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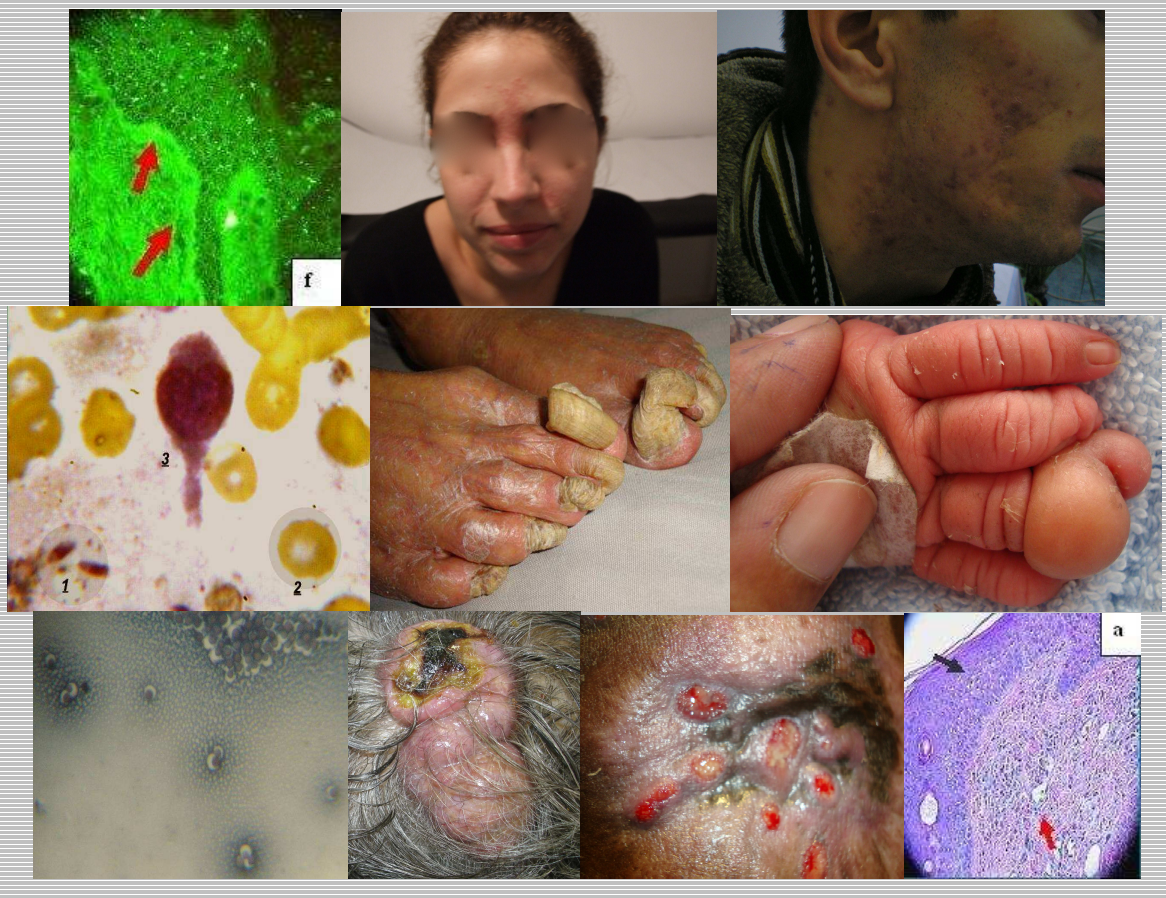

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SKIN APPENDAGEAL IMMUNE REACTIVITY IN A CASE OF CUTANEOUS LUPUS

REAKTYWNOŚĆ IMMUNOLOGICZNA PRZYDATKÓW SKÓRY NA PRZYKŁADZIE PRZYPADKU SKÓRNEJ POSTACI TOCZNIA

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Abstract
Background: Discoid lupus erythematosus is a cutaneous disease with a worldwide distribution, and its pathogenesis remains unclear. **Case Report:** A 41 year old male was evaluated for hair loss, in patches on the scalp. We studied selected adaptor proteins expressed in T, natural killer, neutrophil and mast cells; these proteins are important mediators for antigen receptor signaling in situ. **Methods:** Skin biopsies for hematoxylin and eosin examination, as well as for direct immunofluorescence and immunohistochemistry analysis were performed. **Results:** Hematoxylin and eosin staining demonstrated classic features of lupus with focal dermal scarring; epidermal atrophy was noted, with lymphohistiocytic infiltrates around the skin appendages. Direct immunofluoresence revealed classic, lupus band positive staining along the dermal/epidermal junction. In addition, immune reactants were identified in neurovascular areas, and around pilosebaceous units. Immunohistochemistry staining showed positive staining for the T-cell antigen receptor zeta chain, the linker for activation of T cells, myeloperoxidase, cyclo-oxygenase 2, melanoma-associated antigen 1, B cell leukemia/lymphoma-2 associated X protein, and BCL-2 markers. The positive staining was observed within the dermal inflammatory infiltrate, around pilosebaceous units, upper dermal blood vessels, and focally within eccrine sweat glands. **Conclusions:** The pathobiology of cutaneous lupus involves not only the epidermis, but also dermal pilosebaceous units, eccrine sweat glands and blood vessels. Further studies are recommended, especially in the light of presented data regarding T cell activation and proapototic molecules.

Streszczenie
Wstęp: Dyskoidalny toczeń rumieniowaty jest chorobą skóry z dystrybucją na całym świecie, a jej patogeneza jest niejasna. **Opis przypadku:** 41-letni mężczyzna został diagnozowany z powodu wypadania włosów na skórze głowy. Badano ekspresje selektywnego adaptera białek na limfocytach T, komórkach-natural killer, neutrofilach i komórkach tucznych; białka te są ważnymi mediatorami receptora antygenu sygnalizacji in situ. **Metody:** Wykonano biopsję skóry w barwieniu hematoksyliną i eozyną, jak również immunofluorescencję bezpośrednią i analizę immunohistochemiczną. **Wyniki:** Barwienie hematoksyliną i eozyną wykazały klasyczne cechy tocznia z ogniskową blizną skóry; obserwowano zanik naskórka z lymphohistiocytarnymi naciekami w obrębie przydatków skóry. Immunofluorescencja bezpośrednia wykazała klasyczne, lupus band pozytywne barwienie wzdłuż połączenia skóra / naskórek. Ponadto, immunologiczne reagenty zostały zidentyfikowane w obszarach naczyniowych i w okolicy jednostek włosowo-łojowych. Barwienia immunohistochemiczne wykazały pozytywne barwienie dla antygenu T-cell receptor łańcucha zeta, łącznika do aktywacji limfocytów T, MPO, cyklooksygenazy 2, antygenu związanego z czerniakiem 1, komórek B leukemia/lymphoma-2 związanych z białkiem X i BCL- 2 markera. Pozytywne barwienie stwierdzono w nacieku zapalnym skóry, w obrębie jednostek włosowo-łojowych, górnych naczyń krwionośnych skóry i ogniskowo w ekrynowych gruczołach potowych. **Wnioski:** Patobiologia skórnej postaci tocznia nie dotyczy tylko naskórka, ale również skórnych jednostek włosowo-łojowych, ekrynowych gruczołów potowych i naczyń krwionośnych. Niezbędne są dalsze badania, zwłaszcza w świetle przedstawionych danych dotyczących aktywacji komórek T i proapototycznych cząsteczek.

Key words: cutaneous lupus; ZAP-70; LAT; BCL-2; myeloperoxidase; COX-2; MUM-1; pilosebaceous unit; sweat glands
Słowa klucze: skórna postać tocznia; ZAP-70; LAT; BCL-2; mieloperoksydaza; COX-2; MUM-1; jednostka włosowo-łojowa; gruczoły potowe

Introduction

Cutaneous lupus erythematosus (LE) covers a broad morphological spectrum, extending beyond acute, subacute and chronic cutaneous lupus erythematosus, which are commonly classified as lupus-specific skin disease [1]. Other, less common presentrations include tumid lupus erythematosus, lupus profundus, chilblain lupus, mucosal lupus erythematosus and bullous lupus erythematosus [1]. Possible vascular sequelae of lupus erythematosus include leukocytoclasis, urticarial

vasculitis, livedoid vasculopathy and livedo reticularis [1]. Many previous histologic investigations have emphasized 1) immune deposits at the dermoepidermal junction (DEJ) of the base membrane zone (BMZ), i.e., the lupus band), and 2) the role of autoantibodies and antibody dependent cellular cytotoxicity in the pathogenesis of LE [1]. It is known that autoimmune T helper cells drive pathogenic autoantibody production in LE, but the mechanisms maintaining those pathogenic T cells are unknown. Here, we explore pertinent T cell signaling activators, and the possible roles of other immune cells mediators in lupus erythematosus. We include products derived from activated neutrophils, such myeloperoxidase; and also other molecules located in a lupus susceptibility region on chromosome 1, such as cyclo-oxygenase 2.

Case report

A 41 year old male was evaluated for the presence of hair loss, in patches in the scalp. On physical exam, the patient demonstrated atrophic scaly scalp plaques, with hair loss inside the plaques. A lesional skin biopsy was taken for hematoxylin and eosin (H&E) analysis. In addition, direct immunofluorescence (DIF) and immunohistochemistry (IHC) studies were performed.

Methods

In brief, skin preparations for H&E, DIF and IHC studies were performed as previously described. We utilized antibodies from Dako (Carpinteria, California USA), including 1) anti-human cyclo-oxygenase 2 antibody (COX-2), which does not crossreact with cyclo-oxygenase 1, 2) the T-cell antigen receptor zeta chain (ZAP-70 antibody), 3) the linker for activation of T cells (LAT), myeloperoxidase, 4) mutated melanoma-associated antigen 1 (MUM-1), 5) B cell leukemia/lymphoma-2 associated X protein (BAX) and 6) BCL-2 antibody. The direct immunofluorescence (DIF) and immunohistochemistry (IHC) studies performed as previously described [2-10].

Results

Microscopic description:

Examination of the H&E tissue sections demonstrated classic features of lupus erythematosus; focal atrophy and follicular plugging were noted within the epidermis. The presence of a mild, perifollicular concentric fibrosis and in few areas with some scarring was seen. No significant interface inflammation was noted. A mild, superficial and deep, perivascular and periadnexal dermal infiltrate of lymphocytes, lymphocytes, plasmacytoid lymphocytes and histiocytes was noted. Neutrophils and eosinophils were rare. Mild, perifollicular concentric fibrosis was observed, with additional areas of interstitial scarring. The Verhoeff elastin special stain confirmed the extent of dermal scarring (Fig. 1,2). Focal edema was also appreciated around sebaceous and eccrine sweat glands. DIF demonstrated the following staining results: IgG (++, focal and linear at the epidermal basement membrane zone (BMZ), sebaceous gland BMZs and within selected neurovascular structures; IgA (-); IgM (+, focal, linear epidermal BMZ and dermal perivascular); IgE (+, in selected papillary dermal cells; Complement/C1q (++, focal and linear at epidermal BMZ and sebaceous gland BMZs); Complement/C3 (++, focal linear epidermal BMZ and sebaceous gland BMZs, and surrounding some dermal neurovascular structures; Complement/C4 (++, focal perifollicular and surrounding dermal neurovascular structures); Kappa and Lambda light chains (+, focal punctate epidermal BMZ, and surrounding dermal neurovascular structures and sweat glands); Albumin (++, focal linear epidermal BMZ and sebaceous gland BMZs); fibrinogen (++, focal linear epidermal BMZ and sebaceous gland BMZs) and, finally, ZAP-70, LAT, myeloperoxidase, COX-2, MUM-1, and BCL-2 (+), around dermal sebaceous and sweat glands, and dermal blood vessels (Fig. 1,2). In addition, some of these final antibodies clearly reacted with the BMZs of the sebaceous and sweat glands.

Discussion

Consistent histopathologic features in many cutaneous lesions of LE include a perivascular mononuclear cell infiltrate, with subsequent involvement of the epidermis and dermal appendages. The histologic alterations affecting the epidermis, dermis, and adnexal structures reflect the specific lesion biopsied, and lesional age at the time of biopsy [11,12]. The multiple clinical and serologic forms of lupus erythematosus cannot reliably be distinguished histologically, which supports the premise that lupus erythematosus is a disease presenting a wide spectrum of clinical manifestations and a common underlying pathogenesis [11,1 2].

In our case, we were able to observe how skin adnexal structures and dermal blood vessels interfaced with the pathologic inflammatory infiltrate; specifically, T cells and neutrophil activated, downstream cell signaling molecules seem to play significant roles in the pathophysiology. Most of the markers of immune activation were identified in the adnexal structures. We were able to identify the presence of COX-2, an inducible enzyme that is normally absent in skin cells; however, in response to growth factors, tumor promoters and some cytokines, it exhibits a rapid and transient expression. In addition, ZAP-70 plays a role in lymphocyte activation. It is known that autoimmune T helper cells drive pathogenic autoantibody production in systemic lupus erythematosus (SLE); however, the pathologic mechanisms maintaining these T cells are unknown. Here we demonstrated both TCR (T-cell antigen receptor) mediated and pre-TCR mediated signaling, both in developing and mature T lymphocytes. LAT is involved in FcGR3 (low affinity immunoglobulin gamma Fc region receptor III)-mediated signaling in natural killer cells, as well as FcER1 (high affinity immunoglobulin epsilon receptor)-mediated signaling in mast cells. Coupled activation of these receptors and their associated kinases would then result in downstream intracellular events, including mobilization of intracellular calcium stores, PKC activation, MAPK activation and/or cytoskeletal reorganization through the recruitment of PLCG1, GRB2, GRAP2, and other signaling molecules.

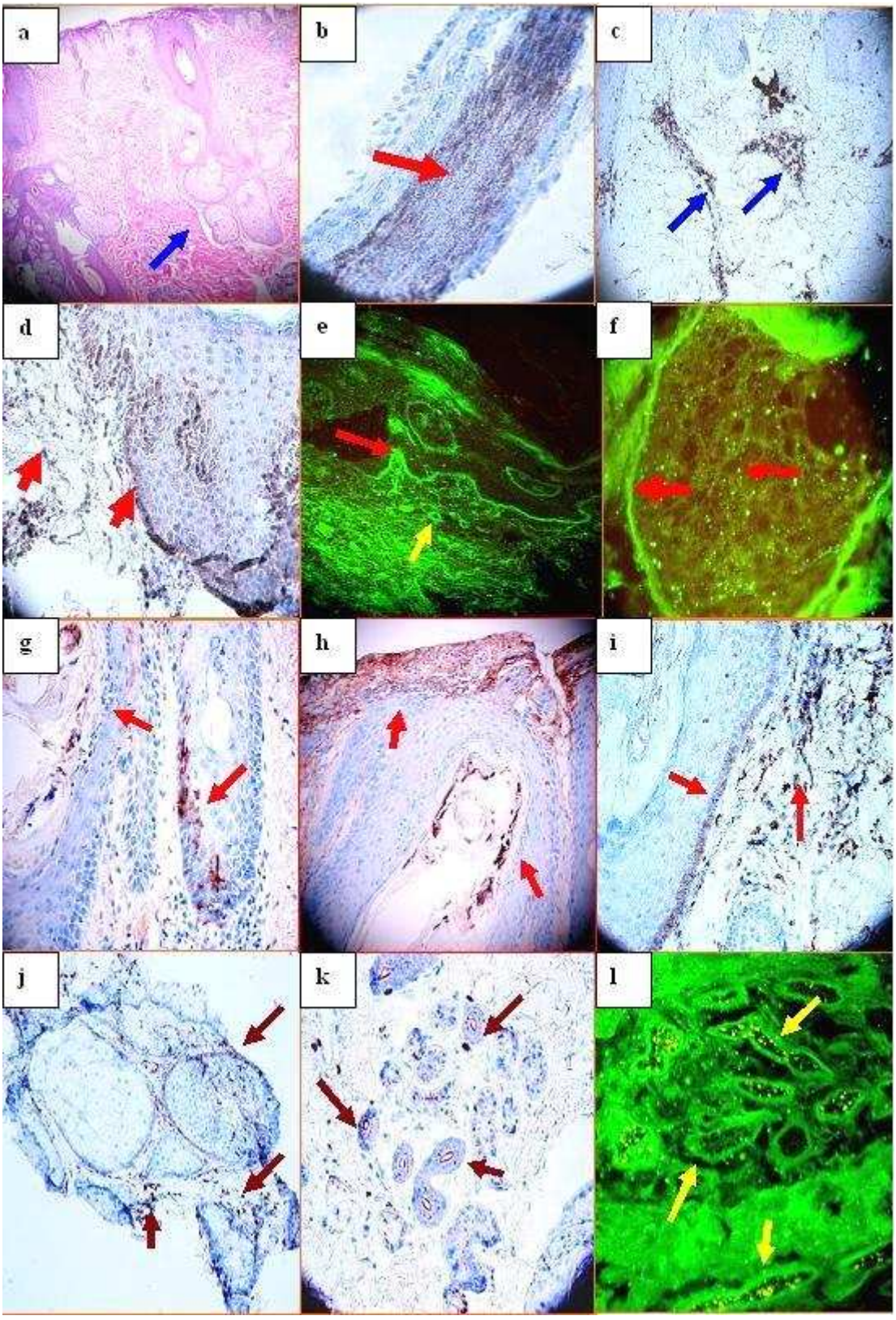

Figure 1. a. H&E, showing atrophy of the epidermis, follicular plugging and no significant hyperkeratosis Mild, perifollicular concentric fibrosis is present, with additional focal interstitial scarring. A mild, superficial, perivascular and periadnexal dermal inflammatory infiltrate is also noted. Focal edema is present within the dermis, especially around hair follicular units (blue arrow). **b.** Positive staining within a hair follicle with the MUM-1 antibody (brown staining; red arrow). **c.** Positive staining with LAT antibody on an upper dermal perivascular infiltrate (brown staining; blue arrows). **d.** Positive staining with anti-BAX antibody accentuated at the basement membrane zone (BMZ) of the epidermis, as well as on individual cells within the papillary dermis(brown staining; red arrows). **e.** Positive staining with FITC conjugated anti-human-Complement/C3 against the BMZ of the epidermis (green staining; red arrow) and against upper dermal blood

vessels (green staining; yellow arrow). **f.** Positive staining with FITC conjugated anti-human-complement/C3 against the BMZ of a sebaceous glands and some areas within the gland (green staining; red arrows). **g.** IHC positive staining of a hair follicle with complement/C3, at the BMZ of the hair follicle and inside the hair follicle (brown staining; red arrows).
h. Complement/C1q positive IHC staining inside the hair follicle shaft and at the epidermal BMZ area (brown staining; red arrows). **i**, COX-2 positive staining at the BMZ of a hair follicular unit and in a perifollicular inflammatory infiltrate(dark staining; red arrows). **j**. IHC positive staining with BCL-2 on inflammatory cells around sebaceous glands (dark staining; maroon arrows). **k**. IHC positive staining with ZAP-70 inside an eccrine gland coil (dark staining; maroon arrows). **l.** DIF positive staining against eccrine sweat glands using FITC conjugated anti-human IgG (green staining; yellow arrows).

Autoreactive T cells are normally eliminated by 1) functional inactivation (anergy) and 2) activation-induced cell death (AICD; directed apoptosis) through death receptor (Fas) signaling**.** Other authors have reported that activated T cells of lupus patients resist anergy and apoptosis by markedly upregulating and sustaining COX-2 expression [13]. Inhibition of COX-2 caused apoptosis of the anergy-resistant lupus T cells by augmenting Fas signaling and markedly decreasing the survival molecule c-FLIP (cellular homolog of viral FLICE inhibitory protein) [13]. Studies with COX-2 inhibitors and COX-2 deficient mice confirmed that this COX-2/FLIP antiapoptosis program has been used selectively by anergy-resistant lupus T cells, and not by cancer cells or other autoimmune T cells [13]. Notably, the gene encoding COX-2 is located in a lupus-susceptibility region on chromosome 1. The same authors also found that selected COX-2 inhibitors were able to suppress the production of pathogenic autoantibodies to DNA by causing autoimmune T-cell apoptosis, an effect that was independent of prostaglandin E_2 (PGE_2) [13]. These findings could be useful in the design of lupus therapies. Our COX-2 data is consistent with expression of this molecule in the context of clinical lupus erythematosus.

The ZAP-70 gene encodes an enzyme belonging to the tyrosine kinase protein family; ZAP-70 plays roles in both T lymphocyte development and activation [14]. The ZAP-70 enzyme, which is phosphorylated on its tyrosine residues upon T cell antigen receptor (TCR) stimulation, functions in the initial step of TCR-mediated signal transduction in combination with Src family kinases. Mutations in the ZAP-70 gene result in a form of severe combined immunodeficiency (SCID) syndrome in humans [14]. ZAP-70 expression is also found in a subset of chronic lymphocytic leukemia patients with unmutated Ig genes and a poor clinical course [14]. LAT, a transmembrane adaptor protein expressed in T lymphocytes, natural killer cells and mast cells, is also an important mediator for TCR signaling [14]. Upon TCR engagement, activated ZAP-70 phosphorylates LAT at multiple conserved tyrosine residues within SH2 binding motifs, exposing these motifs as the docking sites for downstream signaling targets [14]. The phosphorylation process in LAT eventually leads to activation of the corresponding signaling pathways [14].

In regard to the positive staining for BAX, the BAX protein is a pro-apoptotic member of the Bcl-2 family of proteins; BAX is found in many cell types [15]. Bax is a cytosolic protein that translocates to the mitochondria and participates in Cytochrome c release in response to apoptotic stimuli [15]. Aberrant expression of BCL-2 family members can inappropriately promote or prevent apoptosis. Increased expression of BAX is associated with the apoptotic loss of neurons in Parkinson's disease [15]. Given 1) the presence of junctional zone cytoid bodies in many cases of LE and 2) our BAX IHC findings, the role of BAX proapototic molecules in LE warrants further investigation.

Based on our findings, the T cells and the cell signaling cascades are actively targeting not only the BMZ, but also skin adnexal structures; thus, the process may the result in dermal scarring and hair loss in some patients with LE. Additional studies are necessary to further explore and define these pathophysiologic possibilities.

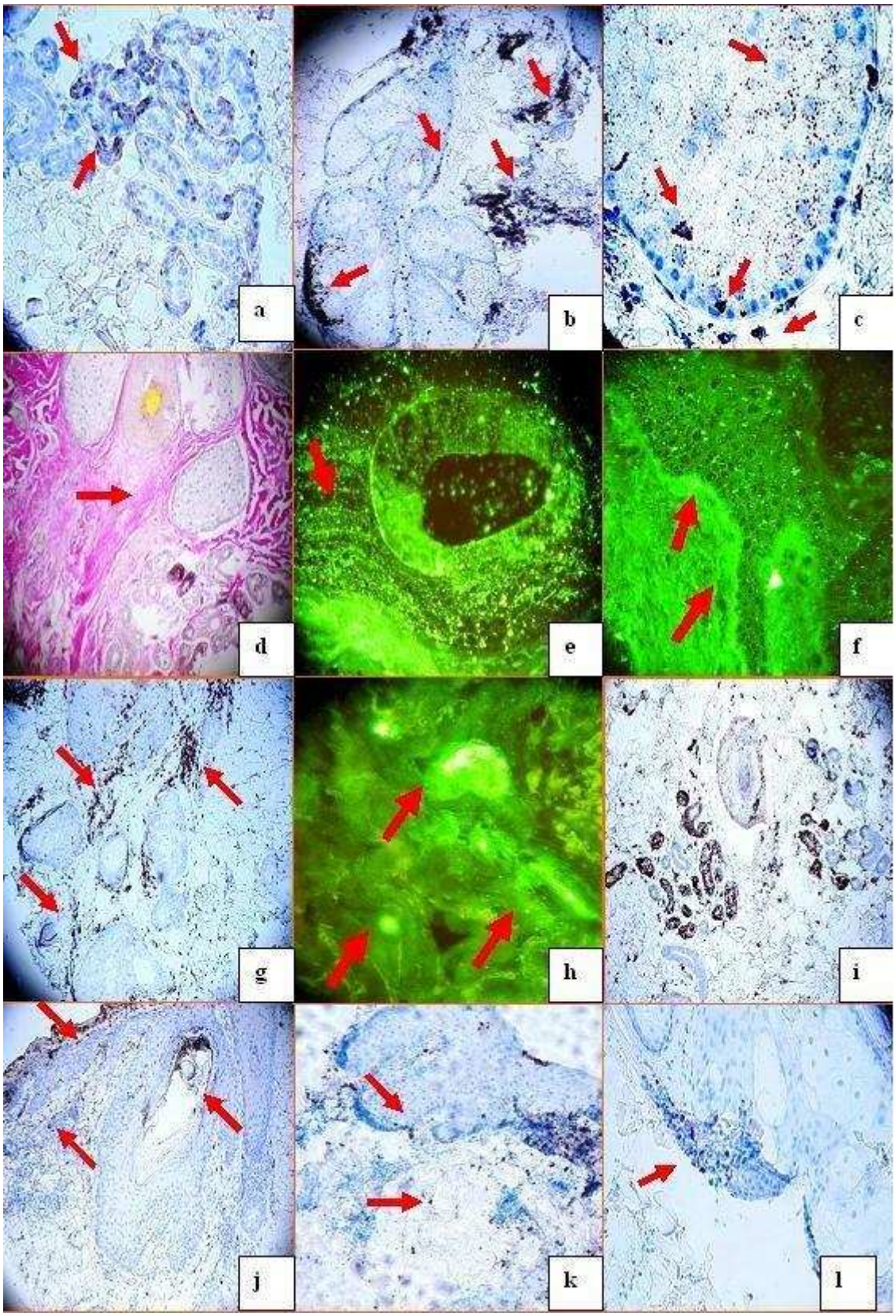

Figure 2. **a**. Positive IHC staining using anti-human BCL-2 antibody against some areas of an eccrine sweat glands (dark-brown staining; red arrows), **b.** Positive anti-human-ZAP-70 antibody staining on a sebaceous gland. Please notice how some staining areas are accentuated along the BMZ of the gland, and some staining areas accentuated within the gland interior(brown staining; red arrows)**. c.** ZAP-70 positive IHC staining in multiple patterns within and around a sebaceous gland. The patterns include dots inside and outside the gland, clustered dot staining, and along the gland BMZ (brown staining; red arrows). **d.** A Verhoeff elastin special stain confirms the extent of dermal scarring (red arrow). **e**, DIF positive

staining with FITC conjugated anti-human-IgG, in a punctate dot pattern around a hair follicle unit (yellow staining; red arrow). **f,** Positive DIF lupus band staining at the epidermal BMZ with FITC conjugated anti-human-complement/C3(faint yellow staining; red arrows). **g** Positive IHC staining with anti-human LAT antibody around a hair follicular unit (brown stain, red arrows). **h.** Positive DIF staining with FITC conjugated anti-human-Complement/C3 against some neurovascular structures (yellow/green staining; red arrows). **i**. Positive IHC staining with anti-human COX-2 in a hair follicle and the eccrine gland coils (brown staining). **j.** Positive follicular and perifollicular IHC staining with anti-human myeloperoxidase (brown staining; red arrows). **k.** Positive IHC staining with anti-human myeloid histiod antigen antibody within and near a sebaceous gland (brown staining; red arrows). **l.** Positive IHC staining with anti-human MUM-1 antibody around a hair follicle (brown staining; red arrow).

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COMPARISON OF SEROPOSITIVITY OF HCV BETWEEN ORAL LICHEN PLANUS AND HEALTHY CONTROL GROUP IN HAMEDAN PROVINCE (WEST OF IRAN)

PORÓWNANIE POMIĘDZY HCV SEROPOZYTYWNYMI PACJENTAMI Z LISZAJEM PŁASKIM JAMY USTNEJ A ZDROWĄ KONTROLNĄ GRUPĄ Z PROWINCJI HAMEDAN (ZACHODNI IRAN)

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Abstract
Background: Lichen planus is an idiopathic inflammatory disease of the skin, nail, hair and mucous membranes. Oral lichen planus (LP) is a chronic inflammatory condition that affects the oral mucous membranes with a variety of clinical presentations. Various etiologies include HCV suggested for LP, and the aim of this study was comparison of seropositivity of HCV in LP patients and control group. **Methods:All** oral LP patients that were referred to dermatology clinic of farshchian hospitalwere entered in the study. Five cc of clot blood was taken from each patient and tested for anti-HCVand when anti-HCV tested positive another 2cc clot bloodwas taken for HCV-Rt-PCR test. The results were analyzed with SPSS 16. **Results:** This prospective cross-sectional study was conducted on 30 oral lichen planus patients [males 13(43.3%) females 17(56.7%)] with mean ages of 46±13.7years and 60 healthy individual [males 26(43.3%) females 34(56.7%)]. There was no oral lichen planus patients who had anti-HCV positive whiles 2 males(3.3%) of healthy group had anti-HCV positive which was confirmed by HCV-Rt-PCR. **Conclusions:** This study showed that there is no correlation between seropositivity of HCV and oral lichen planus in our patients in the west of Iran.

