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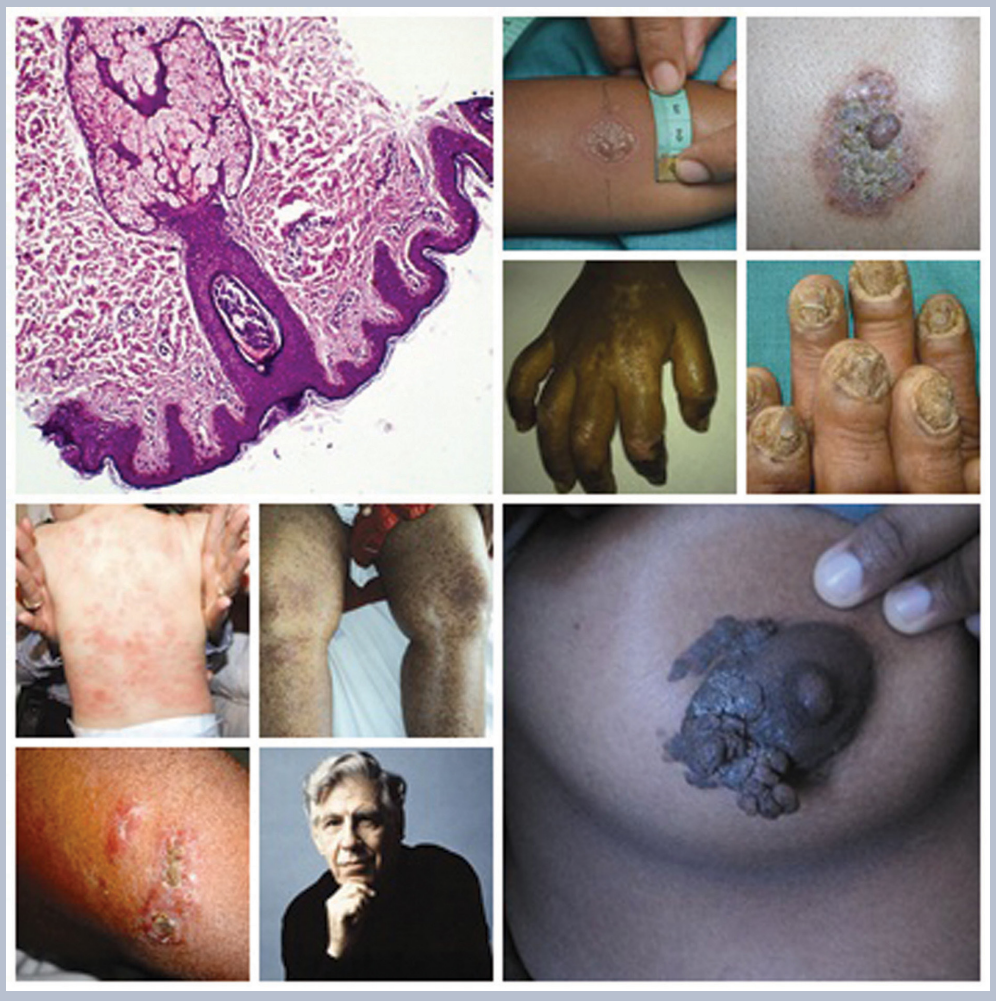

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NASZA DERMATOLOGIA Online
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SERUM LEVELS OF INTERLEUKIN-1 (IL-1A, IL-1B) IN PATIENTS WITH ALOPECIA AREATA

STĘŻENIE INTERLEUKINY-1 (IL-1A, IL-1B) U CHORYCH NA ŁYSIENIE PLACKOWATE

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Abstract

Introduction: Alopecia areata (AA) is disease characterized by focally, nonscarring hair loss on the scalp or other parts of the body. It affects 1-2% population of both genders and occurs at all age groups. The etiology is unknown, although most evidence supports the hypothesis that AA is a T-cell-mediated autoimmune disease of the hair follicle and that cytokines play an important role.

Objective: The aim of our study was to evaluate serum concentrations of IL-1α and IL-1β in patients with AA and healthy subjects and also to asses a possible association between these cytokines and duration of the disease.

Methods: Forty six patients with AA and 20 healthy controls were enrolled in the study. Serum concentrations of IL-1α and IL-1β were measured using enzyme-linked immunoassay techniques.

Results: The serum level of IL-1α in patients with AA was significantly higher than that in the control group (4.34±0.86 pg/mL vs 3.66±0.35 pg/mL, respectively). IL-1β levels were greater in patients with AA than in controls (2.35±0.17 pg/mL vs 2.24±0.30, respectively) but the difference was not significant ($p>0.05$). No correlations were found between duration of disease and the serum levels of IL-1α and IL-1β.

Conclusion: Our results have demonstrated the importance of determining IL-1a concentration in serum in patients with AA. This research could contribute to the interpretation of insufficiently well known views of the pathogenesis role and significance of IL-1α in AA.

Streszczenie

Wstęp: Łysienie plackowate to choroba charakteryzująca się ogniskowym, niebliznowaciejącym łysieniem skóry głowy lub też innych okolic ciała. Choroba ta dotyka 1-2% populacji, bez predylekcji płci ani też wieku. Etiologia choroby pozostaje nieznana, jednakże najwięcej dowodów potwierdza hipotezę, że AA jest chorobą autoimmunologiczną mediowaną za pomocą komórek T, zajmującą korzeń włosa oraz że cytokiny pełnią w tym procesie ważną rolę.

Cel: Celem naszego badania było oszacowanie stężenia w surowicy interleukin: IL-1α i IL-1β u pacjentów z AA oraz u osób zdrowych by wykazać możliwe związki pomiędzy tymi cytokinami a długością trwania choroby.

Metody: Do badania zakwalifikowano 46 pacjentów z AA oraz 20 osób zdrowych. Stężenia cytokin IL-1α i IL-1β były mierzone za pomocą techniki EIA.

Wyniki: Poziomy IL-1α u chorych na AA był znacznie wyższy niż ten w grupie kontrolnej (4.34±0.86 pg/mL vs 3.66±0.35 pg/mL, odpowiednio). Poziomy IL-1β były większe u pacjentów z AA niż w grupie kontrolnej (odpowiednio 2.35±0.17 pg/mL vs 2.24±0.30) jednak statystycznie nieistotne ($p>0.05$). Nie znaleziono korelacji pomiędzy trwaniem choroby a poziomami interleukin IL-1α i IL-1β w surowicy krwi.

Wnioski: Nasze wyniki badań dowodzą wagi pomiaru stężenia IL-1a w surowicy krwi osób chorych na AA. To badanie może przyczynić się do nie do końca poznanej roli IL-1α w patogenezie oraz odkryciu pełnego znaczenia w Alopecia Areata.

Key words: alopecia areata; cytokines; interleukin-1a; interleukin-1β

Słowa klucze: łysienie plackowate; cytokiny; interleukina-1a; interleukina-1β

Introduction

Alopecia areata (AA) is heterogeneous disease characterized by nonscarring hair loss on the scalp or other parts of the body. It affects 1-2% population of both genders and occurs at all age groups [1]. A wide range of clinical presentation can occur-from a single patch of hair loss to complete loss of hair on the scalp (alopecia totalis-AT) or the entire body (alopecia universalis-AU). The course of AA is usually characterized by phases of acute hair loss followed by spontaneous hair regrowth. However, in severe forms hair loss can persist for many years or even life. Although the etiopathogenesis of the disease is not clear, several studies have shown that within the cascade of pathogenesis of AA, cytokines play a crucial role.

It is also considered that a disequelibrium in the production of cytokines, with a relative excess of proinflammatory, versus antiinflammatory cytokines may be involved in the persistence of AA lesions [2-4]. Hair loss may occur because proinflammatory cytokines interfere with the hair cycle, leading to premature arrest of hair cycling with cessation of hair growth [5]. This concept may explain typical clinical features of AA such as a progression pattern in centrifugal waves [6] and spontaneous hair regrowth in concentric rings [7], suggesting the presence of soluble mediators within affected areas of the scalp. Interleukin 1 (IL-1) is a multifunctional proinflammatory cytokine, which has been implicated in the pathogenesis of several chronic inflammatory disorders with an autoimmune component. There are two forms of IL-1: IL-1α and IL-β. Both forms of IL-1 bind to the same receptor and therefore also show similar if not identical biological activities. Studies have shown that IL-1 is a very potent inducer of hair loss and a significant human hair growth inhibitor in vitro [8,9].
Literature data on serum IL-1 in patients with AA are very limited. Therefore, the aim of our study was to evaluate serum concentrations of IL-1α and IL-1β in patients with AA and healthy subjects and also to asses a possible association between these cytokines and duration of the disease.

Methods

The study included 46 patients (29 females and 17 males, median age 36.5, ranging from 5 to 69 years) who presented to the Dermatological Clinic with complaints of hair loss and were diagnosed with AA. Patients with any scalp disorders such as irreversible alopecia, trichotillomania and scalp psoriasis were excluded from the study. The patients who had received any treatment within previous 3 months were excluded from the study, as well as patients with any diseases based on the immune pathomechanism, which could influence serum concentrations of IL-1.

According to the duration of disease, patients were divided into 3 groups:
1. Duration for 6 months or less,
2. Duration of greater than 6 months, but less than 12 months,
3. Duration for year or longer.

The control group consist of 20 generally healthy people (11 females and 9 males, age range 6-63, median age 32.6 years). They did not have any scalp lesions in their personal history or on clinical examination. All subjects gave their informed consent in accordance with the requirements of the Institutional Ethics Committee.
Serum concentrations of IL-1α and IL-1β were measured by an enzyme-linked immunosorbent assay (ELISA) technique, using Quantikine Human IL-1α and IL-1β Immunoassay (R&D Systems, Minneapolis, USA). Briefly, a microplate was coated with a monoclonal antibody that was specific for the cytokines, and standards and samples were pipetted into the wells. After washing, an enzyme-linked polyclonal antibody that was specific for the cytokines was added. The reaction was revealed by addition of the substrate solution. The color development was stopped and the intensity of the color was measured at 450 nm with a photometar (Rider Biotek Elx800).

The data are expressed as mean±standard deviation. The test distribution was done by Kolmogorov-Smirnov test, and comparisons were performed by T-test. The data were considered statistically significant if p values were less than 0.05. Statistical analyses were done by the SPSS software.

Results

In our study, the mean serum IL-1α level in AA patients was 4.34±0.86 pg/mL (mean±SD), with 20% of variation and range 3.40 to 7.10 pg/mL (Tabl. I). Patients with shorter duration of the disease had higher concentration of IL-1α, but not significantly (p>0.05). Correlation between the duration of the AA and concentration of IL-1α: r = +0.273; ρ(rho)=0.097; 95%C.I. (-0.160; 0.343); p>0.05; n.s. Among controls, IL-1α mean is 3.66 pg/mL, with 9% of variation, standard deviation was ±0.35 and range 2.60 to 4.30 pg/mL. Statistical analysis showed significant differences between IL-1α values of patients with AA and healthy controls (p=0.006).
The mean serum level IL-1β in AA patients was 2.35±0.17 pg/mL (mean±SD), with 7% of variation and range 2.10 to 2.60 pg/mL. There was no correlation between the duration of AA and serum IL-1β concentration: r =0.196; ρ(rho)=0.177; 95%C.I. (-0.080; 0.413); p>0.05; n.s. Among controls, IL-1β mean is 2.24 pg/mL, with 13% of variation, standard deviation was ±0.30 and range 1.90 to 3.20 pg/mL. IL-1β levels were greater in patients with AA than controls, but the difference was not significant (p=0.3137).

	Alopecia areata	Control group
Number of patients	46	20
Age (year; mean±SD)	36.5±16.5	32.6±16.1
IL-1α (mean±SD)	4.34±0.86	3.66±0.35
IL-1β (mean±SD)	2.35±0.17	2.24±0.30

Table I. Characteristics of all patients in the study

Discussion

Although the pathogenesis of AA is still poorly understood, a perifollicular, peribulbar and perivascular accumulation of T lymphocytes provide evidence that an immune process is involved, interfering with the hair cycle and leading to reversible hair loss. Recent progress in the understanding of AA has shown that the regulation of local and systemic cytokines play an important role in its pathogenesis [9,10]. In their study, Harmon and Nevis investigated the effects of IL-1α on hair follicle growth and hair fiber production in vitro [11]. They found that incubation with IL-1α resulted in a significant inhibition of DNA synthesis, rapid antiprolliferative effect on hair follicle and inhibition of hair fiber growth.

These observations suggest that IL-1 may play a role in the pathogenesis of AA through direct inhibitory effects on the hair follicle, in addition to its putative proinflammatory role mediated by stimulation of cells of the immune/inflammatory system. Additionally, experiments in cultured human hair follicles by Hoffmann et al. showed that IL-1β completely abrogated hair growth [12]. In vivo studies have shown that IL-1α protects hair follicle from the cytotoxic effects of chemotherapy agents and it has been suggested that this protection may occur as a result of inhibition of hair follicle matrix cell division by IL-1α [13,14]. In vitro studies have shown that IL-1α along with IL-1β and TNF-α, causes vacuolation of matrix cells within the follicle bulb and a decrease in the size of the matrix, as well as disorganization of follicular melanocytes and abnormal differentiation and keratinization of the precortal cells and the inner root sheath [15]. These changes in hair follicle morphology are similar to those reported in AA and suggest that IL-1α and IL-1β may play an important part in the pathophysiology of inflammatory hair disease. Tarlow et al. demonstrated an association between the severity of AA and the frequency of allele 2 of a 5 allele polymorphism in intron 2 of the interleukin 1 receptor antagonist gene [16]. IL-1 gene polymorphysms may be responsible for exaggerated release of IL-1, leading to rapid and more progressive disease. This allele is also associated with severity in other chronic inflammatory autoimmune disorders including systemic lupus erythematosus and psoriasis [17].

In addition, increased serum levels of IL-1α in patients with AA compared with normal controls has been reported, further suggesting a role for this cytokine. The results presented in our study demonstrated that the mean serum levels of IL-1α were significantly elevated in AA patients in comparison to healthy subjects (4.34±0.86 pg/mL vs 3.66±0.35 pg/mL, respectively). Patients with shorter duration of the disease had higher concentration of IL-1α, but not significantly. These results are consistent with a clinical study performed by Teraki et al. [18]. They also recorded a significant increase in serum IL-1α in patients with AA, and there was an inverse correlation between disease duration and the serum levels of IL-1α. After that, Barahmani et al. analyzed serum cytokine profiles in 269 patients with AA and found it that increased IL-1α levels is associated with AA and atopy [19].

A limited number of studies in the literature have evaluated the serum levels of IL-1β in patients with AA. In the study of Nada et al. serum levels of IL-1β in patients with AA did not differ from that in controls [20].

The importance of serum cytokines in dermatology is increasing dramatically. We think that the high serum levels of IL-1α indicate the activation of the immune system in AA and may have a patophysiological role in the disease. Further investigation are required to clarify the pathogenetic role and clinical significance of IL-1, and these findings may provide important clues to assist in the development of new therapeutic strategies for patients with AA.

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A STUDY OF NAIL CHANGES IN VARIOUS DERMATOSIS IN PUNJAB, INDIA

ZMIANY PAZNOKCIOWE W RÓŻNYCH DERMATOZACH W PUNJAB, INDIA

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Abstract
Nails act as a window to diagnosis of skin diseases. Various dermatosis affect the nails and the severity of the skin disorder is reflected in the nails. Nail changes are seen in various dermatosis like psoriasis, lichen planus, onychomycosis, collagen vascular disorders, vescicobullous disorders and other papulosquamous disorders. We will discuss in detail regarding nail changes in various dermatosis.

Streszczenie
Paznokcie są jak okno w diagnostyce chorób skóry. Różne dermatozy wpływają na paznokcie a ciężkość choroby skóry ma swoje odbicie na stanie paznokci. Zmiany w paznokciach widoczne są w różnych dermatozach takich jak łuszczyca, liszaj płaski, grzybica paznokci, kolagenowa choroba naczyń krwionośnych, zaburzenia pęcherzowe i inne zaburzenia grudkowo-złuszczające. Omówimy szczegółowo zmiany paznokci w różnych dermatozach.

Key words: nail disease; psoriasis; onychomycosis; lichen planus
Słowa klucze: choroby paznokcia; łuszczyca; onychomycosis; liszaj płaski

Introduction

Nail disorder comprises approximately 10% of all dermatological condition [1,2]. Any portion of the nail unit may get affected by various dermatological condition, systemic disease, infections, ageing process, internal and external medication, vascular insufficiency, physical and environmental agents, trauma, neurological abnormalities, nutritional deficiency and both benign and malignant tumour [3]. Various nail abnormalities result in pain or interference with functioning or both. Nail disorder may affect walking, picking up of fine objects and protective function. The increasing emphasis on the aesthetic consideration in dermatology means even the slightest nail change may assume significance for the patient [4]. Abnormal nails are of utmost clinical importance, especially when they are the only presenting feature without any other apparent signs and symptom of a disease. Hence nail provides us insight of window looking through which one can establish the diagnosis. Various dermatological conditions that characteristically involve the skin and hair may also involve the nail. The following is the classification of nail disorders:

1. **Genetic disorders:** Epidermolysis bullosa, congenital onychodysplasia of index finger, Racket nail, Dolichonychia, ichthyosis, incontinentia-pigmenti, acrodermatitis enteropathica.
2. **Nail changes in infections:** Various fungal, bacteria, viral, spirochete, yeast, HIV infection, leprosy may affect the nail.
3. **Nail changes in dermatological conditions:** Lichen planus, psoriasis, eczema, alopecia areata, vitiligo and pemphigus vulgaris.
4. **Nails in systemic conditions:**
- Cardiovascular diseases.
- Impaired peripheral circulation.
- Renal diseases: nephrotic syndrome.
- Respiratory diseases: tubercular empyema.
- Endocrine disorder: hypothyroid, hyperthyroid, diabetes mellitus.
- Gastrointestinal and hepatic disorders.
5. **Nail deformities due to trauma:** Nail biting, nail picking, habit-tic deformity, Heller's dystrophy, hang nails and ill fitting shoes.
6. **Occupational nail changes:** Rickshaw pullers, housemaids.
7. **Neoplasm of nails:**
Benign: like glomus tumour, myxoid cyst, periungual fibroma.
Malignant: Malignant melanoma and squamous cell carcinoma.
8. **Drug induced nail changes.**

9. **Cosmetics induced nail changes.**
10. **Nail changes in:** Children, elderly, pregnancy.

Aims

To study the abnormal nail changes in patients coming to the Department of Dermatology.

Material and Methods

For the present study, 500 patients with nail changes coming for various dermatological conditions was selected from the Department of Dermatology. A detailed clinical history regarding onset, duration and associated symptoms was asked. A thorough systemic and dermatological examination was conducted and all details were recorded on a special proforma. Routine investigations like Hb, TLC, DLC, ESR, platelet count, urine complete examination, blood urea, and serum creatinine were carried out to confirm the diagnosis. Special investigations like nail clipping for bacteriological and fungal infection, nail biopsy and skin biopsy were carried out whenever required.

Results

The data was collected, analysed and the following results were obtained.

I. Age Distribution

The above table shows that maximum number of patients with nail changes (40%) were in the age group of 21-40 years, followed by 30% in the age group of 41-60 years, 20% were less than 20 years and 10% were in the age group 61-80 years.

Sr No	Age	No. of cases	Percentage (%)
1	< 20	100	20
2	21-40	200	40
3	41-60	150	30
4	61-80	50	10

Table I. Incidence of nail changes among different age groups

II. Sex Distribution

The above table shows that out of 100 patients, 52% were males, while 48% were females. Male to female ratio was 1.08 : 1.

Sr No	Age	No. of cases	Percentage (%)
1	Males	260	52
2	Females	240	48
	Total	500	100

Table II. Sex distribution of patients with nail changes

III. Occupational Status

Above table shows majority of cases i.e. 34% with nail changes were housewives, whereas 30% of cases were in service or business, 12% were students and 24% were labourers or farmers.

Occupational status	No. of cases	Percentage (%)
Housewives	170	34
Service\business	150	30
Students	60	12
Labourers\Farmers	120	24

Table III. Occupational status of patients with nail changes

IV. Number of Nails Involved

Above table shows that majority of cases i.e. 38% had 6-10 number of nail involvement, 35% patients had 1-5 number of nail involvement,18% patients had 16-20 number of nail involvement and 9% patients had 11-15 number of nail involvement.

No. of nails	No. of cases	Percentage (%)
1-5	175	35
6-10	190	38
11-15	45	9
16-20	90	18
Total	500	100

Table IV. Number of nails involved

V. Nail changes in various dermatosis

The above table shows that majority of cases were of onychomycosis (25%), followed by psoriasis (20%), eczema (20%), paronychia (8%), lichen planus (5%) and dariers disease (4%), to name a few.

Sr No	Dermatosis	No. of cases	Percentage (%)
1	psoriasis	100	20
2	eczema	50	10
3	tinea unguim	125	25
4	lichen planus	25	5
5	paronychia	40	8
6	alopecia	5	1
7	secondary syphilis	4	0.8
8	leprosy	10	2
9	HIV	10	2
10	systemic sclerosis	10	2
11	pemphigus	5	1
12	drug induced	30	6
13	epidermolysis bullosa	2	0.4
14	periungual warts	15	3
15	atopic dermatitis	11	2.2
16	Dariers disease	20	4
17	PRP	7	1.4
18	pachyonychia congenita	1	0.2
19	twenty nail dystrophy	20	4
20	nail changes due to trauma	5	1
21	vitiligo	5	1
	total	500	100

Table V. Nail changes in various dermatosis

VI. Nail changes in psoriasis

It is clear from table VI, that pitting was the most common finding in psoriasis, accounting for 70 % cases. Next most common nail changes were subungual hyperkeratosis in 40% and onycholysis in 52% cases. Discoloration was found in 25% cases followed by paronychia in 10% cases. Splinter haemorrhages were seen in 12% and Beau's lines were observed in 14% cases salmon patches in 10 % cases, longitudinal ridging in 12% cases, longitudinal melanonychia in 4% cases, perilunular erythema/red lunules in 5% cases and twenty nail dystrophy in 3% cases.

Nail changes	No. of cases	Percentage (%)
Pitting	70	70
Subungual hyperkeratosis	40	40
Onycholysis	52	52
Discoloration	25	25
Paronychia	10	10
Splinter haemorrhage	12	12
Beau's line	14	14
Salmon patches	10	10
Longitudinal ridging	12	12
Dystrophy	6	6
Longitudinal melanonychia	4	4
Perilunular erythema/red lunules	5	5
Twenty nail dystrophy	3	3

Table VI. Nail changes in psoriasis (n = 100)

VII. Nail changes in lichen planus

The above table shows that longitudinal ridging was the most common finding accounting for 24% cases. Next most common nail changes were pterygium in 16% and onycholysis in 16% cases. Longitudina melanonychia was found in 20% cases followed by dystrophy in 4% cases. Twenty nail dystrophy was seen in 8% and subungual hyperkeratosis was observed in 12% cases.

Sr No	Nail changes	No. of cases	Percentage (%)
1	pterygium	4	16
2	longitudinal melanonychia	5	20
3	longitudinal ridging	6	24
4	onycholysis	4	16
5	dystrophy	1	4
6	subungual hyperkeratosis	3	12
7	twenty nail dystrophy	2	8

Table VII. Nail changes in lichen planus (n = 25)

VIII. Types of Onchomycosis

The above table shows that longitudinal ridging was the most common finding accounting for 24% cases. Next most common nail changes were pterygium in 16% and onycholysis in 16% cases. Longitudina melanonychia was found in 20% cases followed by dystrophy in 4% cases. Twenty nail dystrophy was seen in 8% and subungual hyperkeratosis was observed in 12% cases.

Sr No	Type	No. of cases	Percentage (%)
1	distal lateral sub ungual onchomycosis	93	74.4
2	superficial white onchomycosis	5	4
3	proximal sub ungual onchomycosis	2	1.6
4	total dystrophic onchomycosis	25	20

Table VIII. Types of onchomycosis (n = 125)

IX. The etiologic distribution of twenty nail dystrophy

The above table shows that the commonest cause of TND was idiopathic (45%). Other causes of TND were psoriasis in 25% cases, lichen planus in 20% cases and alopecia areata was seen in 10% cases.

Sr No	Type	No. of cases	Percentage (%)
1	psoriasis	5	25
2	lichen planus	4	20
3	alopecia areata	2	10
4	idiopathic	9	45
5	Total	20	100

Table IX. Nail changes in twenty nail dystrophy

X. Nail changes in paronychia

The above table shows that absent cuticles and nail fold inflammation were the commonest nail changes seen in all the cases, discoloration in 70% cases, transverse grooves in 50% and onycholysis in 40% cases.

Sr No	Nail Changes	No. of cases	Percentage (%)
1	absent cuticles	40	100
2	nail fold inflammation	40	100
3	subungual hyperkeratosis	6	15
4	onycholysis	16	40
5	discoloration	28	70
6	longitudinal striations	2	5
7	transverse grooves	20	50
8	nail dystrophy	2	5

Table X. Nail changes in paronychia

Discussion

Nail disorders are seen in various dermatosis like fungal infection, psoriasis, lichen planus, vescicobullous and collagen vascular disorders.

Onychomycosis represents a broad term for any fungal infection of any part of the nail unit by dermatophytes, molds or yeast [5].

Onychomycosis caused by dermatophytes is also called as tinea unguium. Fungal infection of nail may be classified as [6]:

1. Distal subungual onychomycosis primarily involves the distal nail bed and hyponychium.
2. Superficial white Onychomycosis is an invasion of the surface of the nail plate.
3. Proximal subungual onychomycosis involves the nail plate from the proximal nail fold.
4. Candidal onychomycosis involves all the nail plates [7,8,9]. There is true invasion of nail plate by candida albicans resulting in dystrophic nail. It occurs in patients with chronic mucocutaneous candidiasis.

Nail involvement of one or all the nail component occur in 10% of patient with lichen planus [10,11]. Severe inflammatory focus in the nail matrix, leads to adhesion formation between epidermis of proximal nail fold and nail bed and result in pterygium formation, which is highly suggestive of LP. Other less common features include onycholysis, shedding of the nail, subungual hyperkeratosis, erythematous patches in the lunula, koilonychia, pitting and nail discolouration may also occur [12,13,14]. Psoriasis is a common disease affecting nails with subsequent dystrophy. Nail involvement has been reported up to 50% of case [15,16], but over a life time, the incidence cumulatively increases to 80-90%. In order of decreasing frequency, nail changes of psoriasis are pitting, onycholysis, subungual hyperkeratosis, nail plate discolouration, uneven nail surface, splinter haemorrhages [2] and lastly acute and chronic paronychia [17,18,19]. Nail changes are common in alopecia areata, ranging from 7% to 66% [20]. Nail changes are not only seen in extensive alopecia areata but may also be present with minimal hair loss and does not imply a poor prognosis for regrowth. Uniform pitting is the most common abnormality seen in alopecia areata. Pits are often uniformly arranged in lines both transversely and longitudinally in a geometrical or scotch plaid pattern [21]. Other nail changes include ridging, onychorrhexis, beau's lines or transversely arranged pits, thinning or occasionally thickening of the plate, koilonychia, onychomadesis leading to nail shedding, leukonychia punctata due to nail bed dystrophy and lunules may be red or mottled. Round finger pad sign could be the early sign of scleroderma. Pterygium inversum unguis may be the helpful diagnostic sign in scleroderma [22]. It is characterized by obliteration of the distal groove due to adherence of the distal portion of the nail bed to the ventral surface of the nail plate. Other nail signs like onycholysis, longitudinal ridging, onychorrhexis, onychogryphosis, haplonychia, longitudinal striation, absent lunulae, periungual vesiculation has been reported in scleroderma. Parrot beak deformity is another distinctive feature of the disease characterized by over curvature of the free margins of the nail over a shortened finger tip. It is due to atrophy of the soft tissue. Twenty nail dystrophy (TND) is a condition in which all twenty nails are uniformly and simultaneously affected [23,24]. Earlier it was called as excessive ridging of childhood or Trachonychia [25]. TND can be idiopathic, congenital or acquired [26]. The acquired type may be related to variety of disorders like lichen planus, psoriasis, alopecia areata, ichthyosis vulgaris, eczema and perhaps Pemphigus. In our study, out of 500 patients,nail changes were seen in various dermatosis. Maximum number of patients (25%), were of onychomycosis (Fig. 1) followed by 20% patients of psoriasis (Fig. 2), 20% patients of eczema, 8%, patients were of paronychia (Fig. 3), 5% patients of lichen planus (Fig. 4) and 4% patient were of dariers disease to name a few. We had one patient of tuberous sclerosis with koenens tumour (Fig. 5). Out of 100 patients of psoriasis the most common changes were pitting, subungual hyperkeratosis, onycholysis and discoloration. Out of 125 cases of lichen planus, the most common changs were longitudinal ridging, pterygium and onycholysis. Twenty nail dystrophy was seen in 20 cases and the commonest cause of twenty nail dystrophy was idiopathic in 45% cases,psoriasis in 25% cases, lichen planus in 20% cases and alopecia areata was seen in 10% cases.

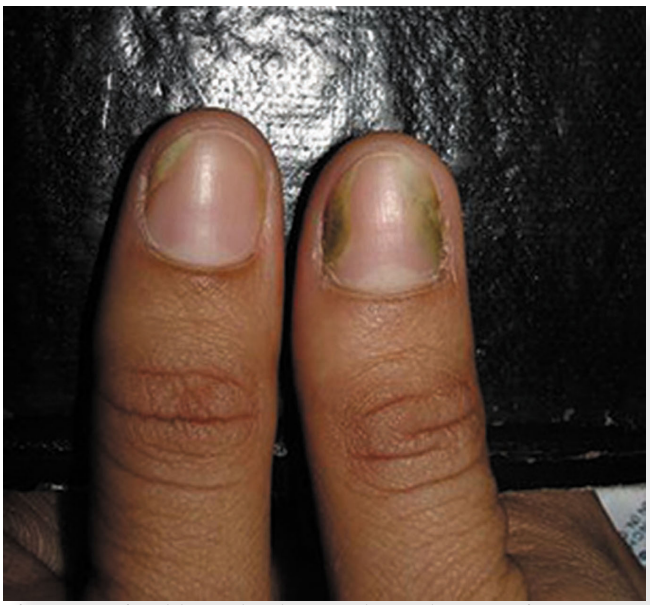

Figure 1. Distal lateral subungual onychomycosis

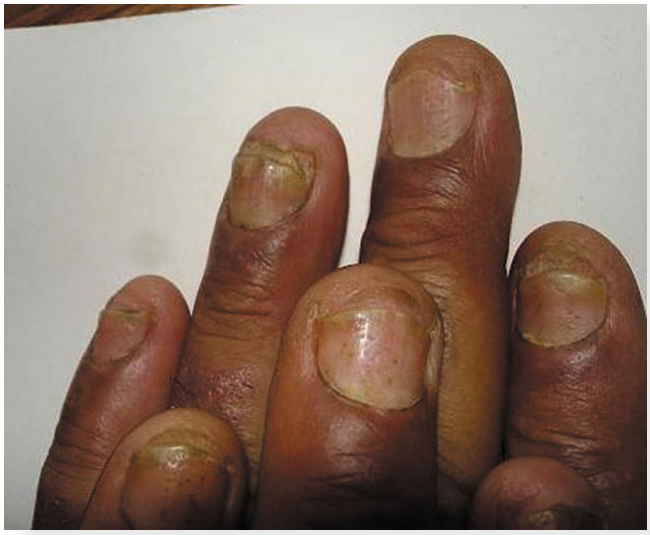

Figure 2. Pitting and onycholysis in a psoriasis

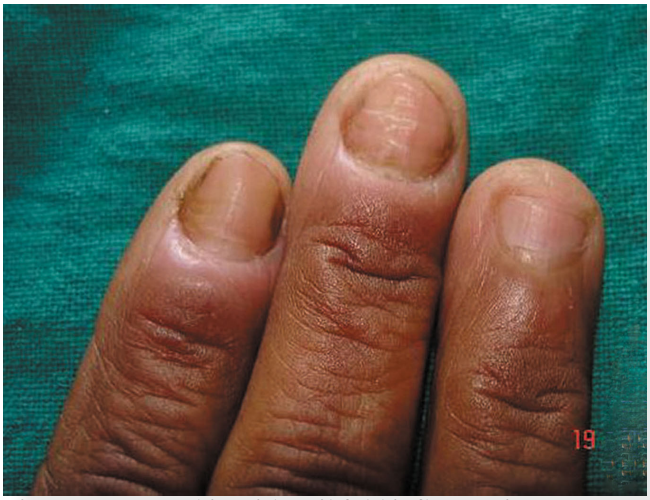

Figure 3. Paronychia with nail fold inflammatio

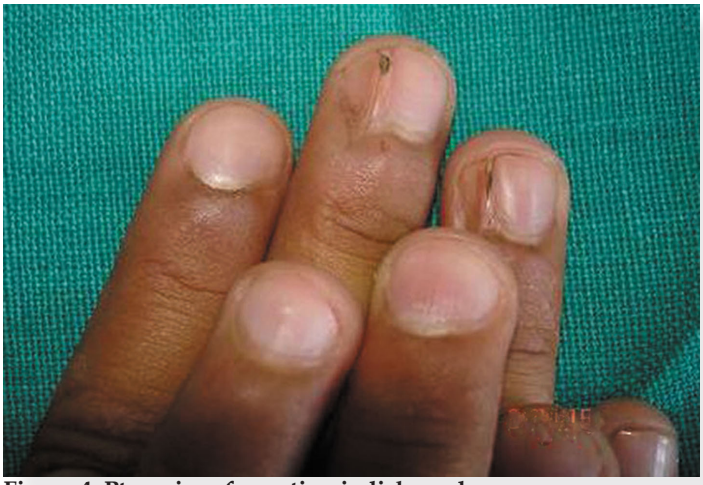

Figure 4. Pterygium formation in lichen planus

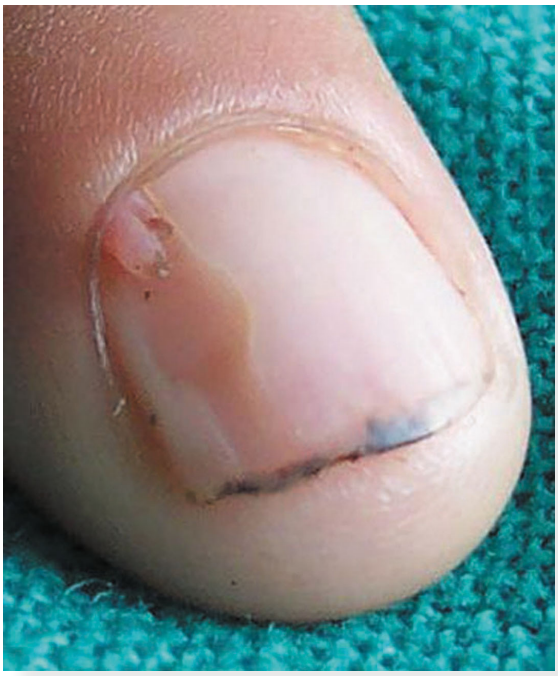

Figure 5. Koenens tumour

Conclusions

From the foregoing account, it can be concluded that a variety of nail changes can occur in various dermatological, systemic and other conditions. The nail unit is capable of only a limited number of reaction patterns, therefore, many diseases share similar changes, but correlation of the nail changes helps dermatologist to reach conclusive diagnosis.
In order to evaluate the nail changes skillfully one must be familiar with the terminology and classification of the nail disorders. Thus knowing the normal and abnormal variants of the nail and their association with wide range of disease is beneficial not only for the establishing diagnosis but also for the specific management of the disease. Hence, no physical examination is complete without the study of nails. However, nails remain an understudied and yet quiet accessible structure that lends itself for examination and evaluation. Hence truly said that nails are the windows through which one can look into the health of the patients.

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A STUDY OF NAIL CHANGES IN VARIOUS DERMATOSIS IN PUNJAB, INDIE

by Neerja Puri, Tejinder Kaur

comment:
Dr. Leonel Fierro, Dr. Alexandro Bonifaz

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The article by Drs Puri and Kaur communicate in a clear and consistent way the various changes that can be found on the nails and their relationship with other dermatoses. As indicated in the beginning, the nails are a „window" to suspect the presence of other associated diseases and to establish diagnostic proposals. Understanding the nail as a „functional unit", the range of entities that the clinician faces is extremely varied, hence the daily practice of dermatologists may encounter inflammatory, autoimmune, infectious, pharmacologic, traumatic and tumoral conditions. Moreover nails reflect signs associated with other dermatoses or with systemic diseases that have no impact to another cutaneous level. The authors obtain a sufficient population to identify some characteristics associated with nail pathology described as: age range, occupation, number of nails involved, diagnosis and some specific findings in states such as nail psoriasis. Based on our experience it is worth noting the contrast at two points in particular, the first is on the prevalence of onychomycosis in our population that exceeds 50% of the cases and is the most common pathology at this level, being the first complaint of nail disease. The second observation is that we have identified that age group with the greatest impact of nail pathology is over 40 years and differs from that discussed in the communication. This may be because in our patients, high rates of associated systemic diseases such as diabetes mellitus and other local processes such as vascular illness, heart disease, tinea pedis, poor hygiene and bad nutrition are factors that may impact adversely on their health, increasing the number of cases with these characteristics. Auxiliary diagnostic techniques currently available as dermatoscopy, laboratory tests and biopsies, are useful tools for more accurate conclusions in the pathology of the nail. Finally there will note the importance of tackling the nail apparatus associated tumors of which the variety of benign strain injuries require experience to establish the diagnostic suspicion and especially melanonychia should be approached with grater care while maintaining the intention of discarding the presence of a malignant melanoma of the serious risks involved. This paper invites in a friendly and clear form to the comprehensive approach on nail diseases, taken into account comorbidities and other conditions. As the article concludes it is important to successful integration and diagnostic correlation, so we can offer the best chance for patients to receive better quality care.

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ONYCHOMYCOSIS - A CLINICAL AND MYCOLOGICAL STUDY OF 75 CASES

GRZYBICA PAZNOKCI - KLINICZNE I MIKOLOGICZNE BADANIE NA 75 PRZYPADKACH

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Abstract
Onychomycosis or fungal infection of the nails is a common disease, especially in older persons. A mycological study of onchomycosis was undertaken in 75 patients. The nails were judged to be infected by their clinical appearance. There were a total of 75 suspected cases of onychomycosis. Of these 75 cases 22.6% were positive by direct microscopy and 33.3% were culture positive. Of these 75 cases, 18 were males (24%) and 57 (76 %) were females, male to female ratio being. The commonest age group was 31-40 years followed by 21-30 years. The finger nails were more frequently involved. i.e. 45 (60 %), followed by toe nails 30 (40 %) and both in 18 (24%) cases. Ratio of finger nail to toe nail infection was 1.5:1. Distal and lateral subungual onychomycosis (DLSO) was the commonest clinical pattern (76%) followed by total dystrophic onychomycosis (18.66%) and then superficial white onychomycosis (4%) and proximal subungual onychomycosis (1.33%). The most common fungal isolates were dermatophytes of which 44% were Trytophyton rubrum, 4% were Trytophyton mentagrophytes. Non dermatophyte moulds constituted 16% of the fungus isolates. Onychomycosis was found to be the commonest in housewives (52%), followed by serviceman / businessman (32%) followed by farmers (8%) and labourer and student 4% each.

Streszczenie
Grzybica paznokci lub zakażenie grzybicze paznokci jest częstą chorobą, zwłaszcza u osób starszych. Mykologiczne badanie paznokci zostało przeprowadzone u 75 pacjentów. Paznokcie uznano za zakażone na podstawie ich objawów klinicznych. Było w sumie 75 podejrzanych przypadków grzybicy paznokci. Z tych 75 przypadków 22,6% były pozytywne w badaniu bezpośredniej mikroskopii, a u 33,3% była pozytywna hodowla. Z 75 przypadków, 18 stanowili mężczyźni (24%), a 57 (76%) stanowiły kobiety. Stosunek mężczyzn do kobiet jest istotny. Najczęstsza grupa wiekowa to pacjenci w wieku 31-40 lat, następnie 21-30 lat. Paznokcie palców rąk były częściej objęte procesem zapalnym, tj. 45 (60%), następnie paznokcie palców stóp 30 (40%) i zarówno palców rąk jak i stóp w 18 (24%) przypadkach. Stosunek zainfekowanych paznokci rąk do paznokci stóp wynosił 1.5:1. Dystalna i boczna podpaznokciowa onychomykoza (DLSO) była najczęstszym klinicznym wzorem (76%), a następnie całkowitej dystroficzna onychomykoza (18,66%), powierzchowna biała onychomykoza (4%) i proksymalna pozpaznokciowa onychomykoza (1,33%). Najczęstszą izolowaną infekcją grzybiczą były dermatofity z których 44% stanowili Trytophyton rubrum, 4% stanowił Trytophyton mentagrophytes. Formy niedermatofitów stanowiły 16% izolatów grzybiczych. Grzybica paznokci została uznana za najczęstszą wśród gospodyń domowych (52%), u żołnierzy / biznesmenów (32%), a następnie u rolników (8%) oraz robotników i studentów po 4%.

Key words: fungi; onychomycosis; nails; culture; infection; dermatophytes
Słowa klucze: grzyby; grzybica paznokci; paznokcie; kultura; zakażenie; dermatofity

Introduction

Onychomycosis refers to the invasion of the nail plate by a fungus [1]. The infection may be due to a dermatophyte, yeast or non dermatophyte mould [2,3]. Predisposing factors of this disease includes presence of positive history of onchomycosis, increasing age, trauma to the nail, diabetes, immunosuppression, poor peripheral circulation and tinea pedis [4]. Perhaps 50% of all nail diseases are caused by fungi that invade the nail unit through the nail bed or nail plate. Often, more than one type of organism is involved. Most cases of onychomycosis in the United States are caused by dermatophytes, but nondermatophyte fungi (molds or yeasts) may also serve as causative agents [5].

Onychomycosis can be classified into 4 types according to the pattern of infection [6,7]. Distal subungual onychomycosis, the most common type, affects the distal portion of the nail bed and the underside of the nail. In proximal white subungual onychomycosis, the fungus enters through the cuticle to invade the proximal portion of the nail bed. The nail plate turns white proximally near the cuticle.

This type is common in immunosuppressed patients, especially those with human immunodeficiency virus infection. In both subungual types, *Trichophyton rubrum* is the most common causative organism. In white superficial onychomycosis, found mostly in the toenails of otherwise healthy individuals, direct fungal invasion of the nail plate surface, usually by Trichophyton mentagrophytes, produces a white, crumbly appearance. The fourth type, Candida & onychomycosis, is usually caused by *Candida albicans* and has three subtypes. Candida paronychia, the most common of the three, is marked by swelling and erythema of the proximal and lateral nail folds. In Candida onychomycosis, the nail plate separates from the nail bed. It is characterized by direct invasion and thickening of the nail plate and associated paronychia. This type of yeast infection occurs mostly in immunocompromised patients. Progression of any of the four types so that the entire nail unit becomes involved is known as total dystrophic onychomycosis [7,8].

