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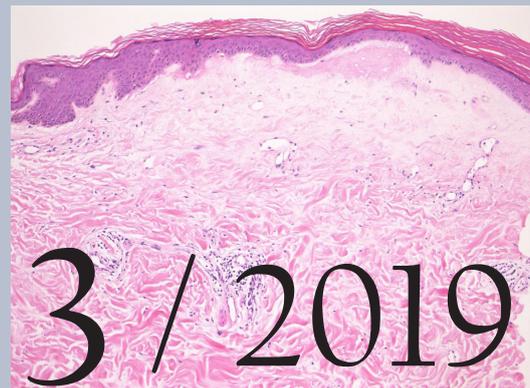
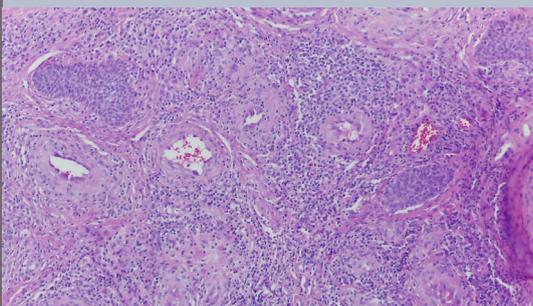
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Editorial Pages

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Decreased serum ferritin and vitamin D levels in patients with recurrent aphthous stomatitis

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ABSTRACT

Background: Recurrent aphthous stomatitis is the most common ulcerative disease of the oral mucosa. Several predisposing factors like oral microbial flora, stress, viral infections, vitamin and mineral deficiencies have been associated with the disease. However, the etiopathogenesis of recurrent aphthous stomatitis remains unknown. **Material and Methods:** The study included 20 patients with recurrent aphthous stomatitis and 20 healthy individuals within the control group. Complete blood count, serum levels of ferritin, folate, vitamin B₁₂, zinc, 25-hydroxyvitamin D, herpes simplex virus type-1 IgG and herpes simplex virus type-2 IgG were evaluated in each participant. **Results:** White blood cell count, platelet count, mean platelet volume, mean serum levels of hemoglobin, folate, vitamin B₁₂ and zinc were statistically similar in patients and healthy individuals. However, the mean serum ferritin level was significantly lower in patients (26.5 ± 25.5 ng/mL) compared to healthy individuals (42 ± 30 ng/mL) ($p=0.04$). The mean serum 25-hydroxyvitamin D level was significantly lower in patients (13.6 ± 6.5 ng/mL) compared to healthy individuals (20.9 ± 10 ng/mL) ($p=0.01$). No significant difference has been observed between two groups in the frequency of positive serum herpes simplex virus type-1 IgG and herpes simplex virus type-2 IgG levels. **Conclusions:** No association has been observed between recurrent aphthous stomatitis and white blood cell count, mean platelet volume, serum levels of hemoglobin, folate, vitamin B₁₂, zinc, herpes simplex virus type-1 IgG and herpes simplex virus type-2 IgG. However, decreased serum levels of ferritin and 25-hydroxyvitamin D were more prevalent in patients with recurrent aphthous stomatitis compared to healthy individuals.

Key words: Aphthous; Ferritin; Recurrent; Stomatitis; Vitamin D

INTRODUCTION

Recurrent aphthous stomatitis is the most common inflammatory disease of the oral mucosa. It is characterized by painful round ulcers with pseudomembranous center and erythematous borders [1,2]. The prevalence of the disease is 0.5% to 75% all over the world. Recurrent aphthous stomatitis is more common in women than in men. The ulcers usually begin during the second decade of life [1]. Recurrent aphthous stomatitis is classified into three clinical types; minor, major, and herpetiform ulcers [2]. Most of the patients (>85%) with recurrent aphthous stomatitis present with minor ulcers that have a diameter less than 1 cm [2]. Major ulcers (Sutton's disease) have a

diameter greater than 1 cm and they can persist for weeks [2,3]. Herpetiform type presents as multiple, small ulcers with 1-2 mm in size, that may heal with scarring [1].

The etiopathogenesis of recurrent aphthous stomatitis remains unknown. However, several predisposing factors have been associated with the disease. Genetic predisposition, iron, vitamin B₁₂ and folic acid deficiencies, nonsteroidal anti-inflammatory drugs, angiotensin converting enzyme inhibitors, gluten sensitive enteropathy, inflammatory bowel disease, stress, trauma to oral mucosa like dental procedures, *Helicobacter pylori* and viral infections have been implicated in the development of recurrent aphthous stomatitis [4].

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The differential diagnosis should include chickenpox, erythema multiforme, erosive lichen planus, Riga-Fede disease, pemphigus vulgaris and pemphigoid [5]. Treatment of recurrent aphthous stomatitis can be difficult particularly in severe cases [6]. It is crucial to rule out an underlying systemic disorder in the management of disease. However, most of the patients are healthy except for painful oral ulcers [7]. Topical therapies include chlorhexidine rinse, antibiotics like doxycycline and minocycline, diclofenac, benzocaine, lidocaine and corticosteroids. Furthermore, penicillin G, rifampin, dapsone, zinc, pentoxifylline, thalidomide, colchicine and ascorbic acid have been used in the management of recurrent aphthous stomatitis [6].

MATERIALS AND METHODS

The study included 20 patients with recurrent aphthous stomatitis and 20 healthy individuals within the control group. Medical records of the participants were reviewed retrospectively between January 2018 and September 2018. Laboratory tests including complete blood count, serum levels of ferritin, folate, vitamin B₁₂, zinc, 25-hydroxyvitamin D (25(OH)D), herpes simplex virus type-1 (HSV1) IgG and herpes simplex virus type-2 (HSV2) IgG were evaluated in each participant. The exclusion criteria were pregnancy, immunosuppression, malignancy, inflammatory bowel disease, diet restrictions, vitamin and mineral supplements.

Data were represented as mean \pm standard deviation or median for quantitative variables; counts and percentage for categorical variables. Differences between two groups were tested with Mann Whitney U test for continuous variables and chi-square or Fisher exact tests as appropriate for categorical variables. The data were analyzed using SPSS 20.0 Statistical Package Program.

RESULTS

The study included 20 patients (15 female, 5 male) with recurrent aphthous stomatitis and 20 healthy individuals (14 female, 6 male) within the control group. The mean age of the patients was 34 ± 12.3 (range: 18-58). The mean age of the healthy individuals was 33.9 ± 13.4 (range: 18-60) ($p=1$). The mean disease duration was 24.6 ± 32.9 months (range: 1-120 months). Three (15%) patients had at least one first-degree family member who suffered from recurrent aphthous stomatitis.

The mean hemoglobin level of the patients and healthy individuals were 13.6 ± 1.5 g/dL and 14.3 ± 1.2 g/dL, respectively ($p=0.1$) (normal range: 11.7-17 g/dL). Two (10%) patients had decreased serum hemoglobin levels, while all the healthy individuals had normal serum hemoglobin levels ($p=0.4$). The mean white blood cell count of the patients and healthy individuals were 7.3 ± 1.6 $10^3/\mu\text{L}$ and 6.7 ± 1.3 $10^3/\mu\text{L}$, respectively ($p=0.3$) (normal range: 4.2-10.2 $10^3/\mu\text{L}$). All the participants had normal serum white blood cell count. The mean platelet count of the patients and healthy individuals were 255.4 ± 68.5 $10^3/\mu\text{L}$ and 252.4 ± 54.5 $10^3/\mu\text{L}$, respectively ($p=0.7$) (normal range: 142-450 $10^3/\mu\text{L}$). One (5%) patient had low platelet count, while all the healthy individuals had normal platelet counts ($p=1$). The mean platelet volume of the patients and healthy individuals were 7.888 ± 1.4 fL and 7.913 ± 0.9 fL, respectively ($p=0.7$) (normal range: 6.4-11 fL). Three (15%) patients and one (5%) healthy individual had low mean platelet volume. Increased mean platelet volume was observed only in one (5%) patient ($p=0.3$).

The mean serum ferritin level of the patients and healthy individuals were 26.5 ± 25.5 ng/mL and 42 ± 30 ng/mL, respectively ($p=0.04$) (normal range: 10-204 ng/mL). Low serum ferritin levels were observed in 6 (30%) patients and 2 (10%) healthy individuals ($p=0.2$) (Fig. 1). The mean serum folate level of the patients and healthy individuals were 5.7 ± 1.8 $\mu\text{g/L}$ and 6.6 ± 2.2 $\mu\text{g/L}$, respectively ($p=0.1$) (normal range: 3.1-20 $\mu\text{g/L}$). All the participants had normal serum folate levels. The mean serum vitamin B₁₂ level of the patients and healthy individuals were 343.7 ± 171.9 pg/mL and 281.6 ± 93.9 pg/mL, respectively ($p=0.2$) (normal range: 190-880 pg/mL). Four (20%) patients had low serum vitamin B₁₂ levels, while 3 (15%) healthy individuals had low serum vitamin B₁₂ levels ($p=1$). The mean serum zinc level of the patients and healthy individuals were 78.8 ± 14.4 $\mu\text{g/dL}$ and 85.3 ± 15.9 $\mu\text{g/L}$, respectively ($p=0.1$) (normal range: 60-150 $\mu\text{g/dL}$). All the participants had normal serum zinc levels. The mean serum 25(OH)D level of the patients and healthy individuals were 13.6 ± 6.5 ng/mL and 20.9 ± 10 ng/mL, respectively ($p=0.01$) (normal range: 20-60 ng/mL). Fourteen (70%) patients had decreased serum 25(OH)D levels, while 9 (45%) healthy individuals had decreased serum 25(OH)D levels ($p=0.2$) (Fig. 2).

The mean serum HSV1 IgG level of the patients and healthy individuals were 52.6 ± 72.4 RU/mL and 80.8 ± 83.9 RU/mL, respectively ($p=0.4$) (range: negative <16, positive >22 RU/mL). Positive serum

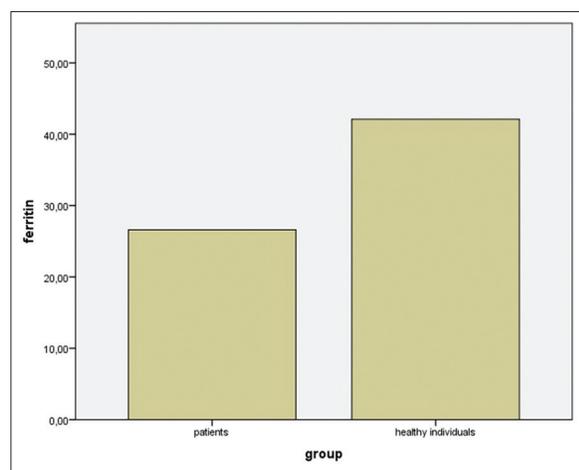


Figure 1: The mean serum ferritin level of the patients (26.5 ± 25.5 ng/mL) was significantly lower compared to healthy individuals (42 ± 30 ng/mL) ($p=0.04$).

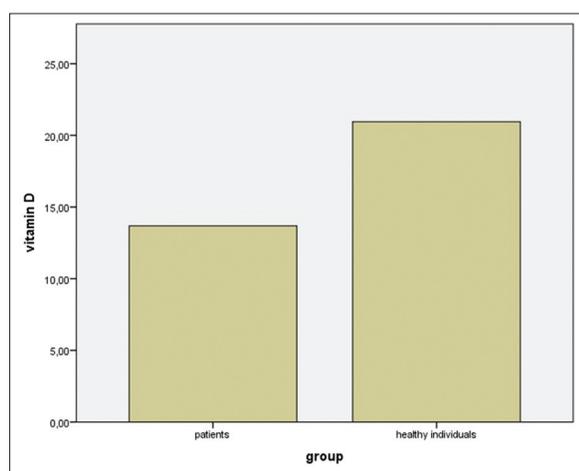


Figure 2: The mean serum 25(OH)D level of the patients (13.6 ± 6.5 ng/mL) was significantly lower compared to healthy individuals (20.9 ± 10 ng/mL) ($p=0.01$).

HSV1 IgG levels were observed in eleven (55%) patients and in 10 (50%) healthy individuals ($p=1$). The mean serum HSV2 IgG level of the patients and healthy individuals were 10.5 ± 34.3 RU/mL and 2.6 ± 2.9 RU/mL, respectively ($p=0.4$) (range: negative <16, positive >22 RU/mL). Positive serum HSV2 IgG level was observed in one (5%) patient. None of the healthy individuals had positive serum HSV2 IgG level ($p=1$).

DISCUSSION

Despite being the most common ulcerative disease of the oral mucosa, the etiology of recurrent aphthous stomatitis has not been clearly identified yet. Therefore, current treatment options are usually aim to reduce symptoms [8]. Impaired mucosal barrier function, stress,

oral microbial flora including *Streptococcus sanguinis* and *Streptococcus mitis*, *Helicobacter pylori* infection, diet, allergies, vitamin and mineral deficiencies, hematological parameters like ferritin, hemoglobin and hematocrit have been associated with recurrent aphthous stomatitis. Moreover, high levels of erythrocyte sedimentation rate, mean platelet volume, white blood cells, neutrophils and homocysteine have been reported in patients with recurrent aphthous stomatitis [8].

Sun et al. investigated hemoglobin, iron, vitamin B₁₂, folic acid, and homocysteine concentrations in patients with recurrent aphthous stomatitis and in healthy controls. Elevated homocysteine levels and decreased levels of hemoglobin, iron, vitamin B₁₂ and folic acid were significantly more common in patients than in control group [9]. Compilato et al. evaluated hematological deficiencies in 32 patients with recurrent aphthous stomatitis and in 29 healthy controls. Serum iron, folic acid and vitamin B₁₂ deficiencies were significantly more common in patient group than in healthy individuals [10]. Chen et al. reported high rates of hemoglobin, ferritin, folic acid and vitamin B₁₂ deficiencies in patients with recurrent aphthous stomatitis [11]. Similarly, Lopez-Jornet et al. reported that iron, folic acid, and vitamin B₁₂ deficiencies were more frequent in patients with recurrent aphthous stomatitis compared to healthy controls [12].

Within this study, the mean serum hemoglobin level was lower in patients (13.6 ± 1.5 g/dL) compared to healthy individuals (14.3 ± 1.2 g/dL). However, no statistically significant difference has been observed in hemoglobin levels between two groups ($p=0.1$). White blood cell count, platelet count and mean platelet volume were similar in patients with recurrent aphthous stomatitis and healthy individuals within the control group. The mean serum ferritin level was significantly lower in patients (26.5 ± 25.5 ng/mL) compared to healthy individuals (42 ± 30 ng/mL) ($p=0.04$). The mean serum folate level was lower in patients (5.7 ± 1.8 μg/L) than in control group (6.6 ± 2.2 μg/L). However, no statistically significant difference has been observed in folate levels between two groups ($p=0.1$). In contrast, mean serum vitamin B₁₂ levels were higher in patients (343.7 ± 171.9 pg/mL) compared to healthy individuals (281.6 ± 93.9 pg/mL). However, no statistically significant difference has been observed in vitamin B₁₂ levels between two groups ($p=0.2$).

Association between serum zinc concentration and recurrent aphthous stomatitis remains controversial.

Ślebioda et al. detected no significant difference in serum zinc levels between patients with recurrent aphthous stomatitis and healthy controls, while Ozler reported a distinct association between zinc deficiency and recurrent aphthous stomatitis [13,14].

Within this study, serum zinc levels of the participants were all in normal limits. Patients with recurrent aphthous stomatitis had lower serum zinc levels ($78.8 \pm 14.4 \mu\text{g/dL}$) compared to healthy individuals ($85.3 \pm 15.9 \mu\text{g/L}$). However, no statistically significant difference has been observed in zinc levels between two groups ($p=0.1$).

Low serum vitamin D level has been implicated as a facilitating factor in the development of recurrent aphthous stomatitis. Oztekin et al. reported significantly lower serum vitamin D levels in patients with recurrent aphthous stomatitis compared to healthy individuals [15]. In contrast, Krawiecka et al. reported similar serum vitamin D levels in patients with recurrent aphthous stomatitis and healthy controls [16].

Within this study, the mean serum 25(OH)D level was significantly lower in patients ($13.6 \pm 6.5 \text{ ng/mL}$) than in control group ($20.9 \pm 10 \text{ ng/mL}$) ($p=0.01$). Vitamin D deficiency was detected in 70% of the patients with recurrent aphthous stomatitis.

It has been suggested that the human herpesviruses (HHVs) may play role in the development of recurrent aphthous stomatitis. However, Brice et al. examined the members of the HHV family including HSV1, HSV2, varicella zoster virus, Epstein-Barr virus, cytomegalovirus, HHV-6, HHV-7 and HHV-8, in the oral mucosa of patients with recurrent aphthous stomatitis and control subjects. The frequency of detection of HHV DNA was not greater in the mucosa of patients than in controls [17]. In addition, Seoudi et al. reported no significant difference between HSV1 IgG and HSV2 IgG levels in patients with Behçet's disease, recurrent aphthous stomatitis and healthy controls [18].

Within this study, a positive HSV1 IgG result was detected in 55% of the patients with recurrent aphthous stomatitis and in 50% of healthy individuals, whereas only one patient was HSV2 IgG positive. The results indicate no correlation between HSV and recurrent aphthous stomatitis.

In conclusion, this study revealed no association between recurrent aphthous stomatitis and mean

platelet volume, white blood cell count, HSV1, HSV2, serum levels of hemoglobin, folate, vitamin B₁₂ and zinc. However, decreased serum levels of ferritin and 25(OH)D were more prevalent in patients with recurrent aphthous stomatitis compared to healthy individuals. Therefore, we suggest that serum ferritin and 25(OH)D levels should be evaluated in all patients with recurrent aphthous stomatitis. Replacement therapies may be helpful in the management of the disease in cases with iron or vitamin D deficiency.

Statement of Human and Animal Rights

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

Statement of Informed Consent

Informed consent was obtained from all patients for being included in the study.

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Clinical and dermoscopic study of melanocytic nevi

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ABSTRACT

Background: A dermoscope is a non-invasive, diagnostic tool which visualizes subtle clinical patterns of skin lesions and subsurface skin structures not normally visible to the unaided eye. Thus, dermoscopy has been introduced as an additional measure to make the diagnosis of melanocytic nevi more accurate. **Objective:** To study clinical and dermoscopic features of Melanocytic nevi. **Materials and Methods:** A total of 50 patients attending Dermatology OPD in a tertiary care centre with melanocytic nevi during the period from March 2018 to August 2018 were consecutively included in the study. A pre-structured proforma was used to collect baseline data. A detailed history was taken, clinical and dermatological examination done. The Melanocytic nevi were evaluated using digital dermoscope. **Results:** Out of 50 cases, age distribution was between 16 years and 54 years. The mean age was 33.9 ± 8.38 yrs. Male to female ratio was 0.78:1. Face was most common site involved. Among local features Pigment network was the most common pattern seen in all lesions. Pigmentation was regular. Dots and Globules were seen in 52% of lesions. There were no streaks, bluish white veil, regression structures or hypopigmentation seen. Among global structures reticular network was seen in 76% of lesions. Globular network was seen in 16% of lesions. Final impression after clinical and dermoscopic examination, incidence of melanocytic nevi was as follows- junctional nevi as most common 48%, followed by compound nevi 36% and congenital melanocytic nevi was 16%. **Conclusion:** Dermoscopy is an evolving science. It serves as a link between macroscopic skin lesions and microscopic histopathological features. Since it is non-invasive, it can be used in all age groups including children and elderly, reducing the need for interventional procedures like skin biopsy. Dermoscopy is a much needed investigative tool in the assessment of Melanocytic nevi.

Key words: Dermoscopy; Melanocytic nevi; Pigment network

INTRODUCTION

A dermoscope (dermatoscope) is a non-invasive, diagnostic tool which visualizes subtle clinical patterns of skin lesions and subsurface skin structures not normally visible to the unaided eye. It has also been called a skin surface microscope, epiluminescence microscope or episcope. Some dermoscopic patterns are observed consistently with certain diseases and these then could be used for their diagnosis. Hence, this office procedure may obviate the need for a skin biopsy for diagnosis and for follow-up. The facility of storage of images and the results being immediately available are added advantages. Basically, a dermoscope is functionally similar to a magnifying lens but with the added features of an inbuilt illuminating system, a higher magnification which can be adjusted, the ability

to assess structures as deep as in the reticular dermis, and the ability to record images [1].

The basic principle of dermoscopy is transillumination of a lesion and studying it with a high magnification to visualize subtle features [2]. Light incident on skin undergoes reflection, refraction, diffraction and absorption. These phenomena are influenced by physical properties of the skin [1].

Dermatoscopes are modified magnifying devices that permit the visualization of pigmented structures or vessels in the epidermis and superficial dermis. Because most dermoscopic structures correspond to specific histo-pathologic correlates, dermoscopy can be regarded as a link between clinical (macroscopic) and histopathologic (microscopic) morphology [3].

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The standard methods to diagnose pigmented skin lesions, such as simple clinical inspection and biopsy, vary in their reproducibility and invasiveness, and there is a need for noninvasive methods that help the clinician in day today practice. The use of dermoscopy improves the diagnostic accuracy and may contribute in discerning the behavior of pigmented skin lesions [4].

The most important parameters that should be assessed when applying dermoscopy in general dermatology include morphological vascular patterns, arrangement of vascular structures, colors, and follicular abnormalities (where relevant)—and the presence of other specific features (clues) [5].

The most studied dermatoscopic method for study of pigmented skin lesion is pattern analysis. Dermoscopic patterns are mostly derived from the presence or union of 3 main dermatoscopic features: pigment network, globules and homogeneous areas. The pigment network is a grid-like network consisting of pigmented lines and hypopigmented “holes” and represents one of the major dermatoscopic features associated with melanocytic lesions [6].

Dermoscopy helps in the diagnosis of many pigmented skin lesions such as seborrheic keratosis (SK), pigmented basal cell carcinoma (BCC), hemangioma, blue nevus, atypical nevus, and cutaneous melanoma. It is 10-27% more sensitive than clinical criteria of ABCD (asymmetry, border regularity, color distribution, and diameter) in the early diagnosis of cutaneous melanoma. Dermoscopy of melanocytic lesions increases the presurgical accuracy rate of clinical diagnosis from 50 to 85% [7].

Undoubtedly, histopathologic examination represents the gold standard of diagnosis in dermatology, and dermoscopy was never suggested as an alternative or competitive method. In contrast, the dermatoscope is a clinical tool that should be considered similar to the stethoscope of general practitioners [8]. Dermoscopy is a valuable tool for evaluating pigmentary lesions, and it greatly enhances the clinical diagnosis of nearly all pigmented skin tumours [9].

The present study has been undertaken to study the clinical and dermoscopic features of melanocytic nevi.

MATERIALS AND METHODS

Objectives

To study clinical and dermoscopic features of Melanocytic nevi.

Methodology

Source of data

A total of 50 patients attending dermatology attending Out Patient Department of Dermatology, Venereology and Leprosy, KVG Medical College and Hospital, Sullia, with melanocytic nevi during the period from March 2018 To August 2018 were consecutively included in the study after taking informed consent.

Method of Collection of Data

Study design

Hospital based cross sectional study.

Sample size

A total of 50 patients with Melanocytic nevi attending the Out Patient Department of Dermatology, Venereology and Leprosy, KVG Medical college and hospital, Sullia will be consecutively selected for the study.

Sample size was calculated based on a study by Senthikumar et al [10]

$$\text{Sample size} = 4pq/d^2$$

$$p - \text{Prevalence} = 0.96\%$$

$$q - 100-p = 99.04\%$$

$$d - \text{allowable error} = 5\%$$

$$\begin{aligned} \text{Sample size} &= 4 \times 0.96 \times 99.04/5^2 \\ &= 380.31/25 \\ &= 15.21 \end{aligned}$$

As the minimum sample size required for the study is 15.21, for the present study 50 patients with melanocytic nevi were selected.

Study instrument

Pre-prepared questionnaire, digital photograph and dermoscope.

Method of collection of data

- 1) Patients coming to OPD with Melanocytic nevi will be selected for the study.

- 2) The patients will be explained about the nature of the study and written consent of the participating patients and the guardians in case of minors will be taken.
- 3) A pre-structured proforma shall be used to collect baseline data.
- 4) A detailed history is taken, clinical and dermatological examination done.
- 5) The pigmented skin lesions will be evaluated using digital dermoscope.
- 6) A skin biopsy and histopathological examination will be done as and when necessary.

Inclusion criteria

- 1) Patients with Melanocytic Nevi

Exclusion criteria

- 1) Patients who have undergone invasive or non invasive procedure on the lesion during past 6 weeks.
- 2) Use of depigmenting agent.

Ethics statement

Ethical committee clearance taken and copy enclosed.

RESULTS

A total of 50 patients with melanocytic nevi were included in the study. The age of the patients ranged between 16-54 years. The mean age of the study is 33.9 ± 8.38 yrs. Majority of the patients were females. Male to female ratio was 0.78:1 (Table 1). Most of the patients gave chronic history. None of the lesions showed any change in appearance of lesion.

On examination face was the most common site (34%) followed by the forearm (16%). Most of the lesions were round in shape (84%) and majority of the lesions had smooth surface (64%). Majority of the lesions (52%) were of size 6-10mm, with mean diameter of 7.04 ± 3 mm.

Out of 50 patients, 11 were diagnosed with junctional nevi. For rest of the lesions, multiple diagnosis were made, because of inconclusive findings.

Dermoscopic Features

Distribution of local features- Pigment network (Fig. 1) was the most common local feature seen in all 50 lesions. Dots and globules (Fig. 2) were seen in 26 lesions (52%). Pigmentation was seen to be

Table 1: Age and sex distribution

Age group	Male	Female
<20	1	1
21-30	5	12
31-40	13	11
41-50	2	2
>50	1	2
TOTAL	22	28



Figure 1: Reticular pigment network.



Figure 2: Dots and globules and few hair follicles.

regular in all lesions (Table 2). Distribution of global structures- globular network was seen in 42 lesions and reticular network was seen in 38 lesions (Table 3).

Final impression after clinical and dermoscopic examination, incidence of melanocytic nevi was as follows- junctional nevi with reticular network (Fig. 1) as most common 48%, followed by compound nevi with dots and globules and few hair follicles (Fig. 2) 36% and congenital melanocytic nevi with fine reticular network with perifollicular hyperpigmentation (Fig. 3) was 16% (Table 4).