Streszczenie
Wstęp: Liszaj płaski jest idiopatyczną zapalną chorobą skóry, paznokci, włosów i błon śluzowych. Liszaj płaski jamy ustnej (LP) jest przewlekłą chorobą zapalną, która wpływa na błony śluzowe jamy ustnej w różnych klinicznych prezentacjach. Jednym z czynników etiologicznych PL jest HCV, a celem tego badania było porównanie dodatniego HCV u pacjentów z LP oraz w grupie kontrolnej. **Metody:** Pacjenci Kliniki Dermatologii szpitala w Farshchian z pełną ustną postacią LP zakwalifikowali się do badania. Od każdego pacjenta pobrano próbki po 5 cm^2 skrzepów krwi i badano na obecność przeciwciał anty-HCV, gdy wyniki anty-HCV były pozytywne skrzepy krwi badano testem HCV-RT-PCR. Następnie wyniki analizowano z SPSS 16. **Wyniki:** To prospektywne badanie przekrojowe było przeprowadzone na 30 pacjentach z liszajem płaskim jamy ustnej [13 mężczyzn (43,3%), 17 kobiet (56,7%)] w średnim wieku 46 ± 13,7 lat oraz na 60 zdrowych osobach, [26 mężczyzn (43,3%), 34 kobiet (56,7%)]. Liszaj płaski jamy ustnej nie występował u pacjentów, którzy byli anty-HCV, z kolei u 2 mężczyzn (3,3%) z grupy zdrowych stwierdzono pozytywne wyniki anty-HCV, które potwierdzone były w badaniu HCV-RT-PCR. **Wnioski:** Badanie wykazało, że nie znaleziono korelacji między dodatnim wynikiem HCV i liszajem płaskim jamy ustnej u naszych pacjentów w zachodniej części Iranu.

Key words: lichen planus; anti-HCV; HCV-Rt-PCR
Słowa klucze: liszaj płaski; anti-HCV; HCV-Rt-PCR

Introduction

Lichen planus is a skin disease with the emergence of clinically flat papules with the appearance of a shiny purple polygon with different sizes. The disease can anywhere affect the body but most common sites are wrist, waist and around the ankle area. Mucosal involvement is very common. Approximately, 30-70% of patients have mucosal lesions [1]. Mucosal involvement, even alone and without skin symptoms can occur. In the mouth, the most common site is buccal mucosa and tongue [1,2]. Oral lichen planus is a chronic inflammatory condition of oral mucous membranes. Patterns of mucosal involvement in oral lichen planus include; reticular, papular, plaque-like, atrophic, and ulcerative. Prevalence of oral lichen planus in the community is 1-4%. It is a disease of middle aged people (between 30 to 70 years) and is more common to women than men [3]. Different etiologies for it include autoimmune disease, drug reaction, diabetes mellitus, hypertension, kidney stones, psychological factors, and bacterial infections [4] And several viruses, including; herpes viruses, immunodeficiency virus, papillomavirus, the hepatitis viruses B and C have also been implicated as etiological agents [5,6]. But in general, etiology of oral lichen planus is still unknown (1). In addition, an autoimmune mechanism which activated T cells directly against basal keratinocyte cells is described [7].

In addition to feeding problems in the patients, the emergence of SCC associated with HCV postulated as a possible etiological factor of oral lichen planus can also create problems specific to HCV infection in an individual patient.

Chronic hepatitis C is often asymptomatic and is usually discovered accidentally. Extrahepatic involvement includes; thyroiditis, delayed skin porphyria, cryoglobulinemia, and glomerulonephritis, especially membranoproliferative glomerulonephritis, sicca syndrome, thrombocytopenia, lichen planus [8,9], diabetes mellitus and lymphoproliferative disorders [10]. The first association between oral lichen planus and hepatitis C virus was reported in 1991 [11] and since then, several articles about the relationship between hepatitis C virus in oral lichen planus have been published [12-17]. Most cases of HCV associated with oral lichen planus have been obtained from studies in the Mediterranean area, whereas in countries like Egypt and Nigeria that have the highest prevalence of HCV, a significant difference has not been reported [13,18]. Therefore, some researchers have suggested that there cannot be explained any relationship between oral lichen planus and HCV only based on the increased incidence in the general population [19] and some workers believe that this controversy is related to different geographical areas [20]. Overall, the relationship between oral lichen planus with HCV infection still remains disputed. Recently, the emphasis on being disputed and the need for more studies based on an accurate methodology without selection bias and the possible confounding factors such as age have risen [21].This study was designed in the West region of Iran considering the importance of relationship between hepatitis C viruses in oral lichen planus .

Methods

This prospective cross-sectional study was conducted on all patients enrolled with a diagnosis of mucosal lichen planus within 18 months from the start of the study who were referred to the department of dermatology or Farshchian hospital (Skin Center in Hamedan province). Five mililiter of blood clots were taken from each patient, and was saved at a temperature of minus 70 degrees Celsius after centrifugation till anti-HCV testing (DIA-PRO kit Italy)was done. Study design was such that if the anti-HCV was positive, two mililiter of blood clots would be taken from a patient in sterile conditions in a special tube for HCV-Rt-PCR test (Sinazhen kit Iran). Controls were randomly selected from the general population, and wereage and gender matched. An inclusion criterion was developing oral lichen planus, which was diagnosed by a dermatologist and defined according to pathology.

Exclusion criteria were lack of consent to participate in the study, and a previous history of hepatitis C in the control group.

The data collection tool for demographic and clinical profile of patients was a questionnaire. Finally, the data were analyzed by SPSS software.

Results

30 patients with oral lichen planus and 60 healthy controls enrolled in the study. Number of patients with oral lichen planus in men and women were 13 (43.3%) and 17 (56.7%), and number of healthy men, and women were 26 (43.3%) and 34 (56.7%), respectively. The mean age of patients with oral lichen planus was 46±13.7 years (range 22-80 years), and control was 46±14 years (range 22-80 years) old. Between the two groups in terms of sex and age, there was no significant difference. Among the oral lesions, 86.7% cases had bilateral involvement. Most patients [29 (96.7%)] had buccal involvement, followed by lip, so that six patients (20%) had involvement in this area. Palatal involvement in a patient (3 / 3%) had the lowest rate (Tab. 1).

Involvement area	Number	Percentage
Buccal	29	96.7
Lip	6	20
Gum	4	13.3
tongue	5	16.7
Palate	1	3.3
Oral floor	4	13.3

Table 1. Frequency of involvement area in patients with oral lichen planus

In most patients who were enrolled in the study, clinical forms of oral lesions, was erosive ulcerative [18 patients (60%)]. And the lowest form of involvement was plaquelike with one case (3.3%) (Tab. 2).

Clinical form	Number	Percentage
Atrophic	3	10
Ulcerative (erosive)	18	60
reticular	13	43.3
plaque-like	1	3.3

Table 2. Frequency of clinical forms of oral lesions in patients with oral lichen planus

Most patients had no clinical symptoms [21 (70%)] and only nine cases (30%) had. Smoking or smoking history existed only five cases (16.7%). None of the patients with oral lichen planus were infection hepatitis C. Two patients (3.3%) in the control group had hepatitis C infection that confirmed by HCV-Rt-PCR. The difference was not significant (Tab. 3).

Study groups	anti-HCV (+) N (%)	anti-HCV (-) N (%)	χ^2	P.value*
Oral lichen planus	0(0)	30(100)	0.8	P=0.21
Healthy	2(3.3)	58(96.7)		
Total	2(2.2)	88(97.8)		

* Pearson chi-square test

Table 3. Comparison of frequency of HCV infection among patients with oral lichen planus, and healthy individuals

Discussion

In the present study, none of the patients was suffering from hepatitis C. This study showed that there is no relation between oral lichen planus and hepatitis C infection in the region of Hamadan (West of Iran). Furthermore, in the present study, confounding factors that could affect the outcome of this study such as age [21] and sex also were eliminated. The lack of association has been reported in several other studies [13,18,23,22].

Most cases of oral lichen planus associated with HCV have been reported from studies in Mediterranean area.

In countries that have the highest prevalence of HCV (such as Egypt and Nigeria), significant difference in oral lichen planus associated with HCV has not been reported in the case group when compared with the control group [13,18].

Although two patients in the control group were positive for hepatitis C which was confirmed by PCR, the two had no clinical manifestations of disease and were found accidentally. Like this study, it has been reported in literature that chronic hepatitis C is often asymptomatic and is often discovered accidentally [8-10]. Statistical analysis of differences in hepatitis C infection in two groups was not significant. Although reports about the association between oral lichen planus and hepatitis C virus infection is contradictory and controversial, and given that a number of researchers said this association depend on geographical areas [20], this study is of the opinion that in Hamedan province (West of Iran) there is no association between them.

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RATIONAL USE OF FLUCONAZOLE PRIOR TO ATTENDING SKIN & VD-OPD IN A TERTIARY MEDICAL COLLEGE HOSPITAL IN BANGLADESH

RACJONALNE WYKORZYSTANIE FLUKONAZOLU PRZEZ PROWADZĄCYCH ODDZIAŁ DERMATO-WENEROLOGICZNY W TERTIARY MEDICAL COLLEGE HOSPITAL W BANGLADESZU

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Conflicts of interest: None

Abstract
This study was done keeping the hypothesis in mind that Fluconazole is used irrationally irrespective of diagnosis. **Material:** All patients attending at Skin-VD OPD of Enam Medical College Hospital during a 6-month time-period (July-Dec, 2009) were considered for this research. Structured questionnaire, check-list and face-to-face interview were used as data collection tools. After careful analysis, 274 cases were found valid out of 976 respondents. The cases were mostly adult (>20 yrs., 70.4%), dominated by male (58%), marriage (54%), literacy (71.6%) and coming from far (>5 km; 65.9%). The referral was made by registered doctors (10.9%), village doctors and/or drug-sellers (50.3%) and self (38.8%). Out of the total Fluconazole-intakers (N=119), it was found that correct prescription done by registered doctors (10.08%), village doctors and/or drug sellers (9.24%) and by self (0.8%) was very few. The respondents wrongly-taken Fluconazole were finally diagnosed as case of psoriasis (21.84%), atopic dermatitis (13.44%) sebrrhoic dermatitis (12.6%) and so on. **Results:** The findings put this recommendation that prior to confirm diagnosis, use of Fluconazole was not rational for generalized skin lessions.

Streszczenie
Badania przeprowadzono stawiając hipotezę, iż Flukonazol jest używany irracjonalnie, niezależnie od diagnozy. **Materiały**: wszyscy pacjenci prowadzeni byli przez lekarzy z Oddziału skórno-wenerologicznego z Enam Medical College Hospital w okresie sześciu miesięcy (lipiec-grudzień 2009). Skonstruowany dokładny kwestionariusz, lista kontrolna i osobisty wywiad zostały użyte jako narzędzia do zbierania danych. Po szczegółowej analizie wyodrębniono 274 z pośród 976 respondentów. Osoby były głownie dorosłe (> 20 lat, 70,4%), przeważali mężczyźni (58%), małżeństwa (54%), piśmienne (71,6%) i pochodzące z daleka (5> km; 65,9%). Skierowania były wystawione przez: zarejestrowanych lekarzy (10,9%), lekarzy praktykujących na wsi i / lub sprzedawców leków (50,3%) oraz osobiście (38,8%). Z ogólnej liczby osób stosujących flukonazol (N = 119) stwierdzono, że jego wypisanie na recepcie było uzasadnione tylko w niewielkim odsetku przez: zarejestrowanych lekarzy (10,08%), lekarze ze wsi i / lub sprzedawców leków (9,24%) a osobiście (0,8%). U respondentów u których niewłaściwie stosowano Flukonazol ostatecznie rozpoznano: łuszczycę – (w 21,84% przypadków), atopowe zapalenie skóry (13,44%) łojotokowe zapalenie skóry (12,6%). **Wyniki:** Wyniki realizacji niniejszego badania pokazały, ze wykorzystanie Flukonazolu do leczenia ogólnych zmian skórnych przed postawieniem diagnozy nie było racjonalne.

Key words: anti fungal drug; rational use of Fluconazole; malpractice; Fluconazole
Słowa klucze: leki przeciwgrzybicze; racjonalne wykorzystanie Flukonazolu; nadużywanie; Fluconazol

Introduction

Bangladesh is a densely populated country with 150 million people in a 144 thousand sq.km. pocket [1]. We are placed in the tropical region with endemic prevalence of communicable [2] diseases, notably over burdened with non-communicable and contagious diseases too. If we sorting out the disease profile [3] of the country, we see, almost 19% of total OPD patients are suffering from skin diseases. As a non-specialized management some common drugs such as Fluconazole, NSAIDs, citrizine, H_2 blockers, prednisolone are used country wide. This study was done to explore the use and

its magnitude and correlations of the commonest anti-fungal drug – Fluconazole [4]. The study period was done in Skin & VD OPD at Enam Medical College and Hospital (EMCH), Savar, Dhaka during July to December 2009. The researchers were intended to assess the prescriber, the diagnosis pattern and its rationality aiming to make some recommendations those can guide and ensure more precise and indicative use of Fluconazole [5].

Objectives:

1. To quantify the patient come after taking Fluconazole.
2. To distinguish the prescriber of Fluconazole by their status.
3. To correlate the use of Fluconazole with its indication.
4. To clarify the prescriber who suggested Fluconazole correctly.
5. To identify the irrational use of Fluconazole.

Methods

This prospective study was carried out in the department of Dermatology and Venereology, at Enam Medical College & Hospital (EMCH), Savar, Dhaka over a period of 6 months, July-December 2009. All patients (976) attending Skin & VD OPD at EMCH were considered with exclusion of those, who (319) came for direct consultation, such as medical related persons (doctors, nurses, medical students, medical staffs and their families); and rest (383), who's treatment had no relationship with Fluconazole (such as erectile dysfunction, anxiety neurosis, STI patients). The ultimate sample size was 274 (valid). Data were collected by structured questionnaire, face to face interview and checklists. Answers were tabulated and checked manually forming table and master-table and analyzed in PC using SPSS version 10.1.

Results and Findings

Demographic Variables		
Age	<20 Yrs. 81 (29.6%)	>20 Yrs. 193 (70.4%)
Sex	Male 159 (58%)	Female 115 (42%)
Marital Status	Married 148 (54%)	Unmarried 126 (46%)
Literacy	Illiterate 78 (28.4%)	Literate 196 (71.6%)
Location	Within Savar 111 (41%)	Outside Savar 163 (59%)
Distance	<5 Km. 34.1	>5 Km. 65-9%

Table 1. The basic demographic information of the respondents by selected variables (N=274)

Valid	Frequency	Percent	Cumulative percent
Registered doctor	30	10.9	10.9
Drug seller/ village doctor	138	50.3	61.2
Self	106	38.8	100
Total	274	100.0	

Table 2. Distribution of patients by the category of preliminary-prescriber (N=274)

	Frequency	Percent	Cumulative percent
Yes	119	43.4	43.4
No	155	56.6	100
Total	274	100	

Table 3. History of taking Fluconazole prior to attend EMCH Skin-VD OPD (N=274)

	Frequency	Percent	Cumulative percent
Yes	24	20.2	20.2
No	95	79.8	100
Total	119	100	

Table 4. History of taking Fluconazole with or without indication (N=119)

Table 1 shows age distribution of the respondents that majority of the cases (65.4%) was in the age group of 21-50 years. 29.6% of patients were shown in lesser age group (0-20 years). Sex distribution showed that majority patients were male(58%) and married (54%). Most of them were literate (71.6%) and attending EMCH from outside Savar or far away (59%) (>5 km; 65.9%). (Please note that, Saver municipality is a peri-urban area where most people use Rickshaw (tri-cycle) as their usual transport and 5 km is not very easy by cost or comfort).

Out of 274 patients, only 10.9% referred by registered doctor but maximum were sent by unregistered-chemist or village doctor (50.3%). A significant potion (38.8%) came to OPD directly (Tab.2).

Table 3 shows that about half of the patient came after taking Fluconazole (43.4%) but the rest came without taking Fluconazole (56.6%).

Table 4 shows that majority of the patient came after Fluconazole-taking without specific indication (79.8%) but a few respondents had indication of Fluconazole (20.2%).

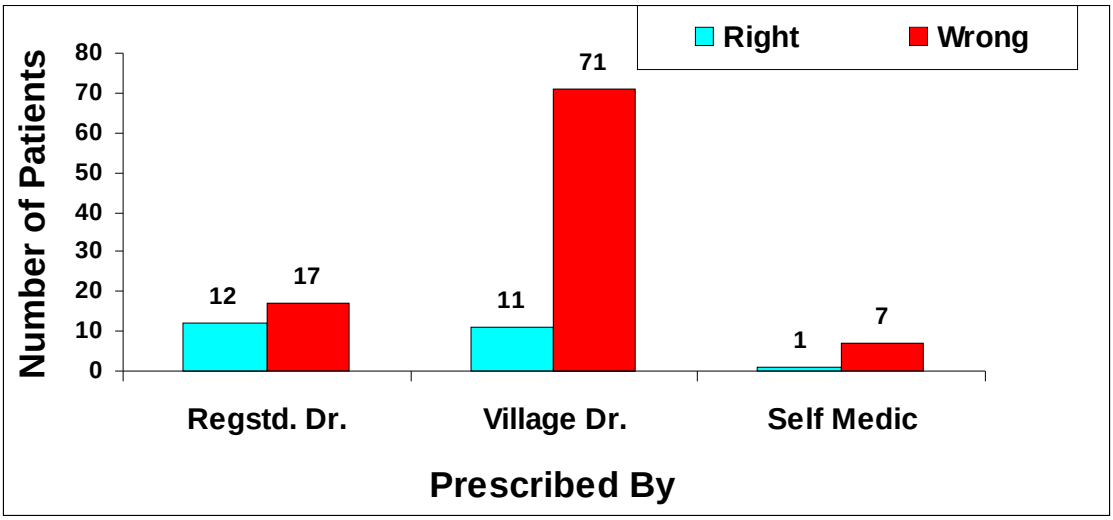

Figure 1. Distribution of the respondents having Fluconazole by correctness and Prescriber-type (N=119)

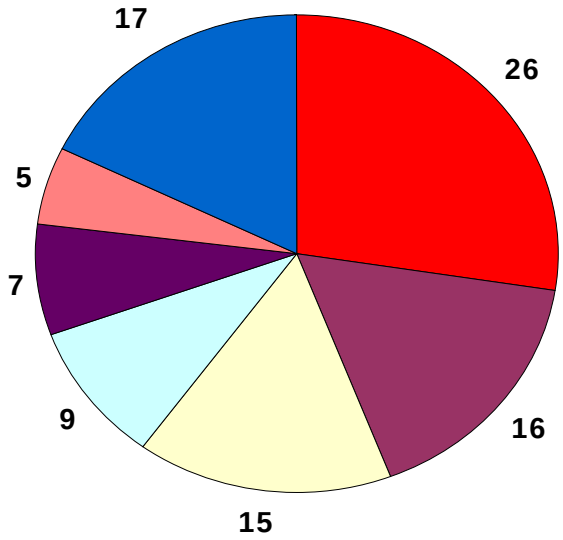

Figure 2. Distribution of the respondents having given Fluconazole, who were finally diagnosed different (N=95)

Figure 1: This Bar chart shows that out of the 119 patients (43.4%)- who had taken Fluconazole before attending OPD, 29 of them (10.78%) were prescribed Fluconazole by registered doctors. However 17 of these prescriptions were not indicative. Whereas the prescription given by the unregistered-chemist or village doctors (quack) were 82 of whom 11 (9.2%) were correctly prescribed and 71 (59.66%) were wrong. Among the self medicated respondents only 1 (0.8%) had correctly prescribed but others were wrong (8; 6.72%).

Figure 2: This pie shown 95 (79.8%) respondents were given Fluconazole as a drug of choice which was done callously. Even psoriasis (21.8%), atopic dermatitis (13.4%), seborrheic dermatitis (12.6%) and the eczema (7.5%) were not diagnosed well beforehand, but treatment started with Fluconazole.

Discussion

This study found that out of the total Fluconazole-intaker (119) only 24 (20.1%) were chosen rightly. In contrast, the study revealed that majority of the patients is treated by Fluconazole which was vague and had determined no clear indication. Indira et al [6] found in their study that out of 876 patients attending OPDs, about 72% were wrongly treated with Fluconazole, which is very close to our findings.

This study also observed that patients were prescribed Fluconazole wrongly even by the registered doctors (58.6% proportional). This happened due to: (1) the prescriber were not very careful about diagnosis (57%); (2) insufficient knowledge on skin- lesion (because, before July 2008, Skin VD was not included into the final professional MBBS examination) [7] (68.3%); (3) any skin manifestation in the tropical zones may be misdiagnosed as fungal disease [8]. (76%). In contrast, Umit N Gundogmus et al [9] from Kocaceli University Medical School, Turkey found causes behind the irrational use of Fluconazole: (1) Careless (53.4%); (2) Less knowledge (20%); (3) Random use (51.6%). But, Indira et al found irrational use of Fluconazole in a different ways 66% in OPD and 41% in IPD among 701 patients.

The very significant observation of the study with those patients- who were treated by Fluconazole thinking the skin lesion as a symptoms of fungal infections, whereas the final diagnosis were too far to assumption, such as– (1) Psoriasis (26, 21.8%); (2) Atopic dermatitis (16, 13.4%); (3) Seborrheic dermatitis (15, 12.6%) and so on. Sommer et al [10], found that the chances of fixed drug eruption, liver toxicity, respiratory distress etc. might happen due to irrational use of Fluconazole, steroid and NSAIDs in 9182 patients in central and northern India in early years of this millennium.

Recommendations

This small scale study revealed that Fluconazole is still prescribed for most of the skin lesion, irrespective of indications. About 80% cases of skin-VD were wrongly treated with Fluconazole. Out of this, registered physicians did wrong 58.6% (proportional), which is very alarming. In contrast to above findings, the study shown that 68.9% patients were prescribed or dispensed Fluconazole by the drug seller or village-doctors (quack). The proportion of this wrong prescription was about 87% - that might be destructive to public health in every consideration.

Basis on the findings, the researchers put forward following recommendations to higher authorities, related professionals and the general practitioners:

1. Common anti-fungal drugs, like Fluconazole, should be chosen after careful analysis or confirmatory diagnosis.
2. All Skin lesions should not be treated with Fluconazole by quick assessment in OPD/clinics considering as simple fungal case.
3. Registered general practitioners should have to undergo short courses or refreshers trainings on Skin-VD for upgrading and updating knowledge and skill.
4. The patients complaining no-prognosis with Fluconazole- must refer to Skin-VD specialties for ethical management.
5. Large scale study is strongly recommended in this field to explore more knots and dots.

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SKIN CANCER KNOWLEDGE, ATTITUDE AND BEHAVIOR TOWARDS SUN EXPOSURE AMONG YOUNG ADULTS IN LITHUANIA

WIEDZA NA TEMAT RAKA SKÓRY, POSTAW I ZACHOWAŃ WOBEC EKSPOZYCJI NA SŁOŃCE WŚRÓD MŁODYCH OSÓB DOROSŁYCH NA LITWIE

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Abstract
Objective: The aim of the study was to assess young adult's knowledge about skin cancer and it's risk factors, attitude towards sun exposure and the interactions of various behaviors in the sun.
Material and methods: The anonymous questionnaire-based inquiry of 750 respondents was created according to the anonymous form filled-in during the annual Euromelanoma Day campaign.
Results: 708 questionnaires were filled-in correctly: 328 (46.3%) by men, 380 (53.7%) by women. Median of age was 21 (women – 22, men – 21). During the sunny days 93.2% of respondents sometimes seek shade, 17.5% of young adults never try to get a tan from 11a.m. to 3p.m. Sunglasses are worn in 52.4% of cases, however 63.1% of them with UV filters. 8,1% of respondents always wear T-shirts, 30.6% cover head in the beach. While sun-bathing one third (32.9%) wear sun protection cream, while working or doing sports outdoors – 8.9%. Majority (57.4%) apply sun protection cream when coming to the beach, 31.4% of them don't use it repeatedly. Those who knew, what is melanoma, were more likely to wear sunglasses (p=0.003) with UV filters (p=0.006), T-shirts (p=0.046), covered head (p<0.0001) and seeked shadow (p=0.002) on the beach; used sun protection cream while working ≥1 hour outdoors (p=0.001) or sunbathing (p<0.0001), and choosed a sun protection cream according to SPF value (p<0.0001).
Conclusion: The data of this study showed that respondents behave careless in the sun. One third of respondents always wear sun protection cream, the majority do not know how to use it properly. More responsible behavior in the sun depends on better knowledge of skin cancer.

Streszczenie
Cel: Celem pracy była ocena wiedzy młodych dorosłych na temat raka skóry i czynników ryzyka, w stosunku do ekspozycji na słońce i interakcji na różne zachowania na słońcu.
Materiał i metody: Stworzono anonimowy kwestionariusz badania oparty na 750 respondentach, którzy anonimowo wypełniali formularz w ciągu całorocznej kampanii Euromelanoma Day.
Wyniki: 708 ankiet wypełniono prawidłowo: 328 (46,3%) przez mężczyzn, 380 (53,7%) przez kobiety. Mediana wieku wynosiła 21 lat (kobiety - 22, mężczyźni - 21). Podczas słonecznych dni w 93,2% respondenci czasami szukali cienia, 17,5% młodych ludzi nigdy nie starało się uzyskać opalenizny między godziną 11.00, a godziną 15.00. Okulary noszone były w 52,4% przypadków, jednak 63,1% z nich posiadały filtry UV. 8,1% respondentów zawsze nosiło T-shirty, a 30,6% nakrycia głowy na plaży. Natomiast jedna trzecia (32,9%) zużywała krem ochronny przed słońcem, a w czasie pracy lub uprawiania sportu na świeżym powietrzu - 8,9%. Większość (57,4%) stosuje krem przeciwsłoneczny, na plaży, 31,4% z nich nie używa go wielokrotnie. Ci, którzy wiedzieli, co to jest czerniak, byli bardziej skłonni nosić okulary (p = 0,003) z filtrami UV (p = 0,006), T-shirty (p = 0,046), nakrycia głowy (p <0,0001) i szukali cienia (p = 0,002) na plaży, używali krem ochronny przed słońcem podczas pracy ≥ 1 godziny na wolnym powietrzu (p = 0,001) lub opalania (p <0,0001) i wybierali krem ochrony przeciwsłonecznej SPF według wartości (p <0,0001).
Wnioski: Dane z badania wskazują, że respondenci zachowywali się nieostrożnie na słońcu. Jedna trzecia respondentów zawsze nakładała krem ochronny przed słońcem, jednak większość nie wie jak go używać prawidłowo. Bardziej odpowiedzialne zachowanie się na słońcu zależy od lepszej wiedzy na temat raka skóry.

Key words: skin cancer; melanoma; sun protection; health education; Euromelanoma Day
Słowa klucze: rak skóry; czerniak; ochrona przed słońcem; edukacja zdrowotna; Euromelanoma Day

Introduction

Over the past few decades the incidence of skin cancer has been rising in Lithuania at an alarming rate. Skin cancer is the most common type of cancer in white population across the globe [1], whereas a non-melanoma skin cancer (NMSC) – most expensive to treat [2]. In 2009, 2272 new cases of skin cancer and 269 of melanoma were registered in Lithuania, while the number of such cases in 1992 were only 938 and 147 respectively [3]. The increase is most likely a result of several factors: depletion of the protective ozone shield due to climate change [4] and people's careless behavior in the sun [5]. Moreover, in the nearest future in Lithuania the incidence of skin cancer might increase even more because of the change in sunbathing habits, e.g. increased frequency of vacations in sunny resorts. It must be acknowledged that the growth in recorded numbers may also be due to better detection methods as well as campaigns for skin cancer prevention, such as annual "Euromelanoma Day" campaign in Lithuania.

Melanoma is considered to be the most serious form of skin cancer due to it's rapid metastasis and a rising morbidity among younger people [6]. This cancer is completely curable if detected at the early stage of disease, unfortunately it is fatal if allowed to progress and spread. The key risk factor for melanoma and NMSC is ultraviolet sun radiation [7]. The majority of lifetime UV exposure is received before the 18 years of age [8].

Extended sun exposure during childhood increase the probability of skin cancer in adulthood [9]. For instance, more than 1 severe sunburn in childhood is associated with a two-fold increase in melanoma risk [10,11]. A higher risk of developing skin melanoma is indicated in fair-haired, blue-eyed, freckled and prone to severe sunburns people, as well as in those who are living closer to the equator, who have experienced severe sunburns once or several times, and in those who are spending their holidays in hot climate zones [12].

