Lichen planus verrucosus in an association with vitiligo
Lichen planus verrucosus in Zusammenhang mit Vitiligo

Abstract

Lichen planus (LP) is a chronic inflammatory disease with characteristic skin lesions and histological findings. Hypertrophic lichen planus, also known as lichen planus verrucosus, is a subtype of lichen planus. Lichen planus verrucosus is clinically characterized by pruritic symmetric hyperkeratotic plaques of red, yellow-gray, red-brown or purplish-grey colour, usually located on the pretibial or perimalleolar areas, less often on the arms or trunk. Lichen planus affects about 0.5–1% of the population. Hypertrophic lichen planus is a less common form of lichen planus. It accounts for 4.7% of lichen planus cases. The disease has a chronic course, and it is often resistant to local and systemic treatment. This article describes a rare lichen planus verrucosus form in association with vitiligo.

Case Report

A 72-year-old woman presented with itchy rash on various parts of the body. The first lesions appeared on the extensor surfaces of the arms four years ago and had now spread to the neck and legs with intensive itching. Medical history included vitiligo beginning at the age of 27, as well as arterial hypertension which was diagnosed 15 years ago and for which she had been treated with Nebivolol 5 mg once daily continuously. Clinical examination revealed round, pink 0.5–1.5 cm nodules with hyperkeratotic surface on the pretibial surfaces, the back, extensor surfaces of the arms and on the neck (Fig. 1a–d) as well as symmetrical vitiligo lesions. Her fingernails and toenails showed yellow discoloration, splitting and were hyperkeratotic (Fig. 1a, c). Milky white reticular papules were seen on the oral mucosa. Patch testing with baseline series of European allergens and topical corticosteroids was negative. Laboratory findings showed iron-deficiency anemia. C-reactive protein, liver enzymes, creatinine, urea and urine analysis were normal. Further investigations including chest X-ray, sonography of abdomen, peripheral lymph nodes and thyroid gland showed no pathological changes. Gastrointestinal endoscopy identified erosive gastritis. Dermatohistological examination of a lesion from the neck area showed hyperkeratosis, hypergranulosis, papillomatosis, acanthosis, band-like lymphocyte infiltration with eosinophils, the presence of civatte and colloid bodies (Fig. 2a–b). These findings confirmed the diagnosis of lichen planus. The patient refused treatment with oral corticosteroids hence symptomatic treatment with oral antihistamines (1st and 2nd generation), topical high potency steroids and UVA phototherapy was commenced. Additionally the patient was recommended to change the antihypertensive medication, which may have been associated with her skin condition.

Discussion

Lichen planus (LP) was first described by Erasmus Wilson in 1869 [1]. The dermatosis (LP) is a chronic inflammatory disease of unknown etiology characterized by pink to purple colour, flattened, pruritic polygonal papules on the skin or milky white reticular papules on the oral mucosa [2]. Hypertrophic LP, also known as lichen planus verrucosus, is a chronic form of LP [3]. LP affects about 0.5–1% of the population [4,5]. Hypertrophic LP constitutes 4.7% of lichen planus cases. The disease has a chronic course, and it is often resistant to local and systemic treatment. This article describes a rare lichen planus verrucosus form in association with vitiligo.

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case_report
11). LP verrucosus diagnosis is based on the clinical and histopathological findings of the lesions. The histopathological findings of LP reveal orthohyperkeratosis, circumscribed wedge-shaped hypergranulosis, acanthosis and – in case of hyperkeratotic form of LP – hyperkeratosis of epidermis. In the upper dermis a band-like lymphocytic infiltrate is observed and vacuolar degeneration with apoptotic keratinocytes (colloid bodies) at the dermoepidermal junction area [12–14]. Hypertrophic LP may resemble chronic lichen simplex, prurigo nodularis, lichenoid cutaneous amyloidosis, Kaposis sarcoma, stasis dermatitis and psoriasis [15]. Alomari et al. suggest that some cases (20.6%) of hypertrophic LP show prominent numbers of eosinophils. Therefore hypertrophic LP should be considered in the differential diagnosis of lichenoid drug eruption [16]. Medications that can cause lichenoid drug eruptions include beta-blockers (latency period, 1 year), penicillamine, antimalarial agents, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, nonsteroidal anti-inflammatory drugs, benzothiadiazides and more [17–19]. Typical histopathological findings of lichenoid drug eruption are focal parakeratosis, presence of eosinophils and plasma cells, and a deeper perivascular and periadnexal infiltrate [20]. The principal differential diagnosis in this case was prurigo nodularis, that affects extensor surfaces of the limbs and presents as pruritic nodules, and is histologically characterised by hyperkeratosis, parakeratosis, irregular acanthosis, the inflammatory infiltrate in the dermis with lymphocytes, mast cells, histiocytes and occasionally eosinophils [21]. In our case hypertrophic LP was associated with vitiligo. S. Baghestani et al. have reported familial coexistence of LP and vitiligo, correspondingly, J. Wyte et al. – a case of long-standing vitiligo that was spared in LP [22,23]. G. Kravvas et al. reviewed many theories of LP association with vitiligo as sun-exposed depigmented areas play an important part in the initiation of LP; the evident manifestation of the Koebner phenomenon in vitiligo area. Another theory is that long-standing vitiligo alters the expression of antigens identified by effector T cells in LP, thus leading to the pathophenotype of LP [24–30]. Longstanding, non-healing, severely itching hypertrophic LP lesions are prone to develop malignancy. There are reports of metastatic squamous cell carcinoma and keratoacanthoma arising from long standing hypertrophic lesions [31,32]. A.R. Bowen et al. study reports, that distinguishing keratoacanthoma and hypertrophic LP can be difficult because both have similar histopathological features, and evaluation of p53, MIB-1

Fig. 1 Pruritic symmetric hard and hyperkeratotic nodules of red, red-brown or purplish-grey colour more prominent on the frontal shins with vitiligo symmetrically area (a–c) and less prominent on the back (d).

Fig. 2 Histopathologic findings: acanthosis, hyperkeratosis, band like lymphocyte infiltration in H+E staining (a), civatte bodies and colloid bodies in PAS staining (b).
and perforating elastic fibers is a tool in differential diagnosis [33]. The first-line treatment of hypertrophic LP is corticosteroids which can be applied topically or given systemically, topical calcineurin inhibitors, phototherapy, systemic retinoids, notably acitretin, systemic cyclosporine, thalidomide and mycophenolate mofetil [2,34]. Itch is also an obsessive and burdensome sympto-
m of hypertrophic LP. According to K. Welz-Kubiak and A. Reich pathogenesis of itch in LP is still not fully understood, so there are no effective therapeutic modalities alleviating pruritus [35]. Som-
times oral antihistamines may be used to control pruritus [34]. In this case lesions were resistant to topical treatment, espe-
cially hypertrophic LP lesions with symmetrical vitiligo areas. Our case doesn't rule out the possibility that hypertrophic LP could be partially induced by antihypertensive drugs. The treat-
ment of LP should be given for a long time and patient follow-up is vital in order to detect malignant transformation of the lesions.

Conflict of Interest

The authors declare no conflict of interest.

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