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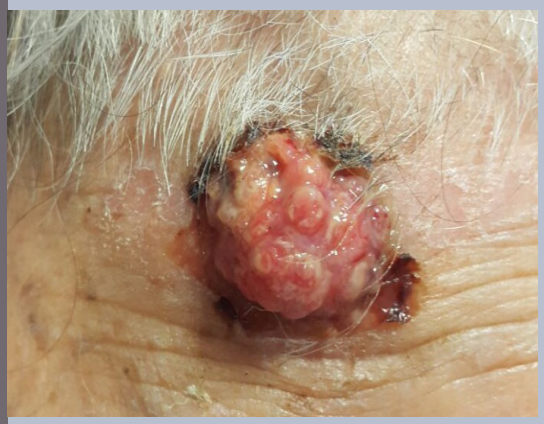

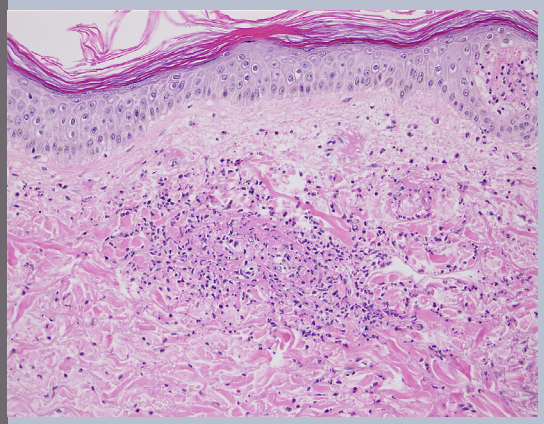

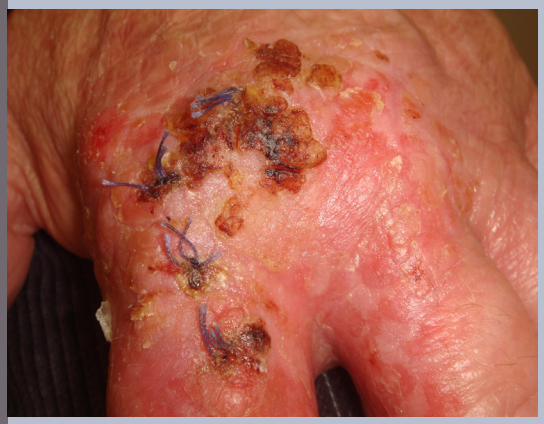

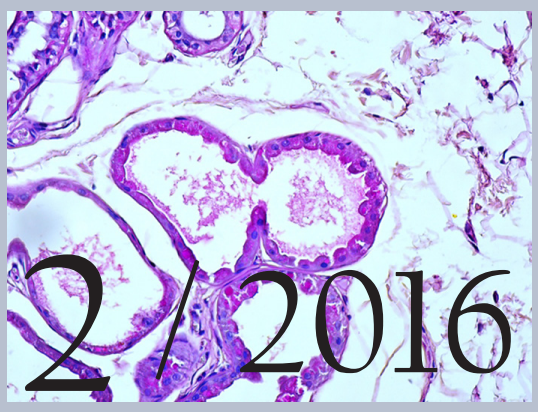

Editorial Pages

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Our Dermatology Online

Original Article

Blood eosinophilia: A poor prognostic factor for primary cutaneous T cell lymphomas? A cohort of 72 cases

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ABSTRACT

Introduction: Blood eosinophilia (BE) is described as a poor prognosis marker for some T cell malignancies. **Objective:** to detect the presence and the prognostic significance of BE in patients with cutaneous T cell lymphoma (CTL). **Methods:** This was a retro prospective study of 72 patients with CTL. Patients with other factors that may increase BE were excluded. **Results:** We had 14 cases of BE, 10 cases were in the erythrodermic stage of the disease and 6 in the tumoral stage and we had 4 cases of death. The BE was associated with deterioration of the general condition (p=0.001); depilation of the body (p=0.04), erythroderma (p=0.008), scalp and nails involvement (p=0.000), high rate of lactate Dehydrogenase (LDH) (p=0.000) and beta 2 microglobulin (B2M), (p=0.000), the histological type of Mycosis fungoides (MF) with positive Immunohistochemistry for CD4 (p=0.014) and CD3(0.05). **Conclusions:** BE was significantly related to MF, to advanced stages of the disease, to pejorative clinical signs and to elevated rate of LDH and B2M which are poor prognostic factors of MF with four cases of death, which prove that BE is also a poor Prognostic factor of MF.

Key words: Primary cutaneous T cell lymphoma; Mycosis fungoides; Blood eosinophilia; Marker of poor prognosis; Noninvasive; Clinical and therapeutic implications

INTRODUCTION

Primary cutaneous lymphomas (PCL) is the second frequent localization of extra nodal lymphomas, and the T cell lymphomas (PCTL) represent almost 80% of PCL. It's characterized by epidemiological, clinical, histologic, immunophenotypic and prognosis variety [1].

PCLT are dominated by the mycosis fungoides (MF), but others entities defined by the European Organization for Research and Treatment of Cancer (EORTC) classification are also reported: $CD30^+$ large T-cell lymphomas ($CD30^+$); Sezary syndrome (SS) and $CD30^-$ large T-cell lymphomas ($CD30^-$), pleomorphic small-to medium-sized CTCLs (PSMs), and it had a prognosis not yet well defined [2].

These lymphomas are becoming more and more aggressive, that's why many studies have attempted to identify some predictors of poor prognosis which will surely have therapeutic implications.

The blood eosinophilia (BE) is well Studied and reported as a marker of a poor prognosis in some T-cell malignancies: nodal T-cell lymphoma and Hodgkin's disease, but this eosinophilia is less studied in PCTL.

The aim of our study was to detect the presence of BE in patients with PCTL and its prognostic significance.

MATERIALS AND METHODS

This was a unicentric retro prospective study (retrospective since 2008 until June 2013 and prospectively from June

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2013 to October 2014) of patients followed for cutaneous lymphomas in the Department of Dermatology of the hospital Hassan II of Fez.

All patients with PCLT were included. The diagnosis of PCTL was based on the combination of clinical, histologic, and immune phenotypic criteria of the EORTC classification.

For survival analysis, we considered only disease-specific death, defined as death related to PTCL or its specific treatment.

Patient data have been collected by our doctors in the Department of Dermatology of the hospital Hassan II of Fez.

The medical records of our patients included: socio-demographic data such as "age, gender, work, ethnicity, the notion of personal or familial atopy, iatrogenic immunosuppression, viral immunosuppression (HIV), exposure to a toxic or irradiation". Clinical data such as: functional signs (Deterioration of the general condition,pruritus, Pain) and physical signs (plaques, nodules, tumors, erythroderma, scalp, nails and mucosal involvement). Paraclinical data: serum rate of lactate dehydrogenase (LDH) and B2 microglobulin, standard histology and immunohistochemistry results.

Deterioration of the general condition or alteration of the general condition (AEG) is a syndrome combining three clinical signs: Anorexia, asthenia and weight loss greater than or equal to 5% of normal weight.

Scalp involvement: especially in folliculotropic lymphoma such as alopecia.

Nail involvement because of the lymphoma or a reaction processe of paronychial inflammation: pachyonychia, xanthonychia, lines of beau.

Raised blood eosinophil count was defined as: >500 elements/mm^3. In patients with BE, an etiologic investigation has been achieved and only patients with a BE proved as "idiopathic" were selected. Patients with other factors that may increase blood eosinophilia were excluded.

High serum level of lactate dehydrogenase (LDH) in Adult was defined as >390 UI/l.

High serum level of B2 microglobulin (B2M) was defined as > 2.5 UI/l.

Two kinds of analysis were Performed: descriptive and univariate analysis.

The analysis was performed using the SPSS 20 software.

RESULTS

Our serie includes 72 cases of PCTL. The average age was 17.6 ± 55.12 years old and most of patients were aged more than 45 years (76.3%) and we had a slight female predominance (54.1%).

The study group included patients with classical and variants of MF (62 cases), Sezary syndrome (1 case), and non epidermotropic lymphoma (NEL): CD30$^+$ anaplastic large T-cell lymphomas (7 cases), CD30$^-$ large T-cell lymphomas (2 cases).

We had 14 cases (19.4%) of BE in our patients (Table 1).

The BE was most notable in patients aged more than 45 years, females with a disease duration between 5 and 10 years. Many clinical signs were noted in these patients (summarized in Table 1) with frequency of unusual pigmented plaques.

Biological abnormal signs found in these patients were: increased rate of LDH and B2M, with histological phenotype of classic, folliculotropic and transformed mycosis MF.

The means of molecular biology for the analysis of lymphocyte clonality were not available in our hospital and have not been used in any of our patient.

8 cases of our patients with eosinophilia were in the erythrodermic stage of the disease and 6 in the tumoral stage and we had 4 cases of disease-specific death.

Finely, in our study population, the BE was significantly related to some clinical and paraclinical signs such as: Deterioration of the general condition, Depilation of the body, Erythroderma, sclap and nails involvement. Increased rate of LDH and B2M, Histological type of MF with positive Immunohistochemistry for CD4 and CD3 (Table 2).

DISCUSSION

Several studies, mostly focusing on the epidermotropic lymphomas such as the MF and Sezary syndrome groups, have attempted to identify clinical, biological,

Table 1: Showed the principal epidemiological, clinical and paraclinical characteristics of patients with and without BE

PCTL (72)	PCTL (N=/%)	PCTL with BE (N/%): 14/19.4%
Epidemiological characteristics		
Age groups		
15-45 years	17/23.6	4/28.5
>45 years	55/76.3	10/71.4
Gender		
F	39/54.1	9/64
M	33/47.1	5/35.7
Disease duration		
<5 years	28/38.9	5/35.7
5-10 years	29/40.2	7/50
>10 years	15/20.8	2/14.3
Clinical characteristics		
Pain	2/27.7	1/8.1
Pruritus	53/73.6	14/100
Deterioration of the general condition	6/83.3	4/28.5
Erythematous plaques	1/1.38	3/21.4
Erythematous scaly patches	32/44.4	8/57.1
Papules	12/16.6	1/7.1
Nodules	14/19.4	4/28.5
Ulceration	15/20.8	4/28.5
Pigmented patches	23/31.9	5/35.7
Tumors	12/16.6	3/21.4
Erythroderma	20/27.7	8/57.1
Depilation of the body	9/12.5	4/28.5
Poikiloderma	4/5.5	1/7.1
Ichthyosiform state	1/1.38	1/7.1
Localization in the hidden areas	59/81.9	6/42.8
Palmoplantar keratoderma (PPK)	13/18	4/28.5
Lymphadenopathies	32/44.4	9/66.3
Mucosal involvement	4/5.5	3/21.4
Scalp involvement	18/25	10/71.4
Nails involvement	17/23.6	7/50
Leonine facie	4/5.5	1/7.1
Paraclinical characteristics		
High serum level of lactate dehydrogenase (LDH)	18/25	11/78.5
High serum level of B2 micoglobulin (B2M)	16/22.2	12/85.7
Sezary cells	1/1.38	0/0
Histological type	MF 62/86.1	
	Classical MF: 52/83.8	10/71.4
	Folliculotropic MF: 8/12.9	1/7.1
	Transformed granulomatous MF CD30+: 1/1.6	1/7.1
	MF transformed into high-grade CD 30- : 1/1.6	1/7.1
Immunohistochemistry	CD4: 43/59.7	CD4: 12/85.7
	CD3: 48/66.6	CD3: 10/71.4
	CD30: 1/1.38	CD30: 1/7.1

histopathologic, or immunophenotypic characteristics that can predict outcome.

Table 2: The clinical, paraclinical and histological signs significantly associated with BE in our patients

BE (14 cases/72 PCTL)	P value
Deterioration of the general condition	0.001
Depilation of the body	0.04
Erythroderma	0.008
Scalp involvement	0.000
Nails involvement	0.001
Increased rate of LDH	0.000
Increased rate of B2M	0.000
Histological type of MF	0.014
Immunohisto chemistry: CD4	0.015
CD3	0.04

So far, the main prognostic factors identified in this group are the type and extent of the skin involvement, extra cutaneous spread of the disease, initial response to treatment, histologic transformation, high serum level of lactate dehydrogenase (LDH), and the detection of a cutaneous or peripheral blood T-cell clone by polymerase chain reaction [3,4].

Besides, there are a few observations that detect the prognostic significance of BE in patients with T cell malignancies and rarely in PCTL.

BE is associated with a number of different etiologies including parasitic diseases, atopy, allergic reactions, inflammatory bowel disease, rheumatoid arthritis, vasculitis and lymphoma [5].

This BE was reported as a marker of a poor prognosis in Some T-cell malignancies: nodal T-cell lymphoma and Hodgkin's disease.

For exemple, there was a study of 99 consecutive patients with "idiopathic" eosinophilia that demonstrated the presence of clonal T-cells in blood, bone marrow, or other tissue samples of 14 patients including 6 patients who had an overt T-cell malignancy [6].

In Hodgkin disease, several studies investigating eosinophilia [7]: found a worse relapse-free survival rate in patients with eosinophilia than in those without [8-9]. In a more recent study, tissue eosinophilia has been shown to be the strongest prognostic factor for poor relapse-free survival and overall survival in nodular sclerosing hodgkin disease.

Concerning PCTL, Some publications reported the presence of BE [10,11] in patients with cutaneous T non-Hodgkin's lymphoma, and was considered as a poor prognostic factor, but these publications were based on a case report.

In two retrospective inception cohort, this BE was also demonstrated to be related to a poor prognosis of the disease.

The first study was a cohort that included 104 patients with cutaneous T-cell lymphoma, BE was a significant indicator of poor prognosis and increased disease-specific death [12].

The second and recent study [13], demonstrated that: BE was a predictive of more advanced disease ($P < .0001$), increased number of treatment types ($P < .002$), and less responsiveness to treatment ($P < .0006$).

In our study, we found 14 cases of BE, which was significantly related to the histological type of MF, to advanced stages of the disease (tumor and erythrodermic stage), to pejorative clinical signs (deterioration of the general condition, depilation of the body and nail involvement) and to the high levels of the LDH and B2M which are known factors of poor prognosis of MF.

We also had four disease-specific deaths in our patients with BE, which leads us to conclude that blood eosinophilia is also a poor prognostic factor for PCTL - especially for MF - in our study.

Some hypothesis were reported to explain this BE in PCTL and in T cell malignancies: It has been related to the predominant secretion of T helper cell type 2 (T_H2) eosinophilopoietic or eosinophilotactic cytokines (interleukin (IL) 3, IL-5, and sargramostim) by neoplastic cells [14,15]. This T_H2 differentiation has been associated with a relative defect of the antitumoral and anti-infectious response [16], so the hypothesis that eosinophilia might be an indicator of poor prognosis was raised, another theory of Depressed cell-mediated immunity and deficiency in IL-2 and interferon γ production and finally Increased production of IL-4, IL-5, and IL-10 [17,18].

Indeed, In view of these notions, these hypothesis must be tested in further prospective, large-scale studies, and such research might help to identify subgroups of patients who might benefit from immunotherapeutic approaches such as IL 12, IL 2, and interferon gamma, which are likely to correct the abnormal cytokine production observed in PTCL [19-21].

This fact that BE is a poor prognosis factor, must leads clinicians to choose some therapeutic options and aggressive treatments from the beginning instead of losing time by applying topical therapies.

This study have some limitations such as: the limited number of patients, the duration of follow-up is unsatisfactory for the study of survival and there was no correlation between the BE and molecular biology to the study of lymphocyte clonality.

CONCLUSION

Blood eosinophilia must be taken Into account in patients having PCTL especially MF because it's a non invasive factor for evaluating the disease prognosis and therapeutic options.

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.

Abbreviations

Blood eosinophilia: BE; Cutaneous T cell lymphoma: CTL; Mycosis fungoides: MF; Beta 2 microglobulin: B2M; Lactate Dehydrogenase: LDH; Primary cutaneous lymphomas: PCL T cell lymphomas: PCTL; Pleomorphic small-to medium-sized CTCLs: PSMs; The European Organization for Research and Treatment of Cancer classification: EORTC classification; Sezary syndrome: SS; Palmoplantar keratoderma: PPK

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Our Dermatology Online

Original Article

Dermatologic challenges of health care for displaced people. lessons from a German emergency refugee camp

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ABSTRACT

Background: The World faces the highest waves of displaced people since World War II. There is limited knowledge about need of dermatological care for refugees and asylum seekers. **Methods:** We report the experience with a temporary emergency refugee camp in Dresden form the viewpoint of a hospital department. This is a descriptive report covering the period of 10 weeks. **Results:** In this refugee camp up to 1 100 people were hosted. The male to female ratio was 5.3. The majority of inhabitants were young males (60%), 20% were children. While 40% of refuges came from Syria, Afghanistan, Iraq and Pakistan were also important countries of origin. Communication war a crucial issue while providing health care. Dermatologic service was granted as consultation, outpatient and inpatient clinic. Most contacts were noted in the outpatient clinic. The majority of patient attended the clinic with communicable diseases such as bacterial or viral infections and infestations. Wounds and chronic inflammatory diseases were rather uncommon. Only 4 patients had to be treated in the hospital (inpatient clinic). **Conclusions:** Displaced people (refugees, asylum seekers) come in big waves to Europe. Dermatologic service is an important part of first aid health care in an emergency camp. Language barriers and cultural barriers have to overcome for optimal service. This is the first report from Germany.

Key words: Refugee camp; Dermatology; Leishmaniasis; Scabies; Communicable diseases; Service

INTRODUCTION

For the first time since 1999 Germany was the World's largest recipient of individual asylum application [1]. This has a manifold impact on society and last not least on health care. Health care is regulated by law and this is also the case for asylum seeker, i.e. national Law on Services for Asylum Seekers (AsylBLG sections 4 and 6). Health care service based on this law is free for asylum seekers.

Since the German health care system does not collect data on health status and health care provision to asylum seekers important data are missing to deal with this growing problem for planning organizing and optimizing health care efforts [2]. Searching on PUBMED® we could identify a single paper on dermatologic problems in asylum seekers from Turkey [3] but none from Germany.

Therefore, we present here an empiric study from Dresden, Germany.

MATERIAL AND METHOD

The increasing numbers of asylum seekers coming from Syria, Iraq, Libya, Eritrea, Afghanistan, Pakistan and Eastern Europe has been a challenge for Europe and in particular for Germany. This has resulted in emergency situations to provide shelter for asylum seekers. On July 23rd, 2015 the German Red Cross (DRK) and the Federal Agency for Technical Relief (THW) established overnight a temporary Refugee Camp with 33 tents, where during the following weeks until October 9, 2015, up to 1,100 asylum seekers found shelter. Each tent had space for about 30 people which translates into 1.5 m^2 per person. Due to the circumstances, no systematic

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preparation of the health care needs was possible. The first local service was provided within the camp by volunteers. With some delay a stable arrangement of first aid services in the camp could be established.

The nearest hospital was the Hospital Dresden-Friedrichstadt, where the Department of Dermatology and Allergology provided dermatologic support. Initially the hospital was the only professional provider of health service except for volunteers. The circumstances caused an additional work load for nurses and doctors mainly in the morning and night shift. Patients usually were companioned by several other people. Health services were free for the asylum seekers.

The major cause for consultations, in- and outpatient treatment was communicable disorders (bacterial and viral infections, scabies and mycosis) in our setting.

Demographics of asylum seekers

On average 60% of asylum seekers were adult males, 20% adult females and 20% children and adolescents. The male to female ratio of all asylum seekers was 5.3. 46% of asylum seekers were married, 11% unmarried, and in 6% the status was unknown.

About 40% of asylum seekers came from Syria, 15% from Afghanistan, 10% from Iraq, 5% from Pakistan, and 30% of various other countries.

Dermatologic service

Dermatologic service was provided as counselling, outpatient treatment and inpatient treatment. Dermatologic service was requested in 19% of all asylum seekers treated by the hospital. Consultations were ordered 8 times for scabies (2x), skin ulcers (2x), Varicella (1x), allergic sting reaction (1x), acute urethritis (1x), and xerosis cutis (1x).

A significant issue is availability of interpreters. English was not spoken by many of the refugees, Arabic interpreters were available. More difficult was communication with people speaking Dari, Pashto, Urdu or Tigrinya. Interpreters were generated from hospital staff and municipal interpreter service (Gemeinde-Dolmetscherdienst).

Outpatient care was the dominant type of service engaged. We saw 52 patients with a wide range of skin diseases. The leading diagnosis was scabies (16x), followed by eczematous dermatitis (5x), and impetigo (4x). About 65% of outpatient diagnoses were infectious diseases and infestations. For further details see Table 1.

Follow-up was realized by the Health Service that had been established in the refugee camp later. After closure of the tent camp and accommodation of refugees in houses, a central outpatient health service was opened in Dresden (https://www.slaek.de/de/01/03Empfehlungen/08Asylbewerber.php).

Inpatient treatment

Only 4 patients needed an inpatient treatment. Three of them had infectious diseases (Varicella, cheek abscess, impetiginized eczema). The other patient suffered from anaphylaxis after an insect sting.

DISCUSSION

There are currently more displaced people around the world since 2nd World War. This has led to emergency situations with an enormous impact on Europe.

In 2011 the Civil War in Syria has started. This had a great impact on neighboring countries such as Jordan, Lebanon, and Turkey. In a study from the southeastern Turkish city of Kahramanmaraş, a more than six-fold increase of cutaneous leishmaniasis had been observed between 2011 and 2013.4 Studies from Jordan where a large population of refugees from Syrian Civil War found shelter suggest that there is a significant percentage of

Table 1: Diagnoses in outpatient treatment of asylum seekers

Diagnosis	Number of patients
Scabies	16
Eczematous dermatitis	5
Impetigo	4
Varicella	3
Folliculitis	3
Abscess	3
Urticaria	3
Cysts	2
Sting reactions	2
Leg and foot ulcer	2
Erysipelas	1
Mycosis	1
Polymorphic light eruption	1
Pyoderma	1
Psoriasis	1
Mollusca contagiosa	1
Gonorrhoea	1
Hyperpigmentation after chemical burn	1

patients with non-communicable diseases, in particular chronic diseases like cardiovascular, diabetes or chronic respiratory [4].

In our specific situation in Dresden, the establishment of an emergency refugee camp in from 23 July to 9 October 2015, with up to 1,100 inhabitants has challenged the organization and structures of the neighboring hospital due to a lack of other official structures of health care. Volunteers cannot replace official and stable infrastructure of health service [5].

More than 65% of dermatological cases were due to communicable infectious (bacterial, viral and mycotic) diseases and scabies. Surprisingly, we did not observe cutaneous leishmaniasis amongst those asylum seekers, although a major part came from Syria. Syria is one of the hotspots for cutaneous leishmaniasis among the Mediterranean countries with *Leishmania major* and *Leishmania tropica* as the main aetiological agents [6]. Other hotspots are Afghanistan and Pakistan with a rising prevalence [7]. Although we found no case of cutaneous leishmaniasis in the temporary refugee camp, the disease will not stop at the borders and we must be aware of this disease [8].

The high prevalence of scabies and infectious disorders argues for a screening when displaced people enter a refugee camp. This was not established in the first weeks after opening of the camp. By this important tool, however, spread of communicable diseases can be prevented.

In case of endemic scabies ivermectin is an alternative to permethrin [9]. In Germany, ivermectin is off-label for scabies. Therefore the treatment was realized by topical permethrin for index patients and family members.

Health service, however, can only be successful when the communication with asylum seekers and health care professionals can be ensured. This is not restricted to the service of interpreters but other issues like overcome of cultural barriers [10].

This is the first empirical study of dermatologic health care needs in refugees/asylum seekers in Germany. Our study is limited by the fact of a temporary tent camp, what will not reflect the situation after prolonged stay of people, and the single center experience. Nevertheless, the data argue for the importance of dermatologic care and treatment in displaced people [11,12].

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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Our Dermatology Online

Original Article

Evaluation clinique du délai de cicatrisation des lésions d'ulcère de Buruli de diamètre inférieur ou égal à 10 centimètres à Pobè (Bénin)

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RESUME

Introduction: L'ulcère de Buruli est une affection cutanée due à *Mycobacterium ulcerans*. L'objectif était d'évaluer le délai de cicatrisation des lésions de l'ulcère de Buruli de diamètre ≤ 10cm. **Patients et Méthodes:** Il s'agissait d'une étude rétrospective, descriptive et analytique, réalisée de 2010 à 2012 à Pobè (Bénin). Ont été inclus, les patients ayant eu une PCR positive au *Mycobacterium ulcerans*, des lésions de diamètre ≤ 10cm et traités suivant les recommandations de l'OMS. **Résultats:** Au total, 104 patients ont été retenus. Les plaques ulcérées représentaient la forme clinique la plus fréquente (57%). Le délai moyen de consultation était de 16,6 ± 19,6 semaines. Les patients présentant une lésion ulcérée et ayant consulté moins de 5 semaines après le début de la maladie, avaient un délai moyen de cicatrisation de 85,1 ± 33,7 jours versus 146,1 ± 80,2jours pour ceux ayant consulté 27 semaines après le début de la maladie *($p < 0,05$)*. Le délai moyen de cicatrisation des formes non ulcérées de diamètre ≤ 5cm était de 105,1 ± 59,5 jours versus 111,2 ± 44,3 jours pour celui des formes ulcérées de 5cm ≤ diamètre ≤ 10cm *($p = 0,04$)*. **Conclusion:** Le retard à la consultation allonge le délai de cicatrisation des lésions. Les lésions non ulcérées quelles que soient leurs tailles ont un délai de cicatrisation plus long que celui des lésions ulcérées. Les résultats de ce travail devraient susciter d'autres études pour de nouvelles perspectives thérapeutiques de cette affection.

Mots clés: Ulcère de Buruli; Diamètre de lésion; Évaluation; Délai de cicatrisation; Bénin

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Our Dermatology Online

Original Article

Clinical evaluation of the deadline of healing of the ulcer of Buruli hurts of diameter lower or equal to 10 centimeters in pobe (Benin)

Christiane Koudoukpo[1], Félix Atadokpede[2], Laouali Salissou[3], Françoise Assogba[1], Fabrice Akpadjan[2], Bérénice Degboe[2], Nadège Agbessi[1], Hugues Adégbidi[2]

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ABSTRACT

Introduction: Ulcer of Buruli is a skin disorder due to *Mycobacterium ulcerans*. The objective was to estimate the deadline of healing of the hurts of the ulcer of ulcer of Buruli of diameter ≤ 10 cm. **Patients and Methods:** It was about a retrospective, descriptive and analytical study, realized from 2010 to 2012 in Pobè (Benin). Were included the patient, having had a positive PCR in *Mycobacterium ulcerans*, hurts of diameter ≤ 10 cm and treated according to the recommendations of the WHO. **Results:** A total of 104 patients have been included. The hurt patches represented the most frequent clinical shape (57%). The average deadline of consultation was 16.6 ±19.6 weeks. The patients presenting a hurt lesion and having consulted less than 5 weeks after the beginning of the disease, had an average deadline of healing of 85.1 ± 33.7 days versus 146.1 ± 80.2 days for those having consulted 27 weeks after the beginning of the disease *(p <0.05)*. The average deadline of healing of forms by diameter ≤ 5cm was 105.1 ± 59.5 days versus 111. ± 44.3 days for that of hurt forms of 5 cm ≤ diameter ≤ 10 cm *(p = 0.04)*. **Conclusion:** Delay in the consultation lengthens the deadline of healing of the hurts. The not hurt lesions whatever are their sizes have a deadline of healing longer than that of the hurt lesions. The results of this work should arouse other studies for new therapeutic perspectives of that affection.

Key words: Buruli ulcer; Hurt diameter; Evaluation; Healing deadline; Benin

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INTRODUCTION

L'ulcère de Buruli est une affection potentiellement handicapante due au *Mycobacterium ulcerans*, caractérisé par une nécrose du tissu cutané et sous cutané avec souvent atteinte des os. Il sévit sous forme endémique dans certaines régions du monde dont plusieurs cas ont été notifiés en Afrique centrale, en Afrique de l'ouest, en Amérique latine, en Asie, dans le Pacifique Oriental, et en Australie [1]. C'est une maladie tropicale négligée car la première publication a été faite en 1948 et ce n'est qu'en 1980 qu'un cri d'alarme a été lancé dans le monde [2]. Sa prise en charge associe un traitement médical et un traitement chirurgical [3]. L'objectif de cette étude était d'évaluer le délai de cicatrisation des lésions de l'ulcère de Buruli de diamètre ≤ 10cm dans le Centre de Dépistage et de Traitement de l'Ulcère de Buruli (CDTUB) de l'Ouémé-Plateau à Pobè (Bénin).

MATERIELS ET METHODES

Il s'est agi d'une étude rétrospective, descriptive et analytique, réalisée 1er Janvier 2010 au 31 Décembre 2012 dans le centre de Dépistage et de Traitement de l'Ulcère de Buruli de l'Ouémé-Plateau (Bénin). Ont été inclus dans cette étude, les patients ayant eu une PCR positive au *Mycobacterium ulcerans*, des lésions de diamètre ≤ 10cm et traités suivant les recommandations de l'OMS qui sont utilisées depuis 2004. Chaque patient a bénéficié d'un protocole de traitement comportant un volet médical et un volet chirurgical. Le traitement médical était fait de biantibiothérapie: la Streptomycine (S) oula Clarithromycine (C) associée à la Rifampicine (R) pendant 8 semaines permettant l'intervention chirurgicale.

RESULTATS

Au total 419 patients ont été admis pour un ulcère de Buruli. Parmi eux, 104 répondaient aux critères d'inclusion. Sur les 104 patients, 71 avaient moins de 15 ans (68,3%) et 33 avaient plus de 15 ans (31,7%). L'âge variait entre 1 et 80 ans, avec une médiane de 11ans. Les femmes étaient plus représentatives soit un sex ratio de 0,89. Soixante dix neuf patients (n = 79) avaient consulté au-delà de 5 semaines après le début de la maladie (Fig. 1).

La plaque ulcérée (Figs. 2 et 3) (n = 60) représentait la forme clinique la plus fréquente (57%). L'ensemble des lésions ulcérées représentaient 73%, et celles non ulcérées 27% (Figs. 3 et 4).

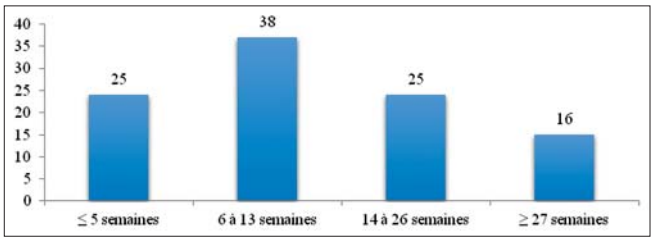

Figure 1: Répartition des sujets en fonction du délai de consultation.

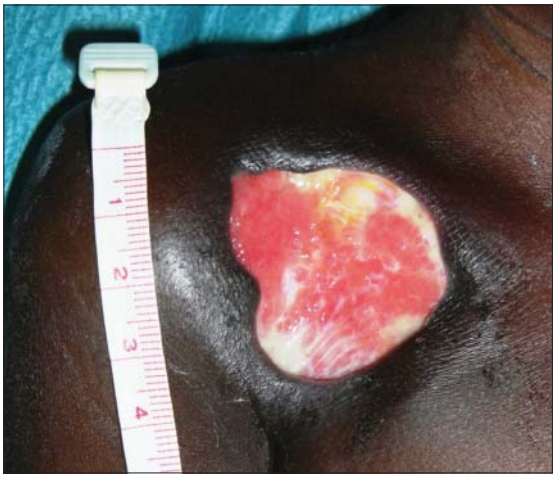

Figure 2: Lésion ulcérée, à bords décollés.

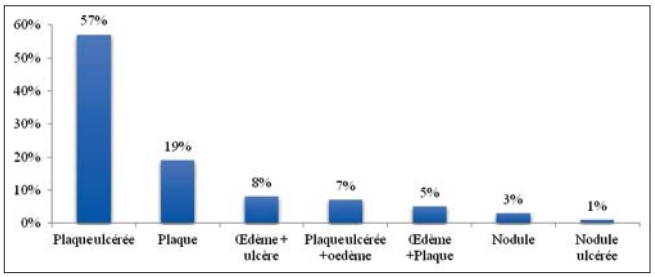

Figure 3: Répartition des formes cliniques des lésions.

La moyenne du délai de cicatrisation (Fig. 5: lésions cicatrisées) était de 92 ± 46,7 jours avec une médiane de 81 jours. Les patients qui avaient une lésion ulcérée et qui ont consulté en moins de 5 semaines avaient un délai moyen de cicatrisation de 85,1 jours et ceux ayant consulté 27 semaines après ont un délai moyen de cicatrisation de 146,1 jours (Tabl. 1).

Les patients qui avaient une lésion non ulcérée et qui ont consulté en moins de 5 semaines ont un délai moyen de cicatrisation de 104,6 ± 66,9 jours et ceux ayant consulté à plus de 27 semaines ont un délai moyen de cicatrisation de 110 ± 4,2 jours (Tabl. 2).

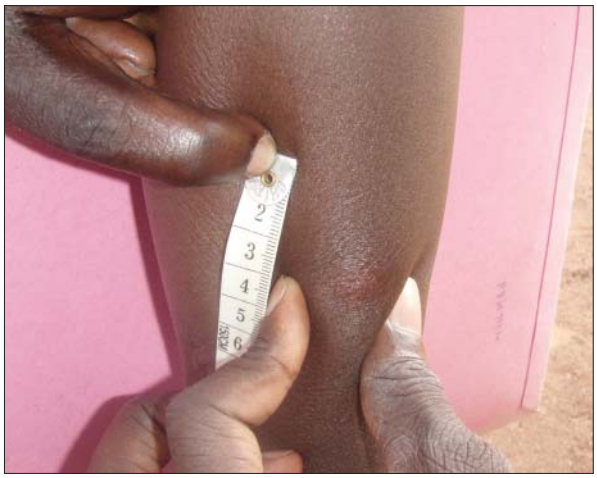

Figure 4: Lésion non ulcérée (nodule).

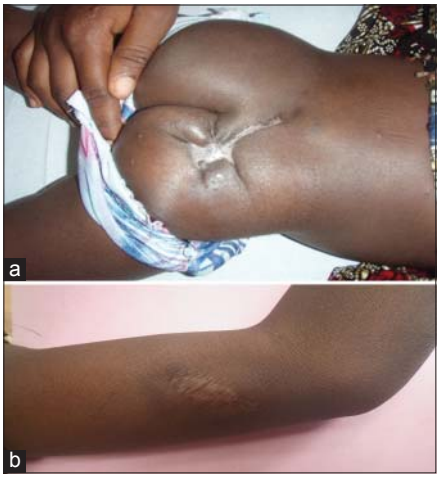

Figure 5: Lésions cicatrisées (a: Ulcérée, b: Nodulaire).

Le délai moyen de cicatrisation des lésions ulcérées de diamètre <5cm est de 89,1 ± 55,1 jours, et celui des lésions dont le diamètre était compris entre 5 et 10 cm était de 93,1 ±41,3 jours (Tabl. 3).

Le délai moyen de cicatrisation des lésions non ulcérées de diamètre <5cm était de 105,1 ± 59,5 jours, et celui des lésions dont le diamètre était compris entre 5 et 10 cm était de 111,2 ± 44,3 jours (Tabl. 4).

Les formes non ulcérées avaient une durée totale d'évolution de la maladie plus longue que les formes ulcérées. La durée moyenne d'évolution de la maladie était de 109,01 ±50,26 jours avec des extrêmes allant de 48 à 377 jours et une médiane de 130 jours.

Les formes non ulcérées avaient une durée d'évolution moyenne de maladie de 109,9 ±52,8 jours alors que les formes ulcérées avaient une durée d'évolution moyenne de maladie de 108,6 ± 49,5 jours (Tabl. 5).

Tableau 1: Délai moyen de cicatrisation des lésions ulcérées en fonction du délai de consultation

Délai de consultation (semaines)	Délai de cicatrisation (en jours)		*p*
	Moyenne	Ecart type	
≤5	85,1	33,7	0,0004
6-13	96,7	33,6	
14-26	114,4	36,7	
≥27	146,1	80,2	

Tableau 2: Délai moyen de cicatrisation des lésions non ulcérées en fonction du délai de consultation (en semaines)

Lésions non ulcérées selon le délai de consultation (semaines)	Délai de cicatrisation (en jours)		*p*
	Moyenne	Ecart type	
≤5	104,6	66,9	0,1
6-13	107,0	39,5	
14-26	143,6	66,5	
≥27	110,0	4,2	

Tableau 3: Répartition du délai moyen de cicatrisation des lésions ulcérées en fonction de leur diamètre

Diamètre des lésions ulcérées	Délai de cicatrisation (en jours)		*p*
	Moyenne	Ecart type	
<5 cm	89,1	55,1	0,04
5 et 10 cm	93,1	41,3	

Tableau 4: Répartition du délai moyen de cicatrisation des lésions non ulcérées en fonction de leur diamètre

Diamètre des lésions non ulcérées	Délai de cicatrisation (en jours)		*p*
	Moyenne	Ecart type	
<5 cm	105,1	59,5	0,04
5 et 10 cm	111,2	44,3	

Tableau 5: Répartition de la durée totale d'évolution de la maladie en fonction de la forme clinique de la lésion

Forme clinique de la lésion	Durée d'évolution totale de la maladie (en jours)		*p*
	Moyenne	Ecart type	
Non ulcérée	109,9667	52,8064	0,6**
Ulcérée	108,6301	49,5548	

Ethics

This study was performed on human subjects; thus, all patients were aware of the presence of the study and they were fully informed about the drug and its side-effects.

DISCUSSION

La population d'étude était composée de 68,3% de patients de moins de 15 ans et de 31,7% de plus de 15 ans avec des extrêmes de 1 à 80 ans et une médiane de 11ans. Ces résultats étaient conformes à ceux d'autres auteurs qui avaient retrouvé une prédominance

chez les moins de 15 ans [2,4]. Pour Delphin et al les moins de 15 ans représentaient 44% [5]. Clancey et al expliquaient cette fréquence élevée d'ulcère de Buruli chez l'enfant par son immunisation faible que celle de l'adulte; mais pourrait être liée aussi à l'exposition constante des enfants lors des activités ludiques menées dans les régions marécageuses [6].

Le sex-ratio était de 0,89. La prédominance féminine retrouvée dans notre étude était en accord avec celle de Barker [2] en 1973 et de Kadio [7] en 1990; cependant, Sopoh et al [8] au Bénin et Van der Werf [9] au Ghana rapportaient une prédominance masculine. Mais selon l'OMS l'ulcère de Buruli touche indifféremment les hommes et les femmes et la prédominance d'un sexe sur l'autre serait en rapport avec les caractéristiques socioéconomiques de la population étudiée et la méthodologie [10].

Dans notre étude 73% des patients avait consulté dans un délai inférieur à 6 mois. Ouattara et al [11] ont rapporté un pourcentage inférieur à celui de notre série, dans une étude réalisée en Côte d'Ivoire avec 55% de patients ayant consulté avant 6 mois. Dans leur étude Delphine al rapportaient que dans le centre de Zagnanado au Benin, 63% des patients étaient plutôt référés [5]. On peut ainsi affirmer que l'amélioration du délai de consultation dans notre étude pourrait s'expliquer par: la création des CDTUB dans les zones endémiques, la forte sensibilisation des populations à risque, le dépistage précoce des cas par les agents de santé et la prise en charge du coût du traitement par le (Programme National de Lutte contre l'Ulcère de Buruli) PNLUB [12].

Dans notre étude, les lésions avaient un délai médian de cicatrisation d'environ 12 semaines (81 ± 46,7 jours). Ce résultat était en accord avec celui de Chauty et al [13] qui avaient noté dans une étude pilote à Pobè (Bénin) un délai médian de cicatrisation de 15 semaines soit 104 jours. Cette légère différence observée serait due à la taille de l'échantillon dans l'étude de Chauty et al (30 patients) [13]. De même Sarfo et al [14] dans une étude sur toutes les catégories de lésions avaient obtenu un délai de cicatrisation de 3 mois.

De nos résultats il ressort que plus précocement le patient consulte mieux la lésion se cicatrise rapidement quelle que soit sa forme clinique initiale ($p < 0,05$ pour les lésions ulcérées et $p > 0,05$ pour les lésions non ulcérées). L'accélération de la cicatrisation chez les patients qui ont consulté tôt serait due aux différents soins (l'antibiothérapie, les pansements, les détersions chirurgicales et la kinésithérapie) dont ont bénéficié les patients dans les CDTUB [14]. Aussi nos résultats ont montré que les lésions de diamètre inférieur ou égal à 5cm ont cicatrisé plus vite que les lésions qui avaient un diamètre compris entre 5 et 10 cm ($p < 0,05$). Ce résultat était en accord avec celui de Chauty et al [13] et compatible avec les objectifs de l'OMS qui encourage le dépistage précoce des cas pour une guérison rapide [15]. Cependant les lésions non ulcérées quelque soit leurs tailles ont un délai de cicatrisation plus long que les lésions ulcérées ($p < 0,05$). Cela serait dû au fait que les lésions non ulcérées passent d'abord par la phase d'ulcération qui est relativement longue d'un individu à un autre; il pourrait également être expliqué par le fait que plus la taille de l'ulcère est grande, plus l'induration est ouverte et plus les nécroses s'éliminent facilement [7,16].

Dans notre série, la durée totale d'évolution de la maladie dans les lésions non ulcérées avoisinait celle des lésions ulcérées (environ 16 semaines). Ce résultat pourrait s'expliquer par le fait que les lésions non ulcérées ont un délai avant consultation court et un délai de cicatrisation long tandis que les lésions ulcérées ont un délai de consultation long et un délai de cicatrisation plus court. La durée totale d'évolution de la maladie a un véritable impact social et économique car elle inactive les patients et leurs assistants. Sachant que la position d'un individu dans la structure sociale est étroitement associée à son état de santé, l'ulcère de Buruli, comme toutes les maladies négligées, est une maladie de la pauvreté avec impact d'inégalité sociale [3,17-19]. De ce fait, le contrôle de l'ulcère de Buruli dépend de l'action sur les déterminants sociaux [20].

CONCLUSION

Le retard à la consultation allongeait le délai de cicatrisation des lésions. Les lésions non ulcérées quelles que soient leurs tailles avaient un délai de cicatrisation plus long que celui des lésions ulcérées. Les résultats de ce travail devraient susciter d'autres études pour de nouvelles perspectives thérapeutiques de cette affection. Cependant la sensibilisation de la population vis-à-vis de cette maladie endémique reste l'arme stratégique pour une prise charge précoce et adéquate.

