disease was exacerbated by herpes simplex and regressed by herpes simplex treatment. We want to emphasize the presence of herpes simplex lesion concomitant with treatment-resistant bullous lesion of Haley- Hailey disease. Hailey-Hailey disease treatment management should bring to mind HSV infections.

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Scleredema diabeticorum

Sir, a 48-year-old male presented with spontaneous onset of pigmentation on his upper arms and back followed by development of progressive thickening of the skin which first started from the neck and gradually extended downwards to involve the upper back and arms of 3 years duration. There was no history of infection prior to development of induration. Our patient was a known diabetic since past 20 years on irregular treatment. Cutaneous examination revealed widespread woody hard induration of the skin over the neck and upper back extending upto the arms with sparing of face and hands (**Figure 1**). The skin of the affected areas could not be pinched. Systemic examination was unremarkable.

Apart from a raised blood sugar level, his other hematological and biochemical parameters such as a complete blood count, ESR, liver and renal function tests, thyroid and immunoglobulin profile were normal. Skin biopsy for histopathologic examination showed a thickened reticular dermis with swollen collagen bundles separated by clear fenestrations suggestive of scleredema (**Figures 2** and **3**).

Discussion

Scleredema, a rare collagen disorder, was first reported by Piffard in 1876. The term scleredema is a misnomer because neither sclerosis nor edema is found on microscopic examination. Clinically it is characterized by symmetric diffuse induration of the upper part of the body due to deposition of hyaluronic acid in the dermis.

Graff described three types of scleredema adultorum.[1] Type 1 is the classic type, which was first described by Buschke in 1902. It characteristically develops following a febrile

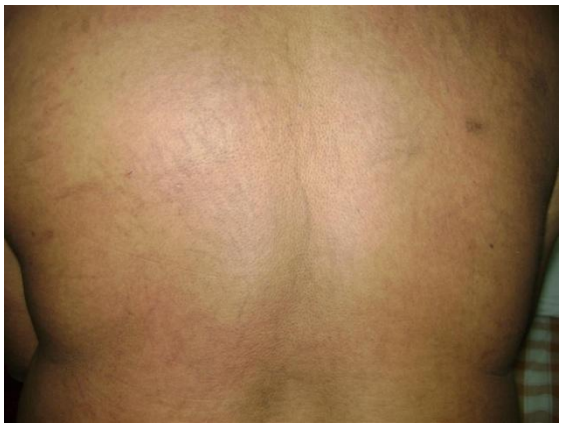

Figure 1 Clinical picture showing indurated skin of the back.

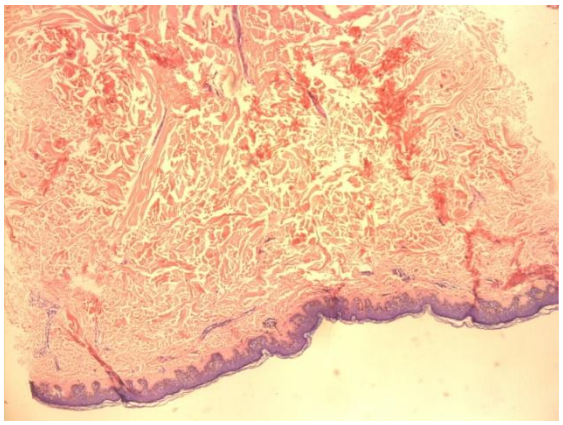

Figure 2 Photomicrograph showing thickened collagen bundles in the dermis (10X).

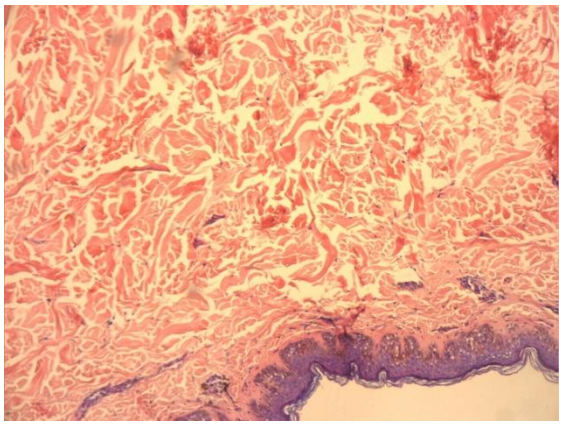

Figure 3 Photomicrograph showing close up of the thickened collagen bundles in the dermis separated by fenestrations (20X).