Table 2: Local features

Local features	Frequency	Percentage
Pigment network	50	100
Dots & globules	21	42
Streaks	0	0
Bluish white veil	0	0
Hypopigmentation	0	0
Regression structures	0	0
Vascular structures	0	0

Table 3: Global features

Global features	Frequency	Percentage
Reticular network	38	76
Globular network	42	84
Cobble stone pattern	0	0
Homogenous pattern	0	0
Starburst pattern	0	0
Parallel pattern	0	0
Multi component pattern	0	0
Unspecific pattern	0	0

Table 4: Final impression

Final impression	Frequency	Percentage
Congenital Melanocytic Nevi	8	16
Compound Nevus	18	36
Junctional Nevi	24	48

DISCUSSION

Nevi are common benign proliferations of uniform melanocytes. Although 20–30% of melanomas arise in association with pre-existing naevi, malignant transformation of naevi is a very rare event [11]. A randomized clinical trial of dermatologists trained in dermoscopy revealed a 42% reduction in the number of unnecessary biopsies compared with using the naked eye alone [12]. Hence regular dermoscopic examination of melanocytic nevi is important for early detection of melanoma.

The most commonly involved age group in this study was 31-40 years with a mean age of 33.9 years \pm 8.38. A study by Piazza et al., mean age was 8.4 ± 3.5 years [13]. A study of dermoscopic patterns of congenital melanocytic nevi by Changchien et al., showed a mean age of 35.2 years [14]. In another study of dermoscopic patterns of congenital melanocytic nevi by Cengiz et al., out of 108 patients Sixty-two participants (57.4%) were aged less than 16 years, and 46 participants (42.6%) were aged 16 and more [15].

In this study there was female preponderance. Male to female ratio was 0.78:1. A study by Piazza et al., both male and females were equally affected [13]. In



Figure 3: Fine reticular pigment network and few perifollicular hyperpigmentation.

a study by Changchien et al., out of a study population of 77 individuals, there were 36 females (47%) and 41 males (53%) with male preponderance [14]. According to a study by Cengiz et al., there was similar female preponderance with 57 females out of 108 and 51 males [15].

On examination face was the most common site (34%) and next was the forearm (16%). In a study by Piazza et al., 40% of lesions were on extremities followed by 37% on trunk [13]. A study by Changchein et al., trunk was the most common site in 59.3% of patients followed by extremities [14]. Another study by Cengiz et al., 52% of lesions were on extremities followed by trunk with 40% of lesions [15].

Out of 50, 8 lesions were present at birth. According to a study by Piazza et al., 5 out of 201 lesions were present at birth. Mean diameter of the lesions was 7.04 ± 3 mm. A study by Piazza et al., the mean diameter of the lesions was 2.3 ± 1.4 mm [13]. In this study 84% of the lesions were round followed by 16% lesions were round to oval. In a study by Changchien et al., 79% of lesions were round in shape followed by irregular shape in 14% [14].

On dermoscopic examination, pigment network was seen in all 100% of lesions, dots and globules were noted in 42%, reticular network was seen in 76% and globular network in 84% of lesions. In a study by Piazza et al., the most prevalent dermoscopic structures observed in the lesions were dots (72.6%), followed by structureless areas (47.8%), pigment network (40.8%) and globules (28.4%). The presence of branched streaks was observed in only 5.5% of the lesions [13]. In a study by Cengiz et

al., after dermoscopy it was found that reticular pattern was in 24.1%, globular pattern in 32.4%, reticular-globular pattern in 12%, homogeneous pattern in 14.8%, reticularhomogeneous pattern in 5.6%, globular-homogeneous pattern in 1.9%, cobblestone pattern in 6.5%, reticular patchy pattern in 2.8%. The globular pattern as the predominant dermoscopic pattern was more frequent in children younger than 16 years old (32.4%) [15].

As presented, our observations were partially comparable with the findings of previous studies. The aim of this work was to evaluate the dermoscopic structures and patterns of Melanocytic nevi.

CONCLUSION

In our study, pigment network was seen in all 100% of lesions, globular pattern was observed in 84% and dots and globules in 42%. Dermoscopy is an evolving science. It serves as a link between macroscopic skin lesions and microscopic histopathological features. Since it is non-invasive, it can be used in all age groups including children and elderly, reducing the need for interventional procedures like skin biopsy. Dermoscopy is a much needed investigative tool in the assessment of Melanocytic nevi.

Statement of Human and Animal Rights

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

Statement of Informed Consent

Informed consent was obtained from all patients for being included in the study.

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A clinicoepidemiological study of facial hypermelanosis among females of reproductive age group

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ABSTRACT

Background: Facial hypermelanosis has great psychological and aesthetic complications attached to it specially in females. A number of factors like genetic, environmental, systemic, work in consortium to give rise to various types of hypermelanosis. Women in the 15-49 years age group, which is the reproductive age group are influenced by various hormonal alterations along with external and systemic agents thus manifesting as various types of hypermelanosis. **Aims and objectives:** This study was undertaken to assess the clinical and epidemiological aspects of various types of facial hypermelanosis and various factors implicated. **Material and methods:** A total of 350 women in the age group of 15- 49 years were taken up for the study. A detailed clinical history, thorough examination was done in all patients. Relevant investigations were carried out. All the results were statistically analyzed and inferences were drawn. **Results:** A total of 350 women were taken up for study. Maximum patients (133,38%) were in the age group of 26 -35 years. Among various hypermelanosis, maximum women(128,36.5%) came with the complaint of melasma, 90(25.7%) women with post inflammatory hypermelanosis, 50(14.3%)females with periorbital hypermelanosis. In our study, melasma, post inflammatory hyperpigmentation due to acne, acanthosis nigricans formed a major share in the 15-49 years age group thus suggesting the role of hormonal alterations and rising PCOS. **Conclusion:** Females are under considerable psychological and societal pressure. Besides adequate treatment, the role of hormonal alterations, life style factors, occupation in the above said age group needs to be ascertained by more such studies.

Key words: Facial hypermelanosis; Reproductive age group; Hormonal alterations

INTRODUCTION

Hypermelanosis affecting the face is a commonly encountered problem among Indian patients seeking dermatological advice. Facial hypermelanosis carries immense psychological impact owing to its evident cosmetic disfigurement and social stigmas attached to it [1]. Moreover, people of Asian and African descent constitutively have a darker phenotype which is more vulnerable to pigmentation [2]. Facial hypermelanosis encompasses a myriad of clinical entities which are commonly encountered: melasma, peri-orbital melanosis, postinflammatory hyperpigmentation, lichen planus pigmentosus, Riehl's melanosis, freckles and

lentigenes, exogenous ochronosis, acanthosis nigricans, erythema dyschromicum perstans, and uncommonly poikiloderma of Civatte, erythromelanosis follicularis of face and neck, nevus of Ota, some to be named. Most of the studies worldwide on facial hypermelanosis have reported a female predominance [3,4]. In India, a great heterogeneity was reported among women in the facial skin color with hypermelanosis accounting for a major share [5]. Besides constitutive pigmentation, a complex interplay of genetic, environmental, systemic diseases, hormonal factors along with use of external agents, have been reported in the causation of various types of facial hypermelanosis [6]. Women in the reproductive age group (15-49 years) undergo hormonal alterations

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right from menarche to menopause thus somewhere making them more prone to hypermelanosis, specially melasma [7]. Also, excessive exposure to sunshine on account of occupation or agricultural work, use of beautifying creams, cosmetics, use of drugs also increase the chances of facial hypermelanosis in the same age group. This study was undertaken to assess the clinical and epidemiological aspects of facial hypermelanosis in women in the 15-49 years age group.

MATERIAL AND METHODS

This was a prospective study carried out over a period of 3 months. A total of 350 women in the age group of 15- 50 years who presented to the outpatient department at our dermatology department with facial hypermelanosis were taken up for the study after obtaining written informed consent. These were divided into various age groups: 15-25 years, 26-35 years, 36-45 years, >45 years. A detailed clinical history regarding age of onset, duration, any aggravating factors, family history was taken. History about various causative factors implicated: sun exposure, occupation, use of cosmetics or chemical agents, drug intake or presence of any systemic illnesses was also noted. Any history of diabetes, thyroid disorders or any hormonal abnormalities was asked too. A detailed general physical examination regarding distribution of pigmentation, whether dermal or epidermal was noted. Also evidence of any other systemic diseases or hormonal abnormalities was also examined. Woods lamp examination was done in all patients and biopsy was taken wherever necessary. Laboratory investigations were done wherever necessary and included complete haemogram, blood glucose levels, serum insulin, renal and liver function tests, thyroid profile and relevant hormonal abnormalities. All the results were statistically analyzed and inferences were drawn.

RESULTS

A total of 350 women were taken up for study. Maximum patients (133,38%) were in the age group of 26 -35 years followed by 105 (30%) patients in the 36-45 years age group. In the 15-25 years age group, there were 65 (18.7%) patients and 47 women were in the 45-49 (13.4%) years age group. Among various hypermelanosis, maximum women (128,36.5%) came with the complaint of melasma (Fig. 1), followed by 90 (25.7%) women with post inflammatory hypermelanosis, 50 (14.3%) females with periorbital hypermelanosis. The

distribution of various types of hypermelanosis is given in Table 1. The age group wise distribution of various hypermelanosis is shown in Table 2.

Females with melasma formed a major share. Most of these were in the age group of 26-45 years. The most common pattern seen was malar seen in 55 (43%) females followed by centrofacial (41,32%) and mandibular patterns (32,25%). Woods lamp examination was done in all patients of melasma. Epidermal pigmentation was seen in 44 (34.3%) patients, dermal in 38 (29.6%) females, mixed in 31 (24.2%) females and indeterminate pigmentation in 15 (11.7%) patients. Among various predisposing factors, pregnancy was the major predisposing factor seen in 75 (58.5%), use of oral contraceptive pills(OCP's) and other hormonal supplements in 23 (17.9%) females. Few females were taking OCP's and hormonal supplements were suffering from PCOS. In 16 (12.5%) females, thyroid dysfunction was seen. 38 (29.6%) out of 128 females gave sun exposure as the



Figure 1: A female with epidermal melasma.

Table 1: Showing distribution of various melanosis

Facial hypermelanosis	Number of patients(%)
Melasma	128
Post inflammatory hyperpigmentation	90 (25.7)
Peri orbital melanosis	50 (14.3)
Freckles/lentigines	31 (8.8)
Lichen planus pigmentosus	14 (4)
Acanthosis nigricans	12 (3.4)
Riehl's melanosis	7 (2)
Pigmented cosmetic dermatitis	5 (1.4)
Ochronosis	5 (1.4)
Naevus of ota	2(5)
Miscellaneous	6 (1.7)
Drug induced pigmentation	2
Porphyrias	1
Systemic diseases	3

Table 2: Showing distribution of various hypermelanosis among various age groups

Types of melanosis	15-25 years	26-35 years	36-45 years	46-49 years	Total
Melasma	10	76	34	8	128
Post inflammatory hyperpigmentation	9	24	33	24	90
Peri orbital melanosis	21	13	10	6	50
Freckles/lentigens	14	9	6	2	31
Lichen planus pigmentosus	3	4	6	1	14
Acanthosis nigricans	2	3	5	2	12
Riehl's melanosis	2	0	3	2	7
Pigmented cosmetic dermatitis	0	1	3	1	5
Ochronosis	0	1	3	1	5
Naevus of ota	2	0	0	0	2
Miscellaneous				0	6
Drug induced	2	0	0	0	
Porphyrias	0	2	0	0	
Systemic	0	0	2	0	
Total	65	133	105	47	350

aggravating factor owing to prolonged exposures to sun during working in fields among rural women or travelling as part of their occupation among others in addition to above causes. The second most common type of hypermelanosis among females was post inflammatory hyperpigmentation (PIH). Maximum cases of hyperpigmentation was attributed to severe acne seen in 56 (62.2%) females and out of these, 39 females had evidence of hormonal imbalance in the form of Poly Cystic Ovarian Syndrome. Females with peri orbital melanosis mostly belonged to <30 years age group and major predisposing factors were genetic predisposition (21 females), stress and inadequate sleep (9), excessive exposure to laptop or mobile sunscreens (8). Peri-orbital oedema and skin laxity also was seen in 6 females. In the rest evidence of atopy and air borne contact dermatitis was seen. Freckles and lentigines (Fig. 2) were seen in 31 females and genetic factors along with sun exposure were implicated. Lichen planus pigmentosus were seen in 14 (4.4%) patients. Sun exposure was the most common aggravating factor. 5 patients reported the use of mustard oil and amla oil which led to the development of lichen planus pigmentosus. The pigmentation ranged from slate gray to brownish black. The forehead and the temples were most commonly involved. Most women belonged to 35-49 years of age group. Among 12 patients of acanthosis nigricans, diabetes was present in 3. In 7 patients, obesity was present leading to altered insulin levels. There were 2 patients of proven PCOS on treatment who had acanthosis nigricans. Other hypermelanosis seen less commonly were: Riehl's melanosis, pigmented cosmetic dermatitis, ochronosis due to abuse of hydroquinone and naevus of ota. Also drug induced hyperpigmentation due to clofazimine and minocycline was also seen.

**Figure 2:** A young female with lentigines

DISCUSSION

Number of females with facial hyper melanosis is seeing an upwards trend among the out patients presenting to dermatologists and various contributing factors are: increased awareness, society and marriage pressures to look more beautiful, use of drugs and cosmetics, increased sun exposure and rising obesity and other hormonal abnormalities due to changing life styles. In our study involving females in the reproductive age group which witnesses multiple hormonal alterations, the most common hypermelanosis seen was melasma present in 36.5% of the patients. Multiple studies across India also have found melasma to be the most common hypermelanosis and maximum women belong to the same age group as in our study [5,8]. In our study malar pattern was the most common in 43% followed by centrofacial seen in 32% and mandibular pattern seen in 25% similar to other studies [8,9]. On the contrary various studies have reported centrofacial

pattern to be the most common [10,11]. The most common precipitating factor was pregnancy (58.2%) in our study followed by OCP's and hormonal supplements (17.9%) thus giving impetus to the role of hormones in its genesis which has been reported in other studies as well [12-14]. Exposure to ultraviolet rays have been found to be crucial in various studies as it leads to increased levels of various hormones: alpha-melanocyte-stimulating hormone, corticotrophin and also interleukin (IL)-1 that, in turn, results in increased melanin production [15]. In our study also sunlight was implicated in the exacerbation of melasma in 29.6% in concordance with other studies which also reported sunlight as the exacerbating factor [16-18]. Epidermal pattern was the most common in our study similar to another study [19]. In our study, the second most common facial hypermelanosis was postinflammatory hyperpigmentation seen in 25.7% of the females. In PIH, skin inflammation results in the production of cytokines, prostaglandins, leukotrienes which further stimulate melanin synthesis. The most common cause attributed was acne. Multiple other studies have also reported similar findings [5,20]. In our study many females with acne were diagnosed cases of PCOS thus implicating hormonal role. In our study we found a lower incidence of periorbital melanosis at 14.3% as compared to other studies which had higher prevalence [20]. Another study reported an even lower prevalence [8]. This could be due to genetic or regional variation. In our study, major factor was genetic predisposition, inadequate sleep, exposure to laptops and mobiles, atopy and airborne contact dermatitis. These were well in concordance with various other studies [8,21,22]. Freckles and lentigenes was also seen in our study with a prevalence of 8.8%. Freckles are mostly seen in fairer skin with sun exposure also implicated. Lentigenes can occur anywhere in body and have a genetic role majorly. Lichen planus pigmentosus is a variant of lichen planus and prevalence in our study was similar to other studies and most of the patients were >35 years well in accordance to other studies [8,23,24]. Acanthosis nigricans comprises of hyperpigmented velvety plaques mostly over the flexures but involves the face as well. In our study obesity and diabetes were the major reasons but PCOS was also seen. Various other studies have reported similar findings [25]. In our study, PCOS resulting in hormonal imbalances and its treatment was implicated in melasma, PIH resulting from acne, acanthosis nigricans thus suggesting rising trend of it in the above studied age group. Also in our study, melasma, post inflammatory hyperpigmentation

due to acne, acanthosis nigricans formed a major share in the 15-49 years age group thus suggesting the role of hormonal alterations and rising PCOS. Riehl's melanosis, pigmented cosmetic dermatitis, ochronosis due to abuse of hydroquinone and naevus of Ota, facial pigmentation due to clofazimine in a leprosy patient and minocycline in another patient were other less common hypermelanosis observed.

CONCLUSION

Facial hypermelanosis is commonly encountered by dermatologists and has multiple patterns. The female gender is particularly vulnerable to the social and psychological constraints of it. The role of hormonal alterations, life style factors, occupation in the above said age group needs to be ascertained by many more such studies.

Statement of Human and Animal Rights

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

Statement of Informed Consent

Informed consent was obtained from all patients for being included in the study.

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Etiologic profile of breast dermatoses in Dakar: A prospective study on 125 cases

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ABSTRACT

Background: The severity of breast dermatoses is related to the existence of an underlying malignant neoplasia with a risk of mastectomy that involves both vital and functional prognosis. The aim of this study was to determine the epidemiological, clinical and etiological aspects of breast dermatoses in Dakar. **Materials and methods:** It was a multicenter descriptive study over a 6-month period in three departments in Dakar. All patients who were referred for dermatosis localized only in the breast were included in this study. **Results:** We collected 125 cases, a hospital frequency of 1.02%. The sex ratio was 0.016. The average age was 45 years [16 -79 years]. The dermatoses of the breast were tumoral in 92 cases, infectious in 25 cases and immuno-allergic in 8 cases. The tumoral pathologies were malignant in 90 cases and benign in 2 cases. Metastases were pulmonary (32 cases), ganglionic (29 cases), hepatic (14 cases), bony (13 cases), renal (1 cases) and cerebral (1 cases). The treatment was etiological with topical corticosteroids, antibiotics and antifungals. The different treatments for the tumors were surgery, chemotherapy, hormone therapy and radiotherapy. A cure was noted in 26 cases (20.8%) and one death in 6 cases. **Conclusion:** We report a high prevalence of breast cancer in adult women and the occurrence of mastitis and puerperal breast abscess in young women and a high frequency of allergic contact dermatitis during atopy.

Key words: Etiology; Breast dermatoses; Dakar

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Profil étiologique des dermatoses du sein à Dakar: Une étude prospective sur 125 cas

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RÉSUMÉ

Introduction: La gravité des dermatoses du sein est liée à l'existence d'une néoplasie maligne sous-jacente avec un risque de mastectomie qui met en jeu le pronostic vital et fonctionnel. L'objectif de cette étude était de déterminer les aspects épidémiologiques, cliniques et étiologiques des dermatoses du sein à Dakar. **Matériels et méthodes:** Il s'agissait d'une étude descriptive multicentrique sur une période de 6 mois dans trois services à Dakar. Tous les patients venus consulter pour une dermatose localisée uniquement au niveau de la région mammaire ont été inclus dans l'étude. **Résultats:** Nous avons colligé 125 cas soit une fréquence hospitalière de 1,02%. Le sexe ratio était de 0,016. La moyenne d'âge était de 45 ans [16-79 ans]. Les dermatoses du sein étaient tumorales dans 92 cas, infectieuses dans 25 cas et immuno-allergiques dans 8 cas. Les pathologies tumorales étaient malignes dans 90 cas et bénignes dans 2 cas. Les métastases étaient pulmonaires (32 cas), ganglionnaires (29 cas), hépatiques (14 cas), osseuses (13 cas), rénales (1 cas) et cérébrales (1 cas). Le traitement était étiologique avec les dermocorticoïdes, les antibiotiques et les antifongiques. Les différents traitements des tumeurs étaient la chirurgie, la chimiothérapie, l'hormonothérapie et la radiothérapie. Une guérison était notée dans 26 cas (20,8%) et un décès dans 6 cas. **Conclusion:** Notre rapportons une prévalence élevée du cancer du sein chez les femmes adultes et la survenue des mastites et des abcès du sein puerpérales chez la femme jeune et une plus grande disposition à développer un eczéma de contact en cas de dermatite atopique.

Mots clés: Etiologies; Dermatoses du sein; Dakar

INTRODUCTION

Les dermatoses du sein regroupent l'ensemble des manifestations dermatologiques localisées à la glande mammaire et/ou de son revêtement cutané ou secondaire à une maladie générale. Les causes de l'atteinte spécifique de la peau du sein sont multiples d'origine le plus souvent infectieuse, tumorale et/ou immunoallergique [1]. La gravité des dermatoses mammaires est liée d'une part à l'existence d'une néoplasie maligne sous-jacente avec une possibilité de mise en jeu du pronostic vital par le biais d'une métastase à distance et d'autre part au préjudice fonctionnel par le risque de mastectomie et d'arrêt de l'allaitement [2-5]. Peu d'études ont rapporté la prévalence et les différentes causes des dermatoses du

sein en Afrique subsaharienne alors que le cancer du sein occupe la première cause des pathologies mammaires au Sénégal [6]. L'objectif de cette étude était de déterminer les aspects épidémiologiques, cliniques et étiologiques des dermatoses du sein à Dakar.

MATÉRIELS ET MÉTHODES

Il s'agissait d'une étude descriptive multicentrique sur une période de 6 mois dans les services de Dermatologie et de cancérologie de l'Hôpital Aristide Le Dantec et le service de Dermatologie de l'Institut d'Hygiène Sociale. Nous avons inclus tous les patients venant consulter pour une dermatose localisée uniquement au niveau de la région mammaire.

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Nous avons exclu les patients qui présentaient une dermatose mammaire relevant d'une maladie générale. Le diagnostic de dermite de contact était basé sur l'interrogatoire (recherche d'une notion d'application de produits), l'examen clinique et l'exploration allergologique par les patch-tests. Le diagnostic de dermatite atopique est basé sur l'interrogatoire à la recherche d'équivalent atopique familial ou personnel (asthme, rhinite allergique, conjonctivite allergique, eczéma) et les critères diagnostiques de l'United kingdom working party. Le diagnostic de l'infection était basé sur les signes cliniques évocateurs et les explorations paracliniques notamment mycologiques, bactériologiques et à l'échographie.

Les causes tumorales étaient confirmées par l'examen anatomo-pathologique soit par le biais d'une biopsie cutanée ou au tru-cut. L'échographie et la mammographie étaient demandées à la recherche de lésions suspectes de malignité. Le scanner thoraco-abdomino-pelvien recherchait une localisation secondaire. Les explorations allergologiques étaient effectuées avec la batterie standard européenne. La saisie et l'analyse des données ont été effectuées sur les logiciels Sphinx version 5.1.0.2 et SPSS version 18.

RÉSULTATS

Nous avons recensé 125 cas de dermatoses mammaires sur 12211 malades vus durant la période de l'étude soit une fréquence hospitalière de 1,02%. Le sexe ratio était de 0,016 soit 2 hommes et 123 femmes. La moyenne d'âge était de 45ans avec des extrêmes de 16 ans à 79 ans. La répartition des tranches d'âge selon les groupes pathologiques est illustrée sur la Fig. 1. Le délai moyen

de consultation était de 8 mois avec des extrêmes de 5 jours à 5 ans.

Les circonstances de découverte étaient la douleur (18 cas), le prurit (8 cas), le placard inflammatoire (25 cas), l'intertrigo (10 cas), l'érythème (2 cas) et une masse mammaire (62 cas). Les malades ont eu recours en première intention au médecin généraliste dans 49 cas et à un tradipraticien dans 25 cas. Les antécédents ou terrains étaient cardio-vasculaires (une hypertension artérielle (12 cas), un diabète (6 cas), une dyslipidémie (2 cas) et une obésité (8 cas), immuno-allergiques (une atopie dans 9 cas et un eczéma des seins dans 4 cas), infectieuses (un abcès dans 2 cas, un intertrigo sous mammaire dans 2 cas) et tumorales (carcinome canalaire infiltrant dans 4 cas). L'état général était conservé dans 118 cas et altéré dans 7 cas. Une fièvre était notée dans 6 cas. Les lésions mammaires étaient unilatérales dans 107 cas et bilatérales dans 18 cas.

Les différents aspects cliniques lésionnels sont répertoriés sur le Tableau I. Nous avons noté trois groupes

Tableau I : Répartition des cas en fonction des lésions mammaires

Aspects des lésions	Effectifs	%
Erythème	82	65,6
Masse tumorale	63	50,4
Induration	42	33,6
Aspect peau d'orange	36	28,8
Rétraction	31	24,8
Écoulement mamelonnaire	16	12,8
Nécrose	10	8
Enduit blanchâtre	9	7,2
Nodule	8	6,4
Vésicules	6	4,8
Troubles pigmentaires	5	4
Lichénification	2	1,6

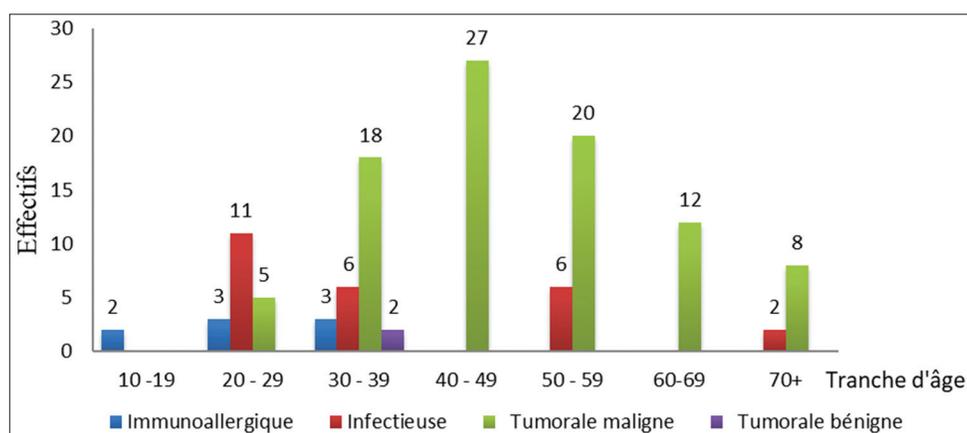


Figure 1 : Répartition des tranches d'âge en fonction des groupes pathologiques.

de pathologies mammaires. Il s'agissait de pathologies tumorales dans 73,6% (92 cas), infectieuses dans 20% (25 cas) et immuno-allergiques dans 6,4% (8 cas). Les pathologies tumorales étaient malignes dans 90 cas et bénignes dans 2 cas.

La répartition des cas en fonction des types de tumeurs est illustrée sur le Tableau II.

Les pathologies infectieuses étaient bactériennes dans 60% (15 cas) à type d'abcès du sein (9 cas) (Fig. 2), de mastites puerpérales (6 cas) et d'origine mycosique (10 cas).

Les pathologies immuno-allergiques étaient à type de dermatite de contact allergique (7 cas) (Fig. 3) et de dermatite atopique (1 cas).