Primary prevention through identification of people at high risk [13], as well as by health education programs aiming to modify behavior in the sun and to promote protective measures, is a long-term approach to avoiding skin cancer in the future [14].

The aim of this study was to ascertain and evaluate knowledge and attitudes of Lithuanian young adult's regarding skin cancer, sun exposure and the interactions of their various behaviors in the sun.

Material and methods

The method employed was anonymous questionnaire-based inquiry. The questionnaire, consisting of 47 questions, was created according to the anonymous form filled in during annual Euromelanoma Day campaign. A pilot study phase enabled us to make possible choices more relevant and appropriate. Exclusion criteria used in this study were: respondents younger than 18 and older than 75; mental or movement disorder that would impair the ability to understand and complete the questionnaire. The following demographical data was asked: gender, age, education and profession. The respondents were asked to evaluate their skin type (Fitzpatrick classification) [15]. In order to check basic knowledge of skin cancer and sun exposure we asked the participants what is melanoma, ABCD criteria, SPF letters on cosmetics, whether skin cancer is contagious and which factors increase the probability of it. The opinion if tanned people are more beautiful and healthier, as well as if respondents pay enough attention to sun protection was also asked. We found out such peculiarities of respondents behavior in the sun: if they wear sun glasses (with or without UV filter) during a sunny day, seek shade in order to avoid direct sun rays, avoid sunbathing from 11 a.m. until 3 p.m., wear T-shirts and head covers (hats, caps, kerchiefs or wide-brimmed sunbonnets) in the beach. We also ascertained the duration of time spent outside on weekdays and weekends as well as whether they experienced sunburns to blisters under 18 years old.

Study included 708 respondents: students of Vilnius University, Vilnius Gediminas Technical University, and Vilnius Pedagogical University, pupils of Vilnius Lyceum, Vilnius High School of Salomeja Neris, Vilnius Jesuit and Garliava Jonuciai High Schools as well as randomly selected 18-75 years old people. The statistical analysis was performed using SPSS 17.00. Distribution of bias was graphically assessed. Analysis of data was performed using X^2 test or Mann-Whitney test. A p value of <0.05 was considered statistically significant.

Results

708 (94.4%) questionnaires out of 750 were filled in correctly and included into data analysis. 328 (46.3%) respondents were men and 380 (53.7%) women. The age of participants ranged between 18 and 75 years, vast majority of them (87.4% (619/708)) were younger than 30 years old. Median of age was 21 (for women – 22 and men – 21). 71.3% (505/708) of participants were either pupils or students. Majority of respondents (80.5% (570/708)) attributed themselves to II or III skin type according to Fitzpatrick scale (Fig. 1).

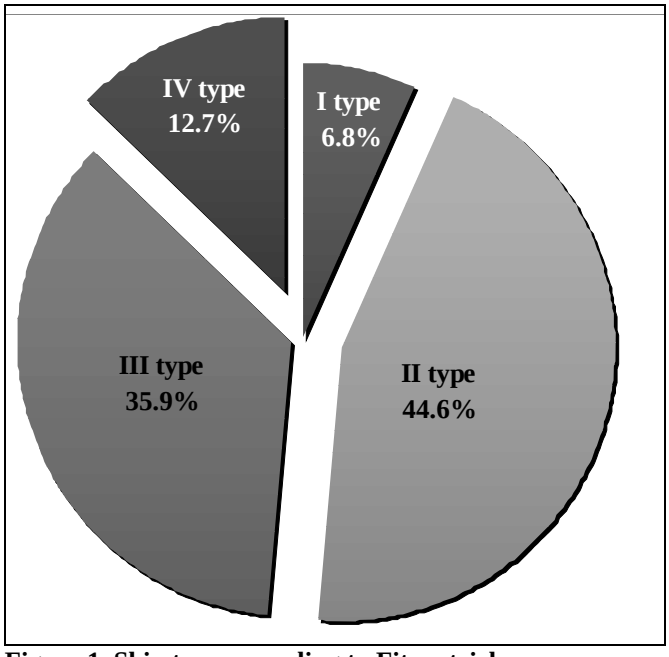

Figure 1. Skin type according to Fitzpatrick

Overall, 45.3% (321/708) of respondents did not know that melanoma is a skin cancer. 83.3% (583/708) did not consider that ABCD criteria define appearance of the atypical mole. Less than half of respondents (46.9% (332/708)) explained that SPF letters on cosmetics is abbreviation of "sun protection factor", only 7.5% (53/708) correctly converted sun protection cream's with SPF30 duration of protection from harmful sun's rays. Only 3.2% (23/708) correctly named all mentioned skin cancer risk factors (Tabl. 1), 98.6% (698/708) of them knew that skin cancer is not contagious.

The number of females, who answered that melanoma is a type of skin cancer, was significantly greater than men: 70.3% (267/380) and 36.6% (120/328) respectively ($p<0.0001$). Women significantly more frequently correctly answered that skin cancer is not contagious 94.2% (358/380) compared to men 83.2% (273/328) ($p<0.0001$). Females also more often named all skin cancer risk factors (females 5.0% (19/380) vs. males 1. 2% (4/328)) ($p=0.005$). Males better answered the question, what ABCD criteria define, (female 76.3% (290/380) vs. male 88.1% (289/328) ($p<0.0001$) and counted sun protection cream's with SPF30 duration of protection from harmful sun's rays (female 50.0% (190/380) vs. male 74.4% (244/328) ($p<0.0001$)). The number of respondents, who were ≥30 years old more often correctly named all risk factors of skin cancer (7.9% (7/89)) than people who were 18-29 years old (2.6% (16/619) ($p<0.0001$)). Respondents, who correctly answered what is melanoma, had significantly higher education degree (graduated/still studying at university) 59.9% (224/374) compared to the people who graduated/still learning at high school 48.6% (161/333) ($p=0.003$). Furthermore, people with higher education claimed more often that skin cancer is not a contagious disease (respectively 93.1% (350/376) vs. 84.6% (281/332) ($p<0.0001$)) and correctly answered what is SPF (respectively 96.5% (363/376) vs. 93.1% (309/332) ($p=0.036$)). No other significant differences were noticed among demographical data, skin type and knowledge about skin cancer and sun exposure. Respondents, considering that tanned people are more beautiful, think more frequently that tanning is healthy compared to people who believe that suntan is not related to beauty standards: 31.0% (149/480) and 8.0% (7/87) respectively ($p<0.0001$) (Fig. 2).

Almost half (47.1% (263/708)) of respondents have been sunburned to blisters at least once before they reached 18 years old. Majority of respondents usually spend ≥1 hour in the sun in a typical weekday 66.4% (470/708), whereas on weekend this number reached 85.3% (604/708). 55.9% (396/708) of respondents admited not paying enough attention to skin cancer prevention. During the sunny days 84.0% (595/708) of respondents sometimes and 9.2% (65/708) of respondents always seek shade and only 17.5% (124/708) never try to get a suntan from 11 a.m. to 3 p.m. Sunglasses are worn in 52.4% (371/708) of cases, however, only 63.1% of them (234/371) have UV filters. Only 8.1% (57/708) of respondents always wear T-shirts and about one third of them (30.6% (217/708)) cover head on the beach. While sunbathing 32.9% (233/708) wear sunscreen, while working or doing sports outdoors – only 8.9% (63/708). 33.3% (172/516) use sunscreen with SPF 12-30. Majority (57.94% (296/516)) of respondents apply sunscreen when coming to the beach, 31.4% (162/516) of them don't use it repeatedly. Respondents, who correctly answered what is melanoma, were more likely to wear sunglasses ($p=0.003$) with UV filters ($p=0.006$), to wear T-shirts ($p=0.046$), to cover their head ($p<0.0001$) and to seek shadow ($p=0.002$) on the beach, to use sunscreen while working ≥1 hour outdoors ($p=0.001$) or sunbathing, and to choose sunscreen according to SPF value ($p<0.0001$) (Fig. 3) (Tabl. 2).

No	Knowledge statements	Correct answer	No (%) (n = 708)
1	Light skin	True	266 (37.6)
2	Presence of family members skin cancer	True	425 (60.0)
3	Fungal disease of face skin	False	49 (6.9)
4	>50 body skin moles	True	343 (48.4)
5	Acne during adolescence	False	20 (2.8)
6	Moles and freckles	True	244 (34.5)
7	Severe skin sunburn during childhood	True	204 (28.8)
8	Long duration sunbathing	True	564 (79.7)
9	Frequent visits of tanning salons		595 (84.0)
	Overall, correctly answered		23 (3.2)

Table 1. Knowledge regarding risk factors of skin cancer

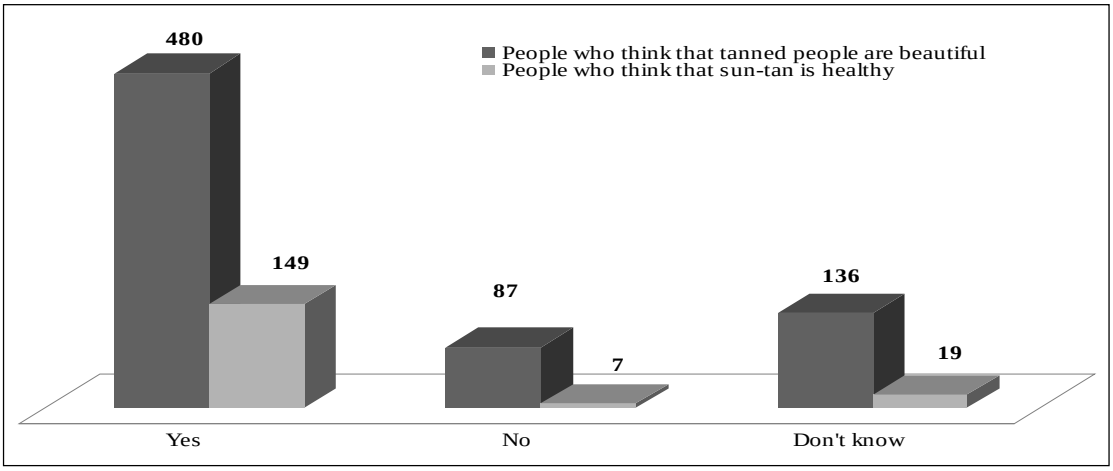

Figure 2. Respondents, who consider that tanned body is a beauty standard, statistically more often think that it is also healthy (31.0% (149/480) vs. 8.0% (7/87) (p<0.0001)

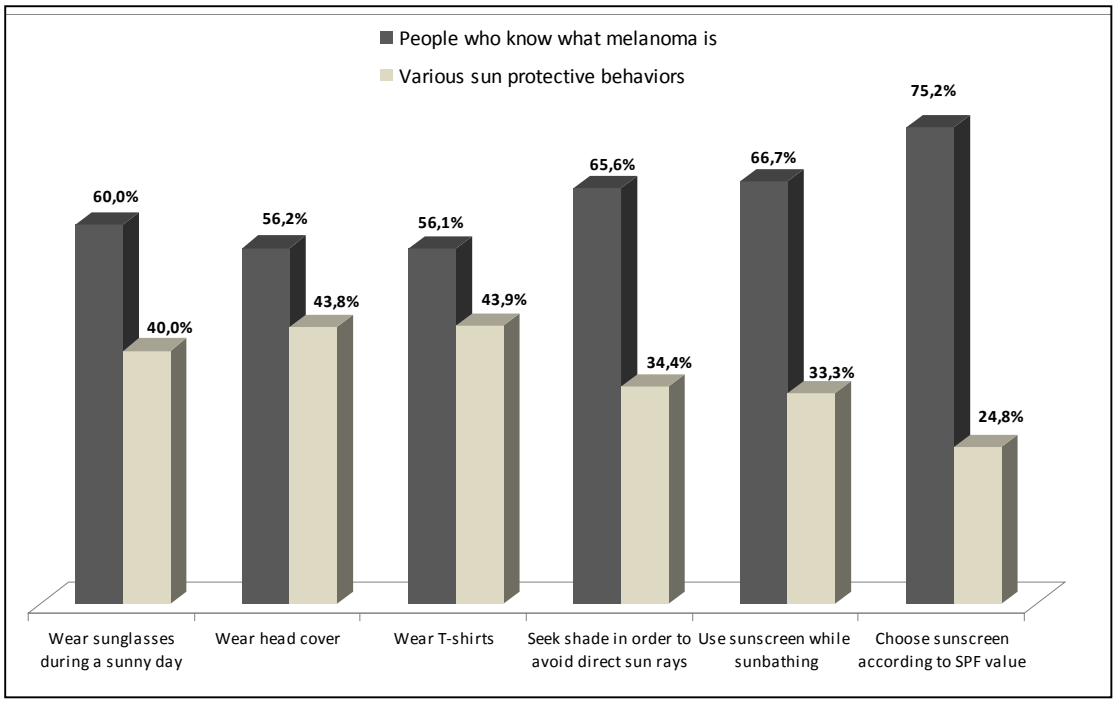

Figure 3. Respondents, who correctly answered what is melanoma, were more likely to wear sunglasses (p=0.003) and T-shirt (p=0.046), to cover head (p<0.0001) and seek shadow (p=0.002) on the beach, to use sunscreen while sunbathing, to choose sunscreen according to SPF value (p<0.0001)

When you go to the beach do you use sun protection cream?	**No (%) (n = 708)**
1. Never	192 (27.1)
2. Seldom	283 (40.0)
3. Always	233 (32.9)
When do you apply sun protection cream?	**No (%) (n = 516)**
1. Half an hour before going outside	137 (26.6)
2. When I go outside	41 (7.9)
3. On the beach	296 (57.4)
4. When I get suntan or sunburn	42 (8.1)
Do you repeatedly apply sun protection cream?	**No (%) (n = 516)**
1. No	162 (31.4)
2. Yes, once in 2 hours	279 (54.1)
3. Yes, once in 4 hours	75 (14.5)

Table 2. Peculiarities of sun protection cream usage

Discussion

The results of the performed study showed that careless behavior, such as staying on the beach between 11 a.m. and 3 p.m. (the dangerous time period for exposure to the highest UVR intensity) without sun-protective clothing was very common. With regard to sun protection cream and it's usage, the situation was terrifying as the vast majority of present study respondents used sunscreen infrequently, inconstantly and incorrectly. The main reason for such behavior might be insufficient knowledge concerning sun's effect on health and the measures which help to protect the skin from harmful sun exposure. This should be a matter of great concern.

Furthermore, 82.4% of respondents were not aware of a simple self-examination ABCD rule for atypical mole and melanoma. Self-examination of moles is important because a mole which is changing in size, shape or colour may develop into melanoma [16]. It is necessary to know how to distinguish mole from melanoma.

The results of this study are comparable to similar studies worldwide. We found that the respondents from Lithuania seem to be less knowledgeable as far as melanoma is concerned. While only 55% of the participants reported knowing what is melanoma, in other countries the percentage of those with this knowledge was much higher (in Australia - 66% [20], in New Zealand - 75% (21), in USA - 80% [22] (Fig. 4).

The generalization of the results also showed that in our country the frequency of sunscreen usage while being on the beach is not high (only 32.9%) compared to other countries (e.g. Israel, Australia, USA) [23-25]. For instance, the reported frequency of sunscreen usage in USA is 83.0%. It is recommended to use sunscreen in order to protect from sun exposure and to prevent sunburn [26]. The usage of sunscreen can prevent squamous cell carcinoma [27] and reduce the number of acquired nevi that are associated with sun exposure as a risk marker for melanoma [28].

Likewise, Lithuanians wear sun-protective clothing and sunglasses with UV filter disappointingly rarely. For instance, a wide-brimmed sunbonnet (a head cover, which gives the best sun protection) was worn on the beach only by 11.6% of young adults. These findings are similar to Lithuanian schoolchildren's. They wear sunbonnets in 10.8% of the cases [29].

In accordance with the results of similar studies, the findings show that a great part of population in many countries fails to avoid intensive sun exposure, and Lithuanians are not an exception. It has been determined that 49% of the present study participants experienced sunburn at least once till 18 years old. These results are similar to the sunburn incidence reported in the studies from France (46%) [30] and Greece (56%) [31]. In similar study performed in USA [32], a sunburn incidence percentage was very high - 83% (Fig. 5).

The majority of the respondents reported that a tanned body is a beauty standard (67.7%). The present findings are identical with the results of study conducted in Greece (67.7%) [31].

Young people from all over the world are likely to stay in the sun for extended periods, especially those aged 13-19 years [33]. Since it has been shown that adults have the lowest probability for change in behavior [33,34], educational efforts should focus mainly on students [25].

A review of recent activities of Lithuanian dermatologists shows that great efforts are being made to assess one's knowledge, attitude and to understand UVR and it's effects on the skin as well as to implement education programs. A health education program "Let's know the sun better" was implemented in several Kaunas city secondary schools. The evaluation of it's results proved both the efficiency and the necessity of the prepared educational program [29]. Furthermore, the annual pan-European campaign of free skin examination "Euromelanoma" was successfully organized in Lithuania for the third time. It's goal, which is consistent with WHO [35] guidelines, is to raise awareness of extended sun exposure risks and to educate the public about the protective measures [36].

On the basis of present study results, we recommend the national cancer prevention program to devote special attention to skin cancer prevention in children (through school curricula) and in the rest of the society through mass media, as it is the main source of information [35].

A few limitations of the study should be noted. A larger sample size and country-wide participation of adult respondents would provide more data about the knowledge about skin cancer and attitude towards sun exposure. The interview of other groups (e.g. younger pupils, parents and teachers) would allow the comparison of their beliefs with the present study participants.

Conclusions

The knowledge about skin cancer and harmful effects of the sun among Lithuanian young adults is insufficient. The irresponsible behavior in the sun should be a matter of great concern. Furthermore, many young Lithuanians associate suntans not only with beauty but also with health. To sum up, poor knowledge, risky behavior and a dangerous standpoint must be challenged by health education and promotion programmes in order to reduce the epidemic of skin cancer in Lithuania in the nearest future.

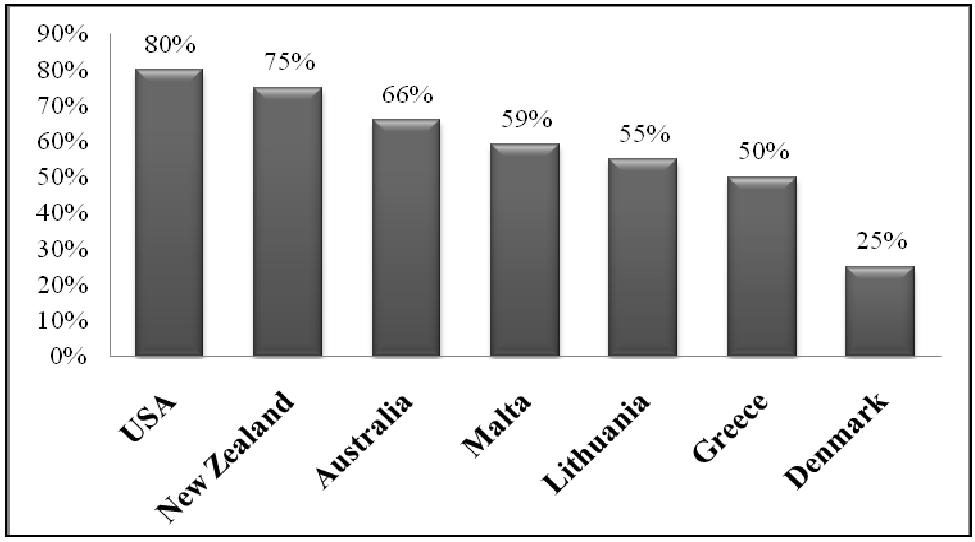

Figure 4. Knowledge of what melanoma is, according to country
***Adapted from M. Saridi et al article [31]**

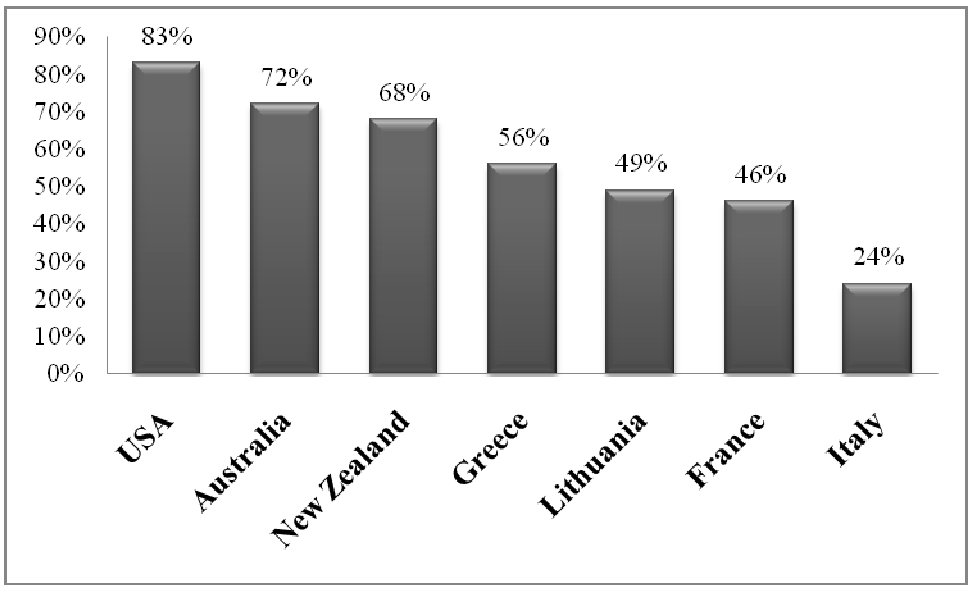

Figure 5. Sunburn incidence, according to country
***Adapted from M. Saridi et al article [31]**

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NASZA DERMATOLOGIA Online
OUR DERMATOLOGY Online

RECOGNITION OF ACTINIC KERATOSIS. A RETROSPECTIVE BIOPSY STUDY OF THE CLINICAL DIAGNOSTIC ACCURACY BY PRIMARY CARE PHYSICIANS COMPARED WITH DERMATOLOGISTS. EXPERIENCE IN MEXICO

ROZPOZNANIE ROGOWACENIA SŁONECZNEGO. RETROSPEKTYWNE BADANIE DOKŁADNOŚCI KLINICZNEJ DIAGNOSTYKI LEKARZY PODSTAWOWEJ OPIEKI ZDROWOTNEJ W PORÓWNANIU Z DERMATOLOGAMI. DOŚWIADCZENIE MEKSYKAŃSKIE

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Abstract
Background. Actinic keratoses (AK) are dysplastic keratinocytic lesions confined to the epidermis. Currently, the standard screening method for detecting AK is performed by a health professional.
Objectives. We seek to determine if were differences in diagnosis of AK by dermatologists (DL) and primary care physicians (PCP) in Mexico.
Material and Methods. The clinical diagnoses of PCP and DL were correlated with histopathologic diagnoses. In total, 285 cases were analyzed.
Results. DL diagnosed 90% (256/285) of the cases compared with 36% (102/285) of PCP (P= .001). Primary care physicians were the group with the lowest diagnostic accuracy rate.
Conclusion: Primary care physician needs to acquire sufficient knowledge of basic dermatology as well as dermatopathology. The overall accuracy of the clinical diagnosis, mainly in hyperplastic AK, depends on the clinicopathologic correlation.

Streszczenie
Wstęp. Rogowacenia słoneczne (RS) są dysplastycznymi, keratynowymi zmianami ograniczonymi do naskórka.
Obecnie, standardowe metody badań przesiewowych w kierunku wykrywania RS są wykonywane przez lekarza specjalistę.
Cel: Staraliśmy się ustalić, czy występowały różnice w diagnostyce RS przez dermatologów (DL) i lekarzy podstawowej opieki zdrowotnej (POZ) w Meksyku.
Materiał i metody. Rozpoznania kliniczne przez POZ i DL były skorelowane z rozpoznaniem histopatologicznym. W sumie analizowano 285 przypadków.
Wyniki. DL zdiagnozowali 90% (256/285) przypadków w porównaniu z 36% (102/285) POZ (P= 0,001). Lekarze podstawowej opieki zdrowotnej byli grupą o najniższej stopie dokładności diagnostycznej.
Wnioski. Lekarze podstawowej opieki zdrowotnej powinni pozyskać odpowiednią wiedzę na temat podstaw dermatologii oraz dermatopatologii. Ogólna dokładność rozpoznania klinicznego, głównie w hiperplastycznych RS, zależy od kliniczno-patologicznej korelacji.

Key words: actinic keratosis; clinicopathologic correlation; dermatologist; diagnostic accuracy; primary care physician
Słowa klucze: actinic keratosis; kliniko-patologiczna korelacja; dermatolog; dokładność diagnostyczna; lekarz podstawowej opieki zdrowotnej

Introduction

Actinic keratoses (AK) are dysplastic keratinocytic lesions confined to the epidermis, which are caused by ultraviolet radiation and are one of the most common reasons for patients to consult a dermatologist, with an estimated prevalence of 7.2

million in 1993-1994 in the United States [1] and increasing to 39.5 million in 2004. [2] Lesions are treated mainly for preventing reasons (malignancy), however AK are also treated for cosmetic and symptomatic purposes. [2,3] Currently, the standard screening method for detecting AK is performed by a health professional (DL detect 83.2% of the patients with AK). [4] Unfortunately, many medical professionals other than DL may not be specifically trained in the detection of AK. [5-8].

We were interested to determine whether there are differences in diagnosis of AK by DL and primary care physicians (PCP) in Mexico.

Methods

In this retrospective study, we retrieved and reviewed the records of skin biopsy specimens submitted to the Dermatopathology department at the Hospital General de México, from June 2006 through June 2010. We will use the term "skin biopsy" as a comprehensive designation of various techniques employed to obtain specimens, as punch and excisional biopsy methods. The histopathological diagnosis was made by 2 Mexican certified dermatopathologists and was compared with the clinical data submitted by the clinician (PCP and dermatologist). All records represent slides with hematoxyllin-eosin-stained sections derived from archival material.

Data retrieval

A total of 285 skin specimens were submitted in the examined time frame by 38 physicians (35 PCP and 3 DL). No repeat excision specimens, in which the diagnosis was known, were enrolled in this study.

Comparison between clinical and histopathological diagnoses

Using the histopathological diagnosis as the "gold standard", we recorded a clinical diagnosis as correct, if the clinician listed several alternatives (eg. squamous cell carcinoma/AK/seborrheic keratosis) and AK was confirmed histopathologically. On the other hand, if only one clinical diagnosis was listed (eg. squamous cell carcinoma) and histopathologically the lesion represented another entity (eg. AK), the clinical diagnosis was considered incorrect.

Statistic analysis

DL and PCP were compared with respect to the frequency of correct diagnoses using the χ^2 test of association. Alternatively, Fisher´s exact test was used when frequencies or group sizes made χ^2 test results questionable (expected values less than 5). Percentages reported in the text are accompanied by 95% confidence intervals with the lower and upper limits in parenthesis. *P* values less than .05 are deemed statistically significant.

Results

The distribution of all AK types and the percentages of correct clinical diagnosis are shown in table 1.

We observed that the most commonly reported type of AK was the hyperplastic type (53/285, 18%), however, several case charts were not classified as well (220/285, 77%). The biopsy method mostly preferred for AK by DL was punch biopsy technique (245/285, 86%).

Forty seven hyperplastic AK (89%) were clinically mistaken with squamous cell carcinoma by PCP, versus 12 (23%) in the dermatologist group ($p = .001$).

When analyzing all lesions combined, DL diagnosed 90% (256/285) compared with 36% (102/285) of PCP ($p = .001$). Primary care physicians were the group with the lowest diagnostic accuracy rate. Of interest was the large number of cases for which only one clinical diagnosis was provided by the clinician. Primary care physician provided only one diagnosis in 237/285 cases (83%), compared with 20% of cases by the DL (58/285) ($p = .001$).

Type of actinic keratoses	n= 285 (%)	Correct diagnoses PCP/DL (%)	*P* value
Actinic keratosis not specified.	220 (77)	94 (43)/207 (94)	0.001
Hyperplastic	53 (18)	6 (11)/41 (77)	0.001
Atrophic	5 (2)	0 (0)/3 (60)	NS
Bowenoid	3 (1)	0 (0)/2 (67)	NS
Pigmented	2 (1)	2 (100)/2 (100)	NS
Acantholytic	1(0.5)	0 (0)/0 (0)	NS
Lichenoid	1 (0.5)	0 (0)/1 (100)	NS

PCP = Primary care physician. DL = Dermatologist

NS = Non significative

Table 1. Distribution of actinic keratoses types and percentage of correct diagnoses

Discussion

Physician office visits for the diagnosis of AK and nonmelanoma skin cancer is increasing, [9-11] such tendency is probably due to the heightened public awareness of the prevalence of precancerous and cancerous skin conditions. In 1997, 60 million of 703 million physician office visits in the United States were for skin examinations. During 1993 and 1994, 13.5 million physician office visits were recorded for AK and nonmelanoma skin cancer alone. [4] While most AK are diagnosed and treated by DL, a smaller percentage of cases are diagnosed and treated by other physician groups, including PCP. [12]

In our retrospective study, we try to determine the accuracy in clinical diagnosis of AK among DL and PCP. The present investigation provides additional information of the superior diagnostic capability of DL versus PCP in the diagnosis of AK. Numerous publications have documented a considerable disparity in the clinical diagnostic accuracy of DL and nonDL for even the most common diseases. [1,4-8,10,13,14] In the current study, we compared the clinical diagnoses made on patients that came to our consultation with the histopathological diagnoses. The clinical diagnoses of a total of 285 physicians referring cases to our Dermatopathology department were evaluated.

