'Deep' white superficial onychomycosis due to molds

To the Editor

A 59-year-old man was referred to our out-patient clinic for nail disorders because of a diffuse white discoloration on the nail of his right big toe; the white mark had appeared 1 year before, after nail shedding due to trauma. The affected nail showed several opaque and yellow-whitish areas especially evident on the lateral sides and proximal edge. The nail plate was opaque and fragile with loss of superficial keratin in several zones (fig. 1). Proximal nail plate involvement progressed proximally underneath the proximal nail fold, and the yellow-white discoloration was visible through the overlying transparent cuticle. Gentle scraping of the nail surface with a curette revealed that nail fragility involved even the depths of the nail plate. KOH preparation was positive and cultures grew Aspergillus candidus (fig. 2) on all inocula in three successive nail samplings. A diagnosis of white superficial onychomycosis (WSO) due to Aspergillus candidus was made and we prescribed topical amorolfine nail lacquer to be applied twice a week. Cure of the onychomycosis was observed after 6 months of treatment.

WSO is usually caused by dermatophytes, particularly Trichophyton interdigitale. According to Zaias 5% of WSO are due to non-dermatophytic molds, especially of the genus Acremonium, Fusarium and Aspergillus. Candida sp. may sometimes be responsible. In our experience, WSO due to non-dermatophytes is not frequent, and in addition to the case reported here we have seen three other cases of WSO due to Fusarium sp., all with similar clinical features, consisting of 'deep' nail plate invasion and diffuse involvement of the nail plate surface.

Differential diagnosis with proximal subungual onychomycosis (PSO) is particularly important, since this pattern of nail invasion is frequently shown by molds. In PSO, however, nail penetration starts from the matrix and the superficial nail plate is normal. Superficial spreading of PSO is rare in children, but it may occur because the nail plate is very thin in the first years of life.

WSO with deep nail penetration seems therefore typical for non-dermatophytic molds. A possible explanation for this 'deep' WSO caused by molds of the genus Fusarium and Aspergillus could be the eroding bodies possessed by these fungi. These organs, characteristic of the saprophytic state of fungi, facilitate penetration of the nail plate that would otherwise be a strong barrier against fungal invasion.

Although diffuse and deep invasion of the superficial nail could be an important factor in limiting the success of topical treatment, application of topical amorolfine was successful in our patient.

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*Fig. 1. White superficial onychomycosis due to Aspergillus candidus: large yellow-white opaque areas also involving the proximal nail plate.

*Fig. 2. Lactophenol-cotton blue mount of Aspergillus candidus colony showing globose vesicles with biseriate phialides covering their entire surface.
Multiple keratoacanthomas arising on skin lesions of pseudoxanthoma elasticum

To the Editor
Keratoacanthoma is a common benign cutaneous neoplasm that most often occurs on sun-exposed sites in light-skinned people of middle age or older; it is rare in subjects under the age of 20. It is a rapidly growing tumour that may involute spontaneously within 2–6 months leaving a scar; the lesion is comprised of keratinizing squamous cells arising from the hair follicles. Etiological factors include ultraviolet energy, ionizing radiation, chemical carcinogens, such as tar and mineral oils, trauma, viral, immunologic, and genetic factors.1

A 20-year-old white man was referred to our department for evaluation of nodular growths on the sides of his neck that had developed on previously present skin lesions of pseudoxanthoma elasticum. The lesions had appeared suddenly 2 months before he presented to us and continued to increase after our first examination. He had suffered from itching, burning and tingling upon exposure to sunlight. His medical history revealed that he has had pseudoxanthoma elasticum since he was 13 years old. There was no familial history of pseudoxanthoma elasticum or keratoacanthoma, and no individual history of sunburn reaction.

On physical examination, yellowish plaques were observed on the sides of the neck, but nowhere else on the body. There were about 15–20 papular and nodular lesions in the plaques of pseudoxanthoma elasticum. Both the papular and nodular lesions were dome-shaped and erythematous with a horny plug in the centre (fig. 1). Ocular examination revealed macular degeneration and bands of choroid rupture in the right eye. The man’s cardiovascular system was normal.

Excisional biopsy taken from a nodular lesion evidenced a large, central keratin-filled crater in the epidermis with irregular epidermal proliferations extending downward from the base of the crater into the dermis. In the midreticular dermis there were calcified altered elastic fibers that stained deeply black with Verhoeff’s stain (fig. 2). Step sectioning did not evidence transepidermal elimination of elastic tissue.

Multiple keratoacanthomas have been observed on areas of severe atopic eczema, drug eruptions, miliaria, erythema multiforme, radiodermatitis, weather-beaten skin, sunburn, xeroderma pigmentosum, folliculitis, stasis dermatitis, insect and tick bites, acne conglobata, lichen simplex chronicus, hypertrophic lichen planus, seborrhoeic dermatitis, psoriasiform dyschromia, linear epidermal nevus, split skin graft donor site, healing herpes zoster sites,
pneumococcal and smallpox vaccination sites, nitrogen mustard patch test sites, arterial puncture for measurement of blood gases, surgical scars and thermal burns; however, it has never before been reported as manifesting on the skin lesions of pseudoxanthoma elasticum.

Pseudoxanthoma elasticum is an inherited disorder of the elastic tissue affecting the skin, eyes, and the cardiovascular system. It is characterized by yellow papules and plaques described as resembling ‘plucked-chicken skin’ in flexural areas. Histopathologic changes include fragmentation, clumping and calcification of elastic fibers as well as basophilic swollen and degenerated elastic fibers in the middle and deep reticular dermis. Perforating serpiginous elastosis, numerous acneiform lesions, and chronic granulomatous nodules may coexist in subjects with pseudoxanthoma elasticum. The nodular and papular lesions in our case were clinically similar to those of perforating dermatoses, but we did not observe typical phenomenon for transsepidermal elimination of perforating dermatoses on microscopic examination.