La topographie des lésions au niveau mammaire était au niveau des 4 cadrans dans 52 cas, la zone péri-aréolaire dans 19 cas, la région sous mammaire dans 10 cas, les aréoles dans 2 cas, les mamelons dans 3 cas, la région pariétale dans 4 cas.

Les adénopathies étaient présentes dans 89 cas soit 70,6% avec un aspect.

Inflammatoire dans 10 cas et non inflammatoire dans 79 cas.

Ils siégeaient aux niveaux axillaires dans 89 cas et sus-claviculaires dans 10 cas. Les métastases notées dans 47 cas étaient pulmonaires (32 cas), ganglionnaires (29 cas), hépatiques (14 cas), osseuses (13 cas), rénales (1 cas) et cérébrales dans 1 cas.

L'histologie a confirmé un carcinome épidermoïde dans 2 cas, un molluscum pendulum (Fig. 4), Une mastite et un lymphome non hodgkinien dans 1 cas.

Tableau II : La répartition des cas en fonction des types de tumeurs malignes

Types de tumeurs	Effectifs	%	
Bénignes	1	0,8	1,6
Adénofibrome	1	0,8	
Molluscum pendulum			
Malignes			72
Carcinome canalaire			
Carcinome infiltrant	49	39,2	
CCI	23	18,4	
Adénocarcinome	6	4,8	
Récidive tumorale	4	3,2	
Carcinome lobulaire	3	2,4	
Carcinome épidermoïde	3	2,4	
Carcinome mucineux	1	0,8	
Lymphome	1	0,8	

La biopsie mammaire au tru-cut a été effectuée dans 86 cas, elle a permis de confirmer un carcinome infiltrant (49 cas) (Fig. 5), un carcinome canalaire infiltrant dans 23 cas, un adénocarcinome dans 6 cas, un carcinome lobulaire dans 3 cas, une mastite aigue dans 2 cas,



Figure 2: Un abcès du sein fistulisé chez une nourrice de 25ans.

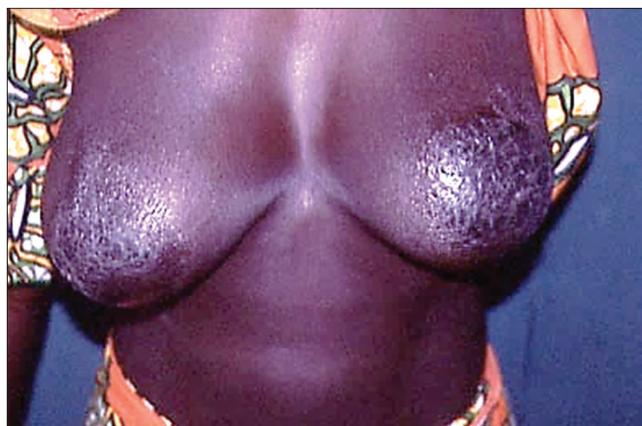


Figure 3: Eczéma de contact des seins au bichromate de potassium et fragrance mix.



Figure 4: Molluscum pendulum chez une femme de 35 ans.



Figure 5: Un carcinome infiltrant inflammatoire du sein gauche.

un carcinome épidermoïde dans 1 cas, un carcinome mucineux dans 1 cas et une tumeur conjonctive bénigne dans 1 cas. L'examen mycologique a isolé le *Candida albicans* dans 2 cas. Les patch-tests étaient positifs au bichromate de potassium et le paraphénylènediamine dans 2 cas. Le bichromate de potassium et le fragrance mix 1 dans 1 cas. Le fragrance mix 1 et la benzocaïne dans 1 cas. Le bichromate de potassium et le clioquinol dans 1 cas.

L'échographie mammaire a décelé une formation tissulaire suspecte de malignité dans 48 cas. La mammographie était réalisée dans 51 cas. Selon la classification de l'American College of Radiology (ACR), les malades étaient classés en ACR 1 (2 cas), ACR 2 (4 cas), ACR 3 (3 cas), ACR 4 (10 cas) et ACR 5 (32 cas). Le scanner thoraco-abdomino-pelvien réalisé dans 80 cas, a permis de détecter des localisations métastatiques secondaires dans 47 cas (52,2%).

Le traitement était celui de la cause. Les dermocorticoïdes et les antihistaminiques dans l'eczéma de contact (8 cas). Les antifongiques dans les infections mycosiques (15 cas), les antibiotiques dans 15 cas d'infections bactériennes associés à un drainage d'abcès dans 9 cas.

Pour les causes tumorales une chimiothérapie a été utilisée en première ligne avec le protocole AC (Anthracycline + cyclophosphamide) dans 59 cas. Le protocole FAC (5-Fluoro-Uracile + anthracycline + cyclophosphamide) dans 13 cas. Les taxanes étaient utilisés en 2^{ème} ligne dans 15 cas. L'hormonothérapie (anastrozole) était utilisée dans 2 cas. Le traitement chirurgical consistait à une tumorectomie dans 2 cas,

une mastectomie dans 1 cas et une biopsie exérèse dans 1 cas. La radiothérapie était utilisée dans 2 cas. L'évolution était favorable pour les causes infectieuses dans 17 cas, pour les causes immuno-allergiques dans 7 cas et les causes tumorales dans 2 cas. Une réponse tumorale partielle était notée dans 9 cas et un décès dans 6 cas secondaire au cancer du sein.

En analyse bi varié nous avons noté une corrélation significative entre l'âge adulte et la survenue du cancer du sein et l'existence d'une atopie au cours de l'eczéma de contact.

DISCUSSION

Notre étude rapporte une fréquence hospitalière de 1,02% des dermatoses mammaires à Dakar. Ces dermatoses se caractérisent par une prédominance des causes tumorales dans 72%, suivi des causes infectieuses et immunoallergiques.

Au Sénégal le cancer du sein et de l'appareil gynécologique occupent le premier rang et représentent 54% des cancers de la femme selon le registre des tumeurs [6].

Par ordre de fréquence, les tumeurs malignes étaient réparties en carcinomes canaux (91,1%), en carcinome lobulaire (3,3%), en carcinome épidermoïde (3,3%), en carcinome mucineux (1,1%) et en lymphome non hodgkinien (1,1%).

La prédominance des carcinomes canaux parmi les types histologiques de cancer du sein est concordante avec les résultats rapportés dans la littérature [7,8]. Les carcinomes épidermoïdes primitifs du sein ou carcinomes à cellules squameuses sont rares et représentent 0,1 à 2% de l'ensemble des carcinomes du sein, ils appartiennent au groupe hétérogène des carcinomes métaplasiques mammaires et sont d'étiopathogénie et de pronostic controversés [9]. Le cancer du sein peut interpeller le dermatologue à différents stades de son évolution. Les principales modifications cutanées induites par le cancer du sein sont: le placard inflammatoire, la rétraction du mamelon, l'induration, l'aspect en peau d'orange ou encore l'ulcération [10-12].

Les dermatoses infectieuses occupaient le second rang des affections mammaires. Les mastites étaient trouvées dans 6 cas soit 4,8% et les abcès du sein dans 9 cas soit 7,2%, ils étaient tous d'origine puerpérale.

Deux formes principales de mastites sont décrites dans la littérature: les mastites puerpérales survenant pendant les périodes de lactation, et les mastites non puerpérales.

La mastite puerpérale touche 10-20% des femmes pendant les six premiers mois de lactation et le risque de développer un abcès en cas de mastite est de 5-10%. La stase laiteuse constitue un facteur de risque pour le développement d'une infection bactérienne des canaux lactifères et lymphatiques, particulièrement en présence d'excoriations au niveau du mamelon [16]. Quant à l'abcès du sein, sa fréquence était plus élevée que celle rapportée dans la littérature: 3% aux États Unis et Allemagne [13-15] et de 0,52% en France [9]. Les causes immunoallergiques occupaient le troisième rang, il s'agissait de 7 cas d'eczéma de contact et d'un cas de dermatite atopique.

L'eczéma de contact était associé à un terrain atopique dans 5 cas soit 71,4% avec un lien statistiquement significatif. Ceci rejoint les données de la littérature qui montre une prévalence plus élevée de l'eczéma de contact du sein avec un terrain d'atopie [17]. Les eczémas de contact au bichromate de potassium peuvent être liés à la composition du textile. Certaines industries du textile utilisent cette substance pour la teinture du coton et la laine notamment pour le linge du corps ou les vêtements et peuvent ainsi être la source allergénique. Le PPD est un colorant noir qui entre dans la composition de certains colorants textiles utilisés dans la fabrication des vêtements ou du linge du corps.

De ce fait, il peut être la source allergénique à l'origine de l'eczéma au PPD.

Le fragrance mix 1 est un mélange de 8 allergènes servant à dépister une allergie aux parfums. On peut le retrouver dans divers produits cosmétiques: parfums, eau de toilette, maquillage, lait de corps [18-20].

Une évolution favorable était notée dans 68% parmi les causes infectieuses, 87,5% des causes immunoallergiques et 2,1% des causes tumorales. Il s'agissait de tumeurs bénignes (molluscum pendulum et adénofibrome) qui avaient reçu une tumorectomie. Une réponse partielle avec diminution de la taille de la tumeur sous chimiothérapie était notée dans 9,8% des cas des causes tumorales.

Une persistance de la tumeur était notée dans 19,6% des cas des causes tumorales.

Cependant le recul était insuffisant pour apprécier l'efficacité définitive de la chimiothérapie.

CONCLUSION

Les manifestations dermatologiques mammaires peuvent être primitives ou secondaires à une maladie sous jacente. Notre étude rapporte une prévalence élevée du cancer du sein chez les femmes adultes entre 40 et 50 ans. La fréquence des mastites et des abcès du sein au cours de la période puerpérale chez les jeunes femmes âgées entre 20 et 30 ans et une plus grande disposition à développer un eczéma de contact en cas de dermatite atopique.

Statement of Human and Animal Rights

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

Statement of Informed Consent

Informed consent was obtained from all patients for being included in the study.

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Meaning of microalbuminuria during the atopic dermatitis of the child

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ABSTRACT

Objectives: Advocate early diagnosis of vascular diseases in children with atopic dermatitis. Determine children at vascular risk during atopic dermatitis. **Method:** The study is transversal and analytical; performed in the University of Brazzaville Medical Center in 18 months. It focuses on children from 0 to 15 years old with atopic dermatitis. The anthropometric, clinical, and antecedent data are collected on cards as well as the balance necessarily including: fasting blood glucose, serum immunoglobulin E (IGE) and microalbuminuria. **Results:** 80 patients were selected, 47 girls and 33 boys. The mean age was 8.9 years SD \pm 4.646. Obesity is found in 21.25% of cases. 82, 5% of children had hypertensive parents or diabetics in the first degree. Microalbuminuria was positive in 53.75% of cases. It was independent of age and sex and more common in children with hyper IgE. **Conclusion:** The study reports arguments for a vascular predisposition and show the interest of achieving microalbuminuria in atopic dermatitis.

Key words: Atopic dermatitis; Arterial hypertension; Diabetes; Microalbuminuria; Child

INTRODUCTION

Atopic dermatitis (AD) is an inflammatory, pruritic and chronic condition of complex etiopathogenesis with implications for hereditary factors and environmental antigens [1]. Atopic dermatitis can be considered as a systemic disease [2], its clinical diagnosis is done by the criteria of the United Kingdom Working Party.

DA is a model of polygenic inheritance that is accompanied by stimulations of secretion of polypeptide substances and chemical mediators with vasoactive activity [1]. Some metabolic manifestations such as obesity and some idiopathic nephrotic syndromes are described in AD [3,4]. The occurrence of arterial hypertension during atopic dermatitis has been suggested [5].

The authors conducted a hospital study to look for a link between AD and common vascular pathologies such as diabetes, high blood pressure and obesity. The

interest of this study is to advocate early detection and prevention of vascular diseases in children with AD.

PATIENTS AND METHOD

It is a transversal and analytical study, carried out in University of Brazzaville Medical Center in consultation from January 2015 to July 2016. The study is made by a Dermatologist and a nephrologist hospital-university.

Inclusion criteria were: patients aged 0-15 years with AD. Exclusion criteria were: urinary tract infection or other active infection, ongoing or long-term steroid therapy.

The clinical and laboratory parameters studied were: body mass index, personal and family history of diabetes, high blood pressure, asthma, ambulatory blood pressure measurement with a suitable cuff, fasting blood glucose, blood count, C reactive protein, cytobacteriological examination of urine, ADDIS count, serum immunoglobulin E (IGE), microalbuminuria.

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Microalbuminuria was performed on urination, it was considered positive when it was greater than 20ml/g.

The diagnosis of AD was made thanks to the criteria of the United Kingdom Working Party. The severity of the AD was defined by the SCORAD index.

The information was recorded on cards. Data analysis was done by SPSS for calculating averages, standard deviations and correlations.

RESULTS

Epidemiological aspects

374 children were received during the study period among them, 87 had an AD (23.6%) and 80 met the criteria of the study. There were 33 boys and 47 girls. The M/F ratio was 0.7. The average age was 8.9 years SD \pm 4.646 years. The average age of girls was 10.19 ± 4.08 and 7.24 ± 4.89 for boys (P = 0.099).

Clinical aspects

There was no diabetes or ongoing hypertension in the patients. 13 children were asthmatic (16.25%), 17 obese children (21.25%), 66 children had parents with diabetes or hypertension (82.5%); distributed as follows: high blood pressure (n = 46), hypertension and diabetes (n = 12), diabetes (n = 8).

Biological aspects

Microalbuminuria was positive in 43 cases (53.75%). Mean microalbuminuria was 24.20 ± 21.40 . This average was evaluated at 27.4 ± 28.57 for boys and 21.93 ± 14.35 for girls (P = 0.016). 80% of children with microalbuminuria had hypertensive or diabetic parents. IgE was elevated in 45% of patients. The average IgE was 339.2 ± 371.5 .

Correlation between microalbuminuria and the different variables studied

Positive microalbuminuria was found in 47.3% of children in the 0 to 4 age group, 50% had positive microalbuminuria in the age group of 5 to 9 years, and 60% between 10 and 15 years old. The Pearson correlation rate is 0.022, bilateral significance 0.845 (Table 1). The averages of microalbuminuria with respect to age are shown in Fig. 1.

Positive microalbuminuria was found in 45% of children who have hyper IgE. The Pearson correlation was 0.212;

Table 1: Correlations between microalbuminuria and different variables

	Age	Microalbuminy	IGE
Age			
Correlation of pearson	1	0.022	0.208
Sig. (bilateral)		0.845	0.063
N	80	80	80
Microalbuminy			
Correlation of pearson	0.022	1	0.212
Sig. (bilateral)	0.845		0.059
N	80	80	80
IGE			
Correlation of pearson	0.208	0.212	1
Sig. (bilateral)	0.063	0.059	
N	80	80	80

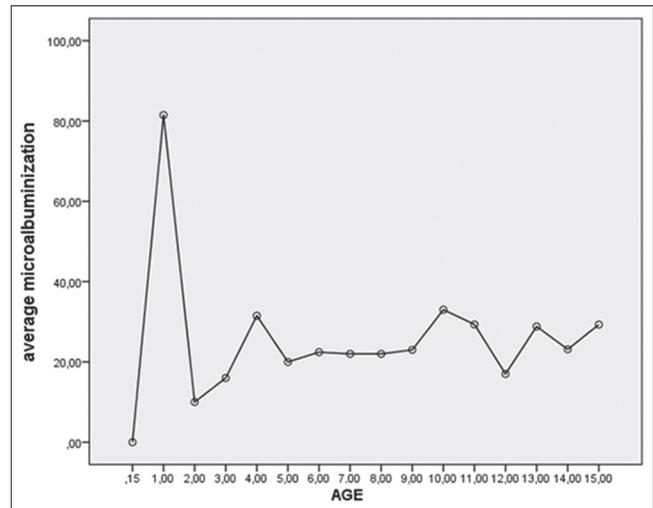


Figure 1: Microalbuminurie and age.

Bilateral significance 0.059. The microalbuminuria averages by age group are shown in Fig. 1. There were 13 asthmatic patients among whom 5 (38.4%) had microalbuminuria. Severe AD exist in 23.75% cases, 73.6% of whom had hyper IgE and positive microalbuminuria.

DISCUSSION

Atopic dermatitis is an inflammatory, pruriginous and spongiotic disease [6]. It is considered a systemic disease [2]. prevalence of AD is high but varies by study [7-9]. The classic feminine predominance is found in our study [10]. The penetrance of AD is variable, comorbidity is common [11] other manifestations of atopy are often associated. In the study, 16.25% of children were asthmatic, 24.7% had AD strict.

The physiopathology of AD is complex [1]; it is accompanied by stimulations, secretions of chemical

mediators and a high cellular activity [12]. These mechanisms are arguments for an alteration of the endothelial tissue. There is a relationship between inflammatory mediators and vascular and metabolic risk factors: interleukin 6 induces insulin resistance [13], cytokines (TNF, IL6, interferon gamma) have a pro-inflammatory role [14], T h 1, T h 2 regulatory T lymphocytes, whose activity is crucial in AD, induce atherosclerosis [15], podocyte damage is found in the idiopathic nephrotic syndromes of atopic subjects [16].

The arguments for a vascular predisposition in our study are: 82.5% of first degree parents were diabetic and or hypertensive, the positivity of microalbuminuria in 53.75% of children.

The association of AD with obesity, diabetes and arterial hypertension is gaining acceptance [17,18]. The link between idiopathic nephrotic syndrome and atopic dermatitis has been suggested [4,16]. In our study 26.25% of children were obese. No child was hypertensive or diabetic. For Zhang [19], obesity is controversial in AD but is frequently reported in studies such as Kusunoki [3] and Lim [20]. The small sample in the study did not allow us to associate obesity with AD. Microalbuminuria reflects either a rise in capillary pressure or an alteration of the triple glomerular barrier or a generalized endothelial dysfunction it is a risk factor cardiovascular whose interest is established in the management of diabetes and high blood pressure [21]. Positive microalbuminuria appeared to exist independently of age, more than half of the patients had positive microalbuminuria. We did not find an explanation for the peak before the age of two. The positivity of microalbuminuria appeared to be related to the elevation of immunoglobulin E and the severity of AD.

CONCLUSION

The existence of pathological microalbuminuria in children with AD seems to be relevant. It seems important to systematically look for personal and familial vascular risk factors in children with AD, to ensure optimal prevention of common vascular diseases.

Statement of Human and Animal Rights

All procedures followed were in accordance with the ethical standards of the responsible committee on

human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008.

Statement of Informed Consent

Informed consent was obtained from all patients for being included in the study.

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Hand eczema and patch testing – A clinico-allergiological study

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ABSTRACT

Background: Hand eczema is a common and distressing condition. Most of the cases of hand eczema have a multifactorial etiology. Most of the cases of hand eczema are irritant contact dermatitis due to irritants like soaps and detergents, but a large number of cases occur due to contact allergy to specific substances. Patch testing is an important diagnostic tool for identification of the probable allergens responsible for the eczema. **Objectives:** The aim of this study was to study the patterns of hand eczema and to identify the most common allergens causing dermatitis using patch testing. **Patients and methods:** Thirty consecutive patients (M: F=11: 19) with hand eczema were examined, detailed history was taken, and the pattern of dermatitis was noted. All of them were subjected to patch testing using the Indian Standard Series. **Results:** The study included 11 (36.67%) men aged between 23 and 54 years and 19 (63.33%) women aged between 28 and 49 years. The majority of patients were in the 21–50 years age group (86.67%; n=26). The most common occupational group among females was housewives (63.15%, n=12) while among the males the most common occupational group was farm workers (54.54%, n=6). Discoid eczema was the commonest morphological pattern seen in 36.67% (n=11) of cases, followed by hyperkeratotic eczema in 30% (n=9). On performing a patch test, 21 (70%) patients showed positive reaction to one or more allergens. In our study, positivity through patch test was found to be highest for nickel (33.33%), followed by potassium dichromate (28.57%), paraphenylenediamine, parthenium, fragrance mix and cobalt chloride. Potassium dichromate was the most common allergen in males while among females, nickel was the predominant allergen. **Conclusions:** Hand eczema is a common problem predominantly seen in females in the middle age groups. It can be both due to exposure to irritants and due to development of contact sensitivity to various allergens with nickel and chromates being the most commonly implicated agents.

Key words: Eczema; Contact dermatitis; Hand eczema; Occupational dermatoses; Paraphenylenediamine

INTRODUCTION

Hand eczema is a chronic and distressing condition with a point prevalence of 1-5% among adults in the general population. Hand eczema is twice as common in women as in men and is more common among people with some kind of occupational exposure [1]. Hand eczema may be endogenous or exogenous in origin and most of the cases of hand eczema have a multifactorial etiology. Endogenous causes of hand eczema are atopic dermatitis, discoid eczema, hyperkeratotic eczema and pompholyx. The most common external cause of hand eczema is contact with irritant or mild toxic agents

like soaps or detergents. Allergic contact dermatitis of hands is much less common than irritant and occurs only in people who have developed a contact allergy to specific substance such as rubber chemical, nickel or other allergens [2,3]. As clinical differentiation between chronic allergic and irritant hand eczemas is often impossible, patch testing becomes an important diagnostic tool for identification of the probable allergens responsible for the eczema.

The aim of this study was to study the patterns of hand eczema and to identify the most common allergens causing dermatitis using patch testing.

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MATERIALS AND METHODS

Thirty consecutive patients with hand eczema were included in the study after taking an informed consent. Pregnant or lactating women were excluded. Patients having acute dermatitis were patch tested after control of their dermatitis, when they were off systemic corticosteroids, or the dose of prednisolone was less than 20 mg/day. Details about age, sex, personal or family history of atopy (nasobronchial allergy, asthma, and childhood eczema), use of various products and its duration, onset, duration, and distribution of dermatitis were noted. The enrolled patients were patch tested by using the Finnchambers method with the Indian Standard Series recommended by the Contact Dermatitis and Occupational Dermatoses Forum of India (Table 1). Patches were applied on the upper back and the patients were asked to return for results after 48 h (day 2) and 72 h (day 3). The results were graded according to the International Contact Dermatitis Research Group criteria [4]. Only reactions persisting on day 3 were considered positive for the final analysis. Relevance of a positive patch test result was determined clinically.

Side effects such as adhesive tape reaction, discomfort and itching, flare of dermatitis, angry back phenomenon, active sensitization, and pigment alteration at test site, when they occurred, were recorded. A patch test for the suspected cosmetic agent itself and the photopatch test were not carried out.

RESULTS

The study included 11 (36.67%) men aged between 23 and 54 years and 19 (63.33%) women aged between 28 and 49 years. The majority of patients were in the 21–50 years age group (86.67%; n=26). The total duration of dermatitis was less than 1 year in 21 (70%) patients, 1–5 years in six (20%) patients, and more than 5 years in three (10%) patients. The minimum duration was 1 month and the maximum duration was 6 years, and the mean duration was 19 months. Eight (26.67%) patients had a history of atopy. The most common occupational group among females was housewives (63.15%, n=12) followed by students (21.05%, n=4) while among the males the most common occupational group was farm workers (54.54%, n=6), followed by office workers (27.27%,

Table 1: Etiological profile of various allergens among patients

Allergens	Number (%)		
	Male	Female	Total
1/Vaseline	-	-	-
2/Black rubber Mix	-	-	-
3/Potassium dichromate	5 (45.45)	1 (9.09)	6 (28.57)
4/Colophony	-	1 (5.26)	1 (4.76)
5/Formaldehyde	-	-	-
6/Nickel sulphate	1 (9.09)	6 (31.57)	7 (33.33)
7/Cobalt chloride	1 (9.09)	1 (5.26)	2 (9.52)
8/Mercaptobenzothiazole	-	-	-
9/Fragrance mix	-	2 (10.52)	2 (9.52)
10/Balsam of peru	-	-	-
11/Paraphenylenediamine	1 (9.09)	2 (10.52)	3 (14.29)
12/Epoxy resin	-	-	-
13/Thiuram mix	-	-	-
14/Neomycin sulphate	-	-	-
15/Benzocaine	-	-	-
16/Nitrofurazone	-	-	-
17/Parthenium	1 (9.09)	1 (5.26)	2 (9.52)
18/Chlorocresol	-	-	-
19/Wool alcohol	-	-	-
20/Parabens mix	-	-	-
Total	9	14	23

n=3). The disease was bilateral in 86.67% patients (n=26). Palms were involved in 46.67% (n=14) patients, fingers in 30% (n=9) patients, dorsa of hands in 16.67% (n=5) patients and whole hand in 6.67% (n=2) patients. Scaling was the most common presentation seen in 70% (n=21) patients followed by erythema, fissuring, hyperpigmentation, papules, papulovesicles, vesicles and oozing. Discoid eczema was the commonest morphological pattern seen in 36.67% (n=11) of cases, followed by hyperkeratotic eczema in 30% (n=9), palmar and vesicular palmar eczema in 13.33% each (n=4) and fingertip eczema in 6.67% (n=2) cases.

On performing a patch test, 21 (70%) patients showed positive reaction to one or more allergens, thus confirming the diagnosis of allergic contact dermatitis. Eighteen patients showed patch test positivity to one allergen while the remaining patients had sensitivity to two or more allergens on patch testing. The etiological allergens in the study population are described in Table 1. Potassium dichromate was the most common allergen in males while among females, nickel was the predominant allergen.

DISCUSSION

Hand eczema is one of the most common dermatological disorders encountered in dermatological practice.

Irritants and contact allergens are the major etiological agents in hand eczema and they frequently co-exist. In most of the cases it is not possible to identify the cause as irritant or allergic. So, patch testing becomes an important diagnostic tool for identification of the allergen or allergens responsible for eczema [5,6].

Hand eczema affects both the sexes but is predominant among the females as reported in various studies [7]. Our study included 11 (36.67%) men aged between 23 and 54 years and 19 (63.33%) women aged between 28 and 49 years. This is probably due to increased exposure of females to wet work, soaps and detergents while washing and to vegetables while cutting and cooking. Occupation has significant bearing on hand eczema because of exposure to various allergens at workplace. The most common occupational group among females was housewives (63.15%, n=12) while among the males the most common occupational group was farm workers (54.54%, n=6). This observation is in agreement with the study conducted on hand eczema by Kishore et al who also found housewives to be the most common occupational group in females and skilled and semiskilled workers among males [8]. There are several types of hand eczema with a distinctive appearance of which the cause is unknown. Discoid eczema was the commonest morphological pattern seen in our study, followed by hyperkeratotic eczema palmar and vesicular palmar eczema. Laxmisha et al, studied 36 cases of hand eczema and found fissuring in 19 cases, hyperkeratotic type in 4 cases, vesicular type in 3 cases, and pompholyx in 1 case [5].