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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Our Dermatology Online

Original Article

Cutaneous hypopigmentary disorders - An observational study

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ABSTRACT

Background: Hypopigmented skin lesions is very common among people of all age groups. There are limited studies in India about evaluation of hypopigmented skin conditions. The aim of my study is to evaluate the different etiologies of cutaneous hypopigmentation. **Aim:** The present study was undertaken to find the relative incidence of the various disorders causing a hypopigmented lesion in a random sample of 200 cases and to study site, distribution and characteristics of the lesions. **Methods:** A random sample of 200 patients presenting with one or more hypopigmented lesions to the outpatient department of Dermatology, Venereology and Leprosy in KVG Medical College and Hospital, Sullia from December 2011 to January 2013 was studied. Detailed history including address and occupation with special reference to onset and duration, preceding skin conditions, exposure to chemicals, topical application and family history was taken. Various characteristics of the lesion like site, size, number, distribution, surface and sensation were studied. After this samples were taken for relevant investigations like complete hemogram, biopsy, slit skin smear, KOH mount and assessed for the causes of hypopigmented lesions. **Results:** In our study, most common cause with cutaneous hypopigmentation was pityriasis versicolor, seen in 52%, followed by post inflammatory hypopigmentation in 32%, pre vitiligo in 6.5%, Hansen's disease, idiopathic guttate hypomelanosis, nevus anemicus each in 2%, Woronoff's ring in 1.5% and miscellaneous conditions in 2% of the cases. Commonest age group affected was 21-30 years. Males (49%) and females (51%) were almost equally affected. **Conclusion:** The study concludes that various conditions comes under hypopigmentary disorders. More common in young adults. Most common scaly condition was pityriasis versicolor and non scaly condition was pre vitiligo. Proper counseling and ruling out Hansen's disease is required to alleviate the patient's anxiety.

Key words: Hypopigmentary disorders; Pityriasis versicolor; Post inflammatory hypopigmentation; Pre vitiligo, Hansen's disease

INTRODUCTION

Skin is the largest organ of the body and the only organ which is visible and is in direct contact with the environment [1]. It has been said that the greatest problems in this world are very tiny, the atom, the ovum and a touch of pigment. The largest organ of the body very commonly suffers from this touch of pigment.

Numerous skin conditions cause alteration in the normal pigmentation resulting in significant psychological morbidity due to cosmetic disfigurement. Pigmentary disturbances may be congenital or acquired, circumscribed or generalised, hypomelanotic or hypermelanotic [2].

This study strives at the various skin conditions presenting as hypopigmentation. With attention to variability of extent of hypomelanosis, history of evolution, attention to hue and awareness of ancillary features, the differential diagnosis will be narrowed down and definite diagnosis will be arrived at with the help of relevant investigations. An attempt will also be made to find the relative incidence of each condition.

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METHODS

A clinical study was conducted on 200 patients who presented with hypopigmented lesions were selected over a period of 2years from out patient department of Dermatology in a tertiary care hospital. The study included patients of pediatric as well as adult age group presenting with one or more hypopigmented lesions. Both scaly and non scaly presentations were included. Cases with depigmented lesions including those of established vitiligo, chemical leukoderma and leukoderma secondary to topical applications were excluded.

Cases with lesions only over the face and/or mucosae and cases with generalised hypomelanosis were also excluded from the study.

Detailed history including address and occupation with special reference to onset and duration, preceding skin conditions, exposure to chemicals, topical application and family history was taken. Various characteristics of the lesion like site, size, number, distribution, surface and sensation were studied along with nail, hair, mucosal examination and examination of the palms and soles. Care was taken to find out any associated conditions coexisting with the primary disease.

Relevant investigations including routine hemogram, scraping for KOH mount, slit skin smear, Wood's lamp examination and skin biopsy were done. Analysis of each of the diseases was done and results compiled.

Ethics

This study was performed on human subjects; thus, all patients were aware of the presence of the study and they were fully informed about the drug and its side-effects.

RESULTS

A total of 200 patients were included in study. There were 98 males(49%) and 102 females (52%) with ratio of 0.96:1 (Table 1). Maximum numbers of cases were seen between 21-30 years of age group comprising 36% of the total. Of the various diseases studied, 52% of the cases were pityriasis versicolor, 32% were post inflammatory hypopigmentation, 6.5% were pre vitiligo, 2% each of leprosy, idiopathic guttate hypomelanosis, nevus anemicus, 1.5% Woronoff's ring and 0.5% each comprised of lichen sclerosus et atrophicus, halo nevus, nevus achromicus and hypomelanosis of Ito (Table 2). Regarding sites involved, many cases had more than one site that was involved. In 89 cases, trunk was the predominant site involved. Of the 64 cases of post inflammatory hypopigmentation, 33 cases (51.57%) cases were polymorphous light eruptions, 15.63% cases were psoriasis, 14.07% cases were pityriasis rosea, 6.25% were lichen striatus, 3.12% each of dermatitis herpetiformis, pemphigus vulgaris, pityriasis lichenoides et varioliformis acuta and 1.56% each of irritant contact dermatitis and pityriasis alba. Of the 104 cases of pityriasis versicolor, 93 cases (89.42%) were KOH positive, showed the presence of fungal elements. 84 cases showed yellow fluorescence under Wood's lamp. One case of borderline tuberculoid leprosy showed the presence of bacilli in slit skin smear examination. Histopathology was carried out for 9 cases. This included 4 cases of Hansen's disease, 2 cases of psoriasis, 2 cases of dermatitis herpetiformis and 1 case of pemphigus vulgaris. In all cases, histopathological findings correlated well with clinical findings.

Of the various diseases studied, 163 (81.5%) cases were classified as scaly and 37 (18.5%) cases were nonscaly (Table 3).

DISCUSSION

Numerous skin conditions cause alteration in the normal pigmentation resulting in significant psychological morbidity due to cosmetic disfigurement. Pigmentary disturbances may be congenital or

Table 1: Age and sex wise distribution

Age in years	Male		Female		Total	
	No	%	No	%	No	%
<1	-	-	1	0.5	1	0.5
1 to 10	10	5	6	3	16	8
11 to 20	23	11.5	24	12	47	23.5
21 to 30	33	16.5	39	19.5	72	36
31 to 40	19	9.5	19	9.5	38	19
41 to 50	10	5	8	4	18	9
>50	3	1.5	5	2.5	8	4
Total	98	49	102	51	200	100

Table 2: Relative incidence of hypopigmented skin conditions

Diagnosis	n	Percentage
Pityriasis versicolor	104	52
Post inflammatory hypopigmentation	64	32
Pre vitiligo	13	6.5
Leprosy	4	2
Idiopathic guttate hypomelanosis	4	2
Nevus anemicus	4	2
Woronoff's ring	3	1.5
Lichen sclerosus et atrophicus	1	0.5
Hypomelanosis of ito	1	0.5
Nevus achromicus	1	0.5
Halo nevus	1	0.5
Total	200	100

Table 3: Scaly and nonscaly lesions

Nonscaly conditions	Scaly conditions
Leprosy	Pityriasis versicolor
Pre vitiligo	Resolving psoriasis
Lichen sclerosus et atrophicus	Polymorphous light eruption
Idiopathic guttate hypomelanosis	Resolving Pityriasis rosea
Nevus anemicus	Pityriasis lichenoides et varioliformis acuta
Nevus achromicus	Pityriasis alba
Hypomelanosis of ITO	Lichen striatus
Woronoff's ring	
Dermatitis herpetiformis	
Irritant contact dermatitis	
Pemphigus vulgaris	
Halo nevus	

acquired, circumscribed or generalised, hypomelanotic or hypermelanotic.

In this study, tinea versicolor formed the majority of cases (104). The male predominance with 60 cases and commonest age group 21-30 years seen in this study correlates with previous studies [3,4]. The commonest distribution was over upper chest, back and neck and lesions were hypopigmented, well defined with pencil line border and branny scaling as documented in literature (Fig. 1) [5]. Positive scraping for the fungus with spaghetti and meatball appearance on KOH mount was found in 93 cases out of 104 (89.42%). A previous study had reported 98% positivity [5].

13 cases of pre vitiligo showed male preponderance not consistent with previous reports [6]. The commonest site was upper back and age group affected 11-20 years also not coincides with recent studies [7,8]. Associated diabetes seen in this study has been documented [9]. Lesions were ill defined, non scaly with associated mucosal involvement and leucotrichosis seen in few cases.

4 cases of Hansen's disease were seen and the female predominance was not consistent with previous studies [10]. The predominance in age group 21-30 was against the reported bimodal distribution. The commonest type seen was borderline tuberculoid which was also reported by Indian studies [11]. Lesions of BT Hansen were non scaly, well defined at some and ill defined at other areas with definite impairment of sensation associated with asymmetrical nerve thickening as cited in literature (Fig. 2). Patient with TT type of leprosy had solitary well defined non scaly hypopigmented patch with loss of sensation and nerve thickening. One case of BT Hansen had positive slit skin smear. Skin biopsy findings were consistent with literature.

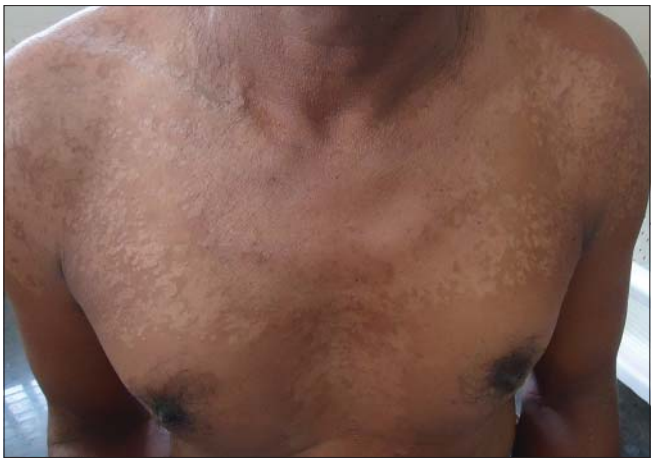

Figure 1: Multiple hypopigmented patches with branny scales in pityriasis versicolor

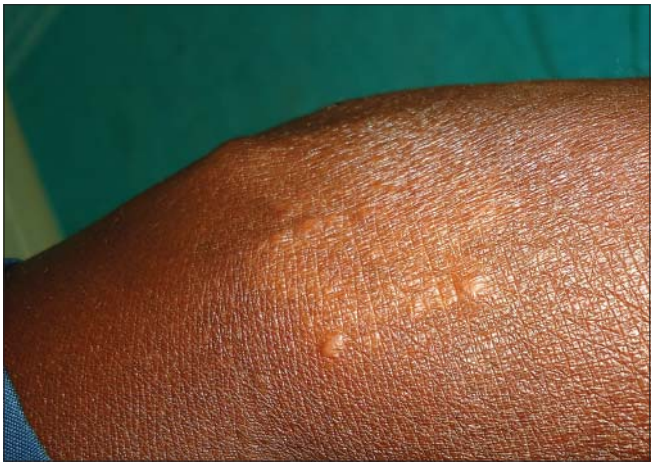

Figure 2: Hypopigmented patch in borderline tuberculoid leprosy

Post inflammatory hypopigmentation formed the second major group in this study. Hypopigmentation following the commonest causes seen in this study i.e. polymorphous light eruption, psoriasis, pityriasis rosea and pityriasis lichenoides et varioliformis acuta has been documented [12]. Polymorphous light eruption was the commonest cause and the predominance of young females (21-30year age group) seen in this study as well as the commonest sites of dorsa of forearms and nape of neck correlates with the description in literature [13]. Polymorphous light eruption was the commonest cause and the predominance of young females (21-30year age group) seen in this study. Psoriasis (Fig. 3), pityriasis rosea, parapsoriasis forms the other conditions leaves behind hypopigmentation.

Idiopathic guttate hypomelanosis is seen in 2 females and 2 males in our study.

The low incidence seen in this study could be due to the fact that the asymptomatic nature and

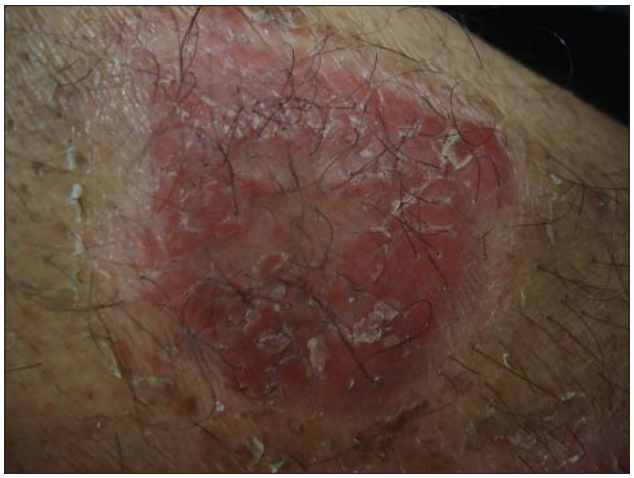

Figure 3: Post-inflammatory hypopigmentation seen around a psoriatic plaque.

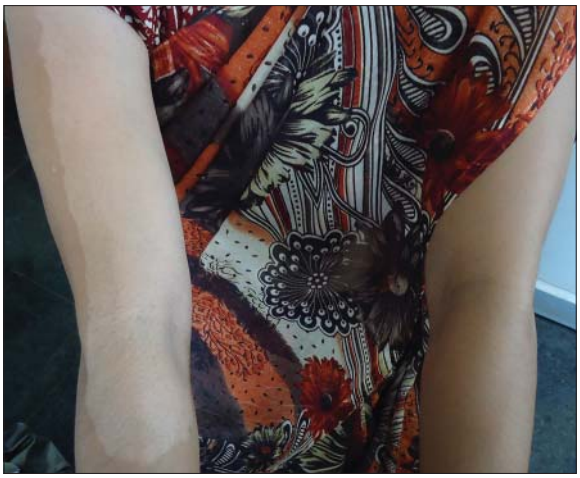

Figure 4: Nevus achromicus

occurrence over cosmetically unimportant sites of this condition prompts patients to ignore it and not seek treatment [14].

A case of nevus achromicus was noted in our study. Lesions were hypopigmented, non scaly, well defined stable since birth and were asymptomatic as described in literature. A study of 20 cases of nevus achromicus showed similar clinical presentation but extracutaneous features like mental retardation and seizures reported in the study were not seen in this study [15].

Hypopigmented atrophic lesions of extragenital lichen sclerosus et atrophicus were seen. The findings of atrophic epidermis with glassy dermal collagen were consistent with literature.

A case with characteristic whorled hypopigmented lesions along lines of Blashko was seen (Fig. 4). No associated extracutaneous manifestations were seen in this case. Though according to literature 75% cases have extracutaneous manifestations, cases without any such abnormalities have been reported [16].

The study concludes that various conditions comes under hypopigmentary disorders. More common in young adults. Most common scaly condition was pityriasis versicolor and non scaly condition was pre vitiligo. Proper counseling and ruling out Hansen's disease is required to alleviate the patient's anxiety.

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Our Dermatology Online

Original Article

Nail disorders in children, a clinical study

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ABSTRACT

Introduction: Aims of the study to investigate the frequency and the nature ofnail disorders in children significant clinical data is available. Nail disorders although common in children in some parts of our country. This study was carried out to document the clinical and demographic pattern of nail disorders in a dermatology outpatient clinic of a pediatric hospital in Ankara, Turkey. **Material and Methods:** All consecutive patients a total of 3000 children from age 0-16 were admitted to dermatology outpatient clinic of Ankara Pediatric Hematology and Oncology Education and Research Hospital during January 2011 to December 2011 were studied and retrospectively evaluated for age, gender, drug use, diseases, systemic or genetic disorders and demographic features. Diagnostic evaluation results were noted and patients were categorized for demographic features and diagnosis. **Results:** These 133 patients (M: F 58:75, %44 vs 56, respectively) were under 16 years of age and have 17 different dermatological disorders related with nail symptoms. Fifty three of (39,8%) these patient were under 2 years of age, 31 (23.3%) were between 3-5 years, 30 (22.5%) were between 6-11 years old, 19 of 133 (14%), 2 were between 11-16 years of age. Through all of ages and independent of gender the most etiologies of nail disorders were, onychomadesis, paronychia, onycholysis, onychomycosis and systemic nail presentation of systemic dermatosis. **Conclusion:** Nail disorders are different in children than in adults. In our study, the first 5 years of age was found in 53% of nail disorders. Nail disorders are uncommon but may be seen as a part of a
systemic disease and may be associated with cosmetic and psychologic problem.

Key words: Children; Nail disorders; Skin diseases

INTRODUCTION

Nails which help the free movement of fingers, protects them from traumas, to be kept small objects, to be used for itching and also important for cosmetic appearance [1]. Nail disorders may cause sociopsychological problems as this will adversely affect the quality of life. Nail disorders in childhood are mainly similar to adults and some physiological changes occur by time and disappear over years. Nail disorders may be a sign of a systemic disease such as congenital and hereditary diseases, infections and dermatoses [2]. Distribution and frequency of childhood nail disordres differs from adults and varies according to the quotation. There are limited number of studies on this nail disorders in childhood. Nail disorders in childhood constitute a small portion of admittions to outpatient clinics of dermatology and pediatrics children and estimates a rate of 0.05-3% of all admissions [1]. In this study, the frequency and distribution of pediatric nail diseases documented and retrospectively evaluated.

MATERIALS AND METHODS

During the period between May to December 2011 at the Children's Hospital Pediatric Hematology and Oncology in Ankara, clinical records of 133 patients admitted for nail disorders at the age group of 0-16 years to dermatology outpatient deoartment were evaluated retrospectively. Our study was approved by the local

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ethics committee. All of the patients' parameters for the purpose of examination and laboratory tests to help diagnose were recorded. To compare the distribution of the patients according to demographic data and diagnoses were four separate categories. Cathegorization made as follows; 0-2 years (infantile period), 3-5 years (pre-school), 6-11 years (school-age period), 12-16 years (adolescent period).

Ethics

This study was performed on human subjects; thus, all patients were aware of the presence of the study and they were fully informed about the drug and its side-effects.

RESULTS

Seventeen different disease diagnosed to 133 patients with various nail disorders. Nail diseases identified in Table 1 are shown. Nail disorders were identified and included in the study of 75 female patients (56%) and 58 males (44%). Male/female ratio was 1.2. Onikomadesis was the most common nail disorders in all age groups 17.2% (n = 23), paronychia 17.2% (n = 23), onycholysis 15.7% (n = 21), onychomycosis 9% (n = 12), dermatosis 9% (n = 12), ingrown toenail 6% (n = 8), periungal wart 4.5% (n = 6), dystrophic onychomycosis 3.7% (n = 5).

Compared by gender onycholysis, onychomycosis, ingrown toenail incidence was higher in girls than boys.

Table 1: The most seen nail disorders in children and distribution due to gender

Disorder	N (girls)	%*	N (boys)	%*	Total
Onychomadesis	11	48	12	52	23
Paronychia	11	48	12	52	23
Onycholisis	14	66	7	34	21
Onychomycosis	9	75	3	25	12
Dermatose nail	8	60	4	40	12
Ingrown toenail	5	62	3	38	8
Dystrophic nail	2	40	3	60	5
Periungal verrucae	3	50	3	50	6
Trachyonychia	2	40	3	60	5
Koilonychia	3	75	1	25	4
Pachyonychia	2	67	1	33	3
Epidermolisis Bülloza	0	0	3	100	3
Ectodermal dysplasia	1	33	2	66	3
Onychogriphose	2	100	0	0	2
Splinter hemorrhage	1	100	0	0	1
Leukonychia	1	100	0	0	1
Median nail dystrophia	0	0	1	100	1
Total	75	56	58	44	133

Paronychia, onychomadezis, dystrophic nails, and epidermolysis bullosa were seen more in boys than girls. Other diseases were as equal.

In the evaluation of the nail diseases by age group, 40% (n = 53), age 0-2, 23.3% (n = 51) and 3-5-age, 22.5% (n = 50) age 6-11, 14.2% (n = 19) were age 11 to 16. In infant age group 0 -2 paronychia, onikomadezis, onycholysis, onychomycosis, age 3-5 onycholysis, paranoşi, onychomycosis, age 6-11, onikomadezis, paronychia, onycholysis, periungal warts, ingrown toenail, age 12-16, dermatosis nail, onychomycosis were frequently seen etiologies (Table 2).

Nail disorders in children with systemic disease was diagnosed in 37 cases (27.8%) (Table 3).

DISCUSSION

Although nail diseases are uncommon in children may have importance in this age group and also may be congenital or acquired. Findings of hereditary diseases of nail usually occur in childhood. A nail disorder in childhood is important because may be a symptom or a marker of a systemic disorder [2].

A ratio of 0.05-3% infants and children are estimated to be presented with the problem of nail [1]. The incidence varies by populations and studies [3-5]. Iglesilas et al. reported that the prevalence of nail disorders was about 11% under the age of 17 [3]. Previous studies in our country revealed that incidence of nail disorders varies between 0.7% -2.3 [6-9]. In this retrospective study with 3000 pediatric patients, the incidence of nail disorders found to be 4.4%.

One of those found in this study onikomadezis was the most frequently seen disorder (in 23 cases). Onikomadezis is segregation of proximal nail fold [10]. Of these 23 patient had a history of immunodeficiency, liver failure, asthma, chronic urinary tract infections, such as strep rash and febrile disease had a history of drug use and associated systemic diseases. That most of the patients were in the range of 0-2 years of age, so that babies are being exposed and more susceptible to infections and diseases that may be the casuse. A study from Taiwan reported that the incidence of onikomadezis in children with hand-foot-mouth disease was 5% [11].

Paronychia is the most common nail infection in children. Nail biting, thumb sucking habits, excessive

Table 2: The distribution of nail disorders due to age subgroups

Disorder	n=133	Total N=3000	Within the group (%)	0-2 years		3-5 years		6-11 years		12-16 years	
		%		N	%	N	%	N	%	N	%
Onychomadesis	23	0,7	17,2	10	18,8	3	9,6	8	26,6	2	10,5
Paronychia	23	0,7	17,2	12	23	6	19,6	4	13,3	1	5,2
Onycholisis	21	0,7	15,7	8	15	8	25,8	4	13,3	1	5,2
Onychomycosis	12	0,4	9	3	5,6	2	6,4	5	16,6	2	10,5
Dermatose nail	12	0,4	9	5	9,4	4	12,9	1	3,3	2	10,5
Ingrown toenail	8	0,2	6	3	5,8	0	0	0	0	5	26,3
Dystrophic Nail	6	0,2	4,5	1	2	1	3,2	3	10	1	5,2
Periungal verrucae	5	0,2	3,7	3	5,8	2	6,4	0	0	0	0
Trachyonychia	5	0,2	3,7	2	3,7	0	0	1	3,3	2	10,5
Koilonychia	4	0,1	3	2	3,7	1	3,7	1	3,3	0	0
Pachyonychia	3	0,1	2,2	0	0	2	6,4	0	0	1	5,2
Epidermolisis Bülloza	3	0,1	2,2	1	2	1	3,2	1	3,3	0	0
Ectodermal dysplasia	3	0,1	2,2	2	3,7	0	0	0	0	1	5,2
Onychogriphose	2	0,06	1,5	0	0	1	3,2	1	3,3	0	0
Splinter hemorrhage	1	0,06	0,7	0	0	0	0	0	0	1	5,2
Leukonychia	1	0,06	0,7	0		0	0	1	0	0	0
Median nail dystrophia	1	0,06	0,7	1	2	0	0	0	0	0	0
Total	133	4,4	100	53	100	31	100	30	100	19	100

Table 3: Systemic disease related to nail disorders

Number of patients	Disorder	Associated systemic disease	Number of patients
23	Onychomadesis	Liver failure	2
		İmmundeficiency	2
		Cronic urinary infection	2
		Asthma	1
		Streptococcic rash	2
23	Paronychia	Leukemia	1
		lymphadenopathy	1
21	Onycholisis	Febrile convulsion	1
		Cronic urinary infection	1
		Drug use	1
		Familial meditarenean fever	1
		Esophageal atresia+hydrocephalus	1
12	Onychomycosis	Down syndrome	1
		Obesity	1
12	Dermatose nail	Asthma	2
		Epilepsy	1
8	Ingrown toenail	Hypogonadism	1
6	Dystrophic nail	none	0
5	Periungal verrucae	None	0
5	Trachyonychia	none	0
4	Koilonychia	Anemia	4
3	Pachyonychia	Anemia	1
3	Epidermolisis Bülloza	Epidermolysis Bullosa	3
3	Ectodermal dysplasia	Ectodermal dysplasia	3
2	Onychogriphose	Epilepsy	1
1	Splinter hemorrhage	Bone hypertrophy	1
1	Leukonychia	Acute rheumatoid fever	1
1	Median nail dystrophia	Hypertension	1
133	Total		37

moisture environment, and chronic irritation may cause [12]. In our study we diagnosed 23 cases with paronychia with an overall incidence of 0.7%. One of these three pateints was due to candida, one of them was herpetic the other were bacterial. Paronychia was mostly seen in 0-2 age group and in boys. Tamer et al found a ratio of 0.5% [13], Nanda et al 0.3% [5], and Fung et al [14] found this rate 4.5%.

Oncholysis is separation of the nail plate from its bed [15]. Acute or repeated minor trauma, infections, systemic diseases and drug use are main causes of

onycholysis [15]. In our study, we found an incidence of 15.7% of 21 cases. We observed that onycholysis was seen mostly in 0-5 years of age and frequently seen in females. In medical history of these patients, hydrocephalus with esophageal atresia, chronic urinary tract infections, epilepsy, drug use and associated systemic diseases such as familial meditarenean fever were observed.

Nail disorders due to systemic dermatologic diseases such as alopecia areata, psoriasis, dermatitis, lichen planus, such as pitting were seen only three of all pateints. Psoriatic nail involvement in children range from 7-39% [16]. Patient with psoriasis (n = 34), even though the psoriatic nail involvement (distal onycholysis, nail bed oil stains and pitting), only seen in 3 patients (2.2%).

One of the most common infections of nails in children is onychomycosis and constitute 20% of all nail disorders and has an incidence of 0.2-2.6%. Cause of disease are dermatophytes, yeasts or molds. Hands are mainly involved under 7 years of age, the feet are involved in older life [17,18]. However, thumb sucking, saliva irritation, use of the pool make a suitable humid environment for the fungus. Down's syndrome, HIV infection, long-term use of cortisone, have a history of tinea capitis, tinea pedis are other risk factors. In this study population Down syndrome was diagnosed in one patient. Gupta et al reported that onychomycosis in children under 18 years of age had a ratio of 0.44% [19]. In our study, the rate was 0.4% overall, while the most commonly between the ages of 0-5, and onychomycosis was frequently seen in girls more than boys. Philpot et al found that onychomycosis incidence was 0.02% [20]. Among Turkish studies this ratio differs between 0.1-3% [13,18,21,22]. Hapçıoğlu and Inanir stated that they saw more than those with low socioeconomic status [21,22] In our study, patients with low levels of socio-economic status and more moderate onychomycosis were seen. A study with 1,588 children under the age of 16, a ratio of 9.7% found for onychomycosis incidence and we also found a similar result (9%).

Ingrown toe nail is quite a common entity affecting thumbs. The curve to be settled congenital thumb nails, false nail cutting, trauma, sports activities and narrow shoes are risk factors for ingrown nail [24]. In our study, 6%, 8 cases of ingrown toenail were seen. Tamer et al [13] found 0.3%, and Sarıfakıoğlu et al [25] reported this ratio as 2.4%.

In our study we found 6 cases, 4.5% with periungal warts. Children under the age of 16 in 1588 children in Poland periungal warts found in 19.5% of their screening population [23]. Iglesilas et al. found 6 of 100 children with warts and nail dystrophy [3].

Dystrophic nails consists of plaque discoloration, deformation, could result from a pterygium formation and finally a permanent loss of the nail occurs. [2]. In our study, five patients (0.2%) improved dystrophic nail due to trauma. Philpot determined dystrophic toenail in 5 of 494 school children [20]. Tamer et al. And Nanda et al. found that the incidence of nail dystrophy were 0.2% and 0.06% respectively [5,13]. Fung et al found this rate as 0.1% [14]. Periungal verrucae disturb the structure of the nail. This lesion grow under the nail and the nail plate to peel painful and cause onycholysis [26]. Periungal verrucae is a complication of nail biting [1].

Nail dystrophy or trachyonychia is coarsening the surface of the nail. The surface roughness is less than bright type and there are also a large number of pits. The exact incidence is unknown, but is most common in children [27]. In this present study, we observed 5 patients (0.2%), found that an incidence of 3.7% of all nail disorders. Some studies found different ratios are as follows, Tamer et al 0,1% [13], Nanda et al. 0.07% [5] Sobjanek et al 2,4%.

Koilonychia is lost the normal appearance and contour of the flat or concave nails. While many reasons determined the most common cause of koilonychia is iron deficiency [28]. In 4 cases (3%) we found iron deficiency anemia. Sobjanek et al. found an incidence of koilonychia 1.22% in nail disorders [23].

Pachyonychia congenita (PC) is an ectodermal dysplasia characterized by hypertrophic nail dystrophy [29]. In our study, we found an incidence of PC as 2.2%.

Ectodermal dysplasias primarily affects the skin and other ectodermal structures are quite large, heterogeneous group of rare diseases [29]. İglesilas et al studied 100 children and found 4 (2.5%) ectodermal dysplasia cases [3]. In our results we found similar findings in three cases, by a rate of 2.2%.

Subungual lesions of epidermolysis bullosa (EB) or blisters may be in periungal location. [29]. In our 3 epidermolysis bullosa case, there were various nail lesions. Gul et al found that an incidence of 5.5% EB in 0-1 age group presented with nail disorder [30].

Our rate for EB was 2.2% and Iglesilas et al observed 5 patients (5%) with EB.

Onychogryphosis occurs due to developmental disorders or trauma. Nails are extremely thick and curved [27]. We had two patients with onychogryphosis.

Median nail dystrophy is a rare, usually bilateral lengthening groove-like dystrophy develops a temporary condition. The exact cause is unknown, but due to a defect in the nail matrix, nail the temporary structure is considered impaired [16]. We found only 1 case in our population with median nail dystrophy.

Punctate leukonychia occurs due to trauma in fingernails in children. [16]. We observed punctate leukonychia lesions only in one patient (0.06%). Tamer et al found an incidence of 0.1% [13].

Splinter hemorrhage is a small bleeding islands under the nail bed presented like longitudinally submerged splints under the nail. Splinter hemorrhage may be associated with systemic diseases [16]. We observed this lesions in one patient (0.06%) in our population.

This case had also bone hypertrophy on his finger.

According to our results, distribution of nail disorders by age were as follows, 0-2 age 40% (n = 53), 23.3% of 3-5 age (n = 31), age 6-11, 22.5% (n = 30), 12-16 age, 14.2% (n = 19) Can et al. found that 35% of nail diseases in 0-2 ages, 3-5 at the age of 35%, 14% of school age children, 14% for adolescents [7]. Nail disorders are common in infants this suggests the hypothesis that the increase in the admission to family physician because of concerns about the nails.

Sarıfakıoğlu et al. studied 250 infants under two years of age and found an incidence of nail disorder with a rate of 6.8% [25]. In this study mostly toe nails were affected and mainly seen types were onycholysis, congenital hypertrophy and ingrown toenails. We detected in the 0-2 age group, 40% (n = 53) of all patients, the most seen type of nail disorders were paronychia, onychomadesis and onychomycosis. This result was mainly due to the type of our hospital which is a referral hospital and as a cause of hospitalization of children with systemic disease may revealed this findings.

Systemic diseases and medications may also associated with nail disorders. Especially they make temporary pause in particular to reduce the rate of growth. Drug-induced toxic effects may lead nail disorders usually affects all the nails [31]. In our study, 37 cases 27.8% had systemic diseases such as liver failure, immune deficiency, chronic infections, asthma, and epilepsy. We also had a history of drug use related to these diseases in these patients. Onychomadesis, onycholysis and paronychia were most common nail findings due to drugs in our study population.

CONCLUSION

Types of nail diseases in childhood, adults varies according to the range and frequency. Although many studies established, investigations on nail disorders in childhood have not been fully satisfactory. Some nail disorders if untreated can lead to permanent nail diseases and can impair quality of life. Because of being a reference hospital and the population type of the patients may help health professions on this topic and also can help caregivers think in mind these etiologies of nail disorders.

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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Our Dermatology Online

Original Article

A study on scar revision

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ABSTRACT

Introduction: Scars are psychologically distressing for the patients and have an impact on the quality of life and self esteem of the patients. Scar revision is an aesthetic skill which is mastered by plastic surgeons and encroached now by dermatosurgeons. Scars on the face are aesthetically unacceptable and various techniques have been improvised for making a scar aesthetically acceptable.Various types of techniques are used for scar revision like W plasty, Z plasty and VY plasty. **Aims:** To see the efficacy of various scar revision techniques including Z plasty, VY plasty and W plasty in 30 patients with disfiguring scars. **Methods:** We selected twenty patients of disfiguring scars for the study. The scars from various causes including trauma and burns were included in our study.Various techniques of scar revision include Z plasty, W plasty and VY plasty were performed according to the type and site of scar. **Results:** Male: female was 1.5: 1. The scar revision surgery yielded excellent results with minimal complications including haematoma formation, secondary infection and delayed healing seen in 5% patients each. Regarding the efficacy of scar revision, excellent improvement was seen in 60% patients, moderate improvement was seen in 30% patients and mild improvement was seen in 10% patients. **Conclusions:** Dermatologists can employ a number of surgical scar revision techniques. While some are better suited to treat specific types of scars, they can be used in combination with each other or with adjunctive therapies to achieve optimal results.

Key words: Scars; Facial; Techniques; W plasty; Z plasty; VY plasty; Revision

INTRODUCTION

Scar evaluation and revision techniques are chief among the most important skills in the facial plastic and reconstructive surgeon's armamentarium. Often minimized in importance, these techniques depend as much on a thorough understanding of facial anatomy and aesthetics, advanced principles of wound healing, and an appreciation of the overshadowing psychological trauma as they do on thorough technical analysis and execution [1,2]. Scar revision is unique in the spectrum of facial plastic and reconstructive surgery because the initial traumatic event and its immediate treatment usually cannot be controlled. Patients who are candidates for scar revision procedures often present after significant loss of regional tissue, injury that crosses anatomically distinct facial aesthetic units, wound closure by personnel less experienced in plastic surgical technique, and poor post injury wound management [3,4]. While no scar can be removed completely, plastic surgeons can often improve the appearance of a scar, making it less obvious through the injection or application of certain steroid medications or through surgical procedures known as scar revisions.There are many variables affect the severity of scarring, including the size and depth of the wound, blood supply to the area, the thickness and color of your skin, and the direction of the scar [5,6].

A scar may cause cosmetic deformity and it may be an unpleasant reminder of a traumatic past and the patient may seek to erase its memories by erasing the scar. Finally, the patient may associate the scar with a personal failure - inability to impress a girlfriend or inability to get promoted and may be looking at treatment of the scar as a means of success in his/her endeavors. An ideal scar is thin and flat, has a good

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color match with the surrounding skin, is oriented along the relaxed skin tension lines (RSTLs), and does not produce any distortion of adjacent tissues.Any scar that does not fit the above definition is a suboptimal scar [7-9]. Every attempt should be made to convert a suboptimal scar into an ideal scar although it may not be always possible.

AIMS

To see the efficacy of various scar revision techniques including Z plasty, VY plasty and W plasty in 30 patients with disfiguring scars.

MATERIAL AND METHODS

We selected twenty patients of disfiguring scars for the study. The scars from various causes including trauma and burns were included in our study. Various techniques of scar revision include Z plasty, W plasty and VY plasty were performed according to the type and site of scar. Written informed consent was taken from all the patients before the study. Prior approval of hospital ethical committee was taken for the study. Pre and post operative photographs were taken of all the patients. Proper preoperative counselling of all the patients was done and the patients expectations were brought to the ground level.

Inclusion Criteria

The following patients were included in our study:
- Patients having facial scars
- Patients having realistic expectations.

Exclusion Criteria

The following patients were excluded from the study:
- Patients having history of keloid formation
- Patients having history of bleeding tendencies
- Patients on oral anticoagulants
- Patients having uncontrolled diabetes.

For assessing the efficacy of treatment, the patients were divided into three groups: Excellent results – More than 90% improvement after scar revision.

Moderate results – 75% - 90% improvement after scar revision.

Poor results – 25% - 50% improvement after scar revision.

Ethics

This study was performed on human subjects; thus, all patients were aware of the presence of the study and they were fully informed about the drug and its side-effects.

RESULTS

The data was collected, tabulated and the results were analyzed statistically using chi square test.

Results was presented in (Tables 1-3).

DISCUSSION

There were 12 males and 8 females and male: female was 1.5: 1. The scar revision surgery yielded excellent results with minimal complications including haematoma formation, secondary infection and delayed healing seen in 5% patients each. Regarding the efficacy of scar revision, excellent improvement (Figs. 1a and 1b) was seen in 60% patients, moderate improvement (Figs. 2a and 2b) was seen in 30% patients and mild improvement was seen in 10% patients.Poor scar is

Table 1: Age distribution of patients

Sr. no	Age distribution	Number	Percentage
1	0-20	6	30
2	21-40	12	60
3	41-60	2	10

Table 2: Improvement after scar revision

Sr. no	Results	Number	Percentage
1	Excellent	18	60
2	Moderate	9	30
3	Minimal	3	10

Table 3: Complications of scar revision

Sr. no	Complications	Number	Percentage
1	Haematoma formation	1	5
2	Secondary infection	1	5
3	Delayed healing	1	5

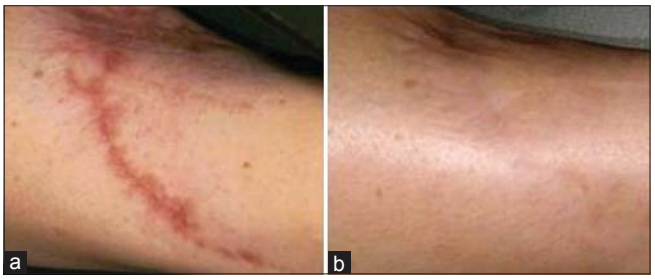

Figure 1: (a and b) A three months old scar before and after treatment.

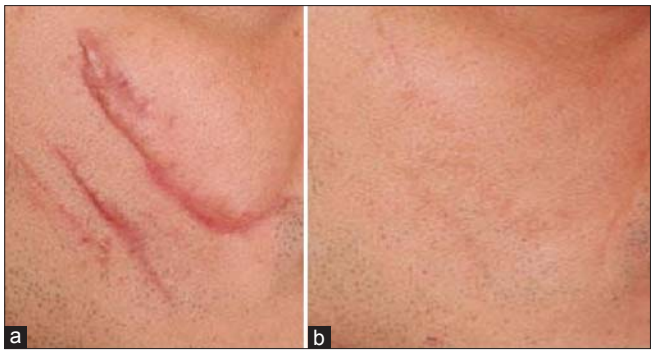

Figure 2: (a and b) A five months old scar before and after treatment.

usually a result of poor technique or post-operative infection. In such cases, simple excision of the scar and resuturing using the above principles may be helpful in attaining a good appearance. Scars can be classified as -Mature scar - A light colored flat scar.

Immature scar - A red, sometimes itchy or painful, and slightly elevated scar in the process of remodelling. Many of these will mature normally over time.

Linear hypertrophic scar (e.g., due to incision) - A red, raised, sometimes itchy scar confined to the border of the original incision. These scars may increase in size rapidly for 3-6 months and then, after a static phase, begin to regress. After maturation, they may have an elevated, slightly rope like appearance with the increased width.

Widespread hypertrophic scar (e.g. due to burns): A widespread red, raised, sometimes itchy scar that remains within the borders of the original injury.

Minor keloid - A focally raised, itchy scar extending over normal tissue. This may develop up to 1 year after injury and does not regress on its own.

Major keloid - A large raised (>0.5 cm) scar, possibly painful or pruritic and extending over normal tissue. This may result from minor trauma and can continue to spread over years.

In addition to the above mentioned varieties, scars may also be atrophic, depressed, hypopigmented, hyperpigmented, or irregular with nodularity.

Scar revision is unique in the spectrum of facial plastic and reconstructive surgery because the initial traumatic event and its immediate treatment usually cannot be controlled. Patients who are candidates for scar revision procedures often present after significant loss of regional tissue, injury that crosses anatomically distinct facial aesthetic units, wound closure by personnel less experienced in plastic surgical technique, and poor post injury wound management. There are various techniques of scar revision including Z plasty, VY plasty,Wplasty and geometric broken line closure. The various techniques of scar revision are as follows:

Z PLASTY

It is a double transposition flap where the scar to be excised lies along the central limb of the Z with two peripheral limbs parallel to each other [10,11]. After transposition, the centre Z-plasty is one of the most versatile scar revision techniques available. As a transposition flap, Z-plasty allows for 2 adjacent undermined triangular flaps, constructed from the same central axis, to transpose over each other and to lie in the other's originating bed. In essence, these 2 triangular flaps are transposed from areas of relative excess into areas of relative deficiency and eventually lie at near right angles to the original central axis.The usefulness of Z-plasty in scar revision rests in its ability to reorient a scar to lie more favorably in the direction of RSTLs; reorient the scar or anatomic landmark into a more favorable location or position; break up the length of the scar, thereby rendering it less visible; increase the scar length (ie, lengthen a contracted scar), thereby decreasing the prevailing scar contractile force and permitting better conformation to contoured surfaces and allow the surface-revised scar to run in a different angle to the deeper, more established scar, thus decreasing the tendency of the final scar to become depressed [12].

W PLASTY

It is designed to make a linear scar irregular, such that majority of the limbs lie along RSTL.While performing the W plasty, some amount of normal tissue is excised along with the scar such that the final scar is irregular, in the shape of multiple W's lined side-by-side [13]. The W plasty consists of multiple small triangular advancement flaps on either sides of the scar such that the closure occurs in an interdigitating fashion. The advantages of W plasty are that it is easy to plan and execute and It breaks a straight scar into multiple small segments many of whom lie along the RSTL [14]. There are various disadvantages of W plasty- it may lead to a longer scar, it needs adjacent tissue laxity and sometimes regular repetitive pattern makes the

scar noticeable W-plasty. The primary utility of the W-plasty (also termed the running W-plasty or zig-zag plasty) is in rendering a lengthy linear scar irregular. In addition to linear scar revision, the W-plasty is useful in the closure of semicircular incisions in which the sweeping unbroken curvilinear scar is more noticeable and under greater tension and, thus, over time more likely to become depressed or pincushioned. Note that while the W-plasty makes irregular a linear scar and spares unwanted lengthening that may arise from using small multiple Z-plasties, the final result is often readily visible because the eye easily can follow the predictable zig-zag configuration [15]. Finally, in its basic execution, this technique incorporates neither transposition nor rotation of adjacent flaps; therefore, the final scar is not elongated but only increased in the final total length.

Geometric Broken Line Closure

It is designed to convert a long linear scar into a randomly irregular scar. Interdigitating geometric lines are drawn in such a manner that triangles, rectangles, squares. and even semicircles are created on either side of the scar in a random fashion. Majority of the lines should lie along the RSTL [16]. After excision along these lines, the advancement flaps from both sides interdigitate so as to create a randomly irregular scar. Like in W plasty, ends have to be closed using 30° angulations to prevent the dog ear which may occur if higher angles are used Unfavorable facial scars result from a variety of influences, over which the reconstructive surgeon often has little initial control.

The decision regarding the location and type of incision used during any scar revision is based primarily on the concept of orienting all incisions perpendicular (as much as possible) to the direction of maximal underlying tension. Incisions made perpendicular (or nearly so) to this direction are better camouflaged and heal more favorably than those made parallel because these contractile forces tend to approximate the wound margins, rather than distract them apart. Understanding this concept is critical because it determines the difference between a long-term mediocre or superior scar revision. For various regions of the face, various scar revision techniques are used [17].

