

Prostaglandin E₁ Improves Necrobiosis Lipoidica

Sir,

Necrobiosis lipoidica (NL) is a granulomatous disorder, which is clinically characterized by discoid, sharply demarcated, atrophic lesions with a yellowish to brownish sclerotic hard centre. Ulceration is a possible complication, and the pathogenesis of this disease still remains unclear. Most commonly the extensor surfaces of the lower extremities are affected. Patients very often report a preceding trauma as causal event (1). Moreover, management of NL is still a challenge with a plethora of frustrating therapeutic approaches. Topical or intralesional application of steroids is most recommended. Recently favourable results have been reported after using a combination of aspirin and dipyridamol, though the reported studies lacked controls (2,3). Improvement was also reported by Littler & Tschen in 1987 (4), who used oral pentoxifylline (400 mg) three times daily on one patient for 10 months, and by Haase et al. (5) who presented a series of 7 patients with this therapy. Here, we report our results with prostaglandin E₁ (PgE₁) treatment on a patient with NL.

CASE REPORT

A 33-year-old woman was referred to the department of dermatology because of painful, reticular, livid red skin lesions located on the heel, the digiti D2 and D3 of the foot and of the left extremity. She was smoking 40 cigarettes a day and was taking a contraceptive. The woman had had a history of claudicatio intermittens for the last 2 months and complained about pain even at rest. A skin biopsy showed marked vascular thrombosis with proliferation of endothelial cells, compatible with thromboangiitis obliterans (TO). As a consequence a digital subtraction angiography (DSA) was carried out, which showed stenosis of a Aae. femoralis communis, profunda femoris, fibularis and tibialis posterior. On admittance to hospital, clinical examination also showed atrophic red-violaceous infiltrated ulcerative patches, each about 5 cm in diameter, on the extensor surface of the left lower extremity, which had a history of about 5 years according to the patient (Fig. 1). Clinically the lesions were diagnosed as NL, which was in accordance with the result of the histopathological examination of a biopsy taken 3 years earlier. Treatment of the TO was started while still performing the DSA by injection of 700,000 IU urokinase and plasminogen (5,000 IU) in combination with a percutaneous transluminal angioplasty. Under this treatment the stenosis of the A. femoralis communis resolved but the other stenoses remained unchanged. An additional therapy with PgE₁ was started with a daily dosage of 4 mg, application intravenously twice daily for 6 weeks. Under this treatment the skin changes referring to TO



Fig. 1. Necrobiosis lipoidica. Status on admittance to hospital.



Fig. 2. Necrobiosis lipoidica. Status after 6 weeks of PgE₁ treatment.

resolved. Moreover, during the treatment the NL lesions also flattened gradually and changed their colour to a more brownish-yellowish appearance with considerable improvement (Fig. 2).

DISCUSSION

Transcutaneous measurement of oxygen and carbon dioxide pressure in the periphery of NL lesions has disclosed hypoxia in NL (5). The mechanism of increasing demand of oxygen and glucose in lesional skin of NL may be partially reversed by PgE₁. Such a mechanism could account for the significant improvement of the lesional skin in our patient. The reported case illustrates a possible pathogenetic role of vascular changes in NL and the potential therapeutic value of PgE₁ in this disease. A controlled study is necessary to evaluate the proposed benefit of PgE₁ in patients with NL.

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