The gold standard for diagnosing allergic contact dermatitis, a type IV delayed hypersensitivity reaction, is patch testing. The basis of the testing is to elicit an immune response by challenging already sensitised persons to defined amounts of allergen and assessing the degree of response. Patch test positivity in the present study was found to be 70% to one or more allergens. Patch test positivity in various studies on hand eczema has been found to vary from 46-82% [5-9]. In our study, positivity through patch test was found to be highest for nickel (33.33%), followed by potassium dichromate (28.57%), paraphenylenediamine, parthenium, fragrance mix and cobalt chloride. In males, potassium dichromate turned out to be most common allergen with 45.45% cases while in females the most common allergen was nickel sulphate with 31.57% cases. This is comparable to a study by Kishore et al, where potassium dichromate was the commonest sensitizer testing positive in 26% of patients while nickel was the

next common allergen testing positive in 18% of the patients [8].

Chromates are present in cements, leather, matches, bleaches, yellow paints, varnishes. Western countries have reported a sharp decline in chromate positivity since the introduction of ferrous sulphate in cement, which converts the easily absorbable hexavalent chromium to the less sensitizing trivalent form. In the present study, potassium dichromate was found to be the commonest allergen positive in masons and labourers. The results of our series are in league with the study conducted by Laxmisha et al in which potassium dichromate was the most common allergen [8]. Patients in our study had significant occupational exposure to chromates, thereby increasing the risk of contact sensitivity to chromates, which could explain the high number of positive patch test reactions to potassium dichromate noted by us. Positive reactions to chromate in metal workers and construction workers was also observed by Ni et al in their study of hand eczema [10]. Nickel is another common sensitizer, perhaps due to its widespread use in imitation jewellery, watches, buttons, zippers, rings, doorknobs, batteries, metal-cutting fluids, coins, orthopaedic plates, keys, spectacle frames and kitchenware [8]. Systemic exposure can take place from diet. The commonest clinical presentation of hand eczema induced by ingested nickel is 'dyshidrotic,' vesicular eczema. Women are more commonly affected and have an earlier age of onset than men [11]. In our study nickel sulphate turned out to be the most common sensitizer in women while no men were found to be sensitive to this metal.

Limitations

Hand eczema can occur due to a wide variety of chemicals other than those available in the Standard Series, which could have led to contact dermatitis. Unavailability of these allergens, unavailability of the photopatch testing, the small number of patients and not testing with patients' own cosmetics may have resulted in our missing some cases of contact dermatitis.

CONCLUSIONS

Hand eczema is a common problem predominantly seen in females in the middle age groups. It can be both due to exposure to irritants and due to development of contact sensitivity to various allergens present in different substances with nickel and chromates being

the most commonly implicated agents. Patch test is an important tool in determining the causative allergens, which will be of help to the patients to avoid the allergens and better management of the disease.

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Subclinical systemic lymphedema and its progression

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ABSTRACT

Lymphedema is a condition stemming from the accumulation of macromolecules in the interstitial space that leads to the retention of fluids. Patient seven-year old male patient with a diagnosis since birth of congenital primary lymphedema in the right lower leg underwent treatments over the years, but the condition progressed to elephantiasis. The patient sought the Godoy Clinic and was submitted to the intensive Godoy Method, nearly reaching normalization. Bioimpedance analysis was performed at the time and continued as a routine monitoring practice over the years, revealing a progression to subclinical systemic lymphedema. The increase in weight and the body mass index (BMI) over the years was accompanied by an increase in water, leading to the accumulation of total body water. An increase in weight in patients with lymphedema can evolve to subclinical systemic lymphedema, further aggravating the primary lymphedema.

Key words: Lymphedema; Subclinical; Elephantiasis; Treatment; Evolution

INTRODUCTION

Lymphedema is a condition stemming from the accumulation of macromolecules in the interstitial space that leads to the retention of fluids. This condition is divided into three clinical stages. In stage I, the patient awakens without edema, but swelling develops throughout the day. In stage II, the patient awakens with edema, which worsens throughout the day. Stage III is the aggravation of stage II and is also known as elephantiasis [1,2].

Godoy & Godoy developed a new concept in the treatment of lymphedema that enables normalization or near normalization [2]. However, different factors can contribute to the aggravation of lymphedema, such as erysipelas, trauma and radiotherapy. Obesity has also been associated with lower limb lymphedema and is therefore yet another aggravating factor [3].

Animal studies have demonstrated that the increase in weight in obesity can trigger changes in the lymphatic pumping mechanism, capillary permeability and the immune system [3,4]. Bioimpedance analysis enables the determination of total intracellular and extracellular water as well as water in isolated extremities and the trunk and has demonstrated an increase in total body water with the progression of lymphedema [5].

This paper reports the evolution of bioimpedance findings with the increase in weight in a patient throughout a five-year period.

CASE REPORT

A 27-seven-year old male patient with a diagnosis since birth of congenital primary lymphedema in the right lower leg underwent treatments over the years,

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but the condition progressed to elephantiasis. The patient sought the Godoy Clinic and was submitted to the intensive Godoy Method, nearly reaching normalization (Figs. 1a and 1b). Bioimpedance analysis was performed at the time and continued as a routine monitoring practice over the years, revealing a progression to subclinical systemic lymphedema. The increase in weight and the body mass index (BMI) over the years was accompanied by an increase in water, leading to the accumulation of total body water. However, the extracellular water/total body water ratio remained within the limits of normality, except in the leg with lymphedema (Table I).

DISCUSSION

The present study shows the progression in the accumulation of water in the entire body with the increase in BMI in a patient who had elephantiasis treated six years ago and remained in clinical stage II of lymphedema. With the increase in weight, an increase occurred in the amount of fluids accumulated in all limbs and the trunk, characterizing what we denominate subclinical systemic lymphedema [6]. At present, bioimpedance analysis has only detected lymphedema in the right leg, which corresponds to clinical stage II.

This study demonstrates that obesity is an aggravating factor of generalized edema, which was congenital primary lymphedema of the right lower limb in the present case. In clinical practice, we noticed that the increase in weight led to the emergence of lymphedema in the lower limb (determined by bioimpedance analysis) stemming from gravitational pressure, which progressed first to the trunk and then to the upper limbs.

In animal studies, an increase in BMI causes damage to the lymphatic pumping mechanism, changes in capillary permeability, an inflammatory process and changes in the immune defense system. These findings are compatible with those seen in humans with the increase in weight. A case study reports an association between lower limb lymphedema and generalized lymphedema.

The present study shows that these changes are systemic rather than localized, with generalized edema accompanying the increase in weight. Therefore, this study makes a novel contribution to the treatment of obesity and lymphedema.



Figure 1: (a and b) Initial treatment and after with nearly normalization.

Table I: Evolution of total intracellular and extracellular water, water in limbs and trunk, and extracellular water (ECW)/total body water (TBW) ratio over five-year period

Age	27.0	28.0	29.0	30.0	31.0	31.0
Height (kg)	93.9	98.4	101.9	103.4	106.8	110.8
Intracellular-water	27.1	28.5	29.0	30.7	30.4	32.1
Intracellular-Normal	29.0	29.0	29.0	29.0	29.0	29.0
Extracellular water	17.3	18.2	18.2	18.9	18.0	19.9
Extracellular-Normal	17.8	17.8	17.8	17.8	17.8	17.8
BMI	30.3	31.8	32.9	33.4	34.5	35.8
Water-R-arm	2.49	2.60	2.65	2.81	2.80	2.95
Water-L-arm	2.39	2.59	2.61	2.77	2.78	2.90
Water-Arm-Normal	2.79	2.79	2.79	2.79	2.79	2.79
Water-R-Leg	9.54	9.17	10.19	10.32	10.41	11.09
Water-L-Leg	7.86	8.36	8.62	8.59	8.48	8.89
Water-Leg-Normal	7.77	7.77	7.77	7.77	7.77	7.77
ECW/TBW-total	0.390	0.390	0.387	0.381	0.382	0.384
ECW/TBW-Normal	0.390	0.390	0.390	0.390	0.390	0.390
ECW/TBW-R-Arm	0.370	0.374	0.365	0.367	0.367	0.365
ECW/TBW-L- total	0.372	0.375	0.365	0.367	0.367	0.365
ECW/TBW-A-Normal	0.39	0.390	0.390	0.390	0.390	0.390
ECW/TBW-Trunk	0.387	0.388	0.384	0.378	0.379	0.380
ECW/TBW-Trunk-Normal	0.39	0.390	0.390	0.390	0.390	0.390
ECW/TBW-R-Leg	0.416	0.405	0.410	0.405	0.408	0.414
ECW/TBW-L-Leg	0.377	0.390	0.377	0.367	0.366	0.366
ECW/TBW-leg-Normal	0.39	0.390	0.390	0.390	0.390	0.390

CONCLUSION

An increase in weight in patients with lymphedema can evolve to subclinical systemic lymphedema, further aggravating the primary lymphedema.

Consent

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

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Helical root chondrocutaneous composite graft for nasal reconstruction: Two case reports

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ABSTRACT

Traumatic loss of any aesthetic subunit of the nose poses a reconstructive challenge for the plastic surgeon. Different surgical techniques are discussed in the literature, ranging from primary closure to free flap reconstruction for complex defects. Due to their cylindrical layered morphology and peripheral location; columella and alar margin are difficult to reconstruct. The Helical root chondrocutaneous composite graft is a single-stage procedure that can be used to restore the lost part of these aesthetic subunit of the nose. We present 2 cases, the first case is about a 3 year old child with prolonged use of nasal CPAP in the NICU resulted in columellar necrosis, and the second case for a 28 year old female patient with history of a childhood post nasal trauma alar rim defect, both cases were reconstructed successfully with helical root chondrocutaneous composite grafts. We provide a thorough explanation of the composite graft design and operative technique. Helical root chondrocutaneous composite graft can produce desirable aesthetic outcomes with minimal donor site morbidity and should be considered in patients presenting with traumatic or iatrogenic loss of the columella and alar margin.

Key words: Columella necrosis; Alar defect; Composite graft; Chondrocutaneous graft

INTRODUCTION

The fragile alar rims and columella are complex structures in which specialized and supportive skin ensures the competence of the external valves and the patency of the inlets to the nasal airways, causative factors for their loss can be post traumatic, post excisional (malignancy) and post infection.

From reconstructive standpoint; alar margin and columellar defects are more distinct than other nasal subunits by their peripheral location and cylindrical layered morphology, it's usually difficult to ensure a good aesthetic outcome provided that most of reconstructive options involve scarring in a visible area of the face.

The first description of composite grafts for nasal reconstruction dates back to Konig in 1902.

Composite grafts for nasal reconstruction have been described in many literatures especially it is used as one stage reconstruction.

CASE REPORTS

The first case is about a male child presented to our clinic with prolonged use of nasal CPAP in the NICU during neonatal period resulted in columellar necrosis (Fig. 1).

On examination there was total loss of columella and anterior nasal septum.

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Looking at the site and size of the defect, a plan was made to reconstruct the defect in a single stage operation, using a composite chondrocutaneous graft taken from the ear root of the helix (Figs. 2a – 2c).

The composite graft was sutured to recipient defect after recipient bed preparation, donor site was closed primarily.

The take of composite graft was adequate with good correction of defect and donor site healed completely with minimal donor site scarring (Figs. 3 and 3b).

The second case is 28 years old female patient with history of a childhood post nasal trauma left alar rim defect (Figs. 4a – 4c).

Also planning was to restore alar rim margin using chondrocutaneous composite graft from ear,root of the helix (Figs. 5a – 5c).

The take of composite graft was excellent and restoration of alar rim with good contour, color and texture and the donor site healed with no significant scarring (Figs 6a and 6b).

DISCUSSION

The nose is corner stone in facial appearance and whole body image, restoration of near normal appearance and function are the definitive goals for reconstruction.

The nose is composed of nine aesthetic subunits: two side walls, two ala, two soft triangles, tip, dorsum and columella. From reconstruction stand point this is very important from aesthetic subunit restoration principle [1-4].

Successful surgical strategies for nasal reconstruction are based on accurate analysis of the defect taking into consideration: the size, contour, shape, layers involved and the aesthetic subunits affected.

The reconstructive ladder in nasal defects ranges from: healing by secondary intention and primary closure to skin grafts to local and regional flaps. The peripheral

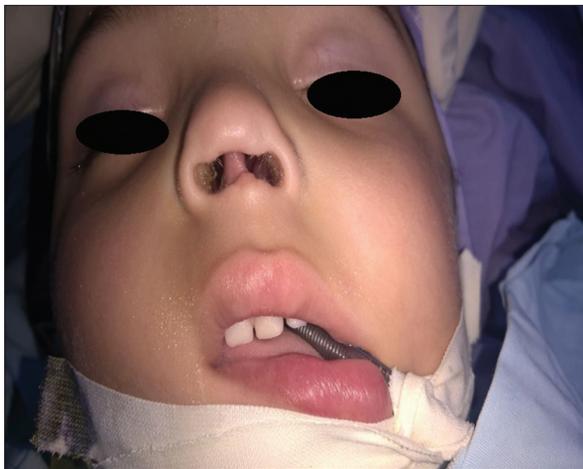


Figure 1: Columellar and anterior septal defect.



Figure 3: Five months follow up with successful aesthetic restoration of columella and minimal donor site morbidity.



Figure 2: Composite graft from root of helix harvest and inset.



Figure 4: Post traumatic left alar rim defect.

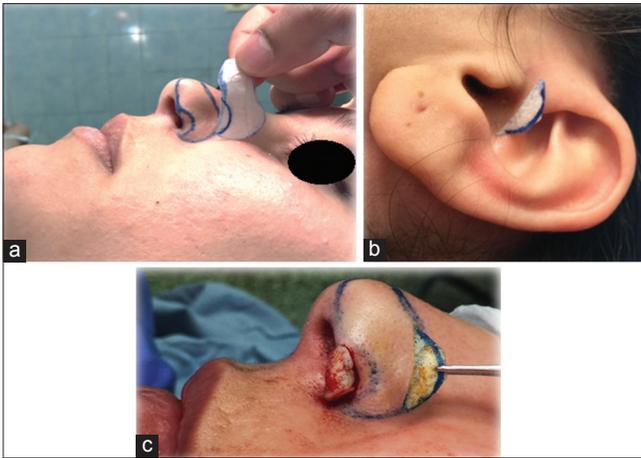


Figure 5: Using paper template from the normal ala, defect was analyzed and restored with composite graft after recipient bed preparation.

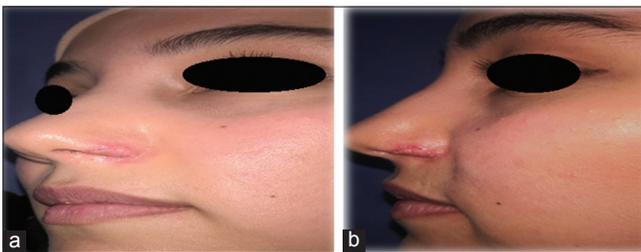


Figure 6: One month follow up showing excellent uptake of the composite graft.

location of columella and alar rim with its cylindrical layered topography and thin skin carry further burden to reconstruction [1-6].

For reconstruction of nasal columella and ala many literatures described loco regional flaps: nasal sulcus flap, forehead flap and nasolabial flaps, but all will add more scars to face and undesirable aesthetic outcomes.

Composite chondrocutaneous graft was described in many literatures, grafts were harvested from helix margin concha which gives traditional wedge shaped of two skin layers separated by cartilage, in our cases report we harvested composite graft from root of helix which gives composite graft of cartilage covered by skin besides it gives better aesthetic outcomes and hidden donor site scar.

Many authors recommended that composite graft size should not exceed 10 mm in diameter for graft survival as if it exceeds 10 mm success rate decrease, composite graft with more than 15 mm chance of survival less than

50 %, in our cases all composite graft size was less than 10 mm [1,2,3,4, 7-9].

CONCLUSION

Helical root chondrocutaneous composite grafts can produce desirable aesthetic outcomes and should be considered in patients presenting with traumatic or iatrogenic loss of columella and alar rim.

Statement of Human and Animal Rights

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

Statement of Informed Consent

Informed consent was obtained from all patients for being included in the study.

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Median canaliform dystrophy of Heller

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ABSTRACT

Median Canaliform Dystrophy of Heller is characterized by midline longitudinal furrow with multiple transverse parallel lines. Repetitive trauma to the nail plate and cuticle may be responsible for some cases, however some case reports have suggested familial occurrence and use of oral retinoids in its causation. We present a special case of Median Canaliform Dystrophy of Heller in middle age Chinese male and discuss the possibility cause in our case. Median Canaliform Dystrophy of Heller case occurred in Chinese patient that has no special related diseases and it resolved simultaneously and happened again.

Key words: Median nail dystrophy; Nail plate; Cuticle

INTRODUCTION

Median Canaliform Dystrophy of Heller or Onychodystrophia Mediana Canaliformis is a relatively rare albeit striking habit tic deformity of thumb nails characterized by midline longitudinal furrow with multiple transverse parallel lines. Although the proposed etiopathogenesis is repetitive trauma in the form of pushing down the cuticle and proximal nail fold, in majority of cases the cause may be obscure. Some case reports have suggested familial occurrence and use of oral retinoids in its causation [1]. Histopathology classically shows parakeratosis, accumulation of melanin within and between the nail bed keratinocytes [2]. Treatment is often prolonged and unsatisfactory, though topical agents like 0.1% tacrolimus have been used successfully [1]. Psychiatric opinion should be taken when associated with the depressive, obsessive-compulsive, or impulse-control disorder. We report a case of 35-year-old Chinese male diagnosed as median nail dystrophy.

CASE REPORT

A 35 year old Chinese male is presented to Dermatology Outpatient Department with complaints of lesion over his thumb nail since 6 months. The patient denied

history of intentional pushing down of cuticular portion of proximal nail fold. The patient had such condition about one year ago and it resolved simultaneously within several months, and had it again. No history of taking oral retinoids or other medications, or history of contact with irritants or allergens was present. The patient didn't have any family history of nail disorders. On examination, there was median split of right thumb nail with transverse furrows extending from longitudinal split (Fig. 1). Rest other finger and toe nails were normal. Systemic examination was unremarkable. The patient refused biopsy. Diagnosis of median nail dystrophy was made on a clinical basis. The patient is still in follow up.

Discussion Median canaliform dystrophy of Heller, also known as dystrophia unguis mediana canaliformis, solenonychia, and nevus striatus unguis, is a dystrophic condition of the nail in which longitudinal splitting occurs [3]. It is almost exclusively seen on the thumbs, often bilaterally. It is characterized by an inverted fir tree-like split or canaliform in the nail plate [3]. The exact etiology of this intriguing condition is yet to be elucidated. However, subungual skin tumors such as glomus tumors, myxoid tumors, have also been described to cause longitudinal grooving, lifting of the nail plate from the bed resulting in a tube-like

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Figure 1: Median split of right thumb nail with transverse furrows extending from longitudinal split.

structure (solenos) distal to it [3]. To our knowledge, only 6 cases have been reported in literature. 4 reported cases occurred in adults and 2 reported case occurred in children. 5 reported cases are associated with trauma. Wu et al (2009) reported a 56 year old female presented with median canaliform dystrophy of Heller with associated swan neck deformity. She had a history of repeated minor injuries over both hands. She didn't have a history of taking any oral medication or have contact with any irritants and no familial history [3]. Madke et al reported a 25 year old female presented with backwardly-angled ridges on her thumbnails resembling a fir tree. She denied a history of repetitive trauma. The patient didn't report any history of contact with known allergens and irritants. She didn't have any family history of nail disorder. The patient was then prescribed topical tazarotene 0.05% ointment to be applied at bedtime and was asked to follow-up [4]. Avhad et al reported a 5 year old Indian boy was presented with single dystrophic thumb nail. He had history of constant biting of thumb nails and was also diagnosed to have attention deficit hyperactive disorder (ADHD) [5]. Kola et al reported a 19 year old female presented with median nail dystrophy. History of biting of thumb nails was present. No history of use of oral retinoids or other medications, or history of contact with irritants or allergens was present. He denied a family history. The patient then was prescribed with 0.1% tacrolimus ointment topically at night and showed some improvement. The patient is still in follow up.[2] Pathania et al reported a 22 year old

Indian male was diagnosed to have Median Canaliform Dystrophy of Heller that affecting both great toe nails and great thumb nails. He was then prescribed topical 0.1% Tacrolimus ointment and Oral Fluoxetine at the dose of 50 mg daily. However, the case was lost to follow up after putting the patient first review [1]. Damevska et al reported 11 year old girl who developed canaliform dystrophy and long-term hypopigmentation following cryotherapy of warts on proximal nail folds [6]. In our patient described herein, this case is special because self-inflicting nail trauma involving manipulation of cuticular portion of proximal nail fold and familial were not present. As the medication history was also absent, and it could resolve simultaneously, also tumor is not considered. The possibility cause in this case remains idiopathic. The patient is still in follow-up.

CONCLUSION

In closing, we report a Median Canaliform Dystrophy of Heller case occurred in Chinese patient that has no special related diseases and it resolved simultaneously and happened again.

Consent

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

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Onychomycosis due to mixed infection with non-dermatophyte molds and yeasts

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ABSTRACT

Onychomycosis is the fungal infection of nails which affects 5.5% of the general population. Etiologic agents include dermatophytes, non-dermatophyte molds, and yeasts. The infection usually occurs due to dermatophytes. However, non-dermatophyte molds and yeasts have an increasing role in the development of onychomycosis. Detecting causative agent is crucial for the appropriate therapy, as non-dermatophytic molds and yeasts are usually resistant to classical antifungal agents which are used in the treatment of onychomycosis. Hereby, we report a 39-year-old Caucasian male patient with onychomycosis of the great toenails caused by *Aspergillus niger complex*, *Chaetomium globosum*, *Cladosporium* species, *Candida* species, and onychomycosis of the left thumbnail due to *Aspergillus niger*, *Chaetomium globosum*, *Cladosporium* species and *Candida lambica*.

Key words: Nail fungus; Non-dermatophyte molds; Onychomycosis; Yeasts

INTRODUCTION

Onychomycosis is the fungal infection of nails caused by dermatophytes, non-dermatophyte molds, and yeasts. Onychomycosis is the most common nail disease with the prevalence of 5.5% all over the world [1]. It usually affects toenails of adults [2]. Onychomycosis can lead to pain, paresthesia, difficulties in daily activities, impaired social interactions, and low self-esteem [1,3]. Trauma, tinea pedis, advanced age, diabetes, psoriasis, malignancy and immunosuppression are regarded as risk factors in the etiology of onychomycosis [1].

Dermatophytes, especially *T. rubrum* and *T. mentagrophytes* are regarded as the most common causative agents in onychomycosis. Non-dermatophyte molds including *Scopulariopsis brevicaulis*, *Aspergillus spp*, *Acremonium*, *Fusarium spp*, *Alternaria alternate*, and *Neosectalidium* are detected in approximately 20% of the patients. Yeasts (*Candida spp.*) are responsible for 10%-20% of cases with onychomycosis [1]. Mixed infections

with dermatophytes and non-dermatophyte molds in onychomycosis have been rarely reported [4].

However, an increasing role of non-dermatophyte molds and yeasts in onychomycosis has been described [5,6]. Furthermore, it has been suggested that non-dermatophytic molds and yeasts are significantly more prevalent causative agents in onychomycosis than dermatophytes [6]. Ovcina-Kurtovic reported *Candida albicans* as the most common fungus isolated from psoriatic patients with nail involvement [7].

Antifungal drug resistance is becoming a healthcare problem as a result of their wide use and availability [8]. Patients with onychomycosis due to non-dermatophytic molds or yeasts may not respond to systemic antifungals like itraconazole, fluconazole and griseofulvin which are frequently recommended in the treatment of onychomycosis [5]. Identification of the causative agent with mycological examination is needed for the appropriate treatment and good clinical outcome [6].

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CASE REPORT

A 39-year-old Caucasian male patient presented with a 5-year history of discoloration and thickening of the toenails. The patient stated that the symptoms started as a yellow discoloration and thickening under the tip of the right great toenail. Then it spread to the rest of his toenails and left thumbnail gradually. The patient had pain and tenderness in the great toenails while wearing shoes. He did not receive any medication previously. The past medical history was remarkable for chronic peripheral venous insufficiency. He has been wearing compression stockings for the last two years. The family history was unremarkable. The patient denied nail injury, getting pedicure at a nail salon, walking barefoot in public areas like swimming pool, sauna or using an immunosuppressive agent.

Physical examination revealed a squamous plaque on the extensor surface of the left thumb; hyperkeratotic, yellow groove in the midline of the left thumbnail extending from proximal nail fold to distal edge; onycholysis, and white, opaque, friable lesions which created a linear plaque on the left thumbnail (Fig. 1). Moreover, subungual hyperkeratosis, yellow discoloration and onycholysis were observed in all toenails (Figs. 2-4).

The laboratory tests including complete blood count, fasting blood glucose, creatinine, total cholesterol, triglyceride, alanine aminotransferase, aspartate aminotransferase, ferritin, folate, vitamin B12, 25-hydroxyvitamin D, zinc and thyroid stimulating hormone levels were all within normal limits.

Mycological examination was performed using conventional methods and matrix assisted laser desorption ionization time of flight mass spectrometry (MALDI-TOF MS). The specimens were obtained from scrapings of left thumbnail and from both great toenails. *Aspergillus niger* complex, *Chaetomium globosum*, *Cladosporium* species and *Candida* species were identified from the scrapings of bilateral great toenails. *Aspergillus niger*, *Chaetomium globosum*, *Cladosporium sp.* and *Candida lambica* were detected from the specimens obtained from the left thumbnail. Thus, the diagnosis of onychomycosis due to mixed infection with non-dermatophyte molds and yeasts was made based on clinical findings and MALDI-TOF MS technique.

The patient was advised to get a skin biopsy from the squamous plaque on the left thumb to rule out psoriasis. However, the patient refused the biopsy. He



Figure 1: Onycholysis, white, friable lesions, and hyperkeratotic, yellow groove of the left thumbnail. *Aspergillus niger*, *Chaetomium globosum*, *Cladosporium sp.* and *Candida lambica* were detected from the specimens obtained from the left thumbnail.



Figure 2: Subungual hyperkeratosis, yellow discoloration and onycholysis of the toenails.



Figure 3: Total dystrophic onychomycosis of the toenails.



Figure 4: Closer view of the great toenails. *Aspergillus niger* complex, *Chaetomium globosum*, *Cladosporium* species and *Candida* species were identified from the scrapings of great toenails.

claimed that the lesion occurred as a result of repetitive picking of the skin due to psychological stress.

