Punch Grafting in Lichen sclerosus et atrophicus
S. Malakar
S. Dhar

Dermatology Division, Duncan Gleneagles Diagnostic and Research Centre, Calcutta, India

Key Words
Punch grafting
Lichen sclerosus et atrophicus

Lichen sclerosus et atrophicus (LSA) is a disease of unknown etiology characterized by porcelain-white sclerotic patches of skin with frequent involvement of male and female genitalia [1]. The condition often coexists with morphea and systemic sclerosis [2, 3]. However, LSA is considered to be a distinct entity having unique clinical and histopathological features [1]. Although various autoimmune disorders have been associated with LSA [4, 5], the autoimmune process per se probably does not play a significant role in the pathogenesis of the disease.

There is no effective treatment for LSA. However, topical steroids [6] and estrogen [7], oral potassium p-aminobenzoate [8] and etretinate [9] have been tried with variable results. We have treated 2 patients with LSA with a new technique, i.e., punch grafting which we describe in this paper.

A 24-year-old male presented with asymptomatic milky white variable-sized patches over his back, right upper arm and left thigh of 7 years duration.
Examination revealed porcelain white atrophic patches varying from 0.5 to 2.5 cm in diameter with follicular plugging. A diagnosis of LSA was made and confirmed by histopathological examination of one of the lesions on the back. There was no improvement of the lesion(s) after 3 months’ application of a moderately potent topical corticosteroid. Punch grafting was done over one of the lesions on the back, measuring 2.5 cm in diameter, according to the standard technique followed in vitiligo surgery [10, 11]. The donor site chosen was the upper and lateral aspect of the left thigh. Ten punch grafts each measuring 2 mm were taken from the donor site to cover the recipient area. A similar procedure was followed over two other adjacent patches of LSA on the back at monthly intervals. The ‘take’ of grafts was 100% and the sclerotic depigmented skin of LSA was replaced by skin that was normal in color and texture. Repeat biopsy and histological examination of the skin from the recipient site revealed no evidence of LSA. During the second follow-up after 1 year, the grafted skin was found to be normal on clinical and histological examinations.

A 32-year-old female presented with asymptomatic white patches over her left-upper arm and forearm of 9 months duration.
Examination revealed porcelain white atrophic patches varying from 0.5 to 2.5 cm in diameter with follicular plugging. A diagnosis of LSA was made and confirmed by histopathological examination of one of the lesions on the left forearm. The ‘take’ of grafts was 100% and the sclerotic depigmented skin of LSA was replaced by skin that was normal in color and texture. Repeat biopsy and histological examination of the skin from the recipient site revealed no evidence of LSA. During the second follow-up after 1 year, the grafted skin was found to be normal on clinical and histological examinations.

A 32-year-old female presented with asymptomatic white patches over her left-upper arm and forearm of 9 months duration. On examination, there were few depigmented patches with atrophy and keratotic plugging. A diagnosis of LSA was made and a biopsy was taken from one of the lesion(s) on the left forearm. The histopathological features confirmed the clinical diagnosis. Punch grafting was one in the same manner as in the prior patient over a large patch of
LSA over the left forearm measuring 3 cm×2 cm. Posttreatment follow-up at intervals of 6 months and 1 year revealed that the skin at the grafted site was clinically and histologically normal. Acceptance of graft at the recipient site further emphasizes that the autoimmune process is probably not triggered in LSA. Although the disease has got a predilection for male and female genitalia, it does not show any specificity as far as the extragenital sites are concerned. It is quite possible that the cytokines, tissue growth factors and tumor necrosis factors present in the donor skin are playing some role in halting the disease process at the site of LSA. However, this observation of ours is in sharp contrast to the observations made in cases of morphea where normal skin grafted over morphea lesion(s) became sclerotic like morphea by 6-9 months [12]. This preliminary work has encouraged us to undertake skin grafting on the LSA Lesions over male and female genitalia which often produce meatal stenosis and ulcerations in males and dyspareunia, ulceration and malignancy in females.

References

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