Cheek

The cheek represents a unique anatomic site in scar revision because the RSTLs do not run straight but rather in a curvilinear fashion from the malar eminence to the inferior border of the mandible. Scars crossing the cheek in the direction of the RSTLs are best treated with a running W-plasty (see image below). The surgeon may use a lateral-end Z-plasty for superior cosmesis. However, scars often run perpendicular (or nearly so) to the RSTL curvature mentioned above. In these cases, better camouflage is achieved by dividing the scar into multiple Z-plasties.

Nasolabial Fold

The pronounced sulcus of the nasolabial fold (ie, cheek-lip fold) is well suited to scar camouflage. Understanding the proper use of Z plasty is critical in this area where Z plasty may be used, either singly or in conjunction with a running W-plasty, for scars extending from the cheek and crossing the nasolabial fold. Of critical importance are the orientation of the lateral limbs and the angle at which they subtend the Z-plasty central limb. In designing the lateral limbs of the Z-plasty, only one combination yields the best cosmetic result and places the lateral limbs nearest the direction of the RSTL.

Mentum

Scars crossing horizontally over the mentum generally follow RSTLs and therefore are best treated with a running W-plasty Laterally based and more obliquely directed scars are good candidates for Z-plasty because the primary objective here is to redirect the scar in the RSTL direction. Often, these scars cross from an oblique lateral to a more horizontal orientation and require a combination of lateral Z-plasty and running W-plasty over the mentum.

Forehead

The underlying frontalis muscle creates unusually prominent forehead RSTLs. These well-defined lines run horizontally in the central forehead with their lateral ends projecting obliquely inferior over the temple region. Pay particular attention to the junction of the glabella and forehead. The vertical RSTLs of the glabella meet those of the forehead in a nearly perpendicular orientation. Correction of scars that cross both of these regions probably requires incorporation of differing revision techniques that redirect by Z-plasty and cause irregularity by W-plasty or that use simple fusiform excision.

Eyebrow

The prominence of the supraorbital rim renders it a probable site of injury in frontal facial trauma. Lacerations frequently cross the forehead to include the eyebrow and are a revision challenge because of their visibility and because they require special techniques to camouflage the scar within the brow hair. Important concepts in eyebrow revision procedures include creating irregularity within the scar and beveling incisions parallel to the hair shaft. W-plasty is the revision procedure of choice and requires particular attention in aligning the superior and inferior borders of the brow.

CONCLUSIONS

Abnormal scarring remains one of the major problems faced by surgeons and their patients. Surgical treatment of a suboptimal scar should be undertaken only after it matures, ideally after a year. A scar can be revised by excision and linear closure, Z plasty, W plasty, geometric broken line closure or excision and cover. Appropriate post-operative care and when indicated, adjuvant therapy form an essential part of the treatment plan. Laser, dermabrasion, and other adjuvant therapies mentioned in previous sections are useful in the management of immature scar. Classification of a scar abnormality guides the choice of treatment technique. A successful scar revision can dramatically improve a patient's quality of life. Dermatologists can employ a number of surgical scar revision techniques. While some are better suited to treat specific types of scars, they can be used in combination with each other or with adjunctive therapies to achieve optimal results.

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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Our Dermatology Online

Brief Report

Cross-linked natural gum resins, when inserted in shampooing product, result infallible to eliminate several metallic ions risky for hair keratin

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ABSTRACT

Aims of my research is to herald the method of eliminating Calcium and Magnesium ions that remain onto hair and scalp keratin after washing with common hard water and trivial shampooing products, but even of removing other metals as Lead, Silicon and Nickel ions which can be retrieved in manifold building materials like mortar, cement, concrete, pozzolans, limestone and asbest, most of workers throughout the world are directly involved with, because of their continuous contact with those chemical materials. I have selected twelve volunteers (workers who are directly in contact with building materials containing Calcium and Magnesium ions) and prayed them to use three types of shampooing products of my invention (containing special gum resins previously cross-linked in order to uptake or sorption the metallic ions) after having used, in precedence, trivial shampoos (bought at the same store) and used the same tap water, since they live all in the same town. I calculated the difference of quantities of Magnesium and Calcium that remain onto hair and scalp keratin, using a general and trivial shampoo respect to my products, apt to remove the same metallic ions. Results are satisfactory and encouraging.

Key words: Metal uptake; Metal sorption; Cross-linked gum resins; German degrees; Water hardness

INTRODUCTION

Aims of my research is to devise the way of eliminating Calcium and Magnesium ions that remain onto hair and scalp keratin after washing with common hard water and trivial shampooing products, but even to build up a rigorous method to totally remove the same Calcium, Magnesium and too often Lead, Silicon and Nickel ions as well from all the total hair of whichever worker who is occupationally involved with the direct and customary contact with mortar, cement, concrete,pozzolans, limestone and asbest, keeping on account that except the following countries: Algeria, Argentina, Australia, Austria, Bahrain, Belgium, Brunei, Bulgaria, Chile, Croatja,Cyprus, CzechRepublic, Denmark, Egyst,Estonia, Finland, France, Gabon, Germany,Greece, Honduras, Hungary,Iceland, Ireland,Israel, Italy,Japan,Jordan, Korea (South), Kuwait,Latvia,Lithuania, Luxembourg, Netherlands, New Caledonia, Norway, Oman, Poland, Portugal, Qatar, Romania, Saudi Arabia, Serbia, Slovakia, Slovenia, South Africa, Spain, Sweden, Switzerland, Turkey, United Kingdom and Uruguay where asbest has been banned since long time, manifold countries exist which have never abolished the use and/or production of asbest, amosite, crocidolite, tremolite, chrysotile, anthophyllite and actinolite, (for instance the exploitment of such minerals are fully allowed in the Republic of India, in the People's Republic of China, in Brazil, Republic of Mongolia, Republic of Seychelles, Singapore or Taiwan (Republic of China or Formosa) or the Democratic People's Republic of Korea, idest North Korea).

Generic mortar, after analysis according to the ASTM C1324, "Standard Test Method for Examination

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and Analysis of Hardened Masonry" appears to contain approximately 60.0% calcium oxide (CaO) and 30.0-to-15.0% silicon dioxide (SiO2). Brucite (magnesium hydroxide) has been also detected at a low amount, indicating the hydrated lime is an impure, high-calcium type.

It is well known that Calcium ion builds up on the hair, leaving the hair feeling dry and weighted down, that can even cause perms and hair sculptures to be relaxed, may cause flaking, too often as dandruff, can clog the hair at the mouth of the follicle, causing the hair to break off, and may coat the scalp, blocking further hair growth. Moreover, some Japanese researchers have recently heralded the existence of the protein S100A3, a unique protein among all members of the calcium-binding S100 family, which is specifically expressed at the inner endocuticle of human hair fibers and upon hair damage the aforementioned protein is released from hair fibers and possibly destabilizes all the hair tissue architecture [1,2].

As far as Magnesium ion is concerned, it attaches to the S-S double bond of the cysteine of hair keratin, leaving it feeling dry and weighted down.Regard to Silicon ions, it is to be stressed that all Sand-like substances can build up on hair, causing dryness, dandruff, weight, and hair loss. Effectively people native to volcanic or desert areas are well acquainted with the fact that sand silica may build up very hard, virtually insoluble deposits on all kind of natural and/or inorganic surfaces, extremely hard to be eliminated. Finally, since Nickel and lead are commonly found in cement in non-negligible concentrations, it is supervacaneous to stress the importance of attempting to remove these ions by cosmetic way from human hair.

It is mandatory to assert that all generic and trivial shampooing products, due to the presence of anionic surfactants, are capable to form aliphatic salts of Calcium or Magnesium or Silicon or Lead, which, for sake that are fully insoluble in water remain attached to hair and are prone to damage it.

So, primarily, I have decided to select twelve volunteers (masons, miners and carpenters) which are always in contact with dangerous building materials containing high percentages of metallic ions that remain inevitably onto their hair or scalp keratin.

Secondarily, I collected all the original rinsing waters of their showers or bath-foams after having used a generic shampooing product and the same tap water, (since they live all in the same city), and the subsequent quantitative detection of the ions, objects of my investigation,by the Boutron-Boudret's idrotimetric method, reviewed by Walter and Gartner, that forecasts the successive titration by $Ba(NO2)^2$ and extrapolation of results in German degrees instead of employing the official measurement by French degrees for the evaluation of water hardness.

It is suggestive to declare that I used in my three formulas the same surface active agent that was used for the determination of the original hardness of the tap water.

Finally, the quantitative detection of the same involved ions after washing and shampooing with three different cosmetics comprising three diverse gum resins apt to uptake and/or sorption the same ions, object of this study.

MATERIALS AND METHODS

It is necessary to explain what the Boutron Boudet's test is: it is based on the capacity Calcium and Magnesium ions that are present in the aqueous solution to create insoluble compounds when mixed together with Castile soaps (idest the savon de Castille, so called from the first Muslim soap-makers in the 12th century in Castilla (Alicante, Malaga, Cartagena) and in Italy (Naples, Savone, Genoa, Bologna and Venice) which only at the end of the 15th century was baptized Savon de Marseille in Marseille France, a worldwide famous surface active agents obtained by the neutralization of olive or laurel oil by strong aliphatic acids).

When a Castile soap solution with known concentration is added drop by drop to a known volume of deionised water and the new-formed solution is drastically agitated, a thick layer of foam appears.

Apposite Boutron Boudet's hydrotimetric bottles are used to determine the degrees that appear on the scale of the cylinder and are called French degrees, that represent indicatively the exact concentration of the Calcium and Magnesium ions pro liter of hard water, but since I use the variant of the Boutron Boudet's method, the Walter Gartner's titration by the use of $Ba(NO3)2$, I express the final results in form of German degrees, since I objectively used a soap deriving form an animal grease (tallow) instead of a soap deriving from the neutralization of a vegetal oil with strong aliphatic acids.

Thus, We have collected the twelve initial rinsing water samples from the 12 volunteers, by reason of 2 lt for each individual, which used the same shampooing-bath-foam purchased in the same store and employed the same tap water, since they live in the same city.

Originally We have scored the value in German degrees of the pure tap water and this value must always be obligatorily subtracted from every result, both after washing using the generic and trivial shampooing product and after washing using the three formulations I gave to the 12 volunteers to test.

We have first determined the quantity of Calcium and Magnesium in the original pure tap water using the Boutron-Boudret's method that expresses the quantity of calcium and magnesium ions in form of German degrees (1 German degree correspond to 1,25 English degrees and to 1,79 french degrees).

The result of this evaluation is 8.96 German degrees (corresponding to 12 French degrees or 16 English degrees).

Secondarily We have determined the German degrees of the 12 rinsing water samples after washing with the generic shampooing product and finally the German degrees of the 12 rinsing water samples after washing using the three formulas I gave the volunteers to test.

It must be kept on account that after every determination it is necessary to subtract the original value in German degree of the pure tap water used for washing.

We used potassium tallowate, as the anionic surfactant required to determine the total degrees of the rinsing water samples, and it can be noticed that the same potassium tallowate will be the same surfactant apt to clean and cleans human hair and scalp, comprised in the three formulas I ideated.

Several are the international patents and papers which disclose the invention of matrices or beads apt to uptake bivalent or trivalent or heavy metals from pure water to make it drinkable or just from wasted water to reuse [3-9].

We have selected three fluid matrices, where three biopolymers (gum resins) were previously cross-linked by the aids of inorganic activators, and thus:

(MATRIX ONE) A 1.5% Locust bean gum aqueous solution cross-linked by borax (2.3%) and condensed in microwave oven for 2 min (potency 200W).

(MATRIX TWO) A 2% Carrageenan acqueous solution cross-linked by Ammonium Bromide (1.4%) and condensed in microwave oven for 1 min (potency 400 W).

(MATRIX THREE) A 1.5% Tara Gum acqueous solution cross-linked by borax (2.1%) and condensed in microwave oven for 1 min (potency 400W).

These three condensed and cross-linked gum resins are able to uptake generically and indifferently Calcium, Magnesium,Lead, Silicon and Nickel ions and are inserted in cosmetic formulas that comprise the same surface agent that had been used to determine the hydrotimetric degrees (German degrees) of the rinsing water samples of the twelve workers after shampooed using a trivial shampooing product.

The following are the formulas of shampooing-bathing-foams I prepared for the volunteers.

It is important to notice that no preservatives, colours and fragrances are included, in order not to distort quantitative detections of the involved ions.

A) Matrix ONE
Potassium tallowate
Glycerin
Decyl glucoside
Sodium cocoyl glutamate.

B) Matrix TWO
Potassium tallowate
Glycerin
Decyl glucoside
Sodium cocoyl glutamate.

C) Matrix THREE
Potassium tallowate
Glycerin
Decyl glucoside
Sodium cocoyl glutamate.

In Tables 1-4 was presented list of German degrees.

Ethics

This study was performed on human subjects; thus, all patients were aware of the presence of the study

Table 1: List of German degrees evaluated in the 12 rinsing waters after simplest washing with trivial shampooing products and tap water (it must be considered that some individual may present higher pools of Calcium and Magnesium ions than others, due to his contacts with chemicals during his job)

Case	A	B	C	D	E	F	G	H	I	L	M	N
German degrees	9.7	9.0	7.9	10.8	9.6	9.7	8.9	10.1	7.8	9.5	11.8	7.8

Table 2: List of German degrees evaluated in the 12 rinsing waters after washing with Shampooing product A containing Matrix ONE

Case	A	B	C	D	E	F	G	H	I	L	M	N
German degrees	5.0	4.1	5.6	6.1	6.2	4.9	6.0	7.0	3.9	4.8	5.2	6.1

Table 3: List of German degrees evaluated in the 12 rinsing waters after washing with Shampooing product B containing Matrix TWO

Case	A	B	C	D	E	F	G	H	I	L	M	N
German degrees	4.8	5.2	6.6	7.1	6.3	6.1	6.8	5.9	6.2	5.7	6.0	7.1

Table 4: List of German degrees evaluated in the 12 rinsing waters after washing with Shampooing product C containing Matrix THREE

Case	A	B	C	D	E	F	G	H	I	L	M	N
German degrees	3.3	4.1	5.5	3.6	4.8	3.7	7.1	4.3	5.4	6.1	3.9	5.0

and they were fully informed about the drug and its side-effects.

RESULTS AND DISCUSSIONS

It is important to refer that I preferred to use the Raghu Raj Bahadur's statistical method that observes the Anderson–Bahadur's algorithm to calculate the final results and thus, after having had all the experimentations made from the 12 volunteers and had all the calculus realised I can absolutely assert that:

The shampooing containing Matrix A (that is locust bean gum cross-linked by borax) evokes a decrease of 41.91% of the original value measured in German degrees measured in the rinsing waters after washing with trivial shampooing product.

The shampooing containing Matrix B (that is Carrageenan cross-linked by Ammonium bromide) evokes a decrease of 32.00 % of the original value measured in German degrees measured in the rinsing waters after washing with trivial shampooing product.

The shampooing containing Matrix C (that is Tara gum cross-linked by borax) evokes a decrease of 48.66% of the original value measured in German degrees measured in the rinsing waters after washing with trivial shampooing product.

All this stands for an approval or indeed a drastic incitement to the proposal of manifold cross-linked natural gums which may be employed in cosmetics apt to wash, rinse and cleanse hair and epidermis in order to remove and eliminate metals that are retrievable in hard waters, atmosphere and more so in cements, mortars, concretes,pozzolans, limestones and asbest.

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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Our Dermatology Online

Brief Report

The diverse and amazing allergic responses to coloured semi-synthetic fabrics in skin of man, woman and transgenders (MTF and FTM)

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ABSTRACT

We have attempted to determinate how three particular synthetic or semi-synthetic fabrics (that are serge, polycotton and spandex) may interact with the skin of Males, Females or Transsexuals (MtFs and/or FtMs), when these individuals put on these fibers directly on nude epidermis. Transsexuals are to be considered, in this seat, all the subjects that take on purpose the relative opposite hormones to try to change or determinately change their sex, that is Men who take estrogens and Women who take Testosterone to grow transgender. Hormonal influence is to be reputed the chief responsible cause of the occurrence of diverse cutaneous manifestations. Results are extremely suggestive and show that hormonal influence is actually the primary cause of histamine and bradikyne cascade, apt to evoke odd cutaneous manifestations in Man, Woman or Transgender, depending on the type, idest, if the transgender is MtF or FtM.

Key words: Azo dyes; Serge; Polycotton; Spandex; MtF, FtM

INTRODUCTION

Since immemorial times Man has been using natural cellulosic fibers, both vegetal as cotton, kemp and linen and animal, as wool, silk and byssus in order to confect his own accountrements, apparel,clothes and equipment.

Organic Chemistry, at the beginning of last century intended to imitate the natural polymers employing patrol derivatives and so began to create a huge assortment of diverse fibers, each of every one quite different from the others, chemically-physically speaking, and researchers used to designate those polymers synthetic fibers tout court [1,2].

More recently these synthetic polymers have been foreseen as backbone of mixed fibers, idest fabrics where natural chains represent the warp of the same natural fibers, which concretely embody the woof, or vice versa: for instance the merino cavalry twill may result the warp of polyester fibers or the serge that is the denim combined with rayon: and it must be stressed that serge is the main constituent of all clothes of military uniforms.

There is even to be kept in account that manifold are the chemical additives which may be included in these combinaisons of semi-synthetic fabrics (synthetic and natural) in order to bestow some physical attitudes like water repellency, crease resistance, antistatic and fireproof properties, but even chemical stuff as artificial dyeings, products for finissage, as some metallic ions, gums and glues and moreover optical brighteners and sometimes biocides [3,4].

It is unequivocal that too often all mixed fibers may yield to severe cutaneous pathologies, like:

Allergic contact dermatitis; Pigmented Purpuric dermatitis (mainly Schamberg's disease); Polymorph

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erythema (erythema multiforme); Fisher's Pigmented dermatitis (evoked usually by all the azo-dyes used for fabrics); Pustular dermatitis; Phototoxic dermatitis; Hives; Eritrodermie; Folliculitis.

The areas of the entire body which are more prone to contract the aforesaid dermatitis are all those zones that are in closest contact with the same fibers, especially all the regions of the epidermis that are not covered by undergarments that are made up by natural fabrics and so axillae, neck, popliteal fossa, antecubital fossa, trunk and chest.

Female socks and collants are the major responsible causes of allergies and dermatosis in the areas of the internal and posterior tighs and often even the back of the feet [5].

It is suggestive that allergies evoked by sockets in Male are very rare, even if the major synthetic fibrous component is rayon, both in female socks and in male sockets.

It is also necessary to add the fact that most of fabrics and tissues hailing from extra EU countries, where rules concerning the banishment of allergenic stuff involved in the productive cycle of the semi-synthetic tissues are scarce and improper and where the technologies exploited to confect tissues are antiquate and obsolete so that these are not capable to remove the chemical and risky substances previously used to create the special textiles.

Manifold consumers declare to have contracted allergies and dermatitis after having worn mixed tissues, but too often these supposed allergies and dermatitis are at last simplest irritations [3,5,6].

The principal negative event that can provoke discomfort in man is generally abundant sweating and the typical rushes that could be defined as "calor et rubor", caused by scarce transpiration and certain ways to evaluate the air and liquid permeability of fabrics exist like TexTest Air Permeability, driven by the aids of Tester FY3300, following standard method ASTM D-737,BS 5636; WIRA Liquid Absorbency Time, that records the time required for complete wetting and WIRA Liquid Absorbency Capacity that scores the amount of liquid each of every specimen can hold after a period of immersion) and all these tests are executed always before to put a newest tissue on the international market.

Last but not least, it must be considered the usage of textile colours, which are classified in seven important categories as far as fabrics are concerned:

a) Acid
b) Basic
c) Vat (that forecasts the usage of Sodium dithionate to transform "leuco" bases in diverse coloured nuances)
d) Direct
e) Dispersed
f) Reactive (which contain a cromphore apt to react with the fibrous substrate)
g) Sulfur dyes (using Cachou de Laval's method based on alkaline sulphide sources or Vidal's method based on mixing aniline with pure sulfur).

And each of everyone must be always chemically linked to a mordant in order to maintain the colouring puissance during the reiterative washing and drying day after day.

Mordants commonly used for tissues are potassium dichromate or metal complexes where generally nickel or cobalt are present in the same molecule.

Almost all the textile colours are azo-dyes and among those, all the ones belonging to the category of dispersed, are allergenic (since they are physically lypophilic and thus may penetrate easily the epidermis barrier), even it is well ascertained that anyway the 40% of all textile colours are allergenic at all when they enter into contact with epidermis and various annexes and mucosae.

Diperse Blues (number 1,3,26,27,9,56,60,106 and 124) are the most hazardous at all.

Here follows Table I where the seven categories of textile colours and their applications to dye diverse fabrics are plotted.

Paraphenylendiammine, banished for dying of artificial tissues, is commonly and legally employed for the

Table 1: The categories of textile colours and their applications to dye diverse fabrics

Type of textile colour	Its peculiar application onto particuliar fabric
Acid	Wool, silk, leather, acrylics
Basic	Acrylics, wool, silk
Direct	Cotton, leather, nylon
Vat	Cotton, rayon, lycra, spandex
Sulphuric	Cotton, cellulosic fibers, orlon
Disperse	Polyesters and acrylics, spandex, lycra
Reactive	Cotton, cellulosic fibers, orlon

confection of kevlar, synthetic fiber retrievable in bulletproof vests, transsexuals and FtMs (idest females which want to appear male by wearing male clothing and apparel like packing underwear) love to put on directly on nude skin, when in society or for fun.

Aims of my research is to determinate how three particular synthetic or semi-synthetic fabrics may interact with the skin of Males, Females or Transsexuals (MtFs and/or FtMs), when these individuals put on these fibers directly on nude epidermis.

Transsexuals are to be considered, in this seat, all the subjects that take on purpose the relative opposite hormones to try to change or determinately change their sex, that is Men who take estrogens and Women who take Testosterone to grow transgender.

Hormonal influence is to be reputed the principal responsible cause of the occurrence of diverse cutaneous manifestations.

MATERIALS AND METHODS

We have selected three synthetic and semi-synthetic fibers, all strictly polychrome, that are:

Serge; Polycotton; Spandex.

And we have chosen for the experimentations 9 volunteers, as follow:

Three Men, and Specifically

A security guard which wears uniforms made of serge and who has been prayed not to use undergarment made of natural fibers for two weeks (during the summer season at hot temperatures and highest humidity level).

A carpenter which puts on a dungaree made of polycotton directly on his nude body.

A diver who continuously wears wetsuits made of spandex (lycra, elastan, creora).

Three Women, and Specifically

A policewoman who wears an uniform made of serge.

An employee in an industry producing jams who wears a dungaree made of polycotton.

A cyclist who puts on short pant made of spandex.

Three Transsexuals, and Specifically

The first is a transgender, that is he underwent castration, who has taking estrogens, from three years and likes to wear military uniforms.

The second is a transvestite (a will be transgender, taking estrogens only from two months and his hormonal asset could be defined quasi-eunuchoid type) who likes to wear coloured pantyhose made of polycotton directly on nude legs.

The third is a FtM who takes testosterone derivatives and who loves to take on packer, breast binding, laces and guipures made of spandex.

All the 9 individuals were prayed not to take (or to reduce dosage), when possible, Antimicrobials (e.g. Tetracyclines or Fuorquinolones), Nonsteroidal anti-inflammatory drugs (ketoprofen,Piroxicam), Phenothiaines, antidepressants, amiodarone, quinidine

Furosemide or thiazide diuretics, for one week before the experiment and for two weeks,during the same experiments, since the capacity to photosensitize skin these medicaments present is well documented [6].

The areas of the body We have scrutinized after two weeks of experimentations in each of every individual are chest in man, breast in woman, armits, popliteal fossae, antecubital fossae and finally the pudenda.

Ethics

This study was performed on human subjects; thus, all patients were aware of the presence of the study and they were fully informed about the drug and its side-effects.

RESULTS AND DISCUSSION

Results are simplest and there should be some argumentation to delve deeply, as far as men and women are concerned.

Objectively the three men, the individuals who wear serge, polycotton and spandex, keeping on account the diverse rate of sweating of everyone, do manifest after two weeks of experimentations (that correspond to the simple wearing the same coloured clothes, washing

themselves only once a day using an oily bath foam, pH5.5) solely symptoms of hives and folliculitis.

The three women, instead, after two weeks, are hit by simplest eritrodermie and Fisher's pigmented dermatitis.

The most suggestive results are represented by the three transsexual individuals and specifically the two MtFs manifest rushes of Schamberg's disease (especially on the legs) and the FtM is injured by a pustular dermatitis.

All these results may be explained by the fact that estrogens on bilateral castrated individuals increase the histamine production [7] and for this fact, the Schamberg's syndrome is well justified in the first case.

As far as the second subject is concerned, it is well known that estrogens when given to eunuchoids, are capable to inhibit the production of glucocorticoids by the adrenal gland, which is generally deputy to secrete cytokines, histamine and bradikinines and because of this constriction, the production of these three protein neurotransmitters tends to grow, owing to the consumption of estrogens by a man who did not undergo castration, although eunuchoid.

Regard the third case, the FtM, it is well documented that testosterone, [8] when given to female, primarily tends to increase the histamine-methylase and thus amplifying the production of histamine, and secondarily lets skin grow thicker, oily and more sebaceous and sebum is inclined to induce pustular dermatosis as well.

So, it is unequivocal that hormones play a very important role in the secretion of histamine and occurrence of dermal manifestations in subjects that consume opposite hormones to try to change gender.

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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Our Dermatology Online

Case Report

Torus palatinus. Report of two cases

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ABSTRACT

The torus is a non-neoplastic slow growing bone protuberance, which is usually manifested before the age of 30; Set in the hard palate is called "Torus Palatinus", and located in the lower jaw - "Torus mandibularis". In most cases, the diagnosis is usually incidental, during clinical examination, due to other reasons. The reason is that they are usually asymptomatic and patients are not aware of carrying a torus; hence the conservation treatment , unless it poses problems for the patient. We report two cases of incidental detected palatal torus in women.

Key words: Exostosis; Torus palatinus; Torus mandibularis

INTRODUCTION

Hyperostosis is defined as a benign neoformation of bone tissue, diffused or localized hypertrophy thereof. In the oral cavity can affect the lingual surface of the mandibular bone (torus mandibularis), the hard palate (torus palatinus) or presented as multiple exostosis [1]. The latin word torus means tumor or circular protrusion and is not considered as a pathological condition, but an anatomical variation. The term torus is introduced by Kupfer and Besselhagen in 1879, and is used to designate the exostosis arising on the midline of the palate and the back and inside of the jaw [2].

These bony growths are usually found in adults and occur after puberty. There is a predilection for females 2:1, in the palatal torus; as reported by several studies mandibular torus occurs most often in men [2,3]. It is estimated that 20-25% of the population, including Asian, Native American Indians and Eskimos have some torus, which are more common in women than men. According to the Institute of Reference of Oral Pathology, University of Chile, the frequency of torus in white Americans is 25%, 19% in black Americans and in Chile the prevalence corresponds to 37% [2].

The prevalence is between 6 and 8% in the United States and reaches 25% in other populations worldwide [1-3], while the incidence of mandibular torus is estimated below 8% [2,4]. Tori are more prevalent between 11 and 30 years old and very rare appearance before the age of 10. According to Eggen and Natvug, it is more frequent between 10 and 49 years old, and rare appearance after 50 [1-3].

Regarding its pathogenesis genetically originated are believed in large measure, but local factors micro stress and trauma can be contributory [3,5,6]. According to a research study conducted by Morrison MD and F. Tamimi, published in 2013, there was a statistically significant association between the presence of temporomandibular joint dysfunction, or the presence of dental wear, hypertension and predisposition to develop palatinus torus [5,6]

Pei-Jung Chao and colleagues [7], by a study conducted in 2013 among 119 hemodialysis patients and published in 2015, ruled the relationship between hyperparathyroidism raised as a cause of development of oral torus in chronic hemodialysis patients.

Clinically [1,2], manifest as compact prominences covered with healthy looking mucosa, they are usually asymptomatic, especially less than 1.5 cm;

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They are characterized by a well defined, unilateral or bilateral slow, benign, circumscribed bone growth. The torus can me located only under masticatory; ulcer resulting from trauma can take weeks to months to heal because the underlying bone tissue is poorly vascularized. They can be confined to the anterior or posterior part of the palate and in some cases may be spreaded throughout the midline from the pit prior to completion cleft of the hard palate. These abnormalities are usually symmetrical. According to their shape, palatal tori are divided into levels, unilobulados, multilobed, nodular, irregular or fusiform; the latter are the most common.

In histopathology [1-3]: the torus is characterized by dense bone outgrowth of a laminated pattern and small spaces occupied by thick bone marrow or scattered fibrovascular stroma, where you can observe minimum osteoblastic or occasional periosteal activity activity.

Tori require treatment when they are large, alter the phonatory function, generate dental displacement or produce trauma and ulceration of the mucosal surface, when they interfere with hygiene and are the cause of halitosis, but especially when preclude the placement and use of total or dentures. When treatment is indicated, the lesions can be cut or removed surgically, cutting from its base binding [1-3].

CASE REPORTS

Case 1

77 years old white female, housewife, from an urban area of Paraguay, hospitalized for community-acquired pneumonia, evaluated by dermatology by a raised lesion about 6-8 years of evolution, which gradually increased in size without accompanying symptoms. Tobacco, alcohol and other underlying pathologies refuses. Using of dental prosthesis smoothly. Family history is negative. Physical exam: unilobulated tumor of about 1 cm, net limits and regular edges, bright erythematous, smooth, solid stone consistency that is located on midline of hard palate (Fig. 1). Punch biopsy was taken for histopathology.

Case 2

30 years old white and healthy female, , from an urban area of Paraguay, with no personal or family history of pathological value evaluated in a routine examination of a raised lesion found on the hard palate; it is present since adolescence and is asymptomatic. *Physical exam*: oval tumor, unilobular, 1.5 x 1 cm in diameters, net limits, regular edges, covered by bright erythematous mucosa, of solid consistency, smooth surface, located on hard palate (Fig. 2). Punch biopsy was taken for histopathology.

Histopathology

At the base of the biopsy mature trabecular bone with osteoblastic rim, with osteocytes in lacunae covered by a cartilage cap formation. The intertrabecular space has vascular connective stroma, with no bone marrow (Fig. 3).

Final diagnosis in both cases: Torus palatinus.

Management

Patients were evaluated by dentistry and maxillofacial surgery, and because they do not have symptoms, periodical controls were indicated.

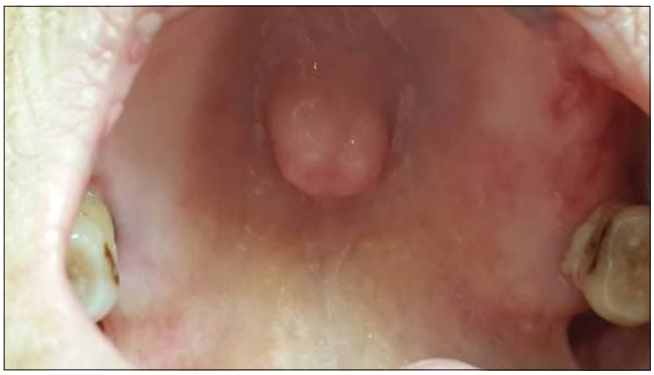

Figure 1: Clinical case 1.

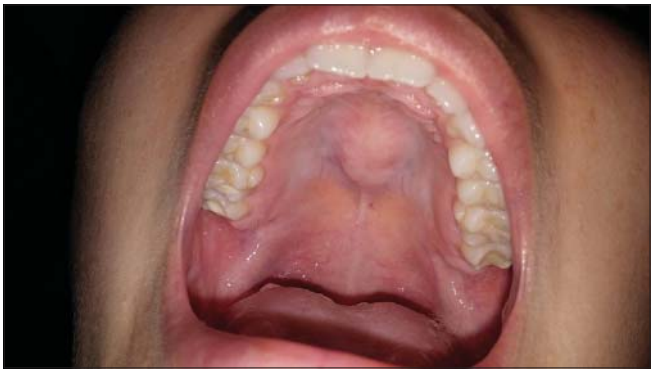

Figure 2: Clinical case 2. In both cases oval unilobular tumor, 1.5 cm in its great dimension, net limits, regular edges, covered by bright erythematous mucosa, of solid consistency, smooth surface, resting on hard palate.

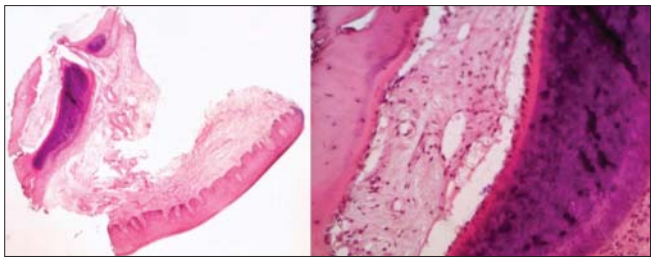

Figure 3: Histopathology. Mature trabecular bone with osteoblastic rim at the base of the biopsy (HE4X left), with osteocytes in lacunae covered by a cartilaginous cap formation. The intertrabecular space has vascular connective stroma, with no bone marrow (HE 40X right).

DISCUSSION

There are several research, case reports and case series publications on the torus, which mention that it is a benign oral pathology, most frequent between the second and the third decades, and is usually discovered unintentionally in the context of a query. According to the retrospective and descriptive study, carried out by Miranda-Gutierrez C., et al. [2], in patients attending the dental clinic at the Regional Military Hospital of Acapulco, during the period from January 1 to May 31 of the year 2013, the prevalence of torus was 3.88% torus. Females had a higher proportion of 1/427, while the male 1/887. In the age range 46-50 years is where the highest prevalence was presented with a percentage of 24%. In most cases (88.6%), did not require any treatment. These data are consistent with our two findings torus in women, young adult, and the other postmenopausal, in whom conservative treatment was raised.

Morrison MD., et al. described the Torus Palatine, as the most common intra oral exostosis among postmenopausal women [5], which could be due to the fact that they are asymptomatic, the diagnosis is made during routine visits or upon need for some type of dental prosthesis.

In fact, despite being a condition that is described as more frequent before 50 years of age, there are several case reports [3,6,8,9] and case series of fortuitous findings in older women as the case of one of our patients. It is rare in men but there are some case reports [10].

CONCLUSION

Torus Palatinus exostosis is a benign pathology, more common in women, and rare appearance after age 50; being asymptomatic in most patients, the finding is usually casual, hence the importance that general practitioners, dermatologists, dentists and pathologists are familiar with this disease, in order to make a correct diagnosis and management, avoiding perform surgical unnecessary treatments.

Consent

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

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Our Dermatology Online

Case Report

Arsenic and skin cancer – Case report with chemoprevention

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ABSTRACT

Introduction: Arsenic is a potentially hazardous metalloid that can cause skin cancer. We want to demonstrate a case of chronic arsenicosis and the potential of chemoprevention with retinoids. **Case Report:** This is a case report of a 72-year-old male patient who was exposed to arsenics by dust and direct skin contact over 3 years in a chemical plant in the late fourties. He developed multiple arsenic keratosis clincialll resembling actinic keratoses, Bowen's disease and palmar minute keratoses. To prevent a transformation into invasive cancer and to lower the burden of precancerous and in situ cancer lesions, he was treated orally with acitretin 20 mg/day. During 9 months of chemopreventive retinoid therapy a partial response of pre-existent skin lesions was noted. Treatment was well tolerated. During follow-up of 5 years no invasive malignancy developed. **Conclusions:** Intense exposure to arsenics during a relatively short period of 3 years bears a life-long health hazard with the delayed development of multiple in situ carcinomas and precancerous lesions. Chemoprevention with retinoids can induce a partial response.

Key words: Arsenics; Metalloids; Occupational hazards; Multiple Bowen's disease; Minute keratoses; Arsenic keratoses

INTRODUCTION

Arsenic is an ubiquitous metalloid which poses health risks for humans. Typical non-occupational and occupational sources of exposure are summarized in Table 1. Chronic exposure possess an increased risk for lung and bladder cancer [1,2].

Arsenic is a primary carcinogen in the skin following ingestion or topical exposure. Chronically exposed patients develop mainly precancerous arsenic keratosis, but in situ carcinoma (Bowen's disease and actinic keratosis) and invasive basal or squamous cell carcinoma have also been observed (Table 2) [3].

There is a number of occupational studies demonstrating a high risk of lung cancer related to arsenic exposure by inhalation. There is less data available on the possible association of occupational arsenic exposure with nonmelanoma skin cancer (NMSC). Recent studies suggest arsenic exposure increases the risk of NMSC as a cofactor to smoking and sunlight exposure [4,5].

The pathways by which arsenic is inducing skin malignancies are yet only poorly understood. In cell culture and animal models cytotoxity leading to increased but disturbed repair activities have been observed. A number of proinflammatory cytokines are released by cells exposed to arsenic including tumor growth factor-alfa. In animals no skin cancer was induced by arsenic. Arsenic is capable to induce post-translational histone modifications that may induce global transcriptional repression including tumor suppressor genes [6,7].

CASE REPORT

A 72-year old Caucasian man presented with multiple keratoses and pits on his palms for years ago. The patient had no history of a familial occurrence of such cutaneous signs. He was not under immunosuppression

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Table 1: Exposure to arsenic

Non-occupational
Drinking water
Food
Betel quid chewing
Chinese traditional medicine
Occupational
Miners
Metallurgy workers
E-material recycling workers
Farmers and tree planters exposed to fertilizers
Cooking plant workers
Outdoor workers exposed to urban air pollutants
Wine makers (and drinkers)

Table 2: Cutaneous manifestations of arsenic exposure

Exfoliative dermatitis	Pimparkar & Bhave 2010
Hyperkeratosis	Pimparkar & Bhave 2010, Wong et al. 1998
Vitiligo	Pimparkar & Bhave 2010; Hall 2002
Spotted melanosis	Hall 2002
Mee's lines on nails	Hall 2002
Bowen's disease [12,20,21]	Yahya & Mohammed 2005; Lee & Bebb 2005; Wong et al. 1998
Basal cell carcinoma [12,22]	Koc et al. 2014; Wong et al. 1998
Squamous cell carcinoma [12,23,24]	Elmariah et al. 2008; Mitropoulos & Norman 2005; Wong et al. 1998

nor had he received an organ transplant. His Fitzpatrick skin type was III. He reported that the lesions developed over four decades. His own medical history was unremarkable.

A thorough clinical examination and a skin biopsy were performed. On his palms small pits were found (Fig. 1). Nails, hair, teeth and oral mucous membrane had no pathologic symptoms. We observed more than 100 hypertrophic keratoses and Bowen's disease randomly distributed over the whole body except his face (Fig. 2). These lesions in conjunction with the pathological report suggest a chronic arsenic intoxication. On request it became clear that he became exposed to arsenics (arsenites and arsenates - dust and direct skin contact) during an occupation in a chemical plant for several years as war prisoner in the 1940ies (1945-1948). After his war prison years he was neither an outdoor worker nor a smoker.

First dermatological consultation was recorded in 1988.

During follow-up over 5 years, several lesions have been removed over time under the suspicion of an early invasive squamous cell carcinoma what could be excluded by histopathology.

There was no development of internal cancer such as lung or bladder cancer.

Figure 1: Palmar hyperkeratosis with minute pits.

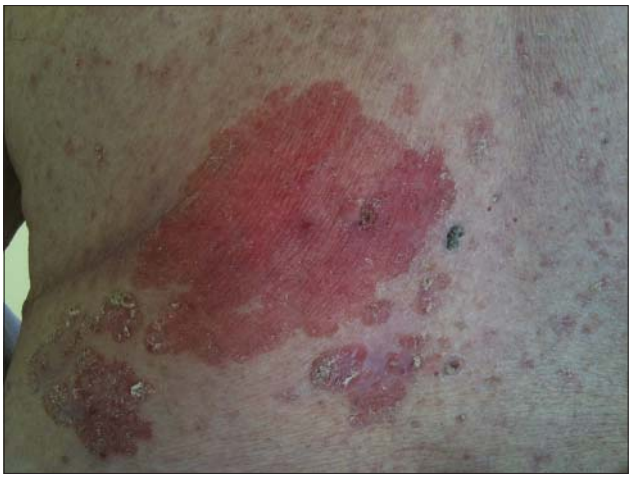

Figure 2: Multiple keratosis (Bowen's disease) on trunk (and extremities).

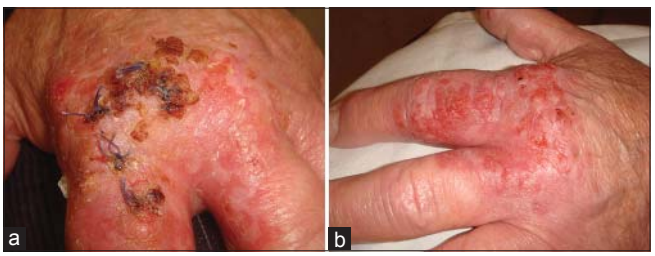

Figure 3: (a) Actinic hyperkeratosis before chemoprevention with acitretin, (b) Partial remission after 9 months of oral acitretin therapy.

He had neither a peripheral neuropathy nor cardiac arrhythmias. Laboratory test for plasma arsenic concentration of 24h-arsenic excretion were not performed since the exposure was decades ago. The diagnosis of chronic arsenicosis was made by medical history, clinical examination and histopathology of selected lesions. Histologically Bowen lesions and hypertrophic keratoses with atypical epidermal keratinocytes were observed.

He was given oral acitretin 20 mg per day as a chemopreventive measure for 9 months. The treatment was well tolerated under strict laboratory control. A partial regression of lesions was seen but not complete remission was achieved (Figs. 3a and b). On the other hand, no progression to invasive skin cancer has been observed.

DISCUSSION

Chronic arsenic exposure poses a health risk for the whole life [8-10]. Skin lesions like palmar pits, multiple keratoses and multiple Bowen's lesions on sunprotected skin are biomarkers for chronic arsenic exposure [11]. The development of precancerous lesions and in situ skin cancer is well known in the medical literature (Table 2) [3,10,12]. Invasive skin cancer such as basal cell carcinoma or squamous cell carcinoma has rarely been observed. Epidemiologic studies suggest that sunlight exposure and smoking are major factors which may become aggravated by chronic arsenic exposure [1,2]. Although skin carcinogenesis by arsenic is not completely understood, aberrant proliferation, release of proinflammatory cytokines, oxygen radical production, disturbed local immune response including impairment of p53 tumor suppressor function, and aggravation of UVB and UVA procarcinogenic effects have been detected [13].

Arsenite inhibits transcription of signaling kinase genes and downstream DNA repair genes DDB2 and RAD23B. Arsenite can displace zinc from the zinc fingers in proteins involved in DNA repair. These effects likely contribute to decreased nucleotide excision repair [14].

In our case no cofactors of arsenic toxicity (smoking or outdoor work) were evident. Drinking water is not a significant factor of chronic arsenic exposure in the region. So the only factor that could be established was a three year unprotected exposure to arsenics in a French chemical plant after World War II. It is remarkable that a relatively short but intense exposure to arsenics can cause ongoing health hazards. Patients with chronic arsenicosis need a lifelong follow-up since invasive cancer can develop with a delay of decades.

