy for diagnosis. The commonly noted dermoscopic findings of PPD include a coppery-red background, red dots, globules and patches, brown dots and globules, red dots, globules, and patches. Less common findings include a brown network, thick

How to cite this article: Sethi P, Maheshwari K. Dermoscopy of Schamberg's disease: Identification of a novel fried-egg appearance. Our Dermatol Online. 2026;17(2):279-280.

Submission: 13.08.2025; **Acceptance:** 11.10.2025

DOI: 10.7241/ourd.20262.32

Table 1: A summary of clinical and dermoscopic features of the differential diagnosis of Schamberg's disease [2-7].

Diagnosis	Clinical Feature	Dermoscopy
Pigmented purpuric dermatoses (Schamberg's disease)	"Cayenne-pepper" petechiae on an orange-brown background, symmetric on legs, mild pruritus	Coppery-red/orange-brown background, red dots/globules, brown dots/globules, reticular network, linear vessels, linear brown lines.
Leukocytoclastic vasculitis	Palpable purpura, often lower extremities, may have systemic symptoms (arthralgia, renal involvement)	Milky-red or livedoid background, red blotches; less commonly, red dots or comma-vessels
Contact dermatitis	Burning/itching associated with an eczematous/purpuric rash localized to contact areas, history of exposure usually present	No specific dermoscopic reports; dotted vessels distributed in clusters or randomly, yellow scales and serous crusts.
Bleeding diathesis	Diffuse petechiae/purpura, mucosal bleeding; lab-confirmed low platelets/abnormal clotting	Dermoscopy reveals homogeneous red/purple splotches and globules on a purple background without vessel morphology
Stasis Pigmentation	Brownish pigmentation, often with varicosities, edema, lipodermatosclerosis	Hemosiderin pigment, dull red to brownish background, glomerular and dotted vessels, scaling
Scurvy	Corkscrew hairs, perifollicular hemorrhages, gum bleeding, easy bruisability	Corkscrew hair; perifollicular erythematous/violaceous macules
Drug hypersensitivity reactions	Purpuric/exanthematous rash, pruritus, systemic symptoms depending on drug and reaction type.	May vary with inflammatory pattern

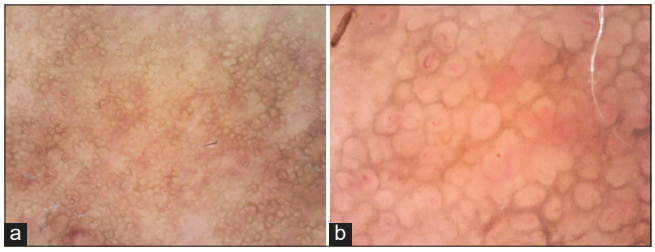


Figure 2: a) Dermoscopy showing a reddish-brown background, brown globules with central red dots, and coiled vessels (Dinolite Edge, 60x). b) Dermoscopy showing a reddish-brown background, brown globules with central red coiled vessels showing the fried-egg appearance, red and brown dots. (Dinolite Edge, 200x).