Aims And Objectives

The aim of our study was:

1. To determine incidence of various types of onchomycosis
2. To find out the occupational consequences related to onchomycosis
3. To find out the fungal infection positivity in nail clippings.
4. To isolate the causative fungal pathogen using fungal nail cultures.

Materials and Methods

The study population comprised of 75 suspected cases of onchomycosis attending the dermatology, outpatient department. Nail scrapings / clippings were obtained according to standard procedures. Detailed history of trauma, infection, occupation, diabetes, personal habits (smoking etc.) were taken. Different clinical patterns (DLSO - Distal and lateral subungual onychomycosis, PSO - Proximal subungual onchomycosis, SWO - White superficial onchomycosis, TDO - Total dystrophic onchomycosis) were recorded separately. All specimens were subjected to direct microscopy in 20% KOH solution for the presence of fungal mycelia and spores. Direct microscopy was used to diagnose whether the fungi were present or absent. Nail scrapings and clippings were inoculated on antibiotic containing Sabourand dextrose agar with and without cycloheximide at 27°C and at 37°C. For culture, 1 of the 2 media contained cycloheximide, which inhibits the growth of many nondermatophytes. Growth on both media suggests a dermatophyte; growth on only the medium without cycloheximide may suggested a nondermatophyte. Treatment was initiated on the results of direct microscopy but was adjusted once the laboratory results were available. The fungal growth was identified by standard procedures. At least three samples from each patient were processed.

Those who grew dermatophytes were classified as dermatophytosis patient. Those who grew a particular mould other than dermatophyte consistently on two or more successive occasions with consistent filaments by direct microscopy at least once and continued to grow the same mould consistently thereafter from the same nail and without growing a dermatophyte on any occasion were classified as opportunistic onchomycosis patient. Those who grew a dermatophyte on one or more occasions and also grew a mould with the same consistency, site specificity and direct microscopic variability as indicated for opportunistic onychomycosis, were classified as mixed infection patients. Because of difficulty in discerning pathogens from contaminants, the guidelines followed were: I). If a dermatophyte was isolated on culture, it was a pathogen, 2) if a nondermatophyte mould (NDM) or yeast was cultured, it was significant only if direct microscopy was positive and 3) NDM required repeated isolation.

Proper specimen collection is essential to accurate diagnosis. First, the nail area was cleansed with alcohol. Then, for distal subungual onychomycosis, the abnormal nail was clipped proximally and the nail bed and underside of the nail plate are scraped with a l-mm curette; the outermost debris was discarded. For proximal subungual onychomycosis, the normal surface of the nail plate was pared down at the lunula and the white debris was collected from the deeper portion of the plate. For white superficial onychomycosis, the white spots on the nail were scraped and the outermost surface was discarded; the white debris directly underneath was then collected. For Candida infection, the material closest to the proximal and lateral nail edges was obtained. In suspected candidal oncholysis, after fifting the nail bed, the undersurface of the nail plate was scrapped. For distal dystrophic onchomycosis, any abnormal area of the nail plate or bed was used as a specimen.

Results (Tabl. I-IX)

The data was collected, analysed and the following results were obtained.

Age group	Number of cases		Total	% Age
	Male	Female		
<20	0	3	3	4%
21-30	4	16	20	26.6%
31-40	9	19	28	37.2 %
41-50	3	12	15	20%
51-60	0	8	8	10.6%
> 60	1	0	1	1.3%

Table I. Age distribution in patients of onychomycosis

Sex	Number of cases	% Age
Male	18	24%
Female	57	76%
Total	75	100

Table II. Sex distribution in onychomycosis
Male: Female ratio = 1: 3.15

Sr No	Type	Number of cases	% Age
1	Distal lateral subungual onchomycosis	57	76%
2	Superficial white onchomycosis	3	4%
3	Proximal subungual onchomycosis	1	1.33%
4	Total dystrophic onchomycosis	14	18.66%

Table III. Types of onychomycosis

Nail changes	Distal lateral subungual onchomycosis N=57	Superficial white onchomycosis N=3	Proximal subungual onchomycosis N=l	Total dystrophic onchomycosis N=14	Total	% Age
Subungual hyperkeratosis	39	2	-	6	50	2.6%
Oncholysis	29	2	1	8	40	53.3%
Discoloration	57	3	1	14	75	100%
Long striature	31	2	-	6	40	53.3%
Transverse grooves	6	1	-	1	8	10.6%
Paronychia	14	2	1	3	20	26.6%
Rough nails	26	2	-	8	38	50.6%
Pitting	2	-	-	1	3	4%

Table IV. Nail changes in different types of onychomycosis (n= 75)

Sites	Number of cases	% Age
Toe nail infections	30	40%
Finger nail infections	45	60%
Both Finger and Nail infections	18	24%

Table V. Incidence of toe and finger nail infections (n= 75)
Finger Nail : Toe nail infections = 1.5: 1

	Before treatment		After treatment	
	Number of cases	% Age	Number of cases	% Age
Nail for Fungus positivity	17	22.6%	3	4%
Fungal nail culture positivity	25	33.3%	5	6.6%

Table VI. Nail fungus and fungal nail culture positivity before and after treatment

Sr No	Occupational status	Number of cases	% Age
1	Housewife	39	52%
2	Farmer	6	8%
3	Labourer	3	4%
4	Student	3	4%
5	Service/ Businessman	24	32%

Table VII. Association of onychomycosis with different ocupations

Number of nails	Number of cases	% Age
1-5	18	24%
6-10	32	40.24%
1-15	11	14.6%
16-20	14	18.6%
Total	75	100

Table VIII. Number of nails involved in onychomycosis

Fungal species	Number of cases	% Age
Trichophyton rubrum	1	44%
Candida albicans	5	20%
Other trichophyton species (Trichophyton mentagrophytes)	1	4%
Mixed infections	4	16%
Non Dermatophyte mould (Penicillium)	4	16%
Total	25	100

Table IX. Organisms involved in fungal nail culture

Discussion

There were a total of 75 suspected cases of onychomycosis. Of these 75 cases 22.6% were positive by direct microscopy and 33.3% were culture positive. Of these 75 cases, 18 were males (24%) and 57 (76 %) were females, male to female ratio being. The commonest age group was 31-40 years followed by 21-30 years. The finger nails were more frequently involved. i.e. 45 (60 %), followed by toe nails 30 (40 %) and both in 18 (24%) cases. Ratio of finger nail to toe nail infection was 1.5:1. Distal and lateral subungual (Fig. 1) onychomycosis (DLSO) was the commonest clinical pattern (76%) followed by total dystrophic (Fig. 3) onychomycosis (18.66%) and then superficial white (Fig. 2) onychomycosis (4%) and proximal subungual (Fig. 4) onychomycosis (1.33%). The most common fungal isolates were dermatophytes of which 44% were *Trichopchyton rubrum*, 4% were *Trichopchyton mentagrophyte*. Non dermatophyte moulds constituted 16% of the fungus isolates. Onychomycosis was found to be the commonest in housewives (52%), followed by serviceman/ businessman (32%) followed by farmers (8%) and labourer and student (4% each).

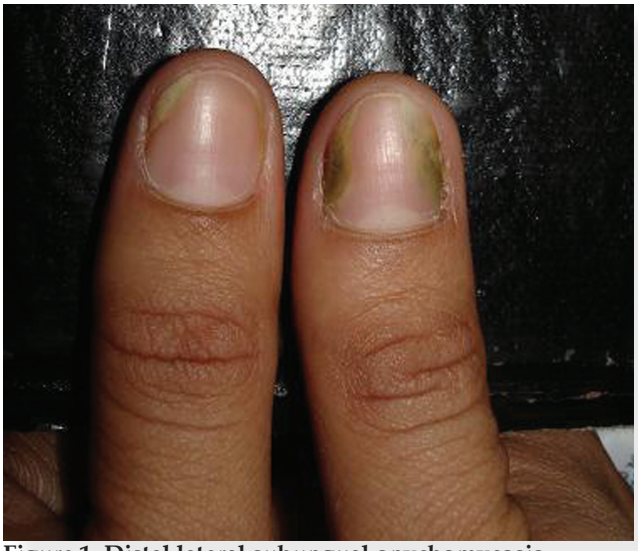

Figure 1. Distal lateral subungual onychomycosis

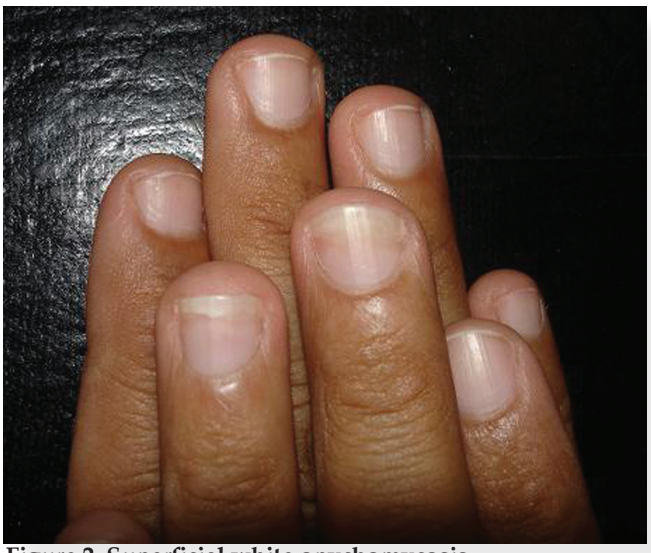

Figure 2. Superficial white onychomycosis

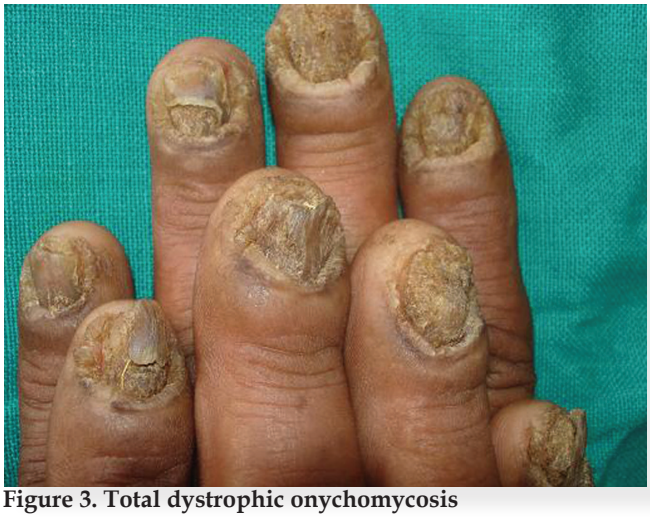

Figure 3. Total dystrophic onychomycosis

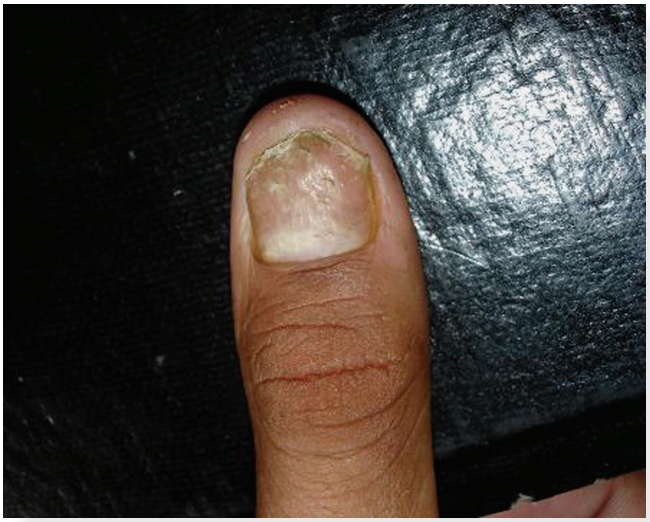

Figure 4. Proximal subungual onychomycosis

Onychomycosis, defined as fungal infection of nail affects approximately 5% of the population worldwide [9-12] and represents around 30% of all superficial mycotic infection and 500.10 of nail disorders. The infection has profound social consequences for the affected patients, who often have diminished confidence or self esteem and experience embarrassment in social and work situations. Onychomycosis in immunocompromised patients, such as those infected with human immunodeficiency virus (HIV), can pose a more serious health problem [13]. Over the recent years, an upsurge in cases of onychomycosis due to nondermatophytes has been documented. Of the nondermatophytic filamentous agents implicated in onychomycosis include members of *Scopulariopsis* and *Scytalidium* (the two most common genera), which are both thought to digest keratin in vivo, as well as members of the genera *Alternaria*, *Aspergillus*, *Acremonium* and *Fusarium*. The most common yeast that is involved in onychomycosis is *Candida albicans*. The fact that the infection is difficult to treat and treatment consists of prolonged courses of potentially toxic drugs makes it imperative to make an early and accurate diagnosis [14,15]. Diagnosis of onychomycosis depends on direct microscopy, supplemented by culture results [16]. Direct microscopy is often time-consuming, because nail debris is thick and coarse and hyphae are usually only sparsely present. Although direct microscopy can provide clues about the identity of the microorganism, careful matching of microscopic and culture results is necessary for the clinician to be confident of the diagnosis. Onychomycosis is a common infection of nails in adults and accounts for prevalence rate of 2 to 50% worldwide and the incidence increases with age [17,18]. In the present study, onychomycosis was found to be commonest in the age group 31-40 years in accordance with most of the studies. Our study reveals that incidence of onychomycosis is increasing with advancing age. Higher incidence was noted amongst females (76%) than males (24%), the ratio being 3.15 : 1, which compares well with most of the studies. Higher incidence in males may be because they are more exposed to household chores and their hands come in contact with water.

Various authors have reported high incidence of onychomycosis of the toenail. In the present study we have come across more cases of fingernail onychomycosis, than toenails with a ratio of 1.5:1, which compares well with other studies. Incidence of increased finger nail onychomycosis may be because of the increased chances of occupation related trauma, also fingernail infection is more likely than the toenail infection to arouse the patients concern, driving them to seek medical attention. In various studies, right thumb was the commonest fingernail involved. We observed that, ring finger and index finger were commonly involved. Greater toenail onychomycosis has been reported frequently, this is in agreement with other studies, because of its bigger size predisposing to increased trauma. The high incidence of DLSO pattern has been reported by various studies. Incidence of DLSO pattern was seen in 76% of our cases, which is comparable with most of the studies. In the present study anthropophilic dermatophytes have been isolated from 29.6% of culture positive cases which is comparable with various studies. *Trichophyton rubrum* was the common isolate i.e., 57.6% in accordance with other studies. *Candida albicans* is reported as the commonest cause of paronychial onychomycosis. This is reflected in our study where all the paronychial cases grew *Candida albicans* on culture. Previously regarded as contaminant, yeast is now increasingly recognized as pathogen in fingernail infections.To conclude, DLSO was the commonest clinical presentation in this study. Ttichophyton rubrum and Candida were major pathogens. This study also stresses the role of non dermatophyte moulds associated onychomycosis.

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CLINICAL SPECTRUM OF NEONATAL SKIN DISORDERS AT HAMDARD UNIVERSITY HOSPITAL KARACHI, PAKISTAN

KLINICZNE SPEKTRUM SKÓRY NOWORODKÓW W SZPITALU UNIWERSYTECKIM HAMDARD W KARACHI, PAKISTAN

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Abstract
Objective: To analyze the clinical spectrum of skin conditions in neonates at Hamdard university hospital. **Study Design:** Descriptive (Observational) cross sectional study.
Methods: This study was conducted from January 2008 to December 2009. All neonates seen at Hamdard university hospital during this period were examined. Neonates with skin conditions with in 28 days of birth were registered on a predesigned questioner by the house officer these cases were confirmed by the pediatric consultant, followed by detail physical systemic examination and skin examination. Dermatologist was involved in the diagnosis of difficult cases.
Results: Total numbers of new born seen during the year 2008- 2009 were 1660, there were 65% males and 35% females, 1360 (81.92%) were above 2.5 Kg at birth, 18.08% were below 2.5 Kg. Numbers of neonates with skin lesions were 577 (34.75%). Neonates with skin infections were 25.12%, 15.59% had with nappy rash and 15.59 % had erythema toxicum neonatrum. Neonates with milia were 60 (10.39%) and with erythema were 27 (4.67%).
Conclusion: Clinical spectrums of neonatal skin are different in this study as compared to other regional and international studies.

Streszczenie
Cel: Analiza kliniczna spektrum chorób skóry u noworodków w szpitalu Uniiwersyteckim Hamdard. **Projekt badania**: Opisowy (obserwacyjny) przekrój badania.
Metody: Badanie zostało przeprowadzone w okresie od stycznia 2008 do grudnia 2009 roku. W tym okresie zbadano wszystkie noworodki widziane w szpitalu uniwersyteckim Hamdard. Noworodki z chorobami skóry w 28 dni od urodzenia były zarejestrowane na gotowych kwestionariuszach przez lekarza rezydenta, następnie te przypadki zostały potwierdzone przez konsultanta dziecięcego, po szczegółowym systemowym badaniu fizykalnym i badaniu skóry. Dermatolog był zaangażowany w diagnostyce trudnych przypadków.
Wyniki: Całkowita liczba noworodków obserwowana w latach 2008 - 2009 wynosiła 1660, w tym 65% płci męskiej i 35% płci żeńskiej, 1360 dzieci (81,92%) było powyżej 2,5 kg po urodzeniu, 18,08% było poniżej 2,5 kg. Liczba noworodków z zmianami skórnymi wynosiła 577 (34,75%). Noworodków z zakażeniami skóry było 25,12%, 15,59% miało pieluszkowe zapalenie skóry a u 15,59% stwierdzono toksyczny rumień noworodków. Noworodków z prosakami było 60 (10,39%) a z rumieniem 27 (4,67%).
Konkluzja: Kliniczne spektrum skóry noworodków w tym badaniu różni się w porównaniu do innych badań regionalnych i międzynarodowych.

Key words: newborn; dermatology; dermatose
Słowa klucze: noworodek; dermatologia; dermatozy

Introduction

Children suffer from different dermatological conditions than adults.

Neonates form special group, the skin of the infant differs from that of the adult, in that it is thinner, delicate, has weaker intercellular attachments and produces fewer sweat and sebaceous gland secretions and is more susceptible to several infections [1]. Pediatric dermatological conditions accounted for large number of referrals [2].

Material and Methods

This study was carried out at Hamdard university hospital, an undergraduate teaching hospital All neonates born at hospital during Jan 2008 to Dec 2009 were included in the study, critically sick neonates on ventilator were not included in the study, a detailed history of the neonates' age

sex, maturity, birth weight, significant maternal history and mode of delivery was elicited. and a pretested questionnaire was used, data was collected by the house officer department of pediatrics, diagnosis was confirmed by consultant, difficult cases were discussed by the dermatologist and diagnosis confirmed by detail physical systemic examination and skin examination. SPSS 15 was used to determine frequencies and endnote for writing references.

Limitation of study: Sort duration of study two year, small number of cases, relation of skin lesions to preterm, term, post term neonates, and low birth weight / normal weight neonate was not studied.

Results

Total numbers of new born during the year 2008- 2009 were 1660 newborns.
Total numbers of male children were 1078 (65%), female neonates were 582 (35%).
Total numbers of children above 2.5 Kg birth weight were 1360 (81.92%).
Number of low birth weight were 300 (18.08%).
442 mothers were between the ages of 19 years to 30 years. (76.62%) 358 mothers delivered vaginally (62%) 219 mothers had instrumental delivery (38%) 352 mothers were given antibiotics 24 hours prior to delivery due to various reasons (61%).
Total numbers of neonates with skin lesions were 577 (34.75%) (Tabl I).
Numbers of neonates with skin infections were 145 (25.12%). neonates with nappy rash were 90 cases (16.0%). There were 120 cases of Mongolian spots (21%) 90 cases of erythema toxicum neonatrum were present in this study (16%) neonates with nappy rash were 90 (16.0%). Numbers of neonates with milia were 59 (10.22%), numbers of cases with erythema were 27 (4.67%), there were 25 cases of neonatal acne (4.33%) there were 10 cases of haemangioma (1.73%) there were 2 cases of café-eu-lail, seborrheic dermatitis, collodian baby one case of harlequin fetus, Epidermolysis bullosa, Sucking blister Scalded Skin Syndrome, Neonatal pustular melanosis.

Dermatological conditions	No of cases	Percentage
Infections	145	25.12 %
Mongolian spots	120	20.79%
Transient toxic erythemia	90	15.59 %
Nappy rash	90	15.59%
Milia	59	10.22 %
Erythema	27	4.67 %
Neonatal acne	25	4.33%
Hamangioma	10	1.73 %
Café- eu-lail	2	0.34 %
Collodian baby	2	0.34 %
Seborrheic Dermatitis	2	0.34%
Epidermolysis bullosa	1	0.17%
Sucking blister	1	0.17%
Scalded Skin Syndrome	1	0.17%
Neonatal pustular melanosis	1	0.17%
Harlequin fetus	1	0.17%
Total	577	

Table I. General distribution of skin conditions

Discussion

Neonates skin condition deserve special attention in hot humid, subtropical climate of Karachi, there is limited data on neonatal dermatology in Pakistan. Five year study by Maqbool S. Razzak S. [3] and Zahoorullaha [4] on skin disorders in children, there is no case report of neonatal skin lesion. Benton EC [5] has not reported a single case of neonatal dermatitis during 25 years of their study period. Some international studies have mainly focused on benign cutaneous lesions in newborns [6,7].
34.75% neonates had skin manifestations during the study period, reported incidence is 27.6% to 31% [8,9].
Maximum numbers of cases in this study were due to Skin infections (25.12%) this is particularly important because our neonates are over covered, in hot humid, subtropical climate. Reported incidence is between 5% to 47.15% [10-11].
There were 21% neonates with Mongolian spots, mainly on buttocks in this study; reported incidence is 56% to 98% [13-14].
There were 15.59% with nappy rash in this study; Ferahbas A et al [14] reported incidence of 2% this difference may be due to financial reasons, our mothers do not change nappies as frequently as required leading to prolong stool contact resulting in nappy rash.
Erythema toxicum neonatrum is the most common pustular dermatitis in newborns a benign condition requiring no intervention, presented in 15.50% of neonates in this study, reported incidence is 21-40% [15,16].

There were 10.22% neonates with milia in this study; reported incidence is 40-50% in other studies [17]. There were 4.33% reported incidence is 40% - 50% in other studies [18].

There were 1.73% neonates with Hemangioma in this study, Mishra PC, et al reported similar incidence [19]. 0.34% neonates presented as collodian baby, seborrheic dermatitis, and café – au-lait in this study. There was one case of Harlequin's fetus (0.17%). Sarkar reported an incidence of 0.11% [20] 0.17% neonates in this study had Epidermolysis bullosa, Sucking Blister Scalded Skin Syndrome, and Neonatal pustular melanosis. The pattern of skin infection in neonate is also different then other studies reported in regional and international studies [21-27].

Limitation of study: Sort duration of study two year, small number of cases, relation of skin lesions to preterm, term, post term neonates, and low birth weight / normal weight neonate was not studied.

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LOW DOSE PENICILLAMINE IN SYSTEMIC SCLEROSIS: IS IT EFFECTIVE?

NISKIE DAWKI PENICYLAMINY W TWARDZINIE UKŁADOWEJ: CZY SĄ EFEKTYWNE?

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Abstract
Low dose D-penicillamine 150mg was given on alternate days to 23 patients of limited cutaneous systemic sclerosis (lcSSC) and 5 of diffuse cutaneous systemic sclerosis (dcSSC) subtypes. Modified Rodnan scoring remained unchanged in 19 and progressed in 3 patients of lcSSc. Only 1 female showed a decrease in the score. In the dcSSc, score decreased only in 1. She had a baseline score of 12 which went down to 4. No new systemic activity was seen in her.

Streszczenie
Niską dawkę D-penicylamine 150mg otrzymywało co drugi dzień 23 pacjentów z ograniczoną skórną postacią twardziny układowej (lcSSc) i 5 pacjentów z rozlaną skórną postacią twardziny układowej (dcSSC). Zmodyfikowana punktacja Rodnan pozostała niezmieniona w 19 przypadkach a postępowała u 3 chorych z lcSSc. Tylko jedna kobieta wykazała spadek w punktacji. W dcSSc, wynik zmniejszył się tylko w 1 przypadku. Pacjentka z 12 punktów bazowych ostatecznie otrzymała 4 punkty. Obserwowano u niej brak nowej ogólnoustrojowej aktywności choroby.

Key words: systemic sclerosis; D-penicillamine; skin diseases
Słowa klucze: twardzina układowa; D-penicylamina; skin diseases

Introduction

Systemic sclerosis is a multi systemic disorder with the cardinal features of Raynauds phenomenon, sclerosis of skin with or without internal organ fibrosis. The basic pathology is believed to be vascular endothelial damage, autoimmunity and increased deposition of insoluble collagen in tissues. The ideal treatment remains elusive. D- penicillamine, a copper chelating agent used in Wilsons Disease, has been seen to block the aldehyde groups involved in the inter- and intra- molecular bonding of collagen helices [1]. This results in the formation of more soluble collagen. D- penicillamine also promotes enzymatic degradation by collagenase enzyme [1]. It has independent immunological effects as well [1]. D- penicillamine at a high dose has been tried for systemic sclerosis [1]. However, laboratory monitoring at this dose is cumbersome and many side effects are seen [2]. Recently low dose penicillamine 125 mg on alternate days has been found to be as effective as high dose therapy with lesser side effects [3-5].

Aim of the study

To report our experience with low dose D- penicillamine 150mg on alternate days for 2 years in Systemic Sclerosis (125mg tablet was not available).

Materials and Methods

This study was carried out in the Department of Dermatology SMHS Hospital (Associated teaching hospital of Government Medical College, Srinagar) between 2005-2010. All the patients of systemic sclerosis registered during this time period (both newly diagnosed as well as follow-up cases) were evaluated. Particular attention was paid to the skin sclerosis in each. Modified Rodnans Skin Scoring (mRSS) system was used to evaluate the extent of skin sclerosis [5]. 17 sites were evaluated: face, anterior chest, anterior abdomen, bilateral sites of upper arm, forearm, dorsum of hands, fingers, upper legs, lower legs, dorsum of foot. Scoring given was 0 if no change was seen, 1-skin thickened, 2-moderately involved cannot be pinched, 3-severely involved cannot be moved. Scoring was done at baseline and in patients put on low dose Penicillamine was repeated at 6 months, 1 year, 18 months, and 24 months. In order to reduce inter observer error, the same observer did a repeat scoring evaluation as far as possible.

The diagnosis of systemic sclerosis was made on the basis of the ARA criteria. A complete history especially regarding the presence of Raynaud's phenomenon, dysphagia, and dyspnoea was taken into account. A detailed physical examination including recording the weight, pulse, and blood pressure, and examination of chest (measuring the chest expansion and auscultation for basal crepitations), cardiovascular system, and abdomen were done. This was followed by a meticulous cutaneous examination. Next the patients were submitted to a battery of investigations including complete hemogram with erythrocyte sedimentation rate [ESR (fasting)], renal function tests (KFT), blood sugar (fasting), estimation of serum electrolytes, liver function test (LFT) with enzymes, chest X-ray [CXR (PA view)], electrocardiogram (ECG) all leads, X-ray hands and feet bilaterally, and urine analysis. Before starting therapy, a representative skin biopsy from fingers was sent for histopathological examination. Ophthalmological checkups for ocular tension and visual acuity was done along with upper GI endoscopy or barium swallow, high-resolution CT scan (HRCT), 24-h urinary protein, creatinine clearance, electromyography, echocardiography, pulmonary function tests (PFT), stool for occult blood, serum iron, and total iron binding capacity (TIBC). A complete collagen vascular profile was done: VDRL, LE cells, ANA, RA factor, anti-ds DNA, anti-RNP, anti-topoisomerase, anti-centromere, and creatinine phosphokinase (CPK) levels. All these investigations were done at baseline. CBC with ESR, KFT, urine exam, and BP recording was done monthly; 24-h urinary protein and CXR were repeated in 6 months. Carbon monoxide diffusion capacity could not be measured due to the nonavailability of facilities for the same.

Results

A total of 63 patients of systemic sclerosis registered for this study. Of these 54 were of limited cutaneous systemic sclerosis (lcSSC) subtype and 9 were of diffuse cutaneous systemic sclerosis (dcSSC). In lcSSc subset, 23 patients had no oesophageal involvement and mRSS of >= 2). In dcSSc, 4 patients had pulmonary involvement at the time of admission as indicated by moderate to severe restriction on PFT. Hence D Penicillamine was given to 23 patients of lcSSc and 5 of dcSSc subtypes. Score remained unchanged in 19 and progressed in 3 patients of lcSSc. Only 1 female (23years old with disease of 6 years duration showed a decrease in the score (from 5 to 2). However, the pinched appearance of nose persisted. In the dcSSc score decreased only in 1. She had a baseline score of 12 which went down to 4. No new systemic activity was seen in her. Incidentally this patient had been on Dexamethasone pulse therapy previously >1 year back and had developed genitourinary tuberculosis due to the same. Duration of disease in her was 7 years. In two patients with mild restriction in PFT at baseline the skin sclerosis progressed and pulmonary function tests showed a worsening. In one patient (a twenty four year old female with hyper pigmentation and skin sclerosis of five years duration) the mRSS worsened from 14 to 24. No new internal activity was however noticed. In one patient (a 35 years old female with four years history of sclerosis and Raynauds phenomenon) followup was poor. She continued to take therapy for 4 years. She reported back with severe skin sclerosis, anaemia, weight loss and pulmonary involvement in the form of severe restrictive lung disease. She had been previously on Dexamethasone pulse therapy and had developed cervical lymphadenopathy. Present investigations revealed pulmonary tuberculosis. Patient was put on antitubercular therapy and was planned to be put on cyclophosphamide later on.

Discussion

The role of D penicillamine in the treatment of systemic sclerosis in various studies was believed to be primarily on Skin Sclerosis with no effect on visceral and vascular symptoms but a few studies claim its efficacy on pulmonary fibrosis [6].Vital capacity and Forced expiratory volume improved but the Diffusion Capacity for carbon monoxide remained unchanged. The effect on skin sclerosis is believed to be not foolproof and its irreversibility is doubtful. Whether it prevents or retards internal organ involvement is also not fully known. As it is believed that extent of skin sclerosis has prognostic significance and it reflects internal organ involvement the therapy assumes importance in this multi system disease [5]. Penicillamine has to be started at a low dose of 250 mg and may have to be gradually increased to 1150mg/day with each dose increment being gradual. Monitoring for side effects is mandatory. In a study by Steen et al, Penicillamine 635mg for 1.8 years was studied [2]. 47% patients had side effects in the form of rash, proteinuria, gastro- intestinal symptoms, dysgeusia, oral ulcers, thrombocytopenia, neutropenia, myasthenia gravis and pemphigus. 29% patients discontinued treatment due to toxicity. Recently it was seen that 125mg penicillamine on alternate days was as effective as high dose therapy, with no advantage achieved on increasing the dose beyond this [3-5]. The recommended duration of low dose therapy is 2 years. Maximum effect is seen in rapidly progressive diffuse cutaneous SSc of less than two years duration [7].

The therapy of this unpredictable disease should serve the following purpose:

Does it decrease skin sclerosis?

Is this decrease maintained on discontinuation of therapy?

Does it prevent or retard pulmonary fibrosis?

Does it prevent development of renal crisis?

Does it decrease new organ involvement?

Hence while on therapy, lookout for fresh activity over baseline was done. In view of this low dose regimen being claimed as being relatively non-toxic we decided to try it in lcSSc without oesophageal involvement and in all dcSSc without internal organ involvement other than mild pulmonary restriction. However, a recent study proves that even low dose penicillamine therapy is not blameless vis a vis the side effect profile [8].

There were limitations in our study as 125mg capsule was not available and we had to use 150 mg instead. Even though low dose penicillamine is recommended only in rapidly progressive dcSSc, we gave it to lcSSc also without oesophageal involvement and dcSSc without systemic involvement irrespective of the duration of therapy the rationale being that therapeutic option in SSc are limited. Skin sclerosis indicates internal organ involvement. Penicillamine affects skin sclerosis and low dose is relatively safe. lcSSc is also a systemic disease with oesophageal and pulmonary vascular involvement. Hence reversal of skin sclerosis would infer retardation of internal organ involvement.

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A COMPARATIVE STUDY ON 100% TCA VERSUS 88% PHENOL FOR THE TREATMENT OF VITILIGO

BADANIE PORÓWNAWCZE 100% TCA W STOSUNKU DO 88% FENOLU W LECZENIU BIELACTWA

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Abstract
There are various medical and surgical modalities for the treatment of vitiligo. Surgical modalities are used in the patients who fail to respond to medical therapy. We selected thirty patients of stable vitiligo from the department of dermatology for the study. The patients were divided into two groups of 15 patients each. In Group I patients application of 100% TCA was done on the vitiliginous sites and in Group II patients 88% phenol was applied on the affected sites. Comparing the results of repigmentation in both the groups it was seen that marked pigmentation was seen in 66.6% patients in the TCA group and 80% in the Phenol group. Moderate pigmentation was seen in 13.3% patients in both the groups and mild pigmentation was seen in 20% patients in the TCA group and 6.6% in the Phenol group.

Streszczenie
Istnieją różne medyczne i chirurgiczne sposoby leczenia bielactwa. Chirurgiczne sposoby są stosowane u pacjentów, którzy nie reagują na leczenie. Wybraliśmy do badania trzydziestu pacjentów ze stabilnym bielactwem z Kliniki Dermatologii. Chorych podzielono na dwie grupy po 15 osób każda. W grupie I u pacjentów aplikowano 100% TCA na bielacze plamy, a w grupie II 88% fenol był stosowany w dotkniętych chorobą miejscach. Porównując wyniki repigmentacji w obu grupach okazało się, że znaczną pigmentację odnotowano u 66,6% pacjentów w grupie TCA i 80% w grupie z fenolem. Umiarkowaną pigmentację odnotowano u 13,3% chorych w obu grupach a pigmentację łagodną zaobserwowano u 20% pacjentów w grupie TCA i 6,6% w grupie z fenolem.

Key words: vitiligo; repigmentation; melanocytes; pigment; TCA; phenol
Słowa klucze: bielactwo; repigmentacja; melanocyty, pigment; TCA; fenol

Introduction

Vitiligo is an acquired pigment disturbance which affects the melanocyte, a dendritic cell producing melanin pigment and which is derived from the neural crest. In the skin, it is located at the basal layer and follicular sheath [1]. The patients refractory to medical therapy are treated by surgical modalities provided that their disease is stable for at least 2 years. Various surgical methods that are being practiced, but in our study we did spot chemical wounding with TCA and Phenol [2].

Phenol or carbolic acid is one of the oldest antiseptic and antipruritic agents. It also acts as a local anaesthetic. Liquified phenol (88%) and TCA have been used for medium depth chemical peeling for facial rejuvenation [3]. In the present study, both TCA and phenol have been successfully used as a medium depth chemical peelant which causes wounding, to treat stable vitiligenous areas and patches of alopecia areata.

Materials and Methods

We selected thirty patients of stable vitiligo from the department of dermatology for the study. The patients were divided into two groups of 15 patients each. In Group I patients application of 100% TCA was done on the vitiliginous sites and in Group II patients 88% phenol was applied on the affected sites. Informed consent was taken from all the patients before the study. Prior approval of hospital ethical was taken. All the patients were photographed before the treatment. Most of the cases had received local or oral steroids or PUVA/PUVASOL prior to the therapy with limited or no improvement. The grading of the pigmentation was done starting from grade 0 to grade 3 as follows:

No pigmentation - Grade 0

Minimal pigmentation - Grade 1

Moderate pigmentation - Grade 2 (Upto 50% of 2x2 cm2 patches)

Marked pigmentation - Grade 3 (>50% of the 2x2 cm2 patches)

Chemical peeling with 88% phenol and 100% TCA was carried on various sites of stable vitiligo. After cleansing and defatting, 100%TCA in Group I and 88% Phenol in Group II was applied on the affected areas till a uniform frost appeared. A routine urine examination, and tests for serum creatinine, blood urea nitrogen, SGOT and SGPT were carried out on all patients prior to the peel. BCG scars or old scars were examined for keloidal tendency. An informed consent was obtained and their blood pressure, heart rate and pulse rate were monitored. The area to be treated was defatted by scrubbing with savlon, followed by spirit and acetone. Both TCA and Phenol were applied then applied gently with uniform smooth strokes so as to cover the entire lesion till an ivory white uniform frosting appeared. Feathering of the borders was done by painting from the periphery of the lesion into the surrounding normal skin. All patients were monitored after half an hour for pulse rate and heart rate. They were asked to, apply mupirocin ointment twice in a day till the lesions healed. After 10-15 days (on completion of wound healing), all patients of vitiligo were started on PUVA/PUVASOL. All patients in both the groups were followed up at weekly intervals for 2 months, 15 days intervals for the next 4 months and at monthly intervals for one year. The procedure was repeated once in a month if required.

Results (Tabl. I, II)

The data was tabulated and the results were analyzed statistically.

SR NO	Pigmentation	Group I (100% TCA)	Group II (88% Phenol)
1	MARKED(> 90%)	66.6%(10)	80%(12)
2	MODERATE(61-90%)	13.3%(2)	13.3%(2)
3	MILD(<60%)	20%(3)	6.6%(1)

Table I. Repigmentation in both the groups

SR NO	Complications	Group I	Group II
1	hyperpigmentation	3(20%)	2(13.3%)
2	hypopigmentation	2(13.3%)	1(6.6%)
3	persistent erythema	1(6.6%)	1(6.6%)
4	secondary bacterial infection	1(6.6%)	-
5	superficial scarring	1(6.6%)	-

Table II. Associations of diabetes mellitus

Discussion

On healing, all the lesions of vitiligo showed perifollicular pigmentation in hairy areas and perilesional repigmentation in non hairy areas. These were further treated with PUVA/PUVASOL. Comparing the repigmentation in both the groups it was seen that marked pigmentation was seen in 66.6% patients in the TCA group (Fig. 1, 1a) and 80% in the Phenol group (Fig. 2, 2a), moderate pigmentation was seen in 13.3% patients in both the groups and mild pigmentation was seen in 20% patients in the TCA group and 6.6% in the Phenol group. Regarding the complications in both the groups, hyperpigmentation was seen in 20% patients in the TCA group and 13.3% patients in the Phenol group, hypopigmentation was seen in 13.3% patients in the TCA group and 6.6% patients in the Phenol group, persistent erythema was seen in 6.6% patients in both the groups, secondary bacterial infection and superficial scarring was seen in 6.6% patients each in the TCA group and in none of the patients in the Phenol group. After the crust fell off, all patients were given PUVA/PUVASOL treatment for the next 2-3 months. Gradually the perifollicular hyperpigmentation started enlarging in size and coalesced together to cover the entire patch. In cases of non hairy sites, the pigment spread slowly from the border of the lesions for a small distance towards the centre. Hypopigmentation seen after TCA and Phenol application was seen in 19.9% patients occurs because the melanin synthesis is impaired temporarily resulting in hypopigmentation. Hyperpigmentation seen in 33.3% patients is due to the fact that skin diseases induce post inflammatory hyperpigmentation. The inciting inflammatory process causes an increase in both melanogenesis and the transferring of melanin granules to the surrounding keratinocytes. Post peel erythema was seen in 13.2% patients and it represents angiogenesis in response to re-epithelialisation and occurs during would healing initially. Secondary bacterial infection occured as a complication in 6.6% patients due to improper wound care on the part of the patients. All of them reported early and their smear examination revelaed Staphylococcus aureus which responded to cephalosporins. Superficial scarring was seen in % patients and this could be because of penetration of the chemical agents which could have seeped in deeper.

Liquified phenol consists of 88% solution of phenol in water and causes kerato coagulation by precipitating the surface proteins [4]. At this concentration, phenol causes medium depth wounding which creates changes through necrosis of the epidermis and part or all of the papillary dermis with an inflammatory reaction in the upper reticular dermis [5]. Re-epithelialisation starts from the 3rd day and is continued till 10th-15th day. The initial event in this process is the migration of keratinocytes from the residual adnexal epithelium at the base of the wound (pilosebaceous follicles and eccrine glands) and also from the wound margin.

Figure 1, 1a. Pre and post treatment photograph of a 35 year old patient treated with TCA

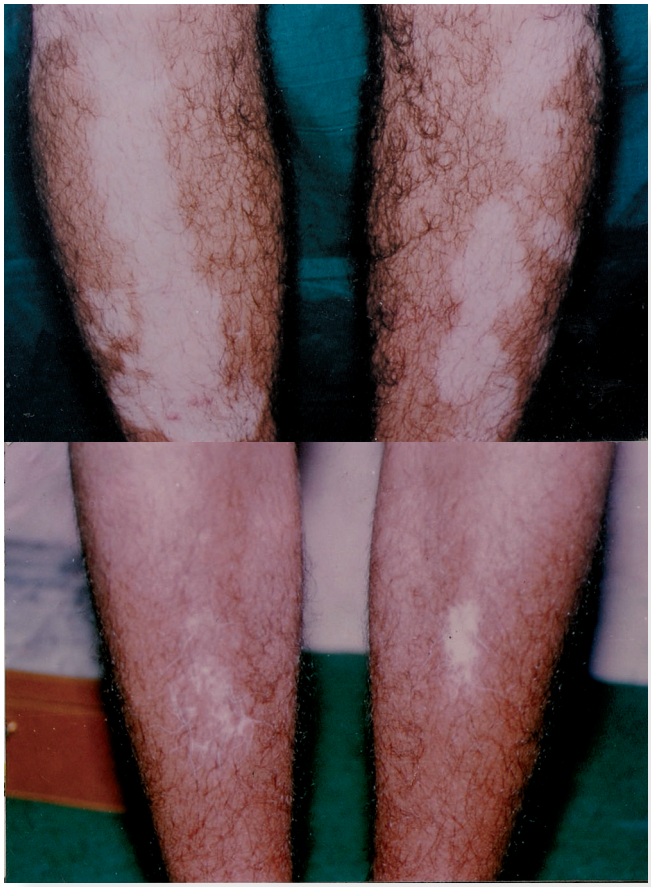

Figure 2, 2a. Pre and post treatment photograph in a 28 year old patient treated with Phenol

Phenol when used for facial rejuvenation, is known to cause cardiac arrythmias, if the quantum of phenol exceeds 3 ml, the duration of application is less than 60 min or when applied to large cutaneous surface areas [6]. This was not seen in any of our patients since precautions were taken not to exceed 1/2- 1 ml in one session. Phenol is also known to be hepatotoxic and nephrotoxic. Diuresis is known to promote metabolism and excretion of phenol. Hence in this study all patients were asked to take plenty of water after the peel. In cases of vitiligo, spot or regional dermabrasion has been used to repigment the hairy areas. It is also known that sometimes topical psoralens with UVA gives rise to blister formation resulting in post inflammatory pigmentation. Both TCA and Phenol in this study have been used for repigmentation on the same principles of wounding [7]. During wounding, there is acute inflammation which is known to induce melanogenesis. During the re-epithelialisation process, melanocytes migrate from the remnants of hair follicles, eccrine glands and also from the surrounding normal skin [8]. In the hair follicles, the inactive melanocytes in the middle and/or lower parts of the outer root sheath divide, proliferate and migrate upwards to near by epidermis [9]. The melanocytes continue to migrate radially to form the pigmented island which is seen as perifollicular pigmentation. Also, the melanocytes from the perilesional normal epidermis migrate towards the centre of the lesions along the border in both hairy and non-hairy areas thus causing perilesional pigmentation. These findings of pigmenting perifollicularlly and perilesionally was observed in our study also. During wound healing, various growth factors are released like endothelial growth factors and fibroblast growth factors which are mitogenic for the melanocytes [10]. Moreover the inflammatory mediators like leukotriene C4 and D4 stimulate the melanocyte proliferation. It is possible that these factors could also have stimulated the pigmentation after a phenol peel wound [11]. Combining the wounding procedure with medical lines of treatment is known to enhance the rate of pigmentation.