In cases of hypertrophic lichen planus, pathogenetic mechanisms, such as chronic inflammation, chronic corticosteroid therapy and chronic mechanical trauma, had been considered. In our opinion, sun exposure and trauma may play a part in the pathologic mechanism concerning the neck site of pseudoxanthoma elasticum with the subsequent tumour formation. We note that this association can not be coincidental because the multiple keratoacanthomas were located only on the plaques of pseudoxanthoma elasticum. The case reported here would be the first example of multiple keratoacanthoma arising on pseudoxanthoma elasticum lesions.

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infiltrate of IgG and IgA classes in the stroma of the tumour and

commonly seen on the neck or face. The linear form is more

puberty. The plaque form is similar to naevus sebaceus and

also excised and histopathologically confirmed as SCAP (fig. 2).

pathological evaluation of the first lesion, two other papules were

papules and one from the scar tissue (fig. 2). After the histo-

physical examination was normal.

Two punch biopsies were obtained, one from one of the pinkish

papules and one from the scar tissue (fig. 2). After the histo-

pathological evaluation of the first lesion, two other papules were

also excised and histopathologically confirmed as SCAP (fig. 2).

SCAP is a rare adnexal tumour with variable clinical appear-

ance. While the plaque or linear varieties present at birth or dur-

ing infancy, the less common solitary nodular form appears at

puberty. The plaque form is similar to naevus sebaceus and

occurs on the scalp as a hairless area. The linear form is more

commonly seen on the neck or face.1,2 These pinkish-brown

papillary and exophytic lesions, some of which have central

ostium, become more verrucous and nodular during puberty.

The solitary nodular form is seen predominantly on the shoulders,

the axillae and genitocrural areas as an up to 1 cm sized, domed

or umbilicated nodule with a friable or crusted surface.

The clinical appearance and location of the lesions of our

patient were compatible with the linear form of SCAP but the

late manifestation, after puberty, led us to question about the
diagnosis of the formerly excised lesion that might have given us a
clue about the origin of the latter.

Because of non-distinct clinical features, SCAP is generally
diagnosed based on its easily recognizable histopathological
findings. Histologically, one or several cystic invaginations lined
in the upper portion by squamous, keratinizing cells similar to
those of surface epidermis, extend downward from a papillo-
matous epidermis. Numerous villous papillary projections are
seen in the lumina of the lower part of these invaginations. Both
the lower part of the invaginations and the papillary projections
are lined by glandular epithelium. Apocrine glands are promin-
ent below these invaginations.1,2 A fairly dense plasma cell
infiltrate of IgG and IgA classes in the stroma of the tumour and

papillary projections is highly diagnostic.1 Features of both
apocrine and eccrine differentiation have been reported. A rea-
sonable explanation for this may be the development of SCAP
from pluripotential cells capable of transforming into different
types of primary epithelial germ structures.2

Interestingly, SCAP is associated with naevus sebaceus in
40% of cases, and secondary basal cell carcinoma develops in 10%
of these coexistent lesions.3,4 In our case, the first excised un-
known lesion may have been a naevus sebaceus or a linear form
of SCAP, however, because there are no reports in the literature
about late development of SCAP in the region of excised naevus
sebaceus or recurrence of SCAP after surgery and as we have no
biopsy report on the previous lesion, this is only an assumption.
Because the papules were located on the scar, the most probable
clinical diagnosis was basal cell carcinoma and squamous cell
carcinoma. We found no reports in the literature about SCAP
arising on scar tissue, and to our knowledge this represents the
first reported case of SCAP arising on postoperative scar tissue.

Differential diagnosis of especially the plaque and linear forms
on the neck and head should include naevus sebaceus. If the
lesion is umbilicated, it can mimic molluscum contagiosum,
and basal cell carcinoma should be considered in the differential
diagnosis, especially when the lesion is located on the eyelid.1

Because of both clinical resemblance and development of secondary basal cell carcinoma, excision is mandatory. If the
location of the lesion is poorly amenable to excision, such as the
ear region, CO2 laser excision is another alternative.10

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plaque parapsoriasis (PP).

These viruses may be excluded from the list of potential initiators of parapsoriasis. These viruses may be excluded from the list of potential initiators of parapsoriasis.

Acknowledgements

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Absence of Epstein-Barr virus and human herpesvirus-6 in pityriasis lichenoides and plaque parapsoriasis

To the Editor

The term parapsoriasis encompasses a spectrum of inflammatory and neoplastic disorders characterized by asymptomatic, persistent, erythematous and sometimes cutaneous eruptions that are resistant to treatment;1 large plaque parapsoriasis (LPP), small plaque parapsoriasis (SPP), pityriasis lichenoides et varioliformis acuta (PLEV A) and chronic (PLC)2,3 are included in this spectrum of diseases. The association or overlap with malignant lymphoproliferative disorders distinguishes parapsoriasis from purely inflammatory dermatoses. Surprisingly the group of parapsoriasis affections is among the least studied dermatological disorders.

Epstein-Barr virus (EBV) and human herpesvirus-6 (HHV-6) are two lymphotropic herpesviruses. We have previously examined mycosis fungoides for the presence of EBV and HHV-6 by PCR (Erlek et al. accepted for publication by JEADV in 2000). We designed a study questioning the potential role for these viruses in subjects with pityriasis lichenoides (PL) and plaque parapsoriasis (PP).

Fifty-four subjects, 37 males and 17 females, were diagnosed as having parapsoriasis or pityriasis lichenoides in our department between January 1989 and December 1998. The subjects ranged in age from 5 to 71 years. Thirty-three cases were diagnosed as PL (15 PLEV A, 18 PLC) and 21 as PP (seven SPP, 14 LPP). A review of patient records showed that nine of the 14 patients with LPP developed typical plaques of mycosis fungoides within a period of 3 months to 8 years, with diagnostic light microscopic findings and clonally arranged T-cell receptors in follow up skin biopsy specimens.