linear vessels, rosette structures, linear brown lines, and follicular openings [2,7]. The term *fried-egg appearance* in the context of Schamberg's disease refers to the typical dermatoscopic pattern observed where red or reddish-brown vascular coiling is noted at the center of darker brown globules like the bright yellow yolk surrounded by the white of a fried egg. By reporting this novel finding, the authors wish to highlight a new observation of the fried-egg appearance, which has not been previously described to the best of the author's knowledge and was seen typically in the patients with the Schamberg's disease subtype of pigmented purpuric dermatoses.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Spigariolo CB, Giacalone S, Nazzaro G. Pigmented purpuric dermatoses: A complete narrative review. J Clin Med. 2021;10:2283.
2. Ozkaya DB, Emiroglu N, Su O, Cengiz FP, Bahali AG, Yildiz P, Demirkesen C, Onsun N. Dermatoscopic findings of pigmented purpuric dermatosis. An Bras Dermatol. 2016;91:584-7.
3. Gaurav V, Gowda P, Bahadur A, Tyagi H, Dev T, Barman KD. Dermoscopy-histopathology correlation of cutaneous small-vessel vasculitis. Cosmoderma. 2024;4:90.
4. Sinha P, Ayub A, Krishnan L, Dash M. Mucocutaneous manifestations in a case of immune thrombocytopenia: Reporting a rare presentation with dermoscopic findings. Clin Dermatol Rev. 2024;8:375-7.
5. Zaballos P, Salsench E, Puig S, Malvehy J. Dermoscopy of venous stasis dermatitis. Arch Dermatol. 2006;142:1526.
6. Wambier CG, Cappel MA, Werner B, Rodrigues E, Welling MS, Netto MR, et al. Dermoscopic diagnosis of scurvy. J Am Acad Dermatol. 2017;76:S52-4.
7. Metin M, Elmas Ö. Dermoscopic profile of pigmented purpuric dermatosis: New observations. Postepy Dermatol Alergol. 2019;36:687-91.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.

Case Letter

Rare metastatic location of a primary cutaneous melanoma

Kacimi Alaoui Imane, Sara Elloudi, Sara El-Ammari, Zakia Douhi, Meryem Soughi, Hanane Baybay, Fatima-Zahra Mernissi

Department of Dermatology, University Hospital Hassan II, Fes, Morocco

Corresponding author: Kacimi Alaoui Imane, MD, E-mail: kacimiimane92@gmail.com

Sir,

Pancreatic metastases from primary cutaneous melanoma are rare, in the order of 2–5%, exceptionally diagnosed when the patient is alive. Herein, we report two new observations.

A 66-year-old patient, followed for a right plantar acrolentiginous melanoma at the metastatic stage at the lymph node and pulmonary level, was readmitted in a picture of cholestatic jaundice and clinical aggravation (Figs. 1a and 1b). A cerebro-cervical-thoraco-abdomino-pelvic CT scan revealed a tissue mass of the head of the pancreas measuring 65 x 50 mm (Fig. 2). The diagnosis of a secondary location was confirmed by the radiologists. Given his multivisceral involvement, the patient was referred to oncology for further management.

A 57-year-old patient, followed for a right plantar acrolentiginous melanoma at the metastatic stage (Fig. 3), having benefited from a lymph node curage with chemotherapy, was readmitted for clinical worsening and installation of new skin metastases. A cerebro-cervical-thoraco-abdominopelvic CT scan revealed a tumor of the head of the pancreas measuring 48 mm, associated with other secondary pulmonary and adrenal localizations. The patient was lost to follow-up.

Melanoma usually metastasizes to the lymph nodes, liver, lungs, or brain. Pancreatic metastases are rare, occurring between 2% and 5%, with 76 cases having been described in the literature [1]. Most come from the kidney, lung, or breast [2]. They present with cholestatic jaundice, abdominal pain, and weight loss. Endoscopy is indicated for gastrointestinal symptoms.

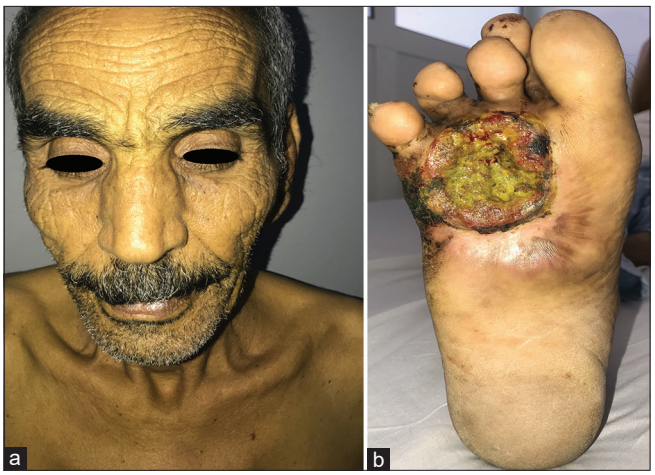


Figure 1: a) Frank cholestatic icterus. b) Plantar ulcerative tumor.