Several previous studies reported on the accuracy of the clinical diagnosis of DL or nonDL, or both, using the histopathological diagnosis as the "gold standard". [4,5,10,14]

We found that DL diagnosed the majority of cases correctly compared with PCP. This can be explained by several ways; the most important explanation is by the different training requirements for DL, and their experience in the management of skin diseases. In a previous study, DL diagnosed 36% (97/270) of AK correctly versus 22% (2/9) of diagnoses made by nonDL. [5] In our study, PCP recognized only 36% (109/285) of all AK, compared with 90% (256/285) of DL.

A limitation of this retrospective study is the use of the clinical data from the charts of the patients and from the pathology requisition form as a surrogate for clinical diagnostic accuracy.

We conclude that PCP needs to acquire sufficient knowledge of basic dermatology as well as dermatopathology. This knowledge is a prerequisite to diagnose (clinically and histopathological) and even treat AK correctly. The overall accuracy of the clinical diagnosis, mainly in hyperplastic AK, depends on the clinicopathologic correlation. Several possible clinical options should be proposed by the clinician, in order to decrease the risk of diagnostic miscorrelation and even increase the usefulness of clinicopathological correlation. Failures in those areas can directly and negatively impact on physician care work and patient benefit.

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APPLICATION OF THE MICROSCOPIC METHOD IN CUTANEOUS LEISHMANIA DIAGNOSIS

ZASTOSOWANIE METODY MIKROSKOPOWEJ W DIAGNOSTYCE SKÓRNEJ LEISZMANIOZY

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Abstract
Introduction: Cutaneous leishmania is spreading fast.
This study aims at developing the microscopic method to achieve a full detection of all positive cases of leishmania.
Methods: 50 human cases have been studied by applying microscopic smears stained with Wright stain. Microscopic photos were taken for the presumed unfamiliar figures.
Results: Mononuclear cells with tails are present at a rate of (98%). They are associated with Leishman Donovan (LD) bodies in 50% of the cases. The polygonal figures and the spherical forms are present at the same rate (60%) and are associated with LD bodies in 24% of the cases. The small promastigote like forms are seen at a rate of (76%) and are associated with LD bodies in 26% of the cases. The giant promastigotes like forms are present in (80% of the cases) and are associated with LD bodies in 28% of the cases. Candle flame forms are present in (40% of the cases) and are associated with the LD bodies in 21% of the cases.
Discussion: It is applicable to use those discovered figures in diagnosing cutaneous leishmania.

Streszczenie
Wprowadzenie: Skórne postacie leiszmanii rozprzestrzeniają się szybko.
Badanie to ma na celu opracowanie takiej metody mikroskopowej, aby osiągnąć pełne wykrywanie wszystkich pozytywnych przypadków leiszmanii.
Metody: 50 ludzkich przypadków badano stosując mikroskopijne rozmazy barwione metodą Wrighta. Mikroskopowe zdjęcia zostały wykonane dla przypuszczalnych nowych danych.
Wyniki: Komórki jednojądrzaste z ogonami są obecne w 98%. Są one związane z ciałkami Leishmania Donovani (LD) w 50% przypadków. Wielokątne figury i kuliste formy były obecne w tym samym odsetku (60%) oraz były związane z ciałkami LD w 24% przypadków. Małe, promastigotyczno podobne formy są dostrzeżone w 76% i są związane z ciałkami LD w 26% przypadków. Gigantyczne, promastigotyczno podobne formy są obecne w 80% przypadków i są związane z ciałkami LD w 28% przypadków. Formy typu płomień świecy są obecne w 40% przypadków i są związane z ciałkami LD w 21% przypadków.
Dyskusja: Mikroskopia jest odpowiednia do zastosowania, z wykorzystaniem tych nowych, odkrytych danych w diagnostyce skórnej leiszmanii.

Key words: cutaneous leishmania; cells; flagellates
Słowa klucze: skórna leiszmania; komórki; wiciowce

Introduction

The Mediterranean area is considered one of the most common places that cutaneous leishmania inhabits [1-3]. Cutaneous leishmania was classified according to its spreading area into two classes:
Old world leishmaniasis: which includes Afghanistan, India, Pakistan and the Middle East countries including Syria and south of Turkey.
New world leishmaniasis: They inhabit South American countries [1,3].
More than two million new cases of leishmania are discovered every year [4]. In addition to that, more than 350 million individuals are prone to leishmania infection [5]. It is highly important to find a suitable, manageable with high sensitivity method for leishmania diagnosis in order to identify those new discovered cases and provide the best available treatment.
The direct microscopic method for cutaneous leishmania secretion is considered one of the simplest methods used to detect any possible infection. This method is considered as a reference method for many sources [5-7]. The microscopic method detects the presence of (LD) bodies Leishman Donovan bodies within the macrophages and multi nucleated giant cells or

Langerhan's cells in the skin lesion [8]. (LD) bodies are nothing but the amastigote forms. They appear inside the cytoplasm of those mentioned cells in a spherical or a spindle like shape containing a nucleus and having a dimension between 2-4 microns in diameter. The principle of the microscopic method depends on the appearance of these LD bodies in the intracellular space for those cells. The presence of those LD bodies will confirm the diagnosis. While absence of those bodies may exclude the diagnosis with the infection. This diagnostic method has been discovered by both the scientists (Leishman- Donovan) more than a hundred years ago. Since that discovery, no developmental work or modification has ever been made on the procedure. Though it is a simple one, it is considered less sensitive and can at best, detect about 60-70% of the infected cases. That means that over 30% of cutaneous leishmania infected cases cannot be detected by this microscopic method and are considered as falls negative.

The purpose of this study is to search the possibility of developing and improving this traditional microscopic procedure so that we are able to detect the additional 30% of the undiagnosed cases where the LD bodies do not appear in the microscopic slides.

Procedures and Methods

50 cases have been studied, 43 males and 7 females who attended the laboratory between April 2006 and July 2008. Samples were obtained from the secretions of what was previously diagnosed by consultant dermatologists as cutaneous leishmania infection. Samples were collected as follows:

1.Slit skin smears: The area was cleaned with 0.9% saline or 70% alcohol. We squeezed the edge of the lesion between thumb and forefinger and made shallow 1 mm slits through skin to dermis with a scalpel then scraped the edges to make slide smears, then made smears as thin as possible, air dried, fixed in methanol, and stained with wright stain.

2.Dermal scrapings: We Obtained 2-4 scrapings from different areas of the lesion. We scraped dermis along the necrotic lip with a scalpel, obtaining as much tissue as possible, then making thin smears on slide, air dried, fixed in methanol, and stained with Wright stain.

In the microscopic study, we looked for the unfamiliar cytomorphologies in the smears, when they repeatedly appear in the different infected lesions, that could be of a diagnostic significance. Microscopic photos for confirmation and documentation were produced for those suggested potential figures featured in each related case. A classification was later made for those different figures and cytomorphologies according to their percentage of appearance among the different smears in a related table.

Results

The attached table indicates the count and the appearance of such unfamiliar figures and their percentage of appearance in the smears. The table nr 1 indicates the association of those figures with the LD bodies (the amastigote forms) in the same smear.

From the table above, it is clear that some new, distinct, cytomorphological figures have been discovered. Those new figures were associated with the infected lesions in more than 50% of the cases. The discovered figures are much different in their morphology than the well-known blood components and the skin tissues. That includes cells like fibroblasts, which appear in normal cases. Here is a brief description for those figures while a detailed description is found in the study (The pathological features of cutaneous leishmania) [9].

Mononuclear cells with tail: The percentage of the appearance of those cells in the smears as noticed from the table above in leishmania infection is as high as (98%) figure 1. The cytoplasmic tail is elongated in some cases up to more than 20 microns in diameter (Fig. 2). Both figures 1,2 show the different cytomorphologic manifestations of those cells. It is important to emphasize the amastigote appearance in the same photos as a confirmatory clue. That proves the possibility of using those new discovered figures to elevate the sensitivity and the specificity of the traditional microscopic procedure in detecting cutaneous leishmania.

Polygons multiformies: Those morphologies are 3-8 microns in diameter. They appear with different sides looking in a way like the neutrophile lobe (Fig. 3). The association of such figures with the amastigote form (LD bodies) is in about 24% of the cases.

Spherical forms: They are spherical in shape, very dense, having no surrounding cytoplasm. The dimension of such forms is between 2-8 microns (Fig. 4). They appear in 60% of the studied cases of leishmania. They are associated with the LD bodies in 24% of the cases.

The big flagellates: They look like the small flagellates in shape but their size is huge. Theycan reach 20-30 microns in diameter (Fig. 5). They appeared in 80% of the studied cases and were associated with LD bodies in 28% of the cases studied.

The candle flame forms: They are formed of chromatin mass with a tiny tail protruding out. Their dimension is 2-4 microns (Fig. 6). They show up in the smears in 40% of the studied cases. They are associated with the LD bodies in 22% of the cases.

The small flagellates: Those small flagellates look very much in shape like the promastigote form of the leishmania (figure 7 shows the different morphologies that this flagellate microscopically appears in the smears). Small flagellates appear in 76% of the infected cases and are associated with amastigote forms in 26% of the cases.

Total count of the cases	Mononuclear cells with tail	Multi formed polygons	Spherical forms measure 2-7 microns	Small promastigote like forms <12 microns	Large promastigote like forms>14 microns	Candle flame forms
50	49	30	30	38	40	20
Percentage	98%	60%	60%	76%	80%	40%
Association with LD bodies	25/50	12/50	12/50	13/50	14/50	11/50
Percentage	50%	24%	24%	26%	28%	22%

Table 1. The count and the appearance of unfamiliar figures and their percentage of appearance in the smears. The association of those figures with the LD bodies (the amastigote forms) in the same smear

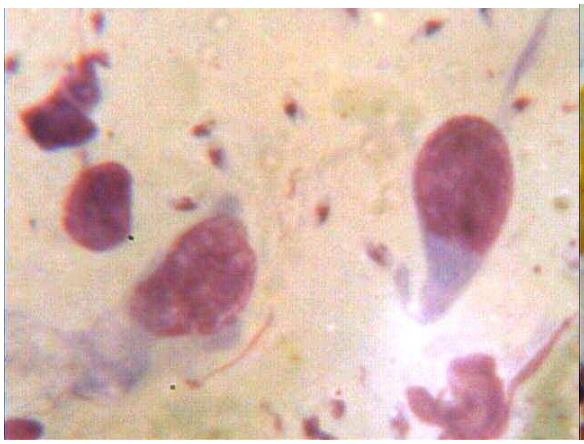

Figure 1. Mononuclear cells with tail

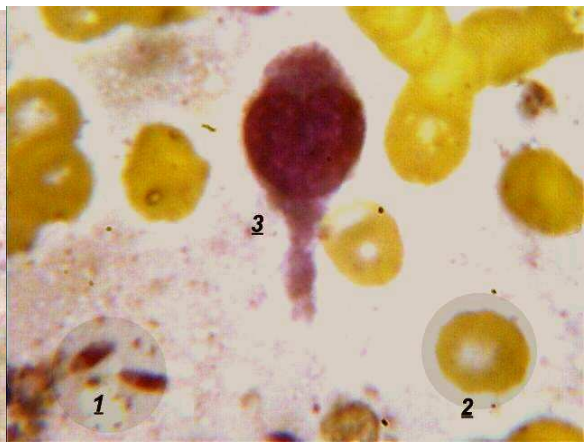

Figure 2. Mononuclear cells with tail

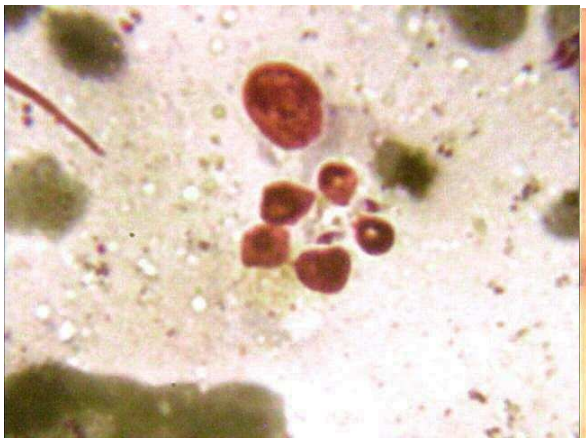

Figure 3. Polygons multiformies

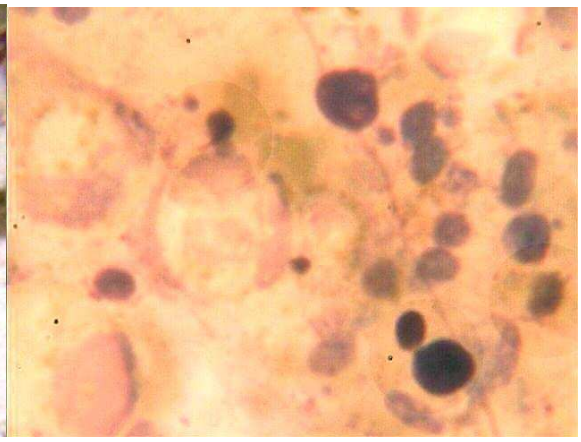

Figure 4. Spherical forms

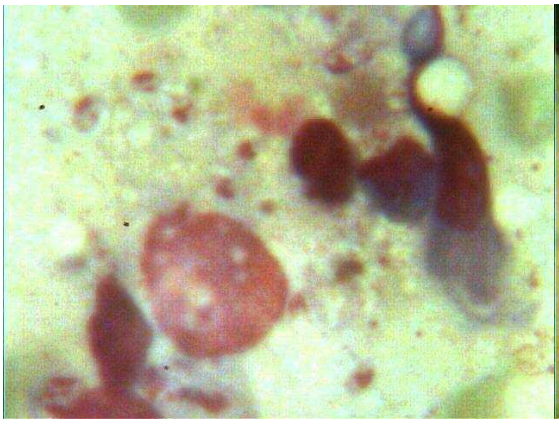

Figure 5. The big flagellates

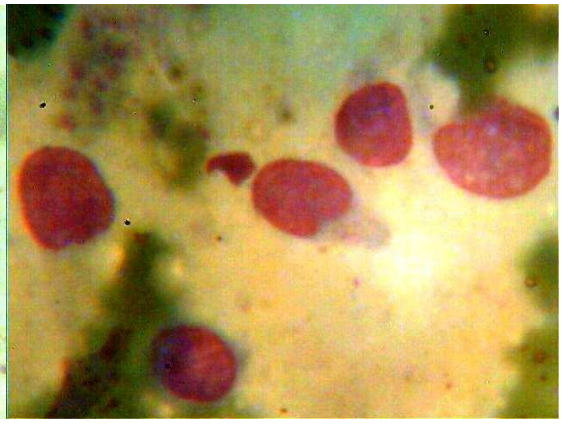

Figure 6. The candle flame forms

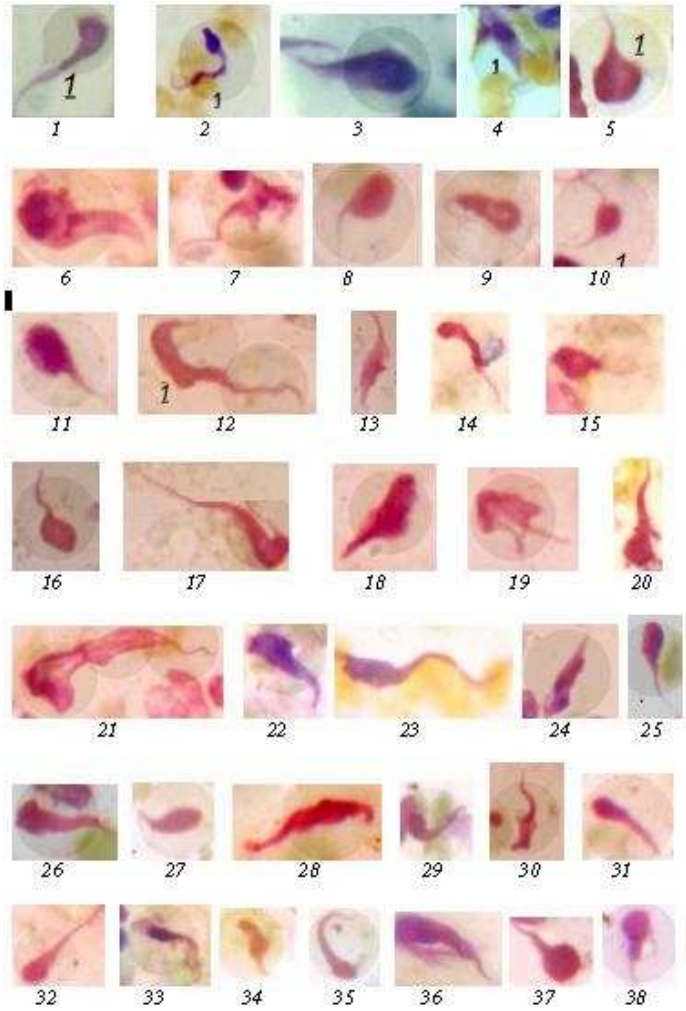

Figure 5. The small flagellates. Different morphologies

Discussion

The organisms with their different cytomorphologies presented above represent a distinguishing mark that could be utilized in diagnosing cutaneous leishmania, especially in cases where the (LD bodies) are totally absent from the microscopic smear. When applying the traditional microscopic reference method that most laboratories rely on, they only detect the LD bodies in the maximum of 70% of the infected cases. It is not yet possible to detect the disease in the other 30% of the positive cases when the LD bodies disappear from the smear. According to many studies [1-6], it seems that at a later stage of the disease process of cutaneous leishmania, the disappearance of such LD bodies together with the macrophages that are infected with, is a fact. The disappearance of such LD bodies then, cannot be explained by the decrease in the sensitivity of the microscopic procedure. It is more or less, due to a real disappearance of such amastigote form during the disease progress. Our study found that in many disease cases of leishmania, even at that stage of the disease process with the (LD bodies) disappearance, the clinical signs continue to show a sharp inflammatory reaction with a deep ulceration, and for a long period still to go [9].

The appearance of such discovered forms of organisms (the mentioned above) in the smear, and their association at different rate with LD bodies in the same smear, is a proof that those cytomorphologic figures are evidence for cutaneous leishmania. And thus, they could be utilized as a diagnostic material for detecting cutaneous leishmania especially in cases when (LD bodies) are not found in the smear . Regardless of those organisms origin, it is obvious that such organisms do not match in their morphology any of the well known cellular components or organelles that are present in the blood or the skin tissues. And because it might still be possible to find these organisms associated with other types of disease, and until we are sure that they are specific for cutaneous leishmania, it is suggested for cutaneous leishmania diagnosis, to consider, not to depend on the appearance of only one type of those organisms in the smear. When LD bodies are absent, it is more applicable to confirm the presence of at least three different types of those organisms in the same smear. In case (LD bodies) are found in the smear, no need then for looking for those organisms.

Conclusion

This study has added a good bonus for the traditional microscopic method in diagnosing cutaneous leishmania. The old method that was used to detect only the presence of LD bodies in the smear is a hundred years old method. It very much lacks sensitivity. And

with the fast spread of the disease in third world countries and with limited resources, it is hoped for an easy, non-costy, fast and reliable method for detecting cutaneous leishmania. The microscopic method when adding those discovered figures and modifications to it, is capable enough to do the job with a 100% sensitivity achievement and almost equal specificity.

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CUTANEOUS CRYPTOCOCCOSIS: A MARKER OF LIFE THREATENING DISSEMINATED CRYPTOCOCCOSIS IN HIV AIDS

SKÓRNA KRYPTOKOKOZA: MARKER ZAGRAŻAJĄCY ŻYCIU ROZSIANEJ KRYPTOKOKOZY W HIV AIDS

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Abstract
Cryptococcosis is an opportunistic infection caused by a ubiquitous encapsulated yeast, *Cryptococcus neoformans*. Affects 5 – 10 % of patients with HIV worldwide. Disseminated cryptococcosis is one of the AIDS defining criteria and the most common cause of life threatening meningitis. Upto 20% of patients with disseminated disease can have skin involvement. Cutaneous lesions in disseminated cryptococcosis are seldom pathognomonic and portent neurological involvement. The significance of skin lesions may provide the first evidence of dissemination and indicate a poor prognosis, however, earlier recognition and treatment would improve survival. Herein we report a case of cryptococcal meningitis with skin lesions in a HIV seropositive patient.

Streszczenie
Kryptokokoza jest zakażeniem oportunistycznym spowodowanym wszechobecnym, otoczkowym drożdżakiem, Cryptococcus neoformans. Dotyczy 5-10% pacjentów z HIV na całym świecie. Rozsiana kryptokokoza jest jednym z kryteriów określających AIDS i najczęstszą przyczyną zagrażającego życiu zapalenia opon mózgowych. U 20% pacjentów z rozsianą chorobą mogą występować zmiany skórne. Zmiany skórne w rozsianej kryptokokozie rzadko są znamienne dla tej choroby, a stan ogólny pogarsza zaangażowanie zmian neurologicznych. Obecność zmian skórnych może być pierwszym dowodem rozsiania oraz wskazać gorsze rokowanie, jednak wczesne rozpoznawanie oraz leczenie może poprawić przeżywalność. Opisujemy przypadek kryptokokowego zapalenia opon mózgowych ze zmianami skórnymi u seropozytywnego pacjenta z HIV.

Key words: cutaneous cryptococcosis; HIV AIDS; dissemination
Słowa klucze: skórna kryptokokoza; HIV AIDS; rozsianie

Introduction

Patients infected with HIV are susceptible to many opportunistic fungal infections. Cryptococcosis is an opportunistic infection caused by a ubiquitous encapsulated yeast, *Cryptococcus neoformans*, present in soil, dust and pigeon excreta. The main route of infection is inhalation of small yeast forms which are aerosolized. The pulmonary infection is primary site and most frequently self-limited and may be asymptomatic [1]. It occurs in 6 to 13% of patients with acquired immunodeficiency syndrome (AIDS), when their CD4 lymphocyte count is below 200/cmm [2]. Currently, AIDS represents the most common risk factor and cryptococcosis at other sites follows dissemination from lungs. Most common recognized site of disseminated cryptococcosis is the central nervous system. Cutaneous cryptococcosis is rare (20%) and is a sign of dissemination and may precede life threatening disease by several weeks. The lesions may vary greatly in morphology and mimic molluscum contagiosum or penicillium marneffei. Other presentations include acneiform papules or pustules, tumors, plaques, abscess, cellulitis, purpura, draining sinus, ulcers, bullae, subcutaneous swelling, herpetiform lesions, violaceous lichenoid lesions, nodular eruption on chin, a warty tumor on foot, a pseudofolliculitis & cryptococcosis mimicking Kaposi sarcoma [1,3]. These lesions are an

ominous sign as they are often the first presenting symptom of systemic disease.

Case Report

A 38-year-old male presented with skin lesions over face, chest & back since 10days and was admitted in neurology ward with severe headache, vomiting and seizures of 1week duration.

Patient was a known HIV seropositive since 3yrs. Patient was asymptomatic till he presented with above complaints. No history of similar complaints in the past. History of extramarital exposure – 5yrs back. No History of any chronic illness and patient did not have antiretroviral therapy.

On Dermatological examination, multiple umbilicated papules and nodules present over face, front of the chest, upper back, upper arms & forearms (Fig. 1,2). Few lesions showed necrosis at the centre (Fig. 3). Excoriations were seen. No lesions were seen over palms, soles, oral mucosa and genitals. There was no cervical or axillary lymphadenopathy. Systemic examination of nervous system was remarkably normal with no signs of meningeal irritation and neck stiffness, deep tendon reflexes were normal.

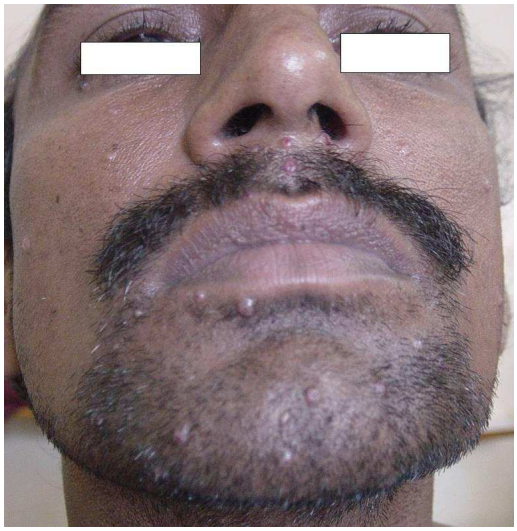

Figure 1. MC like lesions over face

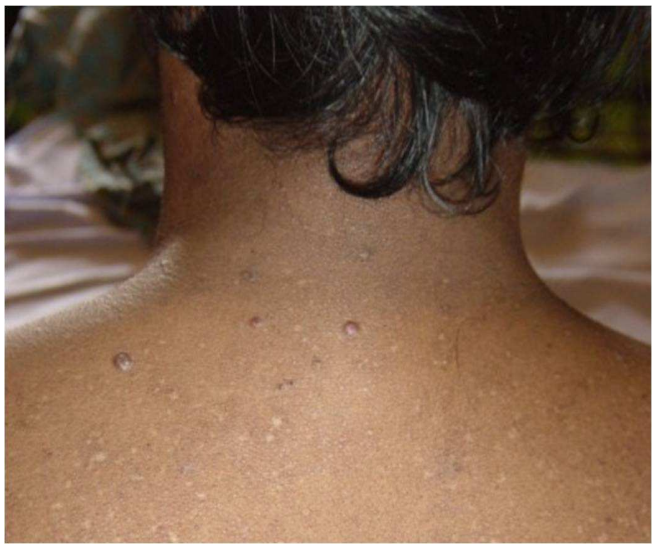

Figure 2. Papules and nodules over the upper back

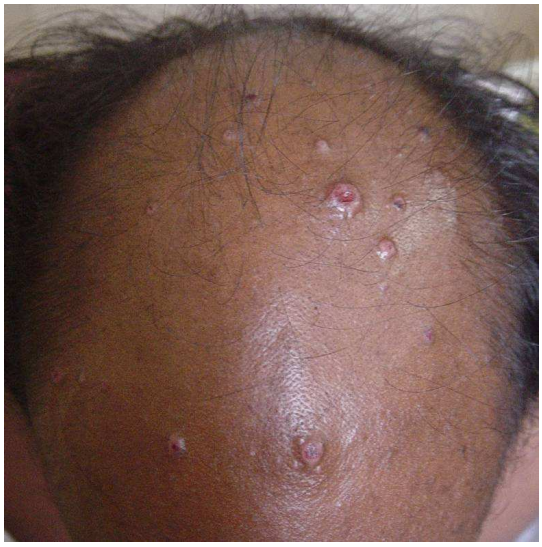

Figure 3. Lesions showing central necrosis

There was no hepatosplenomegaly and lungs were apparently normal. Haematological and Biochemical investigations were within normal limits except ESR was 95mm/1st hour. CD4 counts revealed 140cells/cu mm. CSF examination revealed round bodies arranged singly & budding yeast cells upon gram staining (Fig. 4). Negative staining with congo red with mordant showed typical capsule surrounding budding yeast cells suggestive of Cryptococcus (Fig. 5). Plain CT scan of brain showed normal study. Histopathology of Skin biopsy showed thinning of epidermis and dermis loaded with small round bodies and deep inflammatory reaction in H&E (Fig. 6) and was positive for special stain Alcian blue which confirmed cryptococcosis. Culture showed cream colour mucoid growth seen in saboraud agar media and bio-chemical tests revealed urease positivity which was consistent with *Cryptococcus neoformans var. neoformans* (Fig. 7,8).

The patient was started on Inj. fluconazole IV along with ART and symptomatic treatment. Patient died four days later.