Fifty-four formalin-fixed, paraffin-embedded skin biopsy specimens were studied for the presence of Epstein-Barr virus and human herpesvirus-6 by using the polymerase chain reaction. All samples showed human β-globulin gene amplification by PCR, indicating the absence of major PCR inhibitors. A 549 base-pair long target region of HHV-6 genome was selected by using the primers (H6.1) 5′-CAC TGG GTA GTT CGG GAG CCC CGA G-3′, and (H6.2) 5′-AAG CTT GCA CAA TGC CAA AAA ACA G-3′. Positive control for HHV-6 was provided by Dr. Toshiomi Okuma. As for EBV, a target region of 239 base pairs was selected on gp 220 gene of EBV by using the primers (gp.1) 5′-GGC TGG TGT CAC CTG TGT TA-3′, and (gp.2) 5′-CCT TAG GAG GAA GTC CC-3′. As a positive control sample we used formalin-fixed, paraffin-embedded Hodgkin’s lymphoma lymphoid node tissue that had been shown to be EBV positive by in situ hybridization. Neither EBV nor HHV-6 was detected in any of the patient samples examined.

The etiopathogenesis of parapsoriasis is unknown. There is ample supportive evidence suggesting that owing to similar clinical, histopathological, immunohistochimical and molecular biologic features, LPP actually represents the patch stage of mycosis fungoides (MF).2,4 However, there is no general agreement regarding SPP and PL. PL is included in the parapsoriasis category, although it is distinct from LPP and SPP. Both PL and SPP, however, have been shown to undergo rearrangement of T-cell receptor genes, indicating a clonal T-cell lymphoproliferative process that is part of the cutaneous T-cell lymphoma spectrum.5,6 A vigorous host immune response might prevent these disorders from evolving to true cutaneous lymphomas, although an outcome has occurred rarely in PLEV A, PLC and SPP.7 PL has been proposed to represent an exanthema associated with viral or rickettsial infection or an immune complex-mediated hypersensitivity vasculitis caused by an infectious agent.5,8 Cases of PL in association with adeno virus infection,9 active toxoplasmosis,10 EBV infection11 and HIV-1 infection12 have been reported, suggesting a role for infections in the etiopathogenesis. Latent herpesvirus infections have been hypothesized to act as chronic persistent antigenic stimuli and lead to the development of cutaneous T-cell lymphoproliferative disorders, however, in the present study, EBV and HHV-6 DNA sequences were found negative in PL and PP. Brice et al.13 investigated 10 PLEV A cases (as a control group for mycosis fungoides) for the presence of EBV and HHV-6 and detected no positive result by PCR and Southern Blot, but their series did not include any cases of PP and PLC.

Our findings indicate that latent or active EBV and/or HHV-6 do not exist in skin lesions of pityriasis lichenoides or plaque parapsoriasis. These viruses may be excluded from the list of potential initiators of parapsoriasis.
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References


An extraordinary case of subcutaneous invasive thymoma

To the Editor

Thymoma is the most common tumour of the anterior-superior mediastinum. Malignant thymomas are rather rare neoplasms with an unique clinical association of autoimmune diseases, particularly myasthenia gravis, and are seldom associated with haematological abnormalities. Although representing two different tumour entities, both thymic carcinoma and invasive thymoma have been widely described as malignant thymomas. The former has been defined as cytologically malignant and the latter as cytologically benign. Approximately one third of thymomas are invasive with displacement and infiltration of mediastinal structures and may extend to the whole mediastinum. Complete surgical resection is the treatment of choice. Advanced or inoperable cases of invasive thymoma require adjunctive radiation and chemotherapy to prevent recurrence of the tumour or to achieve local tumour control. A spread of invasive thymoma beyond the thoracic cavity is unusual. Manifestation of invasive thymoma has been observed in sites such as lung, pleura, thoracic vertebrae, but also in the central nervous system, liver, thyroid and extrathoracic lymph nodes. Manifestation of invasive thymoma in the dermis has not yet been reported.

A 71-year-old man was referred to our department in 1999 for a tumour on the upper trunk. The man had undergone multiple thoracic surgeries, receiving coronary-artery bypass grafting in 1974, 1988 and 1990, due to severe coronary artery disease. Clinical examination upon presentation to us evidenced a preteral node (diameter 5 cm) in the deeper dermis and subcutaneous tissue. The node was subtotally excised in our department. Histopathology revealed thymoma of the mixed type with a typical organoid spectrum, a classification into four subtypes (MIB1) of abundant lymphocytes and a discernable amount of epithelial cells. The number of mitotic figures, however, was scanty to absent. Complete staging included computed tomography of the thorax, which demonstrated a great tumour encompassing the sternum with intrathoracic extension into the anterior mediastinum. No association with myasthenia gravis or other autoimmune diseases was found. Further treatment was performed by cardiac surgeons, however, complete tumour resection was not feasible, because the invasion of multiple mediastinal structures (posterior sternum, lateral pericard, myocard of the right ventricle, aorta ascendens, coronary arteries and bypass grafting) became evident intraoperatively. Subsequently the man was treated by oncologists with six cycles of chemotherapy at 3-week intervals, the ADOC scheme (dorzoxibcic, cisplatin, vincristine and cyclophosphamide). The success of the chemotherapy was indicated by partial remission of the remaining tumour mass.

By definition, thymomas are composed of an admixture of epithelial cells and lymphocytes. Although the proportion of lymphocytes and epithelial cell in thymomas appears to comprise a continuous spectrum, a classification into four histologic subtypes was proposed and has been commonly used: subtype 1, predominantly lymphocytic thymoma; subtype 2, predominantly epithelial thymoma; subtype 3, mixed...
and led to cutaneous manifestation in the presternal region. Probably spread per continuitatem, encompassing the xyphoid, astinal structure and organs. Invasive regional growth of prim-placed thymic tissue is capable of growing and invading medi-the mediastinum. It remains debatable whether ectopically possibly be the cause of parts of thymic tissue being scattered in

...was proposed by Bernatz from thymic carcinomas with malignant cytological character-

...viewed as a spectrum encompassing both thymomas and thymic epithelial neoplasms may be

...classified as a mixed lymphoepithelial thymoma (subtype 3

...tended to assume a plump, round to oval or polygonal configuration with scanty cellular atypia, most with moderate amphophilic cytoplasm and round, uniformly dense nuclei (fig. 1). The basophilic lymphocytes (CD3+) were predominantly small and mature, however some appeared 'stimulated' (corresponding to transformed lymphocytes) with enlarged nuclei and open chromatin. Typical pseudorosette formations were seen.

