Letters to the Editor

Modern moulage

The successful treatment of palmoplantar hyperkeratotic lichen planus with enoxaparin

Sir,

The palmoplantar hyperkeratotic variant of lichen

planus (PHLP) is a rare entity. The clinical features of PHLP do not resemble classic lichen planus (LP), and the disease is mostly resistant to treatment. In approximately one-quarter of PHLP cases, typical lichen lesions can also be seen on other skin and mucosal surfaces.[1] Two cases of PHLP, which were successfully treated by subcutaneous enoxaparin without any subsequent adverse effects, are presented below.

Case 1: A 24-year-old male patient was admitted with complaints of thick plaques existing on the palms of his hands. During the physical examination of the patient, yellowish, solid, thick plaque lesions surrounded by a livid margin were observed on both palms and soles [Figure 1a, b]. No pit formation was observed on the solid and thick areas. While no change was observed on the nails, a bilateral reticular appearance on the oral mucosa and scattered livid colored bright papules on the trunk were seen.

Case 2: A 45-year-old male patient was admitted, who had suffered from yellowish solid lesions on his hands and feet for 10 years. During the dermatological examination of the patient, a white reticular appearance was observed bilaterally on the *buccal mucosa,* and multiple solid hyperkeratotic papular lesions livid in color, which included *Wickham* striae, were detected on the flexor surfaces of the arms and on the anterior surface of the tibia.

Common histopathologic features of biopsies taken from the palms of both cases revealed hyperkeratosis, acanthosis, wedge-shaped hypergranulosis within the epidermis, degeneration in the basal layer, and a lichenoid pattern of lymphocytic infiltration at the dermo-epidermal junction [Figure 1c]. These findings were consistent with PHLP.

Enoxaparin (Clexane®, Rhone Poulenc-Cedex-F, France) was administered at a dose of 3 mg subcutaneously into the abdominal wall once a week, after receiving written and verbal informed consent from the patients. HCV, anti-HBs and HBs antigens were found to be negative in laboratory examinations. Platelet count assays and coagulation parameters of the patients (activated partial thromboplastin time, international normalized ratio, prothrombin time) were within normal limits. At the end of 12 sessions, all the skin lesions were seen to heal, leaving postlesional hyperpigmentation [Figure 2a–c], but oral mucosa lesions remained stable. No local or systemic adverse effects were seen in the

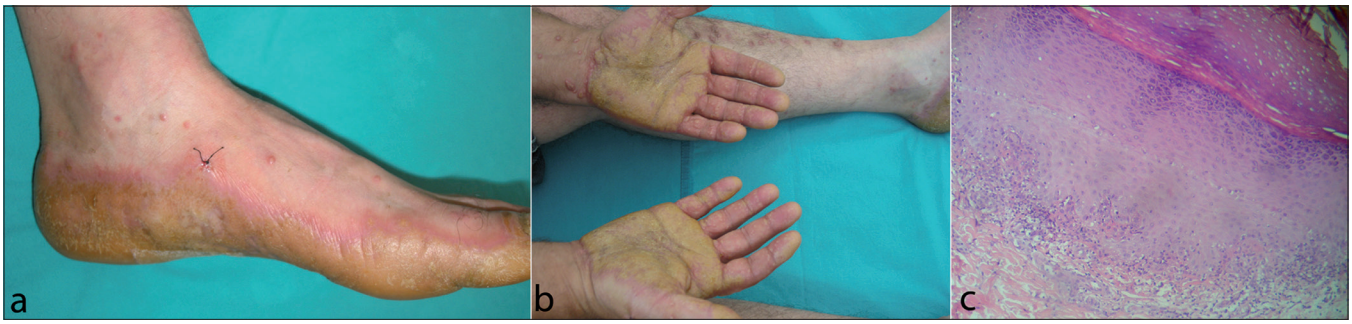

Figure 1: (a,b) Yellow thick plaques existing on palms of the hands and soles of the feet; (c) hyperkeratosis, acanthosis, wedge-shaped hypergranulosis within epidermis, degeneration in basal layer and lichenoid pattern of lymphocytic infiltration at the dermoepidermal junction (H and E, ×200)

