Lichen sclerosus (LS) is a chronic inflammatory disease of uncertain etiology. Initially, it was described at the end of the 19th century by Hallopeau and Darier as a variant of lichen planus; currently, it is considered as a separate entity.

Typical lesions involve sclerotic patches, with central atrophy, generally located in the genital area. Extragenital manifestations are characterized by polygonal whitish papules or plaques, erosions, or lichenification (unique or in combination). Hypokeratosis, vacuolization of the basal cells, and homogenization of dermal collagen with a band-like chronic dermal infiltrate of inflammatory cells is evidenced by light microscopy.1,2

Women are more commonly affected than men, with a bimodal peak of incidence in the prepubertal and postmenopausal age. There is a strong association with human leukocyte antigen class II DQ7 and autoimmune disorders, such as vitiligo, alopecia areata, thyroid disease, and pernicious anemia. There is still controversy regarding the role of infectious diseases (human papillomavirus, Borrelia burgdorferi). Histopathologically, LS is characterized by a band of inflammation as a zone of homogeneous paucicellular superficial dermal edema and sclerosis, with variable vacuolar interface degeneration and epidermal atrophy or lichenification.2 Direct immunofluorescence shows fibrin deposit in the superficial dermis and at the dermal epidermal junction. LS often exhibits a prominent inflammatory infiltrate composed mainly of lymphocytes and plasma cells. The acral form of LS involving the palmoplantar region is rarely reported.3-5

Data from recent case reports show that the most common approach for LS is treatment with immunosuppressant agents (topical and systemic). Surgical procedure is described in only 1 case report (split-skin graft by Sonnex TS in 1985).6

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Herein, we report a case of a 76-year-old man affected for 15 years by chronic plantar ulcers because of acral LS and atrophicus (Figs. 1 and 2).\(^7\)\(^8\) No genital or oral lesions were present, but a severe nail dystrophy had led to a complete absence of the nail plate.

A biopsy taken from a plantar lesion demonstrated epidermal acanthosis with upper hyperkeratosis and compact ortho-parakeratosis. The granular layer was only present focally. There were scattered spongiotic phenomenon with lymphocyte exocytosis and balloon degeneration of basal keratinocytes with acidophilic bodies.\(^9\)

The patient had been successfully treated for many years with immunosuppressive agents (systemic prednisone, cyclosporin A, and azathioprine) and topical application of tacrolimus ointment 0.1% leading to a complete wound healing. Because of metabolic dysfunction (heart failure, diabetes, and increasing of transaminase and creatinine), systemic therapy was reduced to low dosage of steroids with subsequent reactivation of the disease and comparison of plantar ulcerations reappearance (5 × 2 cm).

Therefore, we decided to perform surgery using a graft-derived technique. Reverdin, in 1869,\(^10\) described the skin graft for regenerative purpose for the first time. The skin graft needs a well-granulating wound bed tissue, which chronic wounds often lack. So skin graft is not indicated in many affected patients.

We recently described an innovative surgical technique used for chronic venous ulcers called “nested graft”: the aim of the nested graft is not to cover the wound bed, as with the traditional full-thickness skin graft, but to repopulate the chronic ulcer bed with healthy cells. We also demonstrated that the nested graft is able to induce wound healing through the de-senescence in wound bed fibroblasts.\(^11\)\(^12\) The donor site is prepared using povidone-iodine, and a local anesthesia is achieved using 1% lidocaine.

**Fig. 1.** Right foot before surgery.

**Fig. 2.** Left foot before surgery.

**Fig. 3.** Right foot at 4-month follow-up.
Full-thickness explants, without hypodermal fat, are taken from the donor site using a 6-mm punch biopsy; the donor site is immediately repaired with a simple suture. The graft is then deposited in a Petri dish containing physiological saline. The receiving site (ulcer bed) is prepared with soft curettage, and then full-thickness circular fragments of the ulcer are removed by using a 5-mm punch biopsy. For removing the explants from the donor site, a slightly larger punch (6 mm) is used. Due to the physiological contraction of the transfer tissue, the dimensional match results perfectly.

For the first time, we performed the nested graft on an inflammatory skin ulcer because of LSA. Our patient tolerated the surgical procedure well leading to complete wound healing in 4 weeks, and at the 4-month follow-up, no signs of the LSA were present. The islands of grafted skin maintained their status and were not affected by the underlying inflammatory disease. On the contrary, the grafted skin “healed” the surrounding skin suffering from LS. This could be explained by the proliferation of grafted cells through the skin inducing a microenvironment remodeling process that influenced the epidermal atrophy and lichenoid infiltrate at dermo-epidermal junction. At the follow-up visit, a slow pigmentation grafted island was evident; the pigmentation was because of the presence of resident melanocytes in the donor skin (Figs. 3 and 4). In conclusion, our procedure could well be an innovative surgical treatment for a chronic inflammatory disease.

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