

QUIZ SECTION

Violaceous and Ptechieal Persistent Plantar Plaques: A Quiz

Ignacio Gómez-Martín, Gemma Martín-Ezquerro and Ramon M. Pujol

Department of Dermatology, Hospital del Mar, Passeig Marítim, 25-29, ES-08003 Barcelona, Spain. E-mail: nachgm@gmail.com

A 78-year-old woman was referred to our department for evaluation of persistent whitish and violaceous plaques on both heels. Her medical history included type 2 diabetes mellitus, dyslipidaemia, arterial hypertension and minor depression, with chronic medical treatment. The patient reported a sudden onset of symmetrical hyperkeratotic papules on the soles, which was painful on walking, 2 years previously. Topical antifungals, 10% salicylic acid and curettage had failed to improve the lesions.

Physical examination revealed multiple whitish, punctate, slightly hyperkeratotic papules, 2–5 mm in diameter

that had merged, forming symmetrical plaques, on the posterior area of both soles. The plaques were more extensive on the right foot (Fig. 1a). Ptechieae were also noted. No dilated capillaries were present after curettage. No similar cutaneous lesions were found elsewhere.

A complete blood cell count and biochemical profile showed no abnormalities. Serologies for HIV, syphilis and hepatotropic virus were negative. A skin biopsy was taken (Fig. 1b).

What is your diagnosis? See next page for answer.

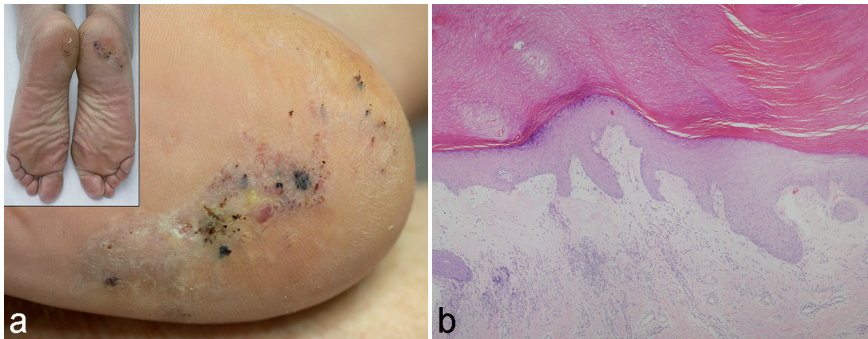


Fig. 1. (a) Hyperkeratotic, whitish and purpuric plaque on the right heel. Inset: violaceous and whitish plaques on both soles. (b) Skin biopsy specimen showing orthokeratosis, liquefaction degeneration of the basal layer, prominent oedema within thickened papillary dermis, vascular dilatation and a lymphocytic perivascular infiltrate in the upper dermis with occasional histiocytes and plasma cells (haematoxylin-eosin, original magnification $\times 100$).

ANSWERS TO QUIZ

**Violaceous and Petechial Persistent Plantar Plaques:
A Comment**

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Diagnosis: Plantar lichen sclerosus

Subsequent physical examination of the genitalia revealed erosions and atrophy of the labia minora and whitish papules and plaques with purpura over the entire vulval and perianal area. A skin biopsy specimen from the vulva revealed characteristic histopathological findings of lichen sclerosus (LS).

Treatment with topical clobetasol propionate 0.05% ointment was prescribed for both genital and plantar lesions. A marked improvement in the plantar lesions and an almost complete resolution of the genital erosions was observed after 3 months of treatment.

LS, formerly known as lichen sclerosus et atrophicus, is a relatively common chronic inflammatory dermatosis, primarily of the anogenital region, in both males and females. The exact prevalence of LS is unknown and is probably underestimated, but estimates range from 1 in 300 to 1 in 1,000 among patients referred to a dermatology department (1, 2). LS predominates in women (approximately 8:1), with a bimodal peak of incidence in prepubertal children and in postmenopausal women and adult men (1). The aetiology remains unknown; however, there is genetic susceptibility and an association with some autoimmune disorders (alopecia areata, vitiligo, thyroid disease and pernicious anaemia) has occasionally been reported (3). Clinically, over 85% of lesions are found on anogenital skin. Genital LS classically manifests as pruritic or painful shiny, white macules and plaques with follicular delving and hyperkeratosis. Occasionally, ecchymosis, bullae and erosions may appear. Without treatment LS may cause atrophy and scarring. LS may be asymptomatic. Development of malignancy (squamous cell carcinoma) has been reported in approximately 5% of genital lesions (1).