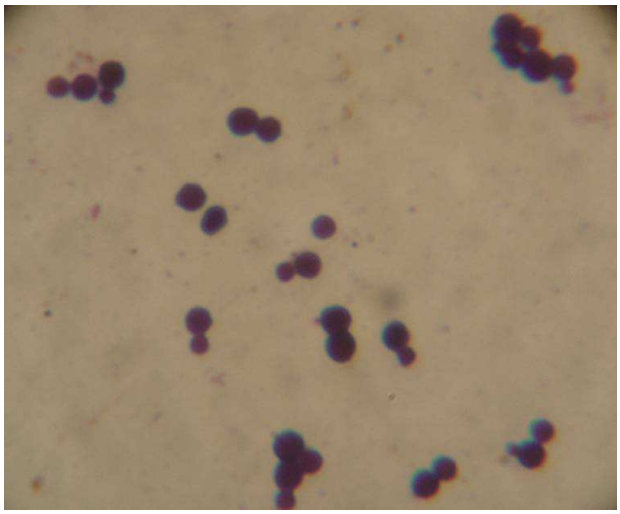

Figure 4. Gram stain showing round bodies arranged singly & budding yeast cells consistent with Cryptococcus

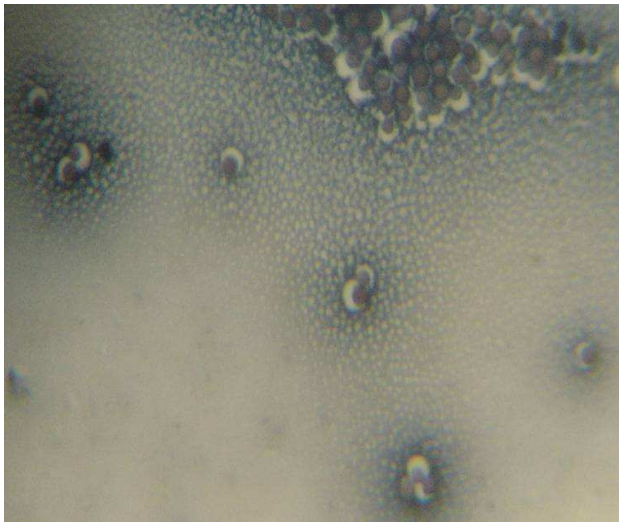

Figure 5. Negative staining with congo red with mordant showing typical capsule surrounding budding yeast cells suggestive of Cryptococcus

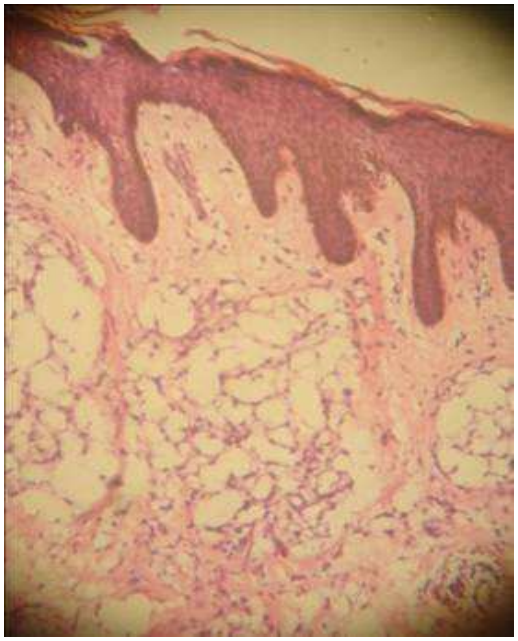

Figure 6. Histopathology of Skin biopsy showed thinning of epidermis and dermis loaded with small round bodies and deep inflammatory reaction. (H&E, 40X)

Figure 7. Culture shows cream coloured mucoid growth seen in saboraud agar media consistent with *Cryptococcus neoformans*

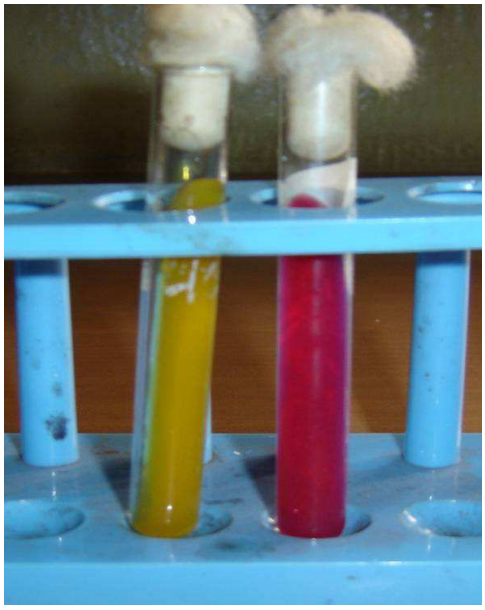

Figure 8. Bio-chemical tests revealed urease positivity which was consistent with *Cryptococcus neoformans var. neoformans*

Discussion

Cryptococcosis is Synonymous with Torulosis and European Blastomycosis. It is an acute, subacute or chronic infection caused by encapsulated yeast '*Cryptococcus neoformans'*. Cryptococcus has a predilection for brain & meninges, occasionally lungs & skin. Other organs involved rarely are bone marrow, heart, liver, spleen, kidneys, thyroid, lymphnodes & adrenal glands. *C.neoformans* was first demonstrated by Busse & Buschke in 1894 *C.neoformans* has two variants: a) *C.neoformans var. neoformans,* b) *C.neoformans var. gattii.* Serotypes A, D, or AD & B or C have been isolated. In Europe and USA, neoformans is found whereas in tropics & Africa gattii is seen. In HIV infection, neoformans variety is most common. neoformans exists as saprophyte, abundant in soil enriched in pigeon droppings. And gattii is isolated from leaf & bark debris from red gum trees. Main route of infection is inhalation of small yeast forms which are aerosolized. Most common age group affected between 30 to 60 years, uncommon in children.

Predisposing factors include immunodeficient states – AIDS, malignant lymphomas, sarcoidosis, collagen disease, carcinoma, systemic corticosteroid therapy & patients with immunosuppression following renal transplantation [4-6].

Cryptococcosis a 'Sleeping giant' among fungal diseases, Ajello in 1980. But after emergence of AIDS, cryptococcosis an 'Awakening giant'[7]. It affects 5 – 10 % of patients with AIDS worldwide. Upto 20% of

patients with disseminated disease have skin involvement, mostly by strains of serotype D. Mortality is high with 30% fatality inspite of antifungals [6].

In immunocompetent individuals, CNS is the most common system involved. It presents as chronic meningitis and focal brain lesions with classic signs of meningismus, changes in consciousness, mental changes & nerve palsies. In AIDS patients, symptoms of meningitis are minimal. Evidence of wide dissemination is by positive blood cultures or multiple skin lesions.

In pulmonary infection, chest signs include nodular shadows, cavitation & pleural effusion.

In disseminated disease, cutaneous lesions may precede or follow the signs of involvement of CNS & lungs. It occurs in about 10% of patients and are seldom pathognomonic. Molluscum contagiosum like lesions, i.e, umbilicated skin-coloured papules or nodules is the most common morphology of cutaneous cryptococcosis in 54% [6,7]. Acneiform papules or pustules are characteristic of widespread systemic infection. Most common sites are head & neck in 78%, but may be widespread [8]. Other cutaneous lesions include pustules, cellulitis, ulceration, panniculitis, palpable purpura, subcutaneous abscess and pyoderma gangrenosum like lesions [6].

In HIV AIDS, cryptococcosis is suspected when papulonodular necrotizing skin lesions like MC are seen with neurological or pulmonary disease. Other varieties described are herpetiform lesions, violaceous lichenoid lesions, acneiform papulopustular & nodular eruption on chin, a warty tumor on foot, a pseudofolliculitis and cryptococcosis mimicking Kaposi sarcoma [6,9]. Commonest differentials are Molluscum contagiosum, other systemic mycoses like Histoplasmosis and infections such as Penicillium marneffi. In all suspicious lesions, it is important to take biopsy & culture.

Systemic diagnosis is done with aid of serology, blood culture and lumbar puncture, CSF serology, culture and India ink staining. Cutaneous diagnosis is confirmed by skin biopsy with special stains for capsule (eg: mucicarmine or alcian blue) and culture or Tzanck preparation.

Direct Microscopy of (Blood or CSF) with India ink or Nigrosin mounts shows large (5-15 micron) budding cells with characteristic capsules. Culture characteristics shows colony growth which is soft, cream to pale brown & mucoid. Microscopy shows yeasts alone and no filaments.

Physiological tests reveal growth at 37^0C, Urease production, Phenoloxidase production and assimilation of creatinine and various carbohydrates. Serological tests are rapid and specific, useful in disseminated or CNS infection by detection of cryptococcal capsular antigen using Latex agglutination test or ELISA assay of blood or CSF. Very high titres are found in AIDS patients in serum & CSF. Non-AIDS patients with single, localised skin lesions are antigen-negative. Histopathology of tissue sections with special stains reveal large encapsulated budding cells with very little inflammation or granulomatous reaction [4-7]. In Non-AIDS Patients, mainstay of treatment is I.V amphotericin B combined with flucytosine. In AIDS patients, I.V amphotericin B with or without flucytosine for 7-14 days to induce remission, followed by long term oral maintenance with fluconazole 200-400 mg/day is recommended [10].

Conclusions

Cutaneous cryptococcosis may resemble molluscum-contagiosum, awareness of this rare opportunistic infection is warranted in clinical practice. Moreover, Cutaneous cryptococcosis lesions may precede the more serious disseminated forms, the early recognition and confirmation of these lesions may help the clinician to start appropriate therapy at the right time.

Acknowledgements

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NASZA DERMATOLOGIA Online
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SEBORRHEIC DERMATITIS AND HOMEOPATHY

ŁOJOTOKOWE ZAPALENIE SKÓRY I HOMEOPATIA

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Conflicts of interest: None

Abstract
Introduction: Seborrheic dermatitis is a common, usually mild skin condition affecting both sexes. Infants as well as adults may be afflicted. It may cause discomfort when not properly treated. Seborrheic dermatitis is in the spectrum of diseases found frequently in HIV infected patients and in people with AIDS. Various treatment modalities exist, all aimed at control and not cure of the disease. Homeopathy is a system of treatment that is cheap, apparently free of side-effects, does not interact with regular medications and is widely applicable in many fields of medicine, including dermatology. Any new, but efficacious, treatment modality is always welcome in dermatology.
Materials and methods: Two patients with seborrheic dermatitis of varying severity and duration were treated with homeopathy and the results documented.
Results: The patients recovered fully and are still in remission years later.
Conclusions: Homeopathy may be of use in the treatment of acute and chronic seborrheic dermatitis. Since it is cheap, free of side-effects and does not interfere with regular medication, it may become an attractive option in the treatment of this disorder, especially in patients with multiple pathologies.

Streszczenie
Wprowadzenie: Łojotokowe zapalenie (ŁZS) skóry jest częstym, zazwyczaj łagodną dolegliwością skórną, występującą u obu płci. Zarówno niemowlęta, jak i dorośli mogą być dotknięci tym schorzeniem. ŁZS może powodować dyskomfort, gdy jest nie właściwie leczone. Łojotokowe zapalenie skóry należy do spektrum chorób często rozpoznawalnych u pacjentów zakażonych HIV i chorych na AIDS. Istnieją różne metody leczenia, wszystkie jednak mają na celu kontrolę, a nie leczenie choroby. Homeopatia jest systemem leczenia, który jest tani, w znacznym stopniu wolny od skutków ubocznych, nie wchodzi w interakcje z regularnie stosowanymi lekami i jest szeroko stosowane w wielu dziedzinach medycyny, w tym w dermatologii. Każdy nowy, pod warunkiem że skuteczny, sposób leczenia jest zawsze mile widziany w dermatologii.
Materiał i metody: Dwóch pacjentów z łojotokowym zapaleniem skóry o różnym stopniu nasilenia i czasie trwania leczono homeopatią, a wyniki udokumentowano.
Wyniki: Objawy ustąpiły całkowicie a pacjenci są nadal w kilkuletniej remisji.
Wnioski: Homeopatia może być użyteczne w leczeniu ostrego i przewlekłego łojotokowego zapalenia skóry. Ponieważ leczenie jest tanie, bez skutków ubocznych i nie koliduje z regularnym podawaniem leków, może stać się atrakcyjną alternatywą w leczeniu tej choroby, zwłaszcza u pacjentów z wieloma patologiami.

Key words: seborrhea; dermatitis; topical steroids; homeopathy; ignatia; magnesia carbonica
Słowa klucze: łojotok; zapalenie skóry; miejscowe steroidy; homeopatia; ingatia; magnesia carbonica

Introduction

Seborrheic dermatitis is a common, usually mild skin condition affecting both sexes. Infants as well as adults may be afflicted. It may cause discomfort when not properly treated. Seborrheic dermatitis is in the spectrum of diseases found frequently in HIV infected patients and in people with AIDS. Various treatment modalities exist, all aimed at control and not cure of the disease. Amongst these are topical antiparasitic, antifungal and steroid creams, all with varying efficacy and limitations of usage. Homeopathy is a system of treatment that is cheap, apparently free of side-effects, does not interact with regular medications and is widely applicable in many fields of medicine, including dermatology. Two cases of seborrheic dermartitis of long-duration treated with homeopathy are presented.

Materials and methods

Two female patients were treated with homeopathy and the results were recorded.

Patient 1

A 25 year-old female, with a facial skin rash that had been on and off for years with a severe flare one month before presentation. She had been treated with methylprednisolone cream, with limited effect. She felt that stress was a major contributing factor to here flares. She had stopped smoking 8 years prior to presentation. Her past medical history was significant for mild hepatomegaly with raised enzyme levels, gastritis and suspected pelvic inflammatory disease. She had also had an appendicectomy. On examination, she was found to have a scaling, erythematous rash, with poorly defined borders, concentrated around the central face. There were no body lesions. She received the homeopathic medicine Ignatia, in the M potency, once. She was asked to stop using methylprednisolone and sulphur creams and to use a cream containing aloe vera as an adjuvant to homeopathic treatment. She also received acupuncture tratment for her abdominal pains. An aggravation followed cessation of the steroid creams and one month later, the lesions had remitted and remain remitted today, 2 years after the onset of treatment.

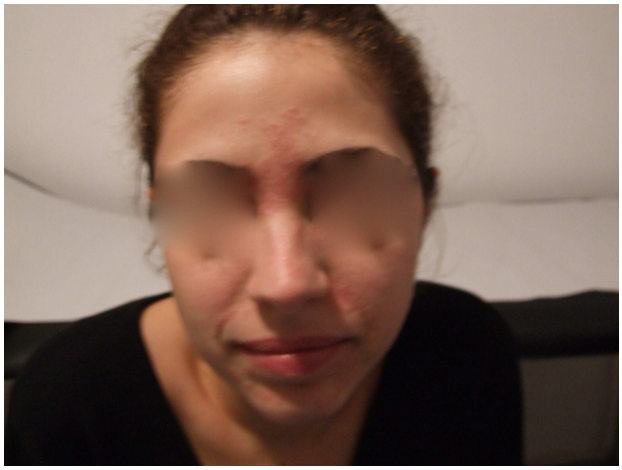

Figure 1. Seborrheic dermatitis

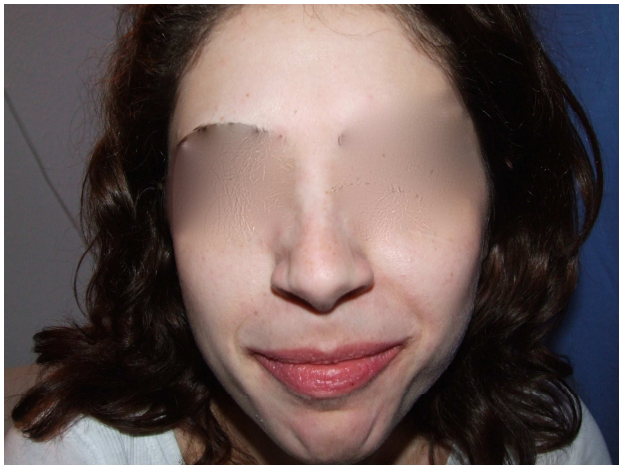

Figure 2. Seborrheic dermatitis

Patient 2

A 42 year-old female presented with an asymptomatic facial and scalp eruption that had been treated for years with methylprednisolone and sulphur creams. The eruption was exacerbated during periods of emotional stress. Her prior medical history was significant for bilateral mammary dysplasia, which had been treated with hormonal and antiinflammatory creams as well as mild lumbar disc disease. On examination, she was found to have a slightly brown, scaling rash extending beyond the borders of the hairy scalp, with circinate borders. She received the homeopathic medicine Magnesia carbonica at CH200 potency (the M potency was unavailable), on a weekly basis. Two months later, the lesions remitted. She has occasional slight exacerbations during periods of great stress, but is otherwise completely lesion free.

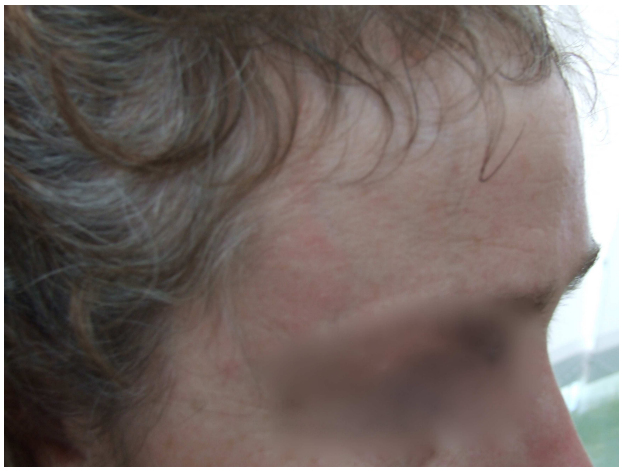

Figure 3. Seborrheic dermatitis

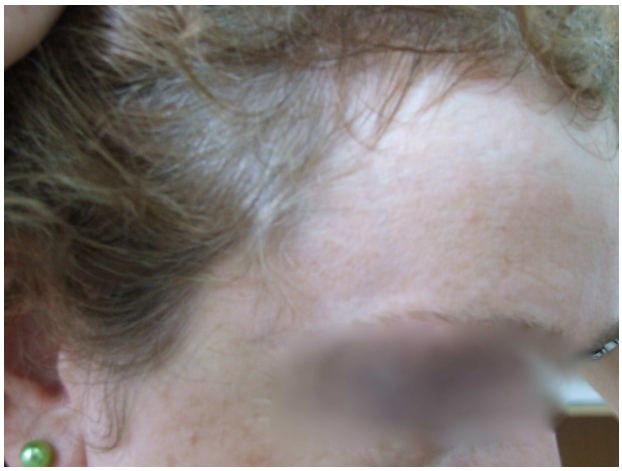

Figure 4. Seborrheic dermatitis

Discussion

Seborrheic dermatitis is a common skin disorder associated with increased activity of the sebaceous glands, though not considered to be a disorder of the sebaceous glands themselves [1,2]. It may affect all ages, but is very common in childhood, affecting about 10% of children, most under 3 months old [3]. It is more frequent amongst HIV-infected individuals[1] and individuals with neurologic disorders [4].

Various treatment modalities have been tried, including topical selenium sulphide, ketoconazole, steroids and topical calcineurin inhibitors, with varying success [1,2,4].

Homeopathy was first started by the German physician S. Hahnemann and its use is widespread in Europe today. It is based on the use of high dilutions of substances and,

until recently, the efficacy of such high dilutions was believed to be scientifically untenable. Recent evidence shows that these dilutions actually work. A team led by the Nobel Prize winner Luc Montagnier showed that bacterial DNA in aqueous dilutions emitted electromagnetic waves [5]. These dilutions of DNA were similar to those dilutions used in homeopathic medicines. The emitted electromagnetic waves were found to be associated with the formation in solution of well-defined polymeric nanoparticles. The presence of nanoparticles of the original substaces in high dilutions of homeopathic medicines was clearly shown by other workers using transmission electron microscopy, as well as inductively coupled plasma-atomic emission spectroscopy [6]. They demonstrated that such dilutions contain something after all. Their work, as well as that of the group led by Prof. Montagnier give credence to the value of homeopathic medicines, though do not yet provide a scientific mechanism for how these remedies work in the human body.

We have found homeopathy to be very helpful in the therapy of recalcitrant verucca vulgaris in patients with diabetes mellitus. In these cases it was also found to be useful improving metabolic control as shown by the reduction in HBA1c levels, though this was not the aim of treatment. It may therefore be of value in improving diabetic control [7]. Homeopathy can also be useful in the treatment of severe acne of long duration [8]. In both situations, homeopathic therapy was found to be free of side-effects, cheap and to not react in any way with the patients concomittant medications, where these were the case.

Conclusions

Seborrheic dermatitis is a common cutaneous disorder and is frequently encountered in children, especially those below 3 months of age. Various treatment modalities exist. Homeopathy may provide a cheap and effective means of treating this disorder. Any new treatment modality that holds the promise of efficacy is always welcome in dermatology. Clinical studies may be required to conclusively establish homeopathy as one of the treatment modalties available to patients for the therapy of seborrheic dermatitis.

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LAT, EGFR -pY197, PCNL2, CDX2, HLA-DPDQDR, BROMODEOXYURIDINE, JAM-A, AND EZRIN IMMUNOREACTANTS IN A RUBBED SPONGIOTIC DERMATITIS

LAT, EGFR-PY197, PCNL2, CDX2, HLA-DPDQDR, BROMODEOXYURIDINE, JAM-A I EZRYNA; IMMUNOREAKTANTY W SPONGIOSTYCZNYM ZAPALENIU SKÓRY

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Conflicts of interest: None

Abstract
Background: Acute and subacute spongiotic dermatitides are among the most commonly diagnosed types of dermatitis. Many patients rub their lesions, with the lesions becoming clinically thickened. The precise immunologic mechanisms within the thickening process are not well defined. **Case report:** An 85 year old male presented with the sudden clinical appearance of erythematous patches and small blisters on the back of his legs, with pruritis. **Methods:** Skin biopsies, one from a rubbed lesion and one from a non-rubbed lesion were submitted for hematoxylin and eosin (H&E), immunohistochemistry (IHC), and for direct immunofluorescence (DIF) analysis. **Results:** The H&E staining demonstrated classic features of a spongiotic dermatitis, but in the rubbed areas psoriasiform hyperplasia was also seen. The psoriasiform areas demonstrated positive, focal IHC staining with bromodeoxyuridine, LAT, EGFR-pY197, PCNL2, CDX2, and HLA-DPDQDR antibodies. DIF staining revealed positive staining of JAM-A and ezrin in the non-rubbed specimens in both the spongiotic epidermis and in the adjacent vessels; normal expression of these markers was appreciated in the rubbed biopsy. **Conclusions:** The immune response seems to be complex when a spongiotic dermatitis is converted from a non-rubbed to a rubbed lesion with histologic features of psoriasiform hyperplasia.

Streszczenie
Wstęp: Ostre i podostre spongiostyczne zapalenia skóry są jednymi z najczęściej diagnozowanych rodzajów zapaleń skóry. Wielu pacjentów drapie swoje zmiany (skórne), co prowadzi do ich pogrubienia. Dokładne mechanizmy immunologiczne prowadzące do pogrubienia (warstw skóry) nie są dobrze zdefiniowane. **Opis przypadku**: 85-letni mężczyzna prezentował nagle pojawiające się rumieniowe plamy i małe pęcherze na tylnej powierzchni kończyn dolnych z towarzyszącym świądem. **Metody:** Analizowano biopsje skóry, jedną z podrapanych zmian i jedną z nie-potartych zmian, którą poddano działaniu hematoksyliny i eozyny (H & E), immunohistochemii (IHC) oraz immunofluorescencji bezpośredniej (DIF). **Wyniki**: Barwienia H & E wykazały klasyczne cechy „spongiotic dermatitis", ale w obszarach potartych obserwowano rozrost łuszczycopodobny. Obszary łuszczycopodobne wykazują pozytywne, ogniskowe barwienie IHC z bromodeoxyuridiną, przeciwciałami LAT, EGFR-pY197, PCNL2, CDX2 i HLA-DPDQDR. DIF ujawnił pozytywne zabarwienie dla JAM-i ezryny w niepotartych obszarach zarówno naskórka spongiostycznego jak i przyległych naczyniach; normalna ekspresja tych markerów została zaobserwowana w preparacie z obszaru potartego. **Wnioski:** Odpowiedź immunologiczna wydaje się być skomplikowana, gdy spongiotyczne zapalenie skóry przekształca się z niepodrapanych w przetarte zmiany z histologicznym obrazem łuszczycopodobnej hiperplazji.

Key words: spongiotic dermatitis; LAT; EGFR-pY197; PCNL2; CDX2; HLA-DPDQDR; bromodeoxyuridine; JAM-A; ezrin; sweat and sebaceous glands
Słowa klucze: spongiotic dermatitis; LAT; EGFR-pY197; PCNL2; CDX2; HLA-DPDQDR; bromodeoksyurydyna; JAM-A; ezryna; gruczoły potowe i łojowe

Introduction

The spongiotic dermatoses, including eczema and its clinicopathologic variants are capable of multiple clinical presentations and are pathologically defined by the presence of epidermal edema that may infrequently form blisters. The immunologic mechanisms which underlie the pathogenesis of these disorders have only recently begun to be elucidated [1,2]. Spongiotic

dermatitis may occur as a result of an allergic reaction. Such allergic reactions include an allergic contact reaction, an allergic reaction to food or a medication, or other allergic reactions. Spongiotic dermatitis is occasionally categorized clinically as eczema. Spongiotic dermatitis may be categorized histologically acute or subacute[1,2]. The main difference between histologic acute and subacute spongiotic dermatitis concerns the size of the vesicles developed by the intercellular edema. Specifically, an acute spongiotic dermatitis has larger vesicles compared with a subacute spongiotic dermatitis [1,2]. Similar to many clinical types of dermatitis, the presenting symptom is often a pruritis. After a variable clinical period, the rash appears [1,2]. The rash is usually initially erythematous, and often turns brown after repetitive rubbing or scratching of the affected areas [1,2]. Histologically, rubbed acute and subacute spongiotic dermatitides commonly develop psoriasiform hyperplasia, with additional hyperkeratosis [1,2]. Few studies have undertaken a simultaneous, comparative examination of non-rubbed acute and lichenified, rubbed lesions in the same patient. Thus, we obtained skin biopsies for hematoxylin and eosin (H&E), direct immunofluoresence (DIF) and immunohistochemistry (IHC) analyses simultaneously from both types of lesions. Our IHC staining was performed as previously described**.** We conducted IHC staining for LAT, survivin, epidermal growth factor receptor pY197 (EGFR-pY197), the proliferating cell nuclear antigen (PCNA), caudal type homeobox transcription factor 2 (CDX2), complement C5b-9 (MAC), junctional adhesion molecule (JAM-A), ezrin and HLA-DPDQDR, as previously described [3,6]. Our monoclonal antibodies were obtained from Dako North America (Carpinteria, California, USA), with the exception of JAM-A and ezrin (Invitrogen, Carlsbad, California, USA).

Case report

A 85 year old male presented with rapidly appearing, inflammatory patches and plaques on the backs of his legs. Clinical examination demonstrated large, hyperkeratotic plaques, as well as separate patch areas with edema and evidence of surrounding inflammation. Lesional skin biopsies were taken from both types of lesions for hematoxylin and eosin (H&E) analysis, for immunohistochemistry (IHC) and for direct immunofluorescence (DIF) comparative studies.

Results

Microscopic description:

Examination of the H&E tissue sections in the non-rubbed biopsy demonstrated diffuse, moderate epidermal spongiosis. The dermis displayed a mild, superficial, perivascular infiltrate of lymphocytes, histiocytes and eosinophils; neutrophils were rare. No vasculitis was present; focal dermal edema was noted around the eccrine and sebaceous glands. The rubbed biopsy demonstrated hyperparakeratosis with overall psoriasiform hyperplasia; focal areas of mild spongiosis were noted, with one small subcorneal blister. Dermal edema surrounding the eccrine and sebaceous glands was also noted. DIF of the non-rubbed biopsy displayed the following results: IgG (-); IgG3 (-); IgG4 (-); IgA (-); IgM (-); IgD (-); IgE (-); Complement/C1q (-); Complement/C3 (-); collagen IV (CIV) (++, normal distribution); JAM-A and Ezrin (+, some accentuation near the basement membrane zone (BMZ) and in the spongiotic epidermis). Albumin (-), fibrinogen (++, around upper dermal blood vessels and eccrine glands). The DIF of the rubbed biopsy was mostly negative (Fig. 1,2). The IHC studies in the rubbed biopsy were patchy positive in areas where the spongiosis seemed to be transitioning into psoriasiform hyperplasia. Some alterations were also seen around dermal eccrine and sebaceous glands, and around dermal blood vessels (Fig. 1,2). In both the non-rubbed and rubbed biopsies, the IHC stains for LAT, EGFR-pY197, bromodeoxyuridine, MAC, PCNA, CDX2, and HLA-DPDQDR were focally positive, primarily in areas of the previously described hyperparakeratosis.