DISCUSSION

Onychomycosis can clinically present with subungual hyperkeratosis, onycholysis, melanonychia, and brown, yellow, orange or white discoloration of the nail plate and friable nails. Onychomycosis due to non-dermatophytes has been associated with a marked periungual inflammation [9]. The non-dermatophyte molds including *Scopulariopsis brevicaulis*, *Aspergillus spp.*, *Fusarium spp.*, *Acremonium spp.*, *Alternaria spp.* and *Neoscytalidium spp.* may be the primary pathogens in the development of onychomycosis. Moreover, they may play role as contaminant agents and secondary pathogens. Yeasts like *Candida albicans* and *Candida parapsilosis* cause nail infections only in patients with predisposing factors such as immunosuppression and diabetes [9].

Clinical diagnosis should be confirmed with a mycological investigation, since the treatment plan depends on the species of fungi and number of affected nails [9]. Treatment of patients with nail infections caused by non-dermatophyte organisms like *Fusarium* is usually difficult [10].

Treatment options include systemic and topical antifungal agents, chemical or surgical removal of the infected nail, and laser therapy. Oral antifungals may have side effects such as liver damage, and they

may cause unwanted drug interactions especially in elderly. Recurrence rate of fungal nail infection is high especially in immunocompromised patients. In addition, diabetes and genetic predisposition to onychomycosis increase the rate of recurrence [10].

Hereby, we report a patient with onychomycosis of the left thumbnail and toenails due to mixed infection of non-dermatophyte molds and yeasts including *Chaetomium globosum*, *Cladosporium* species which are rare in onychomycosis etiology. Detecting the causative agent is crucial for the appropriate therapy, as non-dermatophytic molds and yeasts are usually resistant to classical antifungal agents.

Consent

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

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Staphylococcal scalded skin syndrome: A case report

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ABSTRACT

Staphylococcal Scalded Skin Syndrome (SSSS) is an extensive and exfoliative skin disease which is caused by staphylococcal epidermolytic toxin produced by *Staphylococcus aureus*. A 20-month-old female infant presented with a desquamating rash with flaccid blisters and fever. Skin biopsy revealed superficial intraepidermal cleavage under the stratum corneum. The diagnosis of scalded skin syndrome was assessed and the patient was treated with antistaphylococcal antibiotics with a complete recovery of her skin lesions. SSSS is a pediatric emergency, representing a fatal condition in neonates and a challenge in diagnostic and treatment. Therefore, early diagnosis, prompt treatment, and following strict hygiene measures are imperative and can prevent mortality.

Key words: Scalded skin syndrome; Desquamation; *Staphylococcus aureus*; Exfoliative toxins

INTRODUCTION

Staphylococcal Scalded Skin Syndrome (SSSS) is an extensive and exfoliative skin disease which is caused by Staphylococcal epidermolytic toxin produced by *Staphylococcus aureus*. It occurs in newborns, children less than 5 years of life, and adults with various comorbidities [1,2]. We report a new case of SSSS in a 20-month-old female infant.

CASE REPORT

A 20-month-old female infant presented with fever and a generalized rash since 4 days. There was no family history of similar skin lesions. On physical examination, skin was tender with diffuse erythema and she had superficial erosions and peeling on her trunk (Fig. 1). The superficial erosions started on the axillary region extending to the trunk and limbs with sparing of the mucosa. There were flaccid blisters that ruptured leading to superficial erosions with exfoliation. Nikolsky's sign was positive on the trunk. Other body system examinations were normal.

A skin biopsy was performed on her trunk showing superficial intraepidermal split into the granular layer associated with little inflammatory infiltrate in the superficial dermal layer (Fig. 2). Hence, the diagnosis of SSSS was assessed and the patient was treated with antistaphylococcal antibiotics, administered intravenously 100 mg/kg/day for 10 days with a complete recovery of her skin rash.

Prior to the study, patient gave written consent to the examination and biopsy after having been informed about the procedure.

DISCUSSION

Staphylococcal scalded skin syndrome (SSSS), also known as Ritter's disease, is a rare skin infection seen in neonatal period and early childhood as in our patient. It is a potentially life-threatening skin disorder caused by certain strains of *staphylococcus aureus* that release serine protease exfoliate toxins, exotoxin A (ETA), the most produced one, and the exotoxin B(ETB). These two exfoliative toxins have an affinity to the

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Figure 1: Superficial epidermal peeling on the trunk.

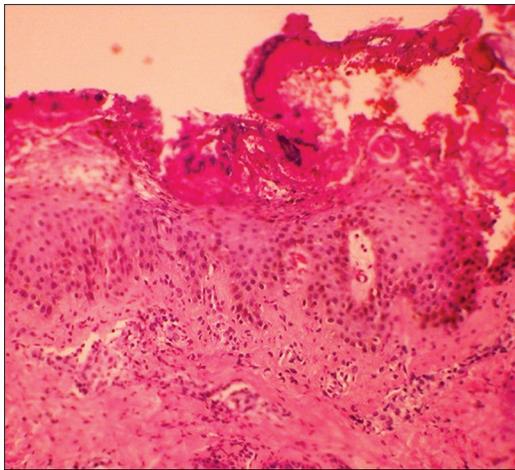


Figure 2: Superficial intraepidermal cleavage under the stratum corneum (HEx100).

glycoprotein, desmoglein-1, present on desmosomes located in the zona granulosa layer of the skin, so they cleave desmosomal cadherins, destroy adhesion between the keratinocytes, leading to epidermolysis and bulla formation [1,2]. Moreover, the lack of protective antibodies to exfoliative toxins and immature renal function, explain the higher risk of SSSS in neonates and children. The diagnosis of SSSS is mainly clinical. It usually presents as generalized erythematous extensive skin lesions associated with the formation of large fragile roofed superficial blisters leading to extended areas of eroded skin, bullae, and desquamation with a scalded appearance especially in friction zones, periorificial crusting, and positive Nikolsky sign [3]. This disease may resemble toxic epidermal necrosis which is a life threatening skin disorder. However, in SSSS there is absence of mucosal involvement as

well as superficial epidermal peeling with absence of necrotic keratinocytes characteristic of toxic epidermal necrosis. It could also resemble pemphigus foliaceus and bullous impetigo (BI) but in BI, the skin lesions are smaller and there is pronounced inflammatory cell infiltrate consisting mostly in neutrophils and the skin [1,3,4]. A prompt administration of intravenous antibiotics mainly antistaphylococcal penicillins generally allows a favorable outcome [5]. Recovery is achieved after 6 to 12 days without scarring. Prognosis is mostly favorable if treatment is begun promptly, and the mortality rate is 4%, due to serious complications like pneumonia, septic arthritis, hypothermia, dehydration, and secondary infections [4].

CONCLUSION

SSSS is a rare cause of skin infection caused by staphylococcus aureus. It is a pediatric emergency, representing a fatal condition in neonate and a challenge in diagnostic and treatment. Therefore, early diagnosis, prompt treatment, and following strict hygiene measures are imperative and can prevent the mortality.

Consent

The examination of the patient was conducted according to the Declaration of Helsinki principles. Written informed consent was obtained from the child's parents for publication of this article.

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Deep chronic cutaneous New World leishmaniasis due to *Leishmania guyanensis* and trichinellosis in a German returnee from Ecuador

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ABSTRACT

In recent years, leishmaniasis has become more common in Germany due to refugees and returnees from endemic areas. However, New World leishmaniasis is less common. We report a 19-year-old female returnee from Ecuador with relapsing deep cutaneous leishmaniasis relapsing after glucantime and unresponsive to paromycine. Species identification by polymerase chain reaction revealed *L. guyanense*. We discuss the molecular background for possible treatment failure to first line therapy. The same patient also had an infestation by trichinellosis.

Infestations and infections need to be considered in returnees from endemic areas. Dermatologists play an important role in clinical diagnosis.

Key words: Travel medicine; Leishmaniasis; Trichinellosis; South America; *Leishmania guyanense*.

INTRODUCTION

Treatment failure and symptomatic relapse are major concerns in New World cutaneous leishmaniasis. Such complications are seen frequently in *Leishmania (L.) guyanensis* infections, in which patients respond variously to first-line treatment and are at higher risk to develop chronic cutaneous leishmaniasis [1].

Leishmaniasis due to *L. guyanensis* has been reported from Argentina [2], Brazil [3], Colombia [4], Ecuador [5], and Peru [6].

The Surveillance of Imported Infections in Germany (SIMPID) Surveillance Network reported 42 cases of leishmaniasis in Germany during the years 2001-2004. Most of them were cutaneous diseases acquired in the Mediterranean countries [7]. Harms et al. (2011) analyzed 23 cases of New World leishmaniasis in returnees from Central or South America to Germany. In all cases *L. braziliensis* had been detected by polymerase chain reaction (PCR) [8].

CASE REPORT

A 19-year-old German Caucasian woman returned from a 12-months stay to Ecuador where she developed ulcero-nodular lesions on the nose and arms. New World leishmaniasis was diagnosed. An intralesional treatment with glucantime once a week was initiated in Ecuador. The lesions disappeared within two months. Once months later, nodular lesions occurred on the left cheek. She also reported diarrhea and temporary muscle pain during her stay in Ecuador.

After return from Ecuador 7 months later, she presented to the clinic for further diagnostics with five deep nodular lesions on the left cheek (Fig. 1). At that timepoint, she had no fever, arthralgia or arthritis, no diarrhea. She suffered from mild fatigue.

Routine laboratory investigations were unremarkable except for revealed increased counts of eosinophils in peripheral blood – 80 Gpt/L (normal range 0-40). Antibodies to leishmania were negative but antibodies

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Figure 1: *Leishmania guyanensis*-infection. Slightly erythematous, deeply seated nodules on the cheek.

against *Trichinella* were positive. A deep skin biopsy was taken for PCR to leishmania species. PCR was positive for *L. guyanensis*.

Diagnostic ultrasound demonstrated normal cervical, slightly enlarged axillary and inguinal lymph nodes, and a mild hepatosplenomegaly. Chest-X-ray was unremarkable.

The diagnoses cutaneous leishmaniasis of the New World and trichinosis were confirmed, although hepatosplenomegaly in conjunction with fever is a symptom of visceral leishmaniasis. However, the patient had no fever, leuko- or lymphopenia, anemia or thrombocytopenia. No circulating antibodies could be detected, and her general health status was good.

We started with topical treatment of leishmaniasis after confirmation of *Leishmania spp.* with paromycin ointment, but it remained unsuccessful. After species identification, we recommended miltefosin 2.5 mg/kg body weight for 28 days.

In addition, albendazole 400 mg twice daily for 10 days was suggested for trichinellosis treatment.

DISCUSSION

In Germany, most cases of imported leishmaniasis belong to the cutaneous and mucocutaneous Old World leishmaniasis, while New World tegumental and visceral types are less common [7-10]. The Institute for Tropical Medicine, Berlin, registered in 2015 16 cases of imported leishmaniasis in Germany including two

New World leishmaniasis infections from Brazil and Peru [11].

Here we report a case of deep cutaneous *L. guyanensis* infection relapsing after glucantime in a German returnee from Ecuador. The treatment failures and relapses with this subtype is a challenge.

L. guyanensis shows a limited response to antimony, since they are capable to detoxify peroxides by trypanothione. Ornithine decarboxylase (ODC) and γ -glutamylcysteine synthetase (GSH1) produce molecules that are direct precursors of trypanothione. Parasites with ODC- or GSH1-overexpression presented an increase of two and four-fold in antimony (III) -resistance index, compared with the wild-type line [12]. Another mechanism involved in antimony resistance are point mutations in the gene encoding aqaglyceroporin 1 [13].

Animal studies revealed that leishmania RNA virus 1 (LRV1), nested within *L. guyanensis* parasites, is able to exacerbate experimental murine leishmaniasis. LRV1 is recognized by the host Toll-like receptor 3 (TLR3) [14]. The virus induces strong interferon type I immune responses that diminish the level of interferon- γ production and release necessary to control leishmania infection [15].

The prevalence of LRV1 in human *L. guyanensis* infection and its relationship to treatment failure and inflammation has been investigated among 75 patients with a diagnosis of primary localized New World cutaneous leishmaniasis. The prevalence of LRV1-positive *L. guyanensis* infection was 58%. All patients infected with LRV1-negative *L. guyanensis* responded to a single or double dose of pentamidine. In contrast, 12 of 44 LRV1-positive patients (27%) presented with persistent infection and symptomatic relapse that required extended therapy and the use of second-line drugs. LRV1 presence was also associated with a significant increase in levels of intralesional inflammation. The LRV1 status in *L. guyanensis* infection is predictive of first-line treatment failure and symptomatic relapse [16]. However, we were not able to analyze our sample for LRV1 presence.

Our patient also suffered from mild trichinellosis. *Trichinella spiralis* and *T. pseudospiralis* occur worldwide. The first one is found in wild and domestic pigs and wild carnivores, the second one infects both mammals and birds. The most important source

of human infection worldwide is the domestic pig. Infection of humans occurs with the ingestion of *Trichinella* larvae that are encysted in muscle tissue and is strongly associated with the consumption of raw or undercooked meat. Diagnostics include clinical symptoms, laboratory findings (eosinophilia, elevated muscle enzymes, and serology), and epidemiological investigations. Treatment options include albendazole or mebendazole monotherapy in case of mild or abortive disease [17].

Porcine trichinellosis in Ecuador is more common in traditional setting in villages compared to farms [18]. In 2015, only 8 cases of imported trichinellosis had been registered by the Robert-Koch-Institute (RKI) in Germany, all of European origin [11].

Consent

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

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Clinical and histological findings of a typical case of angiolymphoid hyperplasia without eosinophilia: a rare and difficult-to-treat disease

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ABSTRACT

Angiolymphoid hyperplasia with eosinophilia (ALHE) is a rare vascular condition characterized by cutaneous papules, plaques or nodules, predominantly located on the head and neck. The main differential diagnosis of ALHE is Kimura's disease. The treatment is a challenge and the gold standard treatment is surgical excision, as relapse is commonly observed. We report a case of a 57-year-old female with ALHE with an unusual clinical improvement at 2-month follow-up after topical corticosteroid without recurrence 12 months after treatment. Due to its rarity, ALHE can be misdiagnosed. In this case, the characteristic lesions in typical location supported our first clinical diagnose, which was confirmed by histological studies. The clinical improvement after topical steroids without recurrence after long period follow-up caught our attention.

Key words: Angiolymphoid hyperplasia; Vascular; Eosinophilia; Kimura's disease.

INTRODUCTION

Angiolymphoid hyperplasia with eosinophilia (ALHE) is a rare, locally proliferative, benign and idiopathic condition that usually affects middle-aged Caucasian women [1]. ALHE pathogenesis remains unknown, but some authors believe that the lesion originates from a vascular tumor or secondary to vascular damage, such as skin trauma, infections (HTLV or herpes virus 8) or hormonal imbalance [2]. ALHE is characterized by cutaneous papules, plaques or nodules, which may be single or clustered, erythematous, violaceous or brown [3]. It is predominantly located on the head and neck [4]. The absence of eosinophilia is not uncommon, but increased serum Immunoglobulin E (Ig E) can be found.

ALHE and Kimura's disease were considered for many years the same entity and to this day they are often confused. Both diseases share common clinical and

histopathological features. However, it is believed that they are two distinct diseases because of their clinical and histopathology differences [4].

The treatment of choice for ALHE is surgical excision, but relapse is common. Cryotherapy, local radiotherapy, topical or intralesional corticosteroids, imiquimod, acitretin, and laser therapy (dye laser, CO₂ laser) are also reported as possible treatments with varying success levels. There are reported cases of spontaneous regression [2].

We report a case of a patient diagnosed with ALHE, without eosinophilia, with good clinical response to topical corticosteroid treatment.

CASE REPORT

A 57-year-old female patient, diagnosed with vitiligo and past history of benign thyroid nodule. She

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complained about pruritic and painful erythematous-violaceous papules located on the tragus (Fig. 1) for 5 years without treatment. She denied any systemic disease or no chronic use of medicaments. Dermatologic examination: achromatic macules on the anterior thorax, forearms and hands, corresponding to the clinical diagnosis of vitiligo. Red-violaceous nodules grouped on tragus and outer auditory channel. The main diagnostic hypothesis was Angiolymphoid Hyperplasia with Eosinophilia, and differential diagnoses were Kimura's Disease and Kaposi's Sarcoma.

A cutaneous lesion biopsy was performed in the lesion on the left tragus and submitted to histopathological examination, which revealed diffuse inflammatory infiltrate with lymphocytes and eosinophils, hyperplasia of the blood vessels and capillaries with prominent endothelium, confirming the diagnosis of Angiolymphoid Hyperplasia with Eosinophilia (Fig. 2).

Laboratory tests did not show eosinophilia or Ig E alterations. She had anti-TPO antibodies positive in high titers, normal thyroid hormones levels, and normal thyroid ultrasonography. Therefore, topical treatment with mometasone furoate cream, once a day for two months was introduced with the aim of relieving symptoms and avoiding surgery.

DISCUSSION

Clinically ALHE present as persistent or recurrent erythematous or hyperpigmented papules or nodules, usually located in the periauricular area and the scalp [1]. In our case, the patient had erythematous-violaceous papules on tragus and outer auditory channel.

The main differential diagnosis of ALHE is Kimura's disease. They seem to be the same disease due to the clinical similarity of cutaneous lesions, however most studies now consider them as two different entities. Clinical differences to distinguish Kimura's disease from ALHE would be a greater diameter of skin lesions, more insidious evolution, and erythematous or purpuric staining of ALHE. This latter characteristic reflects the vascular nature of this process. Kimura's disease presents clinically with one or more subcutaneous nodules, salivary glands involvement, lymph node enlargement and it is commonly associated with peripheral eosinophilia and IgE elevation [2]. In our case, the patient had only cutaneous lesions consistent with the diagnosis of ALHE.

On histopathology ALHE disease usually does not resemble lymphoid tissue, it is primarily a disorder of blood vessels and has structures similar



Figure 1: Erythematous-violaceous papules located on the tragus.

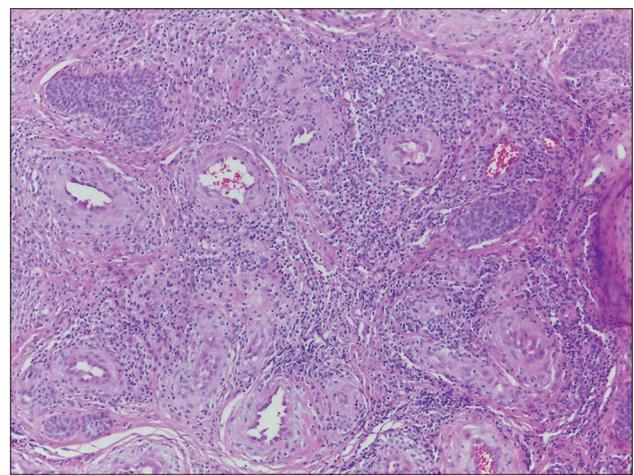


Figure 2: Diffuse inflammatory infiltrate with lymphocytes and eosinophils, hyperplasia of the blood vessels and capillaries with prominent endothelium.



Figure 3: Clinical improvement 12 months after treatment.

to abnormal dilated veins located in the dermis and/or subcutaneous tissue, with abundant mucin deposition on its walls. On the other hand, Kimura's disease is a disorder of lymphoid follicles, which are predominant feature in large quantities [3]. The histopathological examination of our patient revealed diffuse inflammatory infiltrate with lymphocytes and eosinophils, epithelial hyperplasia of the blood vessels and capillaries with prominent endothelium as described for ALHE disease.

The treatment of ALHE disease is always a challenge. The gold standard is surgical excision, but relapse is common. In our patient, we chose mometasone furoate topically applied once a day. A significant clinical improvement was observed at 2- month follow-up treatment with no relapse 12 months after treatment. (Fig. 3).

CONCLUSION

ALHE is a rare difficult-to-treat condition. The characteristics lesions and locations help diagnosis and complementary exams are necessary to exclude other dermatoses. In our case, the rarity, typical clinical and histological findings and the good clinical response to

topical corticosteroid treatment without relapse after a long period follow-up encourage our publication.

Consent

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

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Multiple eccrine spiradenoma in a young Greek patient: case report and review of the literature

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ABSTRACT

Eccrine spiradenoma is an uncommon and benign adnexal tumor characterized by one or more nodules. The present article refers to a young patient with multiple and gradually enlarged nodules which remained undiagnosed and untreated for many years. Lesions were initially considered as multiple leiomyomas whereas skin biopsy revealed the existence of uncommon and benign tumors of eccrine and apocrine glandular adnexa confirming the diagnosis of eccrine spiradenoma. Similarities in clinical appearance and symptoms between tumors may lead to erroneous diagnosis and skin biopsy is an essential tool for differential diagnosis.

Key words: Tumor; Eccrine glands; Pain

INTRODUCTION

Eccrine spiradenoma (ES) represents a rare and benign tumor of sweat glands which was first described in 1956 by Kersting and Helwing. The deep dermal position of the tumor is indicative for its origination from the secretory coil of eccrine glands, even though several lines of evidence also support its possible apocrine differentiation [1,2]. The aim of this article is to present a rare case of a young Greek patient who was diagnosed with multiple ES.

CASE REPORT

A 33-year-old Caucasian woman presented to the outpatient clinic of a dermatological center complaining of multiple and gradually enlarged lesions on the right side of the back. The onset of the lesions was observed at the age of 17 years old. Personal and family medical history was negative for comorbidities except from an excision of a giant cell tumor of tendon sheaths in her right arm one year ago. In addition, anaemia was identified after the performance of blood biochemical test.

Physical examination revealed well defined, round to oval, pink nodules. Their size ranged from 0.5 to 1 cm

and they were arranged in linear pattern. Nodules were painful, tender to palpation, firm and fixed to the skin (Figs. 1 and 2).

Histopathological analysis revealed benign tumors of eccrine and apocrine glandular adnexa of the skin. Morphological features of the tumors were typical to eccrine spiradenoma and nodules were extended to the subcutaneous tissue. Immunohistochemical analysis showed strong expression of cytokeratin 7 (CK7), p40 and p63 and weak expression of the transcriptional factor GATA-3 and carcinoembryonic antigen (CEA). Tumor cells were negative for epithelial membrane antigen (EMA), protein S-100 and gross cystic disease fluid protein 15 (GCDFP-15).

DISCUSSION

ES is an uncommon, benign adnexal tumor characterized by one or more nodules. The tumor has been described as “blue ball in the dermis” because of its blue color, although it might be purple, red, pink or gray [3]. Few clinical variants have been described like solitary ES which is generally accepted as the most common, multiple ES and giant vascular

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Figure 1: Well defined, round to oval, pink nodules, ranging from 0.5 to 1 cm and arranged in linear pattern.



Figure 2: Clinical appearance of lesions after surgical excision.

ES. Review of the literature demonstrates that multiple ES includes less than 2% of all cases and the majority of these tumors are distributed by linear or zosteriform pattern [4]. Pathways underlying the pathogenesis of the tumor are still being elucidated. The potential role of hereditary factors was pointed out by some investigators who suggested autosomal and dominant inheritance but their observations are still not established [5]. Interestingly, trauma has been speculated as precipitating factor leading to the development of ES [6].

Solitary ES occurs more frequently in men, whereas multiple ES appear more commonly in females, with an estimated female to male ratio of 3:1 [7]. The onset of the tumor is typically observed during the second to fourth decade of life. Solitary ES mostly occurs on the upper half of the body, whereas the distribution of multiple ES involves face, neck and lower extremities.

The main symptom and clinical sign is pain and tenderness to palpation respectively [8]. In our patient lesions were initially observed at the second decade of life but they remained undiagnosed and untreated due to their size and lack of symptoms. Their progressive augmentation resulted in pain which motivated the patient to request medical advice.

Diagnosis is established by skin biopsy which reveals the concomitant presence of large, closely packed cells at the central part of the tumor and smaller cells with compact nucleus at the circumference. Immunohistochemistry shows expression of CK, CEA, EMA, and S-100 by the tumor cells [9,10].

Surgical excision of the tumor is the most effective treatment option but it is completely impractical for extensive solitary and multiple ES. Radiotherapy or CO₂ laser are alternative choices which have shown satisfactory results. Intralesional infusion of botulinum toxin A and triamcinolone has been tested providing minimal improvement and its long term efficacy remains questionable [11,12]. It is widely accepted that future studies are needed and until then surgical excision remains the gold standard therapy. The present patient was referred to plastic surgeon and was scheduled for staged excision of tumors. Figure 2 shows the postoperative condition.

CONCLUSION

The presentation of our case report is interesting from the standpoint that tumors were initially considered as multiple leiomyomas which are also rare and benign neoplasms but they arise from smooth muscle cells. In order to design the appropriate therapeutic strategy, our clinical diagnosis needed to be confirmed by a skin biopsy, which showed the existence of multiple eccrine spiradenoma. In conclusion the present article points out that similarities in clinical appearance and symptoms between tumors may lead to erroneous diagnosis and that skin biopsy is essential for differential diagnosis.

Consent

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

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EDSF (McGrath syndrome) - a rare variant of epidermolysis bullosa simplex

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ABSTRACT

Ectodermal Dysplasia Skin fragility syndrome (EDSF) is a rare autosomal recessive syndrome characterized by loss of function mutation in plakophilin 1 gene (PKP1). Around 20 cases of EDSF have been reported in literature with presentations of skin fragility, alopecia, palmoplantar keratoderma, hypohidrosis, nail dystrophy, cheilitis and few uncommon presentations. There are no diagnostic criteria for EDSF syndrome, patient with overlapping feature of ectodermal dysplasia and genetic blistering diseases are included in EDSF syndrome. We report a case of 11 months male infant admitted for pneumonia, with features of skin peeling over pressure sites since 2-3 months of age, along with sparse lustreless hair, absence of eyebrows and delayed dentition. The child was diagnosed as a case of EDSF syndrome.

Key words: McGrath; Ectodermal dysplasia; Skin fragility.

INTRODUCTION

Ectodermal dysplasia-skin fragility (EDSF) syndrome also known as McGrath syndrome, is a rare autosomal recessive genodermatosis, which results from loss-of-function mutations in plakophilin 1 (PKP1) [1]. It is now considered as a specific suprabasal form of epidermolysis bullosa simplex [2]. First case of EDSF syndrome was described by McGrath in a 6 year old boy in 1997 [1]. There are only few case reports of EDSF syndrome, we are reporting this case to add to the number of cases of the rare entity.

