Quiz

Depressed plaques over back in a 35-year-old male

A 35-year-old male presented to our outpatient department with depressed pigmented patches over his back since 1 year. These lesions were non-progressive and asymptomatic. He had no history of trauma, tick bite, infection or injection at these sites.

On examination, multiple well-defined round-to-oval, brownish plaques varying in size from 1 to 8 cm were present over the back [Figure 1]. These were depressed below the level of adjacent skin. There was no induration, sclerosis, tenderness, or perilesional erythema. A punch biopsy specimen was taken from the margin of one of these lesions and was sent for histopathological examination.

HISTOPATHOLOGICAL FINDINGS

Hematoxylin and eosin staining of a skin biopsy specimen revealed dense collagen bundles throughout the dermis. Epidermis was normal. Minimal scattered inflammatory infiltrate was seen. The thickness of dermis was reduced [Figures 2 and 3].

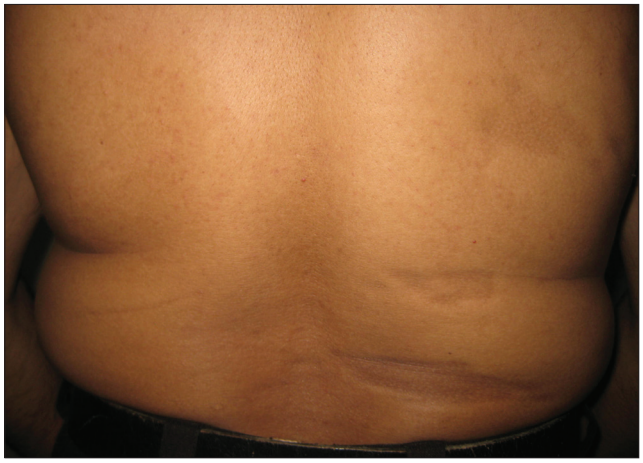

Figure 1: Clinical photograph showing multiple well-defined round-to-oval, brownish plaques varying in size from 1 to 8 cm present over the back

Laboratory studies including a complete blood count, liver and renal function tests, urinalysis, and electrolytes were within normal limits. The chest X-ray was normal. Serum antibody for *Borrelia burgdorferi* and anti-nuclear antibody were negative. Complete ophthalmologic, odontologic, and radiologic surveys revealed no abnormalities.

WHAT IS YOUR DIAGNOSIS?

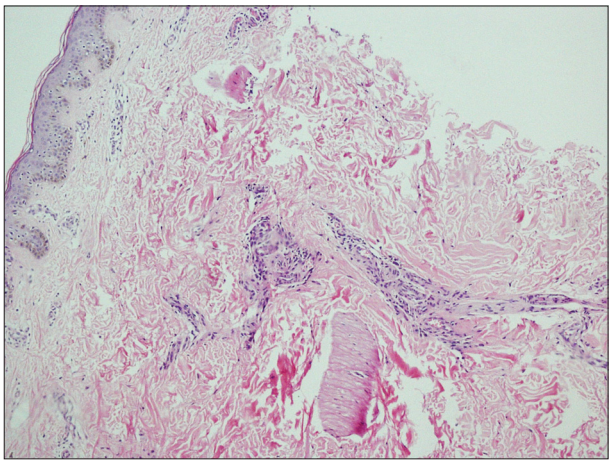

Figure 2: Dense collagen bundles in the dermis (H and E, ×40)

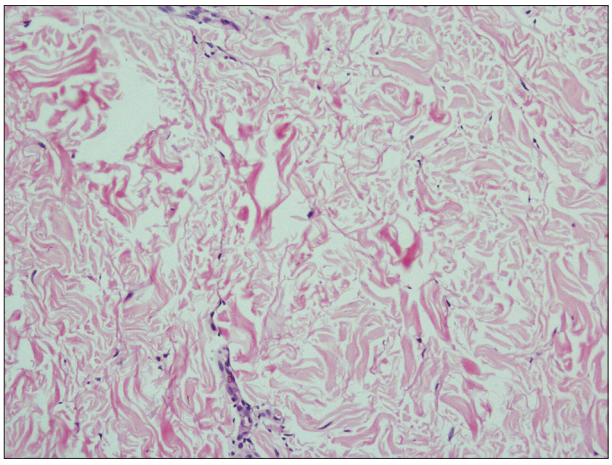

Figure 3: Dense collagen bundles with scattered inflammatory infiltrate in the dermis (H and E, ×100)

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Diagnosis

Idiopathic atrophoderma of Pasini and Pierini

DISCUSSION

In 1923, Pasini[1] described the condition under the name progressive idiopathic atrophoderma. In 1936, Pierini and Vivoli[2] extensively studied and defined the condition and its possible link to morphea. Canizares[3] *et al.*, in 1958 renamed it as idiopathic atrophoderma of Pasini and Pierini (IAPP).

Exact cause of IAPP is unknown. Genetic factors, neurogenic factors, abnormal metabolism of dermatan sulphate, and immunological factors have all been implicated in the pathogenesis of IAPP.[4] IAPP has been considered as a variant of morphea on the basis of certain clinical and histopathological features. These are (a) sclerosis and induration were found in some lesions of IAPP, (b) morphea and IAPP have been observed in the same patient, (c) in its late atrophic stage, lesions of morphea resemble IAPP, (d) homogenization of collagen and a perivascular lymphocytic infiltrate are common histological features encountered in morphea and IAPP, and (f) dermatan sulphate content in involved skin is more than perilesional skin, a pattern that has been observed in scleroderma.[5,6]

However, the following features indicate that IAPP and morphea are distinct entities. Morphea characteristically begins as a discrete circumscribed, erythematous to sclerotic plaque, often with a white center and characteristic peripheral lilac rim. It has an early onset and a longstanding course. IAPP lacks sclerosis, and lesions commonly coalesce over time, producing a moth-eaten appearance that is not consistent with morphea. Atrophy is a primary pathogenetic event in IAPP rather than sequelae as is the case in morphea. Morphea involves all layers of epidermis, whereas IAPP affects collagen in the dermis only.

IAPP is insidious in onset. It is twice as common in females as in males. The most common age group affected is 20-30 years. It is characterized by single or multiple gray-brown or bluish-brown, depressed plaques. Most common site is the trunk, usually on the back or lumbosacral region, followed in frequency by the chest, arms, and abdomen.[4] The affected areas are atrophic, often exhibiting a cliff-drop border of 2-8 mm forming an inverted plateau. The lesions are usually round or ovoid, varying in size from a few millimeters to several centimeters, often orientated along the skin cleavage lines. The lesions are usually asymptomatic and do not show signs of inflammation and sclerosis. The epidermis is not atrophic and markings are preserved. The course of the disorder is benign, except for cosmetic concern.

Histology shows homogenization and clumping of collagen in deeper dermis. Elastic stains show a spectrum of changes ranging from normal to severe diminution and fragmentation of the elastic fiber network.[6]

No effective treatment is known for IAPP. Therapeutic approaches for the sclerosis component of APP have included topical and systemic steroids, antibiotics, antimalarials, D-penicillamine, calcitriol, and phototherapy with variable efficacy. In patients with new early stage IAPP, especially those with a positive *B. burgdorferi* antibody titer, therapy for Lyme disease is recommended. Without treatment, the lesions persist indefinitely. Arpey *et al*.[7] showed the Q-switched alexandrite laser (755 nm) to be effective in diminishing the hyperpigmentation by 50% after three treatments in one case.

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