Discussion

Our results indicate that the immune response is modified in acute spongiosis in rubbed areas versus non-rubbed areas. The rubbing action leads histologically to hyperkeratosis and psoriasiform hyperplasia. Based on our data, we suggest that the immune response may vary vis-à-vis acute or subacute stages in spongiotic dermatitides [8-12]. An initial immune response seems to exist to the initializating allergens, however, subsequent itching and sweating could further irritate the skin, leading to clinical individual rubbing and scratching. These actions could then in turn lead to further immune alterations, inducing further stimulation of the T cell receptor (TCR) as indicated by IHC positivity to LAT. Other pathophysiologic events seem to occur simultaneously, including cell proliferation (indicated by positive IHC staining for PCNL2 and bromodeoxyuridine) [8-12]. The rubbing of the epidermis may induce the IHC positivity seen for the epidermal growth factor receptor; further, the rubbing and/or other signals may induce inflammation around dermal blood vessels and sweat glands, as suggested here by IHC positivity for fibrinogen and other immunoreactants. More specifically, we suggest the epidermal spongiotic areas with hyperkeratotic alterations may upregulate and/or downregulate multiple cell signals that are induced by the rubbing itself [9-11]. In addition, JAM-A and ezrin (cell junction molecules) could be also be upregulated in acute spongiosis, but not in the more chronic lesions. EGPF-pY197 and PCNA seem to be expressed in the hyperkeratotic areas, suggesting creation of a cycle of rubbing and feedback increases in cell proliferation [9-11]. Potentially, the local and subtle expression of PCNA would attempt to prevent cell apoptosis in the edematous skin.

To our surprise, the CDX2 antibody was also positive in same areas as EGFR-pY197 and PCNA; this molecule is strongly expressed in intestinal epithelia. However, it has been shown that the *in vivo* phenotype of each epithelium seems to depend on multiple factors. Specifically, the morphological and biochemical differentiation of a given epithelium may be reversibly modulated by the external environment; thus, extrinsic factors may, under certain conditions, affect the morphological and biochemical differentiation of a given epithelium. Further studies are required to properly identify these molecules, and their roles in regulating epithelial differentiation. In regard to

HLA class II, it has been shown that expression of these molecules on keratinocytes has been confirmed in allergic contact dermatitis, while absent in atopic dermatitis [11]. Other authors have demonstrated keratinocyte HLA staining accompanying epidermal lymphoid infiltrates. In addition, other authors have noted evidence of focal keratinocyte damage in the mid-epidermis in eczema, as we have previously described [5-6].

Identifying the triggers of a spongiotic dermatitis is a challenging process of elimination. However, given our data, we recommend advising the patient to avoid rubbing clinical lesions, thus avoiding further immunologic alterations that may, in turn, autocatalyze clinical lesional development.

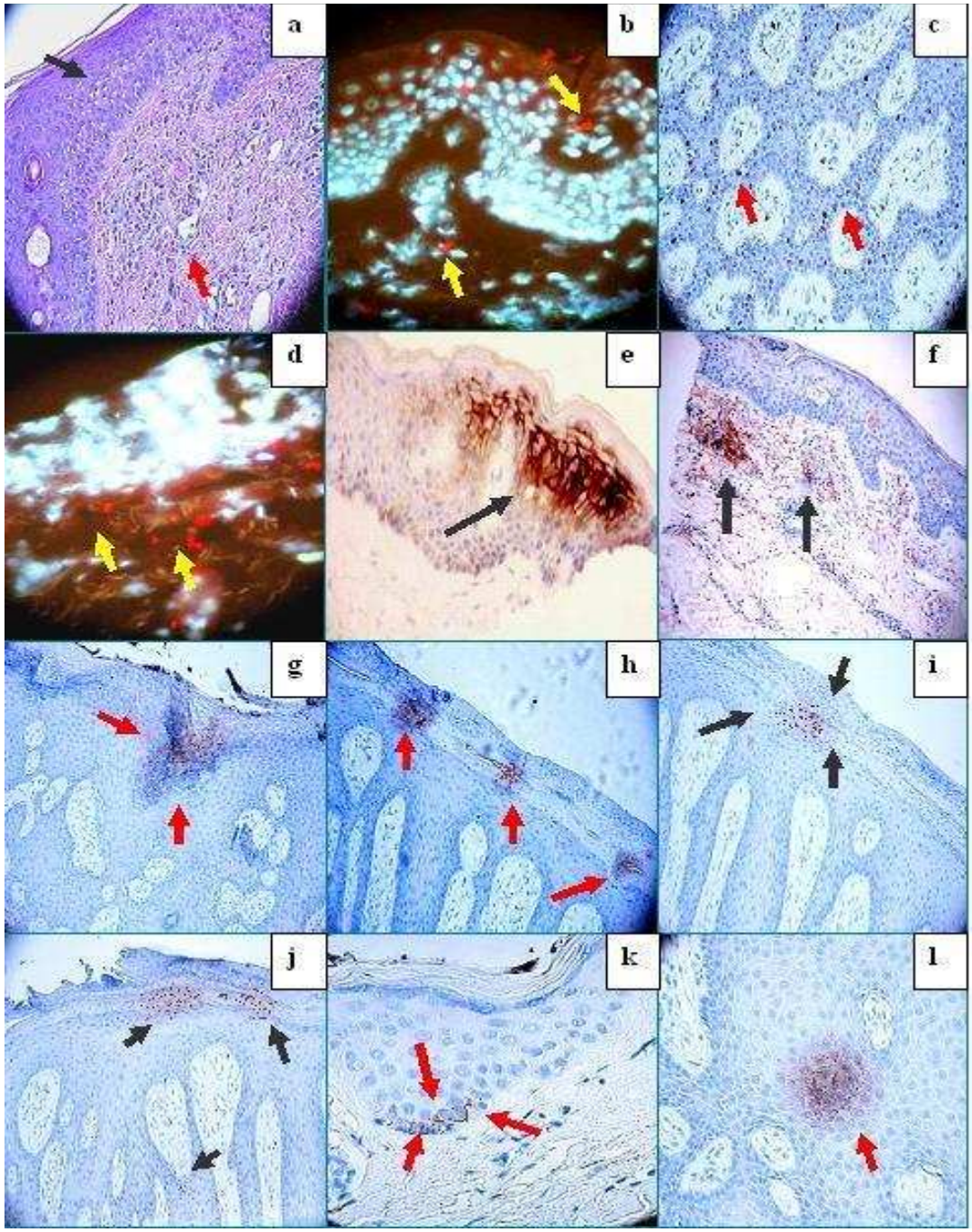

Figure 1. a. H&E demonstratingm focally marked epidermal spongiosis(black arrow) with diffuse edema in the papillary dermis and inflammatory infiltrates around the upper dermal blood vessels (red arrow). **b**. DIF demonstrating focal areas staining positive for Texas red conjugated JAM-A (red staining; yellow arrows) in the epidermis as well as in dermal perivascular areas. Keratinocyte and other cell nuclei were counterstained with 4',6' diamino-2-phenylindole (Dapi) (blue-white staining). **c**. IHC demonstrating positive staining with PCNL2 antibody in several cells along the basement membrane zone of the epidermis (brown staining, red arrows).**d.** DIF demonstrating positive staining with Texas red conjugated ezrin (red stain) in several perivascular areas of the upper dermis (red staining; yellow arrows). Nuclei were again counterstained with Dapi. **e**. and **f** , IHC demonstrating positive staining with HLA-DPDQDR antibody in some of

the epidermal spongiotic areas in **e** and in the dermal perivascular inflammatory cell infiltrate in **f** (brown staining, black arrows). **g** Positive IHC staining for LAT antibodies in a localized area in the upper epidermis where the spongiosis is prominent (brown staining; red arrows). **h** Positive IHC staining for CDX2 in the epidermal corneal layer (brown staining, red arrows). **i**. Positive IHC staining for EGFR-pY197 in focal areas of the epidermal corneal layer where rubbing has occurred (brown staining; black arrows). Notice that the epidermis is displaying having psoriasiform hyperplasia. **j.** Strongly positive IHC staining for PCNL2 within epidermal corneal layer with hyperkeratosis, as well as in some cells along the basement membrane zone (brown staining; black arrows). **k**. Positive IHC staining with bromodeoxyuridine in a cluster of cells along the epidermal basement membrane area (BMZ) (brown staining; red arrows). **l**. Positive IHC staining with LAT in a focal areas of the epidermal stratum spinosum (brown staining; red arrow).

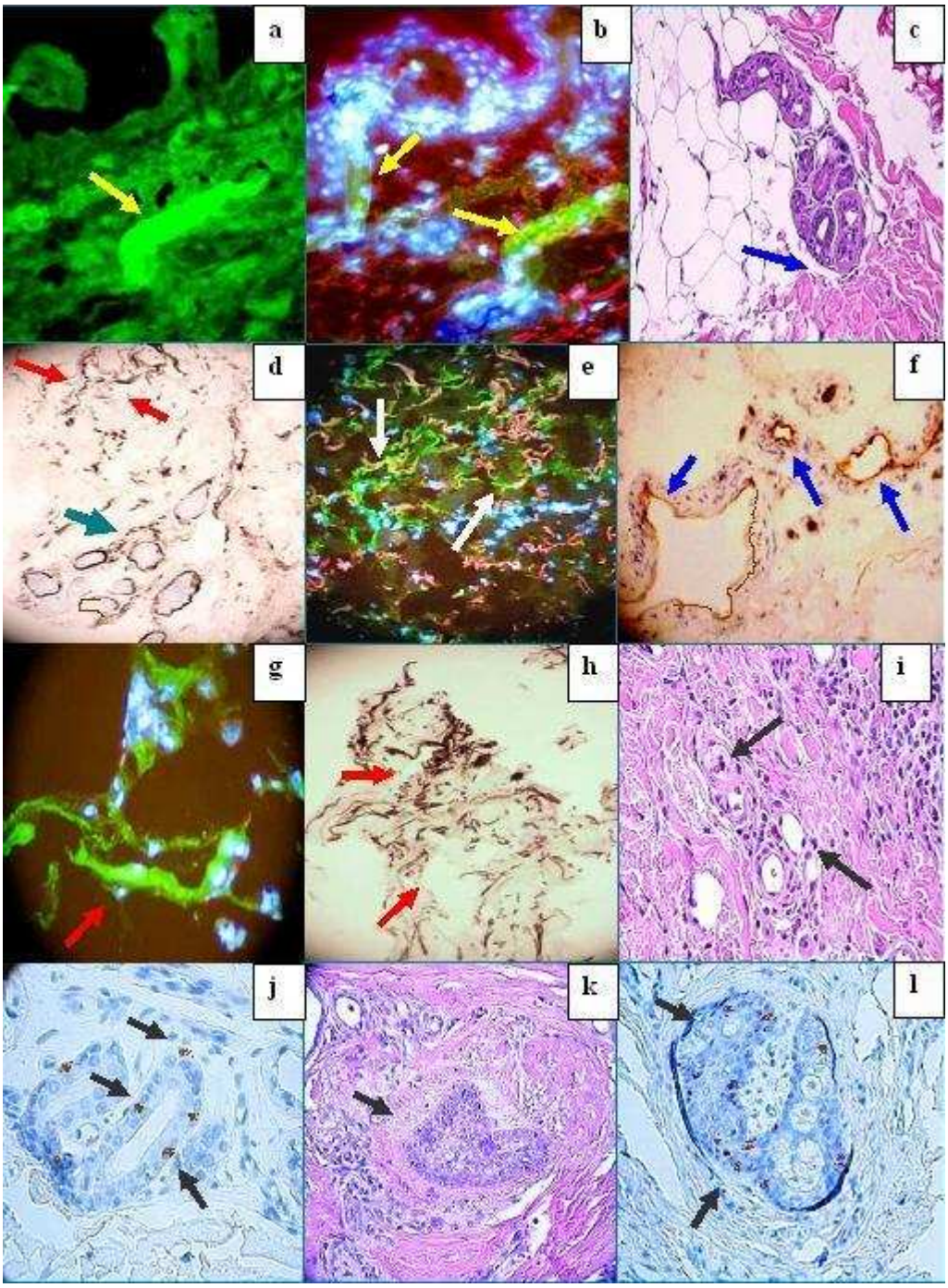

Figure 2. a and **b,** Positive DIF staining with FITC conjugated fibrinogen antibodies against the dermal eccrine ductus (green staining; yellow arrows). Nuclei were counterstained with Dapi (blue-white). In **b**, also note positive staining for Texas red conjugated JAM-A around several upper dermal blood vessels. **c.** H&E demonstrating dermal edema around eccrine sweat glands (blue arrow). **d**. Positive IHC staining for MAC, around dermal blood vessels (red arrow), and around

dermal eccrine sweat glands (green arrow). **e**. Positive IHC staining with anti-human FITC conjugated fibrinogen around several upper dermal vessels (green staining; white arrows). The nuclei were counterstained with Dapi. Positive staining for JAM-A is also present (pink). **f.** IHC positive staining with anti-human HLA-DPDQDR antibody, present around upper blood vessels (brown staining, blue arrows). **g**. Positive DIF staining with FITC conjugated anti-human fibrinogen around the upper dermal blood vessels (green staining, red arrow). The nuclei were counterstained with Dapi. **h**. Detail of positive IHC staining for MAC around upper dermal blood vessels (red arrows). **i.** H&E detailing some mild, chronic inflammation around upper dermal dilated blood vessels (black arrows). **j** and **l**, IHC positive staining with PCNL2 antibody in some cells of the eccrine sweat glands and sebaceous glands, respectively (brown staining, black arrows). **k.** H&E detail of mild inflammation and edema surrounding a portion of a sebaceous gland (black arrow).

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ACNE INVERSA (HURLEY CLINICAL STAGE II): CASE REPORT

ACNE INVERSA (HURLEY - ZAAWANSOWANIE KLINICZNE II'): OPIS PRZYPADKU

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Abstract
We present a case of acne inversa Hurley clinical stage II, to a 28 year-old patient non-obese, smoker, with a long history of firm nodules, large abscesses and sinous tracts, small scars, distributed in the axillary, groin, perianal and infraumbilical areas, associated with lesions on the face. Any therapeutic schemas (antibiotics, Isotretinoin orally, Dapsone, UVB,cryotherapy) was unsuccesfully and we sent the patient to Surgery Department for wide excisions.

Streszczenie
Prezentujemy przypadek trądziku odwróconego w II etapie zaawansowania klinicznego Hurley, u 28-letniego pacjenta bez otyłości, palącego papierosy, z długą historią guzków, dużych ropni, przetok, małych blizn, zlokalizowanych w dołach pachowych, w pachwinach, w okolicy odbytu i w obszarach poniżej pępka, związane ze zmianami na twarzy. Wszelkie schematy terapeutyczne (antybiotyki, izotretynoina doustna, dapson, UVB, krioterapia) był nieskuteczne, w związku z tym wysłaliśmy pacjenta do Oddziału Chirurgii celem opracowania chirurgicznego.

Key words: acne inversa; Isotretinoin; Dapsone; UVB; surgery
Słowa klucze: trądzik odwrócony; Isotretinoina; Dapson, UVB; chirurgia

Introduction

Hidradenitis suppurativa (from the Greek hidros = sweat and aden = glands) is a chronic follicular occlusive disease involving the intertriginous skin of the axillary, groin, perianal, and inframammary regions.

Author	Year	Name of the disease
Velpeau - surgeon from Paris	1839	first description: axillaray, submamary and perianal abcesses
Verneuil -Paris	1854	first name: hidrosadenite phlegmoneuse first pathogenic mechanism: inflamation of sweat glands
Schiefferdecker	1922	association acne-appocrine sweat glands
Pilsburry	1956	acne triad: hidradenitis suppurativa+acne conglobata+perifolliculitis capitis abscendens et suffodiens and the cause: follicular oclusion
Plewig-Kligman	1975	acne tetrad: acne triad+pilonidal sinous
Plewig-Steger	1989	introduced the term: **acne inversa** (term accepted today all the world)
Recent studies	2000	genetic disease?

Table 1. History of the disease

Acne inversa has a typical clinical picture: cutaneous and subcutaneous nodular nflammation, fistulae with malodorous secretion and scaring. It affects men and women, with an incidence between 1-4 %, with a peak in the second and third decade of life, with unknown pathogenesis but with well documented trigger factors: smoking (unclear mechanism), obesity (by maceration and occlusion in the body folds through follicular hyperkeratosis), positive family history and lately genetic backgrounds.

Acne inversa is today regarded as an inflammatory disease of terminal hair follicules and not a disease of appocrine glands [1] that can explain the influence of androgens in the course of the disease, its absence before puberty and some therapeutic results with anti-androgen, although the hormonal levels in all patients are within normal limits [2].

Case report and Conclusion

A 28 year-old patient non-obese, smoker, presented in our department, with a long history of firm nodules, large abscesses and sinous tracts, small scars, distributed in the axillary, groin, perianal and infraumbilical areas, associated with lesions on the face. No fever, but pains and pruritus and an important impairment of the quality of life (Fig.1,2).

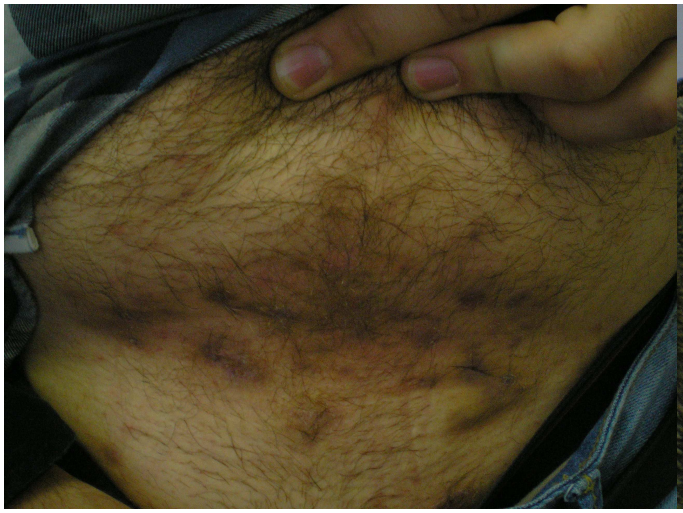

Figure 1. Lesions under ombilicus

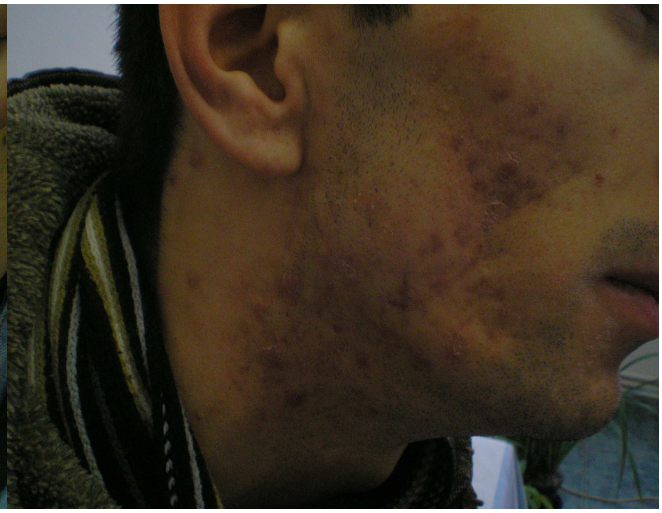

Figure 2. Lesions on the face

All the lab parameters were within normal limits, including androgen level.

Based on clinical aspects: (recurrent abscesses with tract formation and cicatrisation, multiple widely separated lesions with bilateral distribution on specific areas) and on chronicity of the lesions, we established the diagnosis of acne inversa Hurley clinical stage II.

We started immediatly Isotretinoin 20 mg/day increased after 2 months to 40 mg/day with a slight positive evolution in the first weeks of treatment, but with an agressive relapse 3 months later, we stopped the medication after 10 months.

Based on the cultures performed from the axillary and anogenital regions which found Staphylococcus aureus, we introduced antibiotic therapy: Azythromycine, Ciprofloxacine and Oxacilline, but with no improvement.

The next step was the treatment with Dapsone 50 mg/day ,which was also discontinued after two months, for the absence of any therapeutical answer.

Strictly on the lesions, on small areas, we performed cryotherapy interrupted because of pains and later UVB 311 nm with no results.

So we are in front of a patient with a long history of acne inversa, not responding to treatment after one year of trying different therapeutical approaches.

Patient refused any other conservative therapy (such as TNF alfa antagonists or Methotrexat) and decided to accept the surgical treatment: wide excision of lesions in healthy tissue (lateral and deep safety margins). We sent him to the Surgery Department.

The particularity of this case was our fail of therapy and finally the decision to send the patient to Surgery.

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ACNE INVERSA (HURLEY CLINICAL STAGE II): CASE REPORT

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Acne inversa also known as hidradenitis suppurativa is a chronic disease with great burden for patients. There has been much debate about terms and contents. From the histopathologic point of view, hidradenitis is a misnomer but as with other misnomers in medicine it is still in use.

Although smoking and obesity are major known risk factors, stopping smoking after onset of disease does not alter the course so much. Treatment can be a challenge. Drug therapy often does not make a point. Only in early stages there is a temporary release. The more advanced the disease the greater the need for surgery. This has been very nicely shown by the contribution of Anca Chiriac et al. from Romania, who tried to cope with the disease by a broad armentarium of drugs and procedures.

The paper also demonstrates that dermatologic surgery needs to be more developed in Europe. If we as dermatologists want to deal with the more severe dermatoses we have to establish a curriculum in dermatologic surgery. There is a number of very succesfull societies world wide like the American Society for Dermatologic Surgery, the British Society for Dermatologic Surgery, the Indian Society for Dermatologic Surgery or the Polish Society for Dermatologic Surgery just to name a few.

It would be an interesting idea to develop some standards for education and procedures in Europe.

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A CASE OF NOCARDIA MYCETOMA DEVELOPING AT THE SITE OF SKIN GRAFTING

PRZYPADEK ROZWOJU NOCARDIA MYCETOMA W MIEJSCU SZCZEPIENIA

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Abstract
Mycetomas are chronic infections of the skin, subcutaneous tissue and deeper tissues caused by fungi or filamentous bacteria and are characterized by tumefaction, nodules and sinuses. Eumycetomas are caused by fungi and actinomycetomas are caused by filamentous bacteria. Bacteria causing actinomycetomas are saprophytes found in soil, on plants, and on dead and decaying matter. They are aerobic, gram positive and weakly acid fast and form aggregates of micro colonies that appear as grains in the sinuses. Clinically, mycetomas present as areas of tumefaction with nodules and discharging sinuses. Bacterial mycetomas are sensitive to many antibiotics like penicillin, tetracyclines, sulphonamides, rifampcin, aminoglycocides etc, and long term combination therapies with variable success have been reported from different parts of the world. We have presented here a 23 year old male patient who presented with one year history of developing nodules and sinuses in the region of left flank. Patient had undergone skin grafting at the same site 3 years back for a wound he developed in the area following a road accident. He was unsuccessfully treated with antituberculous therapy elsewhere. He was admitted in our institute; the diagnosis of nocardia mycetoma was established and patient was successfully treated with complete clearance of lesions.

Streszczenie
Mycetoma są przewlekłymi, skórnymi chorobami zakaźnymi, obejmujące tkankę podskórną i głębiej położone tkanki, spowodowane przez grzyby lub/i nitkowate bakterie, charakteryzujące się nabrzmieniem, guzkami i zatokami. Eumycetoma są spowodowane przez grzyby, a actinomycetoma są spowodowane przez nitkowate bakterie. Bakterie wywołujące actinomycetoma są saprofitami, które można znaleźć w glebie, na roślinach oraz na martwej i gnijącej materii. Są one tlenowe, gram-dodatnie i słabo kwaśne; występują w formie agregatów w mikro koloniach, które pojawiają się jako ziarna w zatokach. Klinicznie, mycetoma obecne są jako obszary nabrzmienia z guzkami i wydalającymi zatokami. Bakteryjne mycetoma są wrażliwe na wiele antybiotyków jak penicyliny, tetracykliny, sulfonamidy, ryfampicyna, aminoglikozydy itp., a po długim okresie kombinowanej terapii wyzdrowienia ze zmiennym sukcesem zostały zgłaszane z różnych stron świata. Przedstawiamy 23-letniego pacjenta, który prezentował jednoroczną historię rozwoju guzków i zatok w regionie lewego boku. Przed 3-ma laty pacjent przeszedł przeszczep skóry w tym samym miejscu z powodu rany w wyniku wypadku drogowego. Był bezskutecznie leczony antytuberkulinową terapią w innej jednostce. Pacjent był przyjęty do naszego instytutu; postawiono diagnozę nocardia mycetoma i wdrożono skuteczną terapię z całkowitym wyleczeniem zmian skórnych.

Key words: Nocardia; mycetoma; penicillin
Słowa klucze: Nocardia; mycetoma; penicylina

Introduction

Mycetomas are infections caused by fungi or filamentous bacteria and are characterized by tumefaction with nodules and sinuses. Organisms are saprophytes found in soil or on plants [1]. Mostly infections follow penetrating injuries, especially amongst those people who walk barefoot; the disease is more common in tropical and sub-tropical regions [2]. After

gaining access into the subcutaneous space, a swelling develops in the region which is followed by numerous nodules and sinuses over a span of months and sometimes years [3]. Grains that are found in the discharge from sinuses are aggregates of microcolonies of the organisms. Failure to recognize the condition early may lead to deeper involvement of infection causing damage to bones, muscles and other deeper tissues [4]. Early institution of antifungals/antibiotics can lead to complete cure of this condition. Late and neglected case require debridement of tissues and sometimes even amputation of limbs along with pharmacotherapy.

In this article we have discussed a case of actinomycetoma caused by nocardia species.

This patient was diagnosed as cutaneous tuberculosis in some other institute and was treated with anti-tuberculosis therapy. He was admitted in our institute MVJ Medical College and Research Hospital in a rural area of outskirts of Bangalore where he was instigated, and treated initially with amikacin and dapsone; later showed excellent response and complete clearance of lesions with injection crystalline penicillin followed by oral phenoxymethyl penicillin.

Case report

A 23 years old male patient presented with a swelling and multiple discharging sinuses over the left flank one year duration. He had undergone a skin graft 3 years back at the same site after he met with a road traffic accident and sustained a wound in the region. About six months after the skin grafting patient developed swelling and discharging sinuses in the region. His condition was diagnosed as cutaneous tuberculosis in another institute and he was placed on anti-tuberculous therapy for 6 months with no improvement of his condition. Details of the investigations prior to initiation of anti-tuberculous therapy were not available with the patient. On examination, an area of atrophic dyspigmented puckered scarring with nodules and multiple discharging sinuses measuring 15cms x 7cms was present on the left flank. Some sinuses showed seropurulent or bloody discharge while others were covered with hemorrhagic crusts (Fig.1,2). However, no granules were available on expressing the sinuses. Some areas showed moderate tenderness. Systemic examination was normal.

On investigating, his routine blood counts, urine routine examination, biochemical parameters and chest x-ray did not reveal any abnormalities. X-ray of flank was normal.

Ultrasound examination of the region showed multiple subcutaneous and intramuscular abscesses in the left paraspinal area. KOH mounting of the discharge obtained from the lesion did not reveal any fungal elements. Gram staining and modified Ziel-Neelsen staining showed gram positive and weakly acid-fast bacilli. Discharge was sent for culture for fungi, bacteria and mycobacterium species.

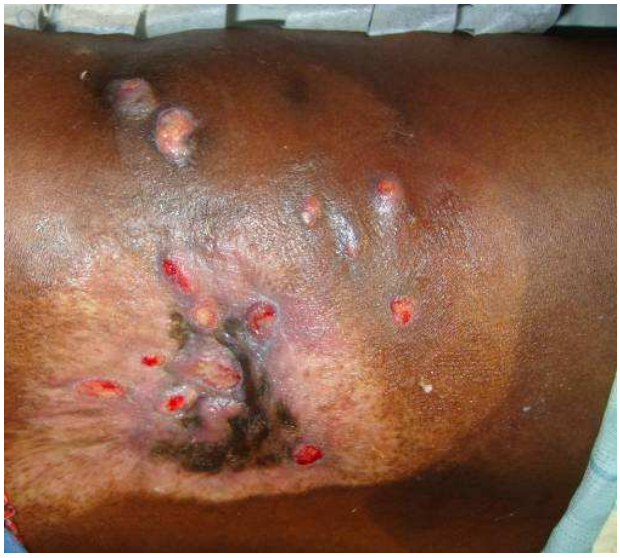

Figure 1. Clinical photograph showing nodules and sinuses

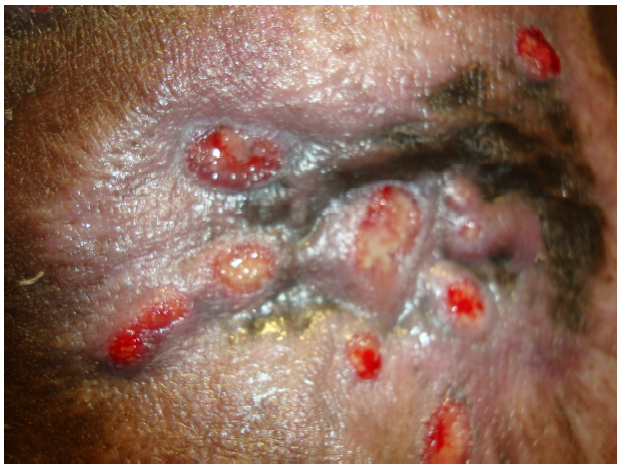

Figure 2. Clinical photograph showing close-up view of lesions

Punch biopsy was performed under local anesthesia and the specimen sent for histo-pathological examination revealed granulation tissue consisting of lympho-histiocytes, polymorphs and fibrous mass. Granulation tissue showed foci of pinkish masses containing filamentous bacteria (Fig.3). Gram staining showed gram-positive colonies surrounded by homogenous pink material (Splendore-Hoeppli reaction) (Fig.4) and staining with modified Ziehl-Neelsen stain showed weakly acid fast bacilli (Fig.5). Meanwhile, culture of the discharge from sinuses yielded a growth of chalky white colonies of *Nocardia spp* (Fig.6). Culture of the biopsied tissue also yielded similar picture, and sensitivity testing of the culture yielded sensitivity to amikacin, gentamycin, tobramycin, erythromycin, cefotaxim and amoxycillin-clavulanic acid and resistence to co-trimoxazole.