The clinical course and behaviour of thymomas are unpredictable. For example, a cytologically benign, well-encapsulated thymoma might be cured by simple resection or may recur beyond the thoracic cavity is very rare. Levine and Rosai6 estimated that probably only 15 reported cases qualified as truly metastasizing thymoma. Predominantly the epithelial and mixed lymphoepithelial subtypes of thymoma were reported to be more aggressive forms with higher tendency to invasion and extrathoracic manifestation. Multiple thoracic surgery may possibly be the cause of parts of thymic tissue being scattered in the mediastinum. It remains debatable whether ectopically placed thymic tissue is capable of growing and invading mediastinal structure and organs. Invasive regional growth of primary thymomas is not infrequent. In the present case, thymoma probably spread per continuitatem, encompassing the xyphoid, and led to cutaneous manifestation in the pretraneous region.

lymphoepithelial thymoma; subtype 4, spindle cell thymoma. In the present case histopathological diagnosis was thymoma classified as a mixed lymphoepithelial thymoma (subtype 3 with a mixture of approximately 33% to 66% of each cell type). The majority of epithelial cells (cytokeratin +) tended to assume a plump, round to oval or polygonal configuration with scanty cellular atypia, most with moderate amphophilic cytoplasm and round, uniformly dense nuclei (fig. 1). The basophilic lymphocytes (CD3+) were predominantly small and mature, however some appeared 'stimulated' (corresponding to transformed lymphocytes) with enlarged nuclei and open chromatin. Typical pseudorosette formations were seen.

The clinical course and behaviour of thymomas are unpredictable. For example, a cytologically benign, well-encapsulated thymoma might be cured by simple resection or may recur locally and invade adjacent organs many years later yet retaining bland cytological features. Thymic simple neoplasms may be viewed as a spectrum encompassing both thymomas and thymic carcinomas, nevertheless a distinction between slow growing thymomas with benign cytological characteristics from thymic carcinomas with malignant cytological characteristics was proposed by Bernatz et al.3 and emphasized by Lewis et al.3 True metastasizing thymomas with embolic spread beyond the thoracic cavity is very rare. Levine and Rosai6 estimated that probably only 15 reported cases qualified as truly metastatic thymomas. Predominantly the epithelial and mixed lymphoepithelial subtypes of thymoma were reported to be more aggressive forms with higher tendency to invasion and extrathoracic manifestation. Multiple thoracic surgery may possibly be the cause of parts of thymic tissue being scattered in the mediastinum. It remains debatable whether ectopically placed thymic tissue is capable of growing and invading mediastinal structure and organs. Invasive regional growth of primary thymomas is not infrequent. In the present case, thymoma probably spread per continuitatem, encompassing the xyphoid, and led to cutaneous manifestation in the pretraneous region.
shoulders, proximal arms and thigh muscles that developed to full stiffness. The weakness increased progressively; she experienced heaviness and stiffness in her neck muscles and maseeters; it became impossible for her to get out of bed and to take care of herself. Two new patchy lesions of morphea appeared on the legs. Serum levels of sarcoplasmic enzymes were increased 3–5 fold, creatine kinase 512 E/L (normal range <170 E/L); ASAT 115 E/L; ALAT 84 E/L; LDH 531 E/L. The ANA titre was 1 : 160 and increased to 1 : 1280 only 5 days later. Anti-histone antibodies were positive. EMG showed myopathic changes. The woman fulfilled three of Bohan and Peter criteria for confirmed polymyositis. The treatment with glucocorticosteroids (betamethasone, 7 mg every 10 days) for 1 month did not change the course and evolution of the myositis. Discontinuation of d-penicillamine intake and administration of methylprednisolone (60 mg/24 h i.v.) led to disappearance of the muscle weakness and myalgia within 1 week with normalization of serological enzyme changes and disappearance of ANA antibodies 2 weeks later.

This patient with localized scleroderm and Hashimoto’s disease developed d-penicillamine induced polymyositis, a frequently observed complication of such therapy. Both the number of observed cases of drug-induced myositis and the list of the culprit medicines have increased in recent years. Forty-one cases with d-penicillamine-induced dermatomyositis or polymyositis are reported in the literature. Treatment of rheumatoid arthritis with this medicine is associated with the development of dermatomyositis/polymyositis in 0.2–1.2% of cases, with onset 1.5–6 months after introduction of the drug. Not all investigators agree with this statement, considering that d-penicillamine induced dermatomyositis/polymyositis depends on neither the dose nor the duration of the treatment and this d-penicillamine induced dermatomyositis/polymyositis depends on neither the dose nor the duration of the treatment and this d-penicillamine induced dermatomyositis/polymyositis depends on neither the dose nor the duration of the treatment. The condition is reversible once the drug is withdrawn. The favourable evolution in our patient, specifically cure of clinical manifestations, normalization of serum levels of enzymes and disappearance of ANA antibodies after withdrawal of the drug, suggests that d-penicillamine was responsible for the polymyositis. The dissemination of localized scleroderma and induced polymyositis in our patient with Hashimoto thyroiditis support the hypothesis that d-penicillamine probably could unmask or provoke certain antigens responsible for these diseases in subjects with immune disorders.

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Is demodex folliculorum larger in diabetic patients?

To the Editor
Demodex folliculorum (DF) which is inhabitant of pilosebaceous follicles has been implicated in rosacea, blepharitis, pyotraumatic folliculorum, pustular folliculitis, and skin lesions of immunosuppressed patients under chemotherapy or with AIDS. The larval form of DF is a vermiform mite about 0.9 mm long that inhabits the sebaceous glands of the nose, especially the alae nasi, and, to a lesser extent, of the forehead, chin, and scalp. Clifford et al. found that mites were more abundant in elderly persons, and persons with diabetes.

The aim of the present study was to investigate the role of diabetes mellitus on carriage of DF, and to evaluate size of DF in type 2 diabetic patients.

