Sclerodermic lesions after liposuction in a patient with Raynaud’s phenomenon and anti-centromeric antibodies

Sirs,
Systemic sclerosis (SSc) is an autoimmune disorder of unknown aetiology. Trauma (1) and several pharmacological substances, including epinephrine (2, 3), have been identified as possible triggering pathogenic factors of this disorder. Here we describe the case of a patient with an overlooked pre-scleroderma condition who developed scleroderma skin lesions after undergoing liposuction.

A 49-year-old Caucasian female was referred to us in June 1999, to evaluate the scleroderma condition who developed anti-centromeric antibodies (ACA) (1/2560) detected by the immunofluorescence technique (Hep 2000, Immunoccepts, Sacramento, Ca, USA). On immunoblotting these antibodies were identified as Cemp-B (80 KD) (Hep-2 Marblot, MarDx Diagnostic Inc, USA). Other serologic and humoral tests were normal including the erythrocyte sedimentation rate, C reactive protein, anti-nDNA antibodies, ENA, rheumatoid factor, cryoglobulins, C3, C4, liver and kidney function tests, thyroid hormones, muscle enzymes, and red blood cell and platelet counts. HLA typing was A2-A30 (19), B51 (5) B18/BW4BW6, CW2CW7, DR3 (17) DR52, DQ2. No signs of oesophageal, lung or cardiac involvement were found on oesophagomamanometry, chest X-ray, respiratory function tests and echocardiogram. Periungual capillaroscopy showed a typical scleroderma pattern characterised by the presence of dilute dilations and some giant loops in the absence of avascular areas. The skin biopsy showed typical scleroderma lesions characterised by the proliferation of collagen fibres arranged in bundles in both the superficial and deep derma, together with scarce mononuclear cell infiltrates and the disappearance of cutaneous appendages.

Liposuction is a surgical technique used to remove fat deposits and, like any operation, presents potential health risks (4). Necrotising fasciitis is the most frequent local complication, while only 3 cases of cutaneous hyperpigmentation due to local iron deposition have been described (5). To our knowledge this is the first case reported in the literature of a patient affected by an overlooked prescleroderma condition who developed these lesions only at the sites of liposuction. It is worth noting that these lesions remain confined to the same areas after 3 years of follow-up. The prescleroderma condition (6) was indeed characterised by RP, typical capillaroscopy findings and by high titres of ACA suggestive of a limited form of SSc. In our case we hypothesize that the surgical trauma or the drugs used for the local anaesthesia, or both factors, might have exert a pathogenic role in triggering scleroderma in a predisposed person. In particular trauma has been described as pathogenic for SSc in predisposed subjects where the predisposition might be represented by HLA-DR52 allele (1). Interestingly this allele was also present in our case. The previous silicon implant might be intriguing but there is a large body of information (7,8) suggesting a random association more than a cause-effect relationship between silicon implants and autoimmune disorders.

In conclusion the present case suggests that in predisposed individuals, among the potential adverse effects of liposuction, scleroderma should be included. Before liposuction, the anamnesis and physical examination should include a careful evaluation of the signs and symptoms of systemic connective tissue diseases and, if found, the consultation of a specialist should be recommended, especially in the case of patients with a history of RP.

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References

Treatment of TRAPS with etanercept: Use in pediatrics

Sirs,
Tumor necrosis factor-alpha receptor-associated periodic syndrome (TRAPS) is a recently described autosomal dominantly inherited disorder that manifests clinically as recurrent episodes of fever, erythematous rash, myalgia, arthralgia, abdominal pain and serositis (1). In contrast with familial Mediterranean fever (FMF), episodic attacks in TRAPS tend to be of longer duration, more responsive to glucocorticoids, and unresponsive to colchicine prophylaxis. The underlying defect in TRAPS is a mutation of the gene encoding the p55 TNF receptor (2). It is not yet clear whether excessive inflammation results from decreas-

Fig. 1. Scleratrophic hypopigmented cutaneous lesions developed in the site of liposuction on the lateral aspect of the right thigh.