With the sensitivity pattern available, patient was started on antibiotics on lines of modified welsch regimen with some modifications, as the culture yield was resistant to co-trimoxazole. Patient was placed on inj. Amikacin 500 mg bid along with tab dapsone 100 mg daily and anoxycillin-clavulanic acid 500/125 mg daily. This combination treatment was given for 3 weeks followed only dapsone and amoxicillin-clavulanic acid

for 2 weeks. Three such cycles were given followed by daily continuous doses of oral dapsone and amoxycillin-clavulanic acid. Patient showed partial healing of the sinuses and decrease in size of nodules as long as he was on cycles of injection amikacin; however the improvement was not sustained while he was switched to oral medication. Fresh sinuses and nodules started appearing and he patient developed tenderness in the region. Attempt at fresh culture for sensitivity testing failed.

We decided to place the patient on benzyl penicillin and started him on 1.2 mega units of benzyl penicillin intravenously 6th hourly, while continuing the dapsone and stopping amoxicillin-clavulanic acid. In four weeks of this therapy patient showed excellent response with near healing of the sinuses and nodules. We decided to continue therapy with oral phenoxymethyl penicillin (pentids) at the dose of 800 mg four times daily while continuing dapsone. Patient has completed 4 months of oral phenoxymethyl pencillin and dapsone. The lesions showed complete resolution with healing of all nodules and sinuses (Fig.7,8).

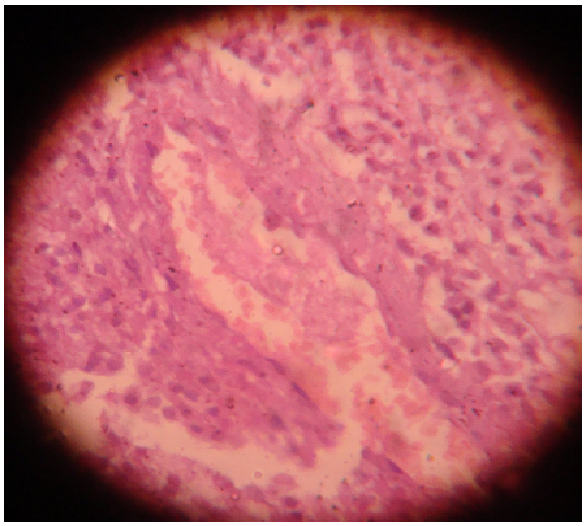

Figure 3. HPE showing grains surrounded by inflammatory cells (H&E Staining, 400 X)

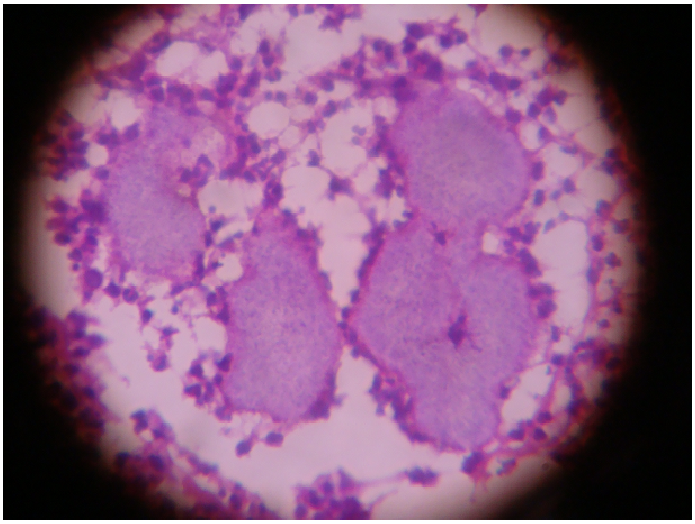

Figure 4. HPE showing grains surrounded by homogenous pinkish material (Splendore-Hoeppli reaction) (Gram staining, 400X)

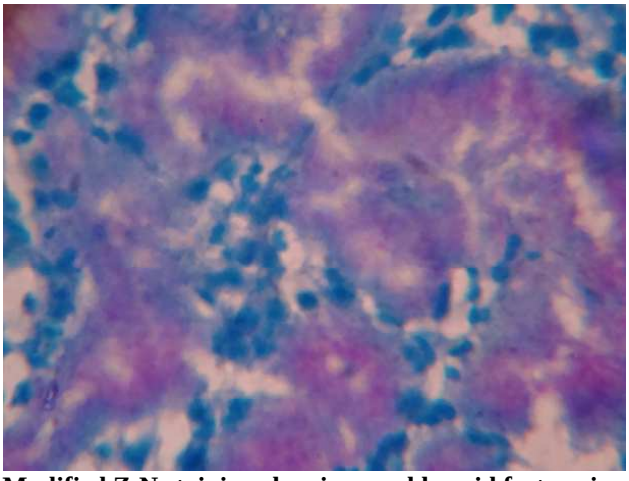

Figure 5. Modified Z-N staining showing weakly acid fast grains (400X)

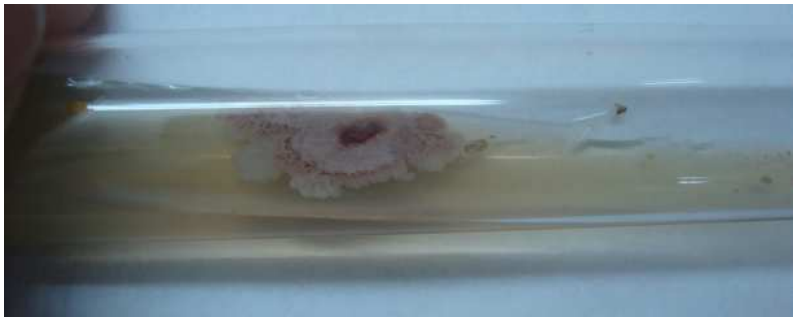

Figure 6. Nocardia colonies in culture tube

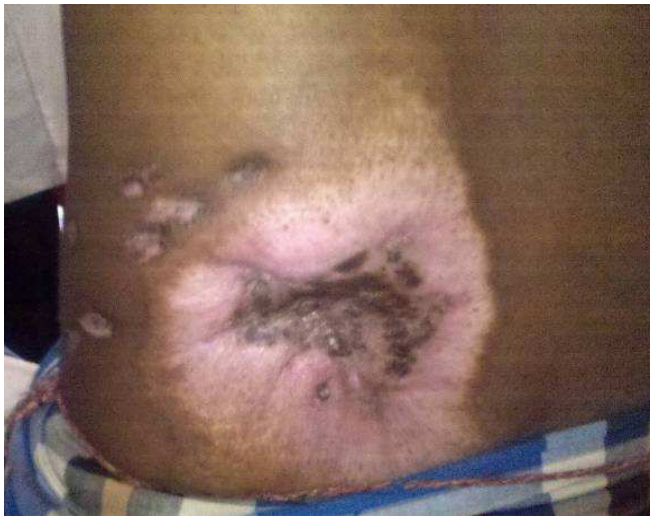

Figure 7. Clinical photograph showing healed area after therapy

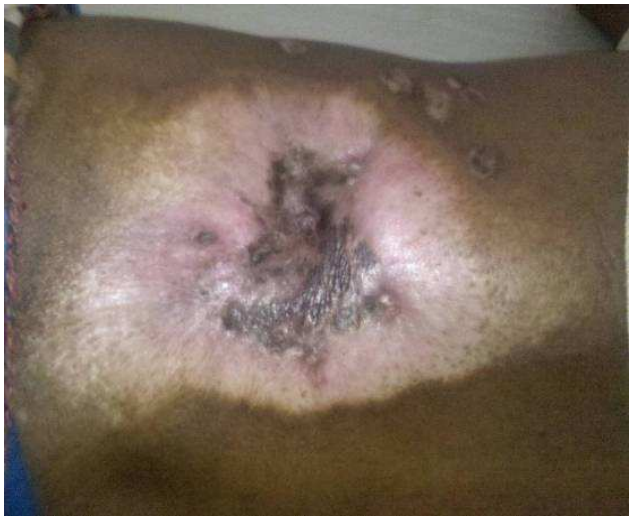

Figure 8. Close-up view of healed area

Discussion

Mycetomas are infections of the skin and deeper tissues caused by fungi or filamentous bacteria and are characterized by tumefaction, nodules and discharging sinuses. Causative organisms are saprophytes present mostly in soil and on plants. From these sources, the organisms are implanted subcutaneously, usually by penetrating injury. Since trauma favors infection, most lesions are on the foot and lower leg, but they may occur anywhere on the body [7].

The grains obtained from the discharging sinuses are aggregates of microcolonies of the organisms. 'Eumycetomas are responsible for about 40 % of all the cases while 'actinomycetomas' are responsible for rest 60% [9].

Actinomycetomas (bacterial mycetomas) are caused by aerobic actinomycetes belonging to the genera *Nocardia* , *Streptomyces* and *Actinomadura* .

Eumycotic mycetomas are caused by a variety of fungi, the most common ones being *Madurella mycetomatis, Pseudallescheria boydii and Acremonium* species [10]. Although reported from all over the world, they are common in tropical and subtropical regions where people walk barefoot. In India, actinomycotic mycetoma is more commonly encountered than eumycotic mycetoma. The genus nocardia consists of aerobic, gram-positive, weakly acid fast bacteria showing filamentous structure.

They are found in soil, wood and water and also in decaying matter. In India the first report on *N. brasiliensis* infection appeared in 1964 [5].

In our case mycetoma occurred at an unusual site (left flank) where skin grafting was done in the region following an accidental injury. *Nocardia spp* was obtained on culture. We started the patient on treatment on lines of regimen described by Welsh [8], with a modification; dapsone was used instead of co-trimaxazole as the organism was resistant to the latter. Since the initial response was not sustainable and fresh nodules appeared at the site after switching the patient from injectable amikacin to oral dapsone and amoxicillin-clauvulanic acid, we started him on intravenous benzyl-penicillin for four weeks with excellent response. This was followed by oral phenoxymethyl penicillin and the healing was near complete.

High index of suspicion, early diagnosis and treatment is necessary in case of mycetomas as delay in therapy may lead to involvement of deeper tissues including bones which can be of disastrous consequences like amputation of limbs and deformities. There are no universally accepted standard treatment protocols since the different species are involved and antibiotic sensitivity varies; treatment should be individualized [11]. Although many antibiotics including the latest ones like imipenam and meropenam [12] are being used for treating actinomycetomas, penicillin, in both injectable and oral forms, can be considered in treating actinomycetomas considering its safety, efficacy and the cost incurred to the patient.

Conclusion

We have reported here a case of nocardia mycetoma occurring at an unusual site. Mycetomas usually occur at the site exposed to penetrating injuries, mostly occurring in tropics and sub-tropics. Early diagnosis and pharmacotherapy remains the mainstay of treatmet. In this case, mycetoma occurred at an unusual site and responded well to injection crystalline penicillin followed by oral phenoxymethyl penicillin.

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OUR DERMATOLOGY Online

JADWIGA SCHWANN AND HER SYNDROME

JADWIGA SCHWANN I OPISANY PRZEZ NIĄ ZESPÓŁ CHOROBOWY

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Abstract
Jadwiga Schwann was a dermatologist from Poland. In the 1960s, Schwann reported a rare congenital genodermatosis. This syndrome is characterized by knuckle pads, leukonychia, palmoplanter keratoderma and sensorineural deafness. This report sheds light on Schwann and the syndrome that bears her name.

Streszczenie
Jadwiga Schwann była dermatologiem z Polski. W 1960 roku Schwann odnotowała rzadką, wrodzoną genodermatozę. Zespół ten charakteryzuje się objawem knuckle pads, leukonychią, rogowcem dłoni i stóp, głuchotą czuciowo-nerwową. Raport ten rzuca światło na J. Schwann i zespół objawów, który nosi jej imię.

Key words: Jadwiga Schwann; congenital genodermatosis; Bart - Pumphrey syndrome
Słowa klucze: Jadwiga Schwann; congenital genodermatosis; Bart - Pumphrey syndrome

Jadwiga Schwann was a dermatologist from Poland. Among her contributions to dermatology, she is credited for describing a syndrome, in German and Polish languagues [1,2]. This syndrome appeared latter in English literature by Robert S. Bart (Dermatologist) and Robert E. Pumphrey (Otolaryngologist) [3]; both from USA, and so the syndrome was then known as Bart - Pumphrey syndrome [4-10].

Schwann syndrome is cited in the Online Mendelian Inheritance in Man [10], as knuckle pads, leukonychia, and sensorineural deafness (OMIM 149200) .It is mapped to, Gene map locus: 13q11-q12 .It is a rare condition, with which few families are affected worldwide [4-10].

It is characterized by knuckle pads, leukonychia, palmoplanter keratoderma (PPK) and sensorineural deafness. However, this syndrome has a considerable phenotypic variability. The clinical features of this syndrome partially overlap with Vohwinkel syndrome and Keratitis-ichthyosis-deafness syndrome [5].

Bart and Pumphrey reported this autosomal dominant condition in a 6-generation family[3].

They disputed whether this complex phenotype could be a monogenic defect with pleiotropic expression [3].

A family reported by Crosby and Vidurrizaga [9] established that keratosis palmoplantaris, probably developing only in older affected persons, is part of the syndrome.

In a multigeneration Polish family with Bart - Pumphrey syndrome, Richard et al [5], reported a novel nonconservative missense GJB2 mutation, segregating with the disorder.

Schwann initially described this condition in families from Poland. Subsequently, cases were also reported from other parts of the world [4-10]. Similar to the condition described by Schwann ; a kindred in which many members had knuckle pads, leukonychia, and deafness due to a lesion of the cochlea, was reported by Bart and Pumphrey [3]. Keratosis palmaris et plantaris was present in some. Male-to-male transmission was thought to have occurred in 2 instances. The presence of leukonychia and the absence of digital constrictions appear to distinguish this disorder from the one listed as 'deafness, congenital, with keratopachydermia and constrictions of fingers and toes' (i.e., Vohwinkel syndrome).

The syndrome is best known currently as Bart - Pumphrey syndrome [4-10].

Although the publication of Jadwiga Schwann on this syndrome [1,2], preceded the publication of Bart and Pumphrey [3] by four years.

Jadwiga Schwann, born in Poland, was precocious. She was working in Szczecin, which is the capital city of the

West Pomeranian Voivodeship in Poland. It is the country's seventh-largest city and the largest seaport in Poland on the Baltic Sea.

The first dermatology researches done in Szczecin, were carried out by Schwann.

Schwann published several papers in dermatology in German and in Polish [11-22]. He has written on different topics in dermatology including mycology and occupational skin diseases [11-22].

I believe that the misnomer Bart - Pumphrey syndrome should be corrected and the syndrome knuckle pads, leukonychia, and sensorineural deafness, should be credited to the right person who reported it first, and should be referred to it as Schwann syndrome.

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JADWIGA SCHWANN AND HER SYNDROME

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Schwann syndrome, also known as Bart-Pumphery syndrome, is an autosomal dominant genodermatosis characterized clinically by knuckle pads, leukonychia and sensorineural deafness and genetically by mutation in *GJB2* gene encoding a gap junction protein, connexin 26. Because Jadwiga Schwann reported this condition in 1963 in German and Polish languages 4 years earlier than the article reported by Bart and Pumphery in N Engl J Med, this disease should preferentially be called as Schwann syndrome. Jadwiga Schwann was a dermatologist in West Poland, who published many articles for both research and clinical topics in the early stage of Polish dermatology.

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ONYCHOGRYPHOSIS

ONYCHOGRYPHOSIS

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Conflicts of interest: None

Onychogryphosis a disorder that is characterized by the hypertrophy and excessive curving of the nails [1], also known as "Ram's horn nails [2] is a hypertrophy that may produce nails resembling claws or a ram's horn, possibly caused by trauma or peripheral vascular disorders, but most often secondary to neglect and failure to cut the nails for extended periods of time and is most common ly seen in the elderly [3]. It can affect the fingernails and toenails [1] are seen in later life especially in the big toe-nail is severely disorted, thickened and interferes with the wearing shoes. May be caused by poor blood circulation to the feet, diabetes, nutritional deficiencies and tight-fitting shoes, foot anomalies such as hallux valgus, old age, uricaemia, ichthyosis, psoriasis, onychomycosis, local injury to the nail apparatus, repeated minor trauma caused by footwear, pathology in the peripheral nervous system, syphilis, phemphigus and variola [1,4]. Etymology: Gk, *onyx* + *gryphein*, to curve, *osis*, condition [5] Onychogryphosis may rarely occur as a development abnormality but is usually acquired, its irregular surface is marked by transverse striations, sometimes this nail is oyster like. Appears in cases of self-neglect and is often seen in tramps and senile dementia. Idiopathic forms are acquired and hereditary [4].

Conservative treatment is especially useful in feet at high risk patients with vascular disease and diabetes and trimming the thickened nail by means of an electric drull and burrs and the removal of subungual keratoses, chemical nail destruction using 40% or 50%, avulsion of the nail plate with surgical destruction of the matrix with phenol or the CO_2 laser, if the blood supply is good.

Figure 1 Onychogryphosis as Ram's horn nails

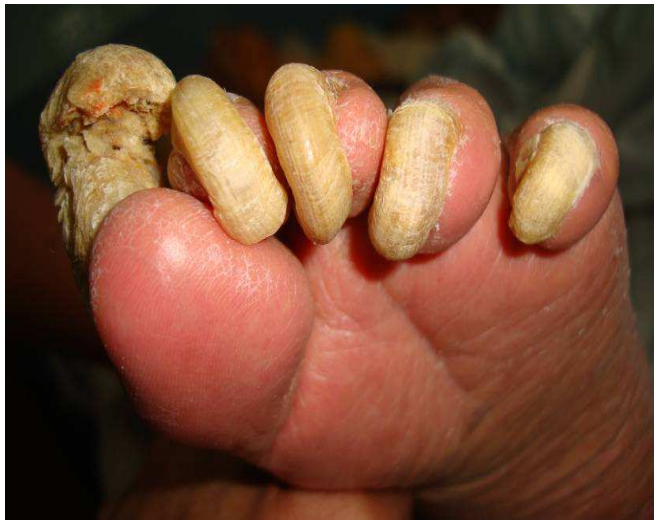

Figure 2 Nails thickened in onychogryphosis

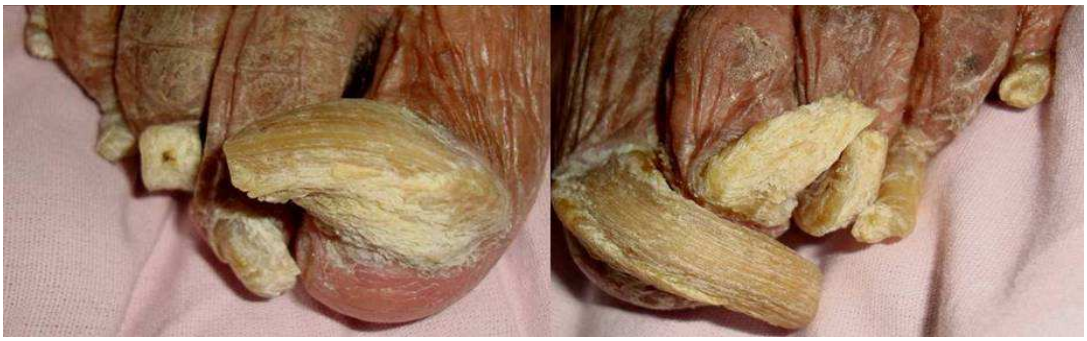

Figure 3 Nails thickened in onychogryphosis

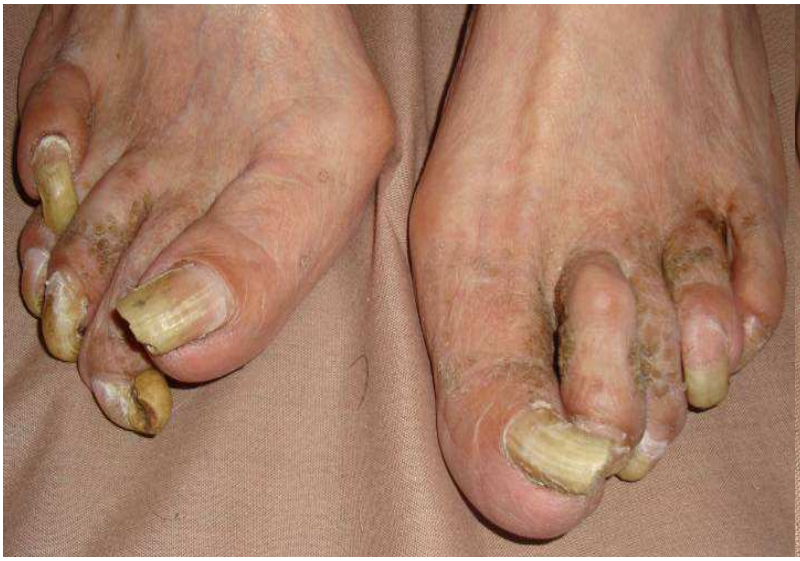

Figure 4 Onychogryphosis in a female patient 80 years old

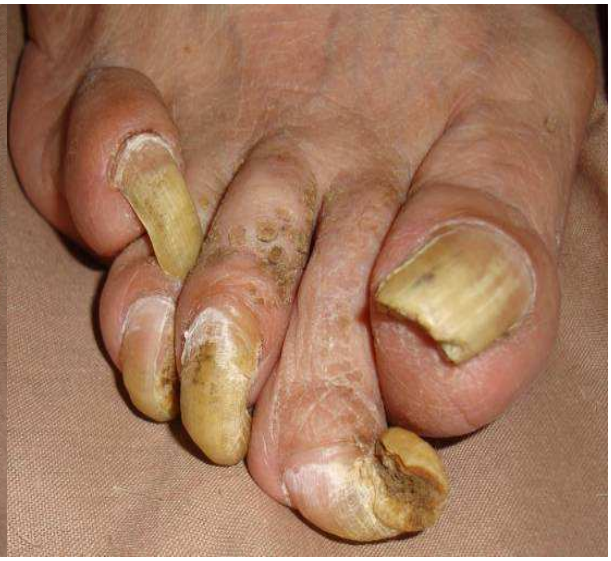

Figure 5 Close up of the nail disease

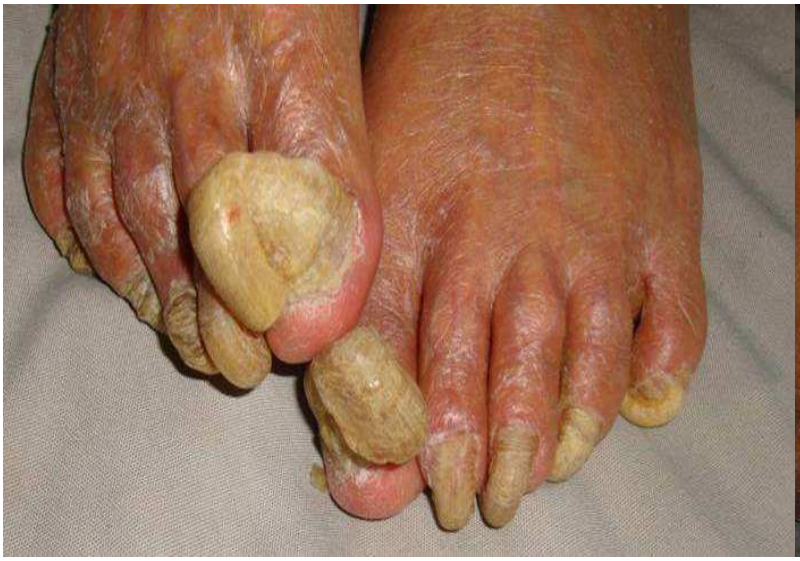

Figure 6 Onychogryphosis of the toenails in male patient 89 years old

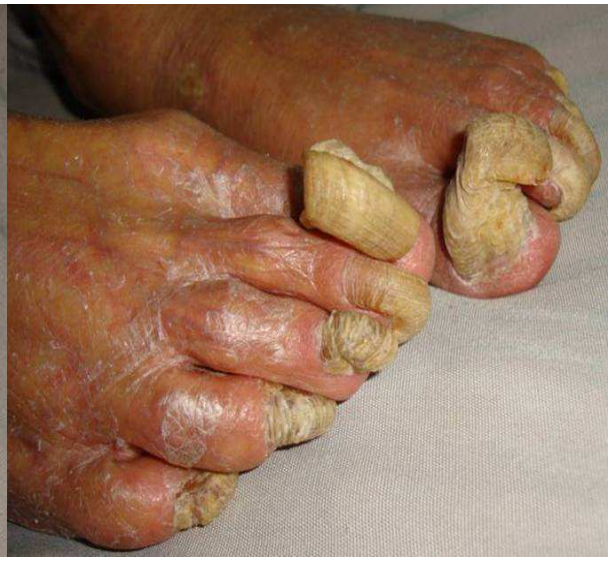

Figure 7 Onychogryphosis of the toenails lateral view

Figure 8 Transverse striations in onychogryphosis

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GIANT CYLINDROMA
GIGANTYCZNY CYLINDROMA

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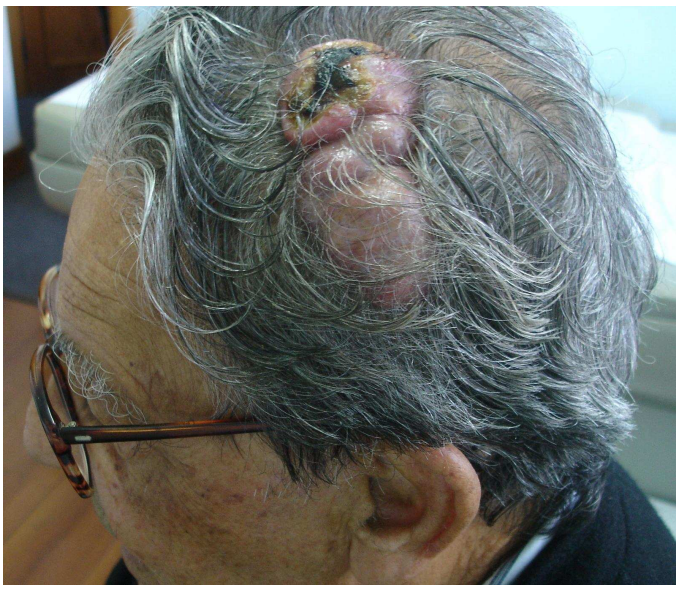
Conflicts of interest: None

Male, age 73
Multiple telangiectatic skin coloured nodules coalescents on the scalp forming a giant mass and the patient refers slow increase in size with the passage of time.