Conclusions

TCA and Phenol peel, as seen in this study is a simple office procedure with no complicated surgery or anaesthesia involved and also needs no expertised training. Discomfort and pain are minimum and hospitalisation or dressings are not required. It can be considered as one of the alternate method to repigment stable vitiligo. Repeat peels can be done on these areas if required. One can cover large areas in multiple sittings.

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A COMPARATIVE STUDY ON 100% TCA VERSUS 88% PHENOL FOR THE TREATMENT OF VITILIGO

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comment:

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The study performed by Puri and Puri appearing in this issue suggested that TCA and phenol, the two medium depth chemical peeling agents, may be used to induce repigmentation of stable vitiligo lesions. Although different modalities have been introduced for vitiligo treatment, surgical intervention is still considered as an important treatment option for stable vitiligo lesions that do not respond to conventional therapies. Different surgical techniques, some requiring cellular cultures, have been established for treating stable vitiligo. More recently, dermabrasion has also been suggested to be an alternative intervention, especially if followed by phototherapy [1]. While it is of academic importance to clarify if the mechanisms involved in repigmentation differ between these different surgical approaches, it is perhaps even more crucial to identify "stable" vitiligo lesions that will most likely respond to surgical interventions.

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STUDY OF FRACTIONAL ABLATIVE LASER IN SURGICAL AND POST TRAUMATIC SCAR

BADANIE NAD WYKORZYSTANIEM ABLACYJNEGO LASERA FRAKCYJNEGO W CHIRURGICZNYCH I POURAZOWYCH BLIZNACH

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Abstract

Introduction: Ablative, fractional lasers generate microscopic columns of coagulated tissue through the epidermis and dermis to evoke a wound healing response. In this study, we examined the efficacy and safety of fractional ablative 2940nm erbium: YAG laser in the treatment of surgical and post-traumatic scars. Fractional laser photothermolysis is the latest in the broad range of Er: YAG laser technique. This technique promises a novel means of providing treatments that would be as effective as traditional Er: YAG, while further reducing their down time and risk.

Aim of the Work: The aim of this work is to assess the efficacy and safety of variable square pulse (VSP) fractional Er: YAG laser for the treatment of surgical and post-traumatic scars; both clinically and histopathologically.

Methods: Clinical studies were conducted on a range of surgical and post-traumatic scars with a 2940nm erbium: YAG fractional ablative laser varying energy, pulse widths, treatment passes, and number of treatments: twenty subjects, with Fitzpatrick skin types III-IV, received two to five treatments at one month interval and a follow up period for 3 months. Clinical and histopathological evaluation of the results was performed.

Results: Almost all patients improved both clinically and histopathologically. Clinical improvement in scars according to investigator assessment: 40% of patients had excellent improvement of 76-100% (grade 3), 50% of patients had good improvement of 50-75% (grade 2), 10% had fair improvement of 26-49% (grade 1) at three month follow up. Histologic findings demonstrated remodeling of scar tissue with renewal and reorganization of collagen fibers in the dermis was noted two weeks post-treatment.

Conclusion: These data illustrate the safety and efficacy of the 2940nm erbium:YAG fractional ablative laser in the treatment of surgical and post-traumatic scars with short down time period, and almost no incidence of complication.

Streszczenie

Wstęp: Ablacyjne lasery frakcyjne generują mikroskopijne kolumny poprzez koagulację tkanek naskórka i skóry właściwej wywołując reakcję gojenia ran. W tym badaniu zbadaliśmy skuteczność i bezpieczeństwo frakcyjnego ablacyjnego lasera erbowego 2940nm: YAG w leczeniu blizn chirurgicznych i pourazowych. Frakcyjna laserowa fototermoliza jest ostatnio techniką o szerokim zakresie Er: YAG. Technika ta obiecuje nowy sposób świadczenia zabiegów, które byłyby tak samo skuteczne jak tradycyjne Er: YAG, przy jednoczesnym zmniejszeniu ich czasu i ryzyka.

Cel pracy: Celem niniejszej pracy jest ocena skuteczności i bezpieczeństwa stosowania zmiennego kwadratowego impulsu (VSP) frakcyjnego lasera Er: YAG do leczenia blizn chirurgicznych i pourazowych, zarówno pod względem klinicznym jak i histopatologicznym.

Metody: Badania kliniczne prowadzone były w zakresie chirurgicznych i pourazowych blizn za pomocą frakcyjnego ablacyjnego lasera erbowego 2940nm: YAG różnej energii, szerokości impulsu, przebiegu leczenia i liczby zabiegów: dwudziestu pacjentów, z typami skóry Fitzpatrick III-IV otrzymało od dwóch do pięciu zabiegów w oresie jednego miesiąca i następnie przez okres do 3 miesięcy. Przeprowadzono kliniczną i histopatologiczną ocenę wyników.

Wyniki: U prawie wszystkich pacjentów zauważono poprawę zarówno kliniczną jak i histopatologiczną. Poprawa kliniczna blizn według oceny badaczy: 40% pacjentów miało doskonałą poprawę w zakresie 76-100% (stopień 3), 50% pacjentów miało dobrą poprawę w zakresie 50-75% (stopień 2), 10% miało zupełną poprawę w zakresie 26 -49% (stopień 1) na trzy miesiące po terapii. Histololigiczne wyniki wykazały, przebudowę blizny z odnową i reorganizacją włókien kolagenowych w skórze właściwej co zostało zanotowane do dwóch tygodni po leczeniu.

Wnioski: Dane te ilustrują bezpieczeństwo i skuteczność 2940nm erbowego: YAG ablacyjnego lasera frakcyjnego w leczeniu chirurgicznych i pourazowych blizn w krótkim czasie i bez prawie żadnego przypadku wystąpienia komplikacji.

Key words: fractional ablative laser; surgical; post traumatic; scar
Słowa klucze: ablacyjny laser frakcyjny; chirurgia; pourazowy; blizna

Introduction

Scars affect approximately 4.5-16% of the general population and arise from either excessive or insufficient new collagen generation during the wound healing process [1]. Hypertrophic scars appear as hypo-pigmented or erythematous raised nodules or plaques containing excessive amounts of collagen, fibrin and proteoglycans [2,3]. In contrast, atrophic scars are dermal depressions with overlying thinned epidermis which results from a loss of dermal collagen following some types of inflammation or traumatic injury such as acne, varicella, post-traumatic wounds or post-operative scars [4].

On the surface, scars may appear to be only a cosmetic concern; however, they can significantly impact the patient on many different physical and psychological levels. Physically, scars can impede the patient's range of motion, and can cause pain, dysesthesia and pruritus [5-7]. Patients with severely disfiguring scars may also experience such psychological symptoms such as low self-esteem and feelings of psychosocial isolation [8,9].

Various treatments have been developed to improve the appearance of scars and to address these adverse effects, including silicone gel sheets, pressure garments, corticosteroid therapy, dermabrasion, surgical excision, chemical peels and more recently, laser treatments [7-11].

While some laser therapies have yielded positive results, others, such as the Nd: YAG and traditional ablative lasers, have actually worsened scar appearance [2,11,12]. Furthermore, traditional ablative treatments result in considerable patient downtime and adverse events such as oozing, infection, and hyper-pigmentation [13-15].

More shallow, full-surface horizontal resurfacing treatments with the short-pulsed ER: YAG laser have advantages of reduced patient downtime and minimal side effects; however, modest clinical results restrict treatment recommendations to only mild atrophic scars [1,16]. While Pulsed dye lasers (PDL) are considered to be the gold standard for scar revision based on their ability to improve scar pliability and texture and decrease erythema [17-23]. Fractional photothermolysis is a new technique for the treatment of scars [24] in which an array of microscopic thermal wounds (microscopic treatment zones) is induced into the skin to stimulate a therapeutic response deep in the dermis. Nonablative fractional photothermolysis at a wavelength of 1,550 nm has been found to be effective for the treatment of melasma, [25] mild to moderate rhytides, [26] acne scars, [27] surgical scars, [28] and even poikiloderma of Civatte [29]. However, this „coagulative" approach is time-consuming and painful, and the results are not always predictable.

Recently, „ablative" fractional photothermolysis using the Erbium: YAG laser (2940 nm) has been introduced as a novel means of providing treatment that would be as effective as traditional ablative approaches while avoiding their high downtime and risks [30,32]. The laser produces thousands of microscopic, clinically inapparent wounds on the skin surface that are rapidly reepithelialized by the surrounding, undamaged tissue, sparing the epidermis.

Materials and Methods

This study was conducted on 20 patients, 12 male and 8 female aged between 12-45 years old. All patients have surgical and post-traumatic scars. Exclusion criteria;History of keloid formation. Squamous cell carcinoma or melanoma. Immunodeficiency disorder. Hypersensitivity to light. Bleeding disorder or use of anticoagulant for which a 10-day washout is not allowed before study treatment. Active local or systemic infection; light-sensitive medication. The use of immunosuppressive medication. Use of botulinum toxin A, dermal fillers in areas to be treated within the previous four or six months. Facelift, use of isotretinoin, or ablative laser to target areas within the past 12 months.Treatment with chemical peels or dermabrasion within the past three months; treatment of the target areas with laser or other device within the past three months. In this study we attempt to confirm the effect of PST (Pixel screen technology) fractional ablative Erbium:YAG (Er:YAG) 2940 nm fractional laser on different types of scars.

The treatment parameters in the present study ranged from 600-1100 mJ fluence, with a spot size of 7 mm, a frequency of 5 Hz and pixel 3 (medium ablation pixel number 30 and pixel size 800 micrometer). Using the soft fractional PS 01 laser hand piece R 04. The number of passes depend on the treated area and type of scars. The patients had two to five sessions.

The clinical assessment was objectively based on clinical photography before treatment and three months after laser treatment by means of clinical improvement, patient satisfaction and histopathological findings.

Clinical improvement assessed by Investigator assessments performed at one- month follow-up visits using the quartile assessment scale („excellent 3 „: 76-100%; „good 2 „: 50-75%; „fair 1 „: 26-49%; „poor 0 „: 0-25%). Two blinded-dermatologists evaluating clinical photographs taken before and after laser treatment. Investigators will be asked to select which image represented the post-treatment image and to rate the percent improvement in the appearance of the surgical or post-traumatic scar. Who will grade the results on a five- point scale, as follows: (excellent, 75% to 100% improvement; good, 50% to 75% improvement; fair, 25% to 50% improvement; poor, 0-25% improvement; or worse, final results were worse than the pre treatment results).

Patients self assessment: Patients will assess their improvement using the following five-point scale: 0=no or minimal improvement, not satisfied (0%-10%); 1=slight improvement, slightly satisfied (11-25%); 2=moderate improvement, satisfied (26-50%); 3=significant improvement, very satisfied (51-75%); and 4=substantial improvement, extremely satisfied (> 75%). Patient assessments will be gathered using phone interviews or written questionnaires.

Some patient will be subjected to two millimeter punch biopsy of hidden scar will be taken prior treatment and two weeks post-treatment, tissue samples will be fixed in formalin and processed for hematoxylin and eosin (H&E) staining and elastic tissue with orcein stain.

All subjects involved in the current work will be informed about the nature and the details of the work and a written consent will be obtained also approval by ethical committee was obtained.

Results

This study included 20 patients having surgical and post-traumatic scars with a broad range of atrophic and hypertrophic scars.

Regarding the age of patients varied from 12-42 years with a mean of 21.15 ± 8.92 years.

The study included 12 males (60%) and 8 females (40%).

The skin type, 12 patients (60%) had Fitzpatrick skin type III and 8 patients (40%) had Fitzpatrick skin type IV.

As regard the cause, 8 patients (40%) were surgical scars (post trauma repair four patients, open repair of fracture two patients, post appendicectomy two patients) and 12 patients (60%) were traumatic scars (fall in sharp or blunt object six patients, scratch marks two patients, burn two patients).

As regard scar location, 7 patients (35%) had scars located on the arm, 4 patients (20%) had scars located on cheeks, 2 patients (10%) had scars located on forehead, 1 patients (5%) had scar located on chin and 1 patient (5%) had scar located in upper lip.

As regard the duration of the scars, it ranged between 5 months and 15 years, with a mean of 4.40 ± 3.20. 5 patients (25%) had scar duration less than 1 year and 15 patients (75%) had scar duration more than one year.

Regarding the clinical improvement of scars to laser therapy (Fig. 1-3a,b), the present study revealed that all patients had clinical improvement in scars according to investigator assessment; 40% of patients had excellent improvement of 76-100% (grade 3), 50% of patients had good improvement of 50-75% (grade 2), 10% had fair improvement of 26-49% (grade 1) at three month follow up.

The clinical improvement according to blinded dermatologist assessment revealed that there was no significant difference between it and investigator assessment.

The degree of patient satisfaction differed from one case to another. Seven patients were slightly satisfied (35%), one patient was satisfied (5%), five patients were very satisfied (25%), seven patients were extremely satisfied (35%). Even, the majority of patients including those who were slightly improved asked for more session.

As regard Pain which is inherently subjective, all patients tolerated the procedure with topical anesthesia (EMLA) for one hour before procedure, no pain to severe pain was reported by all patients, ranged between (0-3) with a mean 1.65 ± 0.88, all patients stated that the discomfort ceased upon removal of the light. 2 patients (10%) had no pain =0.

- 6 patients (30%) had mild pain =1, 9 patients (45%) had moderate pain =2, 3 patients (15%) had severe pain =3.
- As regard crust formation, 2 patients (10%) had crust for four days, 5 patients (25%) had crust for 5 days, 3 patients (15%) had crust for 6 days, 7 patients (50%) had crust for 7 days, with a range of (4-7) days and a mean of 6.05 ± 1.10 days.
- As regard erythema, 2 patients (10%) had erythema for 1 day, 10 patients (50%) had erythema for 2 days, 8 patients (40%) had erythema for 3 days with a range of (1-3) and a mean of 2.3 ± 0.66 days.
- As regard swelling, 16 patients (80%) had swelling for 1 day, 4 patients (20%) had swelling for 2 days. with a range of (1-2) days and a mean of 1.2 ± 0.41 days.
- No patients show permenant hypopigmentation or hyperpigmentation.

Histologic findings (Fig. 4-6a,b) demonstrated remodeling of scar tissue with renewal and reorganization of collagen fibers in the dermis was noted two weeks post-treatment.

The scar tissue had histologically improved by two weeks post treatment as evidenced by a thickened epidermis with normal rete ridge pattern and reduced number of hyperplastic collagen bundles. Two weeks post treatment, there is an increase in elastic fibers particularly in upper dermis and they appear thicker and more randomly distributed was noted.

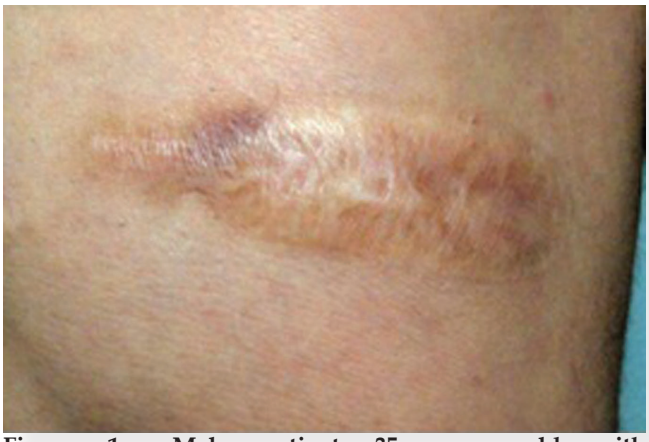

Figure 1a. Male patient 35 years old with hypertrophic scar with blotchy hyperpigmentation in the arm since 3 years (traumatic scar, before treatment)

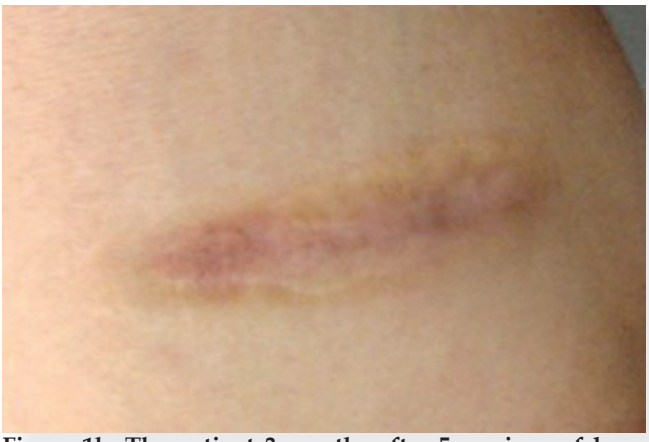

Figure 1b. The patient 3 months after 5 sessions of laser treatment showing flattening, reduced hyperpigmentation and better overall scar quality (excellent improvement 76-100%)

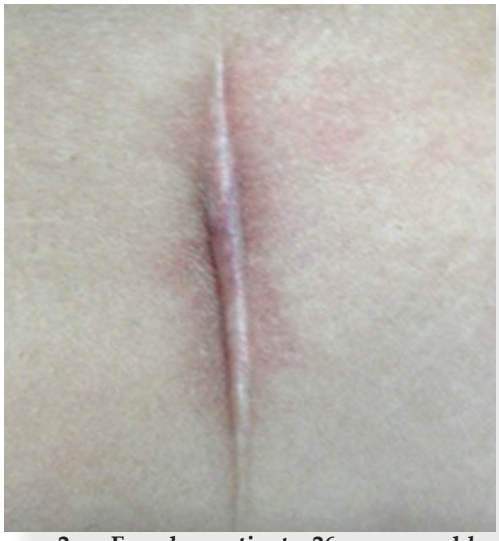

Figure 2a. Female patient 26 years old with hypertrophic and erythematous scar on the chest since 6 months (post-trauma repair, before treatment)

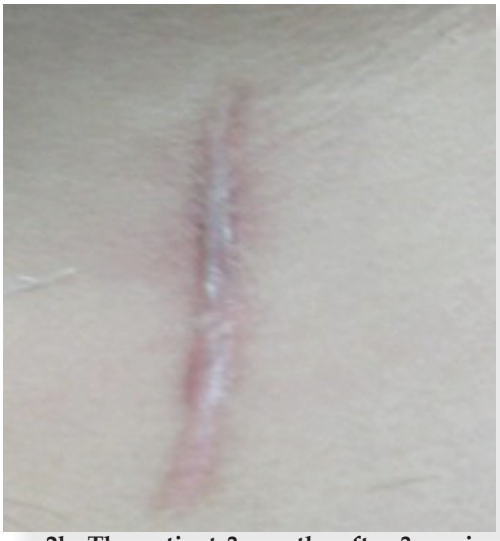

Figure 2b. The patient 3 months after 3 sessions of laser treatment showing reduced erythema and the scar appear flatter (good improvement 50-75%)

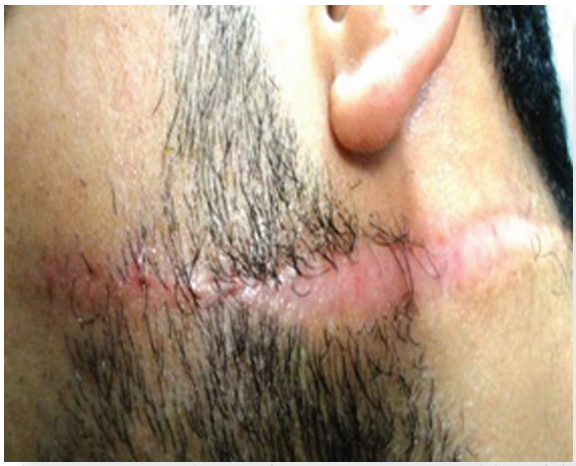

Figure 3a. Male patient 20 years old with erythematous and atrophic scar with elevated medial third in the cheek since 1 year (post-trauma repair, before treatment)

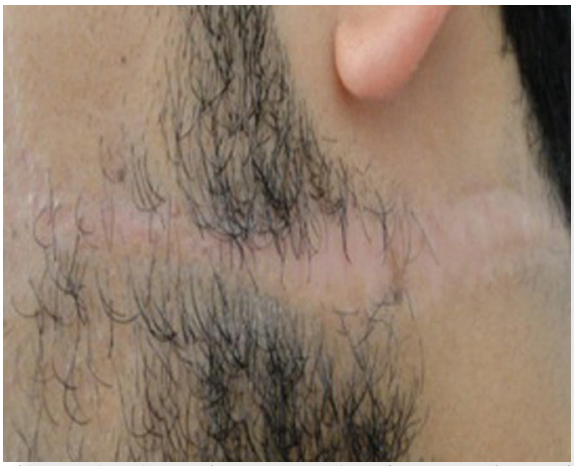

Figure 3b. The patient 3 months after 3 sessions of laser treatment showing decreased erythema and over all flattening of the scar (excellent improvement 76-100%)

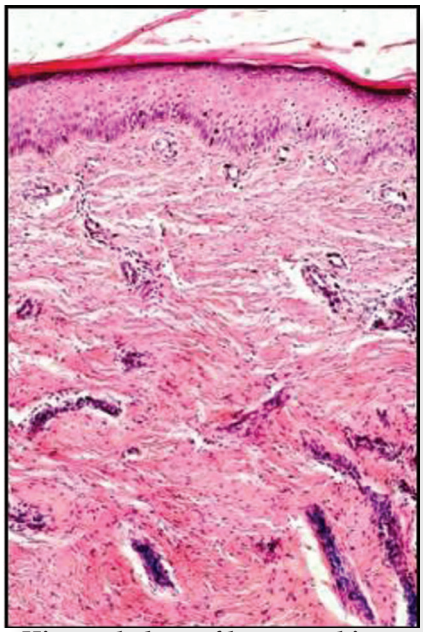

Figure 4a. Histopathology of hypertrophic scar before treatment. There are and significant replacement of papillary dermis with abnormal hyperplastic collagen fibers and excessive inflammatory cells in the scar tissue

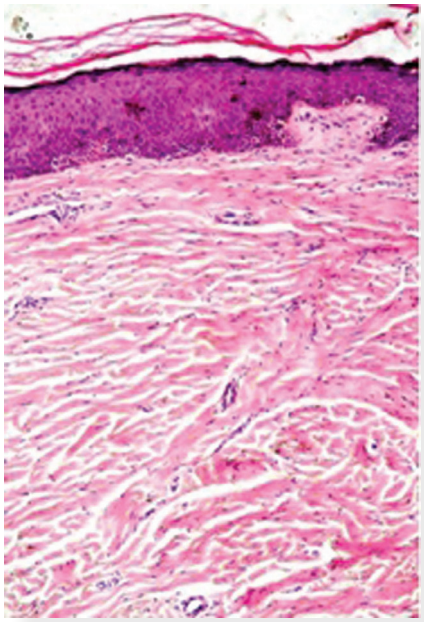

Figure 4b Two weeks post treatment, remodeling of a scar tissue with renewal and reorganization of collagen fibers in the dermis and reduced inflammatory cells was noted. (H&E X 100)

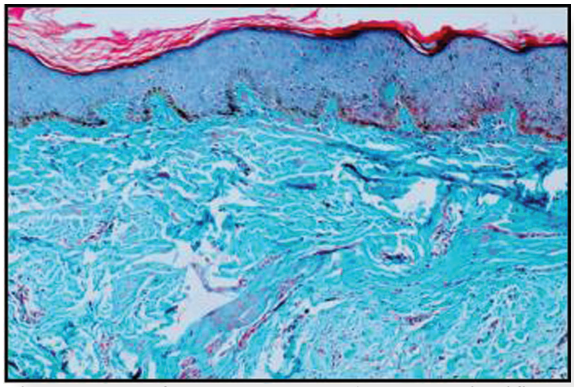

Figure 5a. Before treatment. There are significant replacement of papillary dermis with abnormal hyperplastic collagen fibers in the scar tissue

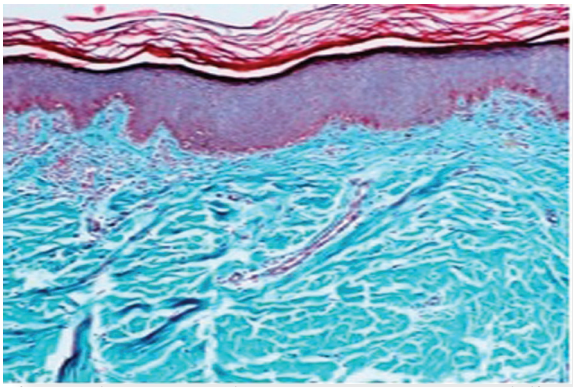

Figure 5b. Two weeks post treatment, remodeling of a scar tissue with renewal and reorganization of collagen fibers in the dermis was noted (Maisson trichrome X 100)

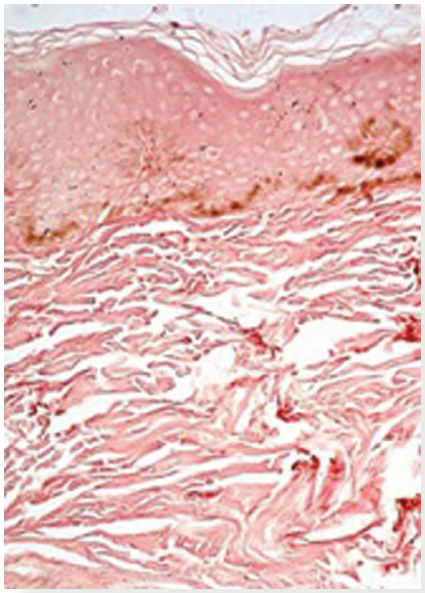

Figure 6a. Before treatment. There are thin, frayed elastic fibers arranged in a parallel orientation

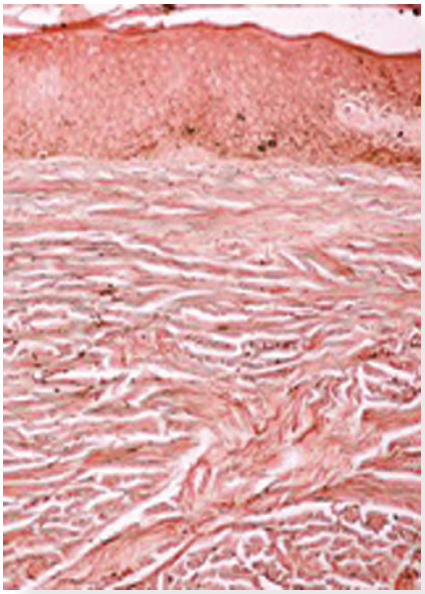

Figure 6b. Two weeks post treatment, there is an increase in elastic fibers particularly in upper dermis and they appear thicker and more randomly distributed was noted. (Orcein staining X 100)

Discussion

In this study, the PST (Pixel screen technology) fractional ablative Er:YAG laser with variable pulse width was used allowing the selection of the effect of the laser from ablation peeling to deeper thermal effect and coagulation. Pixel Screen Technology (PST) divides the basic Er:YAG treatment beam into parallel beam pixels. The advantage of PST compared to other fractional technologies is that it allows the laser beam quality and parameters within the pixels to remain unchanged compared to the basic beam properties. Other fractional technologies use focused beams with modified pulse fluences in the fractional spots and therefore with modified and relatively uncontrolled thermal treatment modalities. PST ensures that the laser fluence in each pixel is exactly as it would be with a standard Er:YAG laser handpiece. PST hand piece has been designed and developed for "stamped" fractional photothermolysis techniques, as opposed to the scanning fractional techniques. The latter technique requires a scanner and in some systems expensive consumables [1,2,4]. In the present study, the clinical assessment was objectively based on clinical photography before treatment and three months after laser treatment by means of clinical improvement, patient satisfaction and histopathological findings. Regarding the clinical improvement of scars to laser therapy, the present study revealed that all patients had clinical improvement in scars according to investigator assessment; 40% of patients had excellent improvement of 76-100% (grade 3), 50% of patients had good improvement of 50-75% (grade 2), 10% had fair improvement of 26-49% (grade1) at three month follow up. In this study, there was improvement in all hyperpigmented and erythematous scars and over all flattening in scars. Hypertrophic scars and atrophic scars responded to treatment with fractional laser photothermolysis. The question elaborated, how the same technology can benefit atrophic and hypertrophic scars is intriguing and deserves further investigation. Perhaps dermal heating normalizes collagen or vascular neogenesis or breaks and realigns abnormal collagen fibers [33-35].

Karen et al [36] investigated an Er:YAG 2940 laser with thermal mode in 12 patients with scars including post traumatic scars and fascial atrophic scars. Two treatment were applied two month apart, at 3–6 months follow-up was graded as excellent in 50%, good in 25%, fair in 25%, and no improvement in 0%. The explanation of such difference can be attributed to the use of different mode (they used thermal mode with sub-ablative fluences of 2.1 and 3.1 J/cm2).

A study was performed by Elliot et al. [37] to investigate ablative fractional laser CO2 which included 15 patients having post-operative and traumatic scars. Each scar received 3 AFR treatments at 1- to 4-month intervals; at six month follow up investigator assessment was graded as 16% of the treated scars achieving excellent 76% or greater overall improvement and 89% of treated scars achieving 51% or greater overall improvement. In the present work, at 3 month follow up 40% of patients had excellent improvement 76% or greater and 90% of patients had 50% or greater improvement. Such difference may be explained by the fact that, we used more number of sessions of laser treatment (up to five treatment sessions at monthly interval) and they used different laser type.

Regarding occurrence of side effects, in the present work, Treatments were well tolerated by all patients no pain to sever pain was reported with a mean1.65±0.88. All patients stated that discomfort ceased upon removal of the light, which was observed by Ane M et al. [38] who recorded that the treatment was well tolerated, without the need for oral analgesic or anxiolytic medication.

Treatment-induced erythema was characterized by all subjects as being both mild and transient and lasted about one to three days with a mean 2.3±0.66. In this respect similar results reported by Hui et al [39].

After treatment, immediate post procedure erythema was noted lasted about three days with a mean 3.6±1.6. Regarding oedema in this study, it persisted for (1 to 2 days) and a mean 1.2±0.41. This was similar to that reported by Elliot et al. [37]. Regarding crust formation, in this study, it persisted for about 4 to 7 days with a mean 6.05±1.10. Similar results reported by Hui et al. [39].

In the present work, no patients reported blistering, persistent swelling or any other adverse events. Of the 20 patients who returned for follow-up three months after their last treatment, none reported any delayed-onset changes in skin pigmentation, erythema, and increased skin sensitivity. No bacterial infections or episodes of viral reactivation occurred during the study.

Karen et al [36] reported that one patient with a history of herpes simplex, a reactivation of latent labial herpes simplex virus infection occurred after the first laser treatment. When acyclovir was given prophylactically before the second treatment, another outbreak could be prevented.

On contrary Rostan et al. [40] reported that ablative resurfacing with the CO2 or Er:YAG laser is associated with considerable downtime and a risk of prolonged erythema, infection, scarring, and delayed hypopigmentation. Moreover, it is painful and usually requires general anesthesia. This was not noted in our study, although the follow-up was relatively short. We speculate that the microscopic pattern of injury induced by the 2,490-nm laser caused only minimal inflammation and therefore led to fewer clinically evident pigmentary changes.

Joy et al. [41] Patients reported moderate pain during treatment. After the procedure, moderate to severe erythema and edema typically resolved within 24 to 48 hours. No additional adverse effects were observed. These findings could be explained by The laser used by Joy et al. [41] was fractional non ablative Erbium-doped fiber laser.

Concerning patients' satisfaction most cases were satisfied at the end of the study period. The degree of patient satisfaction differed from one case to another. Seven patients were slightly satisfied (35%), one patient was satisfied (5%), five patients were very satisfied (25%), seven patients were extremely satisfied (35%). Even, the majority of patients including those who were slightly satisfied asked for more session which explains the psychological aspect of therapy.

Hei et al. [42] found that patient satisfaction with treatment was found to be high, with Five patients stated they were highly satisfied (71.4%) and the remaining two were somewhat satisfied (28.6%).

The clinical improvement according to blinded dermatologist assessment revealed there was no significant difference between it and investigatent.

Histopathology

The scar tissue has histologically improved by two weeks post-treatment as evidenced by a thickened epidermis with normal rete ridge pattern and reduced number of hyperplastic collagen bundles and collagen regrowth as highlighted by Masson's trichrome stain. Orecin staining of untreated scar tissue reveals thin, frayed elastic fibers arranged in a parallel orientation, compared to normal tissue where elastic fibers are thicker in the reticular dermis and more randomly distributed. Two weeks after treatment with the 2940 nm laser, there is an increase in elastic fibers particularly in the upper dermis and they appear thicker and more randomly distributed than in the untreated scar tissue.

In the study conducted by Moshe et al. [32], the biopsy samples clearly showed the thickened epidermis with normal rete ridge pattern and remodeling of scar tissue with renewal and reorganization of collagen and elastic tissue.

These findings support the findings of David et al. [43] who did serial biopsies immediately post-treatment, at 72 hour and two weeks post treatment. The author reported immediate vacuole formation at the dermoepidermal junction is seen with sub-epidermal cleft formation and an underlying zone of dermal coagulation. By 72 hours, the coagulation zone is less defined and complete re-epithelialization has occurred. Although necrotic debris is extruded from the epidermis, it remains entrapped under the stratum corneum and a mild inflammatory reaction is observed. Remodeling of scar tissue with renewal and reorganization of collagen fibers in the dermis was noted two weeks post-treatment.

Histopathological finding in the present study support the previous evidences that the columns of thermal injury characterized by localized epidermal necrosis and collagen denaturation initiate a cascade of events that leads to a normalization of the collagenesis–collagenolysis cycle. This was consistent with Tannous et al. [44] who correlated the histopathological finding and Clinical improvement of pigmented lesions to the formation of microscopically small areas of epidermal necrotic debris and dermal contents containing melanin.

Those necrotic contents migrate and are progressively eliminated, resulting in release of pigment and improvement of pigmented lesions. Also, Goldberg et al [45] who did histologic and ultrastructural analysis reported a decrease in the number of melanocytes and the amount of melanin granules within keratinocytes, consistent with this elimination process. They did not observe post inflammatory hyperpigmentation even in patients with Fitzpatrick skin type IV. It is possible that the micro-beam-composed laser avoids bulk heating of the skin dermis, reducing the risk of post inflammatory hyperpigmentation

Concolusion

From the previous results of this study and in addition to reviewing related internationally published literature, we have the following conclusions and recommendations:

- Scars cause significant impact on the quality of life of the affected patients and compel the search for more effective treatments.
- The ablative fractional laser treatments represent a safe, effective and a promising treatment modality for improving scars due to surgery or trauma with reduced risk and downtime compared to existing laser methods.
- Detailed medical history and drug history should be taken for any case before performing laser treatment.
- Controlled studies are warranted to better understand the efficacy of ablative fractional photothermolysis for the treatment and prevention of scars and to determine optimal parameters.
- Further histopathological studies should be done for more understanding of mechanism of action of laser beam in scar tissue.

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CLINICAL EVALUATION OF LOW LEVEL LASER THERAPY IN TREATMENT OF CUTANEOUS LEISHMANIASIS

KLINICZNA OCENA LASEROTERAPII W LECZENIU SKÓRNEJ POSTACI LEISZMANIOZY

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Abstract
This study aimed to evaluate the effectiveness of Low Level Laser Therapy (LLLT), with specific laser parameters, in the treatment of Cutaneous Leishmaniasis (CL). Thirteen patients, clinically and by positive smear diagnosed as cases of CL, were referred from Khartoum Teaching Hospital and were considered as study population. The Treatment was done using diode laser probe with wavelength of 820 nm, followed by cluster probe (assembly of non-coherent and coherent diodes). The dose was: I. Diode laser probe with energy density of 48 J/cm2 for thirty seconds. II. Cluster probe with energy density of 9.6 J/cm2 for two minutes.
The distance between the probe and the skin was less than 1cm. The frequency of treatment was three sessions weekly for total of ten sessions. The function of LLLT in this study was to reduce inflammation (anti-inflammatory effect) and accelerate healing. The results showed that the response was excellent in the majority of treated patient (92.3 %). The complications were minimal and transient. The results proved that LLLT is a successful treatment method for Cutaneous Leishmaniasis and it is easy to perform.

Streszczenie
Celem badania była ocena efektywności laseroteramii (LLLT), przy określonych parametrach lasera w leczeniu skórnej postaci leiszmaniozy (CL). Trzynastu pacjentów zdiagnozowanych jako przypadki CL (badanie klinicznie i pozytywny rozmaz) z Teaching Hospital w Chartumie zostały zakwalifikowane do badanej grupy populacji. Leczenie wykonywano za pomocą sondy lasera diodowego o długości fali 820 nm, a następnie klasterowej sondy (montaż diod nie-spójne i spójne). Dawka wynosiła: I. Dioda laserowa z sondą gęstości energii 48 J/cm2 do trzydziestu sekund. II. Sonda klasterowa z gęstością energii 9,6 J/cm2 przez dwie minuty.
Odległość między sondą a skórą była mniejsza niż 1 cm. Częstotliwość leczenia wynosiła trzy sesje w tygodniu (łącznie dziesięć sesji).
Funkcją LLLT w tym badaniu było zmniejszenie stanu zapalnego (działanie przeciwzapalne) i przyspieszenie gojenia. Wyniki wykazały, że reakcja była doskonała u większości leczonych pacjentów (92,3%). Komplikacje były minimalne i przemijające. Wyniki wykazały, że LLLT jest skuteczną metodą leczenia skórnej leiszmaniozy i jest łatwa do wykonania.

Key words: cutaneous leishmaniasis; low level laser therapy; anti-inflammatory laser effect; laser in dermatology
Słowa klucze: skórna leiszmanioza; laseroterapia; przeciwzapalne działanie lasera; laser w dermatologii

Introduction

Leishmaniasis is a parasitic disease spread by the bite of infected sand flies. The disease is found in parts of about 88 countries on 4 continents. One of the most common forms of the disease is cutaneous leishmaniasis that occurs most commonly (over 90%). A form that affects some internal organs of the body, visceral leishmaniasis, mostly occurs in Bangladesh, India, Nepal, Brazil and Sudan [1]. Cutaneous leishmaniasis is endemic in many parts of Sudan, in Kordofan province, Darfur province and along the main river Nile. The first indigenous case was described by Archibald in 1911 in a native from the Nuba Mountains of Kordofan province in western Sudan [2]. The Promastigotes of Leishmania are transmitted to human skin by the bite of a sand fly.

Leishmania then invades human macrophages and replicates intracellular. A raised, red lesion develops at the site of the bite (often weeks or sometimes years afterwards). The lesion then ulcerates and may become secondarily infected with bacteria [3].

In many species (for example, *Leishmania major*) the lesion often spontaneously heals with atrophic scarring. In some species (for example, *Leishmania viannia braziliensis*) the lesion may spontaneously heal with scarring, but then re-appear elsewhere (especially as destructive mucocutaneous lesions).

Lesions of other leishmania species may spontaneously heal and then re-appear as satellite lesions around the site of the original lesion, or along the route of lymphatic drainage [4].

The clinical picture tends to vary with the geographic location. The eruption is popular and lasts for months in Africa, whereas in India, the lesions usually start as erythematous and hypopigmented macules that enlarge into patches. Later, these asymptomatic patches may become non-ulcerative erythematous nodules [5].

Exposed parts of the body, easily bitten by the sand fly, are usually affected.

The lesion begins with a nonspecific insect bite-like, erythematous papule(s) at the site of the sandfly bite(s).

Inflammatory satellite papules may develop around the primary lesion representing a reaction to local dissemination of the parasite or its antigenic products [6,7].

Infections persisting for more than 1–2 years are regarded as chronic CL. Patients with chronic lesions have an increased morbidity not only because of the prolonged length of their illness but also because chronic lesions tend to be larger, more diverse in their clinical manifestations, and more difficult to diagnose (absence or small number of organisms in tissue), thus invoking a wide range of differential diagnosis.

Different modes of therapy were used for treatment of Cutaneous Leishmaniasis, like:

Pentavalent antimony [8], Amphotericin B & Liposome Amphotericin B [9].

Pentamidine isethionate, Topical paromomycin [10], Oral antifungals [11], Allopurinol [12], Heat and cryo-therapy, Excision [13], Substituted Quinolines [14].

Recently lasers were suggested as treatment tool due to its successful Clinical applications that show high potential effectiveness in treating soft tissue injury, chronic pain, and wound healing. Resolution of viral and bacterial infections has been claimed, but no plausible mechanism for this has been proposed. One clinical application of interest is the treatment of inflammation, where the possible anti-inflammatory effect of location-and-dose-specific laser irradiation is promising [15].

This work aimed to evaluate the clinical results of treatment of cutaneous leishmaniasis using LLLT, with specific laser parameters.

Material and Methods

Thirteen patients were selected for this study. The Inclusion criteria were:

- Confirmed cutaneous leishmaniasis cases.
- (Confirmed by positive smear for LD-bodies).

The Exclusion criteria were:

- Patients with negative smear for LD-bodies.
- Pregnant women.
- Patients with lesions in glandular areas.
- Patients with epilepsy.

All patients were requested to participate voluntarily and a written informed consent was done with ethical clearance from patient himself before being enrolled in the study. They were informed about the possible side effects and the hazard of laser therapy. Confidentiality of the patient was maintained. The data collection from (Questionnaire sheet) was designed, and filled for every patient (Questionnaire sheet filled by the principle investigator). This sheet was used to record detailed information about personal and medical history of the patient, the site, duration and the characteristics of the lesion, the laser parameters and complication in each follow up visit. Analysis was done using Excel Worksheet Treatment was done using the single probe (820 nm) with energy density of 48 J/cm2 for thirty seconds followed by the cluster probe with energy density of 9.6J/cm2 for two minutes. The distance between the probe and the skin was less than 1cm.