CASE REPORT

Eleven months male infant admitted in paediatric department for pneumonia was referred to us with the complaints of peeling of skin at places and sparse hairs. On detailed interrogation, mother gave history of fluid filled lesions at the sites of friction since 2 months of age, lesions used to rupture spontaneously or with trivial trauma leaving behind raw surface which used to heal in 2-3 weeks time without any pigmentary changes or scarring. There was also history of sparse

scalp hair, absence of eye brows and eyelashes. The child was born at term with normal vaginal delivery with birth weight- 2.6kg and unremarkable antenatal period. There was no history suggestive of lowered immunity. There was no history of consanguinity, similar complaints in the family.

On examination, child had fever with increased respiratory rate and chest indrawing with other vitals normal and was on treatment for pneumonia. Child was weighing 6 kg and other milestones were normal.

On Mucocutaneous examination, there were multiple well to ill defined, erythematous irregular erosions of size approximately 2x2cm to 5x 4cm over back, legs and scalp (Fig. 1). Nikolsky's sign was negative. Scalp hair were sparse and lustreless with absence of eyebrows and eyelashes (Fig. 2). Hair microscopy did not reveal any beading or shaft breakage. Teeth had not erupted and nails were lustreless. Biopsy was not done as the child was sick and parents were reluctant for biopsy.

Systemic examination except respiratory system was normal. Lab investigations and chest radiography

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Figure 1: Back of child showing superficial erosions.



Figure 2: Sparse hair over scalp, absence of eyebrows and eyelashes.

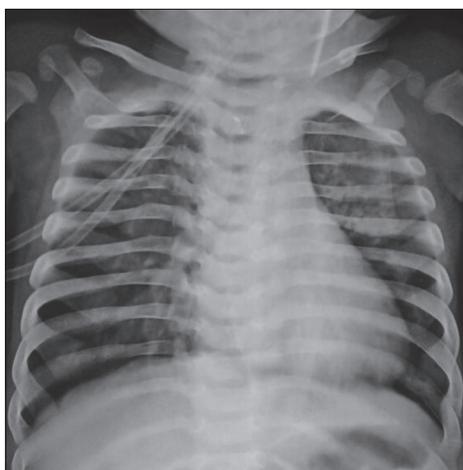


Figure 3: X- ray chest showing heterogenic opacity in left upper zone.

were suggestive of pneumonia (Fig. 3). On the basis of clinical and laboratory findings we diagnosed the case as ectodermal dysplasia-skin fragility syndrome.

DISCUSSION

Ectodermal dysplasias are a group of inherited disorder with developmental abnormalities of two or more of the following: hair, teeth, nails, sweat glands and other ectodermal structures. Genetic blistering disorders are a group of disorders characterized by blistering of the skin and mucosae following mild mechanical trauma. Very rarely, ectodermal dysplasia may co-exist with genetic blistering disorders, first case of which was described by McGrath in 1997 and coined the term “Ectodermal dysplasia-skin fragility syndrome” (EDSF) or McGrath syndrome [1].

EDSF is a rare autosomal recessive genodermatosis, which results from loss-of-function mutations in plakophilin 1 (PKP1), sequencing of genomic DNA revealed a homozygous 5 base pair deletion in exon 5 of the PKP1 gene [2]. PKP1 is member of the armadillo family of proteins which links desmosomal cadherins (desmogleins and desmocollins) to the keratins, contributing to the mechanical integrity of keratinocytes [3].

PKP1 is expressed throughout the epidermis, particularly in suprabasal cells and also in the outer root sheath of hair follicles [4]. Two isoforms of PKP1, 1a and 1b have been described [5]. PKP1a is expressed in both desmosomes and nuclei, whereas PKP1b is expressed only in nuclei. Currently, the specific biologic significance of the two isoforms is unknown [4].

Most common features such as skin fragility, alopecia, palmoplantar keratoderma, hypohidrosis, nail dystrophy, and cheilitis, have been described in patients with EDSF syndrome. Uncommon features such as perioral fissuring, pruritus, growth retardation and dental caries reported in some cases of EDSF syndrome [5].

Unlike PKP 2 and other desmoplakins, PKP1 is not expressed in the heart so cardiac involvement is not seen in EDSF syndrome.

In Hay-wells syndrome patients present with alopecia, skin fragility along with cleft lip/palate and ankyloblephron, which was absent in our case. Mendes de costa syndrome also presents with skin blistering, alopecia, reticulate hyper/hypo pigmentation, but dentition is normal. No pigmentary changes were seen in our patient. A variant of monilithrix with desmoglein 4 mutation also shows scalp erosions and congenital hypotrichosis, but absence of follicular papules and hypodontia in our patient favours EDSF syndrome.

Histopathological examination shows hyperkeratosis, acanthosis with widened intercellular spaces, and acantholytic keratinocytes [6]. Immunohistochemical and electron microscopic studies have demonstrated poorly developed, small desmosomes along with a reduction in the number of desmosomes in the epidermis, particularly involving the lower suprabasal layer [6].

Treatment options are generally limited in the form of counselling, skin care avoiding trauma, use of mild antiseptics. A prenatal diagnosis can be established by preimplantation genetic diagnosis [7].

As EDSF has manifestations of both epidermolysis bulosa simplex and ectodermal dysplasia, but all the clinical features described by McGrath may not be present in every case, especially at early age of presentation. Hopefully, more number of cases in future will help us establish diagnostic features for this syndrome.

Consent

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

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Linear IgA bullous disease presenting as a Hailey-Hailey disease associated with Hashimoto thyroiditis: a case report

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ABSTRACT

Linear IgA bullous disease (LABD) is a rare autoimmune subepidermal bullous disease characterized by linear deposits of IgA anti-basement membrane zone antibodies. The skin lesions are variable: papular, vesicular, bullous, erythematous, edematous, and erythema multiforme-like, mimicking bullous pemphigoid, dermatitis herpetiformis, toxic epidermal necrolysis, mucous membrane pemphigoid or in described case - Hailey-Hailey disease. We present a case of a 17-year-old female patient diagnosed with LABD associated with Hashimoto thyroiditis

Key words: Autoimmune disease; Bullous disease; Clinical dermatology

INTRODUCTION

Linear IgA bullous disease (LABD) is a rare autoimmune subepidermal bullous disease characterized by linear deposits of IgA anti-basement membrane zone antibodies. Most cases of LABD are idiopathic and the etiology of the disease remains unclear, but some cases are occasionally induced by drugs, internal malignancies, and infections [1].

CASE REPORT

A previously healthy 17-year-old female patient presented with a 5-months history of vesicular eruptions. Physical examination revealed multiple vesicles and bullae on an erythematous base. First eruptions appeared in axillae and groins. The initial diagnosis was Hailey-Hailey disease. Topical treatment was established with gradual progression of the disease. The lesions spread on the lower trunk, back thigh surfaces, neck, facial skin around the mouth. The typical bullous eruption with a 'string of pearls' sign

appeared on left forearm. Oral mucosa, eyes, and nails were spared. General condition was good.

The patient's blood test revealed elevated antibodies against thyroid peroxidase (TPO) and antibodies against thyroglobulin (TG). The thyroid stimulating hormone (TSH) was 4,88 mU/L (0,27-4,2), with a free triiodothyronine (fT3) of 2,6 pmol/L (3,1-6,8) and a free thyroxine (fT4) of 9.6 pmol/L (12.0-22.0). Other laboratory studies including hematological examination, blood biochemistry, and urine analysis, antinuclear antibodies were normal. The family history of autoimmune diseases was negative.

Direct immunofluorescence (DIF) of biopsy specimens of normal skin demonstrated linear IgA-deposit pattern at the dermo-epidermal junction (DEJ) in the absence of other immunoglobulins and complement. Indirect immunofluorescence of serum was negative.

With the above findings, a diagnosis of LABD and Hashimoto thyroiditis was made. The patient was

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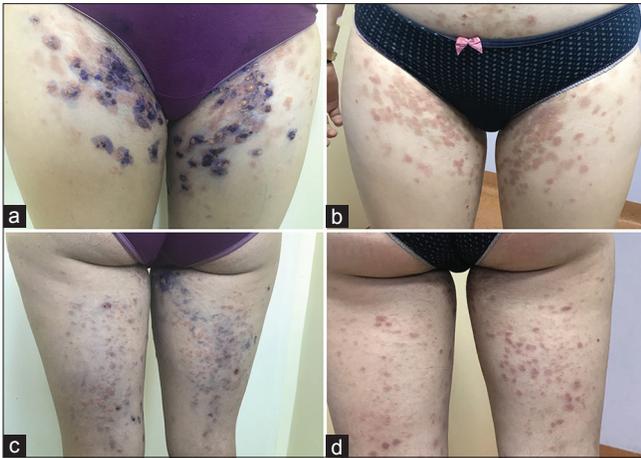


Figure 1: Multiple vesicles and bullae on an erythematous base in groins (a - before treatment; b - 3 weeks after starting an oral course of dapsonsone 50 mg per day) and back thigh surfaces (c - before treatment; d - 3 weeks after starting an oral course of dapsonsone 50 mg per day).

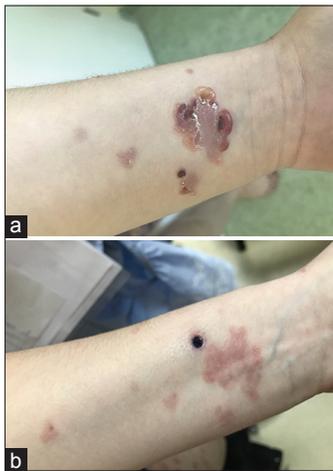


Figure 2: The typical bullous eruption with a 'string of pearls' sign on left forearm (a - before treatment; b - 3 weeks after starting an oral course of dapsonsone 50 mg per day).

started on a course of oral dapsonsone 50 mg daily to which her skin lesions responded (Figs. 1 and 2). The treatment was well tolerated and produced no side effects. The patient is under the care of the Dermatology and Endocrinology Departments.

DISCUSSION

LABD is defined according to the three following clinical and histological criteria: vesicular or bullous eruption involving skin and mucous membranes (MMs), subepidermal blisters infiltrated predominantly by neutrophils in lesion biopsies and a linear IgA-deposit

pattern at DEJ in the absence of other immunoglobulins on DIF [2].

The typical clinical picture is a bullous eruption with a 'string of pearls' sign, especially in childhood. The skin lesions in adult LABD are variable: papular, vesicular, bullous, erythematous, edematous, and erythema multiforme-like, mimicking bullous pemphigoid (BP), dermatitis herpetiformis (DH), toxic epidermal necrolysis or mucous membrane pemphigoid (MMP) [3].

In the described case, the symmetric distribution was confined to the areas typical of Hailey-Hailey disease (areas exposed to friction such as the sides of the neck, axillae, and groins) [4]. The adolescence-onset of disease was also similar to Hailey-Hailey disease.

Furthermore, the patient was diagnosed with Hashimoto thyroiditis which is also an autoimmune disease. Autoimmune diseases are common conditions which appear to develop in genetically susceptible individuals, with an expression of the disease being modified by permissive and protective environments [5].

Consent

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

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Syphilis: case series

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ABSTRACT

Syphilis is a sexually transmitted infectious disease caused by *Treponema pallidum*. This case series reports 3 cases of syphilis and highlights the varied presentation of primary and secondary syphilis which is rare in present day clinical scenario and also the association of syphilis and HIV co-infection. Case 1: A case of primary syphilis presented with solitary painless genital ulcer, associated with lymphadenopathy and VDRL was reactive in 1:32 dilution. Case 2: A retro positive patient presented with primary and secondary syphilitic lesions manifesting as multiple genital ulcers, disseminated skin rashes and oral lesions. VDRL and HIV was reactive. Case 3: A case of secondary syphilis presented with hyperpigmented annular plaques over both palms and soles with healing genital ulcers. VDRL and HIV was reactive.

Key words: Syphilis; HIV; *Treponema pallidum*

INTRODUCTION

Syphilis is an infectious disease caused by *Treponema pallidum*. Transmission occurs through sexual contact, vertical transmission, or less frequently, blood transfusions or reused sharp objects. It is common among patients with HIV infection and the converse is also true. Syphilis is a disease with devastating effects if untreated. Although effective and low-cost treatment is available, syphilis continues to be a public health problem due to lack of awareness [1].

CASE REPORTS

Case 1

18 year old male presented with single painless ulcer over penis since 2 weeks. He gave history of sexual contact with multiple partners since 3 months. There were no other associated signs and symptoms.

On examination multiple, firm, rubbery, non tender lymphnodes were enlarged bilaterally. Single, well defined, indurated, non tender superficial ulcer with clean floor of 2x3 cm present over prepuce. Single, indurated, nontender ulcer 2x4cm over coronal sulcus

(Fig. 1). “Dory flap sign” was present. Diagnosis was confirmed by VDRL (1:32). Patient was treated successfully with inj. Benzathine penicillin 2.4 million units and Tab. Azithromycin 1gm. Counseling was done.

Case 2

28 year old male patient presented with multiple painful genital ulcers of 3 weeks duration. He gives history of genital ulcers in wife 5 months back which healed on treatment. He gives history of sexual contact with multiple partners.

On examination multiple well defined erosions over the glans with clean erythematous floor was seen. Single well defined erosion with erythematous floor which was indurated and non tender over the frenulum (Fig. 2a). Multiple linear fissures with whitish maceration present over the prepuce (Fig. 2b). Inguinal lymphnodes were bilaterally enlarged and non tender. Blood VDRL was reactive in 1:32 dilutions. HIV was also reactive. A diagnosis of secondary syphilis with candidal balanoposthitis with HIV. Patient was treated with inj. Benzathine penicillin 2.4 million units and Tab. Azithromycin 1gm. Systemic and topical antifungals was also given. Counseling was done. Patient was referred to ART center for further management.

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Figure 1: Classical Hunterian chancre.

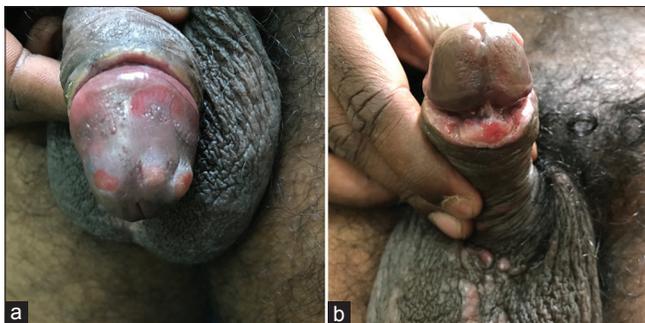


Figure 2a and 2b: A retropositive case with syphilis showing multiple erosions over glans.



Figure 3a and 3b: Palmoplantar lesions in a retropositive patient with secondary syphilis.

Case 3

25 year old male patient presented with annular dark raised lesions over both palms and soles since 20 days. Not associated with itching or pain. History of sexual contact with multiple partners of same sex since 2 years. Last contact was 6 months back. History of significant weight loss in last 6 months. On examination bilateral inguinal, cervical, epitrochlear lymphnodes were enlarged and nontender. Multiple, well defined, polysized, annular hyperpigmented plaques with minimal scaling over both palms and soles (Figs. 3a and 3b). “Buschke Ollendorff sign” was present.

Multiple erosions of 0.5x1 present over shaft of penis. Multiple, smooth, pearly white umbilicated papules over penis and perianal area. Diagnosis was confirmed by VDRL (1:32) and HIV was also reactive. A diagnosis of secondary syphilis with molluscum contagiosum with HIV was made. He was treated with Inj. Benzathine penicillin 2.4million units given IM weekly once for 3weeks. Tab. Azithromycin 1g given. Needling was done for Molluscum Contagiosum. Patient was referred to ART center for further management.

DISCUSSION

Syphilis is a chronic systemic infectious disease caused by *T. pallidum*. It may affect any organ in the body during its course and may result in life threatening consequences that occur in the cardiovascular and nervous systems. It is distinguished by florid manifestations on one hand and years of asymptomatic latency on the other hand. Transmissible to offspring, and treatable to the point of presumptive cure [2]. Syphilis has many uncommon presentations and requires high level of suspicion. The presentation differs in different stages such as primary, secondary, early latent and late latent.

Syphilis and HIV infection are common among patients with HIV infection and the converse is also true. Syphilis produces genital lesions or inflammatory response (macrophages and T cells) that enhance HIV replication. Strong epidemiologic association is observed between HIV and Syphilis. Several unusual manifestations of syphilis observed in the presence of concurrent HIV infection are increased severity of clinical manifestations, rapid progression of syphilis to the tertiary stage within a few weeks or months of initial infection, Sero-nonreactivity of Serological test for syphilis in the presence of an active secondary stage can occur when associated with HIV thus making the dark field examination or skin biopsy essential for the diagnosis of syphilis and relapse in spite of adequate treatment hence aggressive therapy appears mandatory at the initial presentation of syphilis with HIV infection [3]. Syphilis should also be suspected in high risk patients presenting a variety of atypical syndromes such as neurologic symptoms, uveitis or cholestatic hepatitis, especially if palmoplantar lesions are present [4].

CONCLUSION

Syphilis is rarely seen in our present day clinical scenario. This case series is being reported to highlight

the classical and varied presentation of syphilis and its possible association with HIV.

Consent

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

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Necrotizing fasciitis after central venous catheter placement

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ABSTRACT

Necrotizing fasciitis (NF) can appear after various penetrating or non-penetrating skin lesions. This is the second reported case in which NF occurred after a central venous line placement. A 60-year-old woman admitted to intensive care unit for the management of acute coronary syndrome complicated by cardiopulmonary arrest. Ten days after central venous placement in the left internal jugular vein, a reddening and swelling of the insertion site were observed. The evolution was characterized by aggravation of the local symptoms occurred, and the diagnosis of NF was made. The patient died before performing surgically debridement. This report on NF in a critically ill patient due to a subclavian central intravenous line aims to encourage checking for iatrogenic soft tissue condition in sedated intensive care patients. These patients may have a greater risk of developing NF, because they often have predisposing factors such as diabetes, end-stage renal failure, and immune suppression.

Key words: Necrotizing fasciitis; Mortality; Central venous catheter

INTRODUCTION

Necrotizing Fasciitis (NF) is a rare. NF is a bacterial derma-hypodermatitis affecting the soft tissue and muscular fascia. It is an uncommon and severe infection caused by microorganisms called 'flesh eating bacteria', mainly represented by group A beta-haemolytic streptococcus. NF remains a life-threatening condition associated with a high mortality rate [1,2]. Its location to the chest wall is extremely rare [1]. We report the second case in which NF occurred after a central venous line placement.

CLINICAL CASE

A 60-year-old patient, with a clinical history of poorly balanced diabetic, hypertension, and obesity, was admitted to the emergency department for the management of acute coronary syndrome complicated by left ventricular failure. Coronography revealed

an atheromatous coronary network with a long tight stenosis of the first segment of the right coronary and a moderate lesion of the mean coronary. The patient underwent angioplasty of the right coronary with placement of an active stent.

During the post-angioplasty follow-up, the patient presented a haemorrhagic shock following a parietal and retroperitoneal hematoma by breach of the external iliac artery, complicated by Cardio-pulmonary arrest. She was hospitalized in intensive care unit, where she underwent cardiopulmonary resuscitation with placement of a central left internal jugular vein.

The evolution was marked by the appearance of a poorly circumscribed erythematous, hot, painful and infiltrated placard at the left jugular region rapidly extended to the breast region, contralateral jugular region and anterior chest wall, progressing to ulceration and necrosis with a fever at 39 ° C (Figs. 1a-c).

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Biological assessment revealed an inflammatory syndrome (CRP: 405 mg/L), hyperleucocytosis (GB: 43,300 G/L); an increase of lactates to 4.4 mmol/L, CPK to 900 IU/L and transaminases (ASAT: 354 IU/L, ALT: 464 IU/L) and a deterioration of renal function (creatinine: 13 micromol/ Glomerular filtration rate: 42 ml/min). The patient rapidly deteriorated hemodynamically, and required the introduction of vasoactive drugs. Disorders of consciousness appeared, and the patient was therefore sedated and intubated.

An AngioTDM demonstrated a regression of the right parietal hematoma with the appearance of a large infiltration of the cutaneous and subcutaneous soft tissues of the thoracic wall and multiple air bubbles (Figs. 2a and b). The bacteriological study showed Gram-positive Cocci and gram-negative bacilli. Histological study confirmed the diagnosis of necrotizing fasciitis. The patient was made under broad-spectrum ATB. She died of septic shock before surgical debridement

DISCUSSION

Central venous catheters (CVC) cause more than 90% of all catheter-associated infections. Typically,



Figures 1: (a-c) Large necrotic ulcer with exposed fatty tissue.

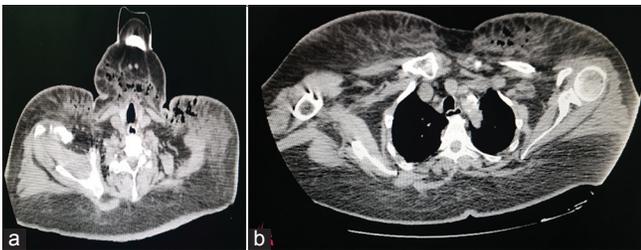


Figure 2: (a and b) An AngioTDM with a large infiltration of the cutaneous and subcutaneous soft tissues of the thoracic wall and multiple air bubbles.

subclavian access is considered as the preferable approach for a CVC from an infectious point of view [3]. However, severe complications such as catheter-related blood stream infections or cellulitis are possible. Therefore, strict criteria for CVC placement must be met and daily monitoring of the insertion site must be performed [3]. Necrotizing fasciitis is a rare but severe soft tissue infection. The incidence is reported to be 0.4 cases per 100,000 populations [4]. NF is associated with a high mortality rate [1].

Three types are distinguished: Type I is caused by a polymicrobial infection consisting with at least one anaerobic or facultative anaerobic bacterium. Type II is monomicrobial, mainly triggered by *β*-hemolytic streptococci or staphylococci. Among those, a large variety of different bacteria causing NF is published in literature. Type III is reported to be induced by *Vibrio* species after minor injuries in salt water [5]. Necrotizing fasciitis can occur after different skin lesions and even in intact skin by hematogenous spreading or by blunt trauma [6]. The first case of Necrotizing fasciitis after CVC placement has been described by N. Leibig et al. in 2014 [3]. Our case is the second case of NF after CVC placement.

A laboratory scoring system was created for NF, namely, the laboratory risk indicator for necrotizing fasciitis (LRINEC) [7]. C-reactive protein, WBC count, and hemoglobin, sodium, creatinine, and glucose concentrations, are integrated into the score. On the day of NF diagnosis, the patient had a LRINEC score of 10, which is believed to be high risk with a probability of † 75% for NF.

Publications show that early diagnosis, within 24 h, is missed in 85%–100% of all cases. Late determination of NF is combined with increasing mortality. If the suspected diagnosis is made, surgical intervention in terms of radical and adequate debridement is required immediately [3].

Sufficient soft tissue coverage after stabilization reduces the rate of re-infection, and all techniques in reconstructive surgery are required including free flaps. In many cases patients should be transferred to specialized centers for plastic and reconstructive surgery, optionally with a burn intensive care unit, for coverage after initial debridement [3].

CONCLUSION

NF is a challenging and potentially lethal disease. Early diagnosis and aggressive multidisciplinary treatment is

mandatory. Such rare complications can be avoided by following strict aseptic precautions when placing the Central Venous Catheter.

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Consent

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

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Milia like lesions in a 21 year male of hypohidrotic ectodermal dysplasia

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ABSTRACT

Ectodermal dysplasia are rare genetic disorders characterized by abnormality in development of skin and its appendages. Depending upon the involvement of eccrine gland they can be divided into different variants. Absence/decreased development of eccrine glands leads to hypohidrotic ectodermal dysplasia. This disorder can be associated with the number of other abnormal morphological features. Milia like plaques have been rarely reported. We present a 21 year male of hypohidrotic ectodermal dysplasia who presented with milia like lesions on the face and scalp.

Key words: Ectodermal dysplasia; Hypohidrotic; Milia like lesion

INTRODUCTION

Ectodermal dysplasia (ED) as the name suggests are inherited abnormalities of structures derived from ectoderm i.e. skin hair, teeth, sweat glands and nails [1]. Depending on the involvement of eccrine gland they may be hidrotic or anhidrotic/hypohidrotic. We are presenting a 21 year old male of hypohidrotic ectodermal dysplasia who presented with alopecia, absence of teeth along with rarely reported milia like lesions on the face.

CASE REPORT

A 21 year old male born to non consanguineous parents by an uneventful normal vaginal delivery with decreased scalp and body hair since birth and history of recurrent febrile seizures. He noticed lesions on the face for past 2-3 years which were asymptomatic and white in color. He also complained of lack of development of teeth. His intellect was normal. Younger brother had similar complaints according to the patient but his sister was normal. There was no history of similar complaints in any of the other family member. On general physical examination he was of average built (height of 159cm and weight of 51.4 kg). His mental, physical and sexual

development was normal. On examination of the face, there was periorbital hyperpigmentation, saddle nose deformity and multiple variable sized papules and plaques studded with milia like lesions (Fig. 1). The hairs on the scalp and beard were sparse, brittle and lusterless with complete loss of eyebrows (Fig. 2). Body hairs were absent. Generalized ichthyosis was present. (Fig. 3). There was decreased dermatoglyphics. Intraoral examination revealed only two peg shaped teeth (right upper incisors) with loss of alveolar ridges and eversion of lips (Fig. 4). Nail and genitalia were normal. On audiometry and eye examination, nothing abnormal was detected. Based on history and clinical examination final diagnosis of hypohidrotic ectodermal dysplasia was made.

DISCUSSION

Ectodermal dysplasias (ED) are rare inherited disorders with a incidence of 1:10⁵ [2]. They are defined by primary abnormal morphogenesis of skin, its appendages and teeth. Thurnam in 1848 first reported a case of ED but the term was coined by Weech in 1929 [3]. There are now more than 192 types of ED which have been classified into 2 major types: hypohidrotic/anhidrotic type and hidrotic type. Hypohidrotic/anhidrotic ED

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Figure 1: Milia like lesions, saddle nose and absence of eyebrows.



Figure 3: Hypodontia with only two persistent peg shaped teeth.



Figure 2: Sparse, thin hairs over scalp with milia like lesions.



Figure 4: Ichthyosis over thighs.

(HED) is also known as Christ-Seimens- Touraine syndrome which can be X linked recessive, autosomal dominant or autosomal recessive, of which X linked recessive variant is the commonest [4].

Clinically HED along with paucity of eccrine glands has hair abnormalities like hypotrichosis with fine, slow-growing scalp and body hair, eyelashes and eyebrows. Dental anomalies are characterized by anodontia/hypodontia in both deciduous and permanent teeth. Teeth are usually conical or peg shaped. Nail though less frequently involved, onychodysplasia may rarely be seen. Skin is thin, wrinkled, dry, with loss of appendages, flexural eczema, peri-orbital hyperpigmentation, saddle nose. Rarely milia like papules may be seen on the face. Dermatoglyphics are reduced. Spock ears may be seen [5]. Patient usually presents at the age of 11-18 yrs. [6].