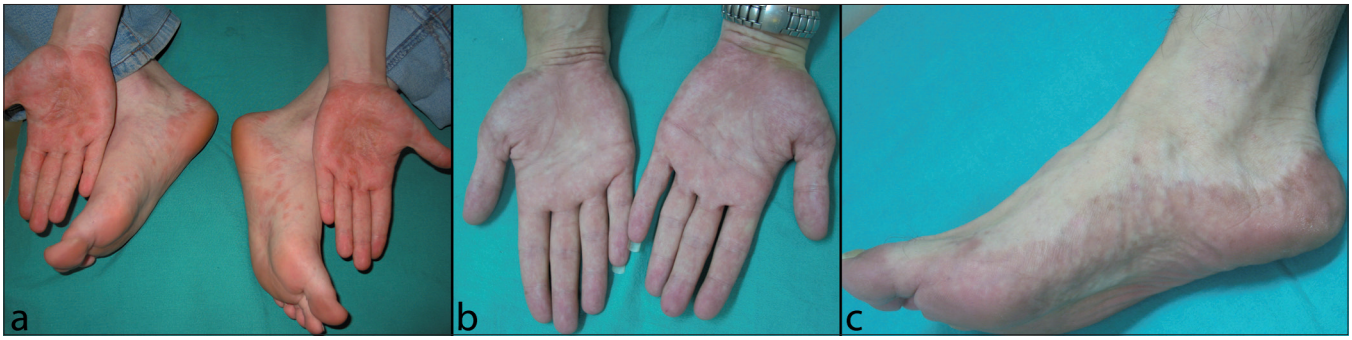

Figure 2: (a–c) Complete response to enoxaparin treatment

patients. No recurrence was seen during a 1-year follow-up of the patients.

The PHLP is a rare variant of lichen which can be easily misdiagnosed as palmoplantar keratoderma, psoriasis, verruca vulgaris, callus, punctate porokeratosis, arsenic keratosis, and lichen simplex chronicus.[2] In PHLP, lesions are frequently highly pruritic, erythematous, squamous and/or hyperkeratotic, well-defined, usually not involving the digits and resemble plaques located on the internal plantar arch.[3] Apart from this classic appearance, PHLP can also be seen with erythematous plaques, punctate keratosis, diffuse keratoderma and ulcerated lesions.[3] The determination of classic LP findings during a histopathologic examination enables a diagnosis.

Enoxaparin was administered for the first time in 1988 by Hodak *et al*. in Israel. Administration was successfully carried out at a dose of 3 mg via subcutaneous injection once a week to 10 LP patients with diffuse skin involvements.[4]

Enoxaparin's mechanism of *action* is based on the inhibition of the heparanase enzyme released by active T lymphocytes. The heparanase *enzyme* emerging on the surface of T lymphocyte passes through blood, cleaves heparan sulfate side chains that have a supporting role in the extracellular matrix, and thus enables the T lymphocyte to penetrate through the upper dermis. Since the heparanase *enzyme* is inhibited by low-dose, low–molecular-weight heparin, T lymphocytes cannot reach their target tissue.[4,5] Because of the disaccharide structure of enoxaparin, it also inhibits the cytokines' role in inflammation, and especially the expression of tumor necrosis factor alpha.[4]

The lesions in PHLP tend to regress spontaneously within 6–18 months. Topical-systemic corticosteroids and other treatment regimens provide healing within 2–9 months in most cases, but recurrence is frequently seen after discontinuation of the drug.[3]

In our cases, complete cure was obtained using enoxaparin administered once a week for 3 months, and no adverse effects were seen. No recurrence was seen in follow-ups performed for 1 year.

Therefore, enoxaparin can offer a successful, particularly safe and long-term therapeutic alternative in PHLP treatment when its adverse effects are compared to those of long-term corticosteroid use.

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