The frequency of treatment was three sessions weekly for total of ten sessions.

Clinical evaluation was done by observation. Photographs for lesions before and after the treatment were used to assess the results. Follow up was done weekly during the period of treatment and up to ten weeks after the last session. Complete healing was achieved when the clinical manifestation was disappeared totally (e.g. erythema, wetness, crusting, ulceration, and pain).

Dressings and antibiotics were the only drugs used to assist in the procedure.

Results

3.1 Sex distribution of the patients:

Gender distribution of the patients included in the study showed that males (Nine patients-69%) were more affected than females (Four patients-31%). The main affected ages were between 1- 9 and 10 to 19 years.

3.2 History of the patients:

3.2.1 Concomitant Diseases: (Tabl. I)

Disease	Number	Percent
Diabetes and Hypertension	1	7.7
Chronic infections	2	15.4
Non	10	76.9
Total	13	100%

Table I. Incidence of concomitant diseases encountered by the patients included in this study

3.2.2 Drug history:
More than 60% of the patients applied topical antiseptic and antimicrobial agents. This can be explained by the nature of the disease itself, because it is wet and oozing in appearance. In addition, doctors might diagnosed the lesions as pyoderma and some health care providers even seem to believe that CL does respond to antimicrobial agents, like antibiotics and antifungal medicines.
Approximately one third of the patients used traditional medicine for the symptomatic relieve from the signs of the disease, because most of the population has a strong believe that CL does not need a specific treatment, and that the disease is self limited.

3.3 Clinical data of the patients:
3.3.1 Number of lesions: (Tabl. II)
3.3.2 Morphology of lesion: (Tabl. III)
3.3.3 Site of lesions: (Tabl. IV)

Number of Lesions / Patient	Number of Patients	Percent
1	3	23.0
2	1	7.7
3	4	30.8
4	2	15.4
6	2	15.4
15	1	7.7
Total	13	100%

Table II. The distribution of the number of lesions per individual seen on the 13 patients included in the study

Lesion	Number	Percent
Papule	2	3,6
Nodule	6	10,9
Ulcer	0	0
Crusted ulcer	32	58.2
Crusted nodule [Nodulo ulcerative]	15	27.3
Total	55	100%

Table III. The distribution of the morphological features of all the lesions seen on the 13 patients included in the study

Region of lesion	Number of lesions	Percent
Scalp	1	1.8
Face	2	3.6
Trunk	2	3.6
Upper limb	20	36.3
Lower limb	30	54.5
Total	55	99.5%

Table IV. Anatomical distribution of lesions on the 13 patients included in the study

3.3.4 Duration of lesions:
61.5% of the patients came at an early stage of the diseases. This can be explained by the fact that the majority of the patients live in Khartoum state & its surrounding areas. Additionally, these patients have an easier access to medical health care providers, who diagnose the diseases and refer them to the appropriate hospital.

3.3.5 Clinical appearance of lesions: (Tabl. V)

Appearance	Number of patient	Percent
Wet	22	40
Oozing	25	45.5
Dry	8	14.5
Total	55	100%

Table V. Incidence of the clinical appearance of lesions observed on the 13 patients included in the study

3.3.6 Associated symptoms:
Most of the patients (69%) suffered from pain, while some of them suffered from itching, fever and fatigue.

3.4 Response of CL to LLLT:
3.4.1 Number of sessions: (Tabl. VI)
Seven patients out of thirteen needed six sessions for treatment of CL by LLLT, this seems very convenient when comparing this mode of treatment to the widely used treatment with medicines, namely Pentostam (Sodium Stibogluconate, SSG.
The majority of the patients (69.2%) completed all planed treatment sessions until they achieved complete heeling. Less than one third did not complete the planed number of sessions (Defaulters). Even though they admitted that they were satisfied by the achieved results.

3.4.2 Patients satisfaction:
More than half of the patients were claimed that they well satisfied from the laser therapy almost at the third session. One quadrant admitted their satisfaction at six sessions. Latest one quadrant their satisfaction not arrived till before ten sessions. No more sessions were needed (Tabl. VII).

Number of sessions	1	2	3	4	5	6	7	8	9	10	>10
Number of patients	0	0	0	1	1	7	0	2	1	1	0

Table VI. Number of sessions done for the thirteen patients

Number of sessions	Number of patients
1-3	7
4-6	3
7-9	3
10->10	0

Table VII. Satisfaction reported by patients in relation to number of sessions

Discussion

According to the clinical evaluation, complete heeling was achieved in majority of the patients (92.3%). Moderate healing was reported in (7.7%) of patients. No failure was recorded. The results of treatment are listed in Table VIII below.

Result	Number of sessions				Total (%)
	1-3	4-6	7-9	≥10	
Complete healing	0	8	4	0	92.3
Moderate healing	0	0	0	1	7.7
Failure	0	0	0	0	0

Table VIII. Results of treatment in relation to the numbers of sessions

Number of sessions of treatment needed was proportional to the period between the detection of the lesions and the initiation of the treatment. That means the earlier the patient arrived the clinic after discovering the lesions, the shorter the time needed to achieve prognosis. A fraction of less than 25% of the patients in this study reported a mild central hypo-pigmentation and peripheral hyper-pigmentation, a color change that is known to vanish with time. None of the patients complained of, nor showed any signs of burns, erythema, tissue inflammation, nor do even any sign of tissue atrophy (Tabl. IX).

The results of this study showed that Low Level Laser Therapy used for Cutaneous Leishmaniasis was a very successful, safe procedure and avoid serious side effects when performed properly. Figures 1 and 2 show examples for some treated lesions.

Side effect	Number	Percent
Hyper-pigmentation	0	0
Hypo-pigmentation	1	7.7
Hyper and Hypo-pigmentation	3	23.1
Burns	0	0
Erythematic	0	0
Inflammation	0	0
Atrophy	0	0

Table IX. Incidence of side effects observed after LLLT in the 13 patients included in the study

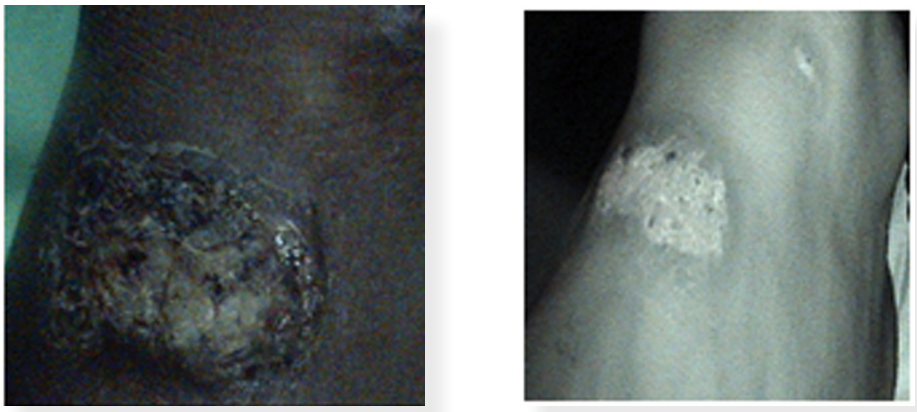

Figure 1a. Before treatment. b. After 6 laser sessions

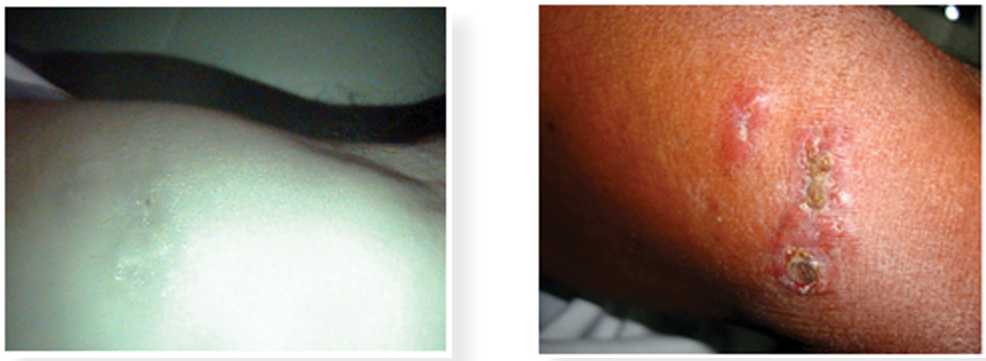

Figure 2a. Before treatment. b. After 6 laser sessions

The response of patients with Cutaneous Leishmaniasis for Low Level Laser Therapy was excellent in the majority of treated patients (92.3%) in a relative short time. LLLT proved to be successful, safe procedure and the complications were minimal and transient. The clinical response was not related to demographic data (age, sex and tribe) or specific factor (family relation, morphology and site of the lesion) but an obvious relation was established for the duration of the lesion in respect to the number of sessions needed.

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IMMUNOLOGIC FINDINGS IN CENTRAL CENTRIFUGAL CICATRICIAL ALOPECIA

IMMUNOLOGICZNE WYNIKI W CENTRALNYM OŚRODKOWYM BLIZNOWACIEJĄCYM ŁYSIENIU

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Abstract
Introduction: Premature desquamation of the inner root sheath is described as a defining histologic feature of follicular degeneration syndrome/central centrifugal cicatricial alopecia; moreover, the immunological features of this disease are not well established.
Case report: A 46-year-old African American female was evaluated for an asymptomatic scarring alopecia after using several chemicals on her hair. The clinical examination revealed visible, well defined patches of hair loss.
Methods: Biopsies for hematoxylin and eosin examination, as well as for direct immunofluorescence and immunohistochemistry analysis were performed. We evaluated molecules involved in signaling of growth factor pathways (e.g. the Akts), specifically VEGF and Oct-4 to investigate involvement of these molecules in this disease. Hematoxylin and eosin staining demonstrated histopathologic findings of premature desquamation of the inner root sheath and eccentric thinning of the follicular epithelium, supporting the diagnosis of central centrifugal cicatricial alopecia. Direct immunofluorescence revealed strong depositions of IgG, Complement/C3 and fibrinogen around the multiple hair follicles and their supply vessels. Immunohistochemistry staining of the base of the hair follicle was seen with fibrinogen and Oct-4 antibodies. Immunohistochemistry also demonstrated increased expressions of VEGF around supply vessels of the hair follicle, as well as some overexpression of anti-human Akt-pS473 phosphorylation site specific antibody.
Conclusions: Our immunologic findings suggest that the etiology of centrifugal cicatricial alopecia includes not only hair traction, but also a possible reactive immune response.

Streszczenie
Wstęp: Cechą definiującą histologiczne rozpoznanie zespołu degeneracji mieszka włosowego / centralnego odśrodkowego bliznowaciejącego łysienia; jest przedwczesne oddzielanie się wewnętrznej pochewki (włosa). Co więcej podłoże immunologiczne prowadzące do tego procesu nie zostało dobrze poznane.
Opis przypadku: 46-letnia afro-amerykanka została oceniona pod kątem bezobjawowego bliznowaciejącego łysienia powstałego po zastosowaniu kilku substancji chemicznych na jej włosy.
Metody: Wykonano biopsje oraz barwienia hematoksyna - eozyna (H-E), a także bezpośrednie preparaty na badania immunofluorescenyjne i immunohistochemiczne. Oceniliśmy cząsteczki związane z sygnalizowaniem działania czynnika wzrostu (np. Akts), a zwłaszcza udział VEGF i Oct-4 w tej chorobie. Barwienia H-E wykazały histopatologiczne cechy pod postacią przedwczesnego łuszczenia się wewnętrznej pochewki korzenia oraz odśrodkowe zcienczenie mieszka włosowego, które to potwierdzają diagnozę centralnego, odśrodkowego bliznowaciejącego łysienia. Metoda immunohistochemiczna ujawniła znaczne depozyty IgG, składnika C3 komplementu dopełniacza oraz fibrynogenu wokół wielu mieszków włosowych oraz ich naczyń zaopatrujących.
Wnioski: Nasze badania immunologiczne tej jednostki chorobowej sugerują nie tylko etiologię związaną z mechanicznym wyrywaniem włosów przez ich pociąganie, lecz także sugerują możliwe podłoże immunologiczne.

Key words: central centrifugal cicatricial alopecia; embryonic stem factor; Oct-4; follicular degeneration syndrome; immunofluorescence; Akt
Słowa klucze: centralne ośrodkowe bliznowaciejące łysienie; macierzysty czynnik embrionalny; Oct-4; zespół zwyrodnienia mieszka włosowego; immunofluorescencja; Akt

Abbreviations and acronyms: Immunohistochemistry (IHC), hematoxylin and eosin (H&E), central centrifugal cicatricial alopecia (CCCA), vascular endothelial growth factor (VEGF), octamer-binding transcription factor 4 (Oct-4).

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Introduction

Follicular degeneration syndrome, also known as central centrifugal cicatricial alopecia is a form of scarring alopecia which is most often clinically first visible as a well defined patch of diffuse hair loss [1-3]. The affected region frequently, although not always, extends centrifugally from the scalp vertex. The disease area may gradually expand in size with time [1-3]. Skin biopsies show that central centrifugal cicatricial alopecia involves inflammation of the affected hair follicles and early desquamation of the hair follicle internal root sheath [1-3].

Case report

A 46-year-old African American female was evaluated for alopecia following several chemical treatments on her hair. The clinical examination revealed visible, well defined patches of diffuse hair loss. The affected region extended centrifugally from the scalp vertex. The patient denied any other systemic disease, and multiple laboratory tests were negative including antinuclear antibodies (ANAs). Biopsies for hematoxylin and eosin (H&E) examination, as well as for direct immunofluorescence (DIF) and immunohistochemistry (IHC) analysis were performed.

Direct immunofluorescence (DIF): In brief, skin cryosections were prepared, and incubated with multiple fluorochromes as previously reported [3-8]. We utilized normal skin as a negative control, obtained from aesthetic plastic surgery patients. We utilized FITC conjugated Immunoglobulins A, E, G, M and Complement/C3 from Dako (Carpinteria, California, USA). We also tested for molecules involved in signaling by the growth factors pathway (e.g. the Akts), specifically VEGF and embryonic stem factor marker Oct-4 (Cell Signaling Technology, Danvers, Massachusetts, USA) to determine involvement of these molecules in this disease. We utilized Alexa 647 (Invitrogen, Carlsbad, California, USA) as a secondary DIF antibody fluorochrome.

Immunohistochemistry (IHC): IHC was performed as previously described. We utilized antibodies against human monoclonal rabbit anti-human Akt-pS473 phosphorylation site specific antibody and monoclonal mouse anti-human VEGF; these antibodies were also obtained from Dako [3-8].

Results

Microscopic description:

Hematoxylin and eosin (H&E) staining demonstrated histopathologic findings of premature desquamation of the inner root sheath and eccentric thinning of the follicular epithelium, supporting the diagnosis of central centrifugal cicatricial alopecia (CCCA). The overall anagen:telogen ratio appeared within normal limits. Focal perifollicular, concentric fibrotic scarring was observed, approximating five (5) per cent of the biopsy area. A mild to moderate perivascular and peri-infundibular inflammatory infiltrate was also noted, with attendant loss of hair follicular units (Fig. 1, 2). The infundibular epithelium was also noticeably atrophic. Focal dermal follicular stelae scars were identified. A Verhoeff elastin special stain confirmed the extent of scarring within the dermis (Fig. 2).

Direct immunofluorescence revealed strong depositions of Complement/C3, fibrinogen and some IgG around the hair follicles and surrounding supply vessels. The immune reactivity colocalized with the presence of Oct-4. Immunohistochemistry demonstrated increased some overexpression of VEGF and anti-human Akt-pS473 phosphorylation site specific antibody in supply vessels around the hair follicles.

Discussion

Follicular degeneration syndrome was also previously known as central progressive alopecia, or hot comb alopecia. The entity was first identified in African-American women, and thought to be secondary to heat of hot combs and oil pomades [1-3,9]. It was originally thought that the oils applied to the hair were heated by the hot comb. The liquid oil was then believed to stream down the hair fiber into the hair follicle opening and irritate the skin, causing inflammation around the upper hair follicle [1-3,9]. Nevertheless, it is now known that although hot combing might elicit follicular degeneration syndrome in some individuals, the disorder may also occur in the absence of any cosmetic procedure.

The clinical differential diagnosis of follicular degeneration syndrome is extensive and includes the following disorders: lichen planopilaris, frontal fibrosing alopecia, fibrosing alopecia in a pattern distribution, central pseudopelade of Brocq, traction alopecia, secondary systemic scarring alopecia (e.g lupus), trichotillomania, chemotherapy alopecia, alopecia mucinosa, keratosis pilaris atrophicans, mycosis fungoides, perifolliculitis capitis abscedens et suffodiens, tufted hair folliculitis, acne keloidalis, acne necrotica, erosive pustular dermatosis of the scalp, pressure alopecia, lipedematous alopecia, and senescent alopecia [1-3,9].

Our immunofluorescence and immunohistochemistry studies reveal that some immune response against the hair follicles and/or their vessels is present. The autoantibodies colocalize with Oct-4, previously characterized as a protein in humans encoded by the POU5F1 gene, and critically involved in the self-renewal of undifferentiated embryonic stem cells [10]. We thus speculate that the hair follicle embryonic cell population could be altered in this disease, although we have analyzed only one case.

Activated Akt phosphorylated protein (also known as Akt2 and protein kinase B; antibody Akt-pS473) functions as an important regulator of various cell processes including apoptosis, proliferation, differentiation and metabolism. We found that this protein seems to be overexpressed around the hair shafts and vessels [11]. Akt2 is a critical downstream effector of PI3-kinase, which mediates signal transduction when initiated by a variety of stimuli including hormones, growth factors and cytokines [11]. Some authors have shown that Akt2 is a determinant of postnatal hair follicle development [11]. Thus, we suggest that more cases of this disease should be compiled to study the significance and colocalization of the immune response around the hair follicles, utilizing our study markers and other antibodies.

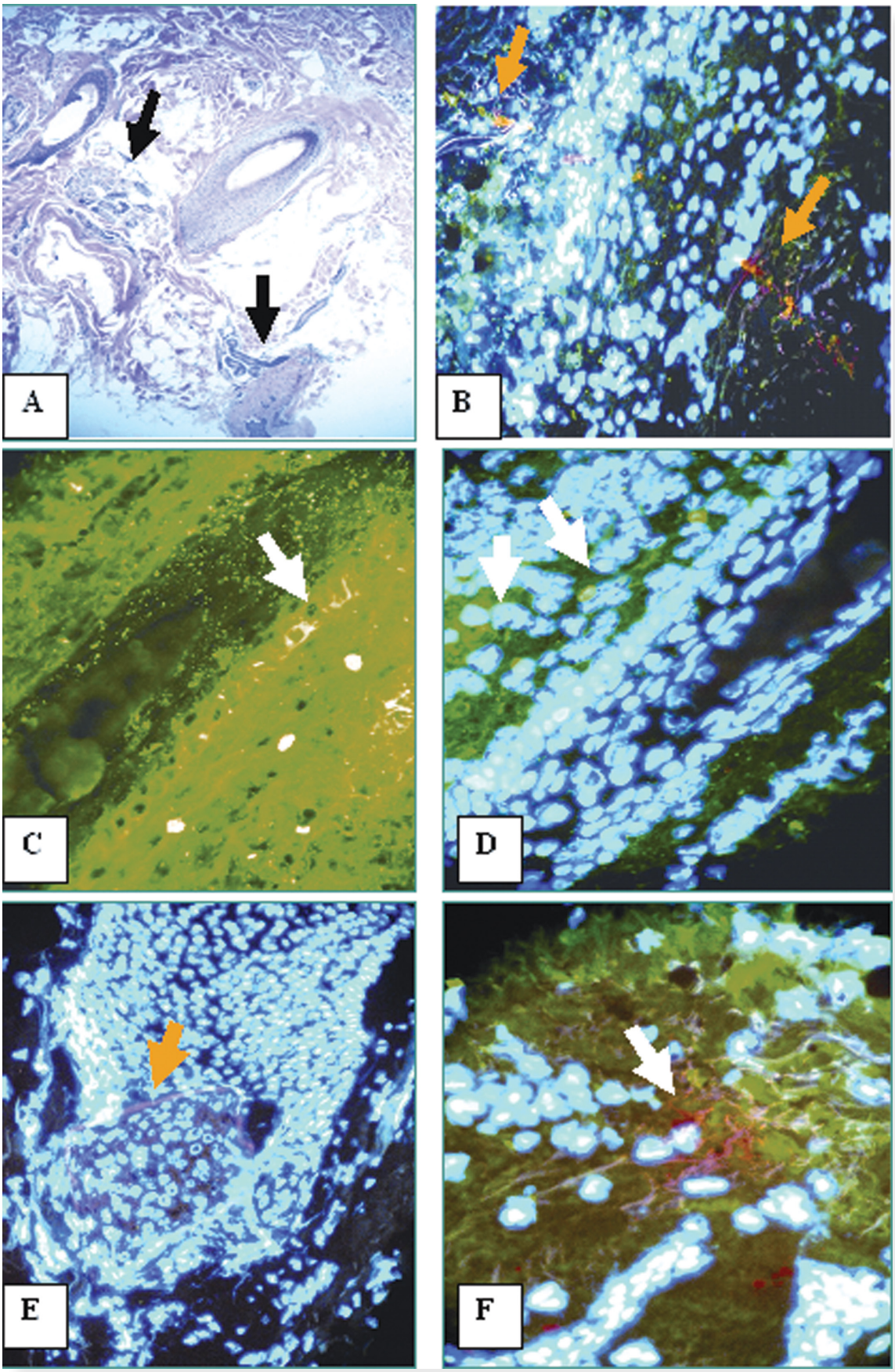

Figure 1. a. The H&E demonstrates the presence of fibrosing alopecia and atrophy of multiple hair follicles (black arrows) (100X). In **b.** DIF double staining with FITC conjugated anti-human fibrinogen (green staining) and Alexa 647 conjugated Oct-4 (red staining) around several vessels surrounding a hair follicle. The follicle nuclei are counterstained with Dapi (light blue/white). In **c**. DIF using FITC conjugated complement/C3 (green staining; white arrow) that shows positivity around a hair shaft and also in some cells around the hair follicle. In **d.** similar to 1c. but using Dapi nuclear counterstaining. In **e.** note positivity in the hair bulb with Oct-4 (red staining; yellow arrow). In **f.** note positivity around hair follicle supply vessels using Oct-4 (red staining; white arrow).

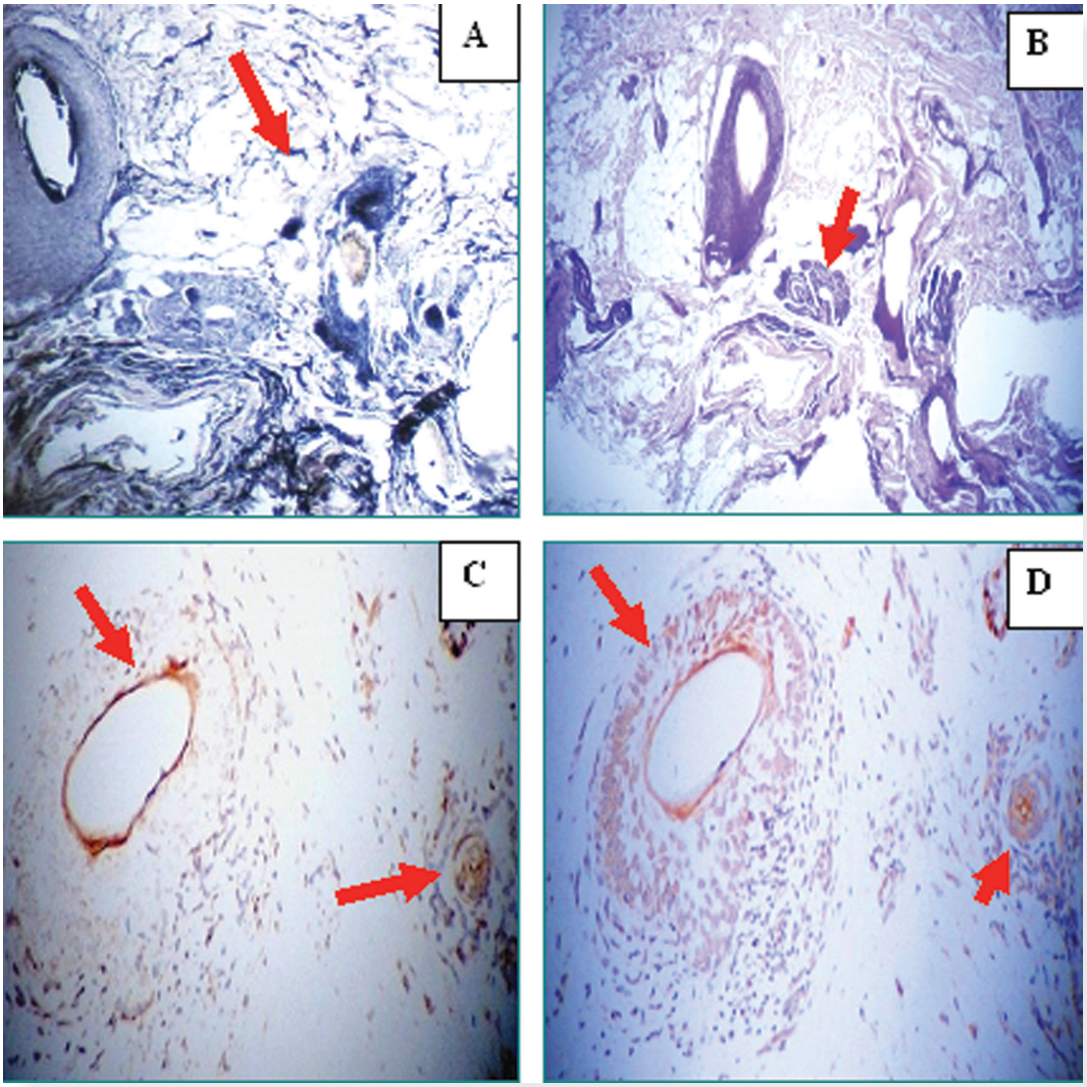

Figure 2. a. Shows a Verhoeff elastin stain, with the red arrow pointing to an atrophic hair follicle. In Figure **b.** H&E staining shows atrophic hair follicles. In **c.** and **d.** VEGF and Akt-pS473 are overexpressed by IHC at the hair follicle and surrounding vessels, respectively (dark staining; red arrows).

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ENIGMATIC NODULES ON THE SKIN – A CASE PRESENTATION

ENIGMATYCZNE GUZKI NA SKÓRZE – PREZENTACJA PRZYPADKU

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Abstract

Among many skin coloured solitary or multiple nodules of the skin with minimal or no symptoms, cutaneous leiomyomas are unique in its clinical presentation, histopathological features and clinically confusing the dermatologists. Though very rare, few cases of segmental cutaneous leiomyomas have been reported in the medical literature.

Cutaneous leiomyomas are rare benign tumors of the skin. Leiomyomas in other regions of the body are on records. Various modalities have been used to relieve pain in such lesions. This presentation highlights the importance of biopsy in all nodular skin lesions of the skin to identify and confirm the disease, plan for the correct treatment, answer questions from the affected patients and to teach dermatology post graduates. Histopathological features of other nodular skin lesions are compared.

Streszczenie

Wśród wielu kolorowych pojedynczych lub mnogich guzków skóry z minimalnymi lub bez objawów, skórne mięśniaki są jedyne w swoim obrazie klinicznym, gdzie cechy histopatologiczne i kliniczne są mylne dla dermatologów. Choć występują bardzo rzadko, kilka przypadków segmentowych mięśniaków skóry opisywano w literaturze medycznej. Skórne mięśniaki są rzadkimi, łagodnymi nowotworami skóry. Mięśniaki w innych rejonach ciała są również dokumentowane. Różne sposoby wykorzystywane były w celu łagodzenia bólu w takich zmianach. Prezentacja ta podkreśla znaczenie biopsji we wszystkich guzowatych zmianach na skórze w celu identyfikacji i potwierdzenia choroby, plan działania naprawczego, pytania i odpowiedzi od chorych pacjentów i nauka absolwentów dermatologii. Porównywane są histopatologiczne cechy innych guzowatych zmian skórnych.

Key words: leiomyoma; erector pili muscle; „Eel"like nuclei; h-caldesmon; MCUL1
Słowa klucze: leiomyoma; mięsień napinający włos; jądro podobne do„Eel"; h-caldesmon; MCUL1

Introduction

Multiple nodular lesions on the skin of human beings create a sense of confusion to the examining dermatologist. He has to rule out many documented nodular skin lesions in an orderly manner before arriving at a final clinically acceptable diagnosis. Even then, he keeps two or more diagnosis for exclusion by the available biochemical and histopathological aids. He will not know till the end what surprise is awaiting him, until when the histopathology shows an entirely different scenario than what he thought of. One among such nodular skin lesions on the human skin, which many dermatologists will never think of while diagnosing clinically is "Cutaneous Leiomyomas"(CL).

Case Report

A 30 yrs old married woman, came to the dermatology department with skin colored nodules on her right upper back near the shoulder and over the right forearm (Fig. 1-3). They were slowly evolving, one by one adjoining each other for nearly 3 months.

The nodules were skin colored, soft, dome shaped, discrete and occasionally painful. There were seven nodules on the right shoulder and multiple over the right forearm. No signs of inflammation or ulceration were seen around the nodules. The nodules over the forearm were slightly hyper pigmented and also discrete. The skin over the nodules was not pinchable, not attached to the underlying structures.

Elsewhere her skin, hairs, nails and mucous membranes were normal. Gynaecological examination was normal.

Clinically the following diagnoses were considered

1. Neurofibroma
2. Neurilemmoma
3. Cutaneous mastocytoma
4. Xanthomas
5. Myxomas

Biochemical studies were normal.

Biopsy of the nodules showed the following features [1].

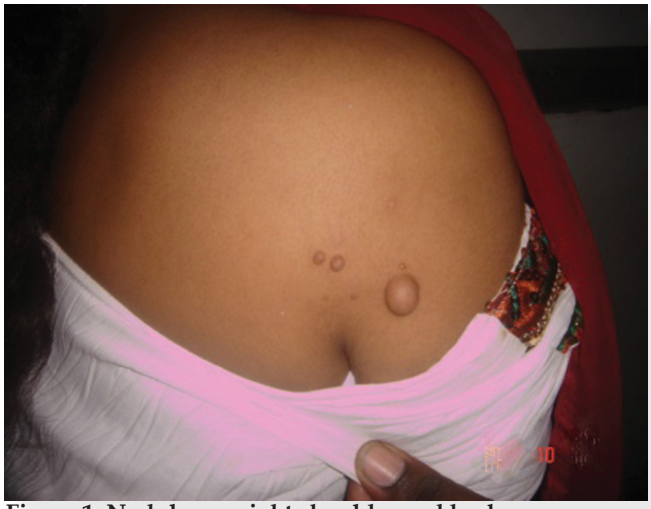


Figure 1. Nodules on right shoulder and back

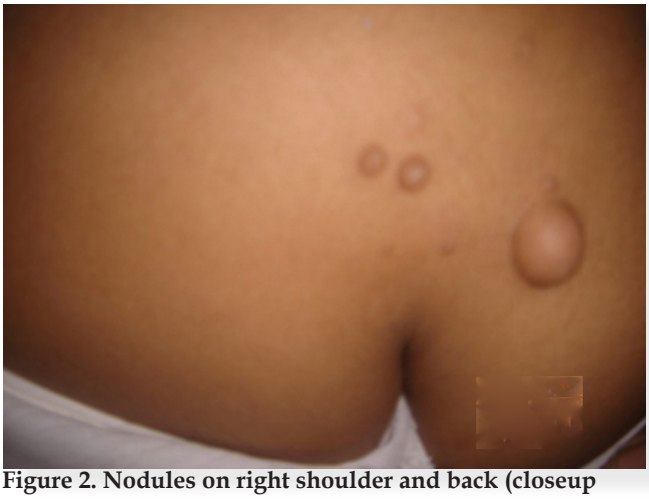


Figure 2. Nodules on right shoulder and back (closeup view)

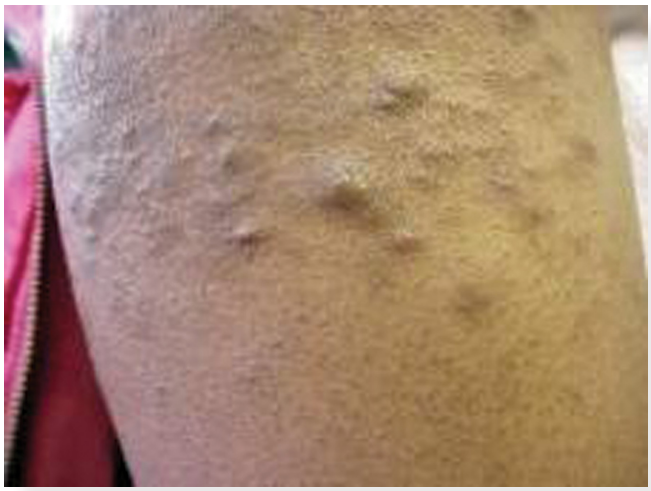


Figure 3. Multiple, slightly hyperpigmented nodules – Right forearm

Figure 4. Picture of eel

1) Eels are long snake like scaleless marine or fresh water fishes.
2) They migrate from fresh water to salt water to spawn.
3) They lack pelvic fins.

Box I.

1. Bundles of smooth muscle interlacing in the dermis (Fig. 4.5).
2. Muscle bundles were straight.
3. Centrally located, thin, very long, blunt edged, "eel-like" nuclei (Fig. 4,6).
4. Intermingling of varying amounts of collagen.
5. No nuclear hyperchromasia, pleomorphism or mitosis.

The nodules on the right shoulder were excised and the outcome was excellent. After six months there were no signs of any recurrence on the excised site or elsewhere.

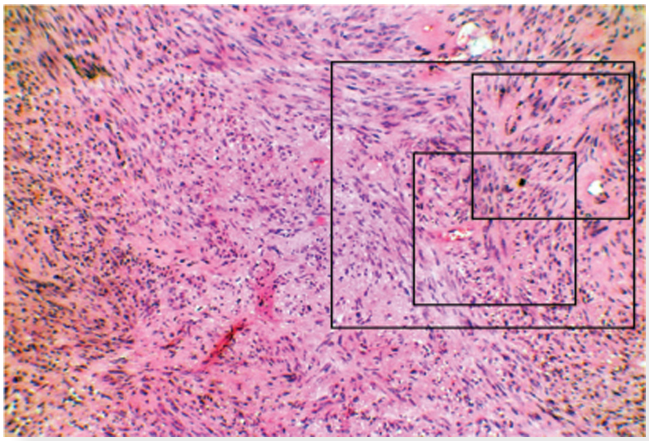


Figure 5. Histopathology of the nodule showing bundles of smooth muscle interlacing in the dermis

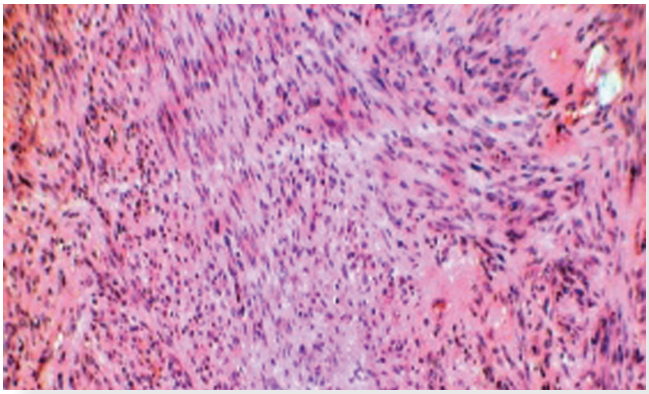


Figure 6. Close up view of Figure 4

Discussion

Cutaneous leiomyomas (CL) are benign tumors originating from the erector pilli muscles. They can develop wherever smooth muscle is present.

They have equal distribution in both sexes. It has a benign clinical course, and most often presents as multiple cutaneous lesions. Nevertheless, some cases have been described in which single cutaneous leiomyoma appear and even cases in which they appear in families [2-4]. There are reports that associate cutaneous leiomyomatosis to tumors located in other organs, specifically in the uterus and kidneys [5-7]. Pleomorphic adenomas (PA) of the parotid are the most frequently found benign tumors of the major salivary glands [8] and their simultaneous appearance with CL has been reported.

First described by Virchow in 1854 [9].

Cutaneous Leiomyomas (CL), are usually not considered while examining any skin coloured nodule with minimal pain, which is sometimes ignored by the patient and missed by the dermatologist. Cutaneous leiomyoma has received little attention in the recent literature.

The histopathological features of Cutaneous Leiomyoma is typical and striking to the eyes which are trained to look for it.

Hereditary form causes multiple leiomyomas [10], noted by Kloepfer et al in 1958 [11].

Malignant transformation probably does not occur.

Three major types of cutaneous leiomyomas exist:

1. Piloleiomyomas, are believed to arise from the errector pili muscle.
2. Angioleiomyomas, originate from smooth muscle (tunica media) within the walls of arteries and veins.
3. Genital leiomyomas.

- They correspond to the histological or anatomic site.
- Menses or pregnancy, temperature and pressure are supposed to be trigger factors for pain [12,13].
- CL are benign tumors that can be exquisitely painful [12,14-16].
- Pathogenesis of pain associated with these lesions is still a mystery.
- The histological findings do not show that prominent nerve fibers are associated with these tumors.
- Others have theorized that specific infiltrating cells may play a role.
- Yet others have suggested that muscle contraction may be pivotal in the induction of pain.
- Genital leiomyomas tend to be the least common of the 3 types.
- Cutaneous leiomyomas with histolopathologic features of uterine symplastic leiomyoma (USL) have also been reported [17,18]. Symplastic leiomyoma is an atypical uterine leiomyoma with cytologic atypia [19].
- Associated morbidity may be due to spontaneous lesional pain, as well as pain evoked by cold and/or tactile hypersensitivity. Additionally, multiple piloleiomyomas have the potential to be cosmetically disfiguring.
- A racial predilection had not been reported.
- The incidence of piloleiomyomas in men and women appear to be equal.
- Symptomatic lesions often necessitate treatment to alleviate discomfort in affected patients.
- Many options are inadequate or create substantial morbidity.
- The search continues for various methods of treatment like CO2 laser ablation, liquid nitrogen cryo, botulinum toxin, nitrous oxide cryo and enucleation [4,20-23].
- A case of cutaneous leiomyomas (CL) arising in a pleomorphic adenoma (PA) of the parotid gland. PA and CL are benign tumours arising from the parotid gland and the erector pilli muscle, respectively [1].
- Cutaneous leiomyomas are more likely to occur in adults than in children.
- Isolated reports of cutaneous leiomyomas in children also exist.
- The most common feature in patients with multiple piloleiomyomas is pain [14,24].

Complications:

1. Erythrocytosis associated with skin leiomyomas [8,25].
2. Pain.

Distribution patterns:

- Bilaterally symmetric,
- Grouped,
- Dermatomal,
- Linear patterns,
- Piloleiomyomas develop in the superficial dermis, therefore it is fixed to the skin.

Recent Research:

- The location of the gene for transmission of dominantly inherited, multiple cutaneous piloleiomyomas associated with uterine leiomyomas in female family members [5].
- As reported by Alam et al., the locus is named MCUL1 (Multiple Cutaneous and Uterine Leiomyomata) [5,26].

Problem in contemporary pathology is:

- The classification and distinction of spindle cell soft tissue tumours of skin.
- Markers such as alpha smooth muscle actin (alpha-SMA) and desmin, considered specific for smooth muscle cell (SMC), have been shown to be expressed in variety of fibroblastic and myofibroblastic processes.

Conclusion

1. Cutaneous Leiomyomas are rare, most of the time not thought of by dermatologists till the histopathology confirms it. Problem in contemporary pathology is the classification and distinction of spindle cell soft tissue tumors of the skin.
2. Markers such as alpha-smooth muscle actin (alpha-SMA) and desmin, considered specific for smooth muscle cell (SMC), have been shown to be expressed in a variety of fibroblastic and myofibroblastic processes. High-molecular-weight caldesmon (h-caldesmon), one of two isoforms, is reported to be expressed exclusively by SMC and shown to be a specific marker of SMC tumors.
3. h-caldesmon is a specific marker of fully differentiated smooth muscle and that it can serve to differentiate spindled SMC soft tissue tumors of the skin from tumors of myofibroblastic and/or fibroblastic origin.

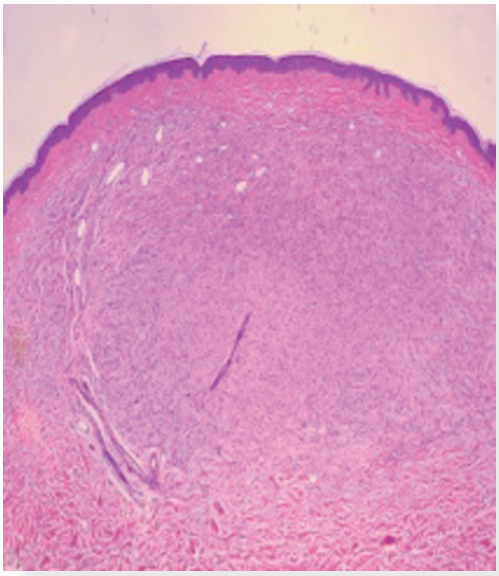
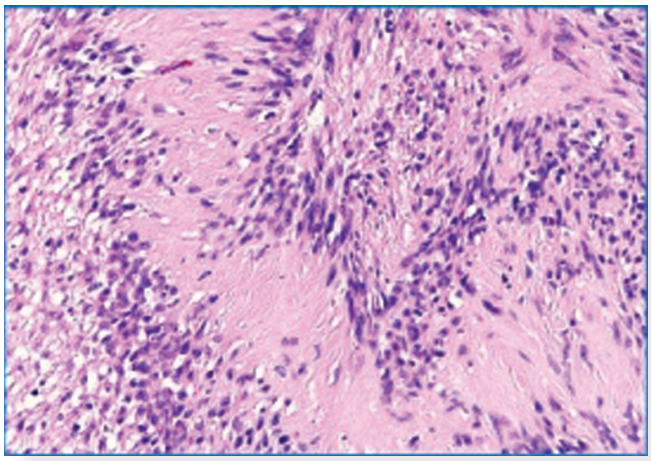
Neurofibroma	Neurilemmoma
	
1. Characteristic round, thin-walled vessels and the mixed nature of the tumor cells. 2. There is no cytologic atypia or mitotic activity. 3. Thin spindle cells associated with thin, wavy collagen bundles. 4. Loosely spaced in clear or mucinous matrix.	1. Small groups of fibrils surrounded by rows of palisaded nuclei. 2. Nuclei in two parallel rows enclosing between them a space nearly homogenous anucleate material.