illness and resolves completely in several months to years. Types 2 and 3 are not associated with a febrile illness, and tend to follow a slow progressive course. Type 2 may be associated with the paraproteinemias including multiple myeloma. Type 3 termed as "diabetic scleredema" is usually seen among obese middle aged men with insulin dependent diabetes mellitus.[2] Risk factors in diabetics predisposing them to scleredema are long duration of the disease, presence of microangiopathy, overweight and the need for insulin.[3] The induration in scleredema is non pitting with no demarcation between normal and abnormal skin. Brownish pigmentation may be seen in the indurated areas. The pathogenetic mechanism of the induration of skin in scleredema is postulated to be due to glycolization of the collagen secondary to hyperglycemia. Consequences of long standing scleredema could be a decrease in motility of the shoulders, impairment of respiratory function and sleep apnea syndrome. Rho *et al*.[4] reported a series of 11 patients with diabetes-associated scleredema, where scleredema lesions improved partially in 5 patients with well-controlled diabetes. This suggests that poor diabetic control may be an etiologic factor in scleredema.

Diabetic scleredema is usually refractory to the treatment with a long protracted course; however, there are isolated reports of improvement with corticosteroids, cyclosporine, methotrexate, UVA1 phototherapy,[5] available in the literature.

Our patient did not have any obvious risk factors for the development of scleredema except for uncontrolled diabetes. His diabetes was brought under control with diet management , exercise and insulin injections and at two months of follow up there was mild softening of the lesions on his back.

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Successful treatment of recalcitrant keloid – a combined approach

Sir, keloids are the result of an overgrowth of dense fibrous tissue that usually develops after healing of a skin injury. The tissue extends beyond the borders of the original wound, does not usually regress spontaneously. Although the basis for keloid formation has not been fully delineated, an imbalance of matrix degradation and collagen biosynthesis resulting in excess accumulation of collagen in the wound have been postulated to be the primary biochemical features of keloidal lesions.[1,2] Fibroblasts of keloidal lesion produce increased amount of collagen per cell compared with normal fibroblasts.[3] There are no set guidelines for the treatment of keloids. Treatment has to be individualized depending upon the distribution, size, thickness, and consistency of the lesions and association of inflammation.[4] Treatment modalities for keloids and hypertrophic scars include compression garments, radiation, excision, intralesional injections, cauterization, cryotherapy, laser surgery, and silicon gel dressings. We report a case of recalcitrant keloid treated successfully with a new combined approach.

A 21-year-old male driver, presented with asymptomatic, solid, raised lesion on the skin over the dorsal aspect of right first metacarpal and thumb since past 5 years. There was history of minor accidental trauma at the site of lesion. Cutaneous examination revealed single well-defined, oblong, stony hard, non tender keloidal growth on the skin involving dorsal aspect of right first metacarpal and thumb (**Figure 1**). The surface was smooth and devoid of hair follicles. Systemic examination and routine blood investigations were within normal limits. We initially planned to give intralesional corticosteroid injections. The lesion was so hard that it was very difficult to insert the injection needle and push the drug intralesionally. We tried topical potent corticosteroid cream and cryotherapy using liquid nitrogen to soften the lesion. But the patient could not tolerate the pain due to cryotherapy and also developed blister. After 1 month, since there was no improvement in the consistency of the lesion, we decided to go for debulking of the stony hard fibrous tissue with the help of radiofrequency surgery under local anaesthesia followed by intra- and post-operative intralesional injection of corticosteroid triamcinolone acetonide. Small area of hard fibrous tissue was debulked initially (**Figure 2**) followed by complete debulking of remaining tissue (**Figure 3**). Initially we injected 40 mg of triamcinolone acetonide injections intralesionally, one just after debulking and other after 3 weeks. Then we injected 10mg injections intralesionally once in 3 weeks for 3 sessions. Gradually the lesion healed up without any major complication. At the end of 3 months, the