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Treatment of Necrobiosis Lipoidica Diabeticorum by Hyperbaric Oxygen

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Necrobiosis lipoidica diabeticorum is a chronic cutaneous complication of diabetes mellitus with microangiopathy as an important pathophysiologic factor. Because of the known success of hyperbaric oxygen in the treatment of chronic non-healing wounds, we used this mode of therapy to treat a diabetic patient with ulcerated necrobiosis lipoidica of 7 years' duration, refractory to medical and surgical treatment. The patient received daily sessions of hyperbaric oxygen therapy. There was considerable improvement during the course of the treatment, with complete closure of all the ulcerations after 98 sessions. The success of this treatment emphasizes the role of hypoxia in the pathogenesis of the lesion. This simple and safe treatment method may be a good solution for patients with chronic non-healing necrobiosis lipoidica which fails to respond to other therapeutic approaches. ***Key words: Diabetes mellitus; Microangiopathy; Tissue hypoxia.***

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Necrobiosis lipoidica diabeticorum is a chronic cutaneous complication of diabetes mellitus. Similar lesions may appear in non-diabetic patients. The characteristic typical plaques, with indurated periphery and central atrophy, are prone to ulceration which is often refractory to medical and surgical treatment (1). Spontaneous remissions occur in less than one fifth of the patients, with residual scarring and atrophy. Systemic corticosteroids with proper adjustment of insulin, fibrinolytics, antiplatelet agents and local surgical treatments are all described as being helpful. Despite the many reports of effective treatment, a critical review of the literature states that none is uniformly effective (2). Corticosteroids were recently reported to be of benefit (3). However, this therapy is not always tolerated in diabetic patients.

Diabetic microangiopathy is an important pathophysiologic factor in the development of necrobiosis. Histologic changes include vasculopathy with deposits of immunoreactants (4), inflammation and thickening of the vessel walls, occasionally leading to occlusion (5). This results in significant hypoxia in the area of the wound (6).

The adjunctive use of hyperbaric oxygen can restore a favourable cellular milieu, in which the wound healing process is enhanced (7). The failure of many different modes of therapy prompted us to administer hyperbaric oxygenation as a rational treatment for long-standing refractory necrobiosis lipoidica diabeticorum.

CASE REPORT

A 44-year-old woman was referred to us because of necrobiosis lipoidica. Her medical history included appendectomy, hysterectomy, peptic ulcer and severe pulmonary infection with ARDS. She had no allergy or atopy. Her father had suffered from pemphigus vulgaris. Insulin-dependent diabetes mellitus had developed at the age of 24. She had been suffering from bilateral peritibial lesions for 7 years. The clinical diagnosis was necrobiosis lipoidica. Biopsy revealed necrobiotic changes in the collagen at the level of the dermis. Inflammatory infiltrates, with a predominance of histiocytes and giant cells, were observed around the necrobiotic foci. Repeated wound cultures were negative. She had been treated by a variety of topical and systemic approaches: topically by antiseptics, hypochloric solution (EUSOL), silversulfa diazine, hydrocolloid dressings and intralesional injections of corticosteroids; systemically with corticosteroids, antiplatelet agents, antibiotics and dapsone. None of these had resulted in improvement.

The lesions were bilateral peritibial plaques 7 cm in diameter, well demarcated, and shiny reddish brown in colour. The centres were atrophic, with severe multiple ulcerations (Fig. 1), which had appeared 2 years after the lesions were first observed. The lesions were not sensitive to touch.

The patient was given 98 daily sessions of hyperbaric oxygen therapy, breathing 100% oxygen at 2.5 atmospheres absolute for 90

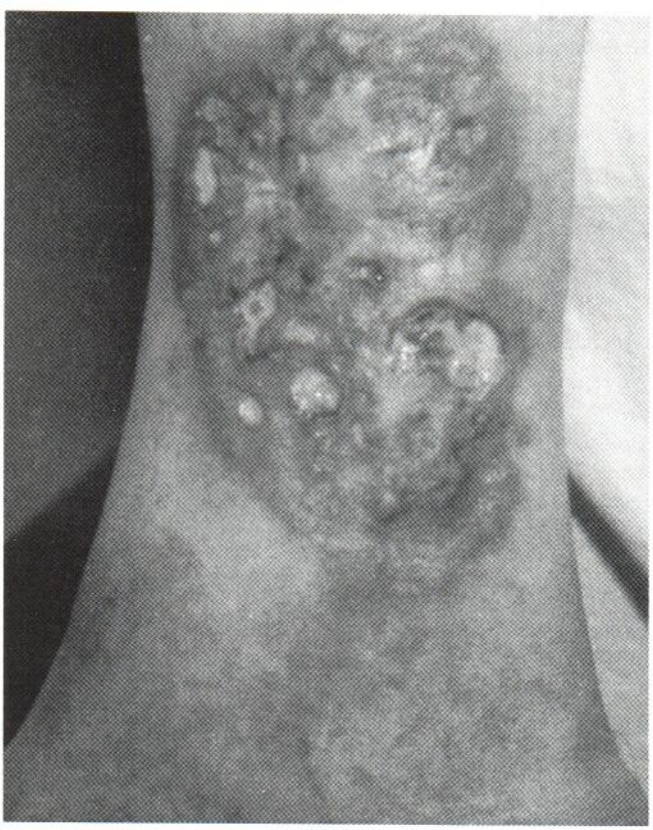

Fig. 1. Typical lesion of ulcerated necrobiosis lipoidica diabeticorum after 7 years of treatment failure.