Table I.Neurofibroma vs Neurilemmoma (HP showing Verocay body)

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KAWASAKI DISEASE WITH PERIPHERAL GANGRENE AND AUTOAMPUTATION - AN EXTREMELY RARE COMPLICATION: A CASE REPORT

CHOROBA KAWASAKI Z OBWODOWĄ ZGORZELĄ I AUTOAMPUTACJĄ - NIEZWYKLE RZADKA KOMPLIKACJA: OPIS PRZYPADKU

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Abstract
Kawasaki disease (KD) is a form of acute vasculitis involving small and medium sized arteries. Ischemic necrosis of the extremities is a very rare and potentially severe complication of KD. Here we present a case of KD with peripheral gangrene and auto amputation, which is the first such case to be reported from India and the fourteenth case in medical literature. A one year old child with KD and peripheral gangrene was started on aspirin. The digital gangrene resolved on follow up with amputation of the tip of the right index finger.

Streszczenie
Choroba Kawasaki (KD) to forma ostrego zapalenia naczyń z udziałem małych i średnich tętnic. Martwica kończyn jest bardzo rzadkim i potencjalnie ciężkim powikłaniem KD. Poniżej prezentujemy przypadek KD z obwodową gangreną i autoamputacją, który jest pierwszym takim przypadkiem zgłoszonym w Indii i 14-tym przypadkiem w literaturze medycznej. U rocznego dziecka z KD i zgorzelą obwodową rozpoczęto leczenie aspiryną. Obserwowano zgorzel palców z amputacją końcówki prawego palca wskazującego.

Key words: amputation; digital gangrene; Kawasaki disease; vasculitis
Słowa klucze: amputacja; zgorzel; choroba Kawasaki; zapalenie naczyń

Introduction

Kawasaki disease (KD) is a form of acute vasculitis involving small and medium sized arteries. Ischemic necrosis of the extremities is a very rare and potentially severe complication of KD. Here we present a case of KD with peripheral gangrene and auto amputation, which is the first such case to be reported from India and the fourteenth case in medical literature [1].

Case Report

A one year old boy was admitted with history of fever for 20 days associated with maculopapular rash, irritability, congestion of the oral cavity and conjunctiva, and peripheral gangrene for the previous one week. The fever was moderate grade, intermittent, associated with irritability. Five days later, the child developed a maculopapular rash which started over the extremities and later became generalised. It was associated with redness of the oral cavity, lips and the eyes which resolved over a period of one week. Gangrene of the toes and the fingers was noticed 10 days after the onset of fever. On examination the child had a height of 75 cms and weight of 8 kgs. He was irritable, febrile (100.6F) with a heart rate of 130/min and BP of 106/68mm hg. All his pulses were well felt with a capillary filling time of less than 3 seconds. Left sided cervical lymphadenopathy was present along with pallor and bilateral pedal and dorsal oedema. There was desquamation of the skin over the upper and lower extremities with gangrene on the right index finger and bilateral great toes. Blackish discoloration was noticed over other toes and fingers (Fig. 1). Systemic examination did not reveal any sign. Child was discharged following 7 days of antibiotics. At follow up the child was afebrile, not irritable and showed autoamputation of the tip of the right index finger (Fig. 2). The other sites of peripheral gangrene had resolved. Investigations revealed a decreased ESR, CRP, Total WBC and platelet count. Treatment was changed over to low dose aspirin for a period of 6 weeks and was gradually stopped.

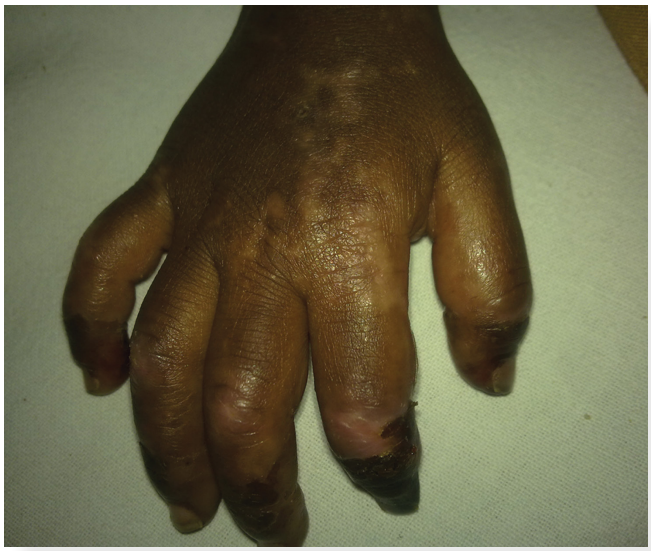

Figure 1. Peripheral gangrene of the fingers

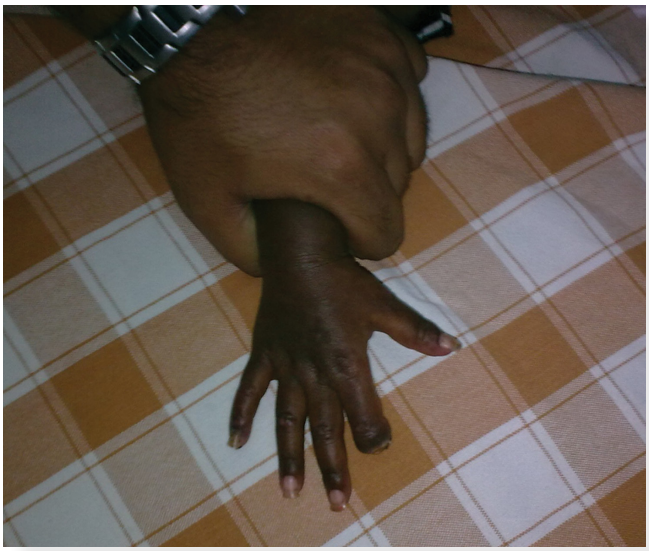

Figure 1. Auto amputation of the tip of the right index finger at follow up

Discussion

Kawasaki disease is an acute systemic vasculitis involving the medium-to-small arteries of young children. Thirteen cases of KD with peripheral gangrene and auto amputation have been reported in the literature; only two of them were Asian children, and none from India [2]. The previous cases of KD complicated with peripheral gangrene were younger than 7 months of age while our patient was around one year of age [3, 4].

A majority of the reported cases eventually suffered from some serious complications, including two deaths. Therefore, peripheral gangrene in KD indicates an underlying severe systemic vasculitis, and predicts serious sequela, especially to the heart. Our case did not have any cardiac involvement even during follow up and did not suffer any other life threatening complication.

Various medications have been tried for treating peripheral gangrene in KD, including heparin, warfarin, urokinase, dipyridamole, nitroprusside, glyceryl trinitrate, sympathetic or caudal block, prostacyclin, and prostaglandin E1 [4]. In our case only aspirin was used as the child could not afford Intravenous immunoglobulin (IVIG). The only sequela to his peripheral gangrene was an amputation at the tip of the right index finger while his peripheral gangrene resolved.

In conclusion since peripheral gangrene in KD is associated with high incidence of amputations and death, an early diagnosis and effective treatment is recommended in all such cases.

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SUCCESSFUL TREATMENT OF INFUNDIBULOFOLLICULITIS WITH TOPICAL TRETINOIN. REPORT OF A CASE

SKUTECZNE LECZENIE INFUNDIBULOFOLLICULITIS ZA POMOCĄ MIEJSCOWEJ TRETINOINY. OPIS PRZYPADKU

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Conflicts of interest: None

Abstract
First described in 1968 by Hitch and Lund. Disseminated and Recurrent infundibulofolliculitis (DRIF) is an uncommon eruption characterized by recurrent, pruritic follicular papules commonly seen on the trunk and proximal extremities. Rarely limited to the neck. It is much more common in black population but has also been reported in other ethnicities including Caucasians. Its etiology has constantly been debated with a few authors describing it as a variant of atopic dermatitis while others have refuted the same as a family history of atopy is not present. Various others have classified DRIF as variants of follicular eczema, along with lichen spinulosus and juxtaclavicular beaded lines. Its treatment also varies with different authors claiming response to steroids, isotretinoin, UVA therapy, keratolytics and tetracyclines.
We describe a case of localized infundibulofolliculitis of the neck diagnosed in a 21 year old female who was successfully treated with a course of topical tretinoin 0.025%. The presenting history, clinical findings, biopsy results and available literature are reviewed.

Streszczenie
Po raz pierwszy opisany w 1968 przez Hitch i Lund. Rozsiane i nawracające infundibulofolliculitis (DRIF) jest rzadkim schorzeniem charakteryzującym się nawracającymi, swędzącymi grudkami przymieszkowymi powszechnie występującymi na tułowiu i proksymalnych częściach kończyn. Rzadko ogranicza się do szyi. Jest o wiele bardziej powszechne wśród czarnej ludności, ale DRIF opisywano także u innych narodowości, w tym u rasy kaukaskiej. Jego etiologia jest stale przedmiotem dyskusji kilku autorów opisujących go jako wariant atopowego zapalenia skóry, podczas gdy inni twierdzą, że wywiad rodzinny w kierunku atopii nie jest obecny. Jeszcze inni klasyfikują DRIF jako wariant rogowacenia mieszkowego, wraz z liszajem kolczystym i „juxtaclavicular beaded lines". Również różnie podchodzi się do leczenia DRIF; autorzy stwierdzają dobre odpowiedzi po sterydach, izotretynoinie, UVA terapii, środkach keratolitycznych i tetracyklinie.
Opisujemy przypadek zlokalizowanego infundibulofolliculitis na szyi rozpoznanego u 21 letniej kobiety, który został skutecznie leczony kursem miejscowej 0,025% tretinoiny. Przedstawiana jest historia schorzenia, objawy kliniczne, wyniki biopsji i dostępna literatura.

Key words: infundibulofolliculitis; neck; Caucasians; female
Słowa klucze: lejkowe zapalenie mieszków włosowych; szyja; Kaukaz; kobieta

Introduction

First described in 1968 by Hitch and Lund [1]. Disseminated and Recurrent infundibulofolliculitis (DRIF) is an uncommon eruption characterized by recurrent, pruritic follicular papules commonly seen on the trunk and proximal extremities. Rarely limited to the neck [2]. It is much more common in black population [3] but has also been reported in other ethnicities including Caucasians [4]. Its etiology has constantly been debated with a few authors describing it as a variant of atopic dermatitis [5] while others have refuted the same [6] as a family history of atopy is not present. Various others have classified DRIF as variants of follicular eczema, along with lichen spinulosus and juxtaclavicular beaded lines [7]. Its treatment also varies with different authors claiming response to steroids [1], isotretinoin, UVA therapy [8], keratolytics and tetracyclines [9].
We describe a case of localized infundibulofolliculitis of the neck diagnosed in a 21 year old female who was successfully treated with a course of topical tretinoin 0.025%. The presenting history, clinical findings, biopsy results and available literature are reviewed.

Case Report

A 21 year old girl presented to our out patient department with complaints of multiple tiny papules present around the neck since 2 years. The lesions were not associated with itching or pain.

She denied application of any topical agents or prolonged exposure to sunlight. She did not give history of atopy.

On examination, multiple, discrete hyperpigmented follicular keratotic papules and few discrete pustules were present around the neck extending from the nape of the neck, lateral and anterior aspects upto the supra sternal space (Fig. 1a,b). There were no other lesions anywhere on the body. Examination of the mucus membranes, hair and nails were normal.

Following differential diagnoses were considered:

1. Infundibulofolliculitis.
2. Pityrosporum folliculitis.
3. Kertosis Pilaris.
4. Pityriasis Rubra Pilaris.
5. Darier's Disease.

Screening for HIV and Syphilis was negative. Scrapings obtained for KOH examination were negative. A punch biopsy of the lesion was obtained which showed multiple polymorphs around the infundibular part of the hair follicle (Fig. 2a,b) suggestive of diagnosis of infundibulofolliculitis. Clinical findings and histopathology report were suggestive of infundibulofolliculitis.

The patient was treated with a two week course of topical tretinoin 0.025% followed by prompt resolution of the lesions (Fig. 3).

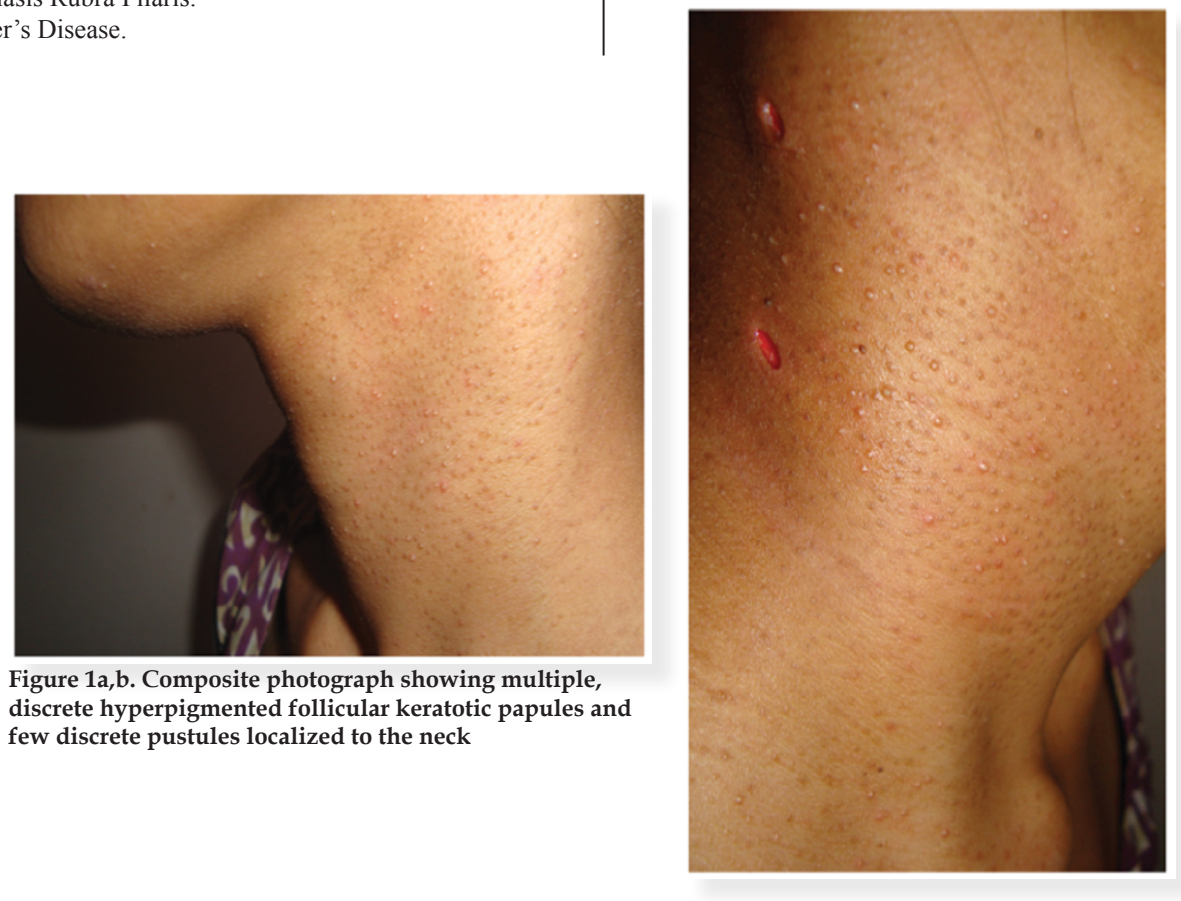

Figure 1a,b. Composite photograph showing multiple, discrete hyperpigmented follicular keratotic papules and few discrete pustules localized to the neck

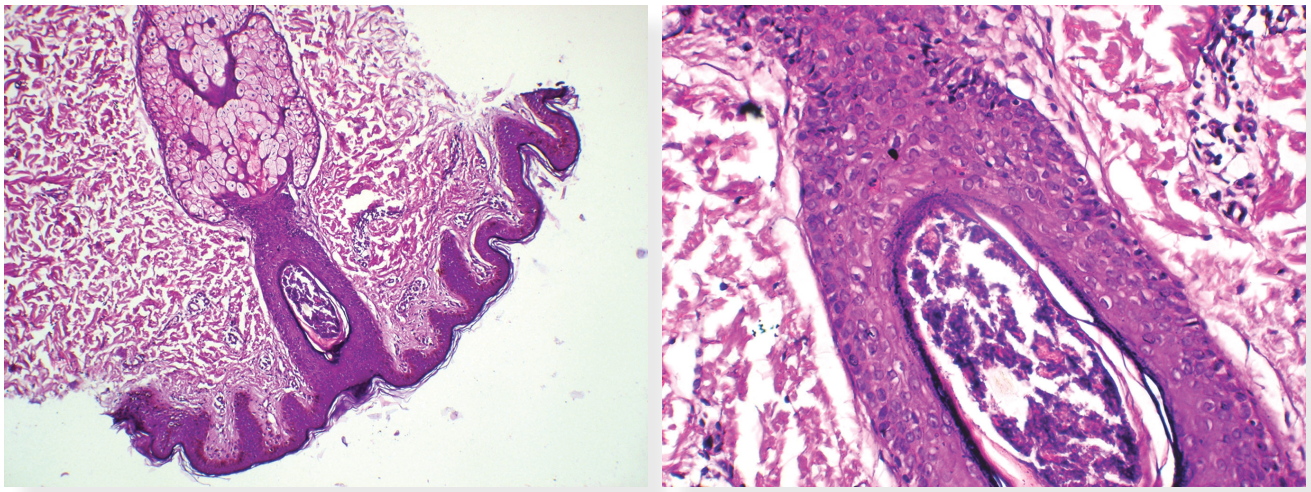

Figure 2a,b. Histopathology of biopsy H and E. (10x and 45x)

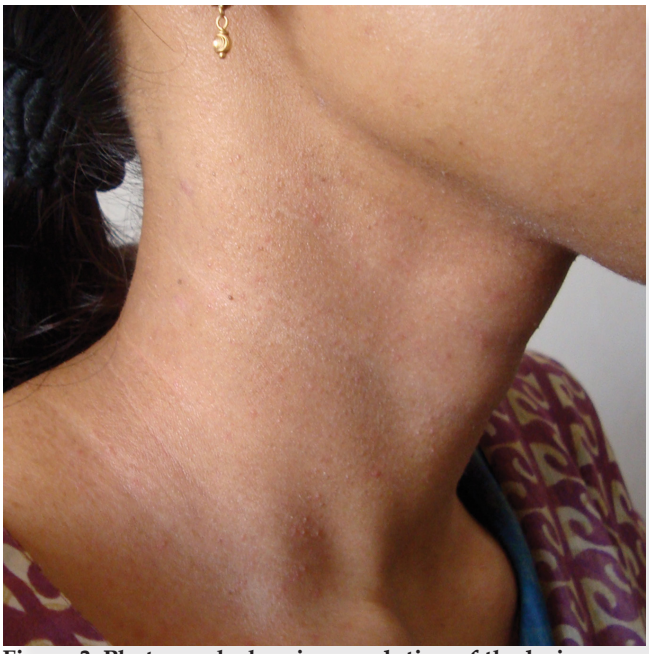

Figure 3. Photograph showing resolution of the lesions following application of topical tretinoin

Discussion

DRIF is an uncommon pruritic follicular eruption of unknown etiology that is predominantly seen in black men [3]. This condition tends to affect the trunk and the upper extremities and is usually unresponsive to local and systemic treatment.

In 1968, Hitch and Lund coined the word disseminated and recurrent infundibulofolliculitis for a patient who had presented with a diffuse, pruritic, skin colored, uniform follicular papular eruption with histologic findings of a perifollicular lymphocytic infiltrate and edema around the infundibular portion of the follicle [1]. Reports of patients with similar symptoms have been described as early as 1959 [1]. Because of the unique combination of clinical and histologic features, a consensus regarding its etiology has not been reached. An atopic etiology [5] has been suggested but refuted by many [6], as a family history is not available. An infectious etiology has also been considered [6] but given that the lesions disappear without antibiotics, it is unlikely. Also there is no evidence clinically or by biopsy of a fungal infection.

DRIF has often been reported to be resistant to treatment. Failure with bland topical agents, antihistamines, tetracyclines, keratolytics and topical retinoin acid [9] has been reported. Mixed results have also been reported with oral vitamin A alone and in combination with vitamin E [3,6]. Topical steroids [9] and tretinoin creams have shown to be of variable effectiveness.

Our patient presented with papular lesions around the neck which on biopsy was suggestive of infundibulofolliculitis. She was treated with a two-week course of 0.025% topical tretinoin and responded well (Fig. 3). She is currently asymptomatic.

Recurrent or persistent disseminated infundibulofolliculitis remains a distinct entity best understood as a specific clinicopathologic response to an unknown cause or causes. Response to therapy is poor, although we report successful therapy with.

Conclusions

1. This case is being reported for its rarity in Caucasian females and only around the neck.
2. Successful therapy with topical tretinoin.

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Dr. S Vasantha Kumar, Principal, MVJ Medical College and Research Hospital.

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NASZA DERMATOLOGIA Online
OUR DERMATOLOGY Online

HYPERKERATOSIS OF NIPPLE AND AREOLA

HIPERKERATOZA BRODAWKI SUTKA I OTOCZKI

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Conflicts of interest: None

Abstract

A 26 year old pregnant female presented with verrucous lesion on nipple and areola of 10 year duration. Histopathology of lesion was consistent with benign pappiloma, which can occur with various clinical differential dignosis which cause clinical picture of hyperkeratosis of nipple and areola. It can occur as isolated entity which is idiopathic or secondary to various causes like epidermal nevus, seborrhoic keratosis, eczema, icthyosis or acanthosis nigricans. Lesion may increase in size in pregnancy and may cause worry to patient regarding nursing the babies in our case. Lesions was removed by radiofrequency cautery.

Streszczenie

26-letnia kobieta w ciąży prezentowała brodawkujące zmiany na brodawce sutka i otoczce trwające 10 lat. Histopatologia zmiany była zgodna z łagodną brodawką, która może służyć do diagnostyki różnicowej różnych obrazów klinicznych o charakterze hiperkeratozy na brodawkach i otoczkach sutka. Może ona występować jako pojedyncza jednostka, która jest idiopatyczna lub wtórna z różnych przyczyn, takich jak znamię naskórkowe, rogowacenie łojotokowe, wyprysk, icthyosis lub rogowacenie ciemne. Zmiana może zwiększyć rozmiar w ciąży i może sprawiać zakłopotanie pacjenta w zakresie opieki nad niemowlęciem, tak jak w naszym przypadku. Zmiany zostały usunięte przez przeskórne przyżeganie.

Key words: hyperkeratosis; nipple; areola

Słowa klucze: rogowacenie; brodawka sutkowa; otoczka

Introduction

Hyperkeratosis of nipple and areola can occur as an idiopathic isolated condition or secondary to localised dermatosis like epidermal nevus or seborrhoeic keratosis or generalised diseases like icthyosis, eczema or acanthosis nigricans. Histopathology of lesion often help in differentiating conditions to some extent. Removal of lesion by cautery, radiofreequency or laser is curative.

Case Report

A 26 year old female primigravida of 16 weeks presented with asymptomatic hyperpigmented warty growth over right nipple of 10 years duration. There is increase in size of lesion since 1 month. Patient has not taken any treatment for this before. She was worried about recent increase in size of lesion and whether it will affect breast feeding. Examination revealed hyperpigmented verrucous plaque with pappillomatous projections over the right areola and adjacent skin (Fig. 1). Left nipple was normal. No other significant skin lesions any where else on body. With the differential diagnosis of hyperkeratosis of nipple and areola, (HKNA) epidermal naevus, seborrhoeic keratosis and verruca vulgaris, an incisional biopsy of lesion was performed. Histopathological examination showed hyperkeratosis, elongated rete ridges pappilomatosis and keratotic plugging with normal dermal architecture (Fig. 2). Histopathological feature is suggestive of benign pappiloma which can be found in most of cases of epidermal nevi seborroeic keratosis old verruca vulgaris and in nevoid HKNA. Thus clinical picture is necessary to arrive at final diagnosis. Based on clinical and histopathological features diagnosis of HKNA type 1 secondary to epidermal nevus was made. Lesion was removed with radiofrequency cautery under local anaesthesia.

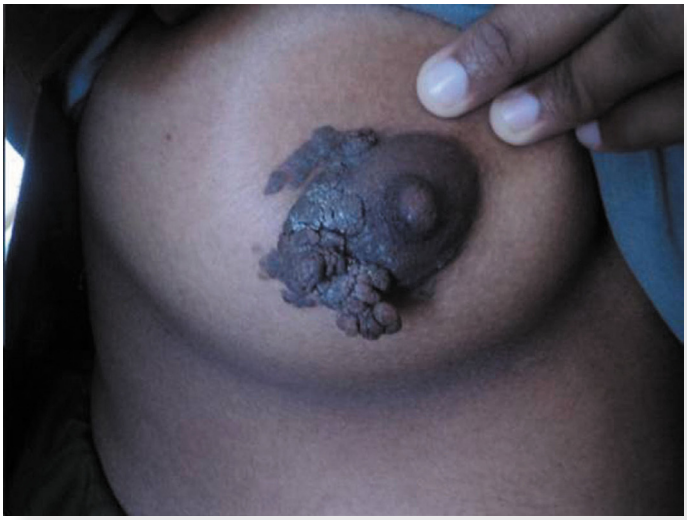

Figure 1. Hyperpigmented verrucous growth over the right areola and adjacent skin

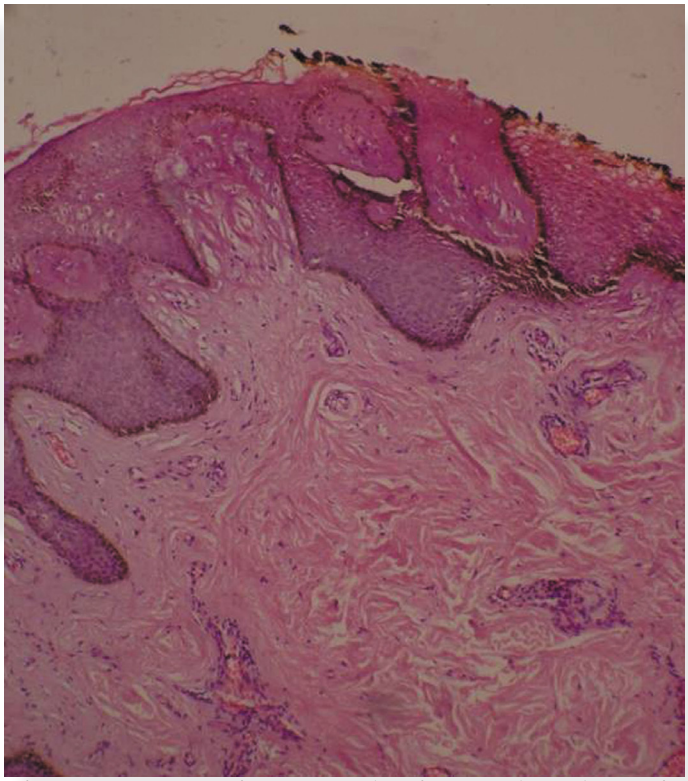

Figure 2. Biopsy showing hyperkeratosis, elongated rete ridges, papillomatosis and keratotic plugging with normal dermal architecture. (H&E – 40x)

Discussion

Hyperkeratosis of nipple and areola (HKNA) is an uncommon benign asymptomatic acquired condition of unknown pathogenesis [1]. Most of the cases are bilateral although unilateral cases can occur [2]. Clinically there is verrucous thickening of nipple and areola. There are usually no associated systemic or dermatological conditions. A change in oestrogen levels has been thought to precipitate this condition [3]. HKNA is seen more in females at puberty and pregnancy and also in males receiving hormonal therapy for prostate cancer [3]. There are doubts on whether n hyperkeratosis of the nipple and areola is a distinct entity or a clinical presentation of various dermatoses. In 1938, Levy and Frankel described three distinct types of hyperkeratosis of the nipple and areola. Type I was hyperkeratosis as an extension of an epidermal nevus, type II was hyperkeratosis associated with other dermatoses (e.g., acanthosis nigricans, icthyosis, lymphoma, chronic eczema, seborrhoeic keratosis or Darier's disease), while type III was nevoid hyperkeratosis, not associated with an epidermal nevus or other dermatoses [4]. Perez-Izquierdo suggested an alternative classification of two types: idiopathic or nevoid, which may be unilateral or bilateral. Other type is secondary to local lesions like verrucous nevus or seborrheic keratosis; or associated with other diseases which include ichthyosis, Darier's disease, acanthosis nigricans chronic eczema, lymphomas, or drug related e.g., diethylstilbestrol and spironolactone [5]. Mehanna et al. suggested that the term ‚nevoid' be replaced by ‚idiopathic' [6]. A histopathological examination is mandatory to rule out dermatological conditions and malignancies The main cause of concern for patients of HKNA is the cosmetic appearance of the nipple and areola. and in some difficulty in feeding the baby. Various keratolitic agents like retinoic acid5 and calcipotriol [7] are found effective. Simple shave excision [8], radiofrequency [9], cryotherapy and co2 laser [10] is also effective.

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ATOPIC DERMATITIS AND HOMEOPATHY

ATOPOWE ZAPALENIE SKORY A HOMEOPATIA

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Abstract

Introduction: Atopic dermatitis (AD) is a chronic, relapsing disorder of the skin associated with allergen sensitization and impaired barrier function. There is often a family history of pruritic skin disease or asthma. **Materials and Methods:** Three cases of atopic dermatitis treated with homeopathy are presented. Case 1 is a case of a 22-year-old female, with AD since early childhood, which had not responded to standard topical therapy. She received several homeopathic medicines, with transitory effect until she finally received the medicine Aurum metallicum, at M potency. At present, 1 year after cessation of treatment, she remains lesion-free. Case 2 is a case of a 10-month-old baby with a an 8-month history of itchy rash and poor sleep, that had failed to respond to treatment. The patient was given the homeopathic medicine Lachesis at C30 potency and responded. The rashes receded and the patient was able to sleep better at night. Case 3 is a case of an 11-month-old boy with a 3-month history of itchy rash, diagnosed as having AD and treated with topical steroids. After 3 months of unsuccessful treatment, the patient was brought in for homeopathic therapy. He received the homeopathic medicine Lachesis, at C30 potency. He improved under this treatment and is currently lesion-free, 6 months after cessation of treatment. **Conclusions:** Three cases of atopic dermatitis that failed to respond to treatment were given homeopathic therapy and responded adequately. The patients remained free of lesions even after cessation of treatment.

Streszczenie

Wprowadzenie: Atopowe zapalenie skóry (AZS) jest przewlekłą, nawracającą chorobą skóry związaną z alergią i upośledzeniem funkcji barierowej. W wywiadzie rodzinnym stwierdzić można świąd skóry lub astmę. **Materiał i metody:** Prezentowane są trzy przypadki atopowego zapalenia skóry leczonego homeopatią. Przypadek 1 jest przypadkiem 22-letniej kobiety z AZS od wczesnego dzieciństwa, które nie odpowiedziały na standardowe leczenie miejscowe. Otrzymała kilka leków homeopatycznych, z krótkotrwałym efektem, do czasu kiedy przyjęła lek Aurum metallicum w potencji M. Obecnie 1 rok po zakończeniu leczenia, pozostaje bez zmian chorobowych. Przypadek 2 to 10-miesięczne dziecko z 8-miesięczną historią swędzącej wysypki skórnej i zaburzeniami snu, nie odpowiadała na leczenie. Pacjentka otrzymała lek homeopatyczny Lachesis w potencji C30 i pozytywnie zareagowała. Zmiany skórne ustąpiły, a pacjentka mogła lepiej spać w nocy. Przypadek 3 to 11-miesięczny chłopiec z 3-miesięczną historią swędzących zmian skórnych, u którego zdiagnozowano AZS i traktowano miejscowymi kortykosteroidami. Po 3 miesiącach nieudanego leczenia, pacjent został poddany homeopatii. Otrzymał homeopatyczny lek Lachesis, w potencji C30. Chłopiec poprawił się w ramach tej terapii i jest obecnie wolny od zmian skórnych, jest 6 miesięcy po zakończeniu leczenia. **Wnioski:** Trzy przypadki AZS, nie odpowiadające na leczenie poddano homeopatii z dobrą odpowiedzią końcową. Pacjenci pozostali bez zmian skórnych nawet po zaprzestaniu leczenia.

Key words: atopic dermatitis; eczema; homeopathy; pruritus; lachesis;Aurum metallicum
Słowa klucze: atopowe zapalenie skóry; wyprysk; homeopatia; świąd; lachesis; Aurum metallicum

Introduction

Atopic dermatitis (AD) is a chronic, relapsing disorder of the skin associated with allergen sensitization and impaired barrier function. There is often a family history of pruritic skin disease or asthma [1]. It appears to be commoner in urban than in rural areas and in the industrialised as opposed to less industrialised countries. In India, the prevalence was 2.4-6% of 37000 children [2], and 8.5% of 1019 south-eastern Nigerian patients had AD [3]. Others suggest an incidence of AD of 15-20% of children in industrialised nations [1]. The exact cause or causes of AD are unknown. However, breastfeeding for at least 3 months appears to reduce the risk in infants of atopic dermatitis [4]. Genetic factors may play a role, as there is a higher rate (77%) amongst monozygotic twins than in dizygotic twins (15%) [5]. Several loci on genes have been linked to atopic dermatitis [5]. There is also an increased concentration of inflammatory cytokines, including interleukins, in atopic skin [6].

Material and Methods

Three patients with atopic dermatitis that had failed to respond to previous treatments were treated homeopathically by this author. Their characteristics and response to treatment are presented.

Patient 1

A 22-year old economics student presented with an erythematous rash on her upper lip, associated with itching (Fig. 1-4). Her past medical history at this visit was not significant, but a later visit revealed a history of itchy rash since early childhood and migraine headaches. After several visits, in which she received homeopathic treatments, the symptoms appeared to worsen with increased rashes, which only showed transitory improvement. Finally, she received the homeopathic medicine *Aurum metallicum* at M potency. The rash disappeared and, 1 year after this treatment, she only has residual mild dry skin around the neck area. The migraines have diminished greatly in intensity and frequency also.

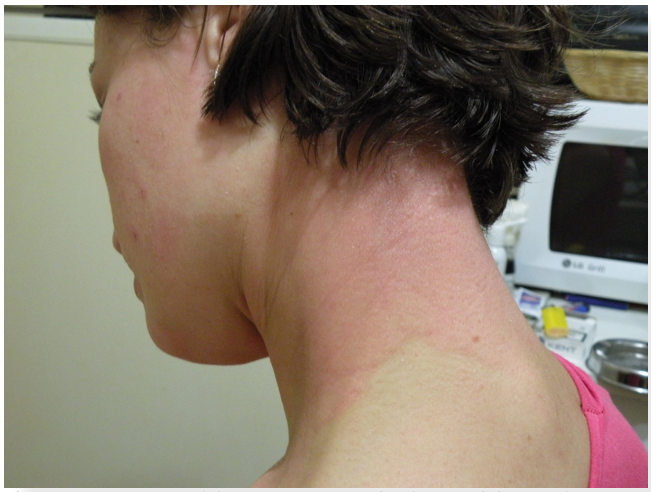

Figure 1. 22-year old woman - atopic dermatitis on the neck - before treatment

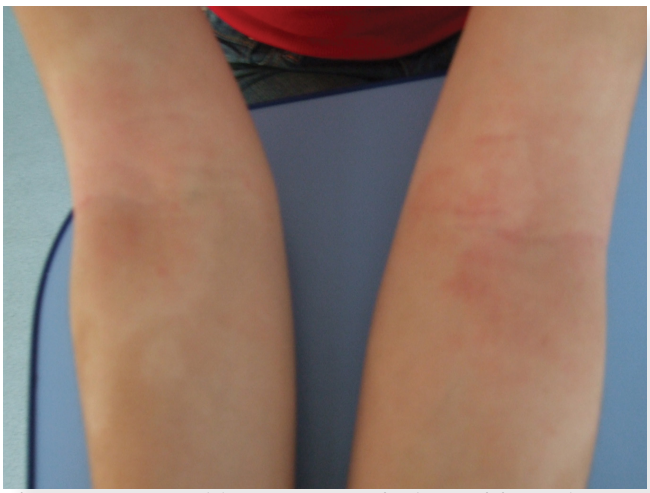

Figure 2. 22-year old woman - atopic dermatitis on the hands - before treatment

Patient 2

A 10-month old baby girl presented with a generalized, itchy rash (Fig. 5-7).

The itch was aggravated by heat and by bathing (the mother had read somewhere that bathing with very warm water was healthy). The patient frequently woke at night and had restless sleep.

Past medical history revealed a history of dry skin in the mother and parapsoriasis in the maternal grandfather. All developmental milestones were met and vaccinations had been given on schedule.

On examination, the child had erythematous, squamous patches, with some weeping lesions that also affected the flexural and extensor areas of the limbs, the face and neck. She received the homeopathic medicine *Lachesis* at C30 potency. The patient continued to improve under this treatment and, 6 months after cessation of treatment, continues to remain almost completely lesion- and symptom-free. The patient has also resumed normal sleep patterns.

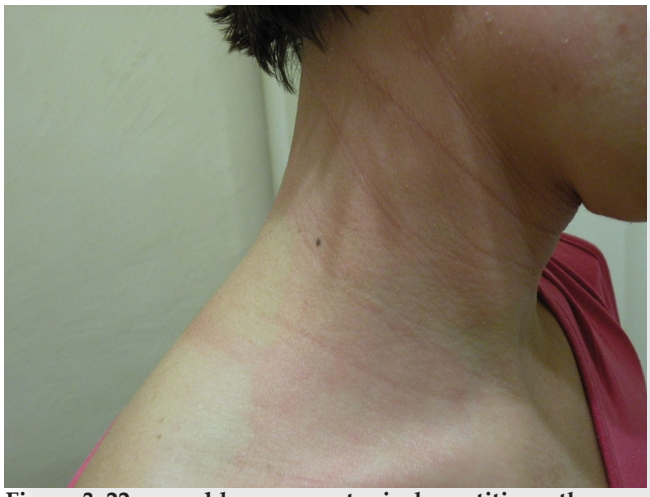

Figure 3. 22-year old woman - atopic dermatitis on the neck - before treatment

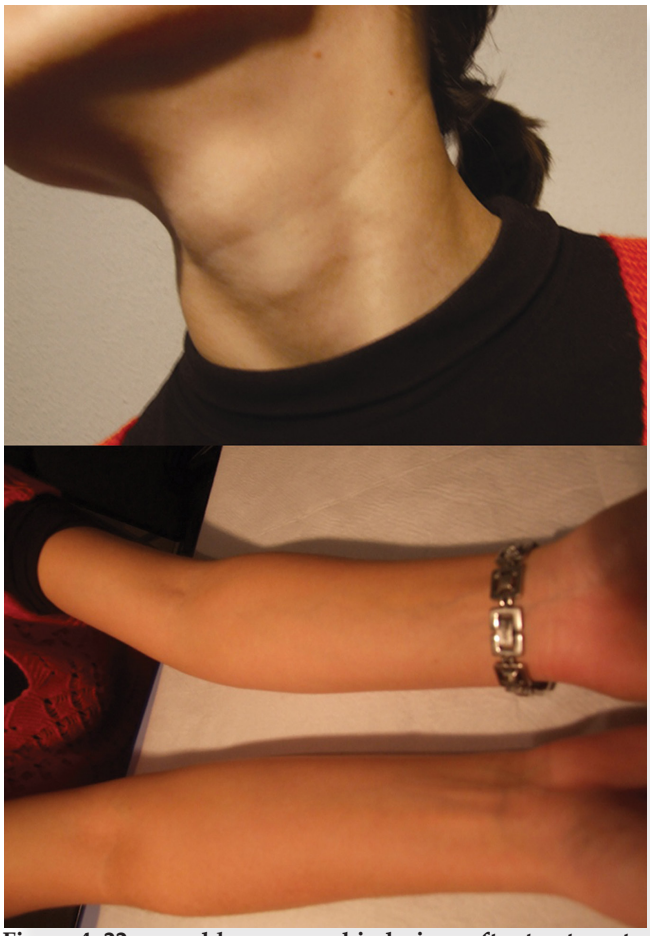

Figure 4. 22-year old woman - skin lesions after treatment

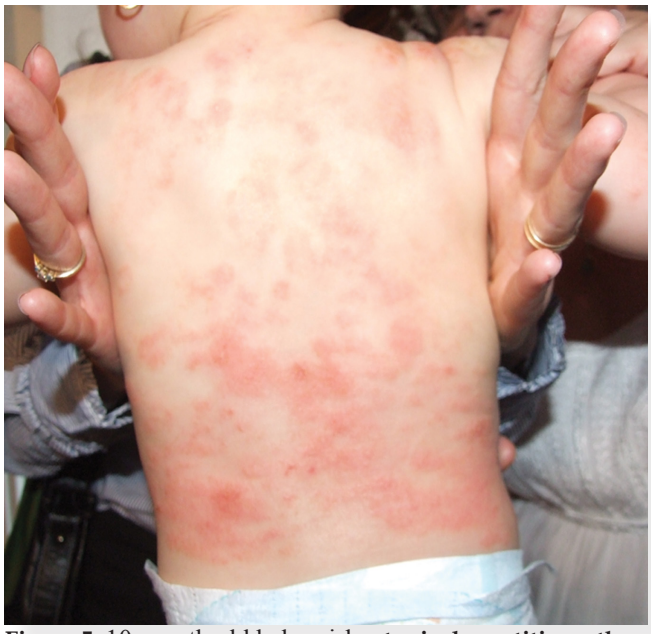

Figure 5. 10-month old baby girl **- atopic dermatitis on the back - before treatment**

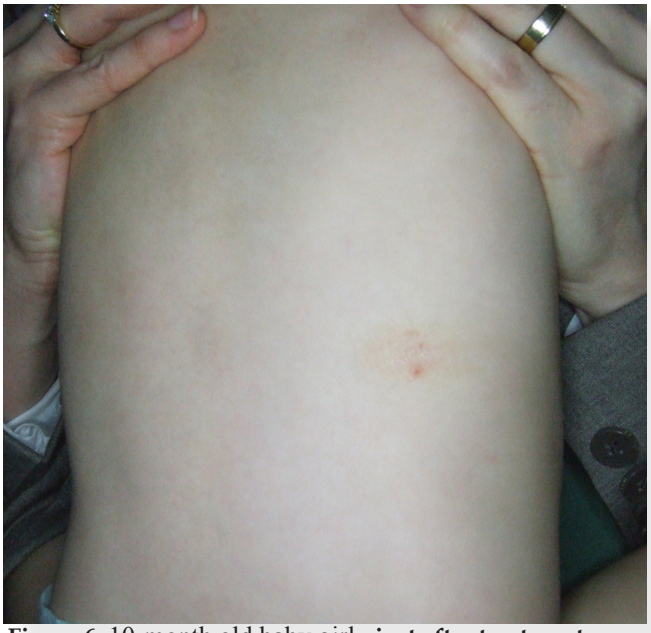

Figure 6. 10-month old baby girl **- just after treatment**

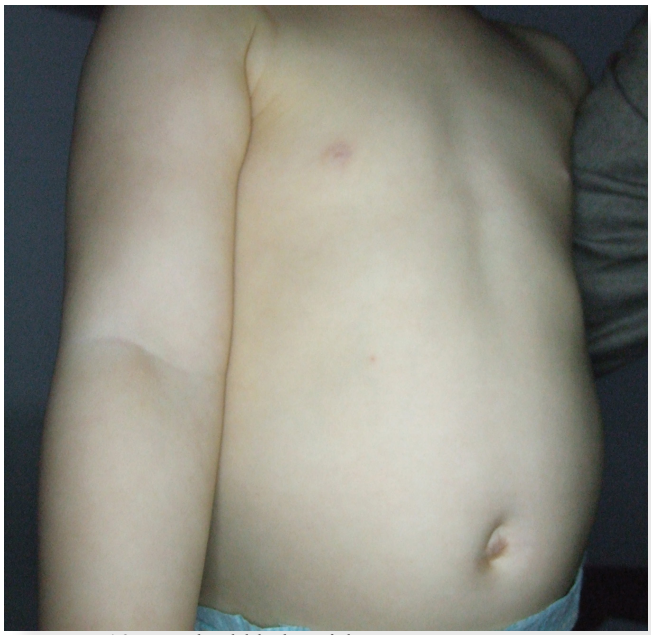

Figure 7. 10-month old baby girl **- 6 months after cessation of treatment**

Patient 3

An 11-month old baby boy presented with a 3 month history of generalized itchy rash (Fig. 8,9).