Chemoprevention of arsenic skin cancer has not really been established. In a phase I trial curcumin was applied in doses of 1 to 12 g per day. No toxicities were observed and a mild cancer protective activity was noted [15]. Other phytochemicals are under investigation [16]. A prospective trial has been initiated in Bangladesh with a 6-year supplementation with alpha-tocopherol (100 mg daily) and L-selenomethionine (200 μg daily) for the prevention of nonmelanoma skin cancer [17]. The future will show if the promises can be fullfilled.

Long term treatment with retinoids, in particular isotretinoin and acitretin, has shown activity in NMSC [18,19].Here we could demonstrate a partial remission of hypertrophic arsenic keratoses. If the treatment prevented lung and bladder cancer in our patient would be a matter of speculation only. Nevertheless, oral retinoid chemoprevention can be a measure in high risk patients after chronic arsenic exposure.

Key messages

- Chronic arsenicosis is a life-long disease with the potential of skin, lung and bladder cancer development.
- Symptomatology occurs after a delay of several years or even decades.
- Chemoprevention by oral retinoids is a measure for high-risk patients.
- A lifelong follow-up is recommended.

Consent

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

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Our Dermatology Online

Case Report

Follicular vitiligo: the present clinical status

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ABSTRACT

Vitiligo is a common autoimmune inflammatory disease where there is damage to the basal melanocytes of the epidermis. Hair follicles are the main reservoir of the melanocytes, and melanocytes stem cells, and these cells will supply the melanocytes for the basal layer of the epidermis when these cells are lost. But when these follicular melanocytes are damaged, this will cause follicular vitiligo either in a form gray/white hair or in form of follicular leukoderma. Although follicular vitiligo is not uncommon variant of vitiligo but rarely discussed and classified.

Key words: Follicular vitiligo; Follicular leukoderma; Follicular pigmentation

INTRODUCTION

Although the etiopathogenesis of vitiligo is still not well formulated but many findings, clinical and immuno-pathological are in favor of autoimmune theory singly or in combination with other etiological factors [1-5].

As a result of this autoimmune reaction, there will be loss of melanocytes from basal layer of the epidermis and the disease will be recovered when there are supply of new melanocytes from the hair follicles or from the residual melanocytes or its stem cells of the epidermis to reach the basal layer of the epidermis [1,5-8].

In some cases of vitiligo,hair follicles will lose their melanocytes resulting in follicular vitiligo [1-4,6-8].

So hair follicle are considered as a reservoir for melanocytes and as has been shown, there are many stem cells in the hair follicles especially in the permanent area of hair follicles so called bulge area and the outer root sheath. These stem cells are melanocytes stem cells, epithelial stem cells and neural crest stem cells, but the most important one is melanocytes stem cells which are closely related with epithelial stem cells.[9,10] Under critical conditions of epidermal melanocytes loss, these stem cells will proliferate, differentiate and move along the outer root sheath to reach the basal layer of the epidermis [9-11].

The hair follicles melanocytes appear more resistant to damage by de-pigmenting conditions like vitiligo, chemical leukoderma, occupational vitiligo and burn leukoderma.In these conditions the melanocytes and melanocyte stem cells of hair follicles will remain alive and active and ready to supply melanocytes to the white areas. In these conditions, hair follicle melanocytes will proliferate making functional cells for the basal epidermis, hence inducing active melanogenesis for the epidermis [9-11].

So following the treatment of these leukodermid conditions, we will see first follicular pigmentation and then re-pigmentation of the whole white abnormal areas (Figs. 1 and 2).

In cases of localized vitiligo when treated by melanocytes transplantation using needling micro-grafting technique, the vitiliginous skin will be covered by melanin in the first month following surgery while the white hair in these vitiliginous areas will take four months to be regimented.In these cases the melanocytes will make reverse journey to travel from basal layer of the epidermis into the outer root sheath, then move down to settle in the

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hair matrix in order to induce neomelanogenesis of the hair shaft [13,14].

FOLLICULAR VITILIGO

In ordinary vitiligo, commonly there is destruction of the basal melanocytes of the epidermis but in certain clinical conditions, there are also damage to hair follicles melanocytes that give rise either to gray/white hair or to follicular leukoderma of the skin [1-8], and all these conditions could be classified as follow:

Premature Grayness of Hair

This condition commonly appears before the age of 40 years and usually gives a diffuse pattern grayness of scalp hair with or without of facial hair but in some cases the picture will be in form of patchy grayness of the hair rather than in diffuse form. The pigment loss of hair matrix either will appear either as partial pigment loss so called early stage I depigmentation where the patients will notice a bunch of blond like (Fig. 3) hair rather than white hair while in late stage II of pigment loss, there is complete loss of hair shaft pigmentation and the patient will present with gray/white hair [2-4,9-12,15].

Follicular Vitiligo that is Associated with Ordinary Vitiligo

In these cases, grey hair has been seen in 15% of case of vitiligo where the patients will notice gray/white hair in the vitiliginous areas [2-4,9-12,15-18].

In cases of gray hairs, the grafted melanocytes will move from regimented epidermis into the outer root sheath then move down to reach the hair matrix thus inducing pigmented hair [13,14].

Follicular Vitiligo in Segmental Vitiligo

Where melanocytes of hair matrix of hair follicles are commonly involved causing grey/white hair in combination with vitiligo of proper skin (Fig. 4) [1-4,9-12,15-18].

Follicular Vitiligo in a form of the Grayness of the Body Hair

It is seen in combination with ordinary vitiligo, and this condition has been reported to involve the coarse hair follicle of the body presenting with whitening of the body hair [1-4,15-20].

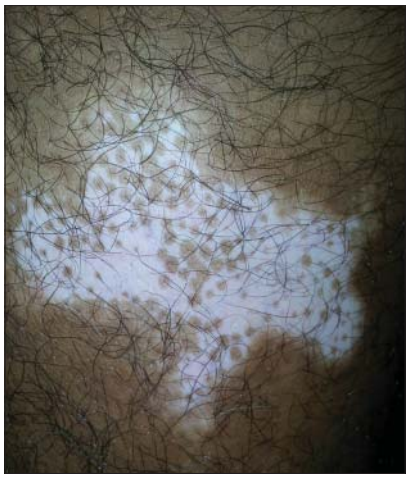

Figure 1: Patient with vitiligo showing a follicular repigmentation.

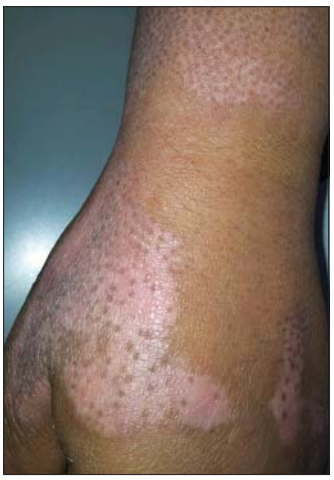

Figure 2: Showing follicular repigmentation of burn leukoderma.

Figure 3: Showing child presented with a bunch of blond hair in case of vitiligo of the scalp hair.

Recently

We noticed cases of vitiligo where the patients presented with follicular leukoderma where there is melanin loss of the skin at the hair follicles orifices

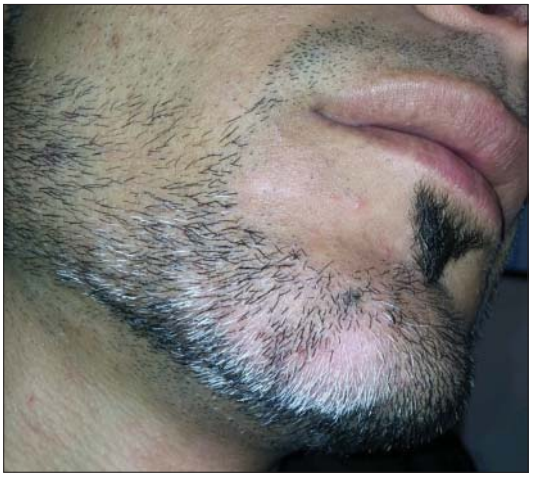

Figure 4: Showing a segmental vitiligo with gray/white hair.

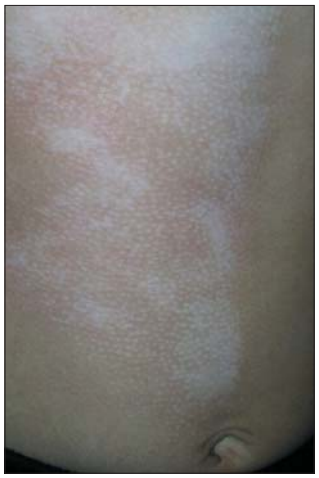

Figure 5: Showing follicular leukoderma of the trunk hair follicles coalescing together to form patches of ordinary of vitiligo.

first and then these follicular leukoderma will coalesce together to form ordinary vitiligo (Fig. 5).

In this type so called follicular leukoderma we assume that there is damage to the melanocytes of the outer root sheath rather than that of the hair matrix then might be followed by loss of hair matrix melanocytes resulting into grey hair.

CONCLUSION

Follicular vitiligo is not uncommon variant of vitiligo where there is damage to the hair matrix melanocytes and the patient will present with gray/white hair which commonly seen in segmental or non-segmental vitiligo or the destruction will involve the melanocytes of the outer root sheath and the patient will present with follicular leukoderma which gradually coalesce together to end with ordinary vitiligo.

Consent

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

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Our Dermatology Online

Case Report

Polypoid melanoma: a rare clinical subtype frequently confused with benign entities

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ABSTRACT

Polypoid melanoma represents a distinct variant of melanoma characterized by an ulcerated exophytic nodule. Although this melanocytic tumor is usually restricted to the papillary dermis, it presents with a thick Breslow level and aggressive course. Its rapidly growing vertical phase and its amelanotic nature frequently simulate benign or non-aggressive entities; leading to a delay in biopsy and resulting in an increased risk of metastasis at the time of the diagnosis.

Key words: polypoid, melanoma, dermatopathology, nodular, breslow

INTRODUCTION

Polypoid melanoma (PM) was first described by Manci in 1981 [1]. Histologically, it is differentiated from classic nodular melanoma by exhibiting an exophytic growth pattern that rarely extends into the reticular dermis [2,3]. This clinicopathologic variant is not common, and may have a delayed diagnosis. We present a case of PM to highlight the important features of this potentially aggressive subtype.

CASE REPORT

A 64 year-old male presented with a 6-month history of a rapidly growing nodule on his left flank. On physical examination, a 3 cm, well-circumscribed erythematous nodule with a partially eroded surface was found. The nodule was attached to the underlying skin by a narrow pedicle (Fig. 1). There was no lymphadenopathy. His past medical history was non-contributory.

Histopathological examination of the lesion showed a polypoid tumor composed predominantly of atypical melan-A-positive melanocytes, with a nodular expansile tumor growth, poor maturation and abundant mitoses (Figs. 2 and 3). An HMB 45 immunohistochemical stain demonstrated diffuse positivity with no evidence of a gradient throughout the lesion. A computerized tomography scan failed to reveal any evidence of metastatic disease. Two sentinel lymph nodes were negative for melanoma after histological evaluation.

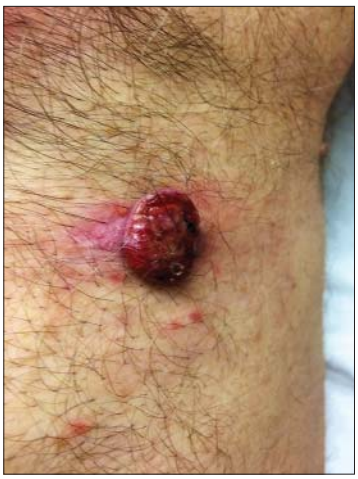

Figure 1: Partially eroded pedunculated tumor attached to the underlying skin by a narrow pedicle.

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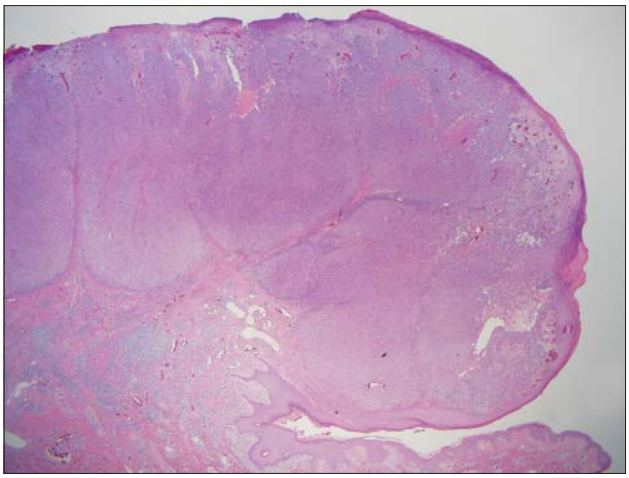

Figure 2: Polypoid neoplasm densely packed with tumoral cells, sparing the reticular dermis. H&E, x20.

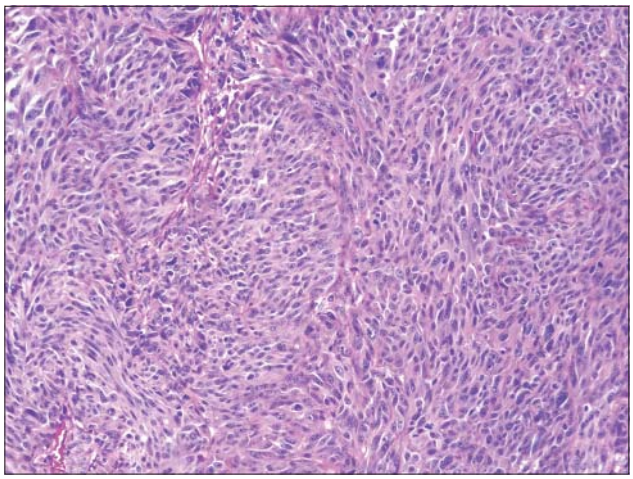

Figure 3: Sheets of atypical epitheliod melanocytes with abundant mitoses. H&E, x400.

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

DISCUSSION

PM is the most common melanoma type of the gastrointestinal tract, frequently presenting as a pedunculated mucosal tumor [4]. In contrast, cutaneous PM represents a rare subtype, reported in less than a third of cutaneous melanomas [5]. Clinically, the initial stage is that of a macule exhibiting a slow radial growth, which subsequently transforms within months into a rapidly growing vertical phase [6]. The clinical differential diagnosis includes benign entities such as pyogenic granuloma, vascular malformation or non-melanoma malignant tumors such as basal cell carcinoma or squamous cell carcinoma.

Traditionally, it has been considered to be part of the nodular melanoma spectrum, however, it may also arise from the acrolentiginous, superficial spreading and lentigo maligna types, displaying a growth pattern restricted to the papillary dermis [6]. Since the tumor typically does not penetrate deeply into the reticular dermis, its aggressive behavior was considered to be somewhat unique to the pedunculated subtype. Nevertheless, it is recognized that the exophytic nature of the tumor contributes to the Breslow level which, in addition to the ulceration, dictate the aggressive course of PMs.

CONCLUSION

It is important to consider PM in the differential diagnosis when assessing pedunculated tumors. The benign appearance of PM and its amelanotic nature may delay the diagnosis, increasing the risk of metastasis and poor prognosis. Awareness of these type of tumors may offer clinicians and patients early detection and appropriate treatment.

Consent

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

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Our Dermatology Online

Case Report

Primary cutaneous follicle center lymphoma: A case report

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ABSTRACT

Primary cutaneous B-cell lymphomas are defined as malignantB-cell proliferations presenting with cutaneous involvement alone and no evidence of extracutaneous manifestationswhen complete staging has been performed. Primary cutaneous folicul centre cell lymphoma (PCFCCL) is a relatively rare entity. Here, we report a 66-year-old female patient presented with six mounts history of erythematous papules and nodules over the skin of neck and body PCFCCL was diagnosed by skin biopsies. No evidence of systemic involvement was present at the time of diagnosis. She was not taken terapy but ones six mounts was done control and she hasn't got new lesions. Twelve months later she remains asymptomatic.

Key words: Primary cutaneous lymphoma; follicular centre cell lymphoma; cutaneous B cell lymphoma

INTRODUCTION

Primary cutaneous lymphomas show characteristic clinical and histopathological features and their biological behaviour is different from nodal lymphomas [1]. The majority of the lymphomas at different stages of development and which have not spread outside the skin are B-cell lymphomas. This type is more rarely observed than the primary T-cell lymphomas of the skin and comprise 20-25% of primary skin lymphomas. They are mostly low grade, progress slowly and have a good prognosis. The view that the lesions should stay limited to the skin for at least 6 months has lost its importance in recent years [2]. According to the classification by WHO-EORTC (World Health Organisation - European Organization for Research and Treatment of Cancer - Cutaneous Lymphoma Project Group), B-cell lymphomas of the skin can be classified into three main groups [3]:

1. Primary cutaneous marginal zone lymphoma
2. Primary cutaneous large B-cell lymphoma
3. Primary cutaneous follicle centre cell lymphoma.

In this case report, a middle aged female patient with multiple, indurated, erythematous papules and nodules in the neck and back area and histopathologically diagnosed with primary cutaneous follicle centre cell lymphoma of the skin (PFMHL) is presented and the disease is discussed in the light of the current information.

CASE REPORT

A sixty year old patient presented to our clinic with a red rash in her neck and back which were present for six months. During the dermatologic examination, numerous erythematous and indurated red and purple papules and nodules were observed in her neck and back (Fig. 1). No lymphadenopathy or hepatosplenomegaly was observed during her systemic examination. Skin biopsies were taken from the lesions with the pre-diagnoses of cutaneous B-cell lymphoma, sarcoidosis, multiple myeloma, lymphomatoid papulosis, pseudolymphoma, cutaneous plasmacytoma, and mastocytosis.

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DOI:10.7241/ourd.20162.49

Histopathologically, nodular lymphoid aggregates with large hyperchromatic nuclei and irregular contours, some with visible nucleoli and narrow cytoplasm, resulting from small to medium diameter, atypical neoplastic lymphoid cell proliferation and mixed with centrocytes and centroblasts were observed in the perivascular areas within the dermis (Fig. 2). Around these lymphoid aggregates, mantle zones were observed and tingible body macrophage loss was detected within the follicles.

The immunohistochemical examinations revealed diffuse and strong staining with CD 20 (Fig. 3), CD79a, Bcl-2 and Bcl-6, and positive staining with CD23 in the focal areas. No immune reaction was observed with CD 10, Tdt and cyclin-D1. In a small number of mature lymphocytes, positive (+) staining was observed with CD3, CD4, CD8 and CD5. Only a few immunoblastic cells were stained using CD 30. The Ki-67 proliferation index was around 20-30% at its highest level (Fig. 4). Based on the morphological and immunohistochemical findings at hand, the patient was diagnosed with atypical cell proliferation and mainly with "follicular centre cell lymphoma".

The patient was referred to the haematology clinic to investigate any systemic involvement. The tests revealed no pathological laboratory findings. The patient's thoracic X-ray was normal. The peripheral smear and bone marrow aspiration and biopsy were evaluated as normocellular. The cervical and pelvic tomographies were observed to be normal. The biopsy of the lesion detected during the abdominal tomography, which was first thought to be a metastasis, pointed out a hemangioma. Since the scans did not reveal any foci, the haematology clinic did not start any treatment protocol and recommended follow up visits

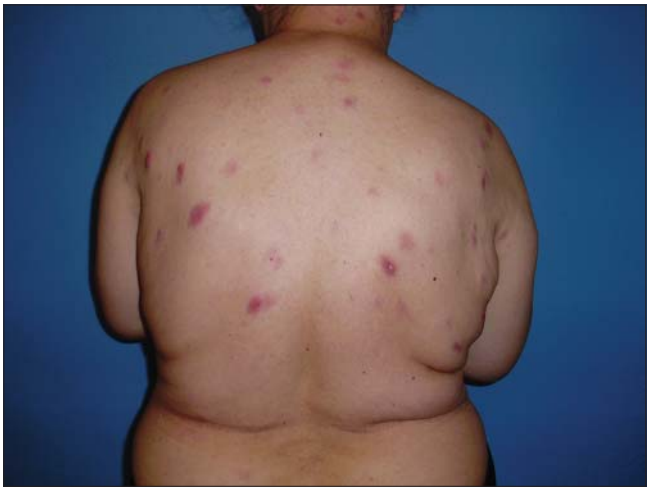

Figure 1: Papulonodular lesions observed in the neck and back.

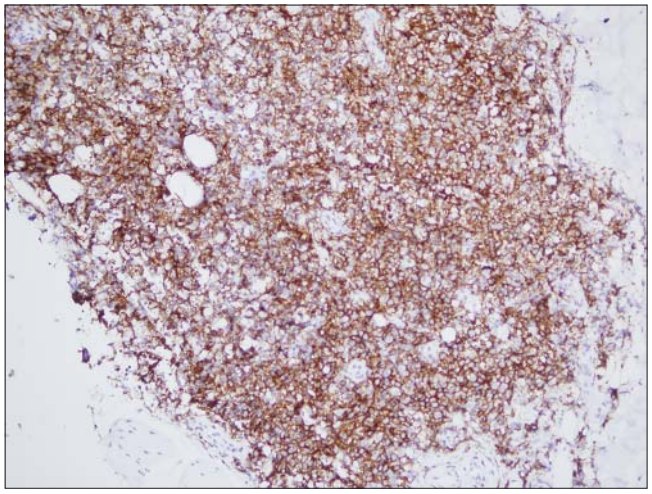

Figure 3: Diffuse staining of strong intensity with CD20 in the lymphoid cells. x200.

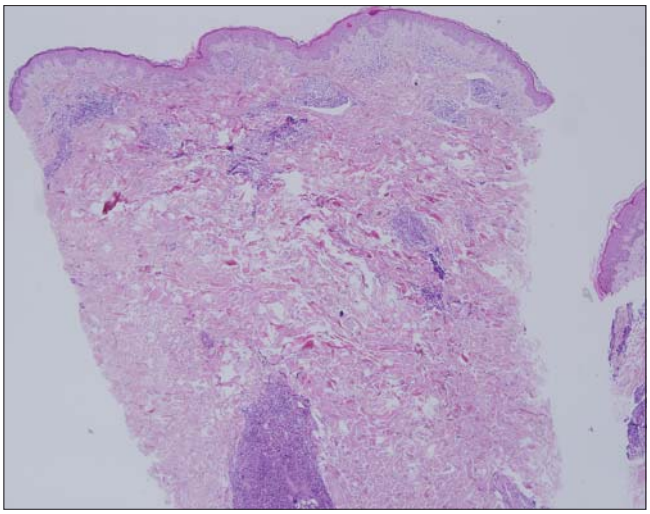

Figure 2: Small nodular lymphoid aggregates in the dermis. H&Ex40.

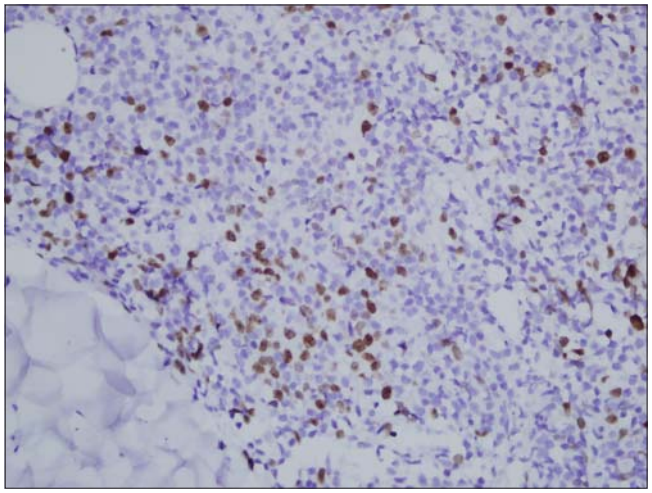

Figure 4: The Ki67% proliferation index was observed around 20-30 in the densest areas. x400.

every 6 months. No systemic involvement was observed during the follow up. In the last visit, the patient stated that some of the lesions had disappeared, only to be replaced by new ones.

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

DISCUSSION

PFMHL is the most frequently observed type of cutaneous B-cell lymphomas comprising approximately 40% of all the cases [1]. It features neoplastic B-cells morphologically and immunophenotypically resembling follicle centre cells and including the centrocytes and centroblasts in the skin. It is mainly located on the head and at the torso, and shows a slow progress. It usually grows diffusely and with few follicular growth patterns. Lesions located at the back are defined as "Crosti lymphoma" or "reticulohistiocytoma of the back" [2-5]. The dermatological examination reveals individual or grouped, red to brown coloured papules, plaques and tumours, while ring-shaped erythema may also be observed around certain lesions. Ulcerations are not commonly observed and the lesions are usually asymptomatic. They are frequently located on the hairy skin, forehead, neck and torso, while the lower extremities are rarely involved [6]. Multifocal lesions are not an indication of a bad prognosis. The clinical development of the disease shows a slow progress though the lesions may spread wider if left untreated. The disease rarely spreads to the other tissues than the skin. It is usually observed in the 5th and 6th decades of life [2], although cases observed in younger patients have also been reported [7]. The thoracic, abdominal and pelvic CT scans and the bone marrow biopsy performed for the purpose of staging is negative during the diagnosis [2].

The histopathological examination reveals diffuse or nodular infiltration in the reticular dermis and subcutaneous tissue. The epidermis is unaffected and there is no epidermotropism. A clear grenz zone is visible in the papillary dermis [2,4-6]. As the tumoral lesions progress, the diameter and number of the neoplastic B-cells increases, while the number of the reactive T-cells decreases. A mix of centrocytes, centroblasts and reactive T-cells is observed in early and small lesions. In large and rapidly growing tumours, monomorphic follicle centre cells, large centroblasts, multilobulated cells and rarely spindle cells are observed. During the immunophenotypic examination, 60-70% of the cases are positive in terms of monoclonal rearrangement. B-cell phenotypes CD19, CD20, CD22 and 79a are positive. While Bcl- 6 is positive, CD- 10 is frequently (+) and CD5 is (-). The negative Bcl-2 protein and no t(14: 18) translocation are the differentiating factors from systemic nodal follicular lymphomas during the diagnosis. The prognosis is disputable in the patients where more than 50% of the neoplastic B-cells produce Bcl- 2. Staining through the active markers of B-cells such as MUM- 1/IRF4 and FOXP1 is negative in most of the cases [2,5]. The t (14: 18) translocation is characteristic in the cytogenic examination of systemic follicular lymphomas [6]. While the t(14: 18) and Bcl- 2 expressions are negative in the majority of the follicle centre cell lymphomas, inactivation of the P15 and P16 tumour suppressor genes is rare. Pseudolymphomas are important in the differential diagnosis [2].

There are varying approaches to the treatment and different protocols are proposed. These include radiotherapy in the early stage; chlorambucil multiple chemotherapy (CHOP), and intralesional anti-CD20 monoclonal antibodies for middle stage; and interferon, IL- 2, systemic steroids in the advanced stage and in case of multiple lesions; and the excision of solitary lesions [1]. Radiotherapy may be added in case of repeating solitary lesions [5,6,8-10]. The prognosis is very good regardless of the growth patterns and the number of the blast cells and the 5-year survival rates are over 95% [3]. The lesions tend to recur in 30% of the cases, although it is not a sign of a poor prognosis [2].

We did not observe any systemic involvement in our patient during the diagnosis and the follow up.

The case of a primary follicle-centre cell lymphoma has been presented due to its rarity.

Consent

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

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Our Dermatology Online

Case Report

Sebaceous carcinoma of the forehead: Case report

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ABSTRACT

Sebaceous carcinoma is an uncommon malign tumor arising from sebaceous glands. Sebaceous carcinoma typically occurs on the eyelid as a slow growing, pink-yellow, painless subcutaneous papule or nodule. Although it is rare, sebaceous carcinoma can also be seen on head, neck, trunk, extremities, breasts, oral mucosa and external genitalia. Sebaceous carcinoma is a locally aggressive tumor, moreover it can metastasize to regional lymph nodes and distant organs. Hereby, we report a 91-year-old Caucasian female with sebaceous carcinoma on the forehead. The extraocular 1,9 cm large sebaceous carcinoma on the forehead was removed surgically with wide local excision under local anesthesia.

Key words: Sebaceous carcinoma; Extraocular; Surgery

INTRODUCTION

Sebaceous carcinoma is a malign tumor of sebaceous glands and it is usually found on the eyelid. However, sebaceous carcinoma can arise anywhere in the body where sebaceous glands exist. Extraocular localization of the sebaceous carcinoma is rare and accounts for 25% of all cases. Sebaceous carcinoma is more common in women between the ages of 60 and 70. Moreover, Asian population is more likely to develop sebaceous carcinoma [1]. Sebaceous carcinoma usually presents as a slow growing, painless, pink-yellow papule or subcutaneous nodule [2]. Sebaceous carcinoma is locally aggressive, therefore it may metastasize to regional lymph nodes and also to distant organs [1]. On the other hand, extraocular sebaceous carcinoma is less metastatic compared to ocular sebaceous carcinoma. Because of the aggressive behaviour of the tumor, a sebaceous carcinoma should be treated with wide surgical excision [3].

CASE REPORT

A 91-year-old Caucasian female presented with a solitary mass on the forehead for further evaluation. The patient admitted that the lesion had gradually increased in size in the last 2 years. The physical examination of the patient revealed a painless, immobile, firm, yellow-pink, 2,5x2x1,5 cm ulcerated exophytic mass with irregular surface on the forehead. A wide local excision was performed under local anesthesia. The patient did well postoperatively and she was discharged home the same day without any complications. The histopathological evaluation of the specimen revealed sebaceous carcinoma with the largest size of 1,9 cm. The tumor was removed completely. Deep and lateral surgical margins were free of tumor cells which were confirmed histopathologically. The patient was referred to the medical oncology department for further close follow-up. Figs. 1-4 show pre- and post operative photographic views of the lesion.

DISCUSSION

Sebaceous carcinoma is a rare, aggressive tumor which arises from both periorbital meibomian and Zeis glands but also from extraocular sites [4]. Sebaceous carcinoma represents less than 0,1% of all cutaneous malignancies [5]. Moreover, extraocular sebaceous carcinoma accounts for 25% of all sebaceous carcinoma cases. Sebaceous carcinoma can be found on trunk, extremities, external genitalia, breasts, head and

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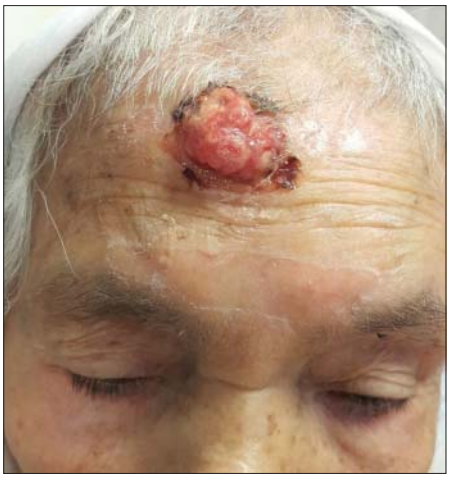

Figure 1: A yellowish-pink, immobile, firm, ulcerated exophytic tumor with irregular surface on the forehead of a 91-year-old female.

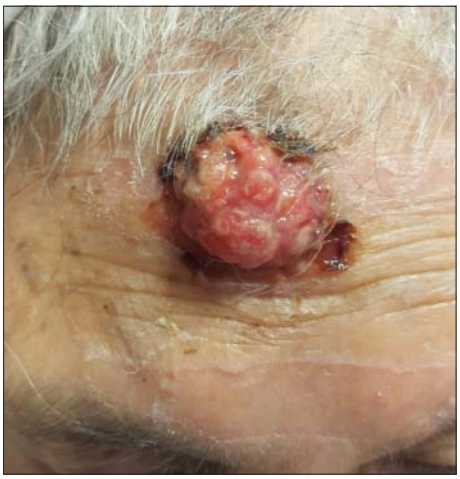

Figure 2: A yellowish-pink, immobile, firm, ulcerated exophytic tumor with irregular surface on the forehead of a 91-year-old female.

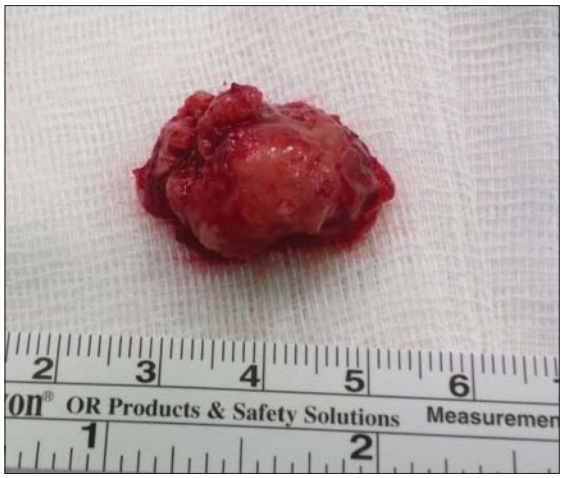

Figure 3: Specimen: 2,5x2x1,5 cm. The histopathological evaluation of the specimen revealed sebaceous carcinoma with the largest size of 1,9 cm.

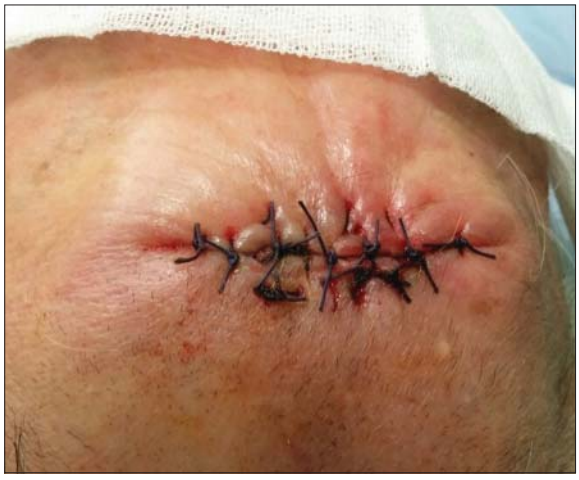

Figure 4: The incision was closed primarily.

neck region such as face, scalp, ears, lips and salivary glands [4,6]. Sebaceous carcinoma presents as firm, painless, pink-yellowish-red subcutaneous nodule or mass which may ulcerate and bleed spontaneously [7]. Risk factors for developing sebaceous carcinoma include older age, history of irradiation, immunosuppression after solid organ transplantation and Muir Torre Syndrome [8]. Muir Torre Syndrome associated with sebaceous carcinoma is an autosomal dominant genodermatosis characterized by sebaceous neoplasms, keratoacanthomas and visceral malignancies [5]. The differential diagnosis of sebaceous carcinoma includes intradermal nevus, chalazion, blepharitis, sebaceous adenoma, basal cell carcinoma and squamous cell carcinoma [4,7]. Histopathological examination is mandatory to reach a definitive diagnosis [5]. Wide local excision is recommended for the treatment of an extraocular sebaceous carcinoma because of the risk of local recurrence. Distant metastasis and mortality have been reported in 20-25% and 20% of all sebaceous carcinoma cases respectively [6]. It has been suggested that Mohs micrographic surgery should be performed to decrease the rate of local recurrence. In elderly patients radiotherapy is the treatment of choice for recurrent tumors and metastatic lesions [7]. The most important prognostic factor for sebaceous carcinoma is early diagnosis. The mortality rate rises from 14% to 38%, if the diagnosis is made after the first six months [9].

CONCLUSION

The case presented above is a rare example of extraocular sebaceous carcinoma on the forehead. Sebaceous carcinoma has a high metastatic potential, therefore early diagnosis of sebaceous carcinomas is of paramount importance to improve survival.

Unfortunately, the presented sebaceous carcinoma lesion reached a size of 1,9 cm within 2 years without medical attention. Therefore, patients should be warned to seek medical attention for any growing cutaneous lesions and sebaceous carcinoma should be kept in mind in the differential diagnosis of cutaneous malignancies of the forehead.

Consent

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

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Our Dermatology Online

Case Report

Pilomatricoma of the orbit

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ABSTRACT

Pilomatricoma is an uncommon, benign skin neoplasm, originating from the matrix of the hair root. These lesions are typically found in the head and neck region, but also occur in the upper extremities and are rarely report in the other sites. It is most commonly seen in children and adolescents with female predominance, but extremely rare in middle age on eyelid or eyebrow. We report a 41-year old male, who developed a single,subcutaneous tumor that gradually and progressively grew over one year period. On physical examination, there was a, firm, painless, non- ulcerated, nodule in the right orbit. Upon computed tomography (CT) scan, well- circumscribed enhancing nodulewas found. The presumed diagnosis of dermoid, epidermoid cyst was made. An excisional biopsy performed for definite diagnosis and treatment. Histopathologic examination with hematoxylin-eosin staining confirmed that mass to be pilomatricoma. Pilomatricoma of the eyelids and orbit is often misdiagnosed clinically and is extremely rare en middle age. This is a report of unusually pilomatricoma in orbit of an adult male. The patient has no symptoms or sign suggestive of recurrence at 5 years after nodule excision.

Key words: Pilomatricoma; Eyelid; Skin neoplasm

INTRODUCTION

Pilomatricoma or calcifying epithelioma of Malherbe is a relatively uncommon originating from the matrix of the hair root [1-4]. Pilomatricoma may be located in any part of the body [4-6] except the palms and soles but, has a predilection for de upper lid and eyebrow [1,3].

It is most commonly seen in children and adolescents [3]. Forty percent of them develop in the first decade of life and other 20% in the second decade [1,7] although can occur at any age. It is more common in female by radio of 3:2, but rarely seen in male adults on the eyelid and eyebrow region [1,5].

Pilomatricoma is usually not considered in differential diagnosis of firm skin nodule on eyelid and orbit. This is a report of an unusually, single, firm, nodular, non-ulcerated, painless orbital pilomatricoma located under right lower eyelid of an adult man. This entity has to be considered in the differential diagnosis of such lesions involving orbit.

CASE REPORT

A previously healthy 41-year old male had a large but asymptomatic lesion in the right orbit (Fig. 1a). He had first noted a small papule that gradually and progressively grew over 1 year period.

On physical examination, the right orbit lesion was a firm, hard, mobile, 10x10- mm nodule in subcutaneous tissue. There was no significant medical or surgical history. The remaining ocular examination was normal. Upon CT scan, there was a circumscribed nodule with peripheral enhancement located at the right eyelid (Fig. 1b).

A presumptive diagnosis of dermoid or epidermoid cyst was made.An anterior orbitotomy was performed under local anesthesia. A skin incision was undertaken in the infraciliar margin. The lesion was meticulously dissected of the surrounding tissues and its complete removal was accomplished (Fig. 1c).

Gross pathology revealed brown-yellowish firm tissue, measuring 11x7x6 mm. Histopathology showed

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fibroadipose tissue with well demarcated nodule, comprised of basaloid and shadow cells with adjacent areas of intralesional calcification (Figs. 2a and 2b). The overall histological findings were suggestive of pilomatricoma. There was no evidence of recurrence at 5 years after nodule excision.

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

DISCUSSION

Pilomatricoma is a benign skin tumor arising from cells of the hair follicle [1,7-9]. Its incidence is 1/800-1000 cutaneous tumors, affecting predominantly women [10]. Four clinical variants have been described: and eruptive type, a perforating type, a familial type associated with myotonic dystrophy and Gardner syndrome [11].

Clinically presents as a firm, single, stony hard slow-growing subcutaneous or intradermical nodule,

Figure 1: (a) Clinical appearance of the swelling under right lower eyelid. (b) Computed tomography showing well circumscribed enhancing nodule. (c) Peroperative exposure of tumor.

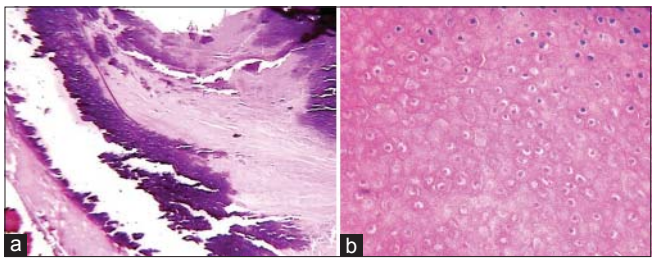

Figure 2: (a) Histological section where the wall of the solid lesion is noted and where appreciates a hypocellular area and calcified areas. H&E x100. (b) Histological section, a high power, where the wall of the lesion was observed, calling attention remnants of epithelial cells (ghost cells). H&E x400.

asymptomatic, adherent to the skin, but not fixed to underlying tissue [1,11-13]. Lesions are usually skin color, but reddish-blue, lesions have been observed probably resulting from the hemorrhage [5].

Periocular tissues are involved in 10-17% of cases and 5% of cases are multifocal [7]. The size of the pilomatricoma varies from the 0.5 – 3.0 cm but giant periorbital lesions up top 3.0 cm have been rarely reported [5].

Most of the eyelid pilomatricoma reported so far have been in young adults and rarely in older patients [5] and female predominance ranging from 55 – 75% [13]. The appearance of multiples pilomatricoma has been occasionally associated with myotonic dystrophy (Steinert disease), Turner syndrome, Gardner syndrome, sarcoidosis [10,13-15] trisomy 9 [12].

Differential diagnosis of pilomatricoma was varied among the three age groups: pilomatricoma in children may be mistaken for epidermoid cyst, sebaceous cyst, dermoid cyst or hemangioma [8]. Differential diagnosis of adult an elderly cases was considered as fibrohistiocytic proliferation (dermatofibroma or fibroma), dermal nevus, adnexal tumor, cutaneous metastases [13] keratoacanthoma [13,14,16] and basal cell carcinoma [1,4].

Preceding trauma, surgery, infection or insect bite [4,12] intramuscular injection at the site of occurrence of tumor has been reported [9]. In general pilomatricoma is not hereditary [4,12] Etiology has been linked to mutations such as B catenin and bcl-2 [15]. It has been showed trisomy 18 as a consistent feature in pilomatricoma [17].

Histopathology examination reveals grossly well circumscribed and firm gritty nodule [7] with basaloid cell and keratinized eosinophilic shadow cells located towards the center of the tumor and occasionally foreign giant body cell reaction [7,11,13]. Calcification (70-80%), ossification (15-50%) were common features. Although can showed cellular evolution toward the other parts of the hair follicle, such as the other an inner root sheaths, sebaceous and infundibular components, therefore, can be considered a panfollicular neoplasm [18].

However, malignant transformation of pilomatricoma was rare, and tended to occur in middle-age or elderly cases [13]. The histopathological features that distinguish a pilomatrixcarcinoma from the

pilomatricoma are: asymmetry of the tumor, infiltrative borders, and predominance of basaloid cells with atypical mitoses, nuclear pleomorphism, and necrotic foci, invasion of vessels and nerves and infiltration into underlying structures [11,13].

The characteristic computed tomography appearance has been described to be that of a non-contrast enhancing, sharply, well-demarcated subcutaneous nodule [15] soft-tissue mass adherent to the skin with or without visible calcifications [11]. The treatment of pilomatricoma including a complete excision biopsy with narrow margin, is usually satisfactory [4,7,9].

