

## The diabetic back: type 3 scleredema

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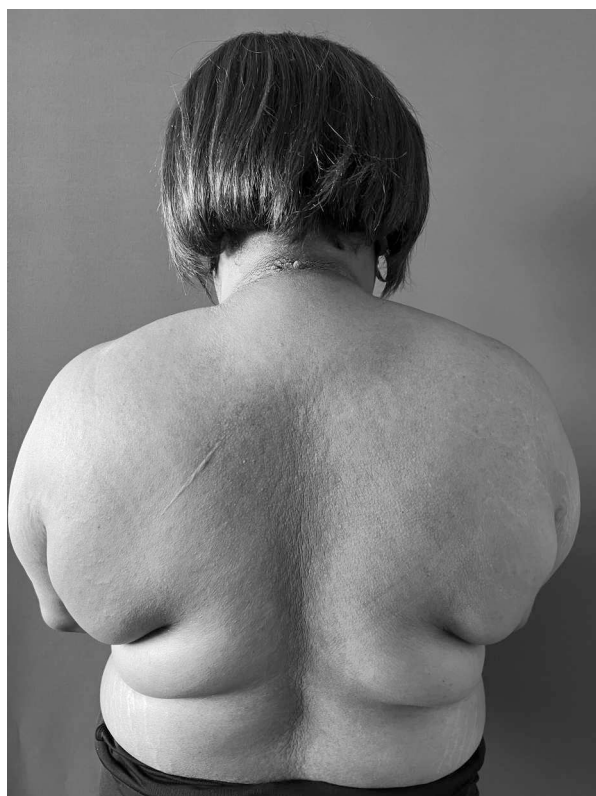
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A woman in her 40s presented to the dermatology clinic with a rash on her back and increasing difficulty changing her clothing for two years. She denied a preceding febrile illness. She had poorly controlled type 2 diabetes mellitus with a glycated haemoglobin (HbA1c) of 12.5%. Physical examination showed (Figure 1):

- (1) hyperpigmented velvety plaques in the axilla and posterior neck;
- (2) exophytic flesh-coloured tags on the neck and
- (3) a hard indurated plaque over the upper back and shoulders with a wrinkled *peau d'orange* skin appearance.

Active shoulder abduction was reduced to 60 degrees. A clinical diagnosis of acanthosis nigricans and acrochordons was made. A punch biopsy of the back showed a normal epidermis, a thickened dermis with broad collagen fibres and widened



**Figure 1:** (1) Hyperpigmented velvety plaques in the axilla and posterior neck: acanthosis nigricans; (2) exophytic flesh-coloured tags on the neck: acrochordons, and (3) a hard indurated plaque over the upper back and shoulders with a wrinkled *peau d'orange* skin appearance: scleredema.

interfibrillar spaces. Periodic acid-Schiff staining confirmed increased mucin. On serum protein electrophoresis no monoclonal band was observed. A diagnosis of scleredema type 3 was made. She received narrow-band UVB phototherapy for three months, resulting in improved shoulder abduction to 90 degrees and reported ease in dressing. Her glycaemic control was concurrently optimised in consultation with endocrinology.

Scleredema is a scleromucinous connective tissue disease characterised by non-pitting induration of the skin, typically affecting the posterior neck, shoulders, and upper back. Unlike systemic sclerosis, acral areas are spared. Although primarily cutaneous, extracutaneous features such as myositis and dysphagia may occur.<sup>1,2</sup> Mechanical sequelae with decreased mobility impact patient quality of life and can be used to monitor disease.<sup>2</sup>

The three types of scleredema are classified by their associations: type 1 with febrile infections (typically viral or streptococcal), type 2 with paraproteinemia – particularly plasma dyscrasia, and type 3 with diabetes mellitus.<sup>2</sup> Type 1 scleredema is often self-limited, resolving within two years. In contrast, types 2 and 3 follow a chronic course. Histologically, the dermis shows broad collagen bundles separated by abundant mucin without an increase in fibroblast number. In type 3 scleredema, chronic hyperglycaemia, and/or hyperinsulinemia are thought to promote non-enzymatic glycation of dermal collagen and increased mucin production by fibroblasts.<sup>3</sup> Type 3 scleredema is estimated to affect 2.5% to 14% of patients with diabetes mellitus and is likely underdiagnosed due to its insidious onset.<sup>4</sup>

Differing treatment for the varied types of scleredema may be warranted. A systematic review of scleredema treatments identified 18 cases of type 3 disease.<sup>1</sup> Successful treatment was reported in all (100%) cases using electron beam therapy ( $n = 5$ ), UVB ( $n = 1$ ), methotrexate ( $n = 1$ ), Factor XIII ( $n = 1$ ), systemic prednisone ( $n = 1$ ), high-dose penicillin ( $n = 1$ ), and penicillamine ( $n = 1$ ). Some 67% improved with PUVA ( $n = 2/3$ , 67%). None improved with topical steroids ( $n = 4$ , 100%).

While these findings are limited by small sample sizes, subjective assessments, and potential publication bias, they support early initiation of physical modalities such as phototherapy. Although the role of improved glycaemic control in modifying disease progression remains debated, it remains essential for overall patient outcomes.<sup>2,4</sup>

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