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Trauma-induced Isomorphic lesions in Morphea
—A Brief Case Report—

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We describe a case of morphea which presented further typical lesions of the disease at the sites of mechanical trauma. It can be suggested that cutaneous lesions of morphea may be locally developed due to physical stimuli as an isomorphic response in patients or subclinical cases of the disease.

Key Words : *Morphea, Trauma, Isomorphic response*

INTRODUCTION

Morphea or circumscribed scleroderma is an uncommon cutaneous disorder characterized by patches of well-demarcated sclerosis occuring in a variety of patterns and locations. The disease occurs most commonly in middle-aged women and in most cases the onset is between the second and fifth decades of life. The etiology of morphea is unknown, however trauma has been thought to trigger it in occasional cases.

CASE REPORT

A 54-year-old woman visited our clinic presenting two oval sclerotic plaques on the anterior and lateral thigh which had first appeared 10 months earlier. The brownish thickened lesions were large coin or walnut-size but she did not complain about any subjective symptoms. The patient denied any previous episodes of trauma or injection on the thigh. On physical examination, the only pertinent findings were confined to the thigh skin, with no evidence suggestive of systemic sclerosis or any other illness. Her past medical history and family history were non-contributory.

A clinical diagnosis of morphea was made and confirmed by histopathologic examination. The patient was treated with intralesional injections of triamcinolone acetonide (5 mg/ml, biweekly), and showed some clinical resolution of the lesions within 4 months. For the next 5 months she maintained remission of the cutaneous lesions.

Four weeks after the last follow-up examination, after suffering a minor accident (a fall down a stairway) causing bruising wounds on her shin areas, she noted the appearance of two egg-size somewhat indurated violaceous patches/plaques on the shin at the sites of injury (Fig. 1). It was recognized on both shins at the fourth week following the accident. A clinical diagnosis of trauma-induced lesions of morphea was suspected. A biopsy specimen taken at the mid-lesional area (performed at the sixth week from the falldown) revealed the findings of morphea : proliferation of collagen fibers in the reticular dermis with downword extension below the eccrine sweat glands ; moderate degree of interstitial edema and some infiltrations of mononuclear cells between the collagen bundles and around the blood vessels (Fig. 2). Thereafter, the patchy lesions gradually became dark-

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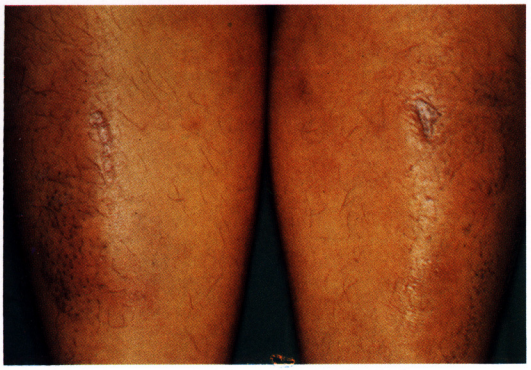

Fig. 1. Indurated erythematous-brownish plaques on the shins. Trauma-scratch scars are seen on both sides.

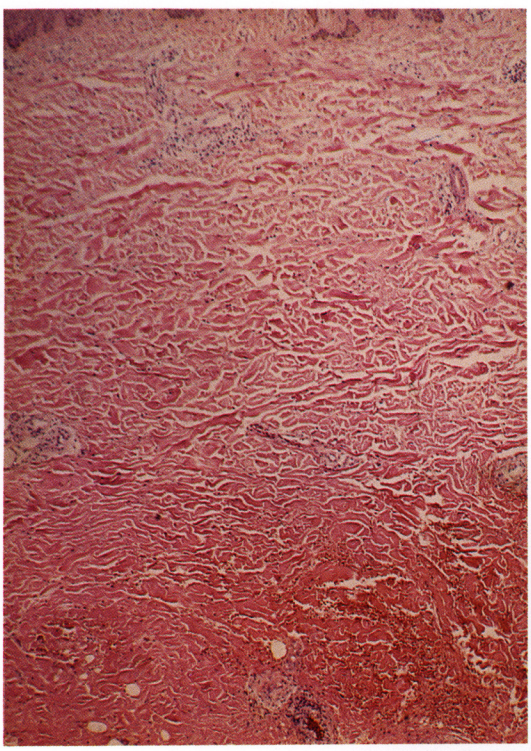

Fig. 2. Increased amount of collagen extended below the sweat glands with some inflammatory infiltrates in the dermis(H & E stain, ×40).

brownish sclerotic plaques.

Laboratory data, including complete blood cell counts with differential, erythrocyte sedimentation rate, urinalysis, serologic tests for complement and immunoglobulin levels, and antinuclear antibodies, obtained around this examination were all within the normal ranges or negative.

The lesions on the shin were treated with intralesional injections of triamcinolone acetonide as applied previously for the thigh lesions. In the following months the lesions showed much clincal improvement.

DISCUSSION

In the above patient, local traumatization of cutaneous tissue appears to be the initiating event for the development of new lesions (isomorphic phenomenon) associated with her morphea-prone constitution.

The lesions of morphea usually occur spontaneously without any specific precipitating factors, though external stimuli such as mechanical traumas including operation/injection injury (Christanson et al., 1956; Tosti et al., 1988), irradiation (Neill et al., 1988; Colver et al., 1989; Trattner et al., 1991), vaccination (Mork 1981), and cutaneous infections (Singh and Beck, 1975; Sahl, 1978) may sometimes appear to be provocational factors for the development of localized cutaneous lesions with latent periods of several weeks to years, as seen in our case. The mechanism involving these local stimuli responsible for the excessive collagen production is difficult to explain. Upon biologic stress or trauma, cutaneous physiology may cause local production and release of inflammatory mediators/cytokines including fibrogenic cytokines, such as transforming growth factor-beta (Kupper, 1990; Smith and LeRoy, 1990; Perez and Kohn, 1993). Perhaps, possible dysregulation of fibrogenic cytokines causing clonal overactivity of fibroblast, and/or other undefined mechanisms may cause excessive synthesis of collagen to the local effects of such triggering factors in susceptible individuals.

Although no definite environmental influences on the development of morphea have been described, mechanical trauma may occasionally be regarded as a contributing factor as seen in this patient.

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