Twenty-eight male and 26 female (total 54) patients with type 2 diabetes mellitus without any skin disease, average age of 49.5 ± 13 years, and 54 age- and sex-matched healthy controls without any skin disease and telangiectasies were included in the study. Patients with a duration of diabetes under five years were excluded from the study. None of the patients had received any topical or systemic immune suppressive drug except oral hypoglycaemic agents or insulin for the preceding 2 months. The verbal consent of all subjects was taken for the skin surface biopsies. Initially a red 10 mm square was printed on a microscope slide. After placing a drop of cyanoacrylic adhesive on the other side, it was applied to three different facial sites of the skin for 45–70 s. After placing a drop of immersion oil and covered with a coverslip. The
Although it seems quite probable that immunodeficiency mechanisms may play a protective role by limiting mite numbers, there are also conflicting results. Fortan et al. did not find any positive effect of HIV infection on mite density.

The mean mite size in diabetic patients was larger than control group statistically. Demodex feeds on sebum and cellular proteins obtained by epithelial and glandular cell destruction through its own enzymatic activity.

We found a positive correlation between mite and HbA1c percent. HbA1c reflects approximately mean level of blood glucose for last 3 months, therefore, a higher HbA1c level demonstrates a higher mean blood glucose level. This result indicates that increased HbA1c levels may have an effect on the size of mites in diabetic patients. In conclusion; the mean mite density on cheeks is higher in diabetic patients than healthy control group. The mean mite size in diabetic patients is also larger than the control group.

samples were studied microscopically at same magnifications of × 40 and × 100 in all subjects (fig. 1). Only clearly identified DF were counted and measured with ocular micrometer. The mite density at each facial site, and the sizes of the mites were recorded. The most heavily infested facial region was the cheek in both diabetic patients and control group. The mean mite density on cheeks were 9.636/cm² in diabetic patients and 4.571/cm² in the control group; the difference was significant statistically (t = 11.533, P < 0.001).

The mean mite size in diabetic patients was 1.056 ± 0.047 mm in the control group and 0.931 ± 0.047 mm in the control group; the difference between two groups was significant statistically (t = 2.162, P = 0.04). The difference between two groups was not statistically significant in terms of mite positivity and mean mite density at other facial regions (Table 1).

The mean mite size in diabetic patients was 1.056 ± 0.022 mm (180 mites) and 0.931 ± 0.047 mm in the control group; the difference between two groups was significant statistically (t = 11.533, P < 0.001).

However it has been documented that DF is a resident of pilosebaceous follicles with ambiguous pathogenicity, conflicting results have also been published in many articles related to pathogenicity of this mite. In our study, positivity of DF in diabetic patients was similar to the control group but DF density was found statistically higher in the cheek region of diabetic patients than the control group. Immunosuppressive conditions may cause increasing of density of DF. The general effects of hyperglycaemia are probably important, and there may be additional immune disturbances in diabetes mellitus.

Table 1

Table 1 Demodex folliculorum frequencies and mean mite densities at facial regions

<table>
<thead>
<tr>
<th>Site</th>
<th>Diabetic patients</th>
<th>Controls</th>
<th>p</th>
<th>Diabetic patients</th>
<th>Controls</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cheek</td>
<td>24 (38.7%)</td>
<td>14 (35.6%)</td>
<td>NS</td>
<td>9.636</td>
<td>4.575</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Forehead</td>
<td>16 (25.8%)</td>
<td>10 (40.0%)</td>
<td>NS</td>
<td>2.625</td>
<td>2.400</td>
<td>NS</td>
</tr>
<tr>
<td>Nose</td>
<td>22 (35.4%)</td>
<td>16 (25.8%)</td>
<td>NS</td>
<td>4.545</td>
<td>2.500</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS, not significant.

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Bakamjian’s flap

To the Editor

Bakamjian’s flap is the best kind of distant flap for repairing defects on the face and neck. It is composed of skin proceeding from the deltoid area that forms the ‘distal racket’, with a horizontal pedicle of pectoral skin that would be the ‘racket handle’. This flap was designed in 1965 for the total reconstruction of the pharynx after laryngectomy (fig. 1), with the upper margin above the clavicle. Nowadays, the incision is performed below the clavicle to avoid problems when suturing after radical neck node dissection. The lower margin runs parallel to the upper margin at a distance of 12–15 cm, approximately three fingers above the nipple, over the fifth rib. The base of the flap lies within the area overlying the first four intercostal spaces, at a distance of 1–2 cm from the lateral edge of the sternum. In its original description, the flap reached the anterior contour of the shoulder (fig. 2A), sufficient to allow reconstructing loss of substance on the neck. If more length is needed to reach the face without promoting tension, the flap can be extended to the posterolateral line of the shoulder (fig. 2B) and even to the lateral surface of the arm (fig. 2C). The vascular flow of the flap involves two vessel systems: The proximal zone is axial, and it depends on the first four perforating branches of the internal mammary artery that run parallel to the skin and emerge in the intercostal spaces, approximately 1 cm from the sternum; the second perforating branch is the most important. The intermediate or lateral deltoid zone is undetermined or randomized. The distal zone is again axial as it receives the blood flow directly from the cutaneous branches of the thoraco-acromial artery (fig. 3). An essential feature of this vessel system is that the veins are located in the deep subcutaneous tissue and can be lifted along with the flap.

The term ‘delay’ refers to the time necessary to obtain good vascular supply through the pedicle. Although Bakamjian considered that delay might not be necessary with an axial, we always use a delay period except when using the open transposition technique. Thus, two types of deltopectoral flaps can be considered: (i) one that has a suitable length and remains totally axial, and (ii) the flap that needs to be extended, reaching the shoulder or the lateral surface of the arm. This is a randomized flap, as the vascular flow is not directly into the flap axis. When more length is required and randomized zones are involved, or when the patient does not have an adequate vascular supply, it is necessary to delay or tube the flap. The delay may be short and accurate if the whole flap is incised, and only the infraclevicular triangle is elevated from the external middle of the upper edge, in order to cut and close the cutaneous branches of the thoraco-acromial artery. Such a flap can be used from 1 week to 10 days later.