HED has to be differentiated from Werner syndrome and odonto-onycho-dermal dysplasia. Werner syndrome

shows features of premature ageing but hypohidrosis is not a feature and sclerosis is seen. Odonto-onycho-dermal dysplasia may have hypodontia, sparse hair, onychodysplasia but have hyperhidrosis of palms and soles.

Genetic evaluation in HED have shown gene locus on Xq11-21.1 mutations. Ectoderm-endoderm interaction is required for formation of various ectodermal structures. Mutations in the genes coding for proteins which are required in signaling these pathways leads to ED. Mutations in EDA (ectodysplastin A, a TNF family ligand), EDAR (ectodysplastin A receptor) and EDARADD (ectodysplastin A receptor associated death domain) genes have been identified to cause HED [7].

A patient of HED, in addition to the psychological support patient needs to be counselled about the risk of hyperthermia because cooling effect due to impaired

sweating is lost [8]. Symptomatic treatment is otherwise given for the rest of the clinical manifestations.

Consent

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

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Validation of Verdura intense moisturizing lotion for its moisturization benefits

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ABSTRACT

Every skin needs moisturization and choosing right moisturizer is very important. No matter what kind of the skin one have, moisturizing it is required. In the current study we have evaluated the moisturizing ability of Verdura intense moisturizing lotion in human volunteers. Dermascope and moisture analyzer was used to know the texture of skin and moisture level respectively. The study shows the intense & prolonged moisturization is achieved with the Verdura intense moisturizing lotion.

Key words: Moisturizing lotion, Analysis of moisturizer, Verdura intense moisturizing lotion

INTRODUCTION

Moisturizing of skin is very vital for healthy skin. Understanding the requirement of moisturization of skin regularly over the past decade, great progress has been made toward elucidating the structure and function of the stratum corneum (SC), the outermost layer of the epidermis [1].

SC cells (corneocytes) protect against desiccation and environmental challenge by regulating water flux and retention. Maintenance of an optimal level of hydration by the SC is largely dependent on several factors. Using of right moisturizers can balance both the moisturization and emolliency of the SC cells which gives healthy skin.

A good moisturizer should be able to provide moisturization to skin and should repair the skin & should retain the moisture for a long time. The moisture level and emolliency of skin can be measured in various methods such as electric conductance, trans epidermal water loss, Fourier transform infrared spectroscopy, photothermal imaging and confocal Raman spectroscopy [2-4]. In these methods measuring moisture level of skin is simple and reliable method to understand the variations in skin hydration & emolliency.

In the present study we have validated the efficacy of the Verdura intense moisturizing lotion by two methods.

1. Moisture level
2. Dermascope images

Moisture levels will give understanding of the moisturization and retention and dermoscopic study shows the repairing ability of the product. Results are presented in the paper.

MATERIALS AND METHODS

Details of investigational product

Verdura intense moisturizing lotion is a product of Dr. JRK's Research and Pharmaceuticals Pvt. Ltd., Chennai, India. It is formulated with *Aloe vera* extracts in a rich base to provide intense and long retention of moisturization.

Clinical evaluation

Study design

Ten volunteers were selected for the study. For analysis volunteers of both male and female in age

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group of 20-45 was considered. Biceps region was selected as wear and tear is less and initial moisture levels were measured and marked these areas for further analysis.

Skin moisture level percentage was measured in 10 volunteers using skin analyzer. (PEPECARE Body Skin Facial Tester Oil Water Moisture Meter LCD Display Monitor Analyzer).

Study was planned for 5 consecutive days in following stages.

1. Initial moisturizing levels was measured - untreated
2. Test product was applied and left for 2-3 minutes and moisturizing levels was measured at 1 hour, 3 hour and 5 hours for all the volunteers
3. Dermoscopic image was taken for all the patients as untreated, treated (3 min) and treated 1 hour to understand the repairing ability

The test sample was applied as 1mg/cm² concentration on marked area. Moisture level was measured for all the volunteers in the time intervals of 3 min, 1 hour, 3 hours and 5 hours respectively. Readings were noted.

RESULTS

The table 1 shows the mean moisture levels of 10 volunteers evaluated for 5 days. There is improvement in the moisture levels of the skin in 5 days treatment.

Table 1: Mean moisture levels in 5 day's treatment

	Untreated	Treated 3 min	Treated 1 hr	Treated 3 hr	Treated 5 hr
Day 1	29±4	36±5	33±2	32±3	29±3
Day 2	32±2	41±2	35±2	32±2	31±3
Day 3	32±2	40±2	36±2	34±2	31±2
Day 4	31±3	55±7	37±3	35±3	33±4
Day 5	32±2	42±2	37±2	35±4	33±2

Table 2: Summary of statistical data

	Treatments					
	1 day	2 day	3 day	4 day	5 day	Total
N	5	5	5	5	5	25
ΣX	156	214	178	168	157	873
Mean	31.2	42.8	35.6	33.6	31.4	34.92
ΣX ²	4874	9366	6348	5654	4941	31183
Standard deviation	1.3038	7.1903	1.6733	1.5166	1.6733	5.3923
Result details						
Source	SS	df	MS			
Between-treatments	452.64	4	113.16		F=9.23002	
Within-treatments	245.2	20	12.26			
Total	697.84	24				

Table 2 shows the f-ratio value is 9.23002. The p-value is 0.000215. The result is significant at p<0.05. Table 3 shows the dermascopic images of the treated and untreated areas for 5 consecutive days. There is an improvement in skin texture was observed.

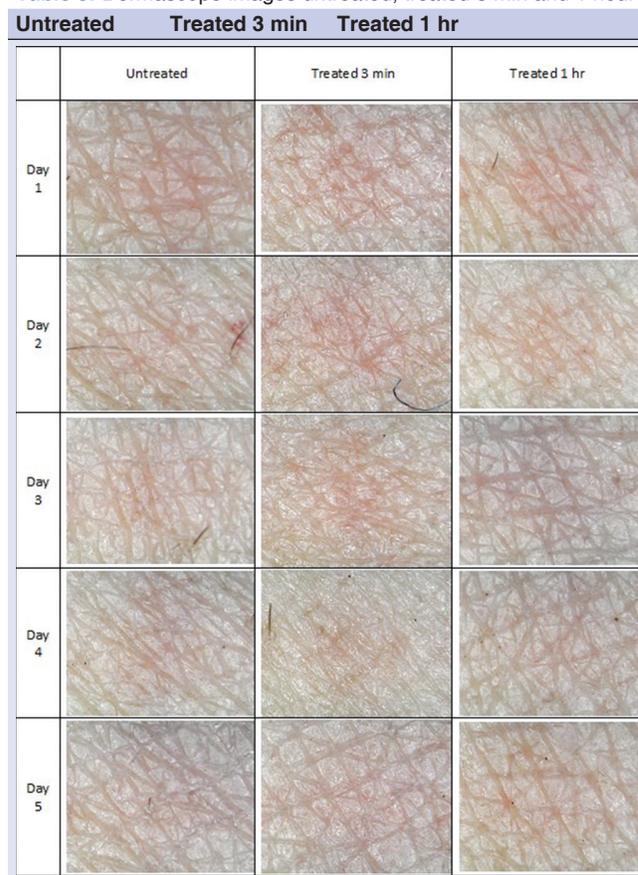
DISCUSSION

Stratum corneum is the outermost layer of the epidermis. It protects the living cells beneath it by providing a tough barrier between the environment and the lower layers of the skin.

The normal functioning of the SC, however, can be disturbed in dry, flaky skin conditions. Moisturizers and emollients are required during these conditions.

In the present study, moisturization benefits was validated for verdura intense moisturizing lotion by measuring moisturizing levels on repeat treatment and the skin structure. Skin structure especially we to see the repair of the SC cells.

Table 3: Dermoscope images untreated, treated 3 min and 1 hour



The moisture analysis conducted for 5 days at untreated zone i.e, before application of lotion at 3 min, 1 hr, 3 hours and 5 hr shows a gradual improvement of moisture levels from 29% to 32% with a standard deviation of 4 and 2 respectively.

In the treated region, at 3 min time also there is sudden raise in moisture levels due to application of lotion. The dermascope image also showed that there is constriction of cells after application of lotion. After an hour there is expansion of cells observed which shows the repair activity of cells. at the same time there is reduction in moisture levels compared to 3 min but more than the untreated i.e 36% (3min on 1st day) and 42 % (3 min on 5th day).

The moisture levels are 33% on day 1 (1 hour reading) and 37% on day 5. The similar moisture levels are observed on 3 hour readings i.e 32% (day 1) and 35 % on day 5.

However, there is reduction in moisture levels by 5 hours but by day 5 there is a significant improvement which shows the repair of dry skin. The moisture levels on day 1 is 29% and improved to 33 by day 5.

CONCLUSION

This study shows that there is improvement in the moisture levels of the skin with regular use of Verdura

intense moisturizing lotion and also it repairs the damaged dry skin. The damage repair can be observed by reduction in the width of the lines between the cells. This reduction shows the improvement in the texture of skin. Thus verdura intense moisturizing lotion can be used for intense moisturization and damage repair of dry skin.

Consent

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

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Cutaneous calcinosis induced by intravenous calcium gluconate administration

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A 78-year-old woman presented to our dermatology department for an indurated plaque on her right wrist since three weeks. There was a history of confusion due to hypocalcemia for which the patient received intravenous injections of calcium gluconate. Calcium gluconate solution was administered through a peripheral vein. An episode of extravasation occurred involving her right wrist causing skin erythema, oedema, pain and cutaneous ulceration. Two weeks later, the erythematous plaque which appeared on her wrist gradually became indurated. Dermatologic examination revealed an indurated plaque measuring 32 mmx35 mm in size located on her right wrist on an overlying erythematous skin (Fig. 1). Cultures were negative for microorganisms. The patient had normal blood analysis including serum calcemia and serum phosphate. A skin biopsy was performed revealing calcium deposition in the dermis. The diagnosis of iatrogenic cutaneous calcinosis due to local tissue injury was assessed. The patient was treated with emollients and keratolytics and the plaque healed progressively leaving an atrophic scar.

Iatrogenic calcinosis cutis is an uncommon complication of the administration of intravenous calcium containing solutions [1]. According to Watanabe et al [2], about 18 cases of calcinosis cutis after intravenous administration of calcium preparations have been reported in the literature. Herein we report a new case of iatrogenic cutaneous calcinosis in an old woman. The precise pathogenesis underlying the development of iatrogenic calcinosis cutis is not well understood. This complication could occur with or without extravasation of calcium. In fact, even minor extravasation of calcium containing solutions can produce lesions of calcinosis cutis.

After extravasation of a calcium containing solution, cutaneous lesions typically develop within 2 weeks. Watanabe et al [2] proposed an explanation to this phenomenon. In fact, it appears according to this author that the introduction of the needle causes tissue injury with capillary disruption in the dermis. In our case, it is iatrogenic due mainly to skin deposits of calcium which cause tissue damage.

Extravasation of calcium solutions may induce a marked inflammatory reaction with soft tissue necrosis and cutaneous calcification. However, extravasation is not a prerequisite for tissue calcification. Alkalinity also favours precipitation of calcium salts [3]. Calcification is defined by the deposition of insoluble calcium salts in the dermis. This deposition leads to a transient increase in calcium concentration in the extracellular matrix between collagen fibers with subsequent binding of calcium phosphate in the dermis and deposition of calcium phosphate crystals in the dermis.



Figure 1: Indurated plaque measuring 32 mmx35 mm in size located on the right wrist on an overlying erythematous skin.

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This iatrogenic calcinosis cutis should be differentiated from metastatic, dystrophic and idiopathic causes of this calcinosis cutis. In fact, metastatic calcification occurs in undamaged tissue and is associated with elevated serum calcium levels. Idiopathic calcification occurs in the absence of evident tissue or metabolic abnormalities. Dystrophic calcification occurs as a result of local tissue injuries or abnormalities [1].

This case is being reported to highlight this possible complication of intravenous calcium administration. A careful medical history taking is important to recognize this complication.

Consent

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

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Sclerema neonatorum in a premature newborn

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Sclerema of the newborn is classified among the lobular panniculitis, it is an extremely rare affection, most often lethal which occurs on a weak ground or sepsis [1]. The sclerema was announced for the first time at the beginning of the XVIIth century and the most authors have confused it with the scleroedema and cytosteatonecrosis of the newborn so the most diverse names have been given to these three conditions combined. Several theories have been proposed to explain his pathogenesis, which remains poorly understood [2]. The diagnosis of sclerema is clinical, it is manifested in newborns during their first week of life by a generalized cutaneous induration which gradually achieves, within a few days, a diffuse sclerous skin condition very paradoxically respecting the hands and feet, but may extend to compromising life-threatening dietary and respiratory functions. Sclerema treatment is based on newborn conditioning, antibiotic therapy, systemic corticosteroids, exsanguino-transfusion and

currently the advent of intravenous immunoglobulins. Despite these treatments, the prognosis of sclerema remains reserved with a high rate of mortality [3].

We report the case of a premature infant on D10 of life, hospitalized in neonatology for acute respiratory distress, which had since birth an induration of the diffuse skin. Dermatological examination showed the presence of a generalized sclerosis taking the whole body respecting the genitals as well as the palmo-plantar region [Figs. 1-3], the diagnosis of newborn sclerema was retained after eliminating other diagnoses including neonatal cytosteatonecrosis and scleroderma, and then was put on cortico antibiotic combination.

Consent

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



Figure 1: Diffuse back sclerosis.



Figure 2: Generalized sclerosis.

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Figure 3: Sclerosis respecting palmar region.

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Melanonychia in children

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A healthy 2 years-old-boy, with no family history of melanoma or other tumors. He was brought in consultation by his mother for evaluation of a congenital lesion of the nail of the thumb of the left hand. Physical examination showed a homogeneous erythematous-brown color band of the nail of the thumb of the left hand. Without hyperpigmentation of the proximal nail fold (Fig. 1). Dermoscopy revealed multiple homogeneous, narrow and regular longitudinal lines in the nail plate with some pigmented globules. Without periungual pigmentation (Fig. 2). Correlation of the age, the congenital onset, clinical and dermoscopic findings suggested melanocytic nevus of the nail matrix as the most likely diagnosis. Given the apparent benign nature of the lesion, close clinical and dermoscopic control was recommended. After 24 months of follow-up, the size of the lesion was unchanged.

Melanonychia is defined as a brown or black pigmentation on a nail as a result of melanin and other exogenous pigments [1]. In rare instances, congenital nevi present subungually, with fewer than 20 biopsy-proven cases reported [2]. Nail matrix nevus usually presents with longitudinal melanonychia, which is also observed in subungual melanoma or other benign conditions such as subungual lentigo. Pigment bands of Nail matrix nevus are well demarcated from adjacent uninvolved nail plate devoid of pigment and the nail plate surrounding the longitudinal melanonychia also presents with a background of brown pigmentation [3]. Hutchinson sign is defined by longitudinal melanonychia extending into the periungual tissues. Whereas it is one criterion for the ABC rule of subungual melanoma and is traditionally considered a worrisome feature, it is certainly not pathognomonic and a malignant cause should not be assumed without thorough assessment [2]. Dermoscopy is especially helpful in the differential diagnosis of nail pigmentations, because it can reduce unnecessary and



Figure 1: Homogeneous erythematous-brown color band of the nail of the thumb of the left hand.

invasive nail unit biopsy. It usually observed as a limited width of brown background with a regular pattern of the longitudinal lines. A previous dermoscopic study of adult and childhood NMNs found that pseudo-Hutchinson's sign, triangle sign, and globules were more frequent in childhood NMN and that Hutchinson's sign and irregular band pattern also tended to be more common in children [1]. The management of pigmented nail lesions will therefore depend on whether the rare but very serious childhood melanoma is suspected. In cases of melanonychia in which malignancy is suspected (a broad band of pigment, Hutchinson sign, irregular dermoscopic features, a dark-skinned patient), the lesion must be completely excised. Dermoscopic and clinical follow-up should be reserved for lesions with low-risk features (narrow bands, uniform dermoscopic characteristics, no changes overtime) [3].

Consent

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

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Figure 2: Homogeneous, narrow and regular longitudinal lines in the nail plate with some pigmented globules.

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Halitosis as presenting feature of Sjogren's syndrome

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Sir,

Sjogren's syndrome (SS) is an autoimmune disorder caused by the lymphocytic infiltration of exocrine glands resulting in glandular dysfunction, preferentially of the salivary and lacrimal glands. It can be classified into two types, namely primary Sjogren's syndrome and secondary Sjogren's syndrome. Primary Sjogren's syndrome (pSS) occurs in the absence of other autoimmune diseases and is characterised by keratoconjunctiva sicca (dry eyes) and xerostomia (dry mouth), collectively called the sicca syndrome. In contrast, secondary Sjogren's syndrome presents along with other autoimmune diseases such as rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE). The most common clinical manifestations of SS include dry eyes, dry mouth and dryness of larynx or pharynx. Xerostomia may lead to secondary problems like oral candidiasis, dental carries and periodontal disease [1-3].

We report the case of a 61-year-old female who presented with a history of halitosis for the last eight years, who on further evaluation, was diagnosed as a case of primary Sjogren's syndrome.

A 61-year-old female presented to us with an eight year history of persistent foul breath. The patient had consulted dentists on numerous occasions and was prescribed antiseptic mouthwashes but there was no sign of any oral disease and the halitosis was persistent, hampering her quality of life. There was no history of any gastrointestinal or sinus problems, nor was any history of any drug intake. The patient gave history of her requirement to drink water frequently, even for swallowing at times, along with recurrent dryness and irritation in eyes for which she hadn't consulted any ophthalmologist. There was no history of any other systemic abnormality. Physical examination revealed

evident halitosis on exhalation through the mouth; the rest of the oral cavity was normal. Laboratory investigations revealed normal hemogram and renal and liver function tests. Indirect immunofluorescence only revealed an antinuclear antibody titer of 1/80 with a speckled pattern and the presence of anti-Ro antibody. Chest radiography and abdominal ultrasound were normal. Schirmer's test revealed moderate dryness of eye with secretion of 7mm after 5 minutes. The patient was advised a salivary gland biopsy which was refused by the patient.

On the basis of history, clinical examination and laboratory investigations, the patient was diagnosed as case of primary Sjogren's syndrome (as the patient fulfilled 4 out of 6 American-European consensus group classification criteria for Sjögren syndrome) which led to halitosis and was managed symptomatically with artificial tears and saliva and was advised frequent intake of water. There was partial improvement in the symptoms over the next three months after which the patient was lost to follow up.

The prevalence of SS is estimated to be approximately 3% in subjects 50 years or older, with a female to male ratio of 9:1. The clinical manifestations are often vague and mistakenly interpreted and attributed to other medical conditions leading to incorrect diagnosis and approximately half of all patients are thought to be undiagnosed [1,2]. Sicca syndrome is the characteristic presentation of SS which is the combination of dryness of the eyes (xerophthalmia), oral cavity (xerostomia), pharynx and/or larynx. Sicca syndrome also includes hoarseness, non-productive cough, skin dryness and dyspareunia in women [2,3]. General symptoms include fatigue, chronic pain and polyarthralgias. Patients with SS have been found to have a higher risk of lymphomas as compared to normal population;

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Table 1: Classification of halitosis

A) Physiologic halitosis (nonpathological oral factors)
B) Pathologic halitosis
I. Oral causes Periodontal disease, stomatitis, pharyngitis, parotid gland dysfunction and neoplasms
II. Extraoral dysfunction
a. Perioral causes: sinusitis, atrophic rhinitis, foreign bodies
b. Gastrointestinal diseases: Zenker's diverticulum, pyloric stenosis, gastroesophageal reflux, peptic ulcer, Helicobacter pylori infection, biliary disease and neoplasms
c. Respiratory diseases: pulmonary infections, bronchiectasis, abscess, tuberculosis and neoplasms
d. Neurological diseases: neurodegenerative diseases, epilepsy and neoplasms
e. Systemic diseases: diabetes mellitus, renal or hepatic failure, dehydration, poisoning, drugs, cystic fibrosis, autoimmune diseases (Sjögren's syndrome, rheumatoid arthritis, systemic lupus erythematosus, scleroderma) and carcinomas
f. Metabolic disorders: Trimethylaminuria, hypermethionemia

most common subtype associated being the mucosa-associated lymphoid tissue (MALT) lymphoma often seen in the parotid glands, which is usually a low-grade indolent neoplasm. Articular involvement may also occur in pSS presenting as symmetric, intermittent, nonerosive arthropathy. Pulmonary involvement in the form of nonspecific interstitial pneumonia and renal involvement as interstitial nephritis are also common in patients of SS [2-4].

The diagnosis of pSS is based on the American-European consensus group (AECG) classification criteria for Sjögren syndrome. These criteria include: 1) subjective presence of ocular dryness, 2) subjective presence of oral dryness, 3) objective measures of ocular dryness by Schirmer's test or corneal staining, 4) focus score > 2 in a salivary gland biopsy, 5) salivary scintigraphy showing reduced salivary flow (1.5 mL in 15 minutes) and/or diffuse sialectasias and 6) positive autoantibodies against SS-A and/or SS-B. SS is diagnosed when 4 out of 6 items are present; either salivary gland pathology or the presence of autoantibodies against SS-A/SS-B is mandatory [5].

Halitosis, also known as foul breath, is a common presentation in patients with periodontal and odontogenic infections. These infections lead to an increase in Gram-negative bacteria in the oral cavity that produce volatile sulfur compounds which impart the bad odor to the breath. Halitosis can also arise from infections like tonsillitis, sinusitis, pharyngitis and lower respiratory tract infections like bronchiectasis, lung abscess and gastrointestinal conditions like gastroesophageal reflux disease and peptic ulcers. Transient

halitosis can also occur with intake of foods like onions, garlic and certain drugs and poisons [5]. The common causes of halitosis are presented in Table 1.

As SS leads to xerostomia and reduced salivary flow, it creates an environment for the increased growth of microorganisms like *Lactobacillus acidophilus* and *Streptococcus mutans* which lead to halitosis, caries and other oral cavity infections.

Although halitosis is not a common presentation of Sjögren's syndrome, but the physicians should be aware of the association and a proper evaluation of patients with halitosis should be done to evaluate the underlying cause.

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Idiopathic pyoderma gangrenosum

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Sir,

A 36-year-old patient with no pathological pathology who presented 6 months before admission pustular lesions increasing in size and number and becoming ulcerated in the legs of centrifugal evolution, painful, without other associated signs including rheumatological digestive or other (Figs. 1a – 1d).

Clinical examination revealed multiple ulcers, well limited, purplish, necrotic in center, of varying sizes, the edges was raised with pus. The lesions were in the legs and there were no extra cutaneous manifestations. Biological tests were normal and the histology was in favor of pyoderma gangrenosum. The Idiopathic one was suggested due to exclusion of other usual associations.

The patient was treated by oral corticotherapy with good evolution.

Pyoderma gangrenosum (PG) is a neutrophilic dermatosis with unclear etiology [1]. Described in 1930 by Brunstig et al [2]. It is a noninfectious disease characterized by necrotizing, ulcerative and painful skin, whose incidence is approximately 3-10 cases per million people per year and it is rarely observed in children, accounting for less than 4% of cases, but primarily affects adults between the ages of 25 and 54 years old without gender preference [3]. PG can associate with inflammatory, infectious or malignant pathologies or be idiopathic [4]. It presents initially with coalescent inflammatory pustules, which fuse progressively leaving reveal a necrotic ulcer, with hypertrophic edges well defined, of purplish color. The base of the ulcer is often under-mined, indicating early subcutaneous inflammatory extension, with secondary ulceration of the epidermis, the lesions predominate classically in the lower limbs or the trunk,

but can be observed on the whole of the cutaneous surface. Exceptionally, it may spread to extracutaneous tissues, with pulmonary infiltrates or joint involvement simulating true septic arthritis.

PG remains at present a clinical diagnosis of exclusion. The histological aspect reveals most often nonspecific, but helps to exclude other potential causes [5].



Figure 1: (a-d) Ulcerations well limited, with raised, erythematous - purplish and inflammatory margins.

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The evocative histology includes a dense and deep dermal infiltrate consisting of neutrophils without vasculitis, associated with a necrotic ulceration appearance, and the treatment remains poorly codified to date. It is mainly based on general or local corticosteroids, immunosuppressors and certain anti-inflammatory antibiotics [2].

Consent

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

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Unusual presentation of pyoderma gangrenosum in an infant

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Sir,

Pyoderma gangrenosum (PG) is a sterile neutrophilic disorder that rarely affects children [1], only 21 cases were reported in literature. It is often associated with a concomitant disease, although up to half of cases are considered idiopathic [2].

We report the case of a 12-year-old child, with no particular antecedent, who consulted for multiple painful and ulcerative lesions evolving for 8 months, he was initially treated for presumed bacterial and viral infections with oral antibiotics and antivirals, with no response.

Physical examination revealed 2 small ulcers and 3 large ulcers measuring 3 to 8 cm with irregular borders, undermined edges and indurated base following a linear distribution along the right leg (Fig. 1).

There was no lymphadenopathy. Hematological and biochemical examination did not reveal any abnormality.

Biopsy was performed from the ulcer edge for histopathologic evaluation and tissue cultures for bacteria, fungi, and atypical mycobacteria.

The histological examination revealed A moderately dense superficial and deep lymphoplasmocytic infiltrate around the vessels (Fig. 2), confirmed the diagnosis of PG. A report looking for associated diseases revealed no anomalies. Oral corticosteroid therapy at a rate of 1 mg/kg/day allowed the healing of lesions with good progress.



Figure 1: Right leg's ulcerations.

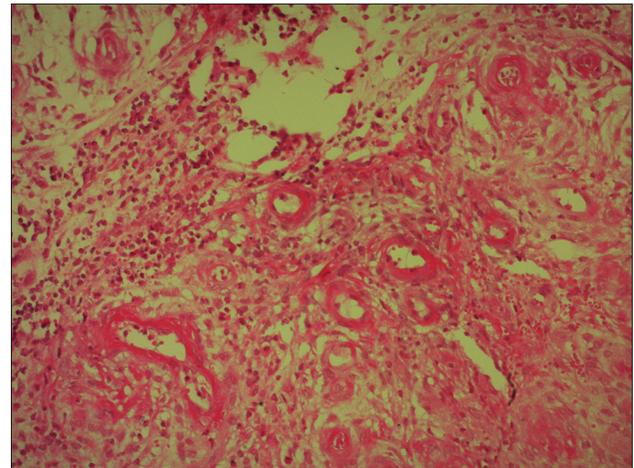


Figure 2: Dense superficial and deep lymphoplasmocytic infiltrate around the vessels with vasculitis. HEx20.