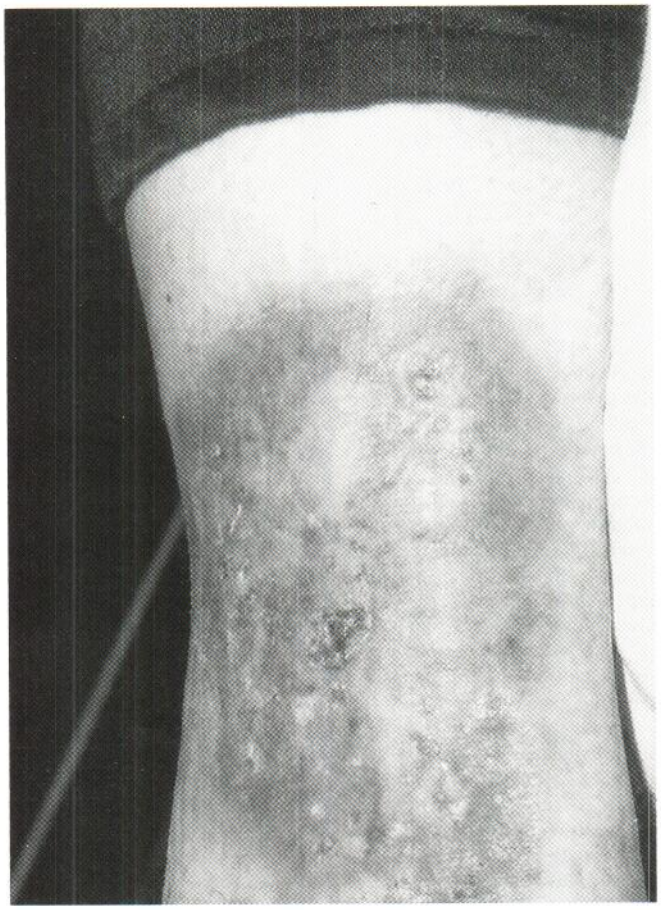

Fig. 2. Closure of all ulcerations after 98 daily sessions of hyperbaric oxygen therapy.

min in a multiplace hyperbaric chamber. During the course of the hyperbaric therapy she continued to receive the same topical treatment. The patient was evaluated after every 10 sessions. There was considerable improvement during the course of the treatment, with complete closure of all the ulcerations after 98 sessions (Fig. 2). Only local atrophy remained. The treatment had no side-effects. After 8 months of follow-up there were no ulcers in any of the lesions.

DISCUSSION

We report a diabetic patient with necrobiosis lipoidica diabeticorum. Hyperbaric oxygen treatment as an adjuvant to the topical management of the disease brought to an end more than 7 years of suffering due to the necrobiotic lesions. No other therapy, including systemic corticosteroids and antibiotics, topical debridement and hydrocolloid dressings, had produced any benefit.

Hyperbaric oxygen therapy is the intermittent inhalation of 100% oxygen at a pressure greater than 1 atmosphere. Animal studies, clinical trials, and clinical experience have shown this therapy to be beneficial in a number of indications (8, 9). Its therapeutic influence is due to the physiologic effect of hyperoxia. The inhalation of oxygen at high pressure leads to an increase in the amount of oxygen dissolved in the plasma. The correction of wound hypoxia by hyperbaric oxygen therapy results in the restoration of fibroblast proliferation, collagen synthesis and capillary angiogenesis (10).

Hyperbaric oxygen treatment is a known therapy for the chronic non-healing wound. The best results are achieved in the diabetic wound, where microangiopathy, ischaemia and hypoxia are important pathophysiologic factors (7). Studies have demonstrated a 70–90% success rate in diabetic wound healing by the addition of hyperbaric oxygen therapy to the treatment regime (11).

Transcutaneous measurement of the oxygen partial pressure in the area of necrobiosis lipoidica reveals significant hypoxia, even more pronounced on the inflamed periphery. The inhalation of 100% oxygen results in a marked increase in the transcutaneous oxygen partial pressure at the site of the lesion, though values are still significantly lower than for normal skin (6). These findings indicate the importance of the vascular-hypoxic milieu of necrobiosis lipoidica and may explain the encouraging success of treatment with hyperbaric oxygen.

Patients with chronic non-healing necrobiosis lipoidica which does not respond to the generally accepted but unsatisfactory therapy might obtain significant benefit from the addition of HBO. Although the latter is expensive, in the cure of such a long-standing disease it is cost-effective. Further experience and a controlled study are recommended to establish the efficacy of this simple and safe mode of therapy.

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