The evolution of the colour of the skin is essential to know whether or not circulation in the flap is adequate. Once the flap is cut and replaced in its original position (first delay), the colour is alabaster white, and only after a few days does it become slightly pink. Oedema is never observed in axial flaps, but it may occur if more length is obtained at the expense of circulation, or when a randomized zone is involved. Oedema can be secondary to compression. The colour and evolution of the skin are complementary in assessing circulation in the flap. However, if progress is observed in the perioperative period, it is not necessary to change the flap to a randomized one, or to perform any other vascular intervention.


arise in distal zones with a random pattern. The skin must be soft and smooth to touch, indicating that lymphatic and venous drainage are adequate. Then, 15–21 days later, the flaps can be again undermined and transposed to cover the defect.

There are many different indications for such flaps, and some require different preparations. Naturally, we are interested in cases were there is much face and neck substance loss after excision of malignant skin tumours. Bakamjian’s flap can be used to repair defects that reach the zygomatic arch.1,4 There are several techniques, and which one depends on the area to be covered: open transposition, transference via flap tubing, pedicle de-epithelialization and reaching the defect under a bridge of normal skin, folding the flap, waltzing migration of the proximal portion of the flap, inverse tubing, application of two dectopectoral flaps and a combination of Bakamjian’s flap with another flap.

Using one or another of these technical variations we have performed surgery using 208 Bakamjian’s flaps, 147 were done by open transposition, which is the most accurate technique.5

The results were excellent in all cases except one involving an obese 82-year-old woman who needed a long flap reaching the lateral surface of her arm, but who suffered from distal venous necroses that required excising 6 cm from the outer portion and then covering the area with a split-thickness skin graft. In two lateral surface of her arm, but who suffered from distal venous necroses that required excising 6 cm from the outer portion and then covering the area with a split-thickness skin graft.

In conclusion, to the best of our knowledge this kind of distant flap is the best option for reconstructions of the face and neck, because it provides smooth and adequate skin to substitute the original skin. We recommend this type of distant flap to all dermatologists who perform surgical dermatology. Finally, we note that this type of flap presents movement similar to that of a small transposition flap; therefore, dermatological surgeons who are familiar with local flap techniques need not worry about performing surgery using these or other types of large flaps.6

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Lichen planopilaris and scleroderma en coup de sabre

To the Editor
Lichen planus and morphea are both cutaneous disorders of unknown aetiology. Their clinical presentation and histological features are different. However, the simultaneous or sequential occurrence of lichen planus and morphea have previously been reported,7–5 suggesting that similar aetiologic events or closely related pathological processes may be involved in both dermatoses. We present a case of lichen planopilaris and scleroderma en coup de sabre occurring together.

A 22-year-old woman presented to the Department of Dermatology, Virgen Macarena University Hospital (Seville, Spain) with an 8-month history of pseudopeladic areas, 2–4 cm in diameter, located on the frontovertical area of the scalp (fig. 1). Her past medical history was not relevant except for repeated episodes of small pseudopeladic alopecic areas concurrent with periods of stress due to university exams. No other lesions were present on the skin or mucosae, and a biopsy from a plaque on the scalp showed features of typical lichen planus and morphea having a history of 8 months.

In conclusion, to the best of our knowledge this kind of distant flap is the best option for reconstructions of the face and neck, because it provides smooth and adequate skin to substitute the original skin. We recommend this type of distant flap to all dermatologists who perform surgical dermatology. Finally, we note that this type of flap presents movement similar to that of a small transposition flap; therefore, dermatological surgeons who are familiar with local flap techniques need not worry about performing surgery using these or other types of large flaps.6
Lichen planopilaris, including a lymphohistiocytic infiltrate surrounding destroyed hair follicles. Two months later she attended again with a slight depression in the middle of her forehead that was clinically diagnosed as scleroderma en coup de sabre (fig. 2). A biopsy on the forehead was not allowed by the patient, but a quite good therapeutic response was observed 8 months later with the coup de sabre lesion using potent topical (0.025% clobetasol dipropionate cream) and intralesional corticosteroids (20 mg triamcinolone acetonide), oral potassic benzoate (PoTaBa 6 g/d), and minoxidil 5% cream, with a partial remission of scleroderma en coup de sabre lesion. Oral doxepine (50 mg/d) was added to improve psychological stress. The lichen planopilaris lesions also improved with the use of intralesional corticosteroids, but small areas of permanent alopecia were still present 8 months later.