The present case has showed no recurrence or metastases for 5 years since the resection.

In conclusion, periocular pilomatricoma is often misdiagnosed clinically and correct diagnosis is only established after excision and histopathological examination, so, it should also be considered in the differential diagnosis of the lesion involving eyelids and orbit.

Most of pilomatricoma have a predilection for the upper lid, eyebrow and canthal area, occur more frequently in children with female predominance and rarely seen in male adults. We have described in middle-age man an extremely rare pilomatricoma of the orbit under the right lower eyelid as this case is showed.

Most case reports were documented in pediatrics, otolaryngology, dermatology literature, ophthalmologists may not be familiar with this entity, and consider it in the differential diagnosis of subcutaneous nodule in the periocular region. The present case has showed no recurrence or metastases for 5 years since resection.

Consent

The examination of the patient was conducted according to the Declaration of Helsinki principles written informed consent was obtained from the patient for publication of this article and any accompanying image.

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Our Dermatology Online

Case Report

Dermatoporosis, an emerging disease: case report

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ABSTRACT

The term dermatoporosis proposed by Kaya and Saurat in 2007, relates to the failure and chronic skin fragility due to aging. The first clinical manifestations appear by the age of 60, but fully developed signs of disease are evident among people between the ages of 70 and 90; affecting 1 in 3 people, with a prevalence of 32%. Typical manifestations of dermatoporosis are skin atrophy, senile purpura, stellate scars, skin lacerations, and dissecting hematomas. Injuries can be classified into primary and secondary. The primary are related to the timing of aging and photoaging; while secondary lesions are associated with chronic use of topical or systemic corticosteroids. The lesions usually appear on sun-exposed areas. They indicate the fourth stage. We report a case of a 74- year- old woman with deep dissecting extremity hematomas.

Key words: Dermatoporosis; Photoaging; Cutaneous dissecting hematomas

INTRODUCTION

The skin is the largest organ of our body and noteworthy it provides a protective barrier from external aggression. With skin aging, normal process mainly characterized by appearance of wrinkles, forms a thinner and dry skin, and all metabolic and physiological functions of loosing skin functionality. When skin aging is mainly exacerbated by lossing or decreaseing amount of intradermal hyaluronic acid, a modification of the extracellular matrix is produced by the likely effect of ultraviolet radiation or the non-enzymatic glycation of structural proteins, causing loosing skin elasticity [1-3].

Skin aging, may also have similar pathological signs to brittle bones (osteoporosis), consisting of an exaggerated skin fragility presenting with erosion and ulcers [4], similar to bedridden patients ulcers where the collagen in the dermis could be affected. This is known as a chronic failure of the skin or dermatoporosis.

Aging skin, weakened by the combination of chronological aging and photoaging, may have diminished the forces of friction and shear tolerance; so both processes, the chronological and photoaging have been cited as risk factors for developing pressure ulcers. Although mentioned the delay in wound healing as a feature of dermatoporosis, there are few publications that relate to the development of pressure ulcers with this condition. [5,6].

This syndrome consists of some structural tissue abnormalities including modifications of the extracellular matrix such as collagen and elastic fibers, and alterations in skin viscoelasticity. Furthermore, the histopathology of the lesions characteristic can show the flattening of the rete ridges and solar elastosis [7]. The lesions usually appear on sun-exposed areas such as the forearms, back of hands, anterior legs and chest; moreover, these areas are accesible to scratching because of the pruritus caused by dry skin itself and in many cases this scratching is the cause of injuries [3,4,8]. Within the morphological signs of dermatoporosis the presence of senile purpura, skin atrophy and scars are included. The loss of protection function results in delayed healing, lacerations after a minor trauma or deep bruises forming dissecting that, if are not drained,

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Table 1: Stages of dermatoporosis

Clinical features	Stage I	Stage II	Stage III	Stage IV
Skin atrophy: Very thinned and numerous wrinkles	+	+	+	+
Senile purpura: Repetitive spontaneous bleeding in the dermis without coagulation abnormalities	+	+	+	+
Skin laceration: Expression of the fragility of the skin following minor trauma	+	+	+	+
Starry scars: Spontaneous skin lacerations with stellar appearance	-	+ (<10)	++ (≥10)	++ (≥10)
Skin dissecting hematoma: Hemorrhage subcutaneous mass, which leads to ischemia and skin necrosis	-	-	-	+

Quoted and modified Kaya and Saurat, 2007 [1,5]

evolving into large necrotic areas [8]. 4 stages have been described [1,5,9] (Table 1).

Stage I is characterized by the presence of senile purpura, skin atrophy and scarring. Stage II also associates some located lacerations. Stage III has multiple lacerations and is associated with delayed healing. Stage IV associated dissecting deep bruises evolving into large areas of necrosis [1,8]. They are deep dissecting bruises on legs after the minor trauma. The vessels, which are fragile by age and placed under an atrophic skin, bleed easily. The bleeding vessel are located between the subcutaneous tissue and muscle fascia, and initially manifests clinically as red, edematous areas, with increased local temperature, which is clinically confused with cellulitis, which are usually treated with oral antibiotics. If the subcutaneous hematoma is not drained at the proper time, the ischemia of skin occurs and appear large necrotic areas requiring wide surgical debridement. The differential diagnosis must include vascular occlusion syndromes that affect the skin, mainly non inflammatory causes, although some may present with minimal inflammatory clinical lesions and skin necrosis. These syndromes result in clinically retiform purple type of injuries that can evolve into necrotic areas [1,8]. Healing of these wounds can be prolonged due to a process in which there is a decreased fibroblastic proliferation, which can be accompanied with local exudates, the risk of bacterial colonization and increased metalloproteases in exudates.

According to the stages [8] management schemes have been proposed: In stage I, II and III, it is proposed to perform applications of paraffin oil, debridement of scabs and small necrotic areas, application of Lanolin-based emollient, zinc oxide and hyaluronic acid throughout the affected area and cover with hydrocolloid meshes to avoid wetting the wound to the bed, to the secondary dressing of calcium alginate or according to the level of exudate gauze compresses applying a clamp or a semi compressive dressing.

Dermatoporosis in stage IV cases, requires a surgical debridement of the hematoma, ablution the lesion with saline solution and application of a desbridante dressing composed of Ringer, along with a cleaning solution with Polihexanide, also checking that the bed of the lesion does not show tunelization.

Once presented granulation tissue in the wound bed, is proposed to continue with the process of healing and epithelization, with the application of hyaluronic acid more topical retinaldehyde; according to some clinical trials performed for the treatment of the dermatoporosis, fragmented hyaluronic acid and the retinaldehyde show an effect dependent on the dose in the correction of atrophy of the skin [8,10].

CASE REPORT

Female, 74, housewife, from an urban area of Paraguay, admitted in an intensive care unit (ICU) of a public hospital for/because of sepsis with starting point in an urinary tract infection, which is treated with ceftriaxone. In its 8th day of hospitalization in ICU appearance of edema of both arms, with redness, prone to spontaneous bleeding and citrine suffusion fluid following minimum procedures. The case is interpreted as cellulite on upper limbs and after clinical worsening ceftriaxone is rotated to piperacilin tazobactam. In its 12th day of hospitalization a dermatological evaluation is performed because of the erythema and bruises on arms and legs.

Personal history: 3-month history of violaceous macules and bruises that appear spontaneously, without accompanying symptoms, difficult to cure, which are increasing in number. Fine, dry and frequently itching skin.

Physical exam: skin atrophy. Bruises of 3-20 cm -some of them qualify to be dissecting and lacerations from 1.5 to 10 cm in upper and lower limbs. Erosion of 15 cm in diameter with necrotic and jagged edges, in legs and thighs (Figs 1 and 2).

Histopathology: epidermal atrophy with loss of rete ridges and basal hyperpigmentation. Orthokeratosis.

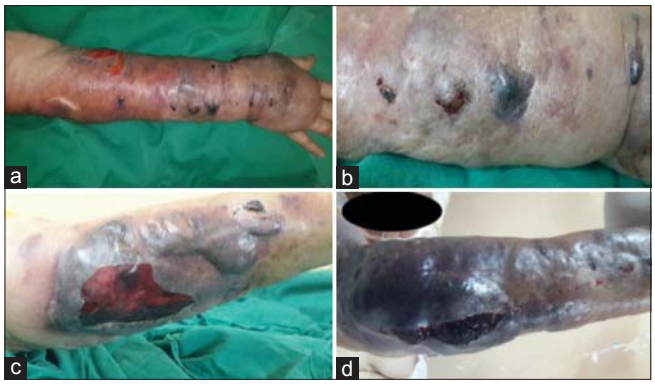

Figure 1: Upper limbs. a. Edematous limb, erythemato purpuric skin atrophy, erosions and hematic vesicles. b. Lacerations, stellate scars and bruises. c and d. Dissecting bruises, lacerations and purpura.

Figure 2: Edematous necrotic ulcerated plaque, defined limits, jagged edges on lower limb. Ulcers from 10 to 15 cm in diameter net limits, jagged edges, granulomatous background. a: left thigh. b: left leg.

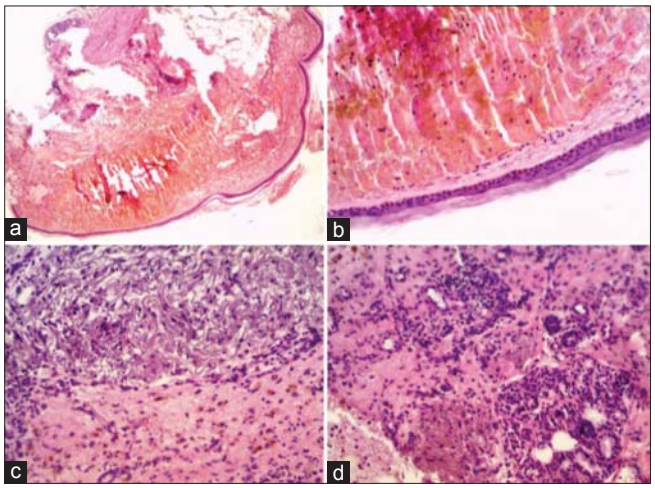

Figure 3: Histopathology. a and b. Epidermal atrophy with lost of rete ridges. Orthokeratosis. Vasocongestion and telangiectasias in dermis with hematic extravasation. c. Degeneration of elastic and collagen fibers of the dermis. d. Scant mononuclear infiltrate and perivascular siderophages.

Vaso congestion and superficial dermal vasodilation with significant hematic extravasation. Degeneration of elastic and collagen fibers in the dermis (Fig. 3).

Ultrasound of soft tissue: subcutaneous edema and important collection of lumps in both arms.

Laboratory: blood count with leukocytosis, neutrophilia and severe anemia (WBC 12,700/mm3 N: 80% L20%, Hb 5.9 g/dl hematocrit 20%); Normal blood pressure (PT 75% Platelets 247,000/mm3; APTT 24 seconds, fibrinogen 300mg/%), urea, creatinine, electrolytes, and normal liver function. Hypoalbuminemia (2,4 gr/L). Urine culture: E. coli..

Dermatological Diagnosis: Stage IV dermatoporosis.

Treatment: Surgical drainage of hematomas on both arms but bleeding persist in extremities. Cures with isotonic saline, vaseline petroleum jelly and gauze to cover.

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

DISCUSSION

Due to population longevity, and therefore to the existence of an older population with multiple pathologies generally considered deleterious to the healing process, and also to polypharmacy including anticoagulants, antiplatelet drugs and steroids, more than a photo prolonged exposure, dermatoporosis constitutes an entity that will increase. One of the greatest expressions of gravity, as presented by our patient, is the development of deep dissecting bruising.

In the case reported by Ramos G., et al [2], it was a 92 years old female patient with factors considered of risk for developing this condition such as been a carrier of Diabetes Mellitus, Hypertension and Chronic Respiratory Failure among others comorbidities, and therefore she had been only treated with antiplatelet agents, and inhaled corticosteroids. She had a dissected hematoma in her left leg confirmed by ultrasound, and proceeded to perform a surgical debridement and subsequent topical treatments, with progressive re-epithelialization.

In the series reported by Kaya G., et al [11], of 34 patients with dermatoporosis over a period of 7 years, the dissecting hematomas were located deep in the legs in all patients. The mean age of patients was 81.7 years and 85.3% of patients were women, being a ratio women/men of 5: 1. Among previous medications prescribed for these patients were inhaled corticosteroids (12%) and anticoagulant medication (29%) (aspirin, clopidogrel and acenocoumarol). Half of the patients was related to slow healing, diabetes mellitus, chronic venous insufficiency, arterial insufficiency and polyneuropathy processes. All patients except two the youngest, had advanced dermatoporosis, and the severe form was observed in older patients who were receiving long-term treatment with systemic corticosteroids. The patients reported a previous legs injury. Initial symptoms in all patients were pain and swelling of the leg. Erythema and edema were observed without fever. The skin necrosis developed as a late manifestation. Erysipelas was initially diagnosed in up to 14 patients, and 8 that had been treated with antibiotics before admission. MRI and histopathology confirmed deep anatomical location deep dissecting hematoma. Hospital treatment consisted mainly of deep incision and debridement followed by direct closure, skin grafts or wound healing by secondary activities. The average hospital stay was 3.5 weeks.

In the case of our patient, she is within the age group considered most affected, comprising from 70 to 90 years old; however had no history of prolonged regular intake of anticoagulants, antiplatelet or corticosteroids. However as influential factors in their photoaging are a Fitzpatrick II skin type, sun exposure without sun protection throughout their live, and limited to no care generally evidenced by a history of skin dryness and frequent itching skin. She had, bruises and scars in the 4 limbs and not only in the legs. The diagnosis is made by histopathology and soft tissue ultrasound; and as in the aforementioned publications, it proceeded to carry out surgical debridement and later topical cures.

Being an emerging, multifactorial and relatively newly diagnosed disease, handling protocols in relation to each of the stages, even drugs are not well define; however the consensus seems to be that before the existence of bruises they must be drawn on brevity.

CONCLUSION

Dermatoporosis is an emerging pathology inherent to the aging population, and deep dissecting hematoma is one of its major complications. Health professionals should know the symptoms and signs of this condition, as well as the risk factors involved, since diagnosis and treatment are important prognostic factors; and given the high cost involved, preventive measures should be applied as soon as possible.

Consent

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

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Our Dermatology Online

Case Report

Bullous reaction to a Mantoux test; a case report and review of the literature

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ABSTRACT

The tuberculin skin test is widely used in the diagnosis of latent tuberculosis infection Blistering skin lesion from the test is rarely observed. In this manuscript, I report a patient who develops a bullous lesion from the test and I review the related literature.

Key words: Mantoux test; Skin test; Tuberculosis

INTRODUCTION

The tuberculin skin test (TST), (Mantoux test), is useful in detecting populations that have been in contact with the tuberculosis bacillus. Live bacteria are not used in the test so there is no chance of developing TB from the test. However, there are few rare reactions from the test. Swelling and redness of the arm, particularly in people who have had tuberculosis (T.B) or been infected previously and in those who have previously had the BCG vaccine, can occur. Anaphylactic reaction, foreign body reaction, regional lymphangitis and adenitis have all been reported.

Likewise bullous lesion from the test is a rare event.

Herein, We report a case that develops a bullous lesion from the test.

CASE REPORT

A 38-years housemaid Filipino female develop a 3 CM blister 1 day after TST (Fig. 1). The patient was tested because, she had progressive enlargement of the left cervical lymph nodes, (Fig. 2), for the last 3 months which were thought to be due to T.B.

The patient reported that she had generalized itching with excoriations all over the body two weeks before

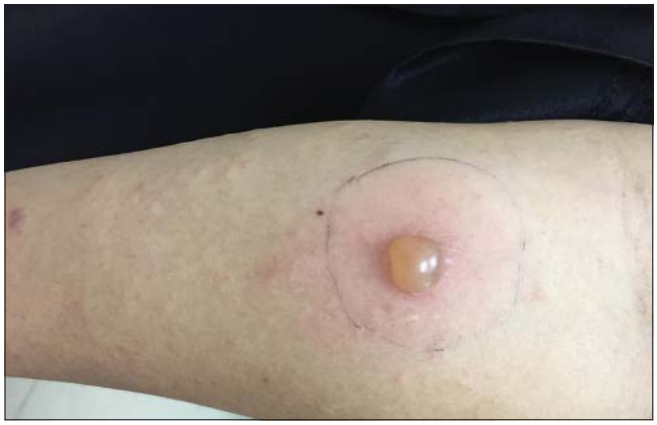

Figure 1: Large bullous lesion at the site of tuberculin test. Note that, the tiny excoriations of supposedly scabies lesions are just visible.

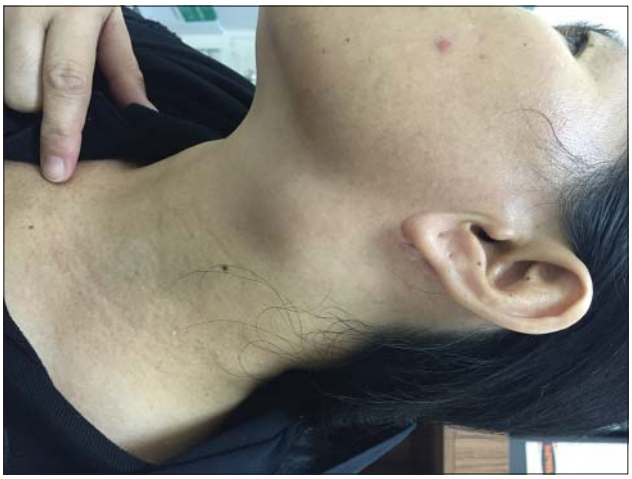

Figure 2: Enlarged cervical lymph nodes.

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the test. Her itchiness was diagnosed by a primary care physician as scabies but she does not know the names of the medications giving to her.

The tuberculin test done for her is 0.1 ml. The contents of which is shown in the Box 1. It is manufactured by BB-NCIPD Ltd., Sofia, Bulgaria.(Marketing authorization number 20000719).Batch NO 4750414. Expiray date 03.2016.

She has no history of cough, hemoptysis, or weight loss. The patient denied any family history of either tuberculosis or allergy.

General physical and systemic examinations were normal, except for single non-tender lymph nodes approximately 4x2 cm in the left side of the neck.

The generalized tiny skin excoriations were compatible with a healed scabies lesions, although skin scrapping failed to show any mites.

Erythrocyte sedimentation rate was 45 mm in the first hour.But, the hemogram, liver and kidney function tests, blood sugar and urine examination were normal. Venereal Disease Research Laboratory (VDRL) and enzyme linked immunosorbent assay (ELISA) for human immunodeficiency virus (HIV) were nonreactive. Chest radiograph did not show infiltration/adenopathy and abdominal ultrasound was normal.

A fine needle aspiration cytology of the left cervical lymph node, carried out later, confirmed evidence of granuloma and necrosis along with acid fast bacilli (AFB). She was diagnosed as tubercular lymphadenitis and started on antituberculous medications.

DISCUSSION

Intradermal tests can be done for the detection of delayed sensitivity to bacterial, fungal and viral antigens. TST is one of these test [1-12].

TST is important for the dermatologists because of an increasing incidence of tuberculosis associated with HIV infection, and also because screening is a necessary part of the work-up before use of antitumor necrosis factor biological drugs (for example, in psoriasis) [2]. Box 2, summarizes important facts about the test.

A positive test can result from clinical or latent tuberculosis infection, from BCG vaccination or from contact with environmental mycobacteria.

The results of this test must be interpreted carefully. The person's medical risk factors determine the size of induration the result is positive (5mm, 10mm, or 15mm). In Table 1, We listed some of the causes of false positive and false negative results.

Normally, a cut-off of 5mm induration is used to determine those at high risk of tuberculosis infection, for example close contacts of an active case, patients with radiographic abnormalities consistent with tuberculosis, those with HIV infection and those

Box 1: The contents of 0.1 skin test done to the patient

Tuberculin purified protein derivative (PPD) (Bioequivalent to 5 IU PPD-S)	5 TU
Tween 80 (as stabilizer)	5 microgram
Phenol (as preservative)	Less than or equal to 0.25 mg
Isotonic phosphate buffer	PH 6.5-7.5
Disodium hydrogen phosphate	0.76 mg
Potassium dihydrogen phosphate	0.145 mg
Sodium chloride	0.48 mg
Water for injection	q.s. 0.1 ml

Box 2: Some facts about TST

- It is given intradermally, on the left forearm as 0.1 ml and read after 48 to 72 hours
- Reading depends on the induration and not the erythema
- The induration is measured as (palpable raised, hardened area) across the forearm (perpendicular to the long axis) in millimeters. If there is no induration, the result should be recorded as "0 mm"
- The higher the risk a person has for developing active tuberculosis, the smaller the diameter criterion used for defining positivity in a tuberculin skin test result
- In case a second tuberculin test is necessary it should be carried out in the other arm to avoid hypersensitising the skin
- It is not specific for TB as PPD is a culture filtrate of tubercle bacilli containing over 200 antigens shared with bacille Calmette–Guérin (BCG) and many non-tuberculous mycobacterium
- For the reaction to be positive, 2 to 12 weeks need to have passed since the tuberculosis infection
- The Mantoux conversion is defined as a change (within a two-year period) of Mantoux reactivity whereas reversion is defined as the change to a negative Mantoux result following a previous positive result
- Giving a second TST after an initial negative TST reaction is called two-step testing. If the test is repeated, a larger reading may be obtained due to the immune response being 'recalled' or 'boosted' by the first test
- Boosting is maximal if the second test is placed between one and five weeks after the initial test, and it may continue to be observed for up to two years
- United States (US) recommends that tuberculin skin testing is not contraindicated for BCG - vaccinated persons, and prior BCG vaccination should not influence the interpretation of the test
- TST is not recommended in the following situations:
  Past Mantoux reactions ≥15 mm, previous TB disease, and Infants under 12 weeks old

Table 1: Causes of false results of PPD

False positive	False negative
• Non - tuberculosis mycobacterium or previous administration of BCG vaccine (BCG may result in a false - positive result for many years after vaccination) • Also when the injected area is touched, causing swelling and itching • Allergic reaction or hypersensitivity (It is advisable that epinephrine is available when giving the test)	Infectious mononucleosis, Sarcoidosis, Hodgkin's disease, Corticosteroid therapy/ Steroid use, Malnutrition, and HIV

immunosuppressed with corticosteroids or other agents. A cut-off of 10mm is used in immigrants from endemic areas, health care workers, the homeless and residents of some inner cities, and those patients with diabetes, renal disease, silicosis and other conditions associated with an increased risk of latent tuberculosis. Finally a cut-off of 15mm is used in those with no risk factors [2,11].

Because the test requires the patient to come to the hospital twice and because of its low specificity, new tests are being developed to replace TST.

The Food and drug administration FDA approved a novel diagnostic test (QuantiFERON-TB GOLD, made by Cellestis, Inc.) for TB. The blood test detects the presence of Mycobacterium tuberculosis (TB) infection by measuring interferon-gamma (IFN-G) harvested in plasma from whole blood incubated with the M. tuberculosis-specific antigens, ESAT-6 (QFT-RD1) and CFP-10 [2].

Reactions from TST are not common [5-12]. The formation of vesicles, bullae or necrosis at the test site indicates high degree of tuberculin sensitivity and thus presence of infection with tubercle bacilli.

To avoid severe skin necrosis, a tuberculin skin test should be avoided in patients with a history of severe reaction.

An exaggerated response causing giant reaction to tuberculin has been occasionally described in patients with lepromatous leprosy [11].

The case I reported has a tuberculous lympahadenitis. She developed a large bulla in just one day, which is not typical for the delayed hypersensitivity reaction seen with TST.

It is difficult to explain for sure the cause of this reaction. However, I think that her presumed scabies infestation facilitate this unusual reaction.

Eosinophils which are one of the important elements of type I hypersensitivity reaction are predominate in scabies and could have switched the reaction from Type IV to Type I or to an unusual Type IV reaction.

Scabies by itself is reported to present with bullous lesion [13].

This report may be a reminder to dermatologists to be involved actively in assessing tuberculin testing as they are the most expert physicians, in interpreting various skin changes associated with intradermal testing.

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Our Dermatology Online

Case Report

Lipedematous scalp: Case report and review of the literature

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ABSTRACT

Lipedematous scalp (LS) is a rare cutaneous disorder of unknown etiology. It is characterized by significant thickening of the subcutaneous layer of the scalp. When it is associated with hair loss it is described as lipedematous alopecia (LA) Lipedematous Alopecia (LA) is a similar disorder associated with hypotrichosis or shortening of hair. Here we report a new case of LS in a 59-year-old Arabic female who presented clinically with a diffuse spongy tender scalp.

Key words: Lipedematous scalp; Alopecia; Hair loss; Scalp

INTRODUCTION

Lipedematous scalp is a rare cutaneous condition of unknown etiology and pathogenesis. It is characterized by thickening of the subcutaneous layer of the scalp. It was first described by Cornbleet in 1935, who reported a 44-year-old black woman who presented with thickening and softening of her scalp [1]. However, the term "lipedematous scalp" was first introduced by Lee in 1994 to differentiate between this condition and others such as lipedematous alopecia [2]. Since then, only 45 cases have been reported in the medical literature.

CASE REPORT

A 59-year-old Arabic female presented to our Dermatology Clinic complaining of thickening of the scalp and headache for the past 2 years. The condition started as a gradual thickening and spongy feeling of her scalp associated with headache. The patient had no history of hair loss or head trauma. Her medical history included Hypertension, Diabetes Mellitus II, Dyslipidemia, Osteoporosis, Osteoarthritis, Cervical spondylosis and Major Depressive Disorder managed medically by the following medications: Aspirin, Pantoprazole, Insulin, Metformin, Escitalopram, and Atorvastatin. There was no history of similar condition in the family. Clinical systemic examination of the patient was unremarkable. Scalp Examination showed normal hair density with no apparent hair abnormality. Scalp skin was normal and hair pull test was negative. On palpation of the scalp it was thick, soft, had a spongy texture without fluctuation and mildly tender all over. There was no pitting of the affected area. No further abnormality was seen in skin, mucous membranes or nails. Complete Blood Count, Liver Function Test, Renal Function Test, Thyroid Function Test and Lipid Profile were within normal limits. Computerized tomography (CT) of the head showed diffuse thickening of the scalp measuring almost 14.4 mm with no brain tissue abnormality (Fig. 1). Scalp incisional biopsy showed normal Epidermis, dermal edema, elastic fibers fragmentation and markedly increased thickness of the subcutaneous fat (Fig. 2). So our final diagnosis was lipedematous scalp. The patient was reassured and a follow up visits to the clinic every 12 weeks was arranged for reevaluation since no effective treatment is available for this condition up to date.

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

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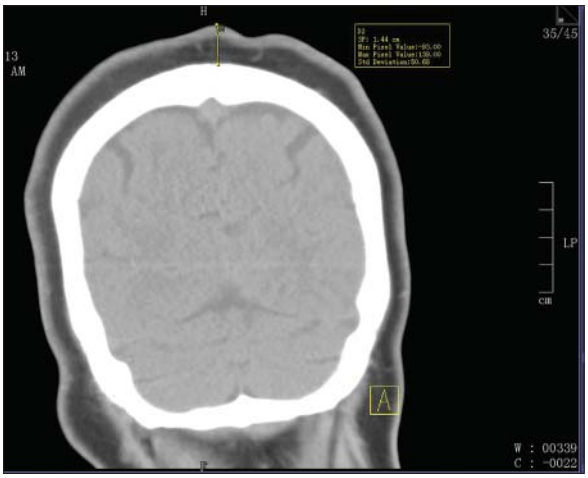

Figure 1: Computerized tomography of the head showed diffuse thickening of the scalp measuring almost 14.4 mm with no brain tissue abnormality.

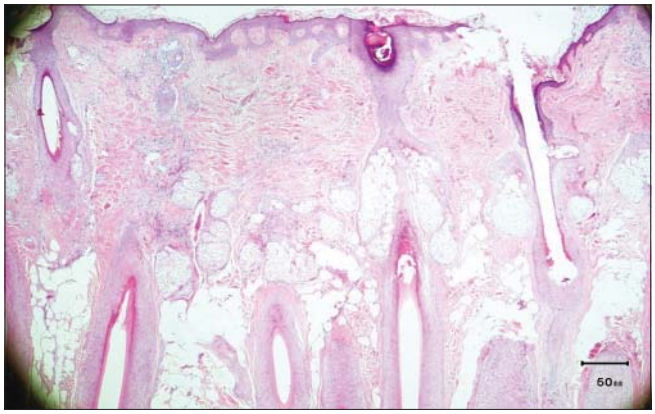

Figure 2: Section of the scalp showing markedly increased thickness of subcutaneous layer. H&E, X10.

DISCUSSION

Lipedematous Scalp is a rare condition of unknown etiology and pathogenesis. It is characterized by thickening of the subcutaneous layer of the scalp. The condition results in a soft, spongy, or doughy quality detected during palpation. When associated with hair loss, it is referred as Lipedematous alopecia. Lipedematous Scalp and Lipedematous alopecia are two entities with similar clinical morphology, imaging features and the slowly progressive course over a period of years. Lipedematous Scalp was first described by Cornbleet in 1935 who reported a 44-year-old black woman with thickening and softening of her scalp which felt like a cotton.1 While Coskey et al in 1961 reported lipedematous alopecia [3]. In 1994, Lee et al reported the second case of lipedematous scalp in a Korean woman [2]. Since then, 45 cases have been reported in the literature [4-16]. Scalp thickening is usually localized at the vertex and occipital areas and may slowly expand to the entire scalp. Patients may have associated symptoms of pain, paresthesias, headache, burning sensations, tenderness, or pruritus. The diagnosis depends upon the clinical features and pathology, which shows thickening of the subcutaneous adipose tissue of the scalp. Both Magnetic Resonance Imaging and head ultrasound scan can be useful to identify and measure the increased scalp thickeness. Different hypotheses have been mentioned in the literature concerning the pathogenesis of lipedematous scalp. There is a debate as to whether leptin plays a role in the development of hyperplasia of subcutaneous fat. This hormone is known to be involved in a long-term feed-back mechanism that regulates fat mass and distribution. Additionally, Yip et al mentioned that leptin plays a role in lipoapoptosis, and serves as a candidate hormone in the pathogenesis of LA and LS [17]. Jone et al suggested adipose metaplasia and the developmental displacement of adipose tissue or possible origin of adipocytes from the wall of dermal vessels in the course of degenerative changes in dermal connective tissue in a manner similar to hamartomatous lesions [18]. Martin et al reported that the presence of edema of the thickened adipose tissue and dilated lymphatic vessels suggest that lymphangiectasia may be responsible for hair loss because it is found only in those patients with lipedematous scalp and alopecia [19]. While some authors relate alopecia to compression of the superficial blood capillaries by the increased volume of the subcutaneous fat layer within the thickened scalp. It seems that both mechanisms play a role in hair loss associated with some cases having lipedematous scalp. Besides, headache may result from pressure on dermal nerves caused by dermal edema and thickening. To date no guidelines for the treatment of Lipedematous scalp or Lipedematous alopecia have been reported. Systemic and intralesional steroids have been utilized with no clear effect. Yip et al reported a successful treatment of Lipedematous alopecia by surgical intervention with de-bulking and scalp reduction [17].

CONCLUSION

Lipedematous scalp and lipedematous alopecia are two rare dermatological disorders with unknown etiology and pathogenesis with no consistent systemic comorbidity. Up to date, only 45 cases have been reported in the world and our case is an addition to this group of patients. Future studies are needed to reveal the pathogenesis and treatment of this rare disorder.

Consent

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

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Our Dermatology Online

Case Report

Lafora disease: A case report

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ABSTRACT

Introduction: Lafora disease is a rare and severe form of progressive myoclonus epilepsy. It is an autosomal recessive disease, genetically heterogeneous. **Aim:** Our aims is to study the clinic-pathological features of this rare entitie We report a case of Lafora disease. **Case Report:** We report the case of a 16 year old girl, which shows from the age of 14 myoclonus epilepsy. Neurological examination showed cerebellar syndrome and intellectual deterioration. Skin biopsy was needed to guide the diagnosis. The Lafora disease has a constantly fatal prognosis. Histological examination confirms the diagnosis and molecular study may help to establish a genetic counseling. **Conclusion:** Lafora disease has significant clinical and evolutionary characteristics that should guide the clinician to achieve axillary skin biopsy to find Lafora bodies.

Key words: Lafora; Epilepsy; Skin biopsy

INTRODUCTION

Lafora disease (LD) is an autosomal recessive disease characterized by a progressive myoclonus epilepsies (PME). The diagnosis is suggested by the association, in an adolescent, of epilepsy, myoclonic seizures and progressive cognitive deterioration. The electroencephalogram is characteristic and confirmation is made by histological examination [1]. The evolution is often fatal. We report the case of a 16 year old girl, who presents from the age of 14 seizures and myoclonus.

CASE REPORT

Our case is a 16 years old girl, born from a first degree consanguineous marriage with two sisters died of the same symptoms. The onset of the disease was at the age of 14 by the appearance of generalized tonic-clonic and myoclonic seizures with progressive cognitive deterioration. The skin examination was normal. Neurological examination found a cerebellar syndrome. The electroencephalogram (EEG) showed an epileptic encephalopathy. All the laboratory tests were normal. The magnetic resonance imaging showed cerebral atrophy. Histological study of skin biopsy performed at the axillary region shows the presence of eosinophils Lafora bodies in cytoplasms of epithelial cells of apocrine sweat glands (Fig. 1). These Lafora bodies were PAS positive (Fig. 2). The diagnosis of Lafora disease was made.

DISCUSSION

Lafora disease is a particularly severe form of progressive myoclonus epilepsy (EMP). It was described for the first time by White in 1988 [1]. The LD is ubiquitous, but more common in the Mediterranean region [2]. Its first manifestations occur during adolescence: generalized tonic-clonic or tonic-clonic seizure, action and rest myoclonus, negative myoclonus, but also partial occipital seizures with amaurosis [3]. The skin lesions are rare.

LD is an autosomal recessive disease. Genetic studies have shown clinical variants of the LD, and spectrum should grow gradually progresses to the elucidation of genetic mechanisms. It has been shown that mutations in Exon 1 of the gene EPM2A could produce a different phenotype with an onset in childhood and sometimes

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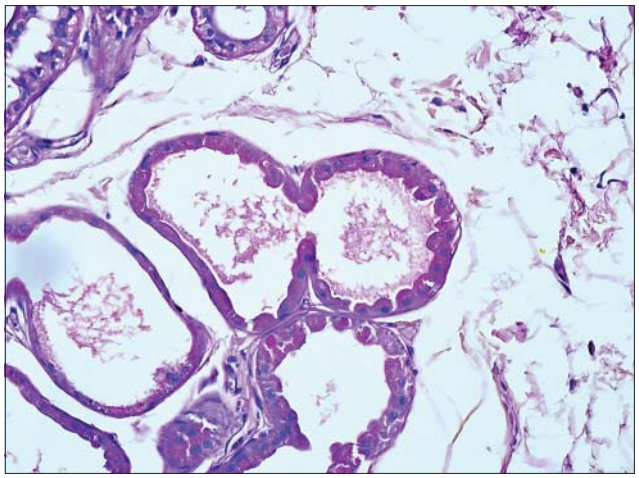

Figure 1: (HEx400) Eosinophilic bodies in the cytoplasm of epithelial cells lining the excretory ducts of apocrine sweat glands.

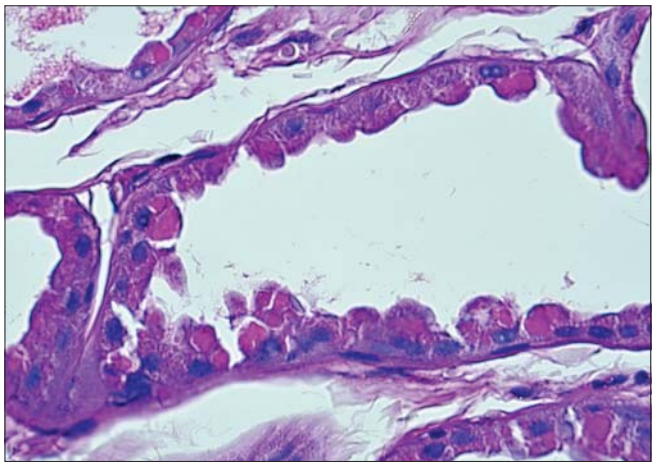

Figure 2: (HEx1000) PAS positive inclusions in the cytoplasm of epithelial cells.

learning difficulties, which are followed later by classic manifestations of the disease [4]. Similarly, EMP2B gene seems to be associated with a slightly longer duration of illness, and a little less severe evolution, independently of the type of mutation or micro deletion observed [5].

The electroencephalogram, which changes may precede the onset of symptoms, initially shows a normal background activity, sometimes slower. In half of the cases there is a diffuse activity polypoints-waves that are sporadic or bursts, spontaneous or provoked by movement or by walking, combined with a gradual slowing of the background rhythm [2]. EEG performed in our patient showed an epileptic encephalopathy. Besides the typical presentation of the disease, there are notable variations that require histological or molecular confirmation.

The role of skin biopsy in the axilla is to confirm the diagnosis of LD. It highlights the Lafora bodies or polyglucosan in the cytoplasm of epithelial cells lining the excretory ducts of apocrine sweat glands. These characteristic PAS positive inclusions are present in several organs such as the brain, heart, liver and skeletal muscle [3]. Lafora bodies are dense polyglycosans and phosphorylated, which resemble to those of normal starch body found in the brains of older people, but their location in the neuron and the dendrites is characteristic of LD. The presence of Lafora bodies in the axillary biopsy in young subjects is pathognomonic of LD. It should however be aware that these characteristic abnormalities may escape some trained eye, and a second reading or new axillary biopsy may be needed [3,6].

Differential diagnoses are discussed in terms of the evolution. At the early stage of the disease, juvenile myoclonic epilepsy or other forms must be discussed. At the status stage the main differential diagnosis is the Unverricht Lundborg disease. However, at the late phase of the LD all EMP etiologies are possible [7].

Drug resistance and psychomotor retardation are limiting factors. Death occurs 2-10 years after the onset with an epilepticus and cachexia [2,5].

CONCLUSION

Lafora disease has significant clinical and evolutionary characteristics. Resistance to antiepileptic and progressive cognitive deterioration should guide the clinician to achieve axillary skin biopsy to find Lafora bodies.

Consent

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

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Our Dermatology Online

Case Report

Linear atrophoderma of Moulin located on the face

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ABSTRACT

Linear atrophoderma of Moulin (LAM) is a rare dermatosis characterized by hyperpigmented and depressed band-like lesions localized along the Blaschko lines. LAM most commonly prefers the trunk and the limbs, while it is more rarely localized in the head and neck region. So far, any case of isolated facial lesion has not been reported. We present a-36-year old male patient with isolated facial lesion. We were observed slightly improvement in the lesion with topical calcipotriole therapy for 2 months.

Key words: Linear atrophoderma of Moulin; Blaschko lines; Topical calcipotriole.

INTRODUCTION

Linear atrophoderma of Moulin (LAM) is a dermatosis characterized by hyperpigmented and depressed band-like lesions localized along the Blaschko lines. This dermatosis was described for the first time by Moulin et al. in five patients with similar characteristics in 1992 and referred to atrophoderma of Moulin, with referrance to the first publication [1,2]. Major characteristics of LAM are being unilateral, following the Blaschko lines, long remain unchanged, onset in the childhood or adolescent period and lack of the induration. The lesions indicate progression over the first few months, then after having a linear atrophic state they stop the progression, limit themselves and become persistent [3]. Despite clinically atrophic appearance of the lesions, elastic and collagen fibers are usually normal in histopathological examination [2]. Etiology of LAM is unknown. Its localization matching the Blaschko lines is thought to be a reflection of mosaicism, which is believed to develop due to a somatic mutation occurring during early embryogenesis [4,5].

LAM most commonly prefers the trunk and the limbs, while it is more rarely localized in the head and neck region. So far, any case of isolated facial lesion has not been reported [3]. Best to our knowledge, the patient who was presented here is the first case with isolated facial lesion.

CASE REPORT

A 36 years old male patient presented with the complaints of line-form depression in the left lower half of his face and darkening of skin color in this area which has been emerged 8 months ago. In dermatologic examination, a purplish brown depressed lesion of about 10 cm length which followed the Blaschko lines was observed (Fig. 1). The patient had not any subjective complaint. There was no family history of similar lesions or skin disease. In skin biopsy from the lesion, there were thinning in the epidermis and flattening of the rete ridges with vacuolar degeneration in the basal layer. In the papillary dermis, mild edema, perivascular mononuclear inflammatory cell infiltration and diffuse melanophages were observed (Fig. 2). No change was observed in the collagen fibers with trichrome stain (Fig. 3). Elastic fibers were found to be normal with elastic staining. Antinuclear antibody, anti-histone antibody, anti-ds DNA and rheumatoid factor were negative. Erythrocyte sedimentation rate, full blood count and biochemical parameters were in the normal range. The patient was diagnosed for LAM with these findings. No any progression or regression

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was observed in the lesion during follow-up over 3 months. For treatment, topical calcipotriene cream was administered two times a day. Two months after treatment with topical calcipotriol, we were observed reduction in pigmentation and atrophic appearance with slightly improvement in the lesion (Fig. 4).

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

DISCUSSION

It has been reported about 30 LAM cases in the literature until now [6]. LAM is most commonly occurs in the trunk. In the reported cases, the most affected regions in order of frequency were the trunk, arms, legs and neck. Cervicofacial localization was reported extremely rare. There was no isolated facial involvement in the two cases which were previously reported to have facial lesions with atypical characteristics for LAM [2]. In a case report by Miteva and Obreshkova, [4] hypopigmented, atrophic and mildly slightly depressed lesions were reported to extend from the axilla to the wrist and mandible in the right arm. Multiple telangiectatic macules were reported in the same patient, which followed the Blaschko in the hip and leg, and the authors suggested that this case might be a different variant of LAM. It was reported in a LAM case by Browne and Fisher [5] that the lesions were bilaterally present in both the lower extremities, right upper extremity, right half of the trunk and right part of the chin. These findigs were not compatible with the unilateral nature of LAM [2].

Differential diagnosis includes some dermatoses following the Blaschko lines such as focal dermal hypoplasia and

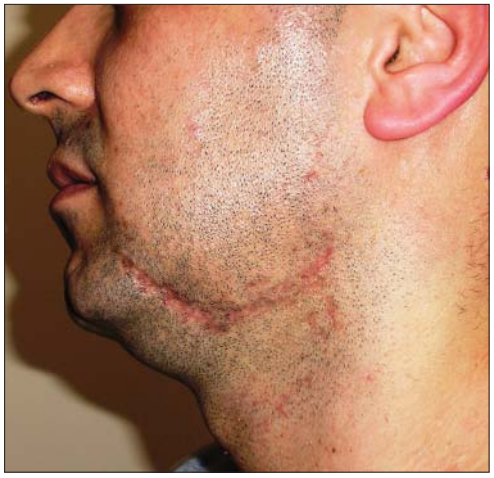

Figure 1: The purplish brown, depressed lesion which followed the Blaschko lines on the chin.