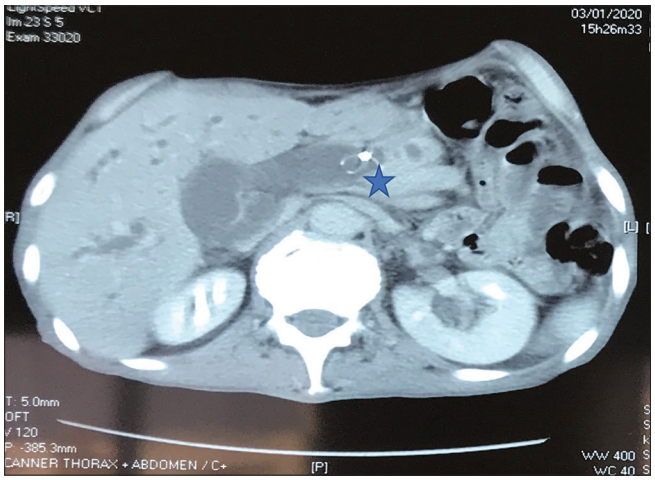


Figure 2: Abdominal CT scan revealing a tissue mass of the pancreatic head.

How to cite this article: Imane KA, Elloudi S, El-Ammari S, Douhi Z, SoughiM, Baybay H, Mernissi F-Z. Rare metastatic location of a primary cutaneous melanoma. Our Dermatol Online. 2026;17(2):281-282.

Submission: 02.03.2023; **Acceptance:** 10.10.2023

DOI: 10.7241/ourd.20262.33

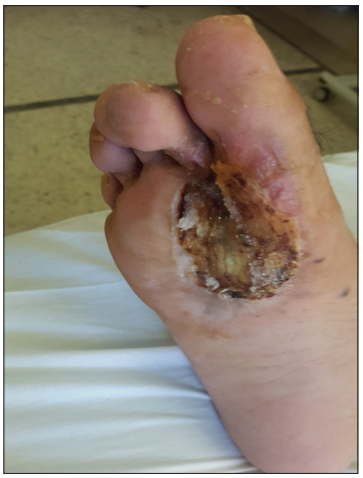


Figure 3: Plantar ulcerative tumor.

Eighty-five percent of pancreatic metastases are incidentally discovered on imaging at an inoperable advanced stage [3]. The choice of treatment should take into account life expectancy, the performance index of the WHO, and the number of metastases. For multivisceral involvement, surgery remains a palliative treatment to relieve symptoms and improve quality of life [3]. Melanoma remains an aggressive malignancy due to its rapid and significant metastatic potential. Although pancreatic metastases are rare, early diagnosis by imaging is crucial.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

REFERENCES

1. Nakamura Y, Yamada R, Kaneko M, Naota H, Fujimura Y, Tabata M, et al. Isolated pancreatic metastasis from malignant melanoma: A case report and literature review. Clin J Gastroenterol. 2019;12:626-36.
2. Chikhladze S, Lederer AK, Kühlbrey CM, Hipp J, Sick O, Fichtner-Feigl S, et al. Curative-intent pancreas resection for pancreatic metastases: Surgical and oncological results. Clin Exp Metastasis. 2020;37:313-24.
3. Kochhar R, Ali H, Mak S, Manoharan P. Metastatic cutaneous malignant melanoma: Spectrum of imaging findings and the role of multimodality imaging. J Med Imaging Radiat Oncol. 2009;53:467-78.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.



















Our Dermatology Online

www.odermatol.com

2.2026 (01.April.2026)