Our case is peculiar because of the rare coexistence of localized scleroderma and lichen planus, and to the best of our knowledge no cases of the association of scleroderma en coup de sabre and lichen planopilaris have been reported. They both are immune-mediated processes and many triggering agents have been implicated, including viruses and drugs. Localized morphoea also occurred after BCG vaccination, varicella, measles, Borrelia burgdorferi infection, radiotherapy, pregnancy, penicillamine and bromocriptine therapies. Familial incidence has been noticed, but no significant HLA associations have been reported, and some cases of the frontal type en coup de sabre appear to have a genetic basis. Localized morphoea also occurred after BCG vaccination, varicella, measles, Borrelia burgdorferi infection, radiotherapy, pregnancy, penicillamine and bromocriptine therapies. Familial incidence has been noticed, but no significant HLA associations have been reported, and some cases of the frontal type en coup de sabre appear to have a genetic basis. Localized morphoea also occurred after BCG vaccination, varicella, measles, Borrelia burgdorferi infection, radiotherapy, pregnancy, penicillamine and bromocriptine therapies. Familial incidence has been noticed, but no significant HLA associations have been reported, and some cases of the frontal type en coup de sabre appear to have a genetic basis. Localized morphoea also occurred after BCG vaccination, varicella, measles, Borrelia burgdorferi infection, radiotherapy, pregnancy, penicillamine and bromocriptine therapies. Familial incidence has been noticed, but no significant HLA associations have been reported, and some cases of the frontal type en coup de sabre appear to have a genetic basis. Localized morphoea also occurred after BCG vaccination, varicella, measles, Borrelia burgdorferi infection, radiotherapy, pregnancy, penicillamine and bromocriptine therapies. Familial incidence has been noticed, but no significant HLA associations have been reported, and some cases of the frontal type en coup de sabre appear to have a genetic basis. Localized morphoea also occurred after BCG vaccination, varicella, measles, Borrelia burgdorferi infection, radiotherapy, pregnancy, penicillamine and bromocriptine therapies. Familial incidence has been noticed, but no significant HLA associations have been reported, and some cases of the frontal type en coup de sabre appear to have a genetic basis. Localized morphoea also occurred after BCG vaccination, varicella, measles, Borrelia burgdorferi infection, radiotherapy, pregnancy, penicillamine and bromocriptine therapies. Familial incidence has been noticed, but no significant HLA associations have been reported, and some cases of the frontal type en coup de sabre appear to have a genetic basis. Localized morphoea also occurred after BCG vaccination, varicella, measles, Borrelia burgdorferi infection, radiotherapy, pregnancy, penicillamine and bromocriptine therapies. Familial incidence has been noticed, but no significant HLA associations have been reported, and some cases of the frontal type en coup de sabre appear to have a genetic basis. Localized morphoea also occurred after BCG vaccination, varicella, measles, Borrelia burgdorferi infection, radiotherapy, pregnancy, penicillamine and bromocriptine therapies. Familial incidence has been noticed, but no significant HLA associations have been reported, and some cases of the frontal type en coup de sabre appear to have a genetic basis.
We conclude that lichen planus and localized morphea may coexist or succeed each other as different stereotype immune reactions against the same antigen(s) within the epidermis, but further studies are required to clarify the pathogenesis of this association.

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Annular leucocytoclastic vasculitis: response to dapsone

To the Editor

The term vasculitis comprises a group of clinical syndromes characterized by the inflammation of blood vessels and the occurrence of vascular wall damage. Fifty per cent of the different varieties of vasculitis are leucocytoclastic vasculitis, affecting skin to a certain extent. Lesions can have annular morphology, although they are usually associated with other typical lesions (purpura, petechia, etc.). The histology is essential for diagnosis and is characterized by the presence of nuclear powder. We report the case of a woman who developed this peculiar and benign variety of leucocytoclastic vasculitis without systemic affection. It has been treated with dapsone with good response and represents a distinct clinicopathological entity.

A 79-year-old woman was admitted to the Department of Dermatology, Hospital Clínico San Cecilio (Granada, Spain) with a medical history of aortic stenosis, high blood pressure and non-insulin-dependent diabetes mellitus. She had a 1-year history of recurrent bouts of several erythematous plaques with active, slightly raised and irregular borders (fig. 1). They develop centrifugally and acquired an annular pattern on the trunk and extremities.

Episodes last for about a week and disappear leaving a residual transient hyperpigmentation. The vital signs were within normal limits in the episodes reported by the patient and there were no associated lesions in the oral mucosa. She has presented comitant facial angioedema twice and has been treated with oral corticosteroids and antihistamines each time.

The laboratory tests were negative or normal, including blood count, erythrocyte sedimentation rate, general biochemistry, urinalysis, latex fixation, antistreptolysin O titre, C reactive protein, antinuclear antibody, serum proteins, immunoglobulins, chest X-ray, serum angiotensin converting...

An unusual habonose patches with polycyclic morphology in the right leg.
enzyme levels, complement levels, viral serology (hepatitis B and C virus, Cocksackie, ECHO, human immunodeficiency virus, and Borrelia serology; however, she had hyperglycaemia (glucose: 156; normal < 120). A biopsy specimen showed epidermal parakeratosis and spongiosis. In the dermis, there was a perivascular mononuclear and neutrophilic infiltrate, with a few eosinophils, and extensive red blood cell extravasation, mild endothelial swelling and mild leucocytoclasia (fig. 2). Immunofluorescence was negative for immunoglobulins, complement and fibrinogen.

Treatment with dapsone 100 mg/d for 2 months was established (after the determination of erythrocyte glucose 6-phosphate dehydrogenase) and after a 4-year follow-up the patient has not reported recurrent episodes.

Clinically, leucocytoclastic vasculitis is defined as the presence of a recurrent, acute or chronic asymmetric exanthema; this is due to an inflammatory process after the deposit of immune complexes of small vessels, mainly venules, and it is noticeable by means of purpuric papules, defined as palpable purpura. The different clinical forms have been categorized: haemorrhagic type, haemorrhagic–necrotic type, polymorphic–nodular type or urticaria-like vasculitis. However, annular purpura has rarely been reported in the clinical spectrum of necrotizing vasculitis, except in infantile acute haemorrhagic oedema of the skin.1,2

In 1996, Cribier et al.3 described a new variety of leucocytoclastic vasculitis, which was first defined as recurrent erythema annulare purpura with purpura. The criteria defining this entity were: (i) multiple attacks for months/years with a sudden onset and spontaneous regression after 7–10 days; (ii) annular purpuric patches that shows a centrifugal extension; (iii) extension over the limbs and trunk creating polycyclic lesions that clear with mild haemoiderin deposition; (iv) no extracutaneous symptoms and good general health during the attacks; (v) histopathological changes of leucocytoclastic vasculitis with mild vascular changes and intense polymorphonuclear cell infiltration; and (vi) complete clearance of all lesions with dapsone therapy. Only a few cases fit the above-mentioned criteria.

Although annular vasculitis was initially described by Degos and Guillane7 in 1962, many new cases have been reported in which the described dermatosis was associated with systemic diseases: sarcoidosis,8 myelomatosis,9 ulcerative colitis10 or monoclonal gammopathy IgA11 as well as other cases reported during pregnancy.12 The differential diagnosis includes several entities: erythema annulare centrifugum (because of its centrifugal evolution), urticarial vasculitis or Sweet syndrome.