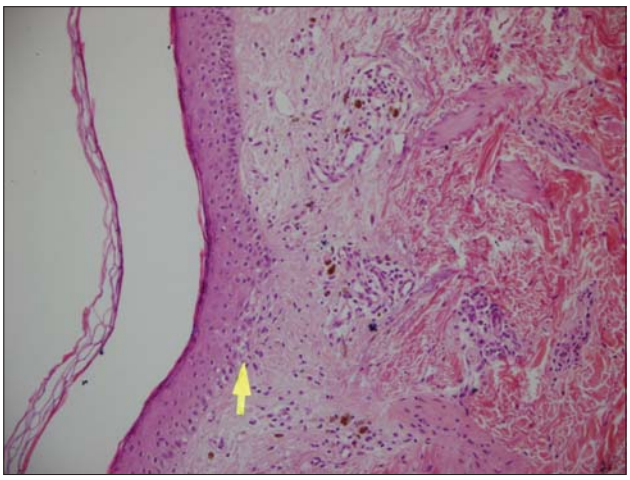

Figure 2: Thinning in the epidermis and flattening of the rete ridges with vacuolar degeneration in the basal layer. Diffuse melanophages in the papillary dermis. H&E, x200

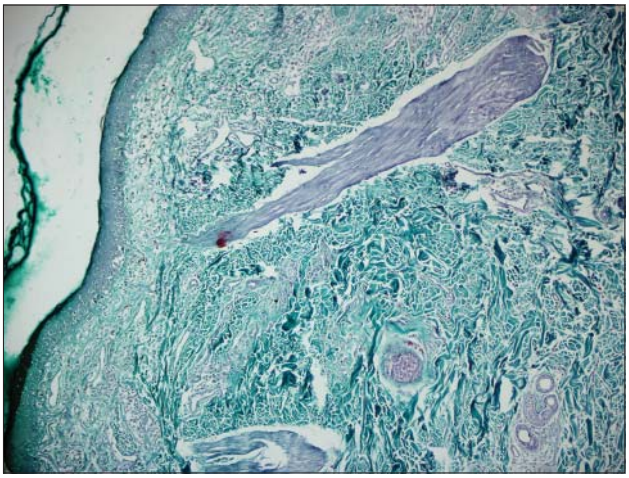

Figure 3: Trichrome stain x100.

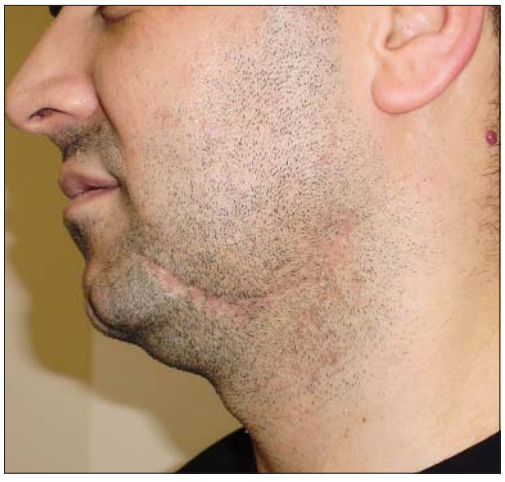

Figure 4: The appearance of lesion two months after treatment with topical calcipotriol.

incontinensia pigment as well as idiopathic atrophoderma of Pasini and Pierini (PPA) and linear morphea [2,7]. LAM and PPA are clinically similar in the presence of asymptomatic, hyperpigmented and depressed plaques and histopathologically lack of sclerosis in both diseases. However, PPA typically tends to be bilateral and simmetric in distribution and does not follow the Blaschko lines. Whereas it is not clear whether linear morphea follows the Blaschko lines or not. Nevertheless, histopathological features of LAM are different from those of morphea [8]. Also, this case was distinguished from linear atrophic lichen planus by absence of lymphohistiocytic lichenoid infiltration in dermis. Histopathological features in LAM are characterized by mild epidermal atrophy, hyperpigmented basal layer, perivascular lymphocytic infiltration and presence of melanophages in the papillary dermis. Despite atrophic appearance of lesions, elastic fibers are usually normal in histopathological examination [2,7]. The present patient was differentiated from PPA and linear morphea with the clinical and histopathological features.

Because of rarity and self-limiting feature of the disease, there is not a proven effective treatment regimen for LAM [6]. High dose penicillin, intravenous penicillin together with topical PUVA therapy, topical corticosteroids, heparin, oral potassium paraaminobenzoate have been used for LAM treatment. These treatment regimens have been found partial effective on some cases but ineffective in most cases [3,6]. Lastly Zaouak et al. [6] reported that they received good response to treatment with 20mg/week methotrexate in the case of extensive LAM. Wongkietkachorn et al. [3] reported a case of LAM with partial response to topical calcipotriol. They suggested that treatment should be started early in order to achieve good response. We also applied topical calcipotriol two times a day in our patient. Two months after treatment with topical calcipotriol, we were observed reduction in pigmentation and atrophic appearance with slightly improvement in the lesion.

Herein presented case is the first with characteristics of LAM which is only facially localized. The outcomes of previous cases and our result we have achieved in this case are important for the assignment of treatment regimens for LAM.

Consent

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

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Our Dermatology Online

Case Report

Henoch-Schönlein purpura (IgA vasculitis) developing after postoperative wound infection by methicillin-resistant Staphylococcus aureus

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ABSTRACT

Henoch-Schönlein purpura (HSP) is an acute small-vessel leukocytoclastic vasculitis, affecting the skin, joints, gastrointestinal tract and kidneys. Its prognosis depends on the severity of nephritis. A wide variety of pathogens, drugs, and other environmental exposures have been associated with HSP. Although group A β-haemolytic streptococcus has been the most studied, the majority of cases showed no direct link to streptococcal infection. Here we report a case of methicillin-resistant Staphylococcus aureus (MRSA) infection-associated HSP. A 68-year-old woman underwent a coronary artery bypass surgery. After the surgery, a postoperative chest wound was infected by MRSA and sternal osteomyelitis developed. Palpable purpura then appeared on the extremities, followed by hematuria, proteinuria and increased serum creatine. Treatments with antibiotics and debridement of the infected wound and sequestrum resulted in rapid improvement of skin symptoms. Renal function partially recovered, however mild hematuria and proteinuria remained. Published work review and the present case suggest that Staphylococcal infection-associated HSP frequently involves kidney disease and its prognosis is likely to be poor compared to a common type of HSP. Further studies are needed to establish an appropriate treatment strategy for Staphylococcal infection-associated HSP.

Key words: Chronic kidney disease; IgA vasculitis, Purpura; Staphylococcus aureus; Vancomycin

INTRODUCTION

Henoch-Schönlein purpura (HSP) (IgA vasculitis) is a systemic small vessel vasculitis associated with IgA1-dominant immune deposits, which may affect the skin, joints, gastrointestinal tract and kidneys [1]. Purpura, arthritis and abdominal pain are known to be the classical triad of HSP, however the prognosis predominantly depends on the degree of renal involvement. A variety of factors, such as infection and drugs, have been associated with the pathogenesis of HSP [1,2]. Although it is well documented that HSP occurs frequently following upper respiratory tract streptococcal infection [3], most cases have no direct link to streptococcal infection [2]. In this report, we describe a case of HSP with renal failure, associated with methicillin-resistant Staphylococcus aureus (MRSA) infection in a postoperative wound after coronary artery bypass surgery.

CASE REPORT

A 68-year-old Japanese woman with a history of hypertension and diabetes mellitus was admitted to our hospital because of unstable angina pectoris. After undergoing coronary arterial bypass grafting, a postoperative wound was infected, and sternal osteomyelitis occurred and worsened due to poor glycemic control (blood sugar level: 400-500 mg/dl). Bacteriological analysis showed methicillin-sensitive Staphylococcus aureus (MSSA) infection. In spite of intensive blood glucose control with insulin and

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intravenous administration of clindamycin, the surgical wound infection with pustular exudates and a high-grade fever continued. One month and a half after the operation, a culture of the wound was positive for MRSA. An antibiotic therapy consisting of vancomycin was initiated with poor response. A few days later, the patient developed multiple purpura on her extremities, and vancomycin was stopped because of suspected drug-induced vasculitis. When the patient presented to our clinic, multiple, slightly elevated purpura were present on the legs and forearms (Fig. 1). She did not complain of either arthralgia or abdominal pain. Periodontitis and swollen tonsils were not observed. Laboratory investigations revealed an elevated white blood cell count of 5300/mm3 (52.7% neutrophils and 1.0% eosinophils); serum creatinine, 0.73 mg/dl; C-reactive protein (CRP), 6.5 mg/dL; HbA1c, 6.9 %; and IgA of 737 mg/dL. Her hemoglobin concentration decreased to 7.9 g/dl. The levels of liver function and serum complements (CH50, C3 and C4) were normal. Anti-streptolysin O, anti-nuclear antigen, MPO- and PR3-ANCA were negative. Urinalysis showed 3+ for blood and slightly positive for protein. A skin biopsy from the leg showed leukocytoclastic vasculitis of the small vessels in the upper and middle dermis (Fig. 2). On direct immunofluorescence examination, IgA and C3 were positive in the vessel walls in the upper dermis. Based on these findings, a diagnosis of HSP was established. We decided to initiate oral administration of tranexamic acid and carbazochrome sodium sulfonate hydrate. However, there was no significant improvement in the purpura. In addition, the hematuria continued, the urinary protein excretion gradually increased (3348 mg/day at the maximum), and the serum creatinine elevated up to 2.21 mg/dl. As a result of poor control of the postoperative suppurative wound, the patient developed sternal osteomyelitis, and the wound was infected with MRSA (3+). We therefore performed debridement and sequestrectomy followed by continuous local washing therapy 2 months after the surgery. Oral linezolid was also initiated. These treatments resulted in reducing bacterial counts of MRSA and levels of CRP, as well as accelerating the wound healing. Two weeks after the debridement, the purpuric lesions on the extremities disappeared. The proteinuria began to ameliorate and serum creatinine decreased to 1.2~1.4 mg/dL 4 weeks post-debridement, however she progressed to moderate chronic kidney disease. In light of the clinical course and pathological findings, we made the diagnosis of MRSA infection-associated HSP.

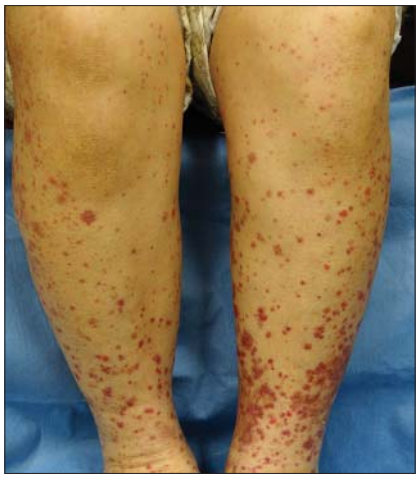

Figure 1: Clinical appearance of the eruption. Multiple, slightly elevated purpura were present on the legs.

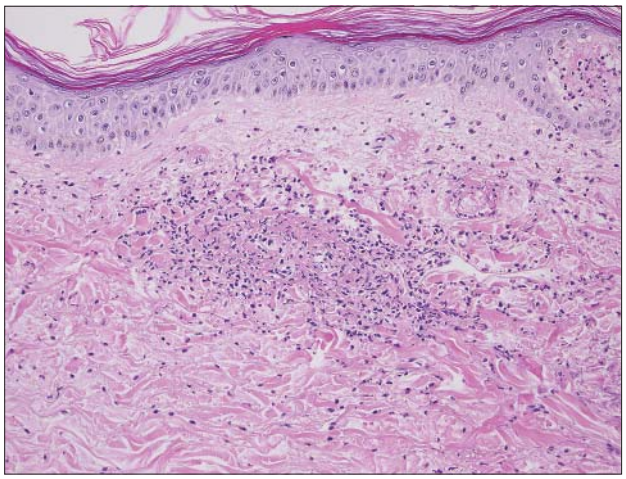

Figure 2: Histological analysis of a skin biopsy from the lesional skin. Hematoxylin-eosin staining showed vascular damage with perivascular neutrophilic infiltrate, fibrinoid change of vessel walls and nuclear debris in the upper and middle dermis, which was consistent with leukocytoclastic vasculitis.

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

DISCUSSION

Herein we presented a case of MRSA infection-induced HSP with a renal dysfunction. Debridement and antibiotics treatment rapidly improved the skin symptoms and prevented deterioration of kidney disease. To date, 22 cases of HSP associated with Staphylococcus aureus infection have been reported in 11 studies [4-14], including the present case. The mean age was 51.9 years (range, 17-90 years). Men were affected more often than women (19 men, 2 women and 1 unknown gender). Twelve cases underwent a

skin biopsy that revealed leukocytoclastic vasculitis. Eight of 9 cases, in which a direct immunofluoroscence examination was performed, showed IgA deposits in vessel walls. MRSA was detected in 15 patients, and MSSA in 7 patients from various infected sites such as skin ulcers/wounds, osteomyelitis, endocarditis, otitis media, external, cutaneous or retroperitoneal abscesses, pneumonia, and sepsis among others. One case developed HSP associated with saphenectomy wound infection after coronary bypass surgery [11]. Twenty-one cases (95%) had glomerulonephritis and/or decreased renal function. With respect to treatments, antibiotics were administered in all cases, and prednisolone and/or immunosuppressive drugs in 14 cases (64%). Renal dysfunction remained or chronic kidney disease was progressed in twelve cases (55%). In two cases, the patients died due to hepatic failure or sepsis [12].

Although common HSP in adulthood presents a variable frequency of renal involvement (40-85%) [1,2,4], the final outcome is good (end-stage kidney disease in 20-30% in long term follow-up) [4]. On the other hand, the renal involvement in Staphylococcal infection-associated HSP was frequent and its renal outcome was poor. In fact, our published work review indicates that 14 (64%) of the 22 Staphylococcal infection-associated HSP cases resulted in chronic kidney disease or death. Therefore, it is important to differentiate patients with common HSP from patients with Staphylococcal infection-associated HSP. Hirayama et al. [12,13] proposed that Staphylococcus aureus enterotoxins may act as superantigens, which could induce strong activation of T cells and cytokine release, and contribute to the pathogenesis of rash and systemic vasculitis. Thus, proper antimicrobial therapy is undoubtedly essential, but resolution of infection does not necessarily result in resolution of skin eruption and nephritis. Some case reports described patients with Staphylococcal infection-associated HSP who were resistant to antibiotics and improved by administration of steroids [8,10]. In contrast to such cases, Fujiwara et al. [7] reported a case where the patient was unresponsive to steroid therapy and improved by antibiotics administration. At present, steroid therapy may exacerbate infection and its therapeutic benefit is controversial.

In conclusion, we report a case with MRSA infection-associated HSP that occurred after coronary artery bypass surgery. By the combination therapy of antibiotics and debridement of the infected wound and sequestrum of the chest, the purpura on the extremities rapidly disappeared but moderate chronic kidney disease remained. In this rare type of HSP, renal involvement frequently occurs and its prognosis is poor compared to a common type of HSP. Further studies are needed to establish an appropriate treatment strategy in Staphylococcal infection-associated HSP.

Consent

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

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Our Dermatology Online

Case Report

Hutchinson-Gilford progeria syndrome: a rare case report

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ABSTRACT

Progeroid syndromes are characterised by clinical features of physiological aging at an early age. Hutchinson-Gilford progeria syndrome is a type of progeroid syndrome, characterised by abnormal facies, bone abnormalities, sclerodermatous skin changes and retarded physical development. Average life expectancy of progeria patients is 13 years. Herein we are reporting a case of progeria who is 21 years old.

Key words: Hutchinson-Gilford progeria syndrome; Short stature, Scleroderma

Introduction

Huthinson-Gilford progeria syndrome is a rare hereditary disorder caused by mutations in lamin A gene which encodes proteins of nuclear lamina, results in production of a truncated protein called progerin [1]. This disorder was first reported by Hutchinson in the year 1886 [2]. Gilford introduced the term progeria. This syndrome along with Werner syndrome are also known as segmental aging syndromes, as they do not feature all aspects associated to physiological aging. Till now less than 100 cases of progeria have been reported in world literature [3].

Case report

A 21 year old male presented with pigmentation of face with itching as major complaint since 6 months. No history of photosensitivity. There was history of delayed physical development and dentition noted at 2yrs of age with no history of convulsions. Intelligence was normal. On examination he had typical bird like facies characterised by large cranium with frontal bossing, prominent eyes, beaked nose, prominent ears, sunken cheeks, micrognathia and mottled pigmentation (Fig. 1). In our patient, unlike progeria there was no loss of eyebrows and scalp hair.

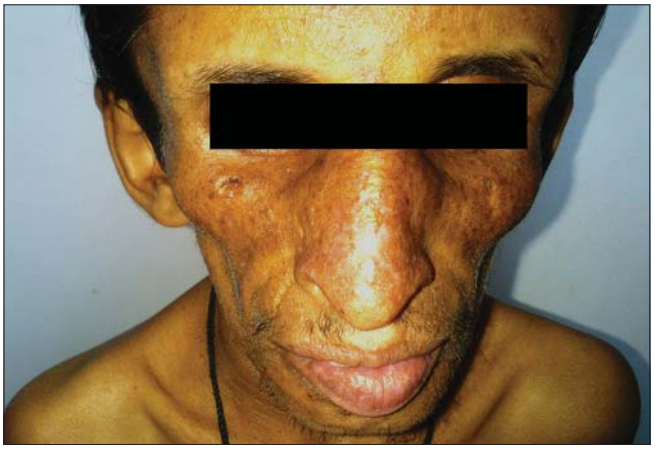

Figure 1: Typical bird like facies with frontal bossing, prominent eyes,& micrognathia.

Detailed examination revealed short stature with height measuring 94 cm (Fig. 2). Nails were thin and brittle. Sclerodermatous thickening of skin noted over the dorsa of hands and fingers. Prominent joints, loss of subcutaneous fat and muscle atrophy were other important features noted. Ocular examination was within normal limit. Systemic examination were within normal limits. Radiological examination of skull, chest and extremities were within normal limits. Electrocardiography showed features of left ventricular failure.

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Figure 2: Short stature of the patient.

Discussion

Hutchinson-Gilford progeria syndrome(HGPS) is a rare disorder, has an incidence of 1 in 4 to 8 billion newborns [4]. It occurs due to sporadic mutation, hence not run in families. Characterised by accelerated aging, short stature, alopecia, generalised atrophy of skin and muscles with sclerodermatous changes over the extremities. The facial appearance is reminiscent of a fledgling bird, with a disproportionately large cranium with patent fontanelles and frontal bossing, prominent eyes and scalp veins, very sparse, downy scalp hair, sparse or absent eyebrows and eyelashes, centrofacial cyanosis, micrognathia, thin lips and a 'beaked' nose [5,6]. Bird like facies is not characteristic of HGPS, also seen in association with Hallermann-Streiff syndrome, familial partial lipodystrophy, Nijmegan breakage syndrome [7].

Progressive mottled hyperpigmentation of skin is noted over exposed parts with aging, but there is no photosensitivity. Abnormal and delayed dentition is noted and associated skeletal abnormalities such as dystrophic clavicles and coxa valga, with joint contractures and a 'horse-riding' stance also observed.

It has to be differentiated from other aging syndrome like Werner's syndrome (adult progeria) which usually presents between 14 and 18 years of age with short stature, immature sexual development, cataract, glaucoma, and sclerodermatous changes, Cockyane's syndrome manifested as facial erythema in butterfly distribution, photosensitivity, ocular defects, and a "micky mouse" appearance with disproportionately large hands and feet and protruding ears, Rothmund-thomson syndrome appears between 3 and 6 months of age with poikilodermatous skin, photosensitivity, and cataract.

Our patient had retarded growth and accelerated aging since 2yrs of age, with typical facial features except for presence of eyebrows and scalp hair, short stature, sclerodermatous changes in skin, loss of subcutaneous fat and atrophy of skin and muscles, radiological features of osteopenia with features of left ventricular failure in electrocardiogram.

Complications include atherosclerosis, angina attacks, cerebrovascular accidents, osteoporosis, pathological fractures and dental anomalies.

Treatment is mainly symptomatic. Long term follow up of patient is required to observe skeletal and cardiovascular changes. Main aim of treatment is to control diabetes, osteoporosis and atherosclerotic changes. Low dose aspirin is used prophylactically to control ongoing atherosclerosis. Pravastatin and bisphosphonates like zoledronic acid to prevent atherosclerosis and osteoporosis is under trial. A phase II trial of lonafarnib, a farnesyl transferase inhibitor, in progeria is currently ongoing in USA [4].

Conclusion

Hutchinson-Gilford progeria syndrome is very rare disorder, less than 100 cases have been reported worldwide. Only few cases have been reported to survive after second decade. Because of its rarity, lack of reporting and long term follow up prompted us to report this rare syndrome.

Consent

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

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Our Dermatology Online

Review Article

The most common childhood skin diseases

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ABSTRACT

The most common childhood skin disorders are usually diagnosed by district nurses, health visitors, panel pediatricians or school doctors. Dermatological or specifically pediatric dermatological examinations are seldom necessary. This article reviews the most common childhood skin disorders and the directives of their treatments.

Key words: Childhood skin disease; Bacterial skin disease; Fungal skin disease; Viral skin disease

HISTORICAL OVERVIEW

It was at the beginning of the 19th century that dermatology separated from internal medicine. The pioneers of dermatology used clinical and histological images to describe symptoms. Later, in the etiological era, the pathogens of certain diseases were demonstrated. This was followed by the exploration of the diseases of the skin and the internal organs.

As it is understood today, several external and internal factors are required for the development of skin disease. The human body should be considered not only as a unit, controlled and coordinated by the hormones of the cerebral cortex, but it is also in close connection with the environment. That is to say, the fact that whether a person is healthy or ill, results from the dynamic relationship between the external and the internal environment.

In the last decades, the discoveries of molecular biology have had a great influence on the development of medicine. Modern dermatology studies abnormal functions behind abnormal morphological lesions, but it also takes into consideration environmental influences as well as changes in the inner organs [1].

A common field for dermatology and paediatry is child dermatology. The knowledge of both professional fields is an essential criterion for successful therapy and prevention.

BACTERIAL SKIN DISEASES

Micro-organisms get onto the infant's skin while traversing the birth canal. The characteristic bacterial flora develops in the first two weeks after birth, the majority being Gram-positive bacteria. Due to their need for a high degree of moisture, Gram-negative bacteria are mostly found in the folds [2].

Impetigo Contagiosa

Impetigo contagiosa is an extremely contagious, superficial skin infection. It appears mostly on young children. According to the statistical data of the Dermatology Department of Heim Pál Children's Hospital Budapest, more than 70% of the cases are caused by aureus, in 20-25% of the cases we can talk about a blended infection (Staphylo- and Streptococcus), and only 5-10% of the cases have Streptococcus as the pathogen [1].

Clinical symptoms: Small, red maculae appear mainly on the face or on the limbs, with vesicles and blisters, which break almost immediately. The serous discharge, drying on the oozing blister base and on the surrounding skin, will generate characteristic honey-yellow slough [3]. Sloughing spreads quickly, not only on the edges, but also to further areas of the body, by autoinoculation, or it may be transmitted by tableware or clothing. The type with an epidemic-like occurrence on young babies is the bullous form, starting

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mainly from around the body orifices [4]. In the case of older children, the appearing symptoms are mostly accompanied by thick sloughing, in most cases on the lower limbs, where there is an injury or bites that have been scratched open.

Treatment: generally topical. Systemic antibiotics are required only in case the infection is extended or reoccurring. The topical treatment is applied in three phases:

a). The removal of sloughs: By means of a neomagnol compress, or antiseptic cream (ung. Antisepticum Fo.No.)
b). We should apply antiseptic paint which does not sting (sol. Merbromini 2%)
c). Cream or ointment again (ung. Prednisolon J., ung. Antisepticum)

Nephritis may develop as a complication of impetigo caused by extended Streptococcus, therefore control (urine test) is recommended for 6 weeks.

Folliculitis

Folliculitis is a pustule, 1-2 millimetres in diameter, surrounded by a red halo of a few millimetres, and with a hair in the centre. In childhood, it appears mostly on the head, the gluteus, or on the limbs. Its larger and deeper type is the boil, developing mostly on adolescents (Fig. 1). It is generally caused by Staphylococcus [5].

Treatment: having removed the hair, apply antiseptic paint, and poultice in the case of a boil. Sometimes surgical intervention or systemic antibiotic treatment is needed.

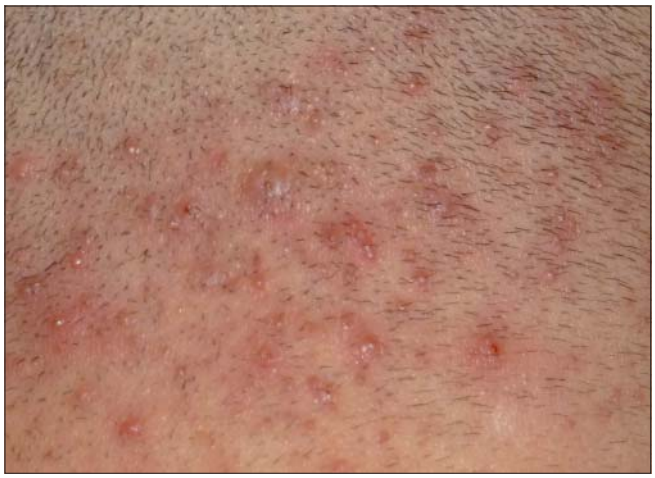

Figure 1: Folliculitis

Ecthyma

Ecthyma is a painful ulcer, 0.5-1 centimetre in diameter, and covered with deep, thick slough, which develops mainly on the legs and gluteus after a trauma or when insect bites have been scratched open. It is caused by Streptococcus heamolyticus [6].

Treatment: systemic antibiotics, topically the same as with the above mentioned [7].

Paronychia

Paronychia is an acute and painful inflammation of the nail-bed and the nail fold. Pressing the nail-bed will drain pus. It is caused by Streptococcus aureus; it develops in areas of thumb-sucking, injury, or ingrown nails on the feet [8].

Treatment: antiseptic soaking, topical or if needed, systemic antibiotics, or possibly surgical opening [9].

Erysipelas

Appearing mainly on the head of infants and babies, erysipelas is superficial dermatitis. The skin is shiny, red, warm to the touch and painful. The erythema has an irregular shape, but it has sharp edges and spreads like tongues of flame. Its pathogen is generally ß-Streptococcus, less frequently Staphylococcus. It gets into the skin through epithelial injuries, and spreads through the lymphatic vessels. It is accompanied by general symptoms, shivering, fever, and malaise.

Treatment: systemic Penicillin or Erythromycin [10]. Topically it is treated with cold packs (preceded by protection with ointment. eg. with Ung. Burow).

Erythrasma

The occurrence of erythrasma is less frequent with young children, and more common with adolescents. Slightly scaly russet colour maculae with sharp edges and a few centimetres in size develop in the axilla or in the folds of the groin. It does not cause subjective symptoms. Pathogen: Corynebacterium minutissimum [11].

Treatment: Erythromycin in full dose [10].

FUNGAL SKIN DISEASES

Fungi are less pathogenic than bacteria or viruses but due to antibiotic and steroid treatments they

increasingly come to the fore. Childhood fungal infections are mostly caused by dermatophytons and proliferous fungi [12]. They can sicken the skin, the hair, the nails as well as the mycoderm.

Tinea Capitis

Tinea capitis is caused by threadlike fungi. The two most frequent types are trichophytia and microsporia, affecting mostly children between 2-5 years of age. One or more plaques with alopecia (bald patches) of a few centimetres are formed. The phenomena may be inflamed or inflammation-free. Raised from the surface of the skin and localized on the hair follicles, confluent and purulent nodes and sloughs are formed. Microsporia causes mildly scaling plaques. The hair shafts break at 2-3 millimetres [13]. The hair stubs are surrounded by a white, powdery capsule and the skin of the scalp looks as if it has been dusted with flour.

Pathogens: dermatophytons [13], which live in the earth, on animals, or on human skin. In recent times, the most frequently occurring infection has been microsporum canis, spreading from animals to children. The pathogen may be carried by symptom-free animals (dogs, cats, guinea pigs and hamsters) as well [14].

Treatment: systemic antimycoticum (Lamisil, Orungal) [15].

Tinea Corporis et Faciei

One or several round lesions, 1-2 centimetres in diameter, appear on the face, neck, chest or arms. The scaly lesions have sharp margins and are surrounded on the edges by vesicles or papules, occasionally forming unbroken lobate areas. They itch, which results in excoriations and sloughs on the skin. The lesions on the face are less characteristic, and are difficult to diagnose [16].

Tinea Inguinalis

Tinea inguinalis affects mainly boys, especially in hot weather. Increased sudation, wearing tight clothing, underwear rubbing the skin, are all predisposing factors. The characteristic fungal symptoms manifest in the folds of the groin.

Tinea Pedis

a). Erosion interdigital: the skin between the toes is macerated, it cracks and itches
b). Hyperkeratotic type: the skin of the sole thickens, scales, and it is slightly erythemic
c). Pustular type: scaling, inflamed plaques, bordered by small pustules, develop on the sole
d). Dyshidrotic type: small, deep-set vesicles develop in the instep, accompanied by oozing and scaling. If it is symmetrical, detected on both soles, it is a secondary symptom, a so-called id reaction. In this case, it is not a case of infection, but a lesion, attached to a primary centre and developing because of an allergy [1].

Tineas are generally treated with disinfectant paint and antifungal ointments (sol. Merbromini 2%, ung. Nizoral). In the case of tinea on the hairy scalp, systematic treatment is necessary [16].

Pityriasis Versicolor

Young children are rarely affected, it appears quite often in puberty. The pathogen lives on the hairy scalp, and there it does not cause any symptoms. After getting on the skin of the trunk, it induces light brown maculae, 0.5-1 centimetre in size, mainly on the shoulders [17]. The maculae occasionally blend together, forming a map-like image, and turning white after sunbathing (Fig. 2).

Treatment: Nizoral shampoo [18], antifungal paint, and cream.

Candidiasis

Candidiasis is caused by candida albicans [19], a facultative parasite of the gastrointestinal tract. Infancy, old age, a tumour, leukaemia, and metabolic diseases

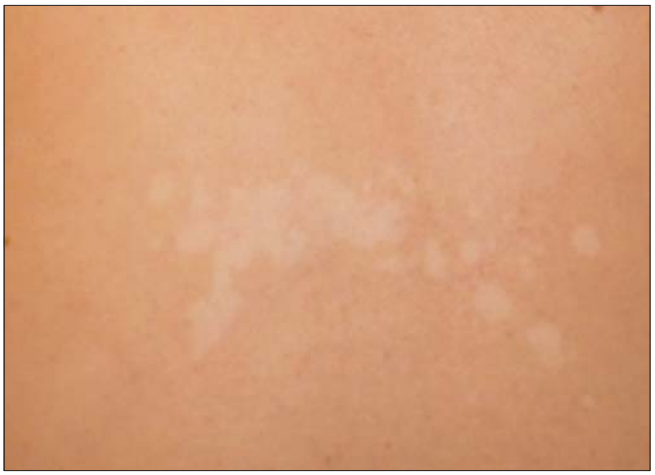

Figure 2: Pityriasis versicolor

are predisposing factors. Candidiasis of the oral mucosa of infants (soor oris) is a frequent phenomenon, since normal bacterial flora has not developed yet. White accretions develop on the oral mucosa, a few millimetres in size and occasionally confluent, which are indelible [1].

Treatment: sol. Canesten, Borax-glicerin solution Fo.No., Nystatin [19].

Angulus Infectiosus

Angulus infectiosus is caused by candida albicans [20]. Inflammation, scaling, or cracks emerge in one or both labial commissures [21].

Treatment: ung. Borosalicylatum Fo.No., Nizoral.

Diaper Dermatitis

Diaper dermatitis is a frequent, polietiological skin disease. It is manifested as tiny pustules and plaques with scaly edges, on the areas of infants' skin, covered with the diaper. Erosions and oozing can be detected in the folds. Apart from the pathogen, candida albicans, getting onto the skin from faeces, a role is played by the occlusive effect of urine and the diapers [22, 23], or if textile diapers are used, the residues of the detergent and the fabric conditioner.

Treatment: see candidiasis. Prevention is of utmost importance, as well as the application of the right skin cleansing oils, emulsions, and adherent protective baby bottom ointment [23].

VIRAL SKIN DISEASES

Herpes Simplex

The primary infection mostly runs its course symptom free. The viruses remain in the spinal ganglia and get onto the skin from there [24].

Herpes simplex recidivans

As a result of fever, trauma, stomach or bowel diseases, herpes simplex recidivans develops around the mouth of children, who are already infected by the virus. Following an itchy, stinging sensation, groups of vesicles [25] are formed on an erythemic basis, which will open and slough in a few days and heal without leaving a trace. There are no accompanying general symptoms, however, painful regional lymph-node swelling may develop even before the appearance of the skin symptoms.

Treatment: having opened the vesicles, apply antiseptic epithelizing paint, or possibly paste (zink-sulfate solution, zink paste) Hevizos cream [1].

Aphthosis

Aphtosis is one or several painful erosions on the oral mucosa, a few millimetres in size, encircled by a red 'halo', and covered with yellow fur. It heals without scars in 7-10 days [26]. It is probably caused by a virus, but an autoimmune origin might be a possibility as well.

Treatment: antiseptic and epithelizing paint.

Diseases Caused by the Verruca Virus

One of the most common skin lesions. A genetic predisposition and reduced cellular immunity result in susceptibility for infection. It is not a severe condition, however, in case of a multiplex occurrence, it is extremely uncomfortable. The incubation period may last as long as six months.

Verruca vulgaris (common wart)

A brownish-grey epitheliolid papula, a few millimetres-1 centimetre in diameter, and with a hyperkeratotic surface [27]. It appears most frequently on the hands, less frequently on the knees, while in the case of nail-biting children, under the nails or around the nail-bed. Filiform increments on the face and neck, flat, corn-like lesions on the soles, which may blend in a larger area like a mosaic (Fig. 3). There is a high rate of reoccurrence, and occasionally there is spontaneous healing. It is extremely infectious.

Treatment: the wart may be removed through peeling by layers with keratolytic paint (Verrumal), or through

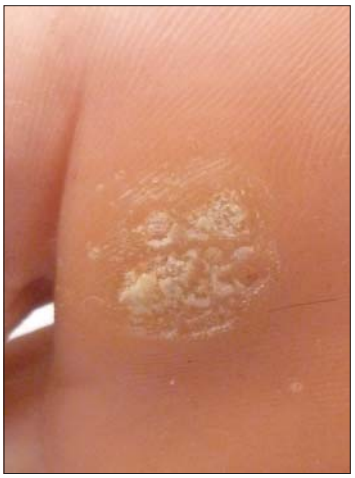

Figure 3: Verruca vulgaris

electrocoagulation under local anaesthesia, or possibly cryotherapy [27,28].

Verruca plana juvenilis (flat warts, plantar warts)
Flat, cuticolour, generally multiplex epitheliolid papules on the face or on the back of the hand [29], 1-2 millimetres in diameter. Their localization is linear, appearing along a wound or scratching.

Treatment: keratolytic paint with a milder effect (Egaverr).

Condyloma accuminatum
Small increments, developing on the genitalia or around the anus [30], effected by moisture and rubbing. It occurs on verruca-infected children and infants .

Treatment: paint with 10% Podophyllin [31].

Molluscum contagiosum ('swimming-pool' warts)
The incubation period may last several months. The semispherical warts are generally small, 1-3 millimetres in diameter [32] and have a flat surface, with a navel-like retraction in the middle and a narrow opening (Fig. 4). When pressed, gritty matter passes. It is extremely infectious. Pearl-like small nodules appear along scratching lines. Warts sometimes heal spontaneously.

Treatment: the warts can be removed with a Volkmann spoon or tweezers, or they may be exfoliated with iodized paint.

CONCLUSION

The majority of childhood skin disorders are of infectious origin. They are caused mainly by bacteria, fungus and viruses. The authors of this article summarized the characteristics of these diseases trusting that reviewing them will lead to early diagnosis, which coupled by adequate treatments speeds up the remission of the affected children.

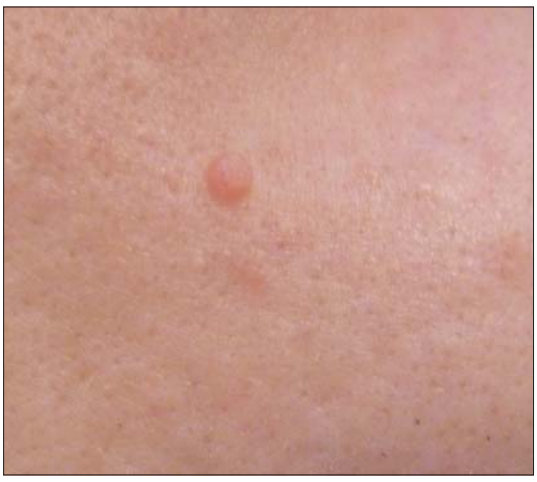

Figure 4: Molluscum contagiosum

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Our Dermatology Online

Review Article

The potential role of cell phones in dissemination of bacteria in a healthcare setting

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ABSTRACT

The mobile phone is an indispensible accessory, both from a social and a professional point of view, which is being used in a variety of environments, including those with high bacteria levels. Medical personnel, patients or even people simply passing by the healthcare facilities, having and using their mobile phones are likely to transmit bacteria, thus it might be suspected that mobile phones are to some extent a link in the cross-contamination. The purpose of this work was to collect information concerning the potential role of cell phones in a dissemination of bacteria based on available research data.

Key words: Cross-contamination; Mobile phone; Bacteria; Microorganisms; Healthcare personnel

INTRODUCTION

Mobile phones are essential accessories that are being used in everyday life, both in its professional and private capacities. These devices are usually stored in handbags and/or in the pockets of their owners' clothing, therefore they are being touched by hands and come in close contact with human skin, not to mention that they are being placed on numerous surfaces countless number of time each and every day what causes the microorganisms to migrate from any other surface that the phone had contact with to a phone itself [1,2]. The average user of a cell phone touches its screen around one hundred and fifty times a day [3] causing the migration of the bacteria from the wireless phone to the skin and vice versa [4,5]. Studies conducted by Grice et al. [6] and Griece and Serge [7] showed that the human microbiota can show interpersonal dissimilarity and that the same rule applies to the microorganisms that can be found on the human skin. Furthermore, Meadow, Altrichter, Green [8] stated in their research that in twenty-two percent of cases studied, the microorganisms that were originally present on the hands of the owners of cell phones, have also been present on the surface of their mobile phones.

Places, which are expected to be contaminated by bacteria in a higher capacity, especially those of a public utility nature like: train stations, airports, shopping malls, schools and also the health care facilities- including the hospitals and dental clinics, are more likely to be involved in the transfer of the bacteria to the other locations by means of cell phones as carriers. Therefore, it can be presumed that the usage of mobile phones in hospitals, both by the patients, healthcare employees and people- including the visitors simply passing by, could potentially cause cross-contamination.

The increasing role of cell phones

In the past few years, the cell phones gradually became more and more involved in our daily life, including

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its private and work-related capacities, acting not only as a primary communication tool, but also being involved in data research and storage and much more beyond that. Therefore, as the significance of wireless electronic devices increased, so did the interest in the probable side effects of their usage. Undoubtedly, a potential role of cell phones as carriers of pathogenic bacteria from one environment to another, or even from one surface to another, can be an example of such side effects. One of many attempts to investigate the correlation between the presence of the bacteria in the hospital environment and on the surface of the cell phone owned by medical personnel in one of the hospitals in Turkey has been conducted in 2007 by Karabay, Kocoglu, Tahtaci [9]. The studies aiming to explore the similar topic have also been conducted in the other parts of the world, since the cell phones became more and more affordable commodities, thus the group that has been researched increased to the point that it can be assumed that nowadays there are almost as many cell phones as there are people on Earth [10]. Based on the available data – the studies conducted by different authors all over the world in a last few years, the attempt has been made to review some of those findings, thus present the current state of knowledge on this particular matter.

OBSERVATIONS

Among the available data regarding the possible role of mobile phones in the spread of bacteria in the healthcare settings, many different approaches to the issue in question might be found. Some of the researchers have been focused on the determination of the rate of contamination, whereas others tried to assess the number of healthcare employees involved in the study who indeed disinfected their mobile phone routinely. Yet another authors aimed to foreseen the impact of the disinfection proceedings in the long run, while others seek to compare the contamination rate between the mobile phones and landline phones, or smart cell phones and non-smart cell phones. Nevertheless, each and every method gave an insight into the matter of the potential role of cell phones in the dissemination of bacteria, yet from different perspectives.

In New York City, NY, USA, Goldblatt et al. [11], in 2007, determined that in about twenty percent of cell phones owned by medical personnel who participated in the research, microorganisms have been present on the surface of these wireless devices. Akinyemi et al. [1] on the other hand, however, stated that based on their research it can be concluded that bacteria are present on the surface of phones in more than half of the cases – in 62% to be exact. In one of the studies conducted in Australia, among 226 wireless phones that belonged to the staff members at one of the local hospitals, in 168 devices that have been screened, the bacteria presence had indeed been discovered. The latter research also noted that the majority of the organisms that have been isolated could be described as a normal flora that can be found on the human skin, whereas only in twelve cases out of 226, the discovered microorganisms could be defined as potentially pathogenic [12].

The issue in question – the potential role of cell phones in dissemination of bacteria has been approached from slightly different angle when in 2014 Vinod Kumar et al. [13] investigated the presence of the antibiotic resistant bacteria on the surface of mobile phones owned by the patients in one of the healthcare facilities in South Arabia. According to their research, 89 out of 106 cell phones have been contaminated with bacteria and the most commonly found type of bacteria was coagulase- negative Staphylococcus that has been present on the surface of 52 mobile phones that have been sampled [13].

Another example of study conducted on the potential role of cell phones in dissemination of bacteria is the research conducted by Jeske et al. [4]. In this study aimed to explore whether, or not, in the exact same conditions and following the same procedure, the surface of the mobile phones and landline phones located in the operating room will be contaminated by bacteria found on physicians' hands in the similar manner. As it turned out, in case of cell phones in 38 out of 40 cases, the bacterial contamination have been found, whereas in four out of 40 instances the human pathogen bacteria have been isolated. For the landline phones, the numbers have been 33 out of 40 and four out of 40 respectively. The authors of this research also noted that the use of cell phones in the operating room could theoretically have more severe hygiene significances since the wireless devices often come in closer contact with the patient than then fixed ones. Borer et al. [5] drew the similar assumptions stating that the stationary phones can potentially be involved in the spread of bacteria, just like wireless phones possibly can, but the latter may prove particularly problematic as they may facilitate the transmission of pathogens on the larger scale.

Yet another attempt to research the potential role of cell phones in the transition of pathogens has been made when Lee et al. [14] studied the correlation between the contamination rate in the case of smart cell phones and non-smart cell phones owned by the healthcare employees. According to the obtained data, the bacteria of a possible pathogenic nature have been isolated in 34.8% of sampled smart mobile phones, while in the case of non-smart phones this number was 20.5%.