There was no family history suggestive of atopy. All developmental milestones were normal and vaccinations were up to date.

On examination there were generalized, erythematous, squamous plaques, concentrated in the upper chest, neck and abdominal areas. There was relative facial sparing.

The patient received the homeopathic medicine *Lachesis* at C30 potency and showed improvement. He was lesion-free by one month and, 3 months later, remains free of lesions.

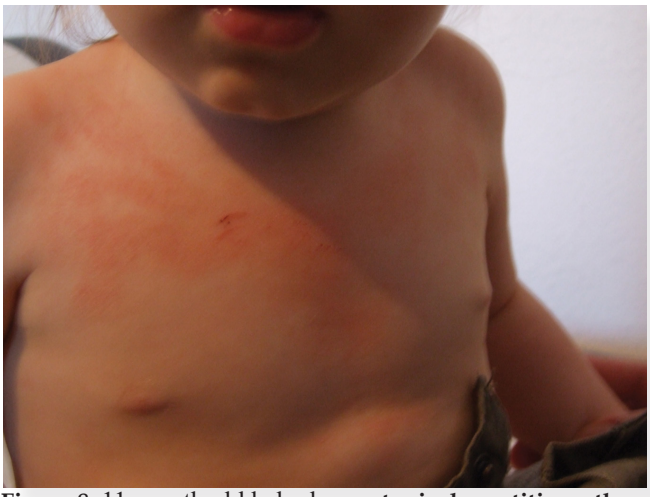

Figure 8. 11-month old baby boy **- atopic dermatitis on the thorax - before treatment**

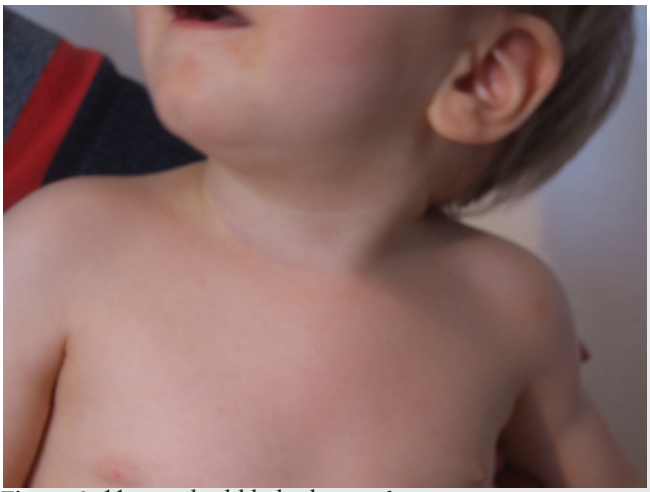

Figure 9. 11-month old baby boy **- after treatment**

Discussion

AD is a fairly common skin condition. It is commoner in industrialised nations, though its prevalence in non-industrialised countries is on the rise. Pruritus and cutaneous irritability are the most important features of atopic dermatitis. Criteria for diagnosis are major and minor. The major criteria include pruritus, rash with typical morphology and personal or family history of atopic disease. Minor criteria include xerosis (dry skin), elevated serum IgE, itchy rash in skin creases. Nipple eczema, cheilitis, Dennie-Morgan infraorbital fold and itch while sweating, amongst others [7]. Various treatment strategies are used for AD, including emmolients, topical and systemic steroids, calcineurin inhibitors, UVB therapy and probiotics [1,7,8]. In this paper, three cases of AD have been presented. They responded positively to homeopathic therapy and continued to remain well after the cessation of treatment. This author has found homeopathy to be useful for various disorders including common warts, seborrheic dermatitis, psoriasis, rosacea and melasma [8-12]. Putative mechanisms have been put forward for the mechanism of action of homeopathy [13,14]. A good review of the principles of homeopathy has recently been published and the case for the use of homeopathy by virtue of its effectiveness was made [15]. Efficacy was defined as the production of a therapeutic effect in clinical trials, while effectiveness the ability to produce an effect in clinical practice [15]. Homeopathy appears to have been clearly effective in AD, in the cases presented.

Conclusions

AD is a cutaneous, inflammatory skin disorder that is common amongst children and is on the increase in both industrialised and non-industrialised nations. Various therapeutic methods exist for the treatment of this disorder. Homeopathy is a cheap, mild form of treatment with almost no known adverse effects, which has been found useful in a number of cutaneous disorders. We present 3 cases of AD that responded to homeopathic treatment and remained in remission after cessation of treatment. Homeopathy may be a useful new treatment modality for AD and further studies and clinical trials are required to establish whether it may be efficacious in the therapy of this disorder.

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THE SELECTION OF THE TYPES OF SHOES AND ITS IMPACT ON THE SKIN OF THE FEET

WYBÓR RODZAJÓW OBUWIA I JEGO WPŁYW NA SKÓRĘ STÓP

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Abstract

The shoes are important for the skin of our feet. It is protecting feet from injurious things in the ground. Some of the dermatoses of the feet are greatly affected by the type of the shoes a patient is wearing.

However, little attention is made on the value and functions of the shoes and its impact on our skin.

This manuscript provides some details on this topic.

Streszczenie

Buty są ważne dla skóry naszych stóp. Jest to ochrona stopy przed szkodliwymi czynnikami zewnętrznymi na ziemi. Niektóre z chorób skóry stóp w dużym stopniu zależą od rodzaju butów, które pacjent nosi.

Jednak mało uwagi zwraca się na wartość i funkcję butów i ich wpływu na naszą skórę.

Rękopis ten dostarcza kilka szczegółów na ten temat.

Key words: feet dermatoses; onychomycosis; shoes

Słowa klucze: choroby skóry stóp; onychomycosis; buty

Looking for a ''healthy shoes'', what you are going to choose?

A shoe is an item of footwear intended to protect and comfort the human foot while doing various activities [1]. Contemporary footwear varies widely in, materials the make the shoes, style, complexity and cost and varies from culture to culture with the types originally being tied to function [1]. Shoes type may include boot, Boat shoes, sandals [2], Flip-flops [3], slippers [4], and others Basic sandals may consist of only a thin sole and simple strap. High fashion shoes may be made of very expensive materials in complex construction and sell for thousands of dollars a pair. Other shoes are for very specific purposes, such as boots specially designed for mountaineering or skiing [1]. Shoes have traditionally been made from leather, wood or canvas, but are increasingly made from rubber, plastics, and other petrochemical-derived materials [1]. A slipper or house shoe is a semi-closed type of indoor/outdoor shoe, consisting of a sole held to the wearer's foot by a strap running over (or between) the toes or instep. Slippers are soft and lightweight compared to other types of footwear [4]. The foot contains more bones than any other single part of the body. It is vulnerable to environmental hazards such as sharp rocks and hot ground, which shoes can protect against [1]. People may choose to wear sandals for several reasons, among them economy (sandals tend to require less material than shoes), comfort in warm weather, and as a fashion choice [2]. Usually, people wear sandals in warmer climates or during warmer parts of the year in order to keep their feet cool and dry. The chance of developing athlete's foot is lower than with enclosed shoes, and the wearing of sandals may be part of the treatment regimen for such an infection [2].

The use of flip-flops has also been encouraged in some branches of European and North American military as sanitary footwear in communal showers, where wearing flip-flops slows the spread of fungal infections [3].

While widely regarded to be comfortable, flip-flops do not provide ankle support, and can cause many foot-related problems [3].

Improper selection of shoes might trigger several skin and orthopedic problems. By precipitating abnormal motion, shoes can result in problems from the foot up into the hips [3].

Skin Problems related to the shoes

1. *Trauma and skin injury:*

For each place (for example, a place containing water, or a mountains containing rocks and stones), there are a proper shoes to be wearied in order to protect the feet. Failure to select the optimal shoes for a given place might subject the feet for injury [3,5].

Some flip-flops, type of shoes, have a spongy sole, so when the foot hits the ground, it rolls inward and the sponge allows it to roll even more than usual. This is known as overpronation and causes many problems in the foot [3].

Poorly fitting shoes may precipitate in growing toes nails and diabetic foot syndrome [6,7]. Increased plantar pressure, especially beneath the metatarsal heads, and the resultant callus play an important role in causing diabetic foot syndrome [6,7].

2. *Skin infection:*

Humidity by a closed type of shoes might precipitate tinea pedis. A case is, also, reported in which the prolonged wearing of combat boots and damp socks caused an acutely inflamed papulopustular candidiasis of the feet [8].Wearing slippers can be used as a way to keep feet clean [4].

Deep fungal infection including Madura foot might result from injury of unprotected feet by a thorn or plant elements.

Cutaneous larva migrans, is a skin disease, manifests as an erythematous, serpiginous, pruritic, cutaneous eruption [9]. It is caused by accidental percutaneous penetration and subsequent migration of larvae of various nematode parasites. It is common among barefoot beachgoers and sunbathers [9]. However, not all the footwear's are protective against cutaneous larva migrans. A case is reported, in which a woman developed cutaneous larva migrans despite wearing, protective' footwear. The authors forwarded a hypothesis by which recently popular water shoes may actually be conducive to the development of cutaneous larva migrans rather than having a protective function [9].

3. *Contact dermatitis:*

Different components of the shoes, like rubber, glue, dyes, may cause contact dermatitis [10]. Some of the chemicals in the shoes may cause leukoderma.

Conclusion

Foot wears are important thing in the life of any person from the time that his or her feet touch the ground.

Particular shoes might be suitable to the feet of one person but not to other. Patients with feet deformities need a designed type of shoes that keep the balance of the person during walking and prevent friction or pressure to any points on the feet.

It goes without saying that torn shoes should be repaired or thrown. Walking with damaged shoes may carry a risk to the person.

People need to know that there is no,'medical shoes'' as such. Instead, there are proper shoes for each purpose [1]. Parents may teach the children the skills of selecting the right shoes for each purpose. What people describe as,'a comfort'' might not equal to,'medical'' or,'healthy'' types of shoes.

Teachers in the schools might also guide the students about the right and the wrong things about their foot wears and may provide proper advices in this matter.

The nature of the,'ground'', a person is going to walk on, is the most important factor that dictates the type of the shoes a person should wear [1].

The term,'medical shoes'' is a real myth generated by shoes industry and has no scientific base.

Particular shoes might be good in one function but not good for other. For instance, a closed type of shoes is good for a protection but also generate a humidity that facilitates the occurrence of tinea pedis.

Another example is that, some people advice the diabetics to wear slippers [4], as diabetes can have effects on blood flow to the extremities of the body. Wearing slippers can offer warmth and comfort that will allow a good flow of blood, but the problem is that, the slippers do not provide the protection needed for the feet of the diabetics.

Most styles of slipper offer little or no support for the tender arch of the human foot. This is essential to children, whose young feet are still developing. The lack of support can allow the foot to roll inwards during walking, which can cause many health issues. Of course, opposing studies suggest that the introduction of rigid heels in slippers and shoes of infants and toddlers can actually inhibit a child's ability to learn to walk as quickly as they would otherwise [3,4].

Some British schools have rules that enforce the wearing of slippers indoors. While this is a good method of regulating hygiene, some rigid-soled slippers can inhibit the correct growth of the child's developing foot. This has caused some concerned parents quite some grief. While wearing slippers can offer comfort, it can also be a danger, in both terms of walking and movement, as well as the development of the young foot [3,4].

But when different features for a shoe is in front of you remember that,'safety'', in a sense that the shoes will not affect the balance and prevent a person from falling down, and,'protection'' of the feet, from injury by the ground are far important over any other features.''Safety, first, protection second'', is a phrase to remember this advice.

In this sense, the closed type of shoes is better, provided that, you wear with it the proper socks that absorb the moisture and try to aerate the shoes from time to time by taking the shoes off.

In addition, foot care after taking the shoes, by washing then drying and using moisturizers, will overcome the problems of humidity that result from using closed shoes for long time.

The inside of shoes can be sanitized with germicidal shoe trees or other cleansing methods to prevent the growth of microorganisms such as odor-causing bacteria or fungi [1].

Selecting the proper size and weight of the shoes, that suit the person's need is also important.

Look carefully to the materials that made the shoes and try to avoid the ones that you are allergic its substances.

Selecting proper shoes are of paramount importance in some high risk medical patients specifically, diabetics. Proper shoes are, with no doubt, instrumental in preventing diabetic foot syndrome and proved to reduce the incidence of amputations in diabetic [6,7].

Diabetics should wear wide, well-fitting shoes without sutures on the inner side. The inside of the shoes should always be checked for foreign bodies or irregular surfaces before they are put on. Diabetics should not walk barefoot [6,7]. Callosities (calluses, corns) should be shown to the podologist or to the doctor.

They are always a sign of increased mechanical stress and therefore, require an adjustment of footwear. Trimming of callosities can only aim at giving symptomatic relief and does not replace an appropriate correction of the mechanical stress [6,7].

Finally, spending more on a better quality, better created shoe can influence the wearer's health and safety [1].

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THE SELECTION OF THE TYPES OF SHOES AND ITS IMPACT ON THE SKIN OF THE FEET

by Khalid Al Aboud

comment:
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The author of the article raises the problem of impact of shoes on feet (skin of feet). Healthy feet are a very important factor in maintaining food physical condition. Well-fitted shoes are a matter of great importance for soldier's fitness and his availability [1]. Because of their static and motion functions, feet have impact on good physical and mental state as well as energetic attitude to life which have a specific/special meaning when the conditions of soldier's life during war as well as at the time of peace are to be considered, because they influence the readiness for battle of the military men.

Skin diseases among soldiers have always been considered as a very important problem demanding effective prophylactics. Detailed health recommendations for soldiers were presented in 1777 in the thesis of Benjamin Rush MD "Doctor Benjamin Rush health recommendations for soldiers"[2].

Skin changes on feet, apart from causing subjective affliction, in which pain is a main factor, are accompanied by objective changes. They appear in the form of inflammatory reactions of different levels of intensification and in consequence, they lead to decrease of the level of soldier's physical condition/ fitness. Intensification of the disease process may be a cause of partial or total exclusion of the soldier from training or battle field [3].

Also causing factors like hyperhidrosis of feet as well as blood supply and innervations disturbances may be important [4]. The main cause of these damages are, in most cases, abrasions and ruptures/breaks of epidermis- results of poorly fitted shoes. Intertrigo mechanical, unguis incarnates, foot blisters, sore spots on feet, congelatio, perniosis, trench foot, clavus (black heel) and phlegmon as an effect of superinfection of skin changes can be added to the list of other skin injuries which are related to poorly fitted shoes. A very common factor which supports inflammatory states of skin of feet are mechanical injuries which are not dressed or are dressed improperly what leads to bacterial and mycosis infections.

Pitted keratolisis is a disease very common among soldiers and at the same time it is a disease typical for military environment [5,6].

This fact is emphasized by Schissel and his partners among American soldiers [7] and by Mathis among soldiers of German army [9]. Jelliffe presents similar data concerning Nigerian soldiers [8]. Brzeziński describes, that percentage of pitted ketarolisis made 51,42% of bacterial diseases and 15,03% of all skin diseases recognized among soldiers in the period 2002-2005 [5,10].

Other social groups among which pitted ketarolisis may appear as frequently are sportsmen [11], farmers [12], people working by silk production [13] and homeless persons [14].

A note of significant increase of confirmed diagnosis of mycosis of feet was taken by Zhang and his partners among Chinese soldiers after a series of sea maneuvers [15]. Noguchi and his partners recognized feet mycosis among Japanese soldiers during summer and winter trainings [16]. Similar conclusions were formulated by Suo and his partners, whose research prove that feet mycosis infections may be a serious problem for soldiers during the peace as well as at the time of war and as a result they can delay soldiers' training and decrease the level of their activity during fight/ battle [17].

Idrosis and blood supply can be a factors which cause skin changes on feet with poorly fitted shoes. This relation was pointed out by Esterman and Pilotto during their researches/ examinations of injuries and microinjuries of feet among soldiers of Australian army [18]. Similar researches/ examinations conducted by Abdel-Fattah and his partners among soldiers of Saudi Arabian army point up/emphasize/ show that feet structure disturbances (platypodia) is not a factor of great risk of elimination of soldier from military training/maneuvers [19].

Di Benedetto and his partners presented extremely interesting thermographical examination of feet of 30 soldiers. Examination was conducted before a series of exercises and after the training. They found that new shoes and discomfort which is related to them can be a cause of feet skin damages detectable through thermography [20]. It was stated during this examination that pathological changes appeared during the exercises which took place between ten and twenty days after enrollment and new, poorly fitted shoes could be a reason of analyzed diseases.

Experiences from stabilization missions in Iraq and Afghanistan show that feet protection against harmful impact of battle field factors can be strengthened only to a very little extent, however there is a possibility of minimizing the influence of mentioned harmful factors , for example, through well-fitted and modern shoes, soldier's education and proper dressing of existing, even the smallest small damages.

Every foot skin injury, even small one, which is not dressed or is dressed improperly can become a cause of serious infection and long-lasting unavailability of the soldier, which, in consequence, weakens fitness of the soldier and the readiness for battle of the fighting unit [21].

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THE SELECTION OF THE TYPES OF SHOES AND ITS IMPACT ON THE SKIN OF THE FEET

by Khalid Al Aboud

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Our feet carry our weight and take us everywhere we go, and our shoes do it as well.
The question of appropriate shoes in different skin diseases is an important topic and every dermatologist has to face it daily. Although the term medical shoes is a myth generated by shoes industry, there is a desperate need of special shoes, let call them therapeutic, in many specific skin conditions. If we only think of all, mostly elderly, with callosities, corns and calluses, that embitter their life to despair because they can not walk. How often do we see feet deformities in patients with varicose veins and leg ulcers? Do you remember the youngster with ingrowing toe nail, hyperhidrosis or pitted keratolysis who refuses to wear any other shoes than the usual rubber shoes? In my experience, these problems are very often not easily resoluble, even when the patient's condition is well defined. The causes are quite complex, depending on the patient, the therapist and the manufacturer. I am going to discuss about some of them from my daily practice viewpoint. Patients often do not like wearing adapted shoes with orthosis. They do not like how they look. Such shoes can be expensive and an unaffordable financial burden. The patients have to travel to a distant centre to procure them. The result of wearing such shoes is often disappointing, only partial and not immediate. The therapist is often not interested in dealing with feet deformities because they are not considered a primarily dermatologic problem. In many patients they are combined with other conditions and the feet problem is frequently left till the last or just forgotten. For example, if we have a patient with leg ulcers, we have firstly to evaluate the venous, arterial, lymphatic and skin conditions of the legs and consider his general condition (heart, diabetes mellitus…), than to prescribe a local ulcer and adjacent skin therapy, further on a compression therapy and eventually a systemic therapy. After that we usually do not have the time and the energy to deal with flat feet and hallux valgus. In certain patients with allergic contact dermatitis of the feet we detect the causative allergen. So we prescribe shoes without, for instance, chromium, p-tert-butyphenol-formadehyde resin, p-phenylendiamine,colophonium, mercaptobenzothiazole or other possible contact senzitizers. The questions are: Where can the patient find a specialized outlet with a qualified vendor able to advise him properly? Are the manufacturer and distributors able to guarantee their shoes are free of a certain senzitizer? Did you know that nowadays in the European Union there is no legislative regulation of the level of chromium released from leather goods? As always, the devil is in the details. Anyway, it is an important and unavoidable topic.

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MAYERSON'S PHENOMENON IN A CUTANEOUS NEUROFIBROMA

ZJAWISKO MEYERSONA W PRZEBIEGU NERWIAKOWŁÓKNIAKA

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We report the case of a 45year old normotensive, nondiabetic, policeman, smoker who presented with a two week's history of red crusted scaly lesion around a skin coloured pedunculated growth over the back. There was associated history of severe itching,but no history of trauma, pain, bleeding, application of any substance or any constitutional features. Examination revealed a well-defined annular moist erythematous scaly crusted plaque around a skin coloured nodule (neurofibroma) which had been present on the back for the past 30 years. There were multiple neurofibromas present over the back and abdomen. Besides routine testing, punch biopsy was taken for histopathological examination. Histopathology of the specimen showed spongiosis with lymphocytic infiltrate and few eosinophils which confirmed the diagnosis of mayerson's phenomenon. Topical steroid (clobetasol propionate,l/a b.d) and an antihistaminic (levocetrizine5m b.d) was prescribed and the condition resolved in a week (Fig 1, 2).

Discussion

Mayerson's phenomenon is an uncommon clinical condition that is characterised by an eczematous halo surrounding a pre-existing melanocytic naevus, and numerous other lesions like molluscum contagiosum, skin tags seborrheic warts and other elevated skin lesions. The etiology of this condition is unknown. It is hypothesised that it occurs because of the interaction between CD4+T lymphocytes and increased expression of ICAM-1.

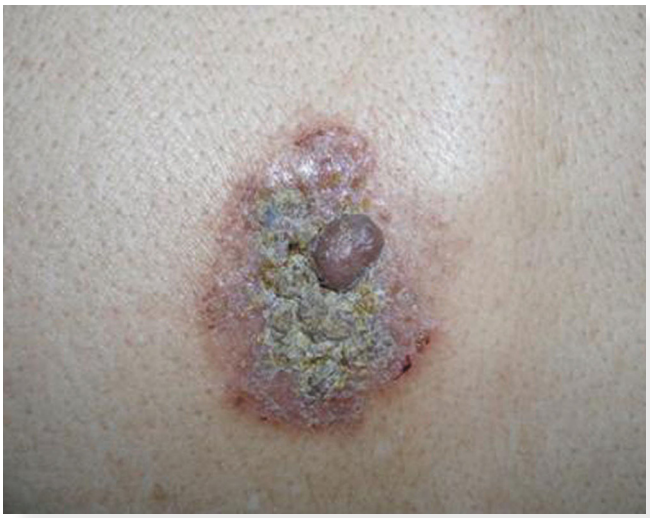

Figure 1. Mayerson's phenomenon

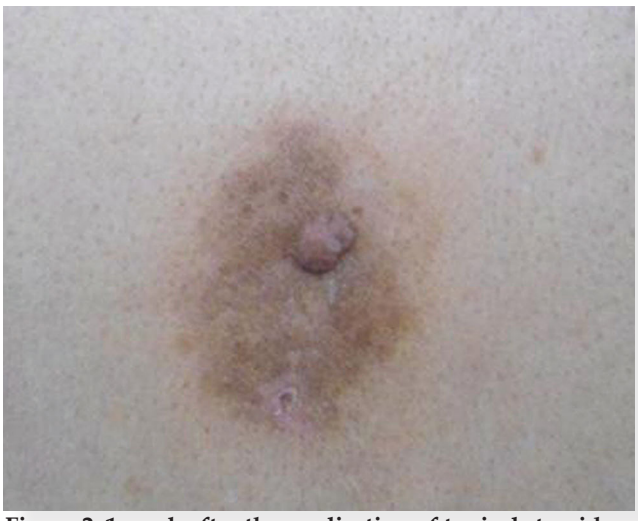

Figure 2. 1 week after the application of topical steroid

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NASZA DERMATOLOGIA Online
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DISSEMINATED SUPERFICIAL POROKERATOSIS AND ANETODERMA DEVELOPING AFTER ACUTE PANCREATITIS

ROZWÓJ ROZSIANEJ POWIERZCHOWNEJ POROKERATOZY I ANETODERMII PO OSTRYM ZAPALENIU TRZUSTKI

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Conflicts of interest: None

Sir,

Disseminated superficial Porokeratosis is a heterogeneous group of disorders characterized by a distinct clinical finding of keratotic ridge with central groove that corresponds to cornoid lamella in histology. Anetoderma is characterised by localised loss of elastic tisssue resulting in herniation of subcutaneous tissue. We describe a rare association of disseminated superficial porokeratosis and anetoderma, which was developed after an episode of acute pancreatitis.

A 56yr old male patient, farmer by occupation, was presented with multiple asymptomatic hyperpigmented annular lesions over the trunk since 8 months and multiple discrete yellowish sac like protrusions over trunk since 6 months. He gives h/o acute pancreatitis 10 months back for which he was treated conservatively. On examination there were multiple discrete annular hyperpigmented plaques of varying sizes with hyperkeratotic grooved borders over anterior and posterior aspects of the trunk and both the upper limbs and neck (Fig. 1). Multiple discrete yellowish sac like protrusions of sizes varying from 5mm to 10mm were seen over both the flanks and posterior aspect of the trunk. Button hole sign was positive (Fig. 2).Routine blood investigations and biochemical investigations were within normal limits. HIV and HBS Ag was negative. Histopathology of keratotic plaques showed foci of epidermal invagination filled with keratin and parakeratotic coronoid lamella ,dermis showed a mild to moderate infiltrate of lymphocytes and evidence of pigment incontinence which was consistent with porokeratosis (Fig. 3). Histopathology of sac like protrusions showed decreased elastic fibres in the papillary and reticular dermis. Staining with verhoeff–van Gieson was consistent with anetoderma (Fig. 4). With the above clinical and histopathological findings the diagnosis of disseminated superficial porokeratosis and anetoderma was confirmed. For larger lesions were treated with topical 5-flurouracil was given and smaller lesions with topical Tretinoin 0.05% cream. Significant improvement was seen within 3 months.

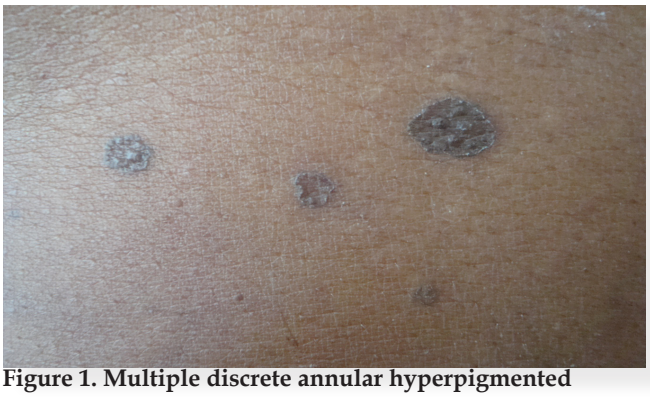

Figure 1. Multiple discrete annular hyperpigmented plaques with hyperkeratotic grooved borders

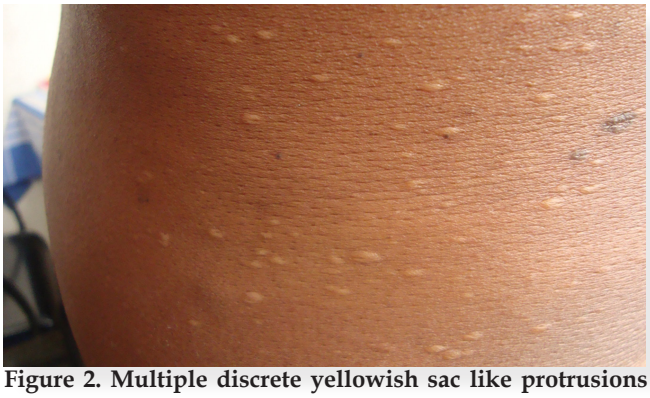

Figure 2. Multiple discrete yellowish sac like protrusions were seen over both the flanks and posterior aspect of the trunk

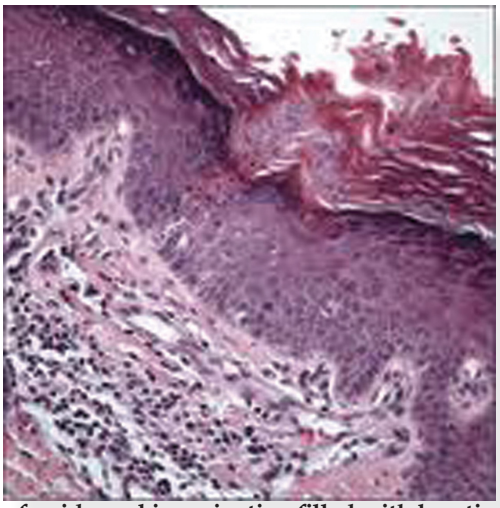

Figure 3. Foci of epidermal invagination filled with keratin and parakeratotic coronoid lamella, dermis showed a mild to moderate infiltrate of lymphocytes (H&E, 40X)

Figure 4. Verhoeff -van Gieson staining showing decreased elastic fibres in the papillary and reticular dermis

Porokeratosis is a heterogeneous group of disorders that are inherited in a autosomal dominant fashion. Different types of Porokeratosis are Porokeratosis of Mibelli, Disseminated superficial Porokeratosis (DSP), Disseminated superficial porokeratosis of immunosuppression, Disseminated superficial actinic porokeratosis (DSAP), linear porokeratosis, porokeratosis Palmaris plantaris et Disseminata and Punctate Porokeratosis. DSP has its onset in 3rd and 4th decade of life. There is a female predominance with 3:1. Early lesions of disseminated superficial porokeratosis (DSP) are small keratotic papules with central dell. They may be erythematous or pigmented. They enlarge to form superficial ring like lesions with slight central atrophy surrounded by discrete ridge topped by furrow. It mainly involves extremities in a bilateral symmetric fashion. Lesions are distributed symmetrically over extremities and trunk with predilection for the extensor surface. Involvement of face is rare. Lesions spare the axillary vaults, inguinal folds, perigenital region, palms, soles and mucous membranes. Dermoscopy reveals a „white track" structure with brown pigmentation on the inside of the track that can be seen at the edge of an individual lesion, corresponding to the cornoid lamella [1]. Centrally, a white area with red dots, globules, and lines is present that corresponds to capillary vessels; it is more easily observed through the atrophic epithelium. Clonal hyperproliferation of atypical keratinocytes leads to the formation of the cornoid lamella, which expands peripherally and forms the raised boundary between abnormal and normal keratinocytes. Local or systemic changes in immune function may allow the development of atypical clones of keratinocytes [2]. The risk factors for the development of porokeratosis include genetic inheritance, ultraviolet radiation, and immunosuppression. Immunosuppression associated with porokeratosis may be secondary to a disease process such as HIV infection or lymphoma or an iatrogenic suppression such as with immune-modulating drugs used to prevent organ transplant rejection or to treat autoimmune diseases. An autosomal dominant mode of inheritance has been established for familial cases of all forms of porokeratosis. O. Ferreira has reported an association of disseminated superficial porokeratosis with acute pancreatitis and suggested that it may be because of immunosuppression [3].

Anetoderma is a rare skin disease with loss of dermal elastic tissue resulting in clinically localised areas of flaccid or herniated sac like skin. It is classified into primary and secondary. Primary anetoderma appears on previously normal skin with unknown pathogenesis. It is divided in to Schweninger-Buzzi type which has no preceding erythema, and Jadassohn-Pellizari type which is preceded by macular erythema or papular urticaria. Secondary anetoderma occurs at sites of skin diseases such as acne, varicella, xanthoma, discoid lupus erythematosus, granuloma annulare, syphilis [4]. The association of lupus erythematosus, and other autoimmune disorders, and primary anetoderma is often cited in the literature. Whether these findings are coincidental or related is unknown. The exact cause of anetoderma is unknown. Possible explanations for loss of elastic tissue include defective elastin synthesis, uncontrolled production of elastolytic enzymes, loss of elastolytic enzyme inhibitors, elastophagocytosis, or degeneration of elastic fibers secondary to local ischemia induced by microthromboses in dermal vessels [5]. Diagnosis relays on histopathology findings which shows decrease in dermal thickness and normal collagen fibres and decrease in elastin fibres in upper dermis. Specific elastin staining can demonstrate a marked reduction in elastin content compared with adjacent normal dermis. No treatment has been found to be beneficial once atrophy is advanced. Colchicine has a preventive role.

A rare association of disseminated superficial porokeratosis and anetoderma developing after an episode of acute pancreatitis is being reported for first time to the best our knowledge.

Acknowledgements

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Dr.Vasantha Kumar S, Principal, MVJ Medical College and Research Hospital.

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NASZA DERMATOLOGIA Online
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PURPURA FOLLOWING A RARE ETIOLOGY; A DIAGNOSTIC DILEMMA

PLAMICA POSTĘPUJĄCA O RZADKIEJ ETIOLOGII; DIAGNOSTYCZNY DYLEMAT

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Sir,

Jatropha Curcas poisoning is a fairly common occurrence in Indian population [1,2]. It follows ingestion of the plant's nuts. It generally manifests with complaints of vomiting, abdominal pain and diarrhea [3]. These are attributed to the Toxalbumin, Curcin and cyanic acid found in these nuts. Though it generally presents with symptoms of mild gastroenteritis, it may sometimes be severe enough to cause hypovolemic shock. We came across an 8 year old child who presented with these symptoms following consumption of the Jatropha Curcas nuts. She also presented with newly developed purpuric spots over whole of the body (Fig. 1). They were especially prominent over the knees. Her blood work-up did not reveal any other cause of the purpurae. There were no signs of vasculitis either. She was managed conservatively with fluid resuscitation. The purpuric spots resolved over 3 weeks.

We conclude that, though poisonong following Jatropha Curcas results in gastroenteritis, it can also result in purpuric spots. This is the first known dermatologic manifestation and may be misinterpreted by the unwary physician.

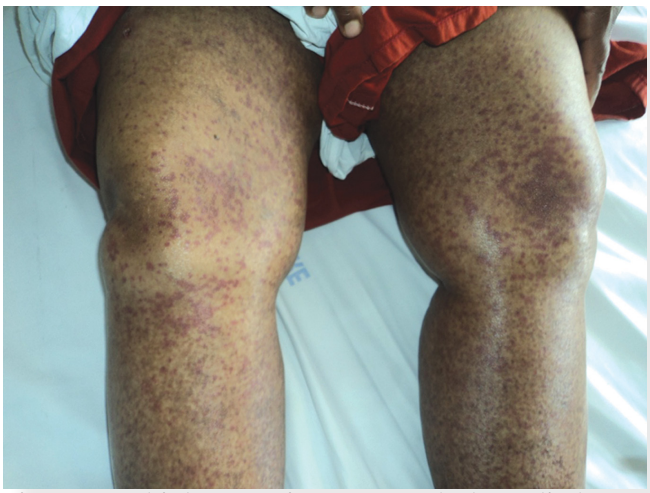

Figure 1. Multiple purpuric spots over the lower limbs

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NASZA DERMATOLOGIA Online
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VITILIGO - ANTI-THYROID PEROXIDASE ANTIBODY

BIELACTWO - PRZECIWCIAŁA PRZECIWKO PEROKSYDAZIE TARCZYCOWEJ

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Conflicts of interest: None

Sir,

Vitiligo is a common skin depigmenting disease, which is thought to have, at least partly, an autoimmune aetiology. The aim of this study was to explore the correlation between vitiligo and autoimmune thyroiditis, especially Anti-thyroid peroxidase antibody.

Our objective was to compare the frequency of thyroid peroxidase antibody (anti-TPO) in vitiligo patients seen in 2011 in our Department.

Methods

57 cases of vitiligo (39 women and 18 men) were enrolled in this study,for a period of 12 months (Tabl. I), (Fig. 1, 2). The clinical type of vitiligo are shown in Table II and Fig 3. Anti-TPO levels were assessed in order to detect any correlation with the onset, the evolution and the treatment of vitiligo.

Patients with vitiligo and with known thyroid disease, history of thyroid surgery and those receiving thyroid medications were not included.

Age (years)	Male	Female	Percentage (of the total number of vitiligo patients)
under 6	0	0	0
6-10	0	0	0
10-18	3	2	8,77%
18-30	7	8	26,31%
30-40	2	8	17,54%
40-60	3	19	38,60%
over 60	3	5	14,03%

Table I. Age and gender distribution of vitiligo patients

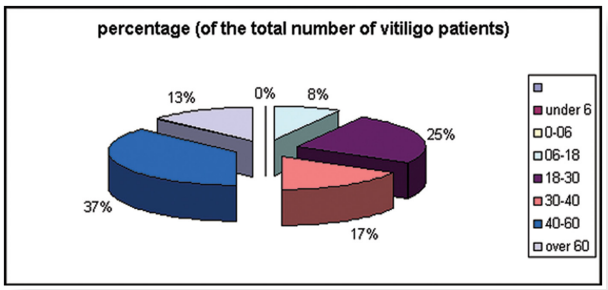

Figure. 1 Percentage (of the total number of vitiligo patients)

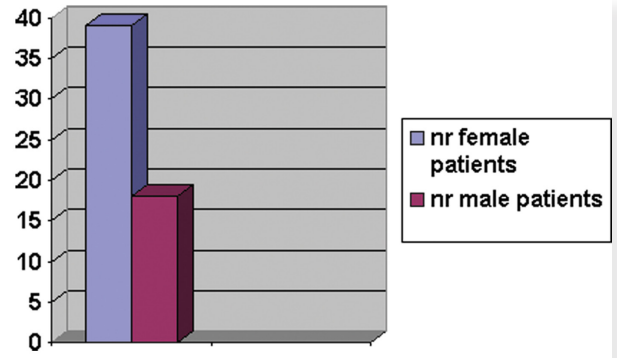

Figure 2. The gender distribution of patients with vitiligo

Clinical forms	Number of patients with vitiligo	Percentage (of the total number of vitiligo patients)	Number of patients with Ac TPO within normal limits (percentage of the total number of vitiligo patients)	Number of patients with Ac TPO high levels (percentage of the total number of vitiligo patients)	Number of patients with unknown value of Ac TPO (percentage of the total number of vitiligo patients)
facial	7	12,28%	3	2	2
acro-facial	4	7,01%	1	0	3
focal	27	47,36%	14	5	8
universal	15	26,31%	6	6	3
acral	4	7,01%	2	2	0
total	57	100%	26 (45,6%)	15 (26,31%)	16 (28,07%)

Table II. The clinical type of vitiligo

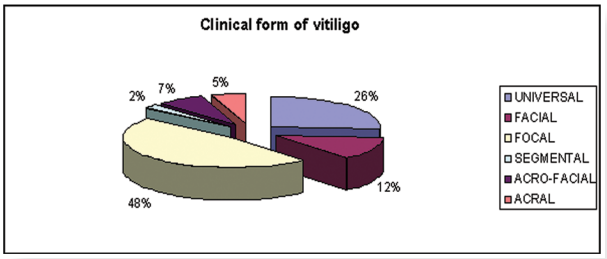

Figure 3. Clinical form of vitiligo

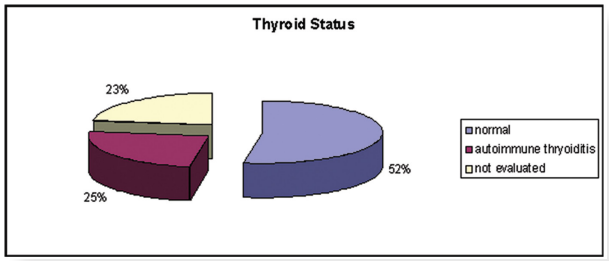

Figure 4. Thyroid status

Of 57 patients with vitiligo: 14 were diagnosed with autoimmune thyroiditis meaning of all patients, 13 patients were not evaluated for thyroid function and 30 had normal thryoid status. (Fig. 4, 5).

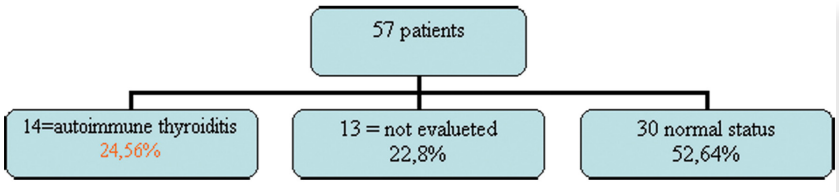

Figure 5. Thyroid status of 57 patients

Conclusions

According to our study, high levels of anti-TPO were shown to be more common in vitiligo patients, especially in young women. As this antibody is a relatively sensitive and specific marker of autoimmune thyroid disorders and considering the fact that vitiligo usually precedes the onset of thyroid dysfunction, periodic follow-up of vitiligo patients for detecting
thyroid diseases is further emphasized especially in young women with increased level of anti-TPO.

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NASZA DERMATOLOGIA Online
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AN ATYPICAL GIANT MANTOUX REACTION

NIETYPOWA OLBRZYMIA REAKCJA MANTOUX

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Conflicts of interest: None

Sir,

Mantoux tuberclin skin test is used for routine screening of individuals with a high risk of Tuberculosis infection and also for diagnosis of tubercular etiology in various illness [1]. A standardized 5 tuberculin units (TU) of purified protein derivative (PPD) is injected intradermally into the volar aspect of the left forearm and the delayed hypersensitivity reaction is noted by measuring the induration after 48-72 hours. Severe reactions with the formation of blisters and necrosis are very rare [2]. We present a child who developed a very rapid and abnormally large lesion after Mantoux testing.

An eight year old child was admitted with history of fever and since the past 2 months. The child had a normal growth for her age. On examination the left posterior cervical lymphnodes were significantly enlarged, discrete ,mobile and non tender. Systemic examination was normal.Her blood picture and Erythrocyte Sedimentation Rate (ESR) were normal. AMantoux test was done with the standard 5 TU Purified Protein Derivative (PPD) given intradermally. An immediate and exaggerated reaction with blisters and induration measuring 25X30mm was noticed within 6 hours after administrating the test (Fig. 1). Enzyme linked immunosorbent assay (ELISA) for human immunodeficiency virus (HIV) was nonreactive. Chest radiograph looked normal and sputum tested did not show any Acid Fast Bacilli (AFB). Her lymph node biopsy showed features of reactive lymphadenitis. Child was not started on antitubercular therapy but was treated with a short course of antibiotics and was asymptomatic at followup.