Extragenital cutaneous LS may be observed as an isolated disorder or in association with anogenital LS (15–20%) (1, 4). Clinically, extragenital LS is manifested by pearly polygonal papules, sometimes confluent, forming atrophic or sclerotic plaques, with keratotic plugs. It usually affects the trunk, the proximal extremities and, more specifically, the neck, shoulders, wrists and sites of physical trauma (koebnerization). LS involving acral areas and, specifically, the palms and soles is an exceedingly rare phenomenon and only a few cases of LS confined to the feet have been reported (4–8). In those areas, LS may present as whitish, hypopigmented, ivory patches/plaques and occasionally telangiectasias, purpura, hemorrhagic blisters and fissures.

In contrast to genital LS, in which the differential diagnosis should be established with intraepithelial neoplasia, erosive lichen planus, vitiligo, lichen simplex chronicus or cicatricial pemphigoid, acral or plantar lesions of LS

should be differentiated clinically from plantar warts, callosities, plantar lichen planus or repeated traumatic petechiae (“black heel”). Histopathological examination is mandatory in order to establish a definite diagnosis. Histological analysis of early lesions of LS show a band-like lymphocytic infiltrate in the upper dermis and vacuolar degeneration of the basal layer. Mild homogenization of the dermal collagen may be present in the papillary dermis. In older lesions, orthohyperkeratosis, thinning of the epidermis and homogenization of the upper dermis, with hyalinization and sclerosis of the papillary dermis, dilated blood vessels and a sparse interstitial lymphocytic infiltrate, are observed. Histologically, the main differential diagnosis should be established with lichen planus and occasionally psoriasis or mycosis fungoides, especially in early lesions when no clear-cut sclerotic changes are present.

LS has no definitive cure, but can be controlled by adequate treatment. Potent topical corticosteroids represent first-line therapy for LS, either genital or extragenital (9, 10). Topical calcineurin inhibitors, topical or systemic retinoids, oral antimicrobials, oral antimalarials, phototherapy and photodynamic therapy have been used as alternative therapies. Surgery is indicated only for complications of scarring or in cases of changes that are suspicious of malignant transformation. Long-term follow-up is mandatory due to the risks of scarring and of developing a squamous cell carcinoma.

REFERENCES

- Powell JJ, Wojnarowska F. Lichen sclerosus. *Lancet* 1999; 353: 1777–1783.
- Wallace HJ. Lichen sclerosus et atrophicus. *Trans St John's Hosp Dermatol Soc* 1971; 57: 9–30.
- Meyrick Thomas RH, Ridley CM, McGibbon DH, Black MM. Lichen sclerosus et atrophicus and autoimmunity – a study of 350 women. *Br J Dermatol* 1988; 118: 41–46.
- Steff M, Toulemonde A, Croue A, Lemerle E, Le Corre Y, Verret JL. Acral lichen sclerosus et atrophicus. *Ann Dermatol Venereol* 2008; 135: 201–204.
- Petrozzi JW, Wood MG, Tisa V. Palmar-plantar lichen sclerosus et atrophicus. *Arch Dermatol* 1979; 115: 884.
- Hammar H. Plantar lesions of lichen sclerosus et atrophicus accompanied by erythralgia. *Acta Derm Venereol* 1978; 58: 91–92.
- Caspary P, Carpena AB, de Almeida H Jr. Plantar involvement in lichen sclerosus. *Int J Dermatol* 2009; 48: 663–665.
- Viana F de O, Cavaleiro LH, Unger DA, Miranda MF, Brito AC. Acral lichen sclerosus et atrophicus – case report. *An Bras Dermatol* 2011; 86: S82–84.
- Neil SM, Lewis FM, Tatnall FM, Cox NH. British Association of Dermatologists's guidelines for the management of lichen sclerosus. *Br J Dermatol* 2010; 163: 672–682.
- Chi CC, Kirtschig G, Baldo M, Lewis F, Wang SH, Wojnarowska F. Systematic review and meta-analysis of randomized controlled trials on topical interventions for genital lichen sclerosus. *J Am Acad Dermatol* 2012; 67: 305–312.