Shakir et al. [15] documented the rate of bacterial contamination of the mobile phones owned by surgeons in a long run – i.e. at the initial sampling, after the disinfection of the wireless phone and after one week of the original testing. The obtained results showed the significant decrease in the rate of bacterial contamination after the disinfection proceedings have been introduced – from initial 83% to 8%, but in following days the recontamination arose – resulting in 75% of sampled cell phones to be contaminated by potentially pathogenic bacteria. In 2012, Brady et al. [16] tested the contamination of the surface of the mobile phones twelve hours after the disinfection proceedings using the 70% isopropyl alcohol, which had been introduced. According to the obtained data, only 16% of the sampled electronic devices did indeed contain the bacteria, bearing in mind that initial rate of contamination was 55%. In other words, the authors noted the significant decrease of 79% in the contamination rate after a single disinfection.

Without any doubt, a lot of other studies have been conducted aiming to investigate the contamination rate of cell phones owned by medical personal, which have not been mentioned in this paper. The emphasis, in this paper, has been put on showing the results published by authors investigating the issue in different countries. Those results have been summarized in the table below (Table 1).

Table 1: The percentage contamination rate of cell phones

Country	Authors of the study	Contamination rate of cell phones (no disinfection proceedings have been introduced) (%)
USA	Goldblatt JG, et al. [2007]	20
Nigeria	Akinyemi K, et al. [2009]	62
Australia	Chao Foong Y, et al. [2015]	74
Saudi Arabia	Vinod Kumar B, et al. [2014]	84
Austria	Jeske H.C, et al. [2007]	95
UK	Brady RR, et al. [2012]	55

Up to this day, many studies have been conducted which concluded that there indeed is a correlation between the bacteria presence on the skin of the owner of the cell phone and the wireless device itself [17-22]. Walia et al. [23] even suggested that the cell phones might, theoretically, act as 'Trojan horses' triggering the development of the diseases caused by pathogens that are usually present mostly in hospitals and in the dental clinics. On the other hand, however, the conclusions drawn by Tacconeslli [24] seem to contradict the thesis about cell phones and their role as the 'Trojan horses', since, according to Tacconeslli, there is no direct correlation between the presence of pathogens on the mobile phones and the frequency of the diseases that are primarily caused by pathogens present mostly at the health care facilities. Furthermore, we lack an unambiguous data stating that cell phones are indeed more likely to be involved in the spread of bacteria form one place to another than any other mobile devices or personal items. Additionally, Karabay, Kocoglu, Tahtaci [9] noted that pathogens involved in development of hospital infection could potentially spread through medical instruments like stethoscopes or personal items-toys in pediatric care units can serve as an example [25], and even by means of the hands of the healthcare personnel.

Still, though, it should be pointed out that according to Datta, Rani, Chander, Gupta [21] in a hospital in India, where they have carried out their research, there were no general guidelines on the way in which the employees have to take care of their phones while at work, nor has it been pointed out where the phones can be used and in which areas it is strictly prohibited. Such lack of basic information and guidelines may increase the probability of cross-contamination and so, the simple methods as disinfection of the cell phones or the restriction of their usage might, at least theoretically, lower the risk of the spread of bacteria. Likewise, Karabay, Kocoglu, Tahtaci [9] drew the similar conclusions and also they pointed out that the restrictions on the use of cell phones in the health care institutions by medical personnel are impractical since those mobile devices can be considered as essential instruments for healthcare workers, therefore the emphasis should be put on the prevention of the spread of bacteria through mobile phones by, for instance, means of proper hand hygiene and disinfection of mobile phones. Borer et al. [5] suggests that strategies that target the behavioral regulation of medical personnel should be applied – like the enforcement of the infection prevention methods, as well as the disinfection methods of the mobile

phones. Furthermore, with no doubt, the hand hygiene among the healthcare workers ought to be monitored and the feedback regarding their performance is more than welcomed.

The similar remarks as to the role of the disinfection of the mobile phones has been made in 2015 by Heyba, Ismaiel, Alotaibi et al. [22] who have noticed that 66.5% of the researched group never had properly disinfected their phones, this number was even higher in the case of studies conducted in Saudi Arabia [24] –76%, whereas in Northern Ireland only 37% of medical employees that have participated in the study had disinfected their wireless electronic devices [26]. In Australia, merely 31% of staff members of local healthcare institution had reported cleaning their cell phones on a daily basis, while 21% of the researched group stated that they use alcohol-containing wipes to do so [15]. Even the lower number of employees of healthcare institution that was involved in the research conducted by Brady et al. [18] admitted to cleaning their cell phones regularly – eight percent to be exact. In the same study, after the mobile phones have been disinfected, the significant decrease (by 79%) in the contamination rate of their surfaces has been reported.

It should be pointed out that the cell phones that can be found in a healthcare facilities are not only those owned by the medical personnel, but also by the patients themselves and the mentioned electronic devices should also be considered as potential carriers of bacteria. Indisputably, the research concentrating on the probable dissemination of pathogens through cell phones owned by medical personnel remains a majority of the studies developed on this particular topic, whereas the probable role in the spread of bacteria of the wireless electronic devices owned by the patients or even the visitors simply passing by the medical facilities, still remains fairly undeveloped matter. Brady et al. [18] in their research had put an emphasis on the bacterial colonization of the mobile phones owned by patients and the patients' awareness of possible cross contamination. Among the group involved in the study, 86.4% of patients who declared owning the mobile phone did bring it into the hospital. The majority of responders –70.3%, stated that they are aware that cell phones can be contaminated by bacteria and that bacteria can spread through cell phones form one location to another. Yet, according to the data provided by the authors, not even a single patient has been informed about mobile phones utilization guidelines during their hospital stay. As far as the disinfection proceedings prior to their hospital stay, 50.9% of patients stated that they have never cleaned their mobile devices, 6.9% admitted to disinfecting them annually, 11.8% monthly, 17.6% weekly, while 12.7% daily. Only 10.8% of the patients involved in a study declared that they have disinfected their wireless electronic devices while staying at the healthcare facility. Also, the authors noted that they did not find a single patient who declared sharing his/hers phone with any other patient, although some of them declared that they probably would, if asked. Therefore, it is advised to introduce the guidelines for patients addressing the proper handling of the cell phones and appropriate disinfection methods that can be both applied on a daily basis and even more importantly after their admission to a hospital.

It seems that more and more health care institutions make an attempt to introduce guidelines addressing the proper handle of electronic devices, including cell phones that aim to prevent the spread of bacteria. In 2012, in Canada, for instance, the CHICA- Canada Practice Recommendations has issued a guideline addressing the matter of the presence and usage of electronic devices, including the wireless phones in the healthcare facilities [27]. In that document addressing the Infection Prevention and Control Related to Electronic (IT) Devices in Healthcare Settings, the standards and protocols focusing on infection prevention and control considerations for electronic appliances have been outlined. Among them, the following recommendations have been made: hand hygiene ought to be performed prior to contact with a patient as well as before and after accessing the electronic appliance; the devices that cannot be properly disinfected should not be used in patient rooms and all surfaces of electronic devices that are accessed at or close to point-of-care must be disinfected with a hospital-grade disinfectant [28]. Nevertheless, in general, there is a shortage of guidelines addressing the issue of proper handling of the cell phones by the patients and visitors while residing at a healthcare facility, nor it is clearly stated in which areas those wireless devices can be used and in which zones it is strictly prohibited. Therefore, it is advised to issue such guidelines, since it may, at least theoretically, increase the awareness of the possible contamination of the surface of the wireless electronic device among people without medical background, and may help prevent the possible spread of pathogens through mobile phones.

CONCLUSIONS

Some of the pathogens that can be found on the human skin basically migrated from other places and surfaces. Each time we touch an object or simply when we are present in a different environment - we are in direct or indirect contact with pathogens that are present on those surfaces or in those places and thus, some of the microorganisms migrate on our bodies and vice versa. In other words, we frequently transfer the microbes from and to our surrounding and that includes our belongings.

The correlation between a person's microbiome and one's health is so to speak extremely complex and still rather poorly comprehended [29]. As the research on this matter continuous, the noninvasive sampling of personal items, like cell phones, especially in case of healthcare employees can possibly be useful in the detection and inhibition of the spread of bacteria, hence improving the prevention of probable cross-contamination. Proper care should be taken while using the wireless electronic devices, especially at the point-of-care. The same rules should also be applied, at least to some extent, to the patients and visitors of healthcare facilities when they are accessing their mobile phones, since pathogens could potentially spread through their personal belongings – including their cell phones, as well. Moreover, the employees of medical facilities and also individuals lacking the medical background- including the patients, should be educated about the possibility of the spread of bacteria through their personal belongings, including their wireless electronic devices, since, at least theoretically, increasing the knowledge about measures to prevent the probable contamination, may indeed led to lower cross- contamination rate.

Mobile phones, generally speaking, are carried by their owners constantly and therefore they are more prone to come in contact with foreign microorganisms in comparison to the objects that are being used less frequently or only in a certain environment, yet cell phones can still be involved in the spread of the pathogens from one place to another, just like any other objects. Therefore, it is essential to increase the awareness of this particular issue, especially among the healthcare personnel, as well as to introduce the means to prevent the spread of bacteria through wireless phones.

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Our Dermatology Online

Review Article

Signalling pathways in dermatology

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ABSTRACT

Different signalling pathways are involved in cellular growth, differentiation, migration and maintenance of stem cells. These include EGF and EGFR, IGF-1R, Hedgehog, WNT, MAPK, PI3K and MC1R-MITF etc. Aberrations or overexpression of these signals result in abnormal proliferation of cells, which may leads to development of tumours. This review stress on different signaling pathways involved in growth, differentiation and maintenance of stem cells, which are important for pathogenesis of many diseases when the pathways are defective.

Key words: EGF; EGFR; MAPK; PI3K; MC1R-MITF; Janus kinase

INTRODUCTION

Human beings have several signalling pathways that are involved in cellular growth, differentiation, migration and maintenance of stem cells. These include EGF and EGFR, IGF-1R, Hedgehog, WNT, MAPK, PI3K, MC1R-MITF, JAK-STAT etc. Aberrations or overexpression of these signals result in abnormal proliferation of cells, which may leads to development of tumours.

EGF AND EPIDERMAL GROWTH FACTOR RECEPTOR (EGFR)

Epidermal Growth Factor (EGF) 6.2kDa protein which stimulates the proliferation and differentiation and migration of epithelial cells and fibroblasts. The biological effects of EGF are mediated by a specific transmembrane receptor which is a 170kDa monomeric glycoprotein which has intrinsic tyrosine kinase activity. When EGF binds to the receptor, it results in autophosphorylation of the receptor and transduction of the EGF proliferative signal. This activated EGF receptor phosphorylates phospholipase C-γ1 (PLC-γ1) and other proteins involved in signal transduction. The activated PLC-γ1 hydrolyses inositol phospholipids and results in increase in intracellular calcium levels, which subsequently activate protein kinase C (PKC) which in turn, attenuates the tyrosine kinase activity of the EGF receptor [1,2].

EGFRs is known to play an important role in regulating the development of the epidermis and its appendages. EGFRs are predominantly expressed in the basal layer of epidermis. As the cell moves up, they are down-regulated as cells commit to terminally differentiate. EGFR has the ability to activate Ras-MAPK signaling, resulting incellular proliferation. It also activate PI3K–Akt signaling which is typically associated with cell survival. AP- epidermal keratinocytes had an inhibitory effect on EGFR promoter activity while the loss of AP-2β results in massive apoptosis. EGFR also activates other pathways such as phospholipase-C and small GTPases such as Rho and multiple signal transducer and activator of transcription (STAT) isoforms. Overexpression of EFGR is associated with tumorigenesis [1,3].

EGFR overexpression and activation results in increased migration of epithelial cells. Which is mediated by Matrix Metalloproteinase (MMP) which can regulate cell growth in different ways (e.g. the release of membrane-bound growth factors like tumor growth factor). E-cadherin and p120 also modulate EGFR Effects upon Cell Adhesion. By promoting increased migration, EGFR is decreases cell adhesion by its interaction with beta-catenin, as the phosphorylated catenin can no longer mediate the connection of the cadherin-catenin complex with the actin-cytoskeleton. EGFR also mediates increased cell aggregation

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through relocalization of p120 which is a component of adherens junctions [1-3].

Insulin-like growth factor 1 receptor (IGF1R)

IGF1R is a transmembrane, ligand-activated tyrosine protein kinase which consists of Alpha-2 Beta-2 heterotetramers held together by disulfide bridges. Both IGF1 and IGF2 exhibit high-affinity binding to IGF1R. Most of the biological effects of IGF1 and IGF2 are mediated by IGF1R. IGF1R-mediated inhibition of apoptosis depends on the activation of PI3K (Phosphatidylinositol-3-Kinase) and Akt/PKB (Protein Kinase-B). The binding of IGF1 or IGF2 to IGF1R activates tyrosine kinase, resulting in the phosphorylation of the IRSs (Insulin Receptor Substrates) which further interacts with the cytoplasmic protein PI3K, leading to the transduction of the functional effects of IGFs, such as enhanced glucose transport, enhanced cardiomyocyte contractility and the inhibition of apoptosis by activating several downstream proteins and molecules. PIP3 binds to the Akt and PDK-1 (Phosphoinositide Dependent Kinase-1). PDK-1 then phosphorylates Akt. PDK-1 also phosphorylates PKC (Protein Kinase-C)[4]. These PKCs, along with Akt facilitate the GLUT4 (Glucose Transporter-4) translocation from the GLUT4 vesicle to the membrane, enhancing the rate of Glucose uptake by the cell. A primary target is the BAD. In its non-phosphorylated state, BAD locates at the mitochondrial membrane where it interacts with BCL2 (B-Cell CLL/Lymphoma-2) and prevents it from performing its anti-apoptotic functions. When Akt phosphorylates BAD, BAD associates with the cytosolic protein and is unable to interfere with BCL2. Akt can also phosphorylate and inactivate Caspase9, preventing the initiation of the Caspase cascade. Akt also phosphorylates several pro-apoptotic members of the forkhead transcription factor family, FKHRL1, FKHR and prevents their activity. Akt decreases expression of FasL (Fas Ligand), thereby decreasing Fas-mediated apoptosis. Akt not only inhibits pro-apoptotic transcription factors, but also increases the levels of anti-apoptotic proteins including BCL2 and BCL-X and several extracellular matrix adhesion molecules [5]. Phosphorylated Akt also stimulate expression of the anti-apoptotic transcription factor NF-Kappa B (by regulating I-Kappa B Kinases). This results in I-Kappa B degradation and allows NF-Kappa B to enter the nucleus and activate transcription of anti-apoptotic genes. Akt also inhibits GSK3 (Glycogen Synthase Kinase-3) which promotes the dephosphorylation and activation of Glycogen Synthase, leading to the stimulation of glycogen synthesis. GSK3 also catalyses the phosphorylation and inhibition of eIF2B (Eukaryotic Protein Synthesis Initiation Factor-2B), thereby inhibiting protein synthesis. Hence, by inhibiting GSK3, IGF1R stimulates the dephosphorylation and activation of eIF2B, leading to an increased rate of protein synthesis. IGF1 promotes protein synthesis by activating eIF4E (Eukaryotic Initiation Factor-4E)[6]. Akt also phosphorylate mTOR. Phosphorylated mTOR promotes phosphorylation and inhibition of 4EBP1 and promotes protein synthesis by relieving 4EBP-mediated inhibition of eIF4E. Upon phosphorylation by mTOR, the ribosomal p70S6K and S6 (Ribosomal Protein-S6) becomes activated and promotes protein synthesis [7].

IGF1R phosphorylation leads torecruitment of protein SHC (SH2 Containing Protein) to the receptor and which is then phosphorylated. Activated SHC then binds the adaptor GRB2 (Growth Factor Receptor Bound Protein-2), recruiting the SOS in an IRS-independent manner. This complex then activates Ras and initiates sequential phosphorylation cascades involving serine/threonine kinase Raf, MEK1/2 (MAP Kinase Kinases) and ERK1/2 (Extracellular Signal Regulated Kinases). This pathway of IGF1R signaling is associated with cell differentiation, migrationand regulation of the machinery of apoptosis [6-7].

Besides, the IGF1R also modify Calcium-dependent signaling pathways. Binding of ligand IGF1R leads to activation of voltage-dependent Calcium channels, thereby, causing large transient increases in the Ca2+ levels, which further regulate Calcium-dependent transcription factors such as MEF2 (Mads Box Transcription Enhancer Factor-2), NFAT (Nuclear Factors of Activated T-cells) and CREB. These transcription factors promote the expression of several anti-apoptotic proteins including BCL2. The increased Ca2+ levels in the cytoplasm disrupt the inhibitory effects of Calmodulin, thereby, activating the protein phosphatase Calcineurin. Calcineurin activation leads to the dephosphorylation of NFAT, allowing it to enter the nucleus, where it cooperates with other transcription factors to bind promoters. The CalmKs (Ca2+/Calm [Calmodulin]-dependent Protein Kinases), activated by the increased Ca2+ levels activate the CREB and the ERK1/2 pathway which promote cell survival [4-7].

HEDGEHOG (Hh) pathways

The Hedgehog signaling pathway is a biological signalling pathway which is important for regulation of

the normal cell-fate specification, tissue polarity and patterning and organogenesis during embryogenesis and tissue homeostasis after severe injuries. The Hh proteins include SHH, Indian hedgehog (IHH) and Desert hedgehog (DHH). All three Hh proteins are able to bind the PTCH1 receptor and activate the Hh pathway in a time and concentration dependent manner [6].

The Hh protein, including SHH, IHH or DHH ligand binds to its 12-pass transmembrane PTCH1 or PTCH2 receptor, relieving the repressive effect induced by this receptor on the activity of its signaling partner, a seven-pass transmembrane coreceptor, SMO. The stimulation of the SMO signaling transduction results in the activation of cytoplasmic GLIs and their translocation to the nucleus, where they participate in the transcription [6,7].

The positive regulatory signalling pathways include EGF/EGFR, Wnt/beta-catenin and TGF-beta, which can cooperate with the canonical Hh ligand-induced signaling to activate GLI proteins and Hh target gene expression. TGF-beta can up-regulate GLI1 and GLI2 expression, thereby contributing tumorigenesis. More specifically, the activation of TGF-beta/TGF-R1-ALK5 system results in the nuclear translocation of Smad3-Smad4 complexes that directly interact with the GLI2 promoter and promote the recruitment of beta-catenin. These nuclear factors can up-regulate the GLI2 expression, which in turn induce the transactivation of Hh target genes, including GLI1 [8-10].

MAPK signalling

MAPK pathways regulates proliferation, growth and migration of cells. When the ligand binds the receptor tyrosine kinase like KIT receptor on the cell surface, the kinase-mediated phosphorylation leads activation of the receptor. The phosphorylation of tyrosine residues give way for binding to adapter protein like GRB2, SOS etc, which initiate the signalling cascade that requires GTPase activity of NRAS and relayed to the kinase activities of BRAF, MEK and ERK. Within the nucleus, this results in transcription of genes involved in cellular proliferation, growth and migration [6,7,11-13].

PI3K signalling

Phosphoinositide 3 kinases are enzymes which regulate growth, proliferation, differentiation, motility and survival of cells. They are activated by RTKs and PIP2 is phosphorylated into PIP3 which act as 2nd messenger. This phsphorylate AKT thereby inhibiting apoptosis by further phosphorylating BAD, leading to loosing of its pro-apoptotic function, increasing transcription of genes, enhancing cell survival and accelerating cell growth via mTOR [6,7,11-13].

WNT signalling

WNT signals are involved in cellular differentiation, migration, proliferation and maintenance of stem cells. When the Frizzled/LRP receptor complexes are not bound by ligands, beta-catenin is phosphorylated which is ubiquitinated and destroyed by proteasome. When WNT binds Frizzled/FLR receptor complex, dishevelled (DSH) is activated and inhibit catenin destruction, thereby stabilizing catenin. The beta- catenin, then enter the nucleus and promotes transcription [12,13].

MC1R-MITF signalling

The Melanocortin-1 receptor(MC1R) is a G-protein coupled receptor. It is activated by Melanocortins (ACTH, alpha-MSH etc). MC1R activates adenylate cyclase, thereby increasing cAMP, which activates Protein kinase (PKA), which, in turn, activates cAMP responsive element binding protein(CREB), which is a transcription factor. MITF is also a trancription factor, which is regulated by MAPK and WNT signalling [6,7,11].

JANUS KINASE

Janus kinase (JAK) is a family of intracellular, non receptor tyrosine kinases. It mediates cellular signal called JAK-STAT pathway. JAK1, JAK2 and TYK2 genes have been mapped to chromosomes 1p13.3. Upon binding of ligand to the receptors, JAK kinase activates Src-kinase cascade, Ras-MAP kinase pathway, PI3K-AKT pathway and STAT signaling. Type I and type II cytokine receptor families possess no catalytic kinase activity, so they depend on the JAK family of tyrosine kinases to phosphorylate and activate proteins involved in their signal transduction pathways. Janus kinases phosphorylate activated cytokine receptors which in turn, recruit STAT transcription factors which modulate gene transcription [14,15].

JAK kinases are involved in signaling by interleukins such as IL-2, IL-4, IL-7, IL-9 and IL-15. Erythropoietin activates JAK 2. IL-3, GM-CSF and IL-5 activate JAK 1 and JAK 2. IL-12 stimulation activates. JAK2 and

TYK2 and plays a critical role in IL-12 mediated T-cell differentiation. IL-2 stimulation leads to the activation of JAK3. IL-2, IL-4, IL-7, IL-15 and IL-19 stimulation activate JAK 3. In short, JAK 1 and JAK 2 mediate Th1 response and JAK 3 mediates Th2 response. JAK kinases also mediate the signal transduction in response to stimulation by growth hormone, Prolactin and G-CSF [16,17].

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Our Dermatology Online

Clinical Image

Dermatitis herpetiformis of Dhuring

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History: Presentation of 15th years old young man with first skin lesions started five years ago as erythematous rush on elbows and knees. After few months the lesions as confluent erythematous plaques on the skin of the extensor side upper and lower extremities, back, neck and both gluteal region (Figs. 1a and 1b). On the same places thera are few singl and linear excoriations and non adheretnt crusts with intensive itching. From first skin lesions till nowadays, patient was treated by several dermatologist under diagnosis of pruritus, atopic dermatitis and eczematous dermatitis and used different types of local and systemic corticosteroids therapy, oral antibiotics antihistamines and one year of Dapson 50mg a day, but without any therapeutic effects.

PHD analysis- (Irregular acanthosis of epidermis. Partially, signs of inter and intra fluid accumulation. Dermal papilla's elongated and edematous. In single papilla's are groups of neutrophilic granulocytes (microabscess). Focal, in papilla's, presens of small splits filled with fibrin and neutrophilic granulocytes. Capillary blood vessels of the upper dermis are dilated, lined with hyperplastic and hypertrophic endothelial cells. Around are lymphocytic and neutrophilic granulocytes (PHD No 508/03.12.2014. confirmed the diagnosis of Dermatitis Herpetiformis)

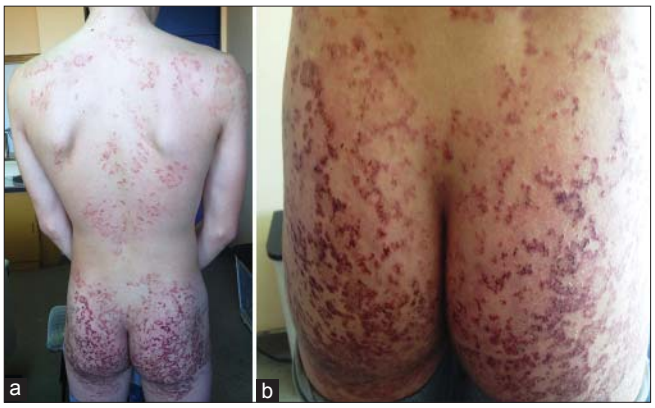

Figure 1: (a) Erythematous, confluent plaques with central regression, "gyres like" edges, marginally lined papules and dotted crusts on the neck, scapular, thoracolumbal and significantly more gluteal area, extensor sides of the upper and lower extremities. (b) Livid reticular plaques covered with dark crusts on the both gluteal areas.

Laboratory Examinations: Tranglutamine Antibody IgA 113 U/ml (lower limit 10 U/ml), Transglutamine Antibody IgG negative. (05.12.2015)

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Letter to the Editor

Long-standing asymptomatic pretibial patch

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Sir,

A 41-year-old woman presented with a long-standing, slow-growing, non-painful lesion on her right lower leg for 5 years. Previous treatments included topical corticosteroids and traditional Chinese medicine without improvement. Upon physical examination, a large-well-circumscribed plaque with a waxy-atrophic center was observed (Fig. 1). Dermoscopic evaluation showed serpentine vessels with multiple anastomosing ramifications over diffuse-patchy yellow-orange areas. Histologic examination revealed granulomatous formation with intermixed areas of collagen degeneration. Histiocytes were arranged in palisades and multiple giant cells were observed horizontally distributed in the observation field (Figs. 2 a and 2b). Laboratory results were notable for slightly elevated glucose levels (104.94 mg/dl) and elevated thyroid peroxidase antibody levels (11.85 lU/mL).

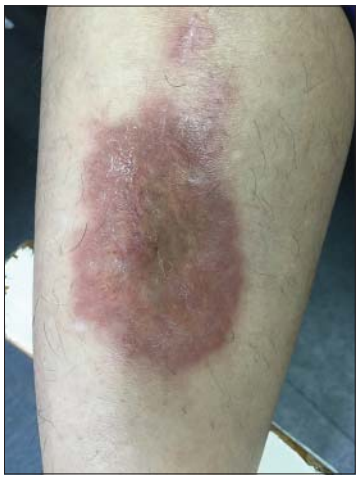

Figure 1: Clinical examination of a 41-year-old woman shows a well-circumscribed plaque, with indurated borders and atrophic center in the right pretibial area.

Necrobiosis lipoidica (NL) was first described in 1929 by Oppenheim and subsequently renamed in 1932 as we know it today [1,2].

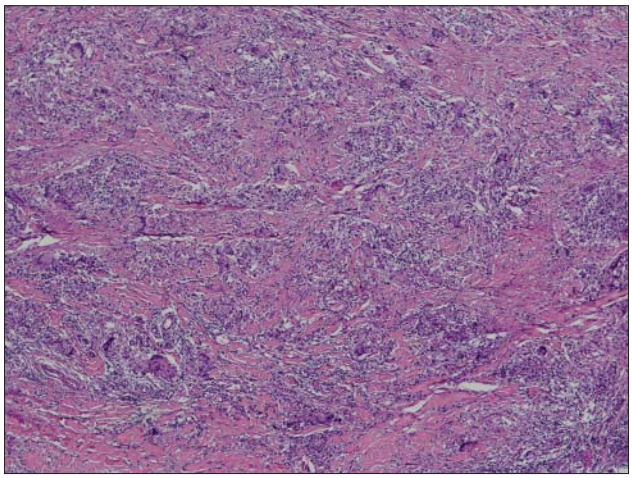

Figure 2a: Dense superficial and deep perivascular lymphoplasmacytic infiltrate accompanied by histiocytes and multinucleated giant cells in interstitial and palisaded array around foci of collagen degeneration. HE 40X.

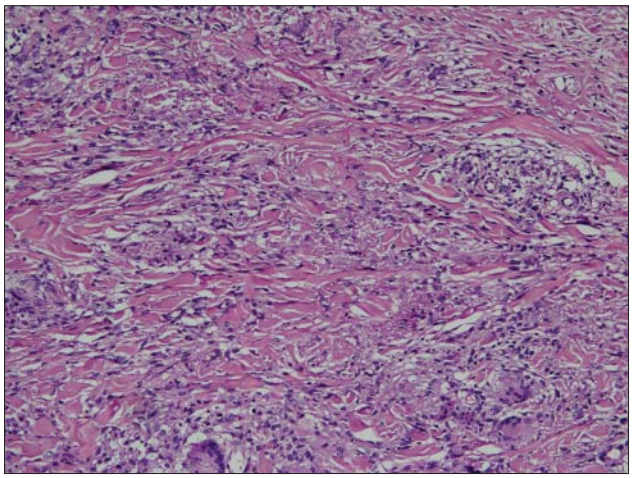

Figure 2b: Mixed interstitial inflammatory infiltrate. Note the presence of histiocytes and multinucleated giant cells. HE 200X.

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Etiology of NL is unclear, however, events such as microangiopathic changes, immunoglobulin deposition, increased collagen crosslinking and impaired neutrophil migration have been hypothesized to be implicated in the pathogenesis of this entity [3,4]. Associations with systemic diseases have been found primarily with diabetes mellitus and autoimmune thyroid disorders [5-7].

Dermoscopy can be a valuable aid to the clinician given that observed features correlate with specific clinical and histological findings. In our case, the presence of a diffuse patchy yellow-orange areas correlated with the presence of a horizontally arranged palisading granulomas on histopathology. These findings are different from those seen in Rosai-Dorfman disease where prominent yellow globules are observed with less conspicuous anastomosing vessels.

In summary, the presence of serpiginous branching vessels with patchy-yellow-orange diffuse areas supported the Dermoscopic diagnosis of NLD in this case.

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Letter to the Editor

Palmar involvement in lichen planus

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Sir,

Lichen planus (LP) is a chronic inflammatory dermatosis with numerous morphological patterns. Palmoplantar LP, acral, localized variant of disease is uncommon and does not usually have typical clinical characteristics of LP [1-3]. Because of presenting with various atypical clinical features, palmoplantar LP may create difficulty in diagnosis [2,3]. We describe a case of a 53-year-old man with hyperkeratotic, pruritic, erythematous, scaly plaques on both palms, especially in the hypothenar area.

CASE REPORT

A 53-year-old man was attended with hyperkeratotic, pruritic, erythematous, scaly plaques on both palms with one month history. The self and family history were unremarkable. On the dermatological examination, hyperkeratotic, erythematous, scaly plaques were observed on both mid-palms and hypothenar eminences, the fingertips were spared (Fig. 1). The other skin surfaces, hair, nails, and oral mucosa were normal. Routine laboratory tests including complete blood count, blood chemistry analysis, and urinalysis were normal. Serological tests for hepatitis B and C viruses, syphilis and autoimmune antibodies were negative. The potassium hydroxide (10% KOH) examinaton and mycologic cultures of scrapings from palmar lesions were also negative. Histopathological examination showed basal cell degeneration with a band-like lymphocytic infiltration in the upper dermis, focal hypergranulosis and irregular acanthosis with a saw-tooth like appearance on the epidermis (Fig. 2). The diagnosis of palmoplantar LP was made with clinical and histopathological features. The skin lesions of the patient improved with topical steroid ointment (0,05% clobetasol-17-propionate) twice a day for 2 months.

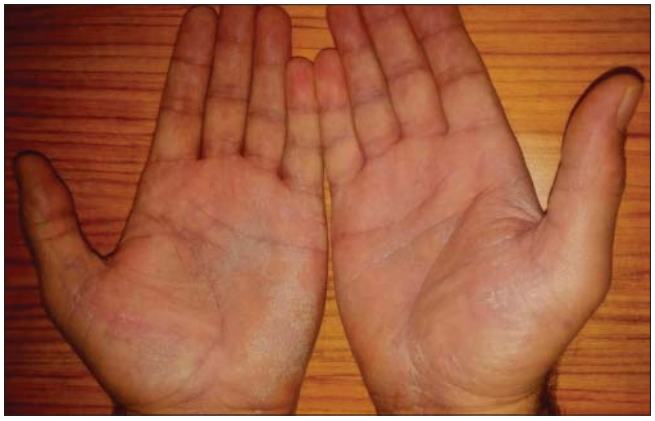

Figure 1: Hyperkeratotic, erythematous, scaly plaques were observed on both mid-palms and hypothenar eminences, the fingertips were spared.

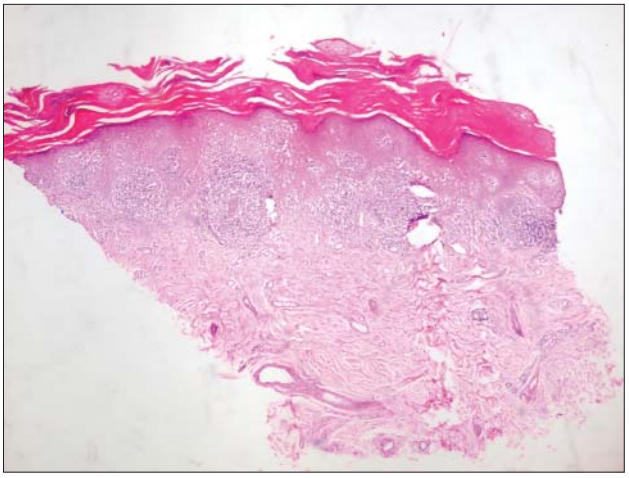

Figure 2: Basal cell degeneration with a band-like lymphocytic infiltrate in the upper dermis along with focal hypergranulosis and irregular acanthosis (H&E, 20X10).

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DISCUSSION

The diagnosis of palmoplantar LP may be difficult, especially when palmar lesions presented as an isolated finding, because palmoplantar lesions do not demonstrate typical polygonal, pruritic lesions of LP and Wickham's striae in contrast to LP lesions [3-5]. Several clinical variants of palmoplantar LP have been described, including erythematous scaly form, firm and rough, semi-translucent and waxy, erosive, hyperkeratotic, punctate keratosis-like, petechia-like and ulcerative forms [1,2-5]. The most common variant is erythematous scaly form in which yellowish, compact keratotic papules or papulonodules are seen on the palms [4-6]. The differential diagnosis of palmoplantar LP includes many dermatologic disorders such as psoriasis, lichen nitidus, acquired palmoplantar keratoderma, tinea, verruca vulgaris, callus, xanthomas, granuloma annulare, Reiter's syndrome, syphilis, punctuate porokeratosis and arsenical keratosis [5-7]. Histopathological examination must be done for the differential diagnosis and histopathology usually shows the characteristic features of LP [3-5].

The first-line treatment of palmoplantar LP is topical or systemic corticosteroids. Other treatment modalities include topical tazarotene, systemic acitretin and cyclosporin therapies. Also, narrow-band ultraviolet B, psoralen and ultraviolet A therapy (PUVA) and PUVA bathing and 308-nm excimer laser therapies may be effective. Surgical treatment with excision and grafting may be used in painful erosive cases [1,4-7]. The present patient was treated with topical steroid ointment (%0,05 clobetasol-17-propionate) twice a day. After 2 months, the skin lesions had improved.

The current report was presented a case of palmar LP because of its rarity and clinical diversity. In conclusion, this case emphasizes to clinicians that palmoplantar LP must be thought among the differantial diagnoses of the palmoplantar lesions and histopathological examination is essential to establish diagnosis.

CONSENT

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

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Our Dermatology Online

Letter to the Editor

Pincer nail

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Sir,

45 years old female with no significant past medical or family history that was seen due to contact dermatitis on her left arm, during her physical examination was observed to have onychophaty on her right toenails, pincer nails was observed on big and second toenail (Figs. 1 and 2), dermatoscopic view of the pincer nails (Fig. 3). The diagnosis pincer nails was done, the patient didn´t return to the another new.

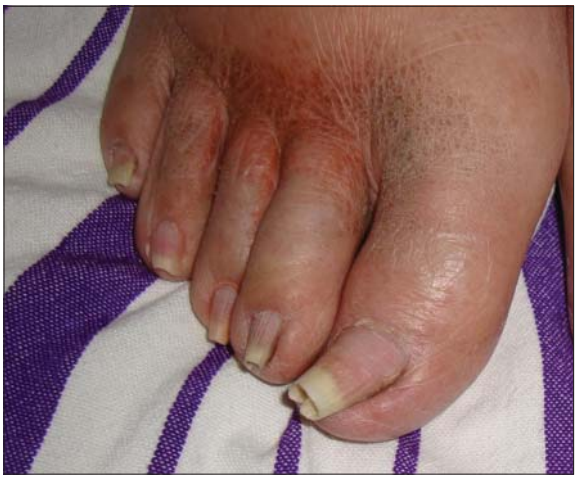

Figure 1: Macroscopic view of pincer nail.

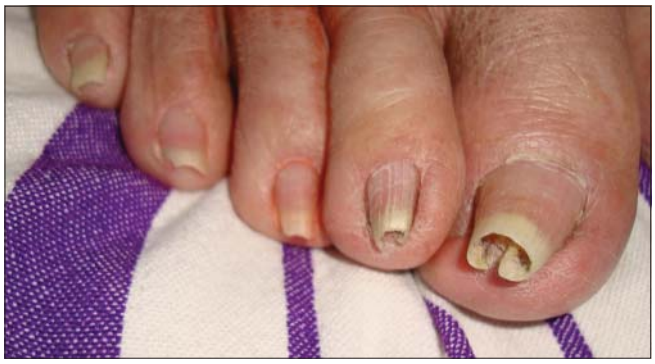

Figure 2: Close up of pincer nail on second and big toenail.

The pincer nail is a dystrophy of the nail probably produced by an enlarged base of the distal phalanx, thus producing an over curvature of the nail along its long axis. It is a frequent condition on toes; other names for this condition are: incurved nails, unguis constringes, trumpet nail or omega nail.

Usually, in toenails, the nail plate curvature is more pronounced than in fingernails, but when this curvature becomes excessive, the lateral border of the nail plate inserts itself into the soft tissue of the lateral grooves pinching the nail bed.

The lateral nail plate margin sinks into the epidermis thus producing granulation tissue, as in ingrown nail [1].

There are three clinical types of pincer nail depending on its morphology: trumpet nail deformity (Figs. 4a - 4e), tile nail (Figs. 5a and 5b) and plicated nail (Figs. 6a - 6c) [1].

Trumpet nail deformity, the most common form of pincer nail, is characterized by the nail plate lateral borders rolling under itself, taking a cylinder or omega shape, the transverse diameter of the nail is decreased and the distal border is lifted up by the traction exerted on the distal dorsal tuft, as a result the nail bed gets pinched. The plicated nail presents moderate convexity with its lateral edges sharply turned down to form a vertical sheet; then, the nails lateral edges press into the lateral nail grooves. The tile nail deformity is frequently seen in tall young people, with no serious symptoms, the nail has a transverse over curvature along its longer axis and the lateral nail edges remain parallel, it forms a tile shape; this type is usually less severe [1,2].

Patients with pincer nails generally have a great toe with the longer axis of its distal phalanx deviated laterally.

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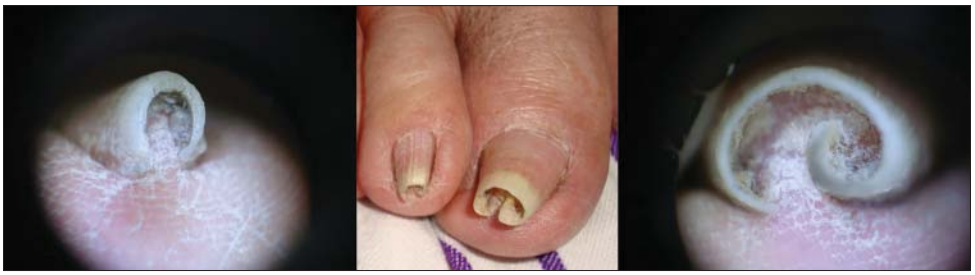

Figure 3: Dermatoscopic view of pincer nails.

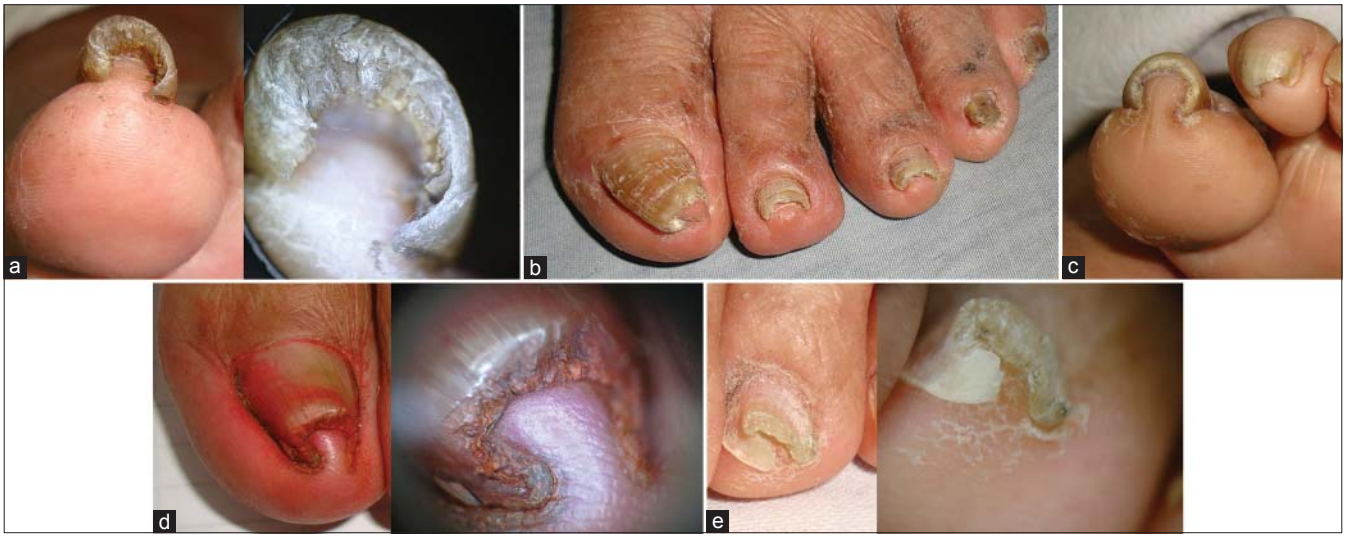

Figure 4: (a- e) Different types of pincer nails.

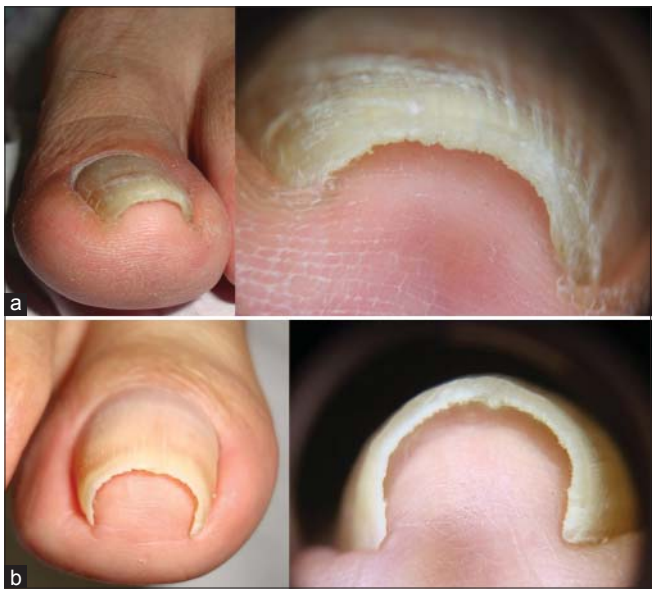

Figure 5: (a and b) Tile nails.

Overcurved nails are even more deviated in this direction. In contrary, lesser toes with pincer nails have a distal phalanx with medial deviation. The overcurvated nail plate also present a distal margin lifted up [1,3].