Our child had demonstrated an atypical and uncommon phenomenon since tubercular response is a delayed type of hypersensitivity reaction. Active tuberculosis, high mycobacterial antigen load or lepromatous leprosy may cause an exaggerated Mantoux response [3]. Patients who have an induration of more than 20mm have a higher chance of developing active tuberculosis than those with 10mm induration [4]. Tuberculin testing is useful for assessing the prevalence of tubercular infection in the developing countries. It should be administered and interpreted with caution and the decision of starting on antitubercular therapy is finally based on the clinical scenario and the results of the other tests for confirming tuberculosis.

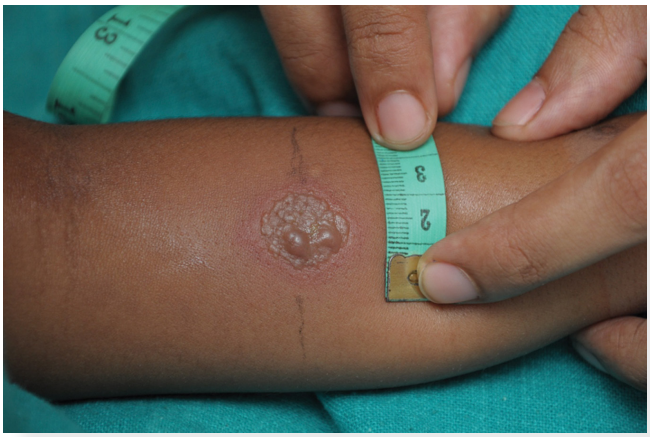

Figure 1. An exaggerated and rapid Mantoux reaction 6 hours after tuberculin administration

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NASZA DERMATOLOGIA Online
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PRIMARY LINGUAL TUBERCULOSIS, A RARE CASE REPORT

PIERWOTNA GRUŹLICA JĘZYKA, RZADKI OPIS PRZYPADKU

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Conflicts of interest: None

Sir,

Lingual tubercular lesions are rare occurrences even in endemic countries like India. The lesions can present in a variety of appearances. They may masquerade as vascular malformations or malignant neoplasm clinically. Cases of lingual tuberculosis have been described from 1888 [1]. There have been occasional reports of lingual tuberculosis since then [2,3]. The clinical diagnosis is often difficult if not impossible as it is rare to suspect especially if it presents as a diffuse swelling. Most often, the diagnosis is made either by fine needle aspiration cytology or histopathological examination of a biopsy from the lesion.

In this case, we present a patient who initially complained of a lingual swelling since 3 years. It had been gradually increasing in size and had occasionally ulcerated with minimal bloody discharge. The swelling was over the dorsal aspect of tongue and measured around 2.5x2.5cms in size (Fig. 1). It was firm in consistency with some areas being harder. There were no palpable cervical lymph nodes. Clinically a diagnosis of lymphatic malformation was made and excision biopsy was done (Fig. 2). Histopathological examination revealed numerous caseating epithelioid and giant cells granulomata, containing scanty acid fast bacilli. A final histopathological diagnosis of tuberculous lesion was later made and was supported by a positive PPD test and Mycobacterial culture from a sputum specimen. The patient was started on antituberculous therapy and follow up showed good initial response.

Many believe that the rarity of oral tuberculosis is due to the continuous cleansing of the oral mucosa by saliva, the presence of a variable normal flora in addition to the presence of submucosal antibodies which gives the buccal mucosa a normal resistance [4]. The case differs from previously reported cases because the patient presented with a nodular and tumor-like mass which was clinically mistaken for a lymphatic malformation.

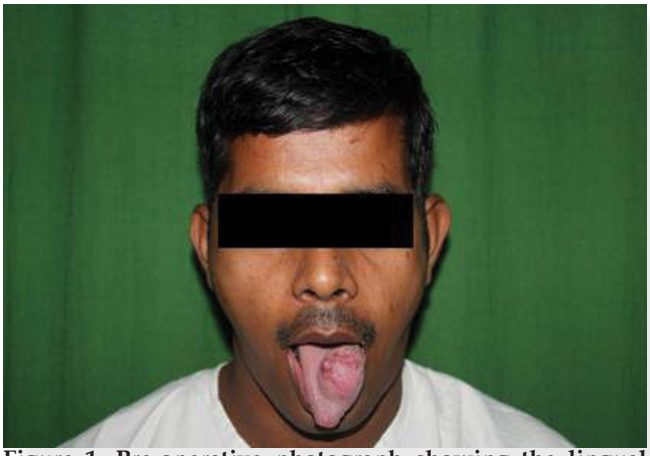

Figure 1. Pre-operative photograph showing the lingual swelling

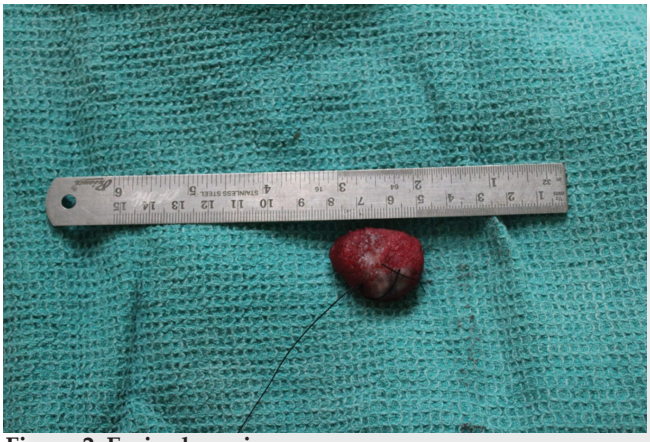

Figure 2. Excised specimen

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NASZA DERMATOLOGIA Online
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THE PROBLEM OF SYNONYMS; MULTIPLE SYMMETRIC LIPOMATOSIS, AS AN EXAMPLE

PROBLEM SYNONIMÓW; MULTIPLE SYMMETRIC LIPOMATOSIS, JAKO PRZYKŁAD

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Conflicts of interest: None

One of the important problems in the medical field is the absent of uniform and standard terminology. This applies to dermatology and to other medical specialties.

Many diseases have multiple names, and may be called differently, even in a single country. This may cause confusion among patients, and health care providers.

For researchers looking for a given disease, absent of uniform nomenclature may put strain on them during their studies. This problem, if not corrected, is expected to get worse in the future due to the progressive increase in the amount of medical literature that might be difficult to search for a disease with several names.

In this communication, I will take Multiple Symmetric Lipomatosis (MSL), as an example, for the problem of synonyms.

MSL is a metabolic condition characterized by the growth of fatty masses around the back of the head, neck, upper arms in a very specific pattern, causing a pseudo athletic habitus.

It is described in adults from 30 to 60 years old, and more common in male. Most of the cases are associated with alcoholism [1]. The cause of MSL remains unknown, but abnormalities in lipogenesis induced by catecholamines and mitochondrial DNA have been observed [2].

Multiple Symmetric Lipomatosis, also spelled as multiple symmetrical lipomatosis, is known in medical literature with other names.

These include Benign Symmetric Lipomatosis, Madelung's Disease and Lanois-Bensaude Syndrome.

MSL was first described by Brodie in 1846. Sir Benjamin Collins Brodie (1783-1862), Figure 1, was an English surgeon. After that, Madelung in 1888 and Launois and Bensaude in 1898 characterized the disease [1]. Otto Wilhelm Madelung (1846-1926), Figure 2, was a German surgeon. Pierre-Emile Launois (1856-1914), Figure 3, was a French physician. Raoul Bensaude (1866-1938), Figure 4, was a French physician [3].

One can see easily that searching scientific databases like MEDLINE (www.pubmed.com), with synonyms of this disorder, as a search words, will end up with different number of citations.

Table I, illustrate the discrepancy in the number of citations obtained using each name for this disease.

Hence, the importance of having a uniform and standard terminology in medical field cannot be overemphasized. It remained a goal which is a difficult to achieve.

Many steps might be needed to improve the problem of synonyms. However, as a starting step, authors and editors should keep the importance of adhering to the most cited international name for a given disease and refraining from mentioning other synonyms.

The names	Number of citations in PubMed using the term as a search words	Number of citations in PubMed using the term as a search words, and limiting the search to the title
Benign Symmetric Lipomatosis	94	60
Lanois-Bensaude Syndrome	101	35
Madelung's Disease	412	94
Madelung-Launois-Bensaude syndrome	4	3
Multiple Symmetrical Lipomatosis	358	37

Table I. The different numbers of citations obtained from searching the PubMed with synonyms of Multiple Symmetric Lipomatosis, as of 23 Feb 2012

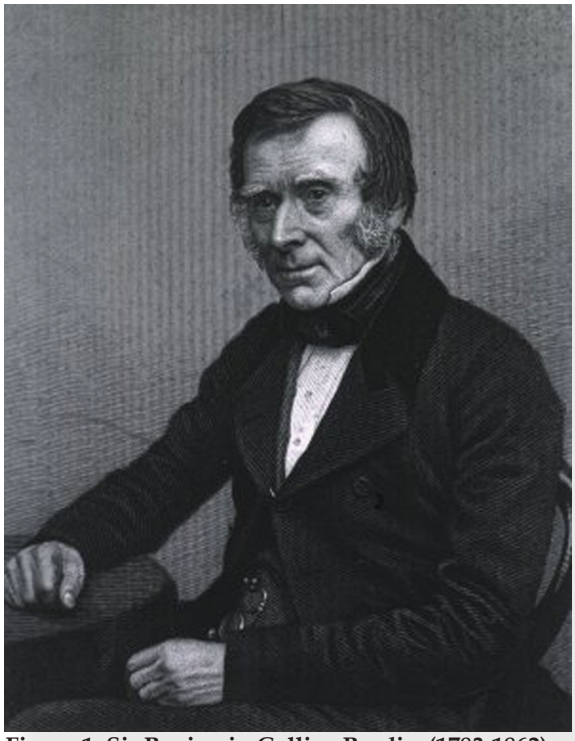

Figure 1. Sir Benjamin Collins Brodie, (1783-1862). Courtesy of the National Library of Medicine

Figure 2. Otto Wilhelm Madelung, (1846-1926). Courtesy of the National Library of Medicine

Figure 3. Pierre-Emile Launois, (1856-1914). Courtesy of the National Library of Medicine

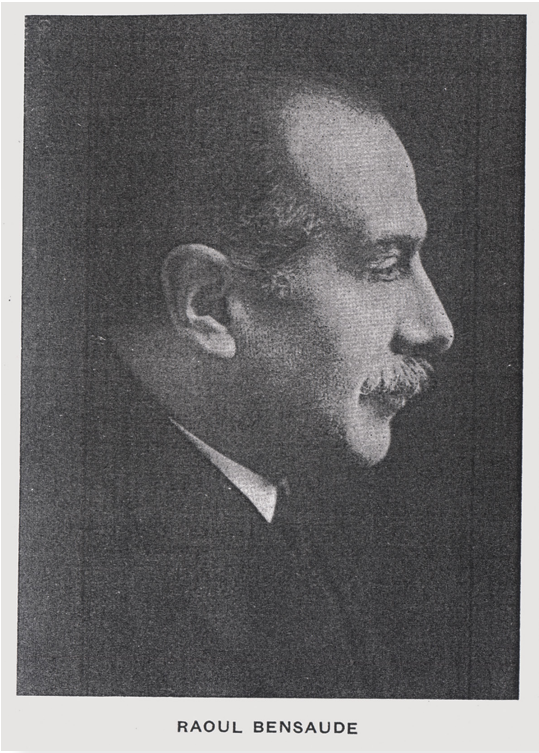

Figure 4. Raoul Bensaude, (1866-1938). Reproduced with permission from reference number 3

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BLAHOSLAV BEDNAR (1916-1998) AND THE TUMOR WHICH BEARS HIS NAME

BLAHOSLAV BEDNAR (1916-1998) I GUZ, KTÓRY NOSI JEGO IMIĘ

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Conflicts of interest: None

Dermatofibrosarcoma protuberans (DFSP) a locally aggressive soft tissue neoplasm with intermediate- to low-grade malignancy [1].

It is reported in the literature as early as 1890. Darier and Ferrand first described it in 1924 as a distinct cutaneous disease entity called progressive and recurring dermatofibroma. Hoffman officially coined the term dermatofibrosarcoma protuberans in 1925 [1].

Pathologically this tumor is characterized in its early stage by large, spindle-shaped nuclei which are embedded fairly uniformly in the collagen stroma, parallel to the skin surface and infiltrating into the subcutaneous tissue in the honeycomb pattern. There is absence of cellular atypia, and mitoses are rare. In the nodular stage, there are irregular, short, intersecting bands of tumor cells forming a storiform pattern. Also typical are cells radiating from a central hub of fibrous tissue forming a cartwheel pattern. Occasionally, DFSP may show focal fibrosarcomatous changes with a characteristic herringbone pattern [1].The tumor is diffusely positive for CD34.

There are several histological variants of DFSP. These include Bednar tumor, fibrosarcomatous, fibrosarcomatous with myoid/myofibroblastic change, myxoid,granular cell, palisaded, giant cell fibroblastoma, combined and indeterminate [2,3].

Pigmented DFSP (Bednar tumor) is morphologically and clinically identical to ordinary DFSP, with the exception of the presence of non-neoplastic melanin-laden dendritic cells which usually found scattered between the neoplastic spindle-shaped cells. It is thought to represent the colonization of DFSP by melanocytes [2]. It constitutes 5%-10% of all cases of DFSP and shows morphologic features that overlap with melanocytic and fibrous proliferations [2-6].

There is one report each of a recurrent DFSP transforming into a Bednar tumor, Bednar tumor with prominent meningothelial-like whorls [4], Bednar tumor with dermal melancytosis, and congenital Bednar tumor in a patient with Fanconi anemia [6]. Rare cases occur with histological overlap between a pigmented dermatofibroma and pigmented DFSP [2]. Bednar tumor has, also, been reported at the site of previous immunization. There is no metastasis reported with this type of DFSP [2].

Figure 1. Blahoslav Bednar (1916-1998). A courtesy of Jaromir Hacek, Editor of Cesko-slovenska patologie Journal
The reprint of this figure is also approved by the committe of the Czech Society of Pathologists

Pigmented DFSP is first reported by Bednar in 1957 [7], while describing a group of nine cutaneous tumors characterized by indolent growth and a prominent storiform pattern and in four cases by the presence of melanin pigment. He regarded these tumors as variants of neurofibroma (storiform neurofibroma) [3].

Blahoslav Bednar (1916-1998), is a well-known Czech pathologist [8-10] (Fig. 1). The Correct spelling for his name in Czech is, Blahoslav Bednáø.

He founded the official journal Czecho-Slovak Pathology, and he modernized Hlava Institute of Pathology [10]. Bednar made a great contribution to the pathology and he left behind hundreds of interesting publications and researches.

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ANDRÉ NANTA (1883-1963) AND THE NEVUS WHICH BEARS HIS NAME

ANDRÉ NANTA (1883-1963) I ZNAMIĘ, KTÓRE NOSI JEGO IMIĘ

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Nevus of Nanta

A nevus of Nanta lesion is characterized by the presence of cutaneous ossification (osteoma cutis) of an existing melanocytic nevus [1-4]. It is postulated that secondary ossification is the consequence of folliculitis, trauma, or neoplastic proliferation, which may induce the dermal fibroblasts to differentiate into osteoblasts [1]. Histologically, the osteomas are composed of well-formed bony spicules with prominent cement lines and calcification. They may demonstrate osteoblasts, osteoclasts, and osteocytes and occasionally may even demonstrate bone marrow elements [2]. Notably, nevus of Nanta has a higher prevalence in women than in men, possibly due to the effect of estrogen stimulation on osteoblasts within the lesion. Of clinical significance, melanoma can occur in association with nevus of Nanta [4].

Using skeletal bone formation as a guide, two mechanisms of bone formation can be expected in the skin. First, enchondral ossification, which occurs in most of the long bones of the skeleton, involves a cartilaginous template that is subsequently ossified and replaced. Or more commonly, membranous bone formation is a result of direct bone formation without the cartilaginous anlage [2].

The osteomas in nevus of Nanta usually occur at the base of the melanocytic proliferation occurring maily in the intradermal nevus but may rarely occur with nevi with junctional activities. In one series of 74 cases of primary and secondary cutaneous ossification, nevus of Nanta represented 26% (19 of 74) of the total osteomas identified and was the single most common lesion with osteoma formation [2].

André Nanta (1883-1963)

André Nanta, French dermatologist, born in 31 May, 1883 [5,6]. He descended from Franche-Comte ancestary. He spent his childhood in toulous. He rapidely ascended into his medical field and become a professor of universities in 1934 [5].

Figure 1. André Nanta (1883-1963). With permission from reference number 5

He had multiple interest and contributions in dermatology.

He presented a thesis on lymphodermia and myelodermia.

He had a special interest in deep fungal infection and made several trips to Algeria for this purpose. This interest in mycosis led him to isolate a special strain in fungus named with his name; aspergillus nantae [5]. He worked also on cutaneous lipidosis, and eosinophilic granuloma [7] (Nanta-Gadrat disease). The Nanta name also remains attached to osteo-nevus described in 1911.

He also contributed with his students Bazex and Dupré in understanding of congenital skin disorders [5].

He had several scientific assignments and he had been a president of Société Française de Dermatologie.

André Nanta died on 27 May 1963 [5.6].

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DERMATOLOGY EPONYMS – SIGN – LEXICON – G

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

Streszczenie
Eponimy stosowane są niemal codziennie w praktyce w klinicznej dermatologii. A jednak informacja na temat osoby związanej z danym eponimem jest trudna do znalezienia. Kto to jest? Jakie jest jego obywatelstwo? Czy jeszcze żyje, jeśli nie to kiedy zmarł? Jak można znaleźć artykuł, w którym osoba ta po raz pierwszy opisała chorobę? Eponimy są używane do opisywania nie tylko choroby, ale również objawu klinicznego, zabiegu chirurgicznego, technik barwienia, preparatów farmakologicznych, a nawet elementów wyposażenia. W tym artykule prezentujemy objawy zaczynające się na literę G. Objawy i ich synonimy oraz tych, którzy opisali ten objaw lub zjawisko.

Key words: eponyms; skin diseases; sign; phenomen
Słowa klucze: eponimy; choroby skóry; objaw; fenomen

GAMBIAN PLAGUE SIGN (Africa)
Lymphadenopathy associated with smallpox type lesions. Caused by contact with the zoonotic *monkeypox virus*, that lives in rodents, and Gambian rats (Gambian giant rats-*Cricetomys gambianus*). Adult ratscan reach a length of 91.44 cm and a weight of 4 kg [1].

OBJAW PLAGI GAMBIJSKIEJ
Powiększenie węzłów chłonnych występujące ze zmianami przypominającymi ospę. Spowodowane przez kontakt z odzwieżęcym wirusem *monkeypox* pochodzących od gryzoni i szczurów z Gambii (gambijskie gigantyczne szczury-*Cricetomys gambianus*). Dorosły szczur osiąga długość 91,44 cm i waży 4 kg [1].

GAMBIAN SLEEPING SIGN (Africa)
Zoonotic trypanosomatosis. Human African trypanosomiasis (HAT), or sleeping sickness, describes not one but two discrete diseases; caused by *Trypanosoma brucei rhodesiense* and that caused by *Trypanosoma brucei gambiense* (Fig. 1). The Gambian form is currently a major public health problem over vast areas of central and western Africa, while the zoonotic, Rhodesian form continues to present a serious health risk in eastern and southern Africa. The two parasites cause distinct clinical manifestations, and there are significant differences in the epidemiology of the diseases caused [2].

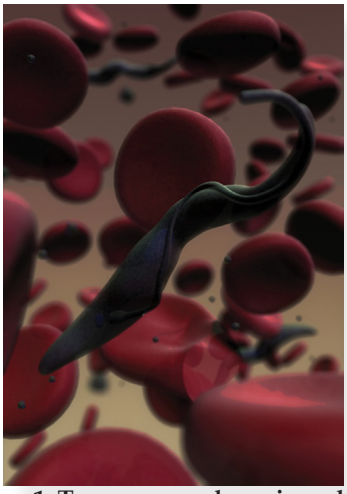

Figure 1. Trypanosoma brucei gambiense

OBJAW GAMBIJSKIEGO SNU (Afryka)
Odzwierzęca trypanosomatoza. Ludzka Afrykańska Trypanosomatoza (HAT) lub śpiączka afrykańska, opisuje nie jedną, lecz dwie oddzielne choroby: spowodowane przez *Trypanosoma brucei rhodesiense* i przez *Trypanosoma brucei gambiense* (Ryc. 1). Gambijska forma jest obecnie głównym problemem zdrowia publicznego na rozległych obszarach środkowej i zachodniej Afryki, a chorobotwórcza, rodesyjska forma nadal stanowi poważne zagrożenie dla zdrowia w Afryce wschodniej i południowej [2].

GANGES SIGN (India)
Epidemic infection by the cholera bacillus occurring during the Hindu pilgrimage and holy days among the crowds at the lower Ganges in India. In one study from December 1985 to November 1987, a bacteriological study of the river Ganges in Varanasi was carried out. In all 407 water samples were collected, 335 from bathing ghats and 72 from sewage openings and were examined for the presence of enteropathogenic bacteria. *Vibrio cholerae* 0-1 (1.72%), Non 0-1 *Vibrio cholera* (3.69%), *Vibrio fluvialis* (0.74%), *Aeromonas sp.* (0.49%), *Plesiomonas sp*. (0.25%), *Salmonella sp.* (0.98%) and *Shigella sp.* (1.23%) [3].

*Ghats (literally steps) is a series of stone steps leading down to the river. It occurs in many places in Asia.

OBJAW GANGESU (Indie)
Epidemia wywołana przez pałeczki cholery występująca podczas hinduistycznej pielgrzymki i święta wśród tłumów w niższych partiach Gangesu w Indiach. W jednym z badań od grudnia 1985 do listopada 1987 roku przeprowadzono badanie bakteriologiczne rzeki Ganges w Varanasi. Wszystkie 407 próbek wody, które zostały pobrane; 335 z ghatów kąpielowych i 72 z otworów kanalizacyjnych przebadano na obecność bakterii enteropatogennych. *Vibrio cholerae* 0-1 (1,72%), nie 0-1 *Vibrio cholera* (3,69%), *Vibrio fluvialis* (0,74%), *Aeromonas sp.* (0,49%), *Plesiomonas Sp.* (0,25%), *Salmonella sp.* (0,98%) i *Shigella Sp.* (1,23%) [3].

*Ghaty (dosłownie stopnie) to ciąg kamiennych schodów, prowadzący w dół ku rzece. Występuje w wielu miejscach Azji.

GARDNER'S SIGN (Gardener sign)
Sebaceus cyst of the skin, polyposis of the large intestine, supernumerary teeth (Fig. 2), and osteomas. A sign of Gardner's syndrome [4].

OBJAW GARDNERA (Objaw Gardenera)
Torbiele łojowe skóry, polipowatość jelita grubego, nadliczbowe zęby i kostniaki (Ryc. 2). Objaw zespołu Gardnera [4].

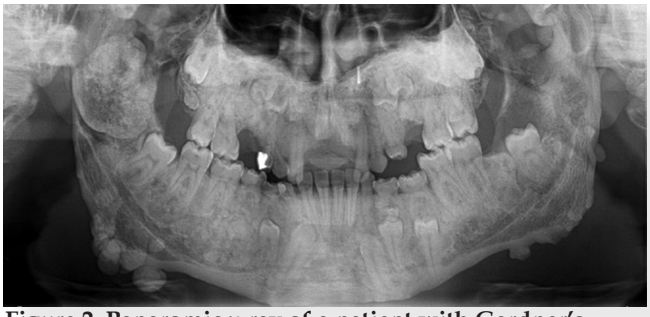

Figure 2. Panaromic x-ray of a patient with Gardner's syndrome

ELDON J. GARDNER
Professor and cancer researcher (1909–1989), a college teacher of genetics (Fig. 3). He described Gardner's syndrome in 1951. His first human genetics paper was published in 1949, and he was active in research as a human geneticist until the day of his death at age 79. Eldon Gardner was a writer; more than 300 publications appear in his vita, including articles in scientific and medical journals, chapters in books, review articles, and books on general genetics, human genetics, and the history of biology [5].

Figure 3. Eldon J. Gardner

Profesor i badacz raka (1909-1989), nauczyciel kolegium genetyki (Ryc. 3). Opisał zespół Gardnera w 1951 roku. Dokument genetyczny został opublikowany w 1949 roku; brał czynny udział w badaniach jako genetyk aż do dnia jego śmierci w wieku 79 lat. Eldon Gardner był pisarzem; ponad 300 publikacji pojawiają się w jego CV, w tym artykuły w czasopismach naukowych i medycznych, rozdziały w książkach, artykuły przeglądowe i książki na temat ogólnej genetyki człowieka oraz historii biologii [5].

GARLIC BREATH 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 sign [6].

OBJAW ODDECHU CZOSNKOWEGO

Przewlekły stan skrajnego bólu i groteskowe zniekształcenie spowodowane przez zatrucie z narażeniem na biały fosfor. Chorzy mają nieprzyjemną cuchnącą wydzielinę z znakiem żuchwy [6].

GAS SIGN

Gas gangrene. *Clostridium perfringens*, type A. As the bacteria grow inside the body, it makes gas and harmful substances (toxins) that can damage body tissues, cells, and blood vessels. Gas gangrene develops suddenly. It usually occurs at the site of trauma or a recent surgical wound. Patients most at risk for this usually have underlying blood vessel disease (atherosclerosis or hardening of the arteries), diabetes, or colon cancer. Symptoms include: air under the skin (subcutaneous emphysema), blisters filled with brown-red fluid (Fig. 4), drainage from the tissues, foul-smelling brown-red or bloody fluid (serosanguineous discharge), tachycardia, moderate to high fever, moderate to severe pain around a skin injury pale skin color, later becoming dusky and changing to dark red or purple, progressive swelling around a skin injury, sweating, vesicle formation, combining into large blisters, yellow color to the skin (jaundice) [7] (Fig. 5).

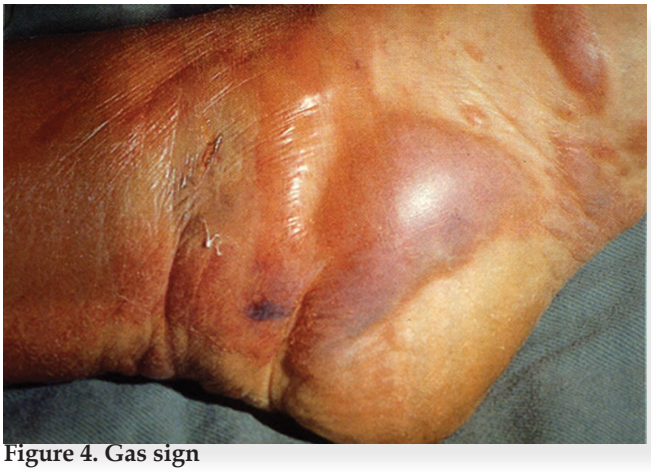
Figure 4. Gas sign

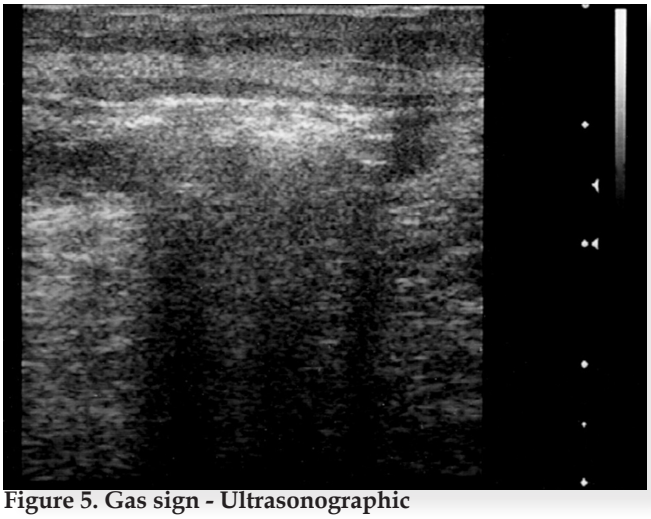
Figure 5. Gas sign - Ultrasonographic

OBJAW GAZU

Zgorzel gazowa. *Clostridium perfringen*s typu A. Ponieważ bakterie rosną wewnątrz ciała, sprawia to rozwój gazu i szkodliwych substancji (toksyn), które mogą uszkodzić tkanki, komórki i naczynia krwionośne. Zgorzel gazowa rozwija się nagle. Występuje zwykle w miejscu urazu lub niedawnej rany chirurgicznej. Pacjenci najbardziej narażeni na to zjawisko mają zwykle chorobę naczyń krwionośnych (miażdżyca lub stwardnienie tętnic), cukrzycę, czy raka jelita grubego. Objawy obejmują: powietrze pod skórą (rozedma podskórna), pęcherze wypełnione płynem brązowo-czerwonym (Ryc. 4), drenaż tkanek, cuchnące brązowo-czerwony lub krwawy płyn (subcutaneous emphysema), tachykardia, umiarkowana do wysokiej gorączka, umiarkowany do ciężkiego bólu wokół uszkodzenia skóry, blady kolor skóry, później staje się ona ciemno czerwona lub purpurowa, progresywny obrzęk wokół uszkodzenia skóry, pocenie się, powstawanie pęcherzyków, łączących w duże pęcherze, żółty kolor skóry (żółtaczka) [7] (Ryc. 5).

GENGOU'S SIGN

Fixation of the complement phenomenon [8].

OBJAW GANGOU

Fiksacja (utrwalenie) zjawiska dopełniacza [8].

OCTAVE GENGOU

Belgian bacteriologist, 1875-1957. He researched with Jules Bordet the *Bordetella pertussis* bacteria. Gengou worked at the Belgium Pasteur Institute in Brussels. With Jules Bordet in 1906 he isolated *Bordetella pertussis* in pure culture and declared is as the cause of whooping cough. 1912 he developed the first whooping cough-vaccine. He also worked on various important fundamental research on a now common test for diseases (e.g. the Wassermann-Test of August von Wassermann) [9].

Belgijski bakteriolog, 1875-1957. Badał razem z Jules Bordet'em bakterie *Bordetella pertussi*s. Gengou pracował w Belgijskim Instytucie Pasteura w Brukseli. Z Julesem Bordetem w 1906r. wyizolowali *Bordetella pertussis* z czystej kultury i ogłosili ją jako przyczynę kokluszu. W 1912 roku opracował pierwszą szczepionkę na koklusz. Pracował także nad szeregiem istotnych badań w kręgu chorób zakaźnych (np. WR-Test Augusta von Wassermanna) [9].

GEOGRAPHIC TONGUE SIGN

Smooth bright red patches on the dorsum of the tongue often with gray, white, or yellow borders (Fig. 6). Also known as migratory glossitis [10].

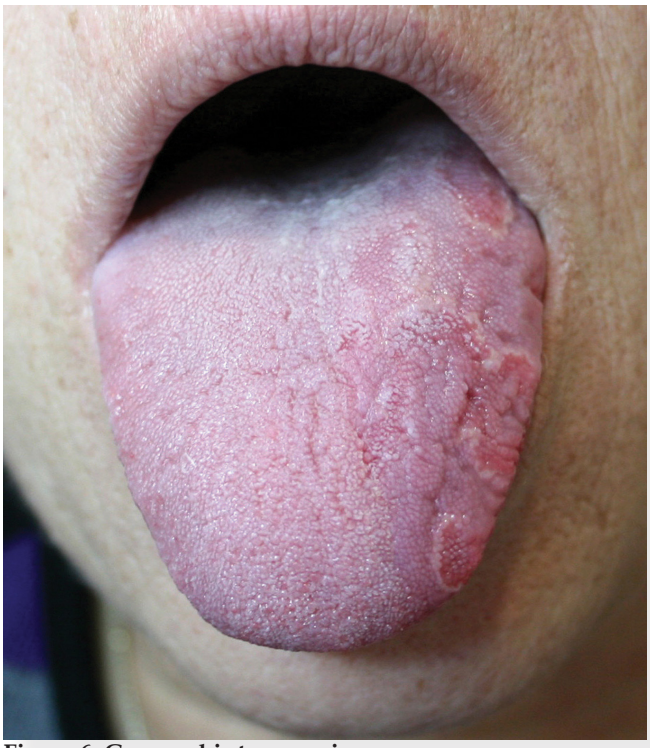

Figure 6. Geographic tongue sign

OBJAW JĘZYKA GEOGRAFICZNEGO

Gładkie jasno czerwone plamy na grzbiecie języka, często z szarym, białym lub żółtym nalotem (Ryc. 6). Znany również jako migrujące zapalenie języka [10].

GERMAN MEASLES SIGN

Syn. congenital rubella (Fig. 7). After birth, infection with rubella virus occurs through inhalation of contaminated droplets. In children infected after birth, rubella viruses usually cause uncomplicated diseases that are associated with an unspecific rash (postnatal rubella). Rubella virus infection during pregnancy can lead to the infection of the fetus. In the first and second trimester of pregnancy, fetal rubella infection often lead to severe abnormalities of the newborn, which are summarized as rubella embryopathy or congenital rubella syndrome. One of these disorders, the Gregg syndrome [11].

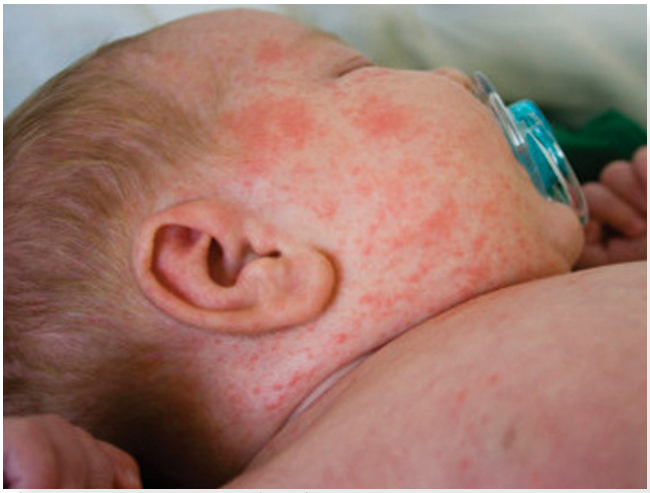

Figure 7. German measles sign

OBJAW NIEMIECKIEJ RÓŻYCZKI

Syn. różyczki wrodzonej (Ryc. 7). Po urodzeniu, zakażenie wirusem różyczki występuje przez wdychanie skażonych kropelek. U dzieci zakażonych po urodzeniu, różyczka zwykle powoduje nieskomplikowane choroby, które są związane z nieswoistą wysypką ust (poporodowa różyczka). Zakażenie wirusem różyczki podczas ciąży może prowadzić do zakażenia płodu. W pierwszym i drugim trymestrze ciąży zakażenie płodu różyczką często prowadzi do poważnych zaburzeń u noworodków, które zostały podsumowane jako embriopatia różyczkowa lub zespół wrodzonej różyczki. Jednym z tych zaburzeń jest zespół Gregg [11].

GIBERT SIGN

Pityriasis rosea. This sign has been listed in past literature also as Gilbert's rosea, due to similar name [12].

OBJAW GIBERTA

Łupież różowy. Objaw ten został opisywany kiedyś w literaturze także jako rosea Gilbert, z powodu podobnej nazwy [12].

CAMILLE MELCHIOR GIBERT

French dermatologist, 1797-1866. He worked at the Broca and Saint-Louis Hospitals. Gibert is remembered for providing the first accurate description of a papulosquamous skin disorder that he named pityriasis rosea. Historically this condition was also called „Gibert's disease". His best known written work on skin diseases was a tome called „Traité pratique des maladies spéciales de la peau". In 1859, with Dr. Joseph Alexandre Auzias-Turenne (1812–1870),

Gibert took part in a controversial experiment in which human patients were deliberately infected with syphilis in order to demonstrate the infectious nature of secondary syphilis. He died in the 1866 Paris cholera epidemic [13] (Fig. 8).

Figure 8. Camille Melchior Gibert

Francuski dermatolog, 1797-1866. Pracował w szpitalach Broca i Saint-Louis. Gibert jest pamiętany za udzielanie pierwszego dokładnego opisu grudkowo-złuszczającej choroby skóry, którą nazwał łupież różowy. Historycznie ten stan nazywany był również „chorobą Giberta". Jego najbardziej znaną pracą na temat chorób skóry była księga zwana „Traité pratique des maladies spéciales de la peau „. W 1859 roku, z dr Joseph Alexandre Auzias-Turenne (1812/70), Gibert wziął udział w kontrowersyjnym eksperymencie, w którym pacjentów celowo zakażano kiłą w celu wykazania charakteru zakaźnego wtórnej kiły. Zmarł w Paryżu 1866 roku epidemii cholery [13] (Ryc. 8).

GIGUERE'S SIGN

A type of modern malnutrition syndrome caused by artificial milk and infant formula substitutes which are deficient in vitaminum D, protein, and essential fats. Presentations include delayed tooth eruption, enamel defects, rickets symptoms, swollen abdomen, as well as, bands of hair discoloration associated with kwashiorkor [14].

OBJAW GIGUERE

Typ nowoczesnego zespołu niedożywienia spowodowanego sztucznym mlekiem i niemowlęcymi substytutami, które są ubogie w witaminę D, białka i tłuszcze. Prezentuje się opóźnionym wyrzynaniem zębów, wadą szkliwa i objawami krzywicy, obrzękiem brzucha, jak również, zespołami przebarwienia włosów związanego z kwashiorkor [14].

MICHELE C. GIGUERE WHITE

American surgeon.

Amerykański chirurg.

GILCHRIST'S SIGN

Blastomycosis, zoonotic fungal pneumonia, dermatologic and bone lesions (Fig. 9, 10). Also called blasto and Chicago disease [15].

OBJAW GILCHRISTA

Drożdżyca, grzybicze odzwierzęce zapalenie płuc, dermatologiczne i kostne zmiany (Ryc. 9, 10). Zwany także blasto i chorobą Chicago [15].

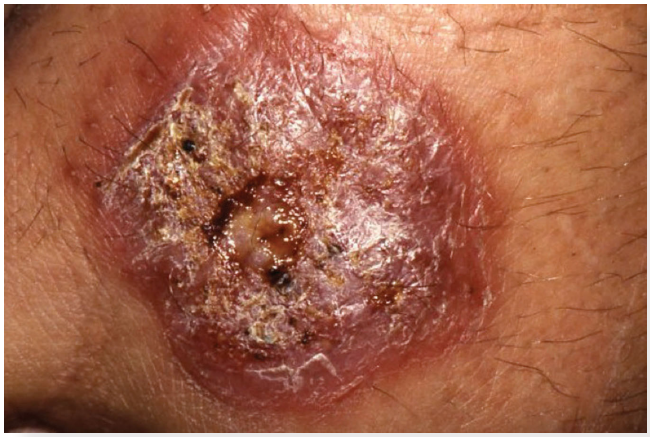

Figure 9. Gilchrist's sign

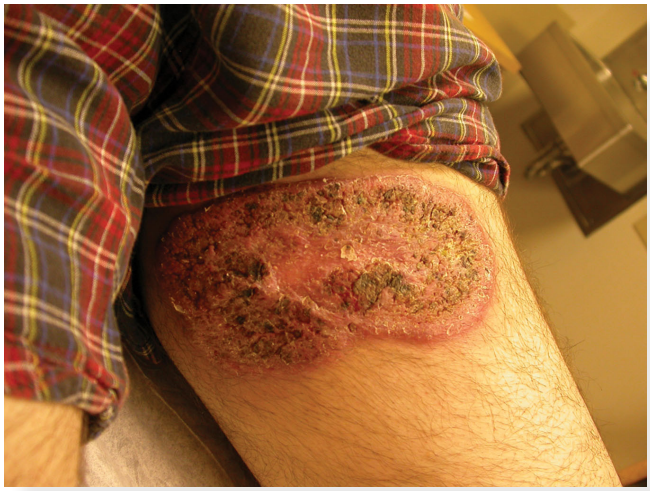

Figure 10. Gilchrist's sign

THOMAS CASPAR GILCHRIST

English-American dermatologist, 1862-1927 (Fig. 11). Studied medicine at the Manchester Royal Infirmary. He left England for America in 1890 and from 1897 he was clinical professor of dermatology at the University of Maryland, from 1898 in the same position at the Johns Hopkins Hospital, Baltimore. In 1907 the University of Maryland conferred on him an honorary M.D. He was president of the American Dermatology Association in 1909. He published extensively in his speciality, on acne vulgaris, experimental urticaria, erypseloid, roentgen dermatitis, porokeratosis, sarcoma of skin, and fatty atrophy [16].

Angielsko-Amerykański dermatolog, 1862-1927 (Ryc. 11). Studiował medycynę w Manchester Royal Infirmary. Opuścił Anglię i wyjechał do Ameryki w roku 1890 i od 1897 był profesorem dermatologii na Uniwersytecie w Maryland, od 1898 roku zajmował tą samą pozycję w Johns Hopkins Hospital w Baltimore. W 1907 roku Uniwersytet Maryland przyznał mu honorowy doktorat. Był prezesem Amerykańskiego Towarzystwa Dermatologicznego w 1909 roku. Wydał obszernie obwieszczenie o swojej specjalności w trądziku pospolitym, pokrzywce eksperymentalnej, różycy, porentgenowskim zapaleniu skóry, porokeratozie, mięsaku skóry i zaniku tkanki tłuszczowej [16].

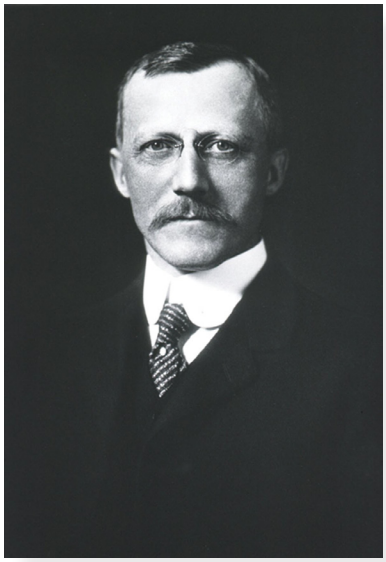

Figure 11. Thomas Caspar Gilchrist

GILL'S SIGN

Myxoedema (Fig. 12, 13). Describes a specific form of cutaneous and dermal edema secondary to increased deposition of connective tissue components (like glycosaminoglycans, hyaluronic acid, and other mucopolysaccharides) in subcutaneous tissue as seen in various forms of hypothyroidism and Graves' disease. It is more common in women than in men [17].