Most of the cases mentioned have a close relationship with the presence of sarcoidosis. They are mainly patients with Löfgren’s syndrome characterized by the presence of fever, polyarthralgia, nodular erythema and bilateral hilar lymphadenopathy. Cutaneous vasculitis occurring in the context of sarcoidosis have been described on some occasions, although always in early stages of the disease. Annular lesions became clearer with the passing of weeks/months, leaving a residual postinflammation hyperpigmentation. It has been suggested that the development of vasculitis in the course of sarcoidosis may fit with the presence of circulating immune complexes, which are present in more than 50% of sarcoid patients. However, these varieties of vasculitis are not the primary events in vascular damage but are simply epiphenomena.10

It is worth mentioning the therapeutic response to dapsone. It is a highly active sulphone that is used for the treatment of diverse varieties of dermatosis, such as benign mucosal pemphigoid, herpetiform dermatitis, subcornea pustulosis and gangrenous pyoderma. Although its mechanisms of action have not been fully analysed, dapsone is used due to its immunosuppressive and anti-inflammatory effect, which seems to be more effective than systemic corticosteroid therapy in this particular variety of leucocytoclastic vasculitis.

In conclusion, this is a benign variety of leucocytoclastic vasculitis with a good prognosis and a difficult clinical diagnosis, which must be taken into consideration in patients with the above-mentioned characteristics.

References

Herpes simplex virus associated erythema multiforme in a prepartum woman without involvement of the newborn

To the Editor

A 26-year-old primigravida female at week 39 gestation, presented with a 2-day history of a slightly pruritic recurrent eruption after a labial herpes infection. On examination numerous reddish papules up to 3 cm in diameter were found on the distal extremities and face, most of them target lesions. A small herpetiform lesion was found on the upper lip. The rest of the physical examination was unremarkable.

Routine blood analysis was normal. Serology was positive for herpes simplex virus (HSV) type 1 (IgG) and negative for type 2, Epstein–Barr virus and toxoplasma. A diagnosis of HSV-associated erythema multiforme was established based on the clinical and laboratory findings. The patient was treated with topical corticosteroids. Fetal monitor was reactive BPS (Bio-Physical Score) 8/8. Two days later the patient underwent elective Caesarean section due to breech presentation. A healthy male baby was born at 3900 g with Apgar 10/10.

Erythema multiforme is a hypersensitivity reaction after exposure to certain antigens. In cases of HSV-associated erythema multiforme, the pathogenesis of the skin eruption is based on a combination of viro- or pathophysiologic effects mediated by herpesvirus proteins, mainly DNA polymerase, and an immunological reaction to viral antigens. Viral DNA and proteins are ingested by macrophages at the herpetiform lesions, and undergo presentation to T cells, which become T-memory cells for HSV. With activation of HSV, herpesvirus DNA fragments are expressed in epidermal keratinocytes and germinative cells. Activated T cells are recruited to the skin site that is expressing viral DNA fragments, causing an inflammatory reaction that is shown by the clinical presentation of erythema multiforme.1–3

The placenta is an effective barrier to the passage of viral particles, including herpes simplex as well as T cells.4 The absence of viral particles and activated T cells in fetal tissues prevent the appearance of erythema multiforme in the fetus or newborn.

We present an interesting observation of maternal erythema multiforme induced by HSV-1 infection that did not affect the newborn.

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Pyoderma gangrenosum in pregnancy

To the Editor

Pyoderma gangrenosum (PG) is a painful, ulcerating skin disease of unknown aetiology. Numerous associations with systemic disease have been noted, including inflammatory bowel diseases, collagen vascular diseases, leukaemia, myeloma and chronic active hepatitis.1 The occurrence of PG during pregnancy is uncommon, with only four cases reported in the literature. We report the case of a pregnant patient with PG, but with no associated systemic disease.

A 37-year-old pregnant woman at 28 weeks of gestation presented with a 4-month history of painful ulcers on the legs and pustules on the upper and lower extremities. Examination of the woman’s lower legs showed an ulcerated lesion, surrounded by a rolled, bluish, raised border (fig. 1). Pustules were seen on the extremities, face and oral mucosa. Sweet’s syndrome-like lesions were seen on the face and upper extremities (fig. 2).
The findings led us to suspect the diagnosis of PG. The diagnosis was supported by negative results of multiple bacterial swabs and the histopathological data that showed dermal neutrophilic infiltration and prominent endothelial swelling without definite vasculitis (fig. 3). Antinuclear antibodies, antibodies to smooth antigen, anticardiolipin antibody, rheumatoid factor and VDRL were all negative. Detailed examinations and laboratory tests did not show any associated systemic disease.

The woman was treated with prednisolone (1 mg/kg per day), with gradual tapering. After delivery, the lesions improved gradually.

The occurrence of PG during pregnancy is uncommon, and in recent reviews of 212 and 863 cases, there was no mention of an association of PG with pregnancy. All the cases were reported individually. To the best of our knowledge, there have been only four other reports of PG during pregnancy as well as three reports of PG following caesarean delivery4–6 and one case with PG during puerperium7.

A 32-year-old woman developed a widespread ulcerative type PG after caesarean delivery.5 Harland et al. reported a woman who developed PG along the scar following an emergency caesarean section for fetal distress at 29 weeks of pregnancy.8 The four cases of PG during pregnancy involved three women with other associated diseases (antiphospholipid syndrome,9 myeloid leukaemia9 and systemic lupus erythematosus10); the fourth woman presented no systemic disease (in 21 months of follow-up) but the PG involved a 15 × 20 cm painful ulceration with inflammatory borders on the upper part of the left breast.11

The precise aetiology of PG is unknown; however, numerous reports support the role of a defective immune system. Pregnancy has been assumed to be associated with suppression of a variety of humoral and cell-mediated immunological functions. In pregnancy, serum may inhibit the generation of interleukin-2 or -1 activity.12 There is evidence, as yet unexplained, that polymorphonuclear leucocyte chemotaxis and adherence functions are depressed, beginning in the second trimester and continuing throughout pregnancy. Furthermore, the levels of several blood coagulation factors are increased during pregnancy.13

It is possible that these alterations in immune functions of pregnant women could play a part in the development of PG.

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