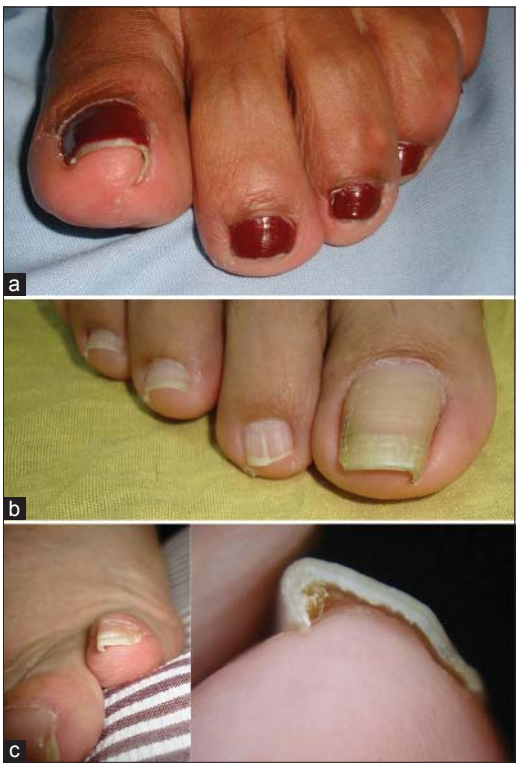

Figure 6: (a-c) Plicated nails.

For patients cutting this overcurved nail becomes painful and difficult; it can interfere with wearing shoes and suppose a cosmetic embarrassment. To improve cosmetic appearance, patients tend to round the distal edge of the affected nail and plate; this reduces pressure on the distal portion of the lateral nail grooves, but remains a risk of leaving a spike that will embed into the soft tissue [1].

Pathogenesis of pincer nail disease is still not clear, but etiology may be divided into acquired or hereditary disorder [1,4].

The acquired pincer nail disorder may be due to numerous factors: ill – fitting shoes or trauma producing phalanx deviation and deformity of the foot; tumors of the nail apparatus such like exostosis, implantation cyst, myxoid pseudo cyst [5]; degenerative osteoarthritis of the distal interphalangeal joints; drugs factors like beta-blocker therapy with practolol and acebutolol [6]; systemic diseases such psoriasis, the most frequent, Kawasaki's disease; the development of pincer nails deformity has been described on a women with chronic renal failure, the nail disease appeared coinciding with a severe decline in renal function [7]; pincer nail has been considered as a marker of gastrointestinal malignancy, specifically metastasing adenocarcinoma [8]; great toenail and thumb nail onychomycosis due to Trichophyton rubrum is related with pincer nail deformity; circulatory disturbance and venous hypertension due to arteriovenous fistula in the forearm may produce the pincer nail disease [1].

Acquired pincer nail deformity reverts after treating the base disease, discontinuation of the causal drug or correcting the causal factors [1].

Hereditary pincer nail deformity is usually characterized by symmetrical changes in the nail plate and similar nail findings may be seen in other family members [9]. The condition has been described like an inherited disorder with autosomal dominant Mendelian characteristics; this condition usually affects the great toes, but the smaller toes may be also involved [1,9].

The development of pincer nail deformity is related with the bone of the nail and with the base of the distal phalanx, which defines the shape of the nail plate. The nail matrix is adhered to the phalanx, when the base of the distal phalanx becomes wider the proximal nail plate curvature decrease and the distal curvature increase, it produces a conical shape of the nail plate.

Imaging studies, like x rays and magnetic resonance imaging, confirm the enlargement of the phalanx, and several times it shows hook-like lateral osteophytes directed distally. There is controversy whether osteophytes are a causal factor or a result of the deformity [1,4,10,11].

Pincer nails are as much of a cosmetic problem as a health problem that causes substantial discomfort and interferes with the daily life; it can cause acute pain, infections of the soft tissues, hinder the walk and impair the quality of life [10,11].

The indications for treatment of pincer nails dystrophy are pain and inflammation which interfere with wearing shoes or/and produce cosmetic discomfort [11].

There is no consensus about subungual hyperkeratosis, as it a common finding in pincer nails but the fungal infection is rare. Some authors recommend initiating proper treatment of hyperkeratosis before treatment of the dystrophy; others, recommend pincer nail correction first and then, with the nail growing better, treatment of fungal infection with a better chance of successful treatment than on the contrary [12].

There is no consensus about the most appropriate way of treatment depending on the severity of the condition, however, recurrence rates are higher with conservative treatment and it may provide only temporary relief [13].

Treatment of the dystrophy will depend on the patient comorbidities, preferences and expectations and the severity of the nail condition to achieve pain relief and good cosmetic outcomes.

Surgical methods include nail avulsion, total or partial excision of the nail bed, phenol matrixectomy of the lateral matrix horns, destruction of the matrices by electro cauterization, removal osteophytes, skin grafting or mucosal grafting of the nail bed, zig zag nail bed flap method, and others. Generally, the surgical procedures are considered an aggressive measure, to treat severe cases or for patients with deformity that is refractory to conservative approaches. The disadvantages of surgical treatment are pain, longer recovery time; secondary infections and some methods may induce cosmetic deformity. Despite this disadvantage, surgical treatment is indicated for long terms results and for severe conditions [10,11,14].

The conservative treatment may include nail grinding, nail brace, urea paste, nickel-titanium wires, and others. Conservative techniques have variable therapeutic results, but these are a useful options, especially, with comorbidities like diabetes, peripheral vascular disease or any other disease that might affect wound healing; and may be indicated for mild to moderate pincer nail deformity [10,14].

Consent

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

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Our Dermatology Online

Letter to the Editor

Desmoplastic trichilemoma of the scalp

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Sir,

A 66 year-old male presented with a 2 cm verrucous, skin colored plaque on the scalp. Histologic examination revealed lobules of glycogenated epithelium with peripheral palisading and a prominent basement membrane. At the center of the lesion, cords of basaloid cells were noted within a dense sclerotic stroma. No atypical features were found in the neoplasm. Immunohistochemical studies were performed showing expression of CD34 in the tumor cells (Figs. 1-3).

Trichilemmoma was described first in 1962 as a benign clear cell tumor with an outer hair root sheath differentiation [1]. Subsequently Hunt and coworkers reported several cases characterized by irregular cords and epithelial cells nests entrapped in a desmoplastic stroma which they called desmoplastic trichilemmoma (DT) [2].

Worldwide, less than 100 DT cases have been published, with a frequency of around 0.003% among skin tumors [3]. DT is usually seen in individuals after their fifth decade of life, affecting most commonly the face; less frequent involved areas such as the scalp, neck, chest and vulva have also been reported [2,4]. While DT is a benign lesion, it can be associated with other tumors such as basal cell carcinoma [5].

Clinically, DT presents as a dome-shaped papule with a smooth or irregular surface. Oftentimes it presents with pearly borders, telangiectasis and superficial ulceration [6,7]. The combination of these features may obscure the initial clinical diagnosis resembling those seen in basal cell carcinoma, verruca vulgaris, sebaceous hyperplasia and squamous cell carcinoma [4].

Histologically, DT is a well-circumscribed lobular lesion. At the periphery, it presents features of trichilemmoma with lobules of glycogenated cells and

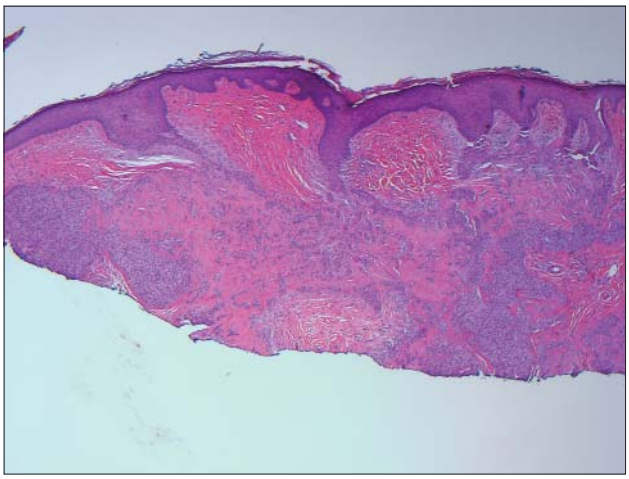

Figure 1: Lobules of glycogenated epithelium with peripheral palisading. At the center of the lesion, irregular cord and nests of basaloid cells in a dense sclerotic fibrocollagenous stroma are seen. HE 100X.

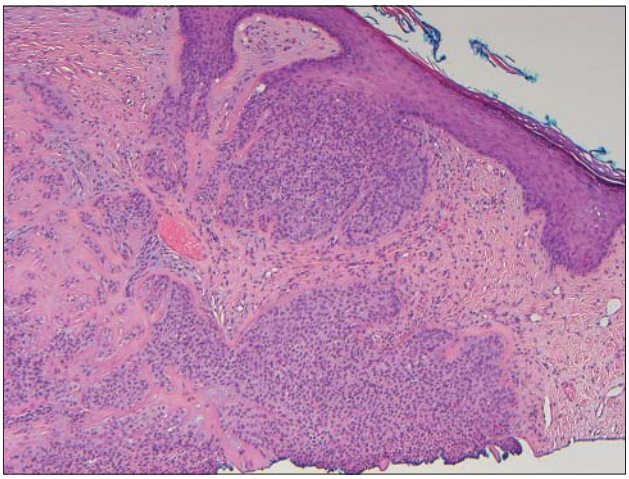

Figure 2: Higher power showing the transition between the lobules with trichilemal differentiation and the irregular cords within a desmoplastic stroma. Note the prominent basement membrane seen at the periphery of the tumor. HE 400X.

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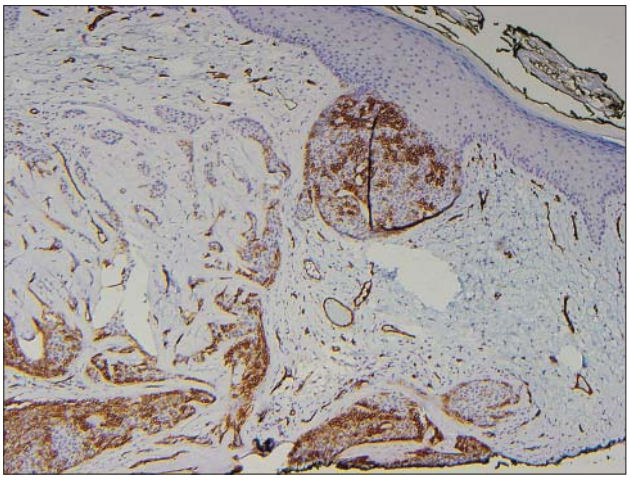

Figure 3: Expresion of CD 34 is observed in the tumor cells.

peripheral palisading, whereas the central part shows irregular cord and nests of basaloid cells in a dense sclerotic fibrocollagenous stroma [6,7]. Extension of the central part of the lesion into the dermis mimics invasion, however, cytological atypia is not usually seen [8]. CD34 is a useful marker which is expressed in DT but not in other neoplasms such as basal cell carcinoma or squamous cell carcinoma [9].

Due to the uncertain behavior of the tumor and the association with other malignant neoplasias, reexcision to ensure complete removal of the lesion is usually recommended. Mohs micrographic surgery has been reported and advocated by some authors as a technique that gives histological control of the margins with maximal preservation of the surrounding tissue [6,7].

Consent

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

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Our Dermatology Online

Letter to the Editor

Penodynia and Depression

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Sir,

Penodynia is a chronic penile pain of duration more than three months clinically presents as burning/vague pain located in the penis in the absence of any objective signs, or positive and relevant investigations to explain such a symptom [1]. Independent penile pain is a rare complaint [2]. Penodynia occurs when the symptoms develop in the absence of observable local disease, infection or a result of referred pain. The exact etiology of this pain is unexplored, because most often the clinical examination and work up unravel a definitive cause. These patients might have psycho social impairment, rarely this symptom (pain) will be the only manifestation of a mental illness [3]. In chronic pain treatment of the organic cause alone may not ease the symptom, especially when psychological and behavioral aspects are involved. This article represents our view on penodynia, depression and the role of amytriptylline.

A 35 years old married male presented with severe burning sensation of shaft of penis present throughout the day since 8 months. History of sexual exposure was present before the onset of symptoms. Clinical examination of scrotal skin, testis, cord, epidydamis, penis and perianal area did not reveal any abnormality. Per rectal examination was done to rule out prostate pathology. Lab investigations like random blood sugar, urine routine and microscopy, X ray lumbosacral spine and ultrasound abdomen, pelvis and scrotum was done to rule out an organic cause. Serological tests to rule out STI were done. The patient had guilt of his sexual exposure. Clinical interview by psychiatrist revealed disrupted sexual activity and psychosocial impairment. A diagnosis of depression was made. Patient was started on amitriptylline 10mg for 1 week then increased to 25mg. He showed gradual improvement in symptoms during the follow up.

This is the third case report of penodynia and first from the Asian country. The first case report on penodynia was published in 2004 by Markos [4] and two french articles by Dauendorffer JN in 2012 and 2014 [5,6]. Unfortunately, penodynia is a condition that has been open to elucidation due to sparse literature on the subject. Here we discuss the clinical presentation, differential diagnosis and management of chronic pain in penis. The etiology of penodynia is not understood, and treatment aspects remain controversial.

Penodynia is a diagnosis of exclusion. Clinicians should first think of the apparent causes of penile pain, such as sexually transmitted infection or trauma. However a wide range of differential diagnoses in patient with penile pain should be considered. The pathologic process within the penis that can result in pain include urethritis, urethral foreign bodies, priapism, Peyronie's disease, balanoposthitis and insect bites (for example ant/spider bites). The other causes of penile pain are due to adjacent structures which include prostatitis and scrotal disorders like testicular torsion, epididymitis, orchitis and direct inguinal hernia. Paraphimosis and balanitis should be considered as the differential diagnosis of penile pain in uncircumcised men [2]. Patients with pudendal neuralgiaand pain disorder associated with psychological factorsmay also experience penile pain which are relatively under-diagnosed [2,7]. So it involves a meticulous elicitation of clinical history and physical examination of the abdomen, buttocks, inner thighs, perineum and male genitalia. If needed repeated virologic, microbiologic, serologic investigations, and imaging like ultrasound, X ray, and MRI has to be done. Since any chronic symptoms can have a concealed psychological problem, other psychiatric illness has to be considered after excluding the possibility of organic source.

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In our case the patient developed depressive symptoms following the guilt of sexual exposure. Male genital pain may present after an episode of inadvertent sexual activity, frequently regretted and inducing guilt, especially in those with a rigid personality. Patients who present with apprehension about sexually transmitted infections frequently go through other ailments also. This may be rationalized into conviction of an infection that is difficult to appease [8]. Hence it is important to rule out STI before appropriate psychological evaluation by a specialist.

There is evidence of a high co-morbidity of chronic pain and depression. Studies found that as many as 75–80% of patients with depression report painful somatic symptoms. Depression and pain share biological pathways and neurotransmitters, which has implications for the treatment of both concurrently. A common theory holds that depression and painful symptoms follow the same descending pathways of the central nervous system. In depression serotonin and nor epinephrine are depleted which effect modulatory system (Limbic structures, Periaquaductal Grey area and on and off cells in Rostral Ventro-Medial Medulla) and then the subject appears to focus, attend to, and rate the pain stimuli as more severe [9].

Antidepressants do not prevent peripheral sensitization, but amitriptyline may reduce peripheral prostaglandin E2-like activity or tumour necrosis factor production. Blockade of peripheral nor-adrenergic receptors by tricyclic antidepressants (TCAs) may contribute to a peripheral analgesic action because peripheral release of noradrenaline (norepinephrine) and serotonin is known to be hyperalgesic. The high association of chronic pain and depression, which should lead clinicians to investigate both dimensions when a patient presents with either pain or depression, as it has shown that the presence of pain tends to negatively affect the recognition and treatment of depression and vice-versa [10].

Patients presenting with penodynia should be evaluated and managed using an interdisciplinary approach. Currently available treatment options are limited. Better understanding of the primary etiopathology of penodynia is required to develop specific treatment strategies.

Consent

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

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Our Dermatology Online

Historical Article

Scientific medical societies for hair; an overview

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ABSTRACT

This is a concise overview for the current non-profit membership-based scientific societies for hair. These societies served several functions in the interests of patients and trichologists. Building educational resources and arranging conferences were possible because of the support of these societies. However, there is a need to encourage all the members to contribute to these societies to make these societies stronger and more valuable.

Key words: Education; Hair; Society

Hair care is an important part of human life. Hair is needed for human for several reasons. Hair remains one of the most important parts of the person's appearance and his or her beauty.

Apart from the long hairs in high demand for making wigs, wig-lets, hair pieces and the modern laced hair systems used extensively for camouflaging the baldness, hair keratins are the richest source of the amino acid L-Cystiene which is used in the food and pharmaceutical industry [1].

Hair science is part of dermatology, and all professional dermatology societies have many educational activities related to hair disorders.

However, there are some scientific societies devoted to hair science, which I list some examples of them in Table 1.

The idea of establishing the first independent, nonprofit organization for hair research was launched on April 1989 by a small group of enthusiastic hair scientists and was soon followed by its formal constitution on the occasion of the first and founding meeting of the European Hair Research Society (EHRS) on November 1989 in Brussels, Belgium [2]. Currently there are 150 active members in EHRS, as of May 2014.

The scientific societies for hair play important role in education by arranging scientific meetings and conferences [3-5].

For example, the North American Hair Research Society (NAHRS) meets twice yearly at the annual meetings of the American Academy of Dermatology and the Society for Investigative Dermatology.

The NAHRS also interfaces with other international hair research societies (e.g., European, Australasian, Korean, Japanese, and Indian Hair Research Societies) and organizes the World Congress for Hair Research every one to three years on a rotating basis. This is the largest and most respected hair research meeting in the scientific community, with proceedings subsequently published in the Journal of Investigative Dermatology.

Table 1: Selected scientific medical societies for hair (listed alphabetically)

The Society	Year of establishment	Current president	Website
European Hair Research Society	1989	Abraham Zlotogorski	http://ehrs.org/
Hair Research Society of India (HRSI)	2004	Patrick Yesudian	http://www.inhrs.org/
Korean Hair Research Society (KHRS)	1998	SIM, Woo-Young	http://www.khrs.or.kr/
North American Hair Research Society (NAHRS)	1990	Wilma Bergfeld	http://www.nahrs.org/
Society for Hair Science Research (SHSR, Japan)	1993	Kensei Katsuoka	http://www.tokyo-med.ac.jp/derma/shsr/

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World congresses for hair research meetings have been held in Brussels (1995), Washington DC (1998), Tokyo (2001), Berlin (2004), Vancouver, BC (2007), Cairns (2010), and Edinburgh (2013).

This communication is just an overview; however, there is an obvious need for more studies to evaluate the activities of these societies and to find a better ways for these societies to improve their performance.

Developing updated website, and creating prizes for distinguished works are important features for any societies and need to be maintained.

Each society has to find the best tools to encourage all the members to be active. Women and young doctors, in particular, should find a place in the executive committees of these socities.

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Our Dermatology Online

Historical Article

Dermatology Eponyms – sign –Lexicon (P). Part 1

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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 (P) and other. The symptoms and their synonyms, and those who have described this symptom or phenomenon.

Key words: Eponyms; Skin diseases; Sign; Phenomenon

Paget's Sign

An increase in the size of the skull, which leads to prominence of the forehead, there is also a increase in the thickness of the vault. Marked enlargement of the maxilla is also a classic presentation of the disease [1-3]. These are signs of Paget's disease of bone, also known as osteitis deformans.

Paget's Eczema Sign

Eczema of the areola as a sign preceding cancer of the breast [4]. Also known as Eczema sign.

Sir James Paget

English surgeon, 1814-1899 (Fig. 1). An outstanding diagnostician, surgeon, and physiologist, Paget also emerged as one of England's finest pathologists.

aget's training began at the age of 16, when he was apprenticed to Charles Costerton, a general practitioner in Yarmouth. During the 4½ years he spent with Costerton, Paget learned a great deal about bones and anatomy.

Paget also was a good artist and botanist. In 1834 (with his brother as a coauthor), Paget published a book entitled A Sketch of the Natural History of Yarmouth and Its Neighbourhood. This remarkable book contained the names of more than 700 insects and 1000 plants.

At the age of 20 years, Paget entered London's St Bartholomew's Hospital as a medical student. During his first year as a student at St. Bartholomew's, he noted some white specks in the muscle of a cadaver he was dissecting. When examinining them with a microscope he found them to be small, encapsulated worms, later named Trichina spiralis by the British anatomist and palaeontologist Richard Owen (1804-1892). This was the first demonstration of trichinosis in man.

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Figure 1: Sir James Paget

James Paget served as surgeon extraordinary 1858-1877 at Bartholomew's Hospital, sergeant surgeon extraordinary 1867-1877. He was demonstrator of anatomy and became professor of anatomy and surgery at the Royal College of Surgeons of England (1847-1852) and was elected fellow of the Royal Society in 1851, its vice president 1873-1874 and president in 1875. He was honorary vice chancellor of the University of London, and was named doctor of honour of law at the universities of Oxford, Cambridge and Edinburgh.

His article, "On Disease of the Mammary Areola Preceding Cancer of the Mammary Gland," was published in St Bartholomew's Hospital Reports in 1874 and refers to Paget disease of the nipple. His 1877 paper, "On a Form of Chronic Inflammation of the Bones" (osteitis deformans, Paget disease) was published in the Transactions of the Medico-Chirurgical Society and remains a classic today [5,6].

Parrot Fever Sign

Pneumonia and sepsis, that can mimic typhoid fever, caused by zoonotic psittacosis [7]. The culprit bacterium *Chlamydophila psittaci* can be found in parrots, pigeons, parakeets, as well some domestic poultry, ruminants, and opossums. According to the Centers for Disease Control and Prevention, there have been fewer than 50 confirmed cases per year in the United States since 1996, although many cases may have gone undiagnosed or unreported (CDC).

Partridge's Sign

The penis drops off, after first drying up and turning black. A complication of typhus fever. Partridge Island, New Brunswick, just outside the main harbour of Saint John, was chosen as the location for a pest house and quarantine station as far back as 1785. In 1847, with a large influx of Irish migrants, the typhus epidemic quickly filled the fever shed with sick and dying. By the 1847 typhus season, 2115 people had died in New Brunswick, with 1196 dying at Partridge Island and in Saint John [8].

Pastia's Sign

1. Hemorrhagic lines appearing in body creases, as in the antecubital fossae. inguinal areas, and the wrists, during scarlet fever; they are visible at the onset of the rash and persist after its desquamation [9]. 2. Associated with scared fever (group A streptococcus or S. aureus rarely); confluent, finely punctate erythema (scarlatiniform) on the lower trunk and thighs with petechiae having a linear configuration in the inguinal region [10,11].

Known as **Thompson's sign.**

Sign was described by Constantin Chessec Pastia - Romanian physician (1883-1926) and Frederick Holland Thomson - British physician (1867-1938).

Pathergy Sign

Pathergy phenomenon is defined as a state of altered tissue reactivity that occurs in response to minor trauma (Fig. 2). This test is used as a criterion in most diagnostic criteria for Behcet's disease e.g., Curth criteria, Japan criteria, Hubault and Hamza criteria, Cheng and Zhang criteria, Dilsen criteria, Japan revised criteria, International study group criteria, Iran traditional criteria, the Classification Tree, the Dilsen revised criteria, the Korean criteria, and the International Criteria for Behcet's Disease.

The pathergy reaction is a unique feature of Behçet's disease and, according to the International Criteria for Behcet's Disease [12].

Patrick Yesudian Sign (Palmar Melanotic Macules Sign)

Palmar melanotic macules (palmar freckling) seen in type 1 neurofibromatosis This sign was first reported by Patrick Yesudian [13]. Multiple melanotic macules of varying sizes were present on the palmar surfaces of 42 of 50 consecutive South Indian patients with von Recklinghausen's disease. The histologic characteristics of the macules showed localized areas of fingerlike prolongations of the rete ridges with increased

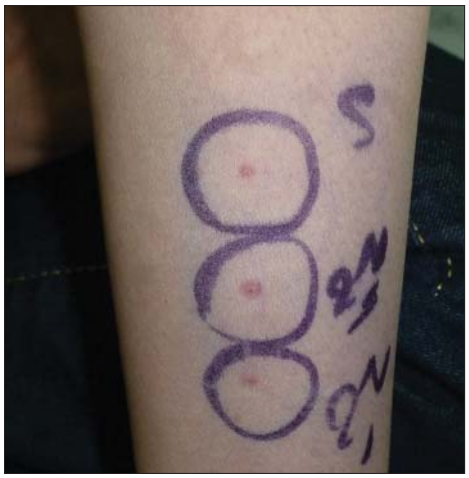


Figure 2: Pathergy sign. 3 pathergy's test: 1- with a 21 needle (marked "N 21" on the skin); 2- with a 25 needle (marked "N 25" on the skin); 3- with a 25 needle and inject one drop of normal saline (marked "S" on the skin)

pigmentation of the basal cells. This epidermal change overlies a small neurofibroma accompanied by thick-walled blood vessels in the reticular dermis [14].

Paullini's Sign

Chromhydrosis (grünen Schweiß), perspiration with a leek-green color. Similar symptoms are: Bartholinus's Sign [15], Lusitanus's sign [16], Chojnowski's sign [17]. Chromhydrosis, or colored sweat, is an interesting anomaly exemplified in numerous reports.

Christian Franz Paullini

German physician and theologian, (1643-1712) (Fig. 3). He studied theology and medicine in Gdańsk, Königsberg, Rostock, Lübeck, Kiel and Copenhagen, was Magister Artium in Wittenberg and received his MD in Leiden. Meanwhile he accomplished study stays and courses in Cambridge, Oxford, Sweden, Norway et Island. He was the Munster Bishop physician and later the Duke of Brunswick physician in Wolfenbüttel. He served as physician to the Bishop of Münster and the Court of Braunschweig, and as Herzoglichen Stadtphysikus (ducal state physician) in Eisenach.

He came back in Eisenach on 1685 and 1689 where he assumed the position of "Ducal Stadtphysicus" i.e. city doctor.

He focused on the therapeutic effect of feces in encyclopedic breadth, including all possible applications from head to foot, and his book went through many editions and reprints.

Figure 3: Christian Franz Paullini

He was a member of numerous learned societies such as Fruitbearing Society, Pegnesischer Blumenorden and German Academy of Sciences Leopoldina. In his long life of approximately 70 years, he wrote 68 books of which several editions were printed.

He made extensive reference and resorted to both ancient and contemporary medical authorities and to folk medicine (sailors, farmers, common people). He wrote a treatise (Flagellum salutis) on the advantage of the whip for curative purpose in various disorders and a handbook on the toad's therapeutic properties (Bufo juxta methodum et leges illustris Academiae Naturae curiosorum breviter descriptus). As a botanist, he gave his name to Paullinia cupana known as guarana, a climbing plant native to the Amazon basin and especially common in Brazil. As a zoologist, he described the kraken in 1706 after Francesco Negri in Animalia fabulosa [18,19].

Pavithran's Nose Sign

It is seen in exfoliative dermatitis in which there is complete absence of erythema and scaling of the nose and perinasal areas. It is hypothesized that sparing of nose in exfoliative dermatitis could be due to greater sun-exposure of nose or it could be explained by the mechanism of island of normal skin [20]. The sign described by K Pavithran. Also known as nose sign.

Paxton's Sign

Trichorrhexis nodosa is the most common hair shaft anomaly, caused by either physical or chemical trauma. It presents with minute grayish nodes along hair shaft and characteristic "thrust paint brushes" appearance on

microscopy. It may be congenital or acquired. Snonyme: lepothrix, trichomycosis chromatica, trichomycosis nodosa, trichomycosis nodularis, trichomycosis palmellina, trichonocardiosis axillaris, trichonodosis, Hodara disease (sign) or Hodarsche disease [21,22].

Periodic Sign

Disease which recurs at regular intervals or at the same period in every year [23].

Perry's Sign

Overdose of vitamin A often leading to death, caused by the consumption of polar bear liver, which can contain lethal concentrations of vitamin A. This condition which can present with findings of vision changes, headache, and altered consciousness includes the signs for pseudotumor cerebri [24].

Pettigrew's Sign

Paternal hereditary ichtyosis, morbid development of the papillae and thickening of the epidermic lamellae. Also called Armadillo sign. Pettigrew mentions a man with warty elongations encasing his whole body. At the parts where friction occurred the points of the elongations were worn off. This man was called "the biped armadillo." The females had normal skins. All the members of the well-known family of Lambert had the body covered with spines [25].

Described by Pattigrew and Ascanius.

Peutz-Jeghers Sign

Melanin pigmentation as spots around the lips associated with the intestinal polyps of Peutz-Jeghers syndrome [26] (Figs 4a and b). Peutz-Jeghers syndrome, first described by Peutz in 1921 and by Jegher in 1944, is characterised by hamartomatous polyps of the gastrointestinal tract (GIT) and mucocutaneous perioral pigmentation.

Johannes Laurentius Augustinus Peutz

Dutch physician, 1886-1957. He was began the study of medicine in 1905. After qualification in 1914 Peutz trained in internal medicine at clinics in Germany, Italy and Belgium.

In 1917 he became principal physician to the hospital of St Joannes de Deo, a Catholic Hospital at The Hague, where

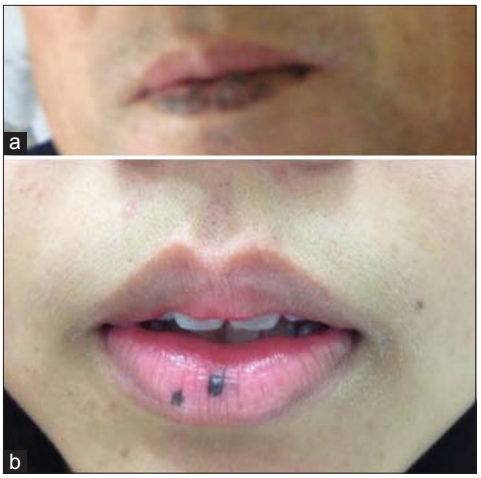


Figure 4: (a and b). Peutz-Jeghers sign

he remained for 34 years, until his retirement in 1951. He is credited with the establishment of an independent department of internal medicine, and a laboratory with electro-cardiographic facilities built to his personal specifications. He also contributed to the establishment of a department of pathology and a serologic laboratory, which was directed by Karl Landsteiner. Peutz was a dedicated clinician with broad scientific interests and in 1921 he obtained a Ph.D. with a thesis entitled "Clinical and experimental contribution to the diagnosis of and internal therapy for pancreas disorders and with particular reference to diabetes mellitus" [27].

Harold Joseph Jeghers

American physician, 1904-1990 (Fig. 5). He got a BSc-degree in biology in 1928 at the Rensselaer Polytechnic Institute, Troy, New York, and received his basic medical education at the Case Western Reserve University Medical School, Cleveland, qualifying in 1932.

He then undertook postgraduate studies, training in internal medicine at the Evans Memorial Institute for Clinical Research in Boston and at the Boston City Hospital, Boston University School of medicine. He became consultant physician at the Boston City Hospital, where he held a teaching post from 1937 to 1946. In 1946 he was appointed professor of medicine and physician-in-chief at Georgetown University School of Medicine, Washington D.C., while at the same time serving as consultant of internal medicine at the Walter Reed Army Medical Center, Washington D.C., and the National Naval Medical Center in Bethesda, Maryland.

Jeghers was appointed professor of medicine at Tufts University Medical School in 1966 and took a special

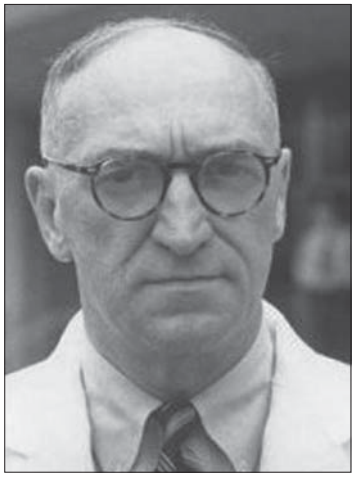


Figure 5: Harold Joseph Jeghers

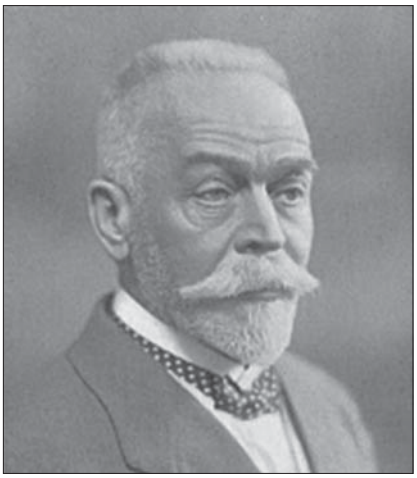


Figure 6: Emil Pfeiffer

interest in the techniques of medical education. He has written two books and more than 70 articles [27].

Pfeiffer's Fever Sign

A rare disease marked by elevation of temperature lasting for a short time and by rapid enlargement of lymph-nodes in the neck [28].

Emil Pfeiffer

German physician and paediatrician, 1846-1921 (Fig. 6). He studied medicine at the universities of Bonn, Würzburg, and Berlin, where he received his doctorate in 1869. Emil Pfeiffer is considered one of the most important of German balneologists. He concerned himself very thoroughly and scientifically with the therapeutic qualities of various mineral waters, particularly those found in his native city. He also worked as a paediatrician, and in this field worked for the establishment of homes for children and day nurseries. Pfeiffer also worked on gout and its therapy, with immunisation against smallpox through vaccination and variolation and described the glandular fever which bears his name [29]. Glandular fever was described by Emil Pfeiffer in 1889.

Phosphorus Sign

Garlic taste, swelling of the tongue, vomiting bilious green, black, and pure blood with repeated fainting. An indication of acute phosphorus poisoning [30].

30. Wiwanitkit V. Acute organo-phosphorus pesticide poisoning, oxidative damage, haemoglobin level and total leukocyte. Afr Health Sci. 2014;14:778.

Phossy Jaw Sign

A chronic condition of extreme pain and grotesque disfigurement caused by poisoning from exposure to white phosphorus. Sufferers have a foul fetid discharge from the jaw with a strong garlic smell. Also known as Garlic Breath sign, the disease, the compo, and the flute [1,20].

Physical Sign

One that can be seen, heard, or felt by the diagnostician. Also known as Objective sign.

Pigeon Nest Sign

Meningitis associated with zoonotic cyptococcosis from exposure to pigeon nests [31].

Pilomotor Skin Sign

The production of goose flesh on stroking the skin; trichographism [32].

Pimply Sign

(fr. Fievre boutonneuse), a zoonotic Rickettsia bacterium African tick typhus. Principle animals are dogs and rodents [33].

Pin Point Pupils Sign

Small pupils seen in narcotic drug addiction or tabes dorsalis or Hashimoto's encephalopathy and Nonketotic hyperglycinemia [34].

Pipe Smokers Sign

Nicotinic stomatitis. Nicotine stomatitis is characterized by the presence of white or gray lesions resembling cobblestones on the palatal mucosa. NS is most frequently related to pipe smoking, but mild cases of the disease can also develop secondary to cigar smoking or, rarely, from cigarette smoking. The palatal mucosa becomes thickened and hyperkeratotic.

Papular elevations with red centres, which represent the inflamed openings of the salivary gland ducts, often develop on the mucosal surface [35].

Pitaluga's Sign

Acquired hypertrichosis of eyelashes due to Kala-azar is called as Pitaluga's sign [36].

Pitres's Sign

1. hyperesthesia of the scrotum and testes. A sign of tabes dorsalis. 2. anterior deviation of the sternum. A sign of pleuritic effusion [37].

Jean Albert Pitres

French neurological physician, 1848-1927 (Fig. 7). He was born in Bordeaux and received his training in Paris, where he was the student of Jean Martin Charcot and Louis-Antoine Ranvier. He was the dean of the Faculty of Medicine of Bordeaux (appointed 1885). He began his medical studies in Bordeaux, later working as an interne to the hospitals of Paris (from 1872). In 1877 he defended his doctoral thesis, and during the following year received his agrégation with a dissertation titled "Les hypertrophies et les dilatations cardiaques indépendante des lésions valvulaires". Later he returned to Bordeaux, where from 1881 to 1919, he was maître to the chair of pathology.

Lessons that Pitres gave in the amphitheater on the following subjects were compiled and published: hysteria and hypnotism (1891), amnesic aphasia (1897), paraphasia (1898) and physical signs associated with pleural effusions (1902). His studies of peripheral neuritis were published in Volume XXXVI of Gilbert and Carnot's "Nouveau traité de médeine et de thérapeutique"[2]. With Leo Testut (1849-1925), he was co-author of "Les nerfs en schémas, anatomie et physiopathologie" (1925). His name became associated with pleural effusion and with tabes dorsalis [38].

Pitted Nails Sign

Psoriasis affecting the nail matrix [39,40] (Figs 8a – 8c).

Platysmal Eye Sign

The act of nipping the platysmamyoides contracts the pupil [41].

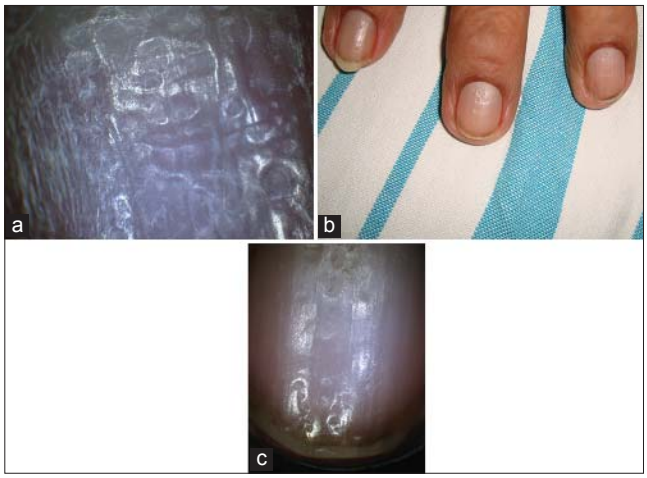
Plumbism Sign (Pierre Francès)

A blue line occurring at the gingival border with teeth (Fig. 9). A sign of chronic lead poisoning. Also known as Burton's line and sign [42].

Henry Burton

English physician (1799–1849). He describing the Burton line in 1840. He was born in London, attended Tonbridge School and studied medicine at Caius

Figure 7: Jean Albert Pitres



Figures 8: (a,b,c) Pitted Nails sign

College, Cambridge where he qualified in 1826. He was a physician at St. Thomas' Hospital in London from 1828, and became a Censor at the Royal College of Physicians in 1838. [43].

Pneumatic Sign

In the labyrinthitis of congenital syphilis, compression of the air in the external auditory canal produces a rotatory nystagmus to the diseased side; rarefaction of the air in the canal produces a nystagmus to the opposite side [23]. Also known as Hennebert's sign or test.

Camille Hennebert

Belgian otologist, 1867-1954 (Fig. 10). His year of death is also given as 1958. Camille Hennebert was affiliated with the Université Libre de Bruxelles. He published extensively. His name is associated with: Hennebert's fistula syndrome, Hennebert's syndrome [23].

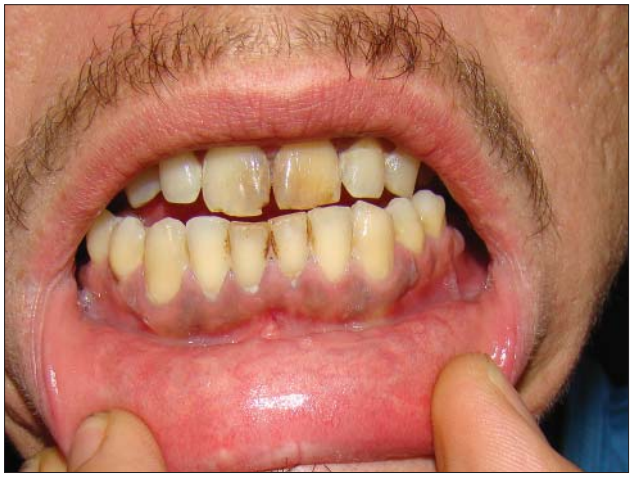


Figure 9: Plumbism sign

Figure 10: Camille Hennebert (Thank you to The Royal Library, Postbox 2149, DK-1016 Copenhagen, Denmark)

Polish Sign

A thick tangling of the hair with a sticky secretion that has a viscid smell of spoiled vinegar, mice, and garlic. The nails are spongy and blackish. Known in Cracow, Poland as weichselzopf, also called by the term plica polonica. If the hair was matted together so as to resemble ropes it is called plica multiformis and if these masses united together to form one single club of hair, like the tail of a horse, it is known as plica caudiformis, plica neuropathica [44]. Psychological disturbance is a risk factor for plica formation.

Plica first appeared in Poland in 1288 during the reign of Leszek II the Black (High Duke of Poland since 1279).

Perzyna Ludwik (1742-1800), a Polish doctor, monk, writer - popularizer of medical knowledge. In his medical books Perzyna conscious of the need for hygiene, traffic and moderation in eating and drinking. Proposed the introduction of regulations limiting the right to exercise the profession of pharmacist and physician to people ending their specialized schools. He demanded universal liquidation of the plica.

Józef Dietl (1804-1878) (Fig. 11). He was an Austrian-Polish physician. He studied medicine in Lviv and Vienna. He was a pioneer in balneology, and a professor of Jagiellonian University, elected as its rector in 1861. Dietl described the kidney ailment known as "Dietl's Crisis" as well as its treatment. He described about plica in 1858.

Figure 11: Józef Dietl

Henryk Franciszek Dobrzycki (1841-1914), Polish physician, philanthropist, musicologist and composer. A pioneer in the field of climate and sanatorium treatment in Poland. He described plica in 1877.

Ercole Sassonia, also known as Hercules de Saxonia, Hercules Saxonia Patavinus, or Hercules of Saxony (1551-1607) (Fig. 12), Italian physician. He was one of the great Italian clinicians of the Renaissance. He was educated in his hometown, and graduated with a degree in medicine from the University of Padua. In 1575 he became the professor of medical practice at the University. Becoming famous as a teacher, he was invited to Vienna by Emperor Maximilian II, where he remained until 1600. His chief scientific works were in the fields of diagnostics, skin diseases, and venereal diseases. Together with Thomas Minadous described plica in 1610.

But in 6900-6300 BC, in Israel, the Neolithic cave named 'Nahal Hemar', which is 14C dated to 6900-6300 BC, contained a mummy with matted hair and lice eggs [45].

Porcupine Sign

Ichthyosis, morbid development of the papillae and thickening of the epidermic lamellae [46]. Also called Steinhausen's sign

Porphyria Sign

Urine that darkens on standing to a port wine colour and fluoresces in ultra-violet light. A sign of porphyria [47].

Figure 12: Ercole Sassonia

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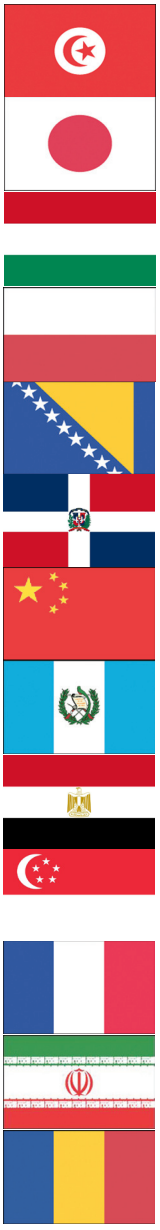

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