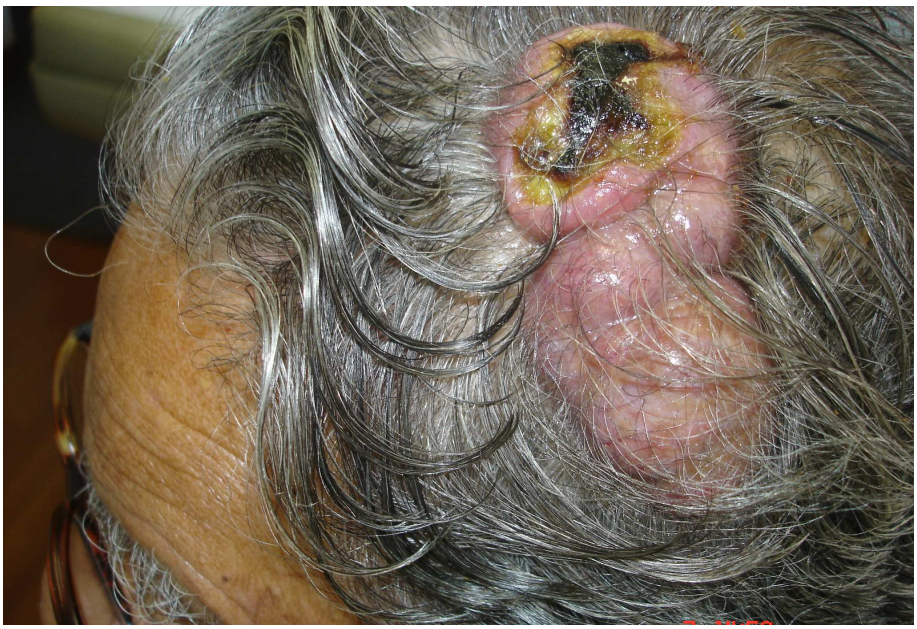


Figure 1, 2. Giant cylindroma

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POST ACNE HYPERPIGMENTATION: A BRIEF REVIEW

HIPERPIGMENTACJA POTRĄDZIKOWA: KRÓTKI PRZEGLĄD

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Conflicts of interest: None

Sir,

Postinflammatory hyperpigmentation, or PIH, is the medical term given to discoloration of the skin that follows an inflammatory wound. It is the skin's natural response to inflammation. PIH presents itself as a flat area of discoloration on the skin (macule) ranging from pink to red, purple, brown or black, depending on skin type and depth of the discoloration. PIH is characterized by an acquired increase in cutaneous pigmentation secondary to an inflammatory process. Excess pigment deposition may occur in the epidermis or in both the epidermis and the dermis [1].

PIH is very common among acne sufferers. It can occur in all skin types, although it is more common in darker skin types. It affects both men and women equally. PIH is not a true scar. Postinflammatory hyperpigmentation may be a sequela of conditions such as acne, allergic reactions, drug eruptions, papulosquamous disorders, eczematoid disorders, and vesiculobullous disorders etc [2,3].

PIH is caused by 1 of 2 mechanisms that result in either epidermal melanosis or dermal melanosis. The epidermal inflammatory response results in the release and subsequent oxidation of arachidonic acid to prostaglandins, leukotrienes, and other products. These products of inflammation alter the activity of both immune cells and melanocytes. Specifically, these inflammatory products stimulate epidermal melanocytes, causing them to increase the synthesis of melanin and subsequently to increase the transfer of pigment to surrounding keratinocytes. Such increased stimulation and transfer of melanin granules results in epidermal hypermelanosis. On the contrary, dermal melanosis occurs when inflammation disrupts the basal cell layer, causing melanin pigment to be released and subsequently trapped by macrophages in the papillary dermis, also known as pigmentary incontinence [1].

In case of Acne, papules and pustules, infection may spread to deep skin layer called dermis. Infected area produces more melanin than normal causing unusual darkness. Thus, infection of hair follicles and sebaceous glands are the real causes of Hyperpigmentation. In most cases, if acne is not severe, it does not leave Hyperpigmentation. Squeezing and popping the pimples also produce Hyperpigmentation. Sun exposure is a leading cause of acne and Hyperpigmentation. Melanocytes are activated by sun light (ultra violet rays) to produce excessive melanin [4].

PIH will fade away over time, even without treatment. It can take three to 24 months for PIH to fully fade, although in some cases it may take longer. The length of time it takes for PIH to fade depends on how dark the PIH macule is compared to skin tone. The bigger the contrast between the macule and natural skin tone, the longer it will take to fade.

There are various treatment options available to help fade postinflammatory hyperpigmentation more quickly [5]. However, acne should be under control before beginning any treatment for PIH. Otherwise, each new pimple could cause another PIH macule, reducing the effectiveness of treatment.Whatever treatment option, improvement will take time of months rather than weeks.

Firstly - avoid sun exposure

Ultra Violet light can cause hyper-pigmented areas to darken further and thus prolong them. Use non-comedogenic facial moisturisers or facial sunscreens which contain a high SPF of at least 15+.

Topical Treatments for PIH

Typically treatments for PIH bleach pigment OR block pigment formation OR accelerate the rate of exfoliation OR a combination.

Bleachs pigment OR block pigment formation
Hydroquinone
Kojic Acid
Benzoyl peroxide
Retinoids
Azeliac Acid
Steroids

Accelerate the rate of exfoliation
The Tape Method of Exfoliation
The Vinegar Method of Exfoliation
Alpha Hydroxy Acid (i.e. Lactic Acid, Malic Acid, Fruit Enzyme etc.)
TCA, GA, SA chemical peels
Mandelic Acid

Non-topical Treatments for PIH

Microdermabrasion and non ablative lasers are being used specifically for treating pigmentation problems. These treatments may be unsuitable for people who suffer from active acne. Laser treatment is generally expensive and carries a risk of causing new acne, PIH and scarring.

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Conclusion

Post inflammatory Hyperpigmentation usually occurs after severe acne has healed. It may take years to disappear if acne is not properly treated immediately. Squeezing the acne spread infection up to dermis. The deeper the infection, the darker the pigmentation will be. Vinegar is the safest and most effective natural treatment for Hyperpigmentation. With all topical and laser treatments for PIH there is a some risk of causing new outbreaks, new pigmentation problems and possibly even new scarring. Risk of these occurences will probably grow with increasing strength or invasiveness of topical or laser procedures. Some treatments are NOT suitable for people with active acne, sensitive skin or darker skin tones. There is no single treatment that works for everyone. The effectiveness of each treatments varies and treatments may have to be used in conjunction with each other.

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AMNIOTIC BANDS WITH INFANTILE DIGITAL FIBROMATOSIS

ZESPÓŁ PASM OWODNIOWYCH Z DZIECIĘCĄ FIBROMATOZĄ PALCÓW

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A Newborn baby presented with deformed digits of both hands since birth. Baby was born as full term normal baby of weight 2.2kg to non consanguineous parents. No history of any medications taken or infections during antenatal period. Mother's. HIV and VDRL tests were non-reactive. There was no family history of similar illness. On examination child was healthy and alert. Vitals were WNL. Both hands showed multiple constriction bands over fingers with loss of distal phalanx of some digits. Portion of digit distal to bands showed diffuse swelling. There was a firm nodule of about 5mm diameter was present on right ring finger and left index finger. No other cutaneous or systemic abnormalities were observed. X-ray and Ultrasound scan of digital nodules revealed fibrous nature of digital nodule. Biopsy or FNAC of nodule could not be done due to lack of consent from parents. Child was referred to orthopaedic surgeon for release of adhesion bands and child is under regular follow up for nodular lesion which was diagnosed as Infantile Digital Fibromatosis clinically and confirmed by USS.

Amniotic bands are congenital constriction bands which occur due to rupture of amniotic membrane which happens usually before 12 weeks of gestation [1]. Small strands of amnion encircle developing structures commonly digits causing constriction bands (pseudoainhum) pseudosyndactyly, auto amputation or if occlusion is partial leading to distal lymphoedema [2]. Large bands can cause decreased foetal movements which can be detected in utero by USS [3]. Bands in ankle joint can cause club foot or if it is in trunk can cause scoliosis. Facial clefts in association with amniotic bands are reported [4]. No two affected babies will have exactly the same features and there is no single feature that occurs consistently in all cases. Examination of placenta may reveal strands of amnion rolled up at the base of placenta. Results of amnion rupture are external so no internal anomalies are associated. Constriction bands can also be seen due to external forces like hair or thread. It can occur secondary to diseases like palmoplantarkeratoderma or after infection, trauma and some times seen associated with Michelin tyre baby [5]. Auto amputation occurring in utero has to be differentiated from hypoplasia aplasia and acromelia. Amniotic adhesions are also seen with limb body wall complex defects which is due to different pathomechanism.

Infantile Digital Fibromatosis are rare cutaneous juvenile Fibromatosis [6]. They are also called as Rey tumours as they are first described by Rey in1965. They are usually present at birth as nodules over 3rd to 5th digits or may occur after birth. Histopathologically show paranuclear eosnophilic inclusion bodies inside interlacing bundles of myofibroblasts [7]. Inclusion bodies stain red with massons trichrome stain. Electron microscopically these are actin filaments. Infantile digital Fibromatosis has to be differentiated from conventional fibromatosis. So biopsy is mandatory. Conservative treatment is recommended due to benign nature and spontaneous regression. How ever rapid growth and functional impairment may necessitate surgery. 60% shows recurrence after surgery. Intralesional steroid and bleomycin has been found effective [8].

Association of amniotic bands with infantile digital fibroma has not been reported in literature to our knowledge.

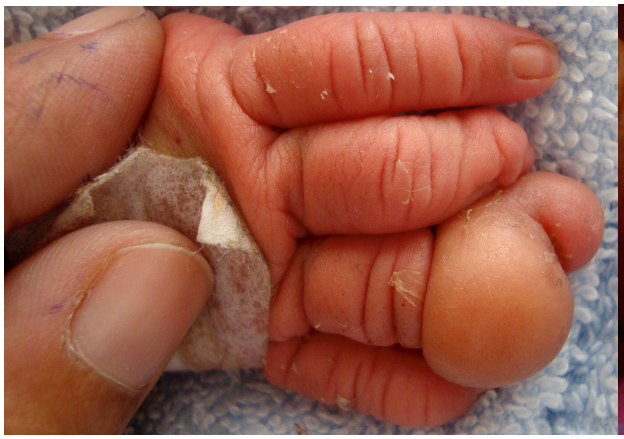

Figure 1. Infantile digital fibromatosis

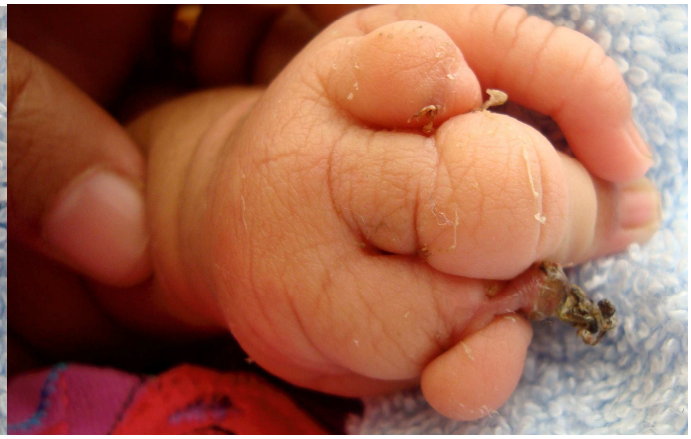

Figure 2. Infantile digital fibromatosis

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AMNIOTIC BANDS WITH INFANTILE DIGITAL FIBROMATOSIS

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Amniotic bands are uncommon conditions that may lead to malformations and fetal-infant death [1]. Amniotic bands are congenital constriction bands from the outer membrane surface into the amniotic cavity, that occur when the amniotic membrane ruptures [2]. As baby develops, amniotic bands can trap extremities and may cause immobilization, constriction or even amputation of the structure [2]. It is not so common for Dermatologists. Even Fitzpatrick's textbook does not have section for it. However, as amniotic bands compress skin directly, dermatologist should have the knowledge of it. In this report, Dr. Ambika and his colleagues showed a complication possibly caused by amniotic bands [2]. They showed that infantile digital fibromatosis was associated with amniotic bands in a newborn baby [2].

Infantile digital fibromatosis is characterized clinically by asymptomatic, flesh-colored and firm nodules affected on the fingers and toes in infants [3]. It may be present at birth. The most affected sites on fingers are the third to fifth digits [3]. It is histopathologically characterized by poorly circumscribed, interlacing bundles of myofibroblasts, in which eosinophilic, Masson trichrome stain-positive paranuclear inclusion bodies are observed [2,3]. These inclusion bodies are the hallmark of infantile digital fibromatosis, differentiating this from other conventional fibromatosis [2,3].

The unfortunate thing is that the patient did not consent to perform skin biopsy of the lesion. Therefore, as mandatory (the authors used this word in the article) biopsy for the diagnosis of infantile digital fibromatosis was not performed, it is possible that the diagnosis may have been wrong.

However, we agree the clinical diagnosis of infantile digital fibromatosis because of the typical and convincing clinical photos they showed in the article and the result of ultrasound scan.

The pathogenesis of infantile digital fibromatosis is unknown. However, it has been suggested the roles of transforming growth factor-□1 mediated differentiation of myofibroblasts from fibroblasts and of bone morphogenetic protein-mediated apoptosis [4,5]. Therefore, it is tempting to speculate that amniotic bands may enhance the expression of these growth factors in the fetus.

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DERMATOLOGY EPONYMS – PHENOMEN / SIGN – LEXICON (E)

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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 (E). 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 E. 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

EAST INDIA SIGN (Furunculus orientalia)

Sharply punched-out ulcer often on the face, feet and the back of the hands, with deep scar. Found among the tea trade routes. Also known as Oriental boil, Aleppo boil, Delhi boil, and Biskra button.

OBJAW WSCHODNICH INDII (czyrakowatość orientalna)

Ostro wycięty wrzód często na twarzy, stopach i rękach z tyłu, z głęboką blizną. Spotykany wśród szlaków handlowych herbaty. Znany również jako Oriental boil, Aleppo boil, Delhi boil, czy Biskra button.

EBOLA SIGN

Rapid viral symptoms accompanied with maculopapular desquamative rash, red eyes, hiccups, and interial or extrernal haemorrhage. Mortality can be 90 percent, caused by the zoonotic Ebola hemorrhagic fever Filoviridae virus.

OBJAW EBOLA

Gwałtowne wirusowe objawy z grudkowo-złuszczającą wysypką, zaczerwienieniem oczu, czkawką oraz wewnętrznym i zewnętrznym krwotokiem. Śmiertelność może wynosić 90%; spowodowane przez odzwierzęcą krwotoczną gorączkę Ebola - Filoviridae wirus.

ECHINOCOCCUS-DISEASE SIGN

Synonym: Hydatid disease. Symptoms depend on the location of the cyst within the body and develop as a result of pressure, leakage or rupture. The most common site for the cysts is the liver. Less commonly brain, lungs and kidneys are affected. Skin lessions: jaundice, Spider angiomas, urticaria, erythema.

OBJAW ZAKAŻENIA BĄBLOWCEM

Synonim: Hydatid disease. Objawy zależą od lokalizacji torbieli wewnątrz ciała i rozwijają się na skutek ciśnienia, wycieku lub pęknięcia. Najczęstszą lokalizacją torbieli jest wątroba. Rzadziej mózg, płuca i nerki. Zmiany skórne: żółtaczka, naczyniaki, pokrzywka, rumień.

ECZEMA SIGN

Eczema of the areola as a sign preceding cancer of the breast. Also known as Paget's eczema sign.

OBJAW WYSPYSKU

Wyprysk w kształcie otoczki jako objaw poprzedzający raka piersi. Znany również jako objaw wyprysku Pageta.

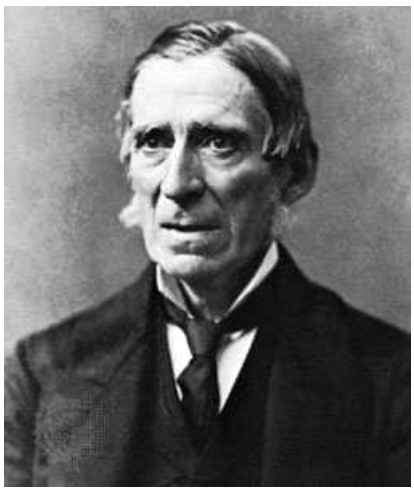

Figure 1. Sir James Paget

Sir JAMES PAGET

English surgeon and surgical pathologist, 1814-1899. 1st. Baronet. Father of modern pathology. The court surgeon Prince of Wales and Queen Victori. Working at St. Bartholomew's Hospital, London (1834–71), Paget discovered (1834) in human muscle the parasitic worm that causes trichinosis. Paget was a professor of anatomy and surgery (1847–52) and was later vice president (1873–74) and president (1875) of the Royal College of Surgeons. He rendered excellent descriptions of breast cancer, an early indication of breast cancer known as Paget's disease, and Paget's disease of bone.
A surgeon of international repute. Among his works are Lectures on Tumours, Lectures in Surgical Pathology, and Clinical.

Angielski chirurg i patolog, 1814-1899. Baronet. Ojciec nowoczesnej patologii. Nadworny chirurg Księcia Walii i Królowej Wiktorii. Pracował w szpitalu Świętego Bartłomieja w Londynie (1834/71), Paget odkrył (1834) w ludzkich mięśni pasożyty, które powodują włośnicę. Był profesorem anatomii i chirurgii (1847/52), a później wiceprezesem (1873/74) i prezesem (1875) Royal College of Surgeons. Opisał doskonałe raka piersi i wczesne objawy raka piersi znane jako choroba Pageta, czy choroby Pageta kości.
Chirurg o międzynarodowej renomie. Wśród jego prac są wykłady na temat nowotworów, wykłady z chirurgii patologicznej oraz klinicznej.

EEC SING, Enterohemorrhagic Escherichia Coli

A zoonotic bacteria found in cattle and humans, causes hemolytic uremic syndrome.Usually acquired thru the ingestion of undercooked ground beef.

OBJAW EEC, Enterokrwotoczny szczep Escherichia Coli

Chorobotwórcza bakteria znaleziona u bydła i ludzi, powoduje zespół hemolityczno-mocznicowy. Możliwość nabycia poprzez spożycie niedogotowanego mięsa zabrudzonego zanieczyszczoną ziemią.

EHRLICH'S SIGN

Human monocytic chrlichiosis or human granulocytic ehrlichiosis, caused by a zoonotic tick borne bacterium. Principle animals are deer, horse, dogs, and rodents found in the USA and Japan. The average reported annual incidence is 0.7 cases per million population. Five species have been shown to cause human infection: Anaplasma phagocytophilum, Ehrlichia ewingii, Ehrlichia chaffeensis, Ehrlichia canis, Neorickettsia sennetsu. The most common symptoms include headache, muscle aches, and fatigue. A rash occurs but is uncommon. Ehrlichiosis can also blunt the immune system, which may lead to opportunistic infections such as candidiasis.

OBJAW EHRLICHA

Ludzka monocytarna chrlichioza lub ludzka granulocytarna ehrlichioza, spowodowana przez odzwierzęce chorobotwórcze bakterie. Zwierzętami z reguły są jelenie, konie, psy i gryzonie występujące w USA i Japonii. Średnią roczną zapadalność odnotowano na 0,7 przypadków na milion mieszkańców. Pięć gatunków okazało się być przyczyną zakażeń u ludzi: Anaplasma phagocytophilum, Ehrlichia ewingii, Ehrlichia chaffeensis, Ehrlichia canis, Neorickettsia sennetsu. Najczęstsze objawy to bóle głowy, bóle mięśni i zmęczenie. Wysypki, są niezbyt często. Ehrlichioza może osłabić układ odpornościowy, co może prowadzić do zakażeń oportunistycznych, takich jak kandydoza.

Figure 2. Amblyomma americanum. Vector Human monocytic ehrlichiosis

Figure 3. Ixodes scapularis. Vector Human granulocytic anaplasmosis

Figure 4. Ixodes pacificus in California Vector Human granulocytic anaplasmosis

PAUL EHRLICH

German physician, 1854-1915. Scientist in the fields of hematology, immunology, and chemotherapy, and Nobel laureate. He is noted for curing syphilis (Salvarsan (arsphenamine, "compound 606") and for his research in autoimmunity, calling it "horror autotoxicus". He coined the term chemotherapy and popularized the concept of a magic bullet. Paul Ehrlich's life and achievements were filmed 1940 in Hollywood by William Dieterle in Dr. Ehrlich's Magic Bullet.

Figure 5. Paul Ehrlich

Nniemiecki lekarz, 1854-1915. Naukowiec w dziedzinie hematologii, immunologii i chemioterapio, laureat Nagrody Nobla. Znany jest z leczenia kiły (Salvarsan (arsphenamine, "związek 606") i jego badań w autoimmunizacji, nazywano go "horror autotoxicus". Wprowadził termin chemioterapii i spopularyzował pojęcie magicznej kuli. Życie i osiągnięcia Paula Ehrlicha zostały sfilmowane w 1940 roku Hollywood przez Williama Dieterle w „Magic Bullet dr Ehrlicha".

EICHSTEDT'S SIGN

Synonym - tinea versicolor

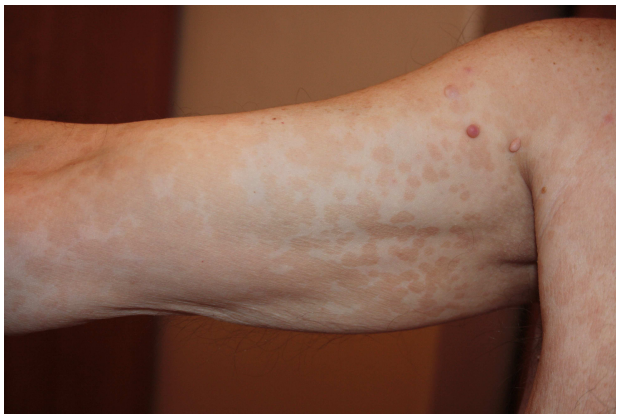

Figure 6. Eichstedt's sign

OBJAW EICHSTEDTA

Synonim - tinea versicolor

KARL FERDINAND EICHSTEDT

German gynecologist and university professor, 1816-1892. Was the son of the General Counsel of the University of Greifswald. He attended high school in Greifswald, earned his doctorate at the Universities of Berlin and Greifswald. First Eichstedt worked in his hometown as a general practitioner. From 1849 he was lecturer, then a professor at the University of Greifswald Obstetric Clinic. Carl Eichstedt is considered the discoverer of the 'Pityriasis versicolor' (1846). Discovered of the contagious nature of pityriasis, who identified a fungus as the cause (1846), later named by Robin C. - Microsporon furfur (1853).

Niemiecki ginekolog i profesor uniwersytecki, 1816-1892. Był synem Radcy Prawnego Uniwersytetu w Greifswaldzie. Uczęszczał do gimnazjum w Greifswaldzie, uzyskał doktorat na uniwersytecie w Berlinie i Greifswaldzie. Na początku pracował w swoim rodzinnym mieście w charakterze lekarza ogólnego. Od 1849 był wykładowcą, następnie profesorem na Uniwersytecie w Greifswaldzie w klinice położniczej. Uważany jest za odkrywcę "łupieżu pstrego" (1846). Odkrył zakaźny charakter łupieżu pstrego i przedstawił

grzyby jako jego przyczynę (1846), później nazwane przez Robina C. - Microsporon furfur (1853).

Figure 7. Karl Ferdinand Eichstedt

EIGHT DAYS SIGN

A signs of tetanus neonatorum at it usually appears at eight days due to umbilical sepsis. In Africa and the West Indies it is a ceremonial custom to place dung on the umbilical cord of newborns.

OBJAW ÓSMEGO DNIA

Objawy tężca noworodków, pojawia się zazwyczaj ósmego dnia z powodu posocznicy pępowinowej. W Afryce i Indiach Zachodnich jest uroczysty zwyczaj umieszczania błota na pępowinie noworodków.

Sir WILLIAM OSLER

Canadian physician, 1849-1919. 1st Baronet. He attended the Toronto Medical College and in 1872 received his M.D. degree from McGill University in Montreal. He studied in London, Berlin, and Vienna. Osler was elected a fellow of the British Royal College of Physicians in 1883. Osler adapted the English system to egalitarian American principles by teaching all medical students at the bedside. William Osler's book, The Principles and Practice of Medicine, first published in 1892, supported his imaginative new curriculum. Another eponyms: Osler's sign; Osler's nodes; Rendu-Osler-Weber disease; Osler-Vaquez disease;
Osler-Libman-Sacks syndrome; Osler's filaria; Osler's manoeuvre; Osler's syndrome; Osler's triad; Sphryanura osleri.

Figure 8. Sir William Osler

Kanadyjski lekarz, 1849-1919. 1-y baronet. Uczęszczał do Toronto Medical College, a w 1872 roku uzyskał stopień doktora w McGill University w Montrealu. Studiował w Londynie, Berlinie i Wiedniu. Osler został wybrany członkiem brytyjskiego Royal College of Physicians w 1883 roku. Dostosowane angielski system do egalitarnych amerykańskich zasad poprzez nauczanie wszystkich studentów medycyny przy łóżku. Książka Williama Oslera, The Principles and Practice of Medicine, po raz pierwszy została opublikowana w 1892 roku, ukazywała jego wyobraźnię nowego programu nauczania. Inne eponimy: Osler's sign; Osler's nodes; Rendu-Osler-Weber disease; Osler-Vaquez disease; Osler-Libman-Sacks syndrome; Osler's filaria; Osler's manoeuvre; Osler's syndrome; Osler's triad; Sphryanura osleri.

EL NIÑO PHENOMENON

Weather anomalies. The phenomen El Niño that affected Peru at 1998, made possible the growth of copious vegetation in traditionally dry places. Cause is - Paederus irritans, dipterous of the order Coleoptera (Paederus dermatitis, a type of irritant contact dermatitis). The population increases rapidly at the end of the rainy season (November and December) and then rapidly diminishes with the onset of dry weather in January. A rapid increase in their population has been attributed to the increased rains associated with the el Niño phenomenon.

Figure 9. Paederus irritans

ZJAWISKO EL NIÑO

Anomalia pogodowa. Zjawisko El Niño, opisano w Peru w 1998 roku, umożliwiło wzrost obfity roślinności w miejscach tradycyjnie suchych. Przyczyną jest - Paederus irritans, dwuskrzydły owad z rzędu Coleoptera (Paederus dermatitis, rodzaj zapalenia skóry z podrażnienia). Populacji gwałtownie wzrasta pod koniec pory deszczowej (listopad i grudzień), a następnie gwałtownie maleje wraz z pojawieniem się suchej pogody w styczniu. Szybki wzrost ich populacji przypisuje się zwiększonej porze deszczowej związanej ze zjawiskiem El Niño.

ELLIOT'S SIGN

1. Induration of the edge of syphilitic skin lesion.
2. A scotoma extending from the blind spot and made up numerous points or spots (ophthalmology).

OBJAW ELLIOTA

1. Stwardnienie krawędzi syfilitycznych zmian skórnych.
2. Mroczki rozciągający się od martwego punktu i składające się liczne punkty lub punkcików (okulistyka).

GEORGE T. ELLIOT

American dermatologist. 1851-1935.

Amerykański dermatolog. 1851-1935.

ERSATZ CROHN'S SIGN

Violent abdominal pain, coughing, and eosinophililc granulomas in the intestine. This is initiated through the ingestion of undercooked fish, as well squid oad octopus. The disease is caused by the parasitic zoonotic Anisakis roundworm.

OBJAW ERSATZA CROHNA

Gwałtowne bóle brzucha, kaszel i eozynofilowe ziarniniaki w jelicie. Zainicjowane przez spożycie niedogotowanych ryb, a także ośmiornic, kalmarów. Choroba wywoływana jest przez pasożytnicze odzwierzęce glisty Anisakis.

ESKIMO SIGN

The black facial appearance found in some Eskimo women. This is a sign of ceremonial matron tattooing of the face with lamp-black

Figure10. Eskimo sign

OBJAW ESKIMOSA

Czarny wygląd twarzy widywany u niektórych kobiet eskimoskich. Jest to znak uroczystego tatuażu matrona o twarzy z czarną lampą.

EXTINCTION SIGN

Extinction of the eruption over an area of skin about the size of the palm when normal injected intra cutaneously. Also known as Schultz-Charlton reaction.

OBJAW ZANIKU

Zanik zmiany na powierzchni skóry o wielkości dłoni, podczas typowego wstrzyknięcia śródskórnego. Znany również jako reakcja Schultza-Charltona.

WILLY CHARLTON

German physician, 1889-?.

Niemiecki lekarz, 1889-?.

WERNER SCHULTZ

German internist, 1878-1947. Schultz is best known for his classic description of agranulocytosis in 1922. He did his research on anaphylaxis independently of Dale and carried out his in vitro testing using the intestinal muscles of guinea pigs.

Niemiecki internista, 1878-1947. Schultz jest najbardziej znany ze swojego klasycznego opisu agranulocytozy w 1922 roku. Opisał swoje badania o anafilaksji niezależnie od Dale i przeprowadzał badania in vitro używając mięśni przewodu pokarmowego świnek morskich.

EXTRA-FACIAL PHENOMENON

The phenomenon of ‘extra-facial’ lesions in rosacea. Known as rosacea disseminated or extra-facial rosacea.

ZJAWISKO ZEWNĄTRZ TWARZOWE

Zjawisko "zewnątrz-twarzowych" zmian w trądziku różowatym. Określane jako rozsiany lun poza twarzowy trądzik różowaty.

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