Pyoderma gangrenosum is relatively rare in the pediatric population with only 4% of PG occurring in children.

The most common presentation of PG in children is disseminated ulcerative lesions with lower extremities most frequently affected when lesions are localized [1].

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The cause of PG is unknown, but some studies have suggested that abnormal neutrophil chemotaxis is the primary process.

PG is an inflammatory condition of the skin first described by Brunsting and colleagues in 1930. PG begins as a pustule or vesiculopustule that progresses to an ulcer or deep erosion with violaceous overhanging or undermined borders [3].

In adults, an associated pathology is present in 74% of cases including a chronic inflammatory bowel disease, hemopathy or rheumatoid arthritis.

While in children, pyoderma gangrenosum is idiopathic in 50% of cases.

Our patient had no associated pathology.

The sporotrichoid character has never been described before, it may be explained in our patient by repeated trauma.

Optimal treatment for infantile PG is not established due to disease rarity and paucity of data. Treatment is based on systemic corticosteroids. In case of corticoreistance, the use of other immunosuppressors could be effective.

CONCLUSION

Through this observation, we report a new case of PG in children with no associated diseases and with sporotrichoid-like presentation which has never been described before.

Consent

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

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Extra-genital lichen sclerosis in a patient with anti-SS-A antibody

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Sir,

Lichen sclerosis (LS) is a rare chronic inflammatory skin disease generally affecting oral and genital areas, and rarely occurs on the neck, shoulders, upper trunk, axillae, and thighs. We describe herein a case of extra-genital LS involving a large area on the back, in which serum anti-SS-A antibody was detected.

A 25-year-old female was referred to our department, complaining of symptomless, whitish macules on the nape and upper back that appeared six months previously. On physical examination, macular lesions were located on the nape, upper back, and lower back (Fig. 1a). A number of whitish spots were observed within the large round, slightly shiny macules on the nape extending to the upper back (Fig. 1b). On the lower back, a few erythematous macules also had pigmented spots (Fig. 1c). There was no skin sclerosis. A biopsied specimen taken from the upper back showed that the epithelium was reduced in thickness, and moderate liquefaction was found in the basal layers. The most remarkable feature was homogenized and hyalinized degeneration of collagen fibers with marked edema just below the epidermis (Fig. 2). Laboratory data on blood chemistry including liver and renal function were within the normal range. Antinuclear antibodies (ANA) (1:80, homogeneous and speckled) and anti-SS-A antibody (83.0 U/ml) were detected in the serum, whereas rheumatoid factor, and serum antibodies against SS-B, thyroid and microsome were all within normal ranges. A diagnosis of LS was made. There were no other similar appearing lesions on her body including genitalia. We applied topical corticosteroid (betamethasone butyrate propionate) ointment, and the lesions gradually improved after two months. In the present case, atrophic macules

were observed relatively diffusely on the back without vulvar involvement. The differential diagnosis of LS includes generalized morphea and vitiligo-like depigmentation associated with systemic sclerosis, which were denied by histological examination. LS is sometimes associated with other autoimmune diseases, among which autoimmune thyroid disease is the most frequent [1-3]. In our patient, serological examination revealed positive ANA (1:80) and anti-SS-A antibody, whereas anti-SS-B antibody was within normal ranges. Additionally, she denied sicca syndrome. Detailed tear and saliva examination denied Sjögren's syndrome (SjS). To date, anti-SS-A antibody has been detected in only one co-existence case of LS and systemic sclerosis [4]. A previous study examined the association of LS and autoimmune disease, and found no association with SjS in 190 female patients with LS [1]. Also, other studies examined autoantibodies in a number of patients with LS; however, anti-SS-A antibody was not detected [2,3]. In a recent report



Figure 1: Macular atrophic lesions on the nape, upper back, and lower back (a-c).

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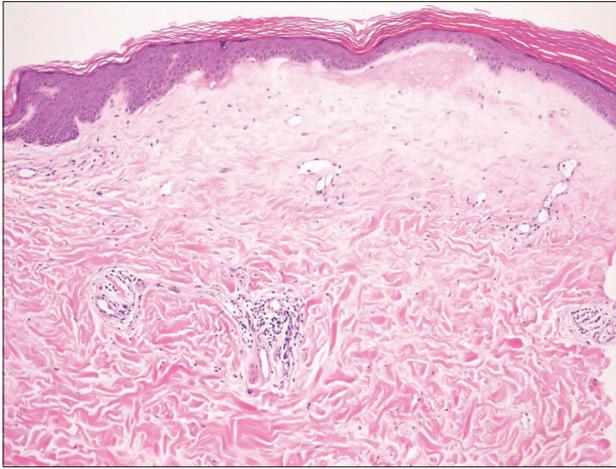


Figure 2: Histological features showing epidermal atrophy and homogeneous degeneration beneath the epidermis.

from Lebanon, nine out of 60 LS patients (15%) had at least one associated autoimmune disease, among which five cases of localized scleroderma, three cases of autoimmune thyroid disease, and one case of SLE were observed [5], but none had SjS. In conclusion, to our knowledge, there have been no reports on associated cases of LS and SjS; however, our patient is under follow-up to examine whether SjS will develop in the future.

Consent

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

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Looking through the dermoscope in a case of piebaldism

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Sir,

Piebaldism is an uncommon autosomal dominant disorder characterized by the congenital absence of melanocytes in localised areas of the skin and hair due to *c-kit* gene mutation, which affects the differentiation and migration of melanoblasts from the neural crest during the embryonic life. This condition is clinically characterised by the presence of localized stable hypo to depigmentation of the skin and hair, and by a characteristic distribution that involves the anterior trunk, extremities, the central portion of eyebrows, and the midfrontal portion of scalp with resultant white forelock, which is also the most common and persisting feature [1].

A 6 year old girl, first child born of non consanguineous marriage presented with asymptomatic white patches on the upper chest which remained stable since their appearance (Fig. 1). The child had no obvious ocular, hearing or neurological defects. The physical and mental development was normal. The audiogram of the child was normal. Routine biochemical tests were also normal. Family history was non contributory.

The examination revealed a well circumscribed white forelock in midfrontal region. Depigmented irregularly shaped macules were seen on the right upper chest. The entire back, abdomen, hands and feet, lower portions of forearm and legs and mucosae were completely spared. There was no evidence of involvement of eyebrows or eyelashes. The differentials considered at this point were piebaldism, nevus depigmentosus and congenital vitiligo.

Dermoscopic evaluation of the depigmented lesions revealed distorted melanocytic network with reduced pigment intensity (red arrows), absence of



Figure 1: Depigmented white patches over the anterior chest with irregular margins.

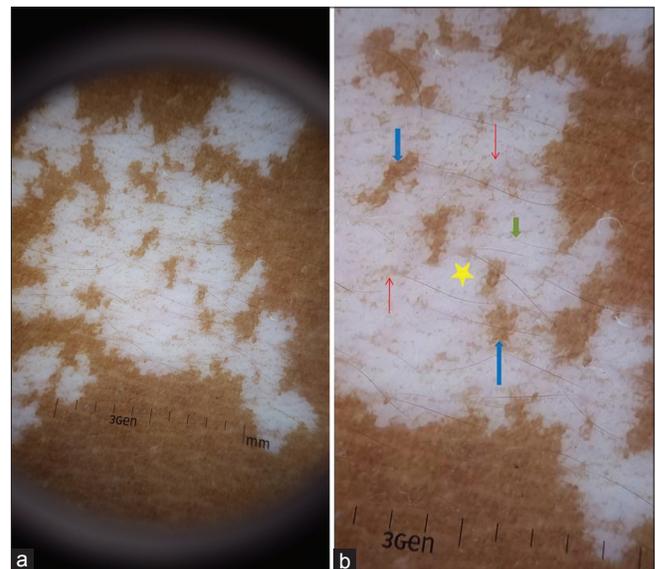


Figure 2: (a-b) Dermoscopy reveals distorted melanocytic network with reduced pigment intensity (red arrows), absence of perifollicular pigment (green arrow), vast areas of depigmentation (yellow star) and islands of normal skin or hyperpigmentation.

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perifollicular pigment (green arrow), vast areas of depigmentation (yellow star) and islands of normal skin or hyperpigmentation (Figs. 2a and 2b).

Based on clinical and dermoscopic features a diagnosis of piebaldism was established.

Piebaldism is a rare autosomal dominant disorder characterized by the congenital absence of melanocytes in affected areas of the skin and hair due to mutations of the *c-kit* gene, located on Chromosome 4q12, which affects the differentiation and migration of melanoblasts from the neural crest during the embryonic life [2]. Melanocytes are absent or considerably reduced in depigmented patches histologically and ultrastructurally. They are normal in number in the hyperpigmented areas.

Piebaldism results from defective migration of melanoblasts from neural crest to the ventral mid line thus manifesting dermoscopically as vast areas of depigmentation interspersed with islands of normal skin or hyperpigmentation. The second defect in the differentiation of melanoblasts to melanocytes

results in distorted melanocytic network with reduced pigment intensity. We hypothesize that the absence of perifollicular pigment may be due to the defect in transfer of melanosomes from the melanocytes in the hair bud to the surrounding keratinocytes, however further studies and ultra structural analysis will be necessary to substantiate the same.

Herein we report the first ever description of dermoscopic features in a case of piebaldism and state that further continuous documentation of observations in these rare cases will help us in arriving at more specific features.

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Rosettes within rosacea

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Sir,

A 43-year-old woman presented with recurrent flushing of the face and redness, erythematous lesions of the face with hypersensitivity to heat. These symptoms had persisted for 4 years, with intermittent remissions lasting up to 2 months. A physical examination revealed facial erythema, telangiectasia, papules and pustules of the midfacial region with some scales and crusts. (Fig. 1) Dermoscopy revealed linear vessels characteristically arranged in a polygonal network, creamy and whitish linear areas and a clear rosette sign. (Fig. 2) The rest of the somatic examination was without abnormalities. An ophthalmological examination showed no evidence of keratitis, conjunctivitis or blepharitis. On these bases a diagnosis of papulopustular rosacea was made. The patient was treated with doxycycline for a total of 12 weeks, which led to a significant improvement.

The rosette sign has been previously observed by Cuellar and colleagues and has been described as a new dermoscopic sign in actinic keratoses, which may be due to alternating areas of orthokeratosis and parakeratosis [1]. Recently, Liebman and his collaborators have pointed out that the rosette sign is an optical effect of polarized light and that its interaction with keratin-filled adnexal openings is observable in a wide range of cutaneous neoplasia [2]. This correlation could also explain the presence of this sign in rosacea too.

Consent

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

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Figure 1: Facial erythema, telangiectasia, papules and pustules of the midfacial region with some scales and crusts.



Figure 2: Dermoscopy showing linear vessels characteristically arranged in a polygonal network, creamy and whitish linear areas and a clear rosette sign.

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Ekbom syndrome: a case report

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Sir,

Ekbom Syndrome is also called delusory parasitosis, psychogenic parasitosis, or dermatozoic delusion [1]. It is an uncommon psychiatric condition, characterized by the continuous belief of being infested by parasites, although no medical evidence of infestation is found [2]. These patients seek advice from multiple practitioners and various specialties are often involved. They generally refuse psychiatric referral or treatment, and often refer to dermatologists.

We describe one case of a patient with delusory parasitosis who consulted many dermatologists for pruritic skin lesions.

A 56-year-old man presented to our clinic with a four-month history of recurrent pruritic papular eruption evolving in erosive lesions covered by crusts. The lesions had begun on his face, and gradually progressed to his upper arm. The disease was reported as heavily troublesome, and significantly impaired the everyday activities of the patient. He had a history of choroidal melanoma diagnosed 7 years ago and treated by protontherapy. There were no local or systemic complications of his melanoma. The man denied any drugs intake. The patient had been seen by several physicians and had experienced different treatments without improvement. Physical examination revealed several erosions, hematic and serous crusts on a slightly erythematous background. The lesions were located on the left half of the face (Fig. 1), and the right upper limb (Fig. 2). No other significant skin or mucosal lesions were seen. General examination was otherwise unremarkable. No primary dermatological diagnosis was made. Skin biopsy showed chronic nonspecific

inflammation of the dermis. The patient underwent routine biochemistry assays, research for parasites, chest-X-ray, abdomen ultrasonography and brain CT-scan. All those diagnostic procedures provided negative. At a more detailed interview, the patient reported that there are small living creatures in his skin which cause the itching. A psychiatrist was consulted, who diagnosed an Ekbom's syndrome. The lesions were provoked by the patient who was trying to eliminate the parasites of his skin. The patient was treated by anti psychotic drugs associated to psychotherapy with good evolution.

Delusory parasitosis is uncommon psychiatric disorder that may represent a challenge for the dermatologist, as it was in our case. It is characterized by the fixed belief to be infested with parasites or small living creatures, although there is no medical evidence for this [1-2].

The Ekbom syndrome is relatively rare. The majority of the patients are women. The average age at the beginning of the disease is 55,6 years [3].

Patients usually experience itching, which they attribute to the presence of animals in or under the skin. They often try to self-treat the disorder by scratching or by using disinfectants or pesticides. This causes skin lesions and itching, which in turn confirm the patient's belief of being infested and leads to a vicious circle of skin lesions, itching and delusional beliefs [2]. Cutaneous lesions are various, including discreet bruises, nodular pruritus and even ulcers and scars [3]. Like in the case presented, skin lesions typically predominate in those body regions that can be easily reached, whereas the body sides opposite to the patient's handedness lesions are usually absent.

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Figure 1: Erosions located on the left half of the face.

There are many psychiatric disorders associated such as anxiety, phobia, hypochondria, non-organic and organic delirious disorder [3].

Some cases are associated to organic diseases like hypothyroidism, diabetes, cortical lesions, mental retardation, kidney failure, hepatitis, severe anemia, intoxication by medication and cardiopathies, substance abuse, infectious and endocrine disorders [3].

The management of this condition is difficult, as patients with this disorder reject psychiatric diagnosis and treatment and often consult many specialists (dermatologists, internists or allergists). Antipsychotic drugs lead to improvement; pimozide was recommended as the drug of choice. Full remission is obtained in only half of the cases with antipsychotic treatment. Psychotherapy support is also needed [4].

Delusory parasitosis must be known by the dermatologist. This syndrome requires a multi-disciplinary approach.



Figure 2: Multiple erosions with hematic and serous crusts located on the right upper limb.

Consent

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

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Dermoscopy of notalgia paresthetica

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Sir,

A middle aged female complained of certain sensations on the back in the subscapular region. On clinical examination, there was a circumscribed, hyperpigmented patch. A provisional diagnosis of notalgia paresthetica was made (Fig. 1). Dermoscopy was performed using a DermLite DL3N at 10x magnification under polarised light.

Dermoscopy of this patch revealed alternating patches of hyperpigmentation and hypopigmentation (Fig. 2a). The hyperpigmented patches were brown to slate gray in colour and were present in various forms. They were arranged in an irregular net like pattern. In some areas the pigmentation was scattered with various patterns around a central hub which was either hypopigmented or hyperpigmented. Shiny white streaks were observed within the hypopigmented patches (Fig. 2b).

Notalgia paresthetica occurs due to nerve root entrapment of T2-T6 rami causing paresthesia resulting in chronic itching and friction. This friction leads to degeneration of keratinocytes and deposition of amyloid fibrils in cases of notalgia paresthetica. Amyloid fibrils are abnormally polymerised cytokertain derived material. These fibrils are birefringent and under polarised dermoscopy appear as shiny white streaks



Figure 1: A case of notalgia paresthetica in a middle aged patient.

streaks [1]. Thus, shiny white streaks in notalgia paresthetica correspond histologically to amyloid fibrils [2]. Hyperpigmentation of various patterns seen dermoscopically corresponds to either basal pigmentation of the epidermis or melanophages with pigment incontinence in the dermis.



Figure 2a: Dermoscopic overview shows alternate hypopigmented and hyperpigmented patches throughout the lesion.

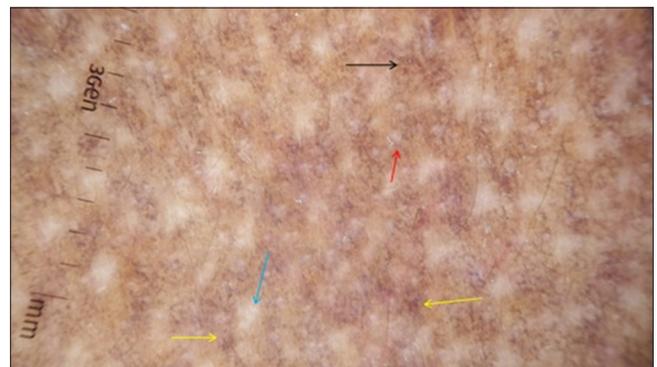


Figure 2b: Hyperpigmented patches are brown or slate gray in color. The pigment pattern may take the formation of irregular nets (black arrow), may have central hypopigmented hub (red arrow) or hyperpigmented hub (yellow arrow) surrounded by pigment configurations of different shapes and patterns. Shiny white streaks are also seen (blue arrow).

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Due to its presentation, notalgia paresthetica may mimic macular amyloidosis not only clinically but also dermoscopically. However, shiny white streaks are absent in macular amyloidosis on dermoscopy providing a vital dermoscopic clue in differentiating the two conditions.

Consent

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

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Anetoderma through the dermoscope

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Sir,

Anetoderma is a rarely encountered benign dermatological condition in which the elastic tissue in the dermis is lost, resulting in localized areas of flaccid or herniated saclike skin [1]. Currently, anetoderma is classified as either primary (idiopathic), or secondary anetoderma (which is associated with a variety of skin conditions, penicillamine use, or neonatal prematurity). Lesions appear on the upper arms, trunk, and thighs. This rare disorder occurs mainly in women aged 20–40 years, but is occasionally reported in younger and older patients of both sexes. In the most usual form, crops of round or oval, pink macules 0.5–1 centimeter in diameter develop on trunk, thighs and upper arms, less commonly on the neck and face and rarely elsewhere. Elsewhere. The origin may be primary or secondary, primary causes include Schweningen-Buzzi type which has no preceding erythema, and Jadassohn-Pellizari type which is preceded by macular erythema or papular urticaria. Secondary anetoderma occurs at sites of skin diseases such as acne, varicella, xanthoma, discoid lupus erythematosus, granuloma annulare, syphilis [2].

A 32 year old lady presented with asymptomatic lesions over the upper back slowly progressing in number and extent since one year. The family and past history was non contributory. She did not give any history of lesions prior to the occurrence of the current complaints. Dermatological examination revealed multiple skin colored to faintly erythematous macules over the upper back. Few of the lesions were slightly depressed as compared to the normal skin (Fig. 1). Dermoscopy of the lesions shows a background of erythematous hue with partially obliterated pigment network. The skin of the affected area has a translucent appearance as compared to the surrounding skin (Fig. 2). On examining of another dermoscopic field multiple lesions are noted



Figure 1: Asymptomatic macular lesions over the back.



Figure 2: Dermoscopy shows a background of erythematous hue with partially obliterated pigment network. The skin of the affected area has a translucent appearance as compared to the surrounding skin.

showing erythematous background with multiple linear vessels and translucent appearance of the skin (Fig. 3).

Venencie et al. [3], in their report suggested that the degradation of elastic fibres in patients with anetoderma is caused by enhanced expression of progelatinases A and B and production of the activated form of gelatinase A, where the lack of

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Figure 3: Multiple lesions are noted showing erythematous background with multiple linear vessels and translucent appearance of the skin. The vasculature becomes visible due to thinning of the skin resulting from degradation of elastin.

control of these enzymes by tissue inhibitors of metalloproteinases results in the development of the anetoderma lesions. The translucent nature of the skin noted on dermoscopy results from the loss of elastin tissue that is seen on histology in these lesions. The vasculature becomes visible due to thinning of the skin resulting from degradation of elastin. The background erythema results from the underlying inflammation. It may be postulated that the mild inflammation may interfere with the melanocytes in the basal layer leading to partial obliteration of the pigment network.

Dermoscopy may provide an important clinical clue towards the diagnosis of this rare entity by highlighting the translucent nature of the skin pointing towards the diagnosis. To the best of our knowledge this is the first report of dermoscopic findings in anetoderma and we believe that dermoscopy may be an important auxiliary tool for the quick and effective diagnosis of this disorder although histology remains the gold standard for diagnosis.

Consent

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

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The mystery of Juliet's death after Romeo's kiss. Romeo had drunk a powerful venom

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Sir,

Before to begin this dissertation it is better to explain three important physiological and chemical behaviours, that could embody finally the simplest explication of this strange pharmacological demeanour.

- a) The benzodiazepines nitrazepam and clonazepam were found to be unstable in saliva at room temperature and nitrazepam was converted into 7-aminonitrazepam. The conversion rate of nitrazepam was strongly dependent on the composition of the subject's saliva, and for this reason both nitrazepam and clonazepam did not induce a real drowsiness in the woman who underwent the cunnilictus [1].
- b) There exist a racial difference: some A.A. referred that individual differences exist between patients, and, for topical therapy, differences in skin due to race had be taken in consideration. Pharmacological response depends upon the percutaneous absorption and the inherent activity of the chemical once absorbed into the biological system. Our objective was to determine the in vivo percutaneous absorption of three test chemicals in human subjects with Asian (A), black (B) and Caucasian (C) ethnic skin. Following a 30 min topical application on the upper outer arm of 1 Mmol/cm ²¹⁴C-labeled chemical, percutaneous absorption was determined by both urinary excretion and the stripping technique. Amounts absorbed were: for benzoic acid 1.43 ± 0.27% (SD) (A), 1.07 ± 0.18% (B), 1.2 ± 0.19% (C); for caffeine 1.06 ± 0.17% (A), 1.01 ± 0.19% (B) and 0.96 ± 0.12% (C); for acetylsalicylic acid 1.8 ± 0.31% (A), 1.59 ± 0.31% (B) and 2.12 ± 0.36% (C). No statistical difference ($P > 0.05$) was found in percutaneous absorption of benzoic acid, caffeine

or acetylsalicylic acid between Asian, black and Caucasian subjects [2-7].

- c) There are physiological and physipathological factors to be observed before experimentations. Factors related to the vaginal physiology include pH of vagina (3.5 to 4.9), effect of the estrus cycle on the permeability of the vaginal mucosa, thickness of vaginal epithelium, vaginal fluid volume, chemical composition of fluid, pH, viscosity and surface tension and the pressure exerted on the dosage form by the rectal wall, play a vital role in vaginal drug absorption and sexual arousal, mucociliary clearance (MCC), vaginal obstruction, etc. which affect either the mucus or ciliary heating and vaginal blood flow. 2) Physicochemical Factors: Factors related to the dosage forms are physicochemical characteristics of the active ingredients; pH and mucosal irritancy; osmolarity; viscosity (solution, gels) and density (powder, tablet) to the formulation; concentration and volume administration; and type of dosage forms; particle size of the molecule of drug, hydrophilicity or lipophilicity of drug molecule, molecular weight of drug molecule, chemical nature, ionization surface charge, etc.

In tables was presented the list of the benzodiazepines and their relative half lives the A.A (Table 1) and the hours of drowsiness induced in the women after having undergone the cunnilictus (Table 2).

It is very odd the phenomenon that the longer is the half life of the benzodiazepines, the shorter is the induced drowsiness in the woman.

Exhaustive efforts have been made toward the administration of drugs, via alternative routes, that

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Table 1: All the kinds of hypnotics the AA assumed to make their proofs

Type of benzodiazepines	Half life (h) of the benzodiazepines
Bromazepam	20-40
Cinazepam	4-5
Cinolazepam	60
Clonazepam	9
Diclazepam	10-18
Estazolam	42
Etizolam	10-31
Flubromazepam	6
Flunitrazepam	100-220
Lorazepam	9.5-20
Metizolam	12
Nimetazepam	14-30
Nitrazepam	17-48
Quazepam	120
Temazepam	10
Triazolam	2

Table 2: The hours of drowsiness induced in the women after having undergone the cunnilictus

Case	Hours of induced drowsiness in woman
I	3
II	4
III	8
IV	0.30
V	2
VI	3
VII	5
VIII	1
IX	3
X	4
XI	2
XII	3
XII	0
XIV	1
XV	6
XVI	2

are poorly absorbed after the oral administration. The vagina as a route of drug delivery has been known since ancient times. In recent years, the vaginal route could be rediscovered as a potential route for systemic delivery of benzodiazepines and other therapeutically

important macromolecules. However, successful delivery of drugs through the vagina remains a challenge, primarily due to the poor absorption across the vaginal epithelium [7].

It is quite interesting to notice that Case IV (Clonazepam) that has been destroyed by saliva is not able to induce drowsiness in the woman. Nitrazepam too is inactive at all.

Consent

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

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Dermatology Eponyms – sign – Lexicon (U)

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ABSTRACT

Eponyms are used almost daily in the clinical practice of dermatology. And yet, information about the person behind the eponyms is difficult to find. Indeed, who is? What is this person's nationality? Is this person alive or dead? How can one find the paper in which this person first described the disease? Eponyms are used to describe not only disease, but also clinical signs, surgical procedures, staining techniques, pharmacological formulations, and even pieces of equipment. In this article we present the symptoms starting with (U) and other. The symptoms and their synonyms, and those who have described this symptom or phenomenon.

Key words: Eponyms; Skin diseases; Sign; Phenomenon

UGLY DUCKLING SIGN

In 1998, Grob and Bonerandi introduced the “ugly duckling” concept to demonstrate that nevi in the same individual tend to resemble one another and that atypical mole often deviates from the individual's nevus pattern. In other words, nevus that does not resemble other nevi is more likely to be suspicious of melanoma [1,2].

UKRAINIAN BLACK TONGUE SIGN (C. 1771, MOSCOW)

Bubonic plague. Black tongue in Bubonic plague. A similar sign – Antichrist sign, where black lips are a classic indication of an infection with *Yersinia pestis* [3-5].

ULNAR LEPROSY SIGN

Thickening of the ulnar nerve at the elbow [6,7]. A sign of tuberculoid leprosy. Also known as Elbow sign.

UMBILICAL SIGN

Umbilical sign is seen in Marfan syndrome. It is the unusual ability to touch the umbilicus with the right hand, crossing the back, and approaching from the left side, indicating increased length of upper extremity [8].

UREMIA TONGUE SIGN

The tongue is dry in cases of uremia [9].

URIOLLA'S SIGN

The presence in the urine of malarial patients of minute black granules of blood pigment [10].

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