OBJAW GILLA

Myxoedema (obrzęk śluzowaty) (Ryc. 12, 13). Opisuje specyficzną formę obrzęku skóry wtórnego do zwiększonego odkładania się w tkance łącznej komponentów (t.j/ glikozaminoglikany, kwas hialuronowy i inne mukopolisacharydy) w tkance podskórnej; w różnych formach niedoczynności tarczycy i chorobie Gravesa-Basedowa. Występuje częściej u kobiet niż u mężczyzn [17].

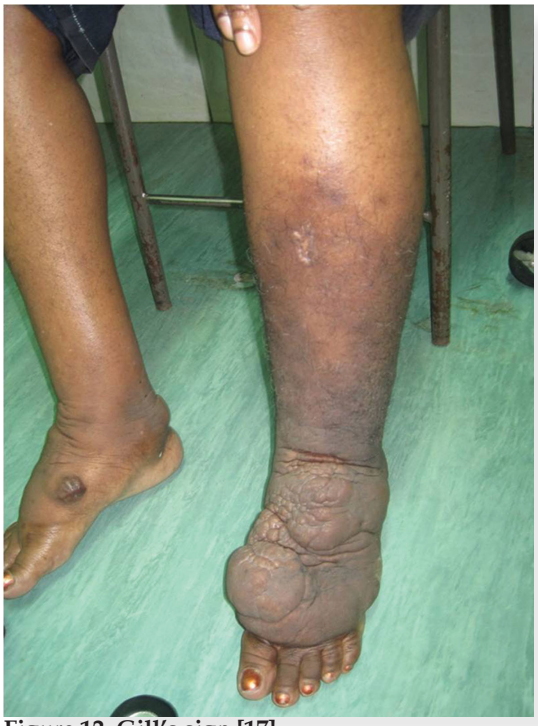

Figure 12. Gill's sign [17]

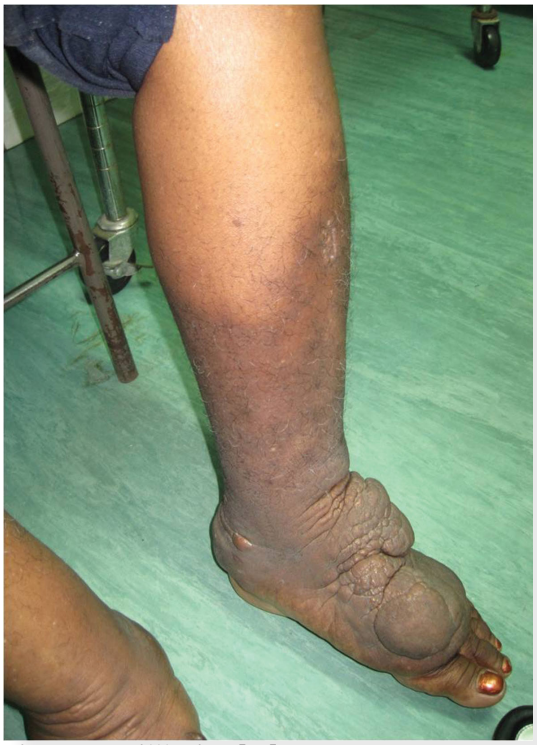

Figure 13. Gill's sign [17]

GIOVANNINI'S SIGN

Nails of the hand are entirely white and opaque, with the look of ivory. A sign found following an attack of typhoid fever. Also called canities unguium. Synonym for leukonychia, achromia unguium, leukopathia unguis [18].

OBJAW GIOVANNINI

Paznokcie dłoni są całkowicie białe i nieprzezroczyste, z wyglądu przypominają kość słoniową. Objaw obserwowany po ataku duru brzusznego. Zwany także canities unguium. Synonim dla leukonychii, achromia unguium, leukopathia unguis [18].

SEBASTIANO GIOVANNINI

Italian dermatologist, 1851-1920. In 1877 he graduated in medicine and surgery. Received the title of Professor in 1884. In 1889 he was appointed professor of skin and venereal diseases at the University of Modena.

Włoski dermatolog, 1851-1920. W 1877 ukończył studia w zakresie medycyny i chirurgii. Uzyskał tytul Profesora w 1884 roku. W 1889 roku został mianowany profesorem chorób skóry i chorób wenerycznych na Uniwersytecie w Modenie.

GIOVANNINI'S HAIR SIGN

A rare nodular disease of the hair produced by a fungus. Giovannini disease (fungal infection of the hair).

OBJAW WŁOSÓW GIOVANNINIEGO

Rzadka guzkowa choroba włosów wywołana przez grzyby. Choroba Giovanniniego (grzybica owłosionej skóry głowy).

GLEICH SIGN

Gleich's syndrome or episodic angioedema with eosinophilia is a rare disease in which the body swells up episodically (angioedema), associated with raised antibodies of the IgM type and increased numbers of eosinophil in the blood (eosinophilia). It was first described in 1984. Its cause is unknown, but it is unrelated to capillary leak syndrome (which may cause similar swelling episodes) and eosinophilia-myalgia syndrome (which features eosinophilia but alternative symptoms). Moreover, it is not a form of hypereosinophilic syndrome as there is no evidence that it leads to organ damage. Some studies have shown that edema attacks are associated with degranulation (release of enzymes and mediators from eosinophils), and others have demonstrated antibodies against endothelium (cells lining blood vessels) in the condition. Gleich syndrome has a good prognosis. Attack severity may improve with steroid treatment [19].

OBJAW GLEICHA

Zespół Gleicha lub epizodyczny obrzęk naczynioruchowy z eozynofilią jest rzadką chorobą, w której ciało brzęknie epizodycznie (obrzęk naczynioruchowy), w związku z podwyższonymi przeciwciałami w klasie IgM i zwiększoną liczbą eozynofilów we krwi (eozynofilia). Po raz pierwszy został opisany w 1984 roku. Jego przyczyna jest nieznana; nie ma związku z zespołem przesiąkania włośniczek (który może powodować podobne epizody obrzęku) i zespołem eozynofilia-myalgia (w którym eozynofilia jest objawem alternatywnym). Ponadto, nie jest formą zespołu hipereozynofilowego i nie ma dowodów, że prowadzi do uszkodzenia narządów. Niektóre badania wykazały, że ataki obrzęku są związane z degranulacją (wydzielanie enzymów i mediatorów w eozynofili) a inni wykazali przeciwciała przeciw endothelium. Zespół Gleicha ma dobre rokowanie. Nasilenie ataków można poprawić leczenie steroidami [19].

GERALD J. GLEICH

Professor of dermatology and internal medicine (Fig. 14). Gerald J. Gleich of the University of Utah, together with Hans-Uwe Simon of the University of Bern have discovered an unusual mechanism by which eosinophils, the cells involved in immune response, inactivate the bacteria attack the digestive tract.

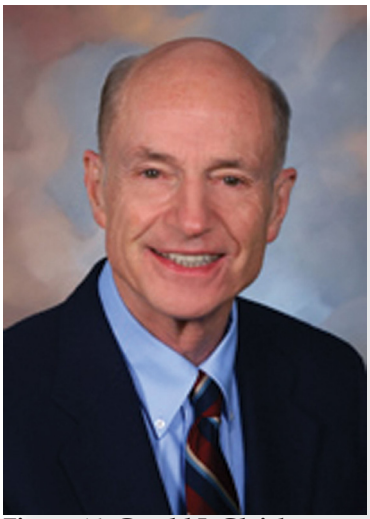

Figure 14. Gerald J. Gleich

Profesor dermatologii i chorób wewnętrznych (Ryc. 14). Gerald J. Gleich z Uniwersytetu Utah razem z Hans-Uwe Simona z Uniwersytetu w Bernie odkryli niezwykły mechanizm, dzięki któremu eozynofile, komórki zaangażowane w odpowiedź immunologiczną, unieszkodliwiają bakterie atakujące przewód pokarmowy.

GOOSE BUMPS SIGN

Presence of goose bumps or goose pimples without temperature cause (Fig. 15), a sign of hypothalamus disorder [20].

Figure 15. Goose bumps sign

OBJAW GĘSIEJ SKÓRKI

Obecność gęsiej skórki bez związku z temperaturą (Ryc. 15), objaw choroby podwzgórza [20].

GORLIN'S SIGN

The ability to touch the tip of nose with the tongue in patients with Ehlers-Danlos syndrome [21].

OBJAW GORLINA

Możliwość dotykania czubka nosa językiem u chorych z zespołem Ehlersa-Danlosa [21].

ROBERT JAMES GORLIN

American pathologist and geneticist, 1923-2006 (Fig. 16). Was a professor and researcher at the University of Minnesota known for pioneering research into craniofacial disorders. He fought in the 2nd world war. Gorlin received numerous honorary degrees from prestigious universities all over the world. He was one of the founders and a diplomate of the American Board of Medical Genetics. His name is associated: Goltz-Gorlin syndrome, Gorlin's cyst, Gorlin's sign, Gorlin's syndrome II, Gorlin's syndrome III, Gorlin-Chaudry-Moss syndrome, Gorlin-Cohen syndrome, Gorlin-Goltz syndrome, Gorlin-Sedano syndrome [22].

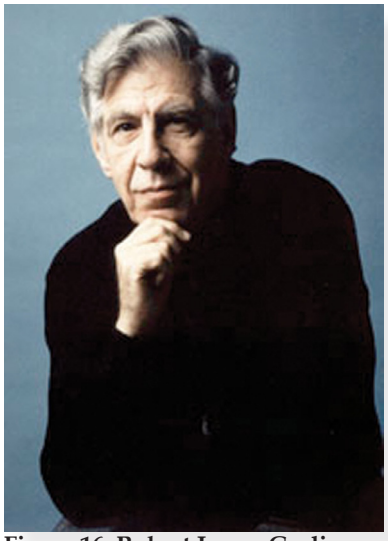

Figure 16. Robert James Gorlin

Amerykański patolog i genetyk, 1923-2006 (Fig. 16). Był profesorem i badaczem z Uniwersytetu Minnesota znany z pionierskich badaniań nad zaburzeniami twarzoczaszki. Walczył w II wojnie światowej. Gorlin otrzymał wiele doktoratów honoris causa z prestiżowych uniwersytetów na całym świecie. Był jednym z założycieli i dyplomatą Amerykańskiej Rady Genetyki. Z jego nazwiskiem związane są: Goltz-Gorlin syndrome, Gorlin's cyst, Gorlin's sign, Gorlin's syndrome II, Gorlin's syndrome III, Gorlin-Chaudry-Moss syndrome, Gorlin-Cohen syndrome, Gorlin-Goltz syndrome, Gorlin-Sedano syndrome [22].

GOTH SIGN

Black lips, as if wearing Goth style makeup, with fever and headache. An early sign of septicaemic plague infection [23].

OBJAW GOTHA

Czarne usta, jakby makijaż w stylu Goth, gorączka i ból głowy. Wczesny objaw posocznicy [23].

GOTTRON'S SIGN

Bluish red plaques on the backs of the fingers, especially over the knuckles, seen in dermatomyositis (Fig. 17,18), and sometimes preceding the onset of muscle weakness by weeks or as long as two to three years [24].

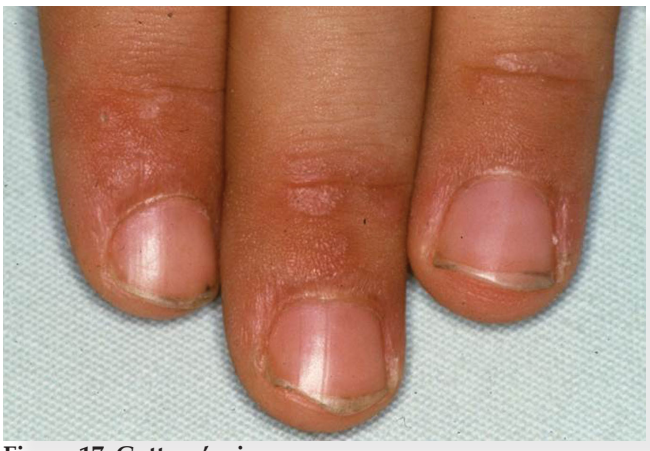

Figure 17. Gottron's sign

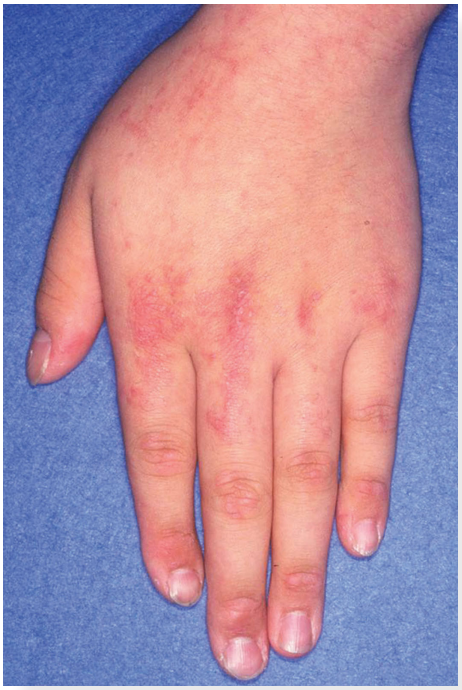

Figure 18. Gottron's sign

OBJAW GOTTRONA

Niebieskawo czerwone blaszki na grzbietach palców, zwłaszcza na kostkach, obserwowane w dermatomiositis (Ryc. 17,18). Czasami poprzedzające początek wystąpienia osłabienia mięśni od tygodni do dwóch, trzech lat [24].

HEINRICH ADOLF GOTTRON

German dermatologist, 1890-1974 (Fig. 19). He published a wide range of articles on contact dermatitis, amyloid and leukaemic infiltrates and skin manifestations. His name is associated: Arndt-Gottron syndrome, Gottron's papules, Gottron's sign, Gottron's syndrome [25].

Niemiecki dermatolog, 1890-1974 (Ryc. 19). Opublikował szereg artykułów na temat kontaktowego zapalenia skóry, amyloidu i białaczkowych nacieków oraz objawów skórnych. Z jego nazwiskiem związane są: Arndt-Gottron syndrome, Gottron's papules, Gottron's sign, Gottron's syndrome [25].

Figure 19. Heinrich Adolf Gottron

GOWERS SIGN

1. Symmetric, progressive, proximal muscle weakness. Gowers' sign is classically seen in Duchenne muscular dystrophy, but also presents itself in dermatomyositis, centronuclear myopathy, myotonic dystrophy and various other conditions associated with proximal muscle weakness [25]. 2. The abrupt intermittent oscillation of the iris under the influence of light. A sign of tabes dorsalis.

OBJAW GOWERSA

1. Symetryczne, postępujące osłabienie mięśni proksymalnych. Objaw Goversa klasycznie występuje w zapaleniu skórno-mięśniowym, dystrofii mięśniowej Duchenne'a, ale również prezentuje się w centralnej miopatii, dystrofii miotonicznej i różnych innych schorzeniach związanych z proksymalnym osłabieniem mięśni [25].
2. Nagłe przerywanie oscylacji tęczówki pod wpływem światła. Objaw w tabes dorsalis.

Sir WILLIAM RICHARD GOWERS

Britisch neurologist, 1845-1915 (Fig. 20). During the latter part of the nineteenth century, made important studies in nervous system physiology and pathology. Gowers was the inventor of the haemoglobinometer in 1878. In 1881 he made a classical description of epilepsy. He was also among the first to recognise the importance of the ophthalmoscope with diseases of the nervous system. In 1892, Gowers was one of the founding members of the National Society for the Employment of Epileptics (now the National Society for Epilepsy). Gowers gave his name to Gowers' sign (a sign of muscular weakness), the Gowers' tract (tractus spinocerebellaris anterior) in the nervous system and Gowers' Round (the National Hospital for Neurology and Neurosurgery's weekly case presentation and clinical teaching session) [26].

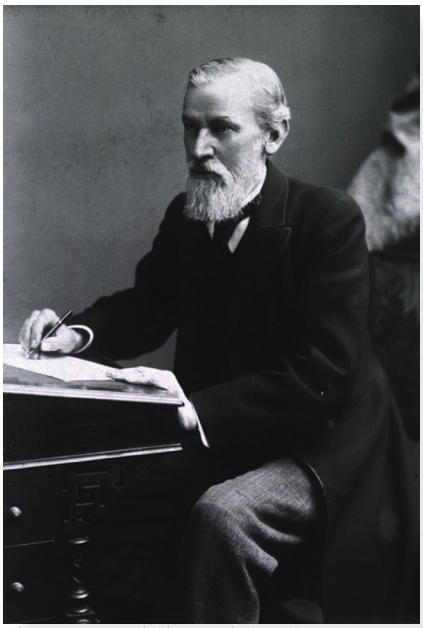

Figure 20. William Richard Gowers

Angielski neurolog, 1845-1915 (Ryc. 20). W drugiej połowie XIX wieku, wykonał ważne badania układu nerwowego w dziedzinie fizjologii i patologii. Gowers był wynalazcą hemoglobinometeru w 1878 roku. W 1881 roku sporządził klasyczny opis padaczki. Był także jednym z pierwszych, którzy zauważyli znaczenie oftalmoskopu w chorobach układu nerwowego. W 1892 Gowers był jednym z członków założycieli Narodowe Towarzystwa Epileptyków (obecnie Narodowe Towarzystwo Padaczki). Gowers dał swoje nazwisko dla objawu Gowersa (objaw osłabienia mięśni), droga Gowersa (droga rdzeniowo móżdżkowa przednia) w układzie nerwowym i „Okrągły Gowers" (tygodniowa prezentacja przypadków i sesje klinicznego nauczania w Narodowym Szpitalu Neurologii i Neurochirurgii) [26].

GRAVIDARUM STRIAE SIGN

White striae on the abdomen which occur in pregnancy [27] (Fig. 21).

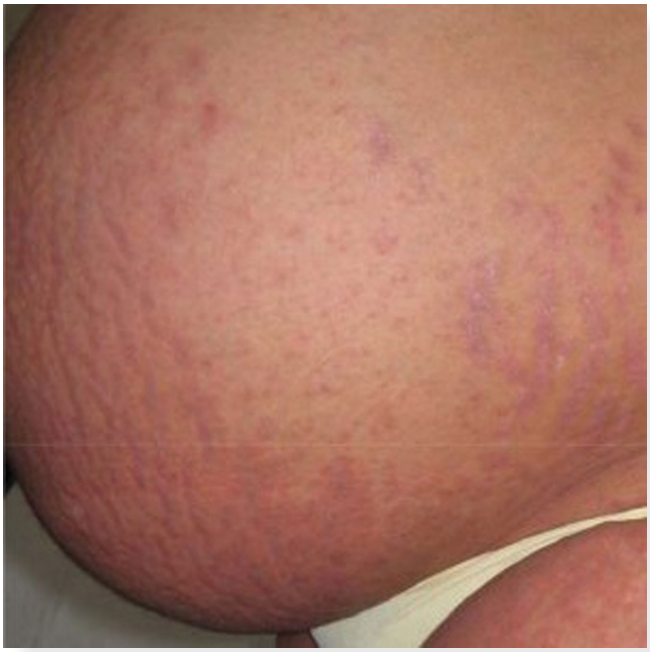

Figure 21. Gravidarum striae sign

OBJAW ROZSTĘPÓW CIĄŻOWYCH

Białe rozstępy na brzuchu, które występują w czasie ciąży [27] (Ryc. 21).

GREEN HAIR SIGN

Green hair as seen in workers of copper smelters [28].

OBJAW ZIELONYCH WŁOSÓW

Zielone włosy u pracowników huty miedzi [28].

GREENBLATT'S SIGN

Linear depression over Poupart's ligament separating draining lymph nodes in lymphogranuloma venereum [29].

OBJAW GREENBLATTA

Linijne zagłębienie wzdłuż więzadła Pouparta oddzielające odprowadzenie węzłów chłonnych w przebiegu lymphogranuloma venereum [29].

GREENHOW'S SIGN

1. Parasitic melanoderma; discoloration of the skin in persons of filthy habits, caused by the irritation of lice. 2. A pigmentary process from an itching disease like prurigo and pityriasis stimulating morbus Addisoni, particulary found in vagrants and tramps. Also called Vagrant's disease and sign [30].

OBJAW GREENHOWA

1. Pasożytnicza melanoderma; przebarwienie skóry u osób o brudnych zwyczajach, spowodowane drażnieniem przez wszy. 2. Barwnikowy proces choroby przebiegającej ze świądem jak prurigo i łupież stymulujące chorobę Addisona, szczególnie występujący u włóczęgów. Zwany także chorobą i objawem włóczęgów [30].

EDWARD HEADLAM GREENHOW

English physician, 1814-1888 (Fig. 22). He studied medicine at Edinburgh and Montpelier. In 1855 he was appointed lecturer on public health at St. Thomas's Hospital. In 1875 he delivered the Croonian lectures at the Royal College of Physicians on Addison's disease. Greenhow wrote: 1. 'On Diphtheria,' 1860. 2. 'On Addison's Disease,' 1866. 3. 'On Chronic Bronchitis,' 1869. 4. 'Croonian Lectures on Addison's Disease,' 1875. He also prepared the following parliamentary reports: 'The different Proportions of Deaths from certain Diseases in different Districts in England and Wales,' 1858 [30].

Figure 22. Edward Headlam Greenhow

Angielski lekarz, 1814-1888 (Ryc. 22). Studiował medycynę w Edynburgu i Montpelier. W 1855 roku został mianowany wykładowcą w zakresie zdrowia publicznego w Szpitalu św. Tomasza. W 1875 roku wygłosił wykłady w Royal College of Physicians o chorobie Addisona. Greenhow napisał: 1. „O błonicy," 1860. 2. „O chorobie Addisona," 1866. 3. „O przewlekłym zapaleniu oskrzeli," 1869. 4. „Croonian-Wykłady o chorobie Addisona," 1875. Przygotowywał również następujące parlamentarne raporty: „Różne proporcje zgonów z powodu niektórych chorób w różnych dzielnicach w Anglii i Walii" 1858 [30].

GREEN NAILS SIGN

Pseudomonas aeruginosa growing within the nail bed [31] (Fig. 23).

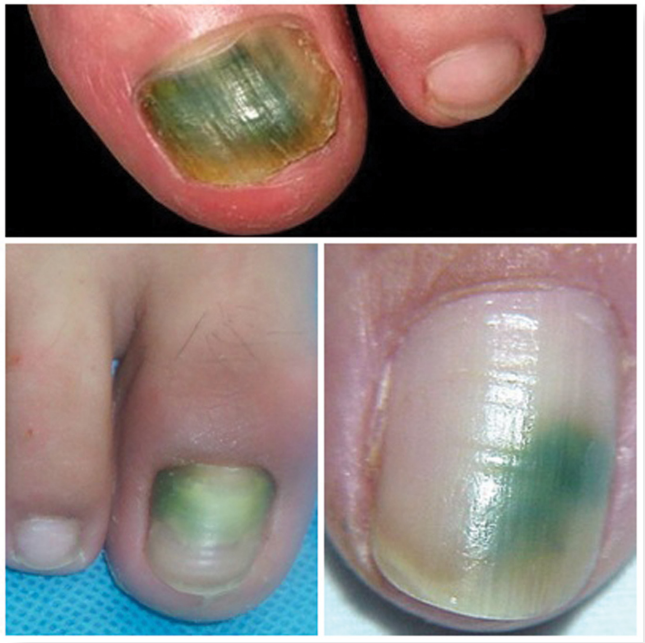

Figure 23. Green nails sign

OBJAW ZIELONEGO PAZNOKCIA

Pseudomonas aeruginosa rosnące w obrębie łożyska paznokcia [31] (Ryc. 23).

GREEN RING SIGN

A thin green stained band of plaque located on the tooth near the gingiva. With other indicators like dry mouth and tar stained fingers, this sign is associated with heavy somking of cannabis.

OBJAW ZIELONEGO PIERŚCIENIA

Cienkie zielone przebarwienie znajdujące się na zębach w pobliżu dziąseł. Inne wskaźniki to, suchość w ustach oraz palce smołowato zabarwione, objaw ten związany z paleniem konopi.

GREY TURNER'S SIGN

Discoloration (bruising) of the skin of the loin in acute hemorrhagic pancreatitis; adverse prognostic (Fig. 24). Also as Turner's sign [32].

OBJAW GREY TURNERA

Przebarwienia (siniaki) skóry w okolicach lędźwi w ostrym krwotocznym zapaleniu trzustki; rokowniczo niekorzystny (Ryc. 24). Zwany również objawem Turnera [32].

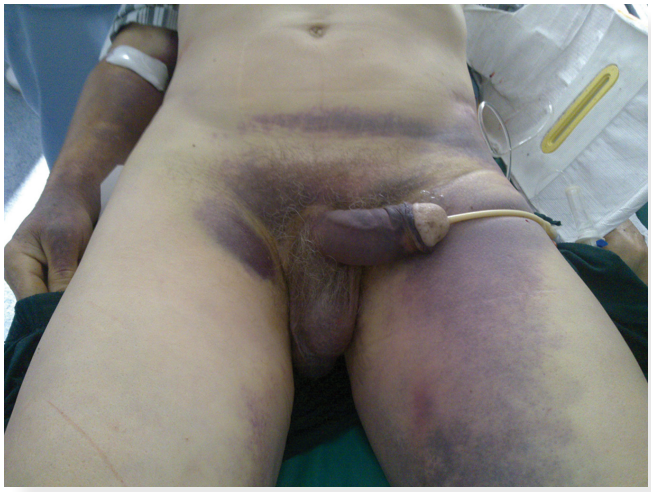

Figure 24. Grey Turner's sign

GEORGE GREY TURNER

English surgeon, 1877-1951 (Fig. 25). Served with the Royal Army Medical Corps in the First World War. First described in 1920 Grey Turner's sign, in the British Journal of Surgery, it was described as a sign of hemorrhagic pancreatitis. As a young surgeon, he travelled around the world, being received by the Pope, Benito Mussolini, the King of Italy and King Alfonso of Spain. Five years before his death, Grey Turner was made President of the International Society of Surgeons. After the war, Grey Turner was briefly famous for performing one of the earliest operations to attempt the removal of a bullet from a soldier's heart. The bullet was never removed, but Grey Turner's surgery saved the patient's life. Worked with early cancer research, and anticipated the development of chemotherapy [33].

Figure 25. George Grey Turner

Angielski chirurg, 1877-1951 (Ryc. 25). Służył w Royal Army Medical Corps w pierwszej wojnie światowej. Pierwszy opisał w 1920 r. objaw Grey Turnera, w British Journal of Surgery, który został opisany jako objaw krwotocznego zapalenia trzustki. Jako młody chirurg, jeździł po całym świecie, był przyjmowany przez papieża, Benito Mussoliniego, króla Włoch i króla Hiszpanii Alfonsa. Pięć lat przed śmiercią, Grey Turner został przewodniczącym Międzynarodowego Towarzystwa Chirurgów. Po wojnie, Grey Turner był znany z wykonania jednego z pierwszych operacji próby usunięcia kuli z żołnierskiego serca. Kula nigdy nie została usunięta, ale chirurgia Grey Turnera uratowała życie pacjenta. Pracował nad początkiem badań nad rakiem i rozwojem chemioterapii [33].

GRIESINGER'S SIGN

Anemia with dropsy, caused by *Ancylostoma duodenale* (Fig. 26) and general malnutrition. Called also cachexia aquosa [34].

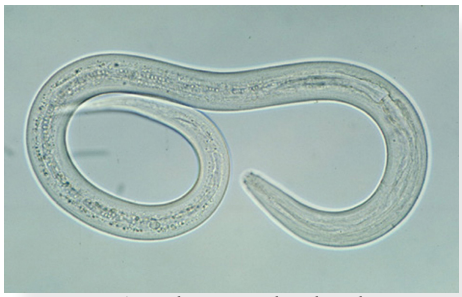

Figure 26. Ancylostoma duodenale

OBJAW GRIESINGERA

Niedokrwistość z obrzękiem spowodowana przez *Ancylostoma duodenale* (Ryc. 26) i ogólne niedożywienie. Zwany również cachexia aquosa [34].

Figure 27. Wilhelm Griesinger

WILHELM GRIESINGER

German neurologist anf psychiatrist, 1817-1868 (Fig. 27). He entered the University of Tübingen as a medical student. Griesinger was not impressed by what his teachers had to offer, stating that he would rather read Johannes Müller (1801-1858), than the obsolete dictates of his teachers. He also read Gabriel Andral's (1797-1876) "Traité d'anatomie pathologique" (3 volumes, Paris, 1829). Obtained his doctorate with a dissertation on diphteriae. In 1840 he seized the opportunity to assume a position as assistant at the lunatic asylum in the royal castle of Winnethal under the directorship of Ernst Albert von Zeller (1804-1877). During the two years he spent here, he gathered a wealth of experience which became the foundation for his famous textbook on mental diseases, first published in 1845.

In 1854 he became full professor of clinical medicine and director of the medical clinic in Tübingen. He founded the "Medicinisch-psychologische Gesellschaft and the Archiv für Psychiatrie und Nervenkrankheiten", soon to become one of the world's leading journals in its field. We also owe to Griesinger the introduction into clinical psychiatry of pathological anatomy [35].

Niemiecki neurolog i psychiatra, 1817-1868 (Ryc. 27). Wstąpił na Uniwersytet w Tybindze jako student medycyny. Griesinger nie był pod wrażeniem tego, co jego nauczyciele mieli do zaoferowania, stwierdzając, że woli czytać Johannesa Müllera (1801-1958), niż to co dyktowali jego nauczyciele. Również czytał Gabriela Andrala Ludowa (1797-1976) „Traité d'anatomie pathologique" (3 tomy, Paryż, 1829). Uzyskał doktorat z pracy doktorskiej na temat diphterii. W 1840 roku skorzystał z okazji i podjął posadę asystenta w przytułku dla obłąkanych na Zamku Królewskim w Winnethal pod kierownictwem Ernsta Alberta von Zellera (1804-1877). Podczas dwóch lat spędzonych tutaj, zebrał ogromne doświadczenie, które stało się podstawą dla jego słynnego podręcznika chorób psychicznych, opublikowanego w 1845 roku. W 1854 roku został profesorem medycyny klinicznej i dyrektorem kliniki medycznej w Tybindze. Założył „Medicinisch-Psychologische Gesellschaft i Archiv für Psychiatrie und Nervenkrankheiten", który wkrótce stał się jednym z wiodących światowych czasopism w swojej dziedzinie. Zawdzięczamy mu wprowadzenie do psychiatrii klinicznej anatomii patologicznej [35].

GRISOLLE'S SIGN
1. If on stretching an affected portion of erupted skin the papule becomes impalpable to the touch. This is a sign of measles. 2. If on stretching an affected portion of erupted skin the papule can still be felt. This is a sign of small pox.

OBJAW GRISOLLE
1. Jeżeli na rozciągniętej, objętej zmianami grudkowymi skórze stają się one niewyczuwalne przez dotyk to jest to objaw odry. 2. Jeżeli na rozciągniętej, objętej zmianami grudkowymi skórze grudki mogą być nadal wyczuwalne, jest to objaw ospy.

Figure 28. Augustin Maurir Grisolle

AUGUSTIN MAURIR GRISOLLE
French physician, 1811-1869 (Fig. 28). Grisolle was a professor to the Paris faculty of medicine and a member of the Académie de Médecine. He was the author of the two-volume Traité élémentaire et pratique de pathologie interne (1844) [36].

Francuski lekarz, 1811-1869 (Ryc. 28). Grisolle był profesorem na wydziale paryskiej medycyny i członkiem Académie de Médecine. Był autorem dwóch wydań „Traité élémentaire et pratique de interne pathologie" (1844) [36].

GRITS SIGN
A diet of maize leads to the niacin deficiency known as pellagra.

OBJAW GRYSU (kasza, łuszczony owies)
Dieta kukurydziana prowadzi do niedoboru niacyny (pellagra).

GROOVE SIGN
1. Enlargement of the nodes above and below the inguinal ligament in patients with lymphogranuloma venereum. Enlargement of the nodes above and below the inguinal ligament [37]. 2. Loss of perivascular fat tissue accounts for the groove sign when the arm is elevated in eosinophilic fasciitis [38] (Fig. 29).

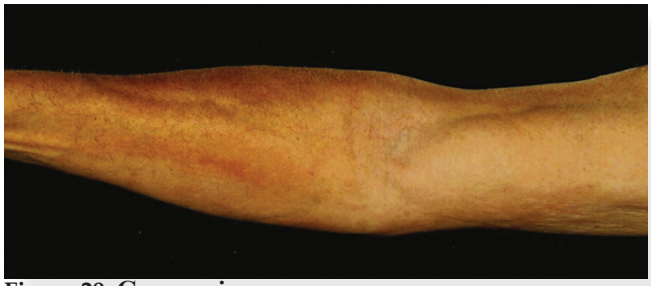
Figure 29. Groove sign

OBJAW ROWKA
1. Powiększenie węzłów powyżej i poniżej więzadła pachwinowego u pacjentów z Lymphogranuloma weneryczna. Powiększenie węzłów powyżej i poniżej więzadła pachwinowego [37]. 2. Utrata okołonaczyniowego tłuszczu wokół tkanek, gdy ramię jest podniesione [38] (Ryc. 29).

GRUBY'S SIGN
A form of tinea seen in children, and due to the fungus *Trichophyton tonsurans* (Fig. 30, 31). It is a common cause of tinea capitis [39].

OBJAW GRUBY'a
Forma grzybicy u dzieci, wywołana przez *Trichophyton tonsurans* (Ryc. 30, 31). Jest przyczyna grzybicy skóry owłosionej głowy [39].

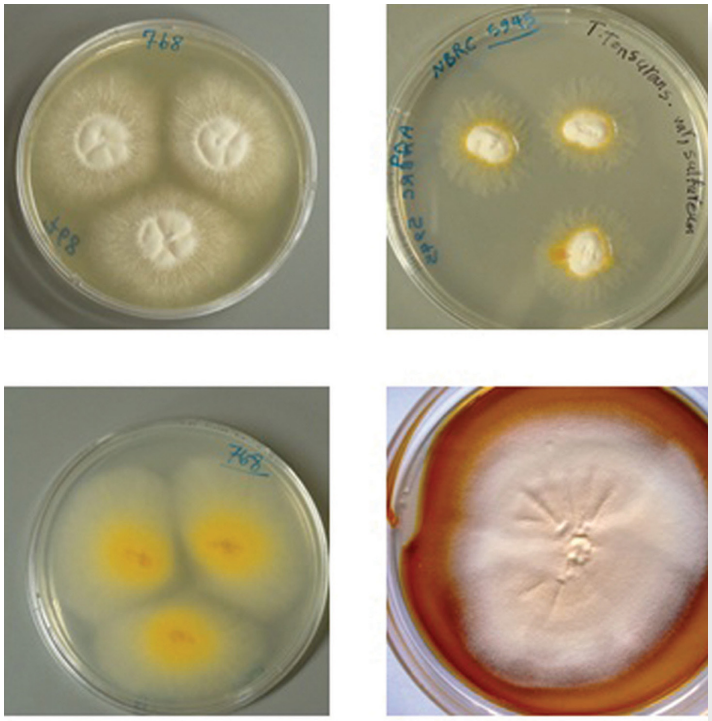

Figure 30. Trichophyton tonsurans

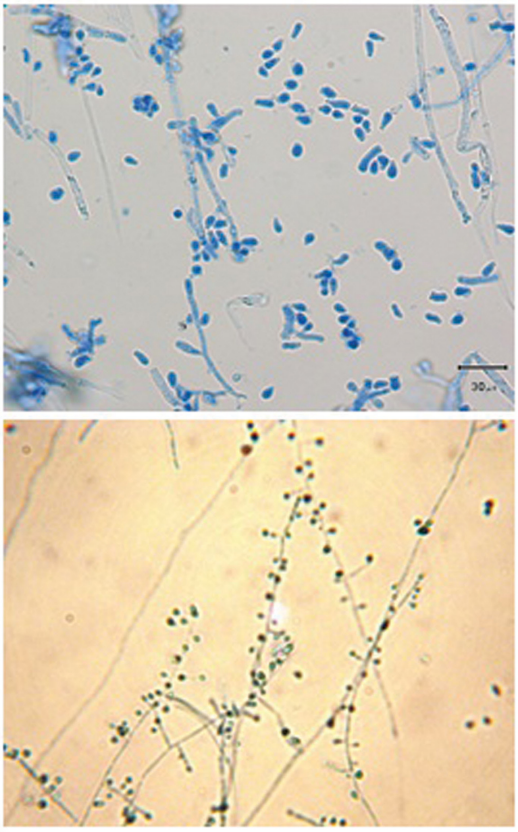

Figure 31. Trichophyton tonsurans

DAVID GRUBY

Hungarian physician, 1810-1898 (Ryc. 32). He received his doctorate in Vienna and performed scientific research in Paris. Gruby is remembered as a pioneer in the fields of microbiology and medical mycology. In 1841 he described the fungus that causes favus. This discovery was independent of Johann Lukas Schönlein's findings. Later, this fungal parasite was called *Achorion schoenleinii* in Schönlein's honor. In 1842 he described a microscopic cryptogam (*Trichophyton ectothrix*) which causes a sycosis barbae. Gruby also discovered *Candida (Monilia) albicans*, the cause of candidiasis, and in 1843 he described a fungus (*Microsporum audouini*). This fungus was named after naturalist Jean Victor Audouin (1797–1842). During the early years of anaesthesia, he performed important experiments with chloroform and ether on animals [40].

Węgierski lekarz, 1810-1898 (Ryc. 32). Doktoryzował się w Wiedniu i wykonywał badania naukowe w Paryżu. Gruby jest wspominany jako pionier w dziedzinie mikrobiologii i mikologii lekarskiej. W 1841 roku opisał grzyba powodującego „favus". Odkrycie to było niezależne od wyników Johanna Lukasa Schönleina. Później ten grzybiczy pasożyt został nazwany na cześć Schönleina *Achorion schoenleinii*. W 1842 roku opisał mikroskopijny kryptogam (*Trichophyton ectothrix*), który powoduje figówkę brody. Gruby odkrył również *Candida (Monilia) albicans*, przyczynę kandydozy, a w 1843 roku opisał grzyb (*Microsporum audouini*). Grzyb ten został nazwany na cześć przyrodnika Jeana Victora Audouin (1797-1742). W pierwszych latach anastezji, dokonywał ważnych eksperymentów z chloroformem i eterem na zwierzętach [40].

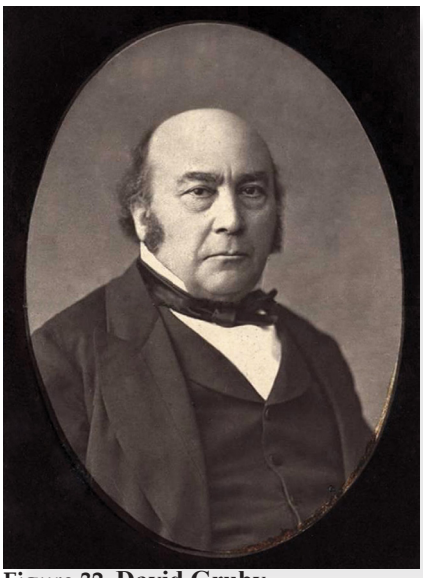

Figure 32. David Gruby

GUBLER'S SIGN

A distinct swelling on the wrist in lead poisoning paralysis [41] (Fig. 33).

OBJAW GUBLERA

Wyraźny obrzęk na nadgarstku w przebiegu porażenia ręki w zatruciu ołowiem [41] (Ryc. 33).

Figure 33. Gubler's sign

ADOLPHE MARIE GUBLER

French physician and pharmacologist, 1821-1879 (Fig. 34). Originally a student of botany, he began his medical studies in 1841 at Paris. In 1845 he became an interne des hôpitaux, earning his doctorate in 1849. Afterwards he worked as a physician at the Hôpital Beaujon. Gubler made a number of contributions in the fields of medicine and pharmacology. He is credited with being the first physician to differentiate between hemotogenous and hepatogenous icterus. His name is associated with „Millard-Gubler syndrome". He was the author of many works on botany, clinical medicine, physiology and pharmacology. Among his better known publications was an 1856 work on hemiplegia titled "De l'hémiplégie alterne envisagée comme signe de lésion de la protubérance annulaire et comme preuve de la décussation des nerfs faciaux". Gubler was a founding member of the Société de biologie [42].

Figure 34. Adolphe Marie Gubler

Francuski lekarz i farmaceuta, 1821-1879 (Ryc. 34). Początkowo student botaniki, rozpoczął studia medyczne w 1841 roku w Paryżu. W 1845 roku został internistą, zdobywając doktorat w 1849 roku. Następnie pracował jako lekarz w Hôpital Beaujon. Gubler dokonał kilku odkryć w dziedzinie medycyny i farmakologii. Przypisuje mu się jako pierwszemu lekarzowi odróżnienie żółtaczki hematogennej i hepatogennej. Jego nazwa wiąże się z zespołem Millard-Gubler. Był autorem wielu prac na temat botaniki, medycyny klinicznej fizjologii i farmakologii. Wśród jego bardziej znanych publikacji była praca z 1856r. nad hemiplegią zatytułowana: „De l'hémiplégie alterne envisagée comme signe de lésion de la protubérance annulaire et comme preuve de la décussation des nerfs faciaux". Gubler był członkiem i założycielem Société de Biologie [42].

GUILT SIGN

Abnormally feeling guilty, can progress to feeling paranoid, accompanied with paralysis and fever. An indication of B-12 deficiency caused by a parasitic tapeworm infection [43] (Fig. 35).

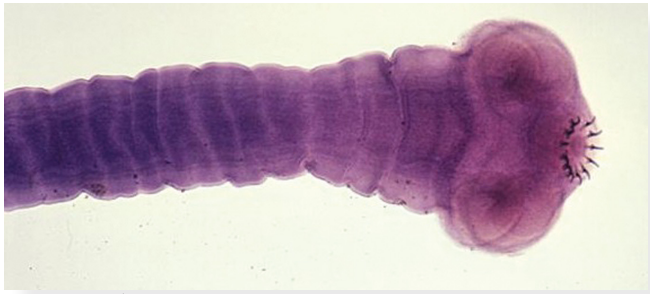

Figure 35. Adult tapeworm

OBJAW WINY

Nienormalnie uczucie winy, może przejść do uczucie paranoi, wraz z paraliżu i gorączką. Związane z niedoborem witaminy B-12 spowodowane przez pasożytnicze zakażenie tasiemcem [43] (Ryc. 35).

ACKNOWLEDGEMENT

to Figure 12 and 13 [17].
I published the pictures in as a reference, in keeping with copyright: Priya Gopie and Vijay Naraynsingh. The International Journal of Lower Extremity Wounds, June 2011; vol.10, 2: pp. 91-92., first published on May 26, 2011 http://online.sagepub.com.

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