Wound Healing with a New Growth Factor Formula

Sir,

Venous ulcers continue to be a major socioeconomic burden. The prevalence has been estimated to be as high as 0.5% of the total population. The etiology of these ulcers has not been fully elucidated. At present, the ulcers are normally treated at out-clinics or by district nurses, costing an enormously large amount of money. Many treatment modalities are currently available for wound healing, such as dressings, foams, creams and ointments. Normally, patients are also treated with compression bandages. However, many of the wounds continue to be a burden for patient and doctor.

The culturing of human cells has been developed in an effort to provide a biological covering of epidermal cells over extensive burn wounds (1). The optimal culture medium gel for keratinocytes contains amino-acids, vitamins, sugars, inorganic salt, trace elements, growth hormone, insulin, triiodothyronine and transferrin. Recently a new wound healing gel, “Cariel Dermal” (Life Medical Sciences, New York, USA), has been developed containing all these elements. This gel has been demonstrated to be effective in accelerating the wound healing process in animal studies and unrandomized human studies (2). We set up a well-controlled trial to evaluate the effects of this new formula. In this pilot study we treated two groups: one with the new formula and one with the best standard treatment for leg ulcers, which is in our opinion Comfeel hydrocolloid dressings (Coloplast, Humlebaek, Denmark). Dressing changing took place every other day and the patients were treated at home. Each ulcer was diagnosed as a venous leg ulcer by Doppler sonography and light-plethysmography. Each patient was furthermore treated with standard non-elastic compression bandages. Evaluations took place before treatment, at 4 and 8 weeks. Standardized slides were used at each visit. They were processed by a specially designed computer program to analyse the progress in wound healing. The total amount of patients needed to reach statistical significance was calculated at 60 patients in each group.

So far we have treated 10 patients in the study group and 10 in the control group. After 4 and 8 weeks of treatment the study group already showed a mean decrease in wound surface of 201 and 340 mm² (s.d. 211 and 281). In the control group these figures were 164 and 252 mm² (s.d. 147 and 326). Some patients showed a mild maceration around the wound edge, which improved in time. No other side-effects were seen.

The new growth factor gel Cariel Dermal seems to be a new

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Discoid Lupus Erythematosus during Treatment with Cyclosporine

Sir,

Few studies have investigated the use of cyclosporine in the treatment of discoid lupus erythematosus (DLE) (1–3). We report an unusual observation of a case of DLE occurring in a patient who was treated with cyclosporine because of severe plaque psoriasis.

CASE REPORT

A 48-year-old man was admitted to our department in 1992 for evaluation of persistent psoriasis. The disease, which had been recurring for 5 years, was characterized by large plaques affecting his trunk, limbs and scalp. Topical corticosteroids, dithranol, keratolytics and sun exposure had been ineffective. Physical examination, apart from his skin, was normal. Laboratory investigation showed all values to be within normal range, apart from a hypertriglyceridemia and hypercholesterolemia. For 6 months the patient was treated with PUVA therapy, with a slight improvement of the skin lesions. In January 1993 the patient was again admitted to our department because of a severe relapse of his psoriasis. Cyclosporine was started at the dosage of 3.5 mg/kg/day. Blood pressure, serum creatinine, potassium, urine acid, liver function and full blood count were monitored. His psoriasis responded well to this regimen; at the end of the first month of treatment the dosage of cyclosporine was reduced to 3 mg/kg/day. The treatment was continued for 5 months with good results, without changes in hematological or biochemical parameters. No systemic therapy was given for 7 months. In January 1994 the patient experienced a severe relapse of his psoriasis. Cyclosporine was restarted at the dose of 3 mg/kg/day. No concomitant drugs, apart from topical calcipotriol, were given. After 3 months' treatment, in a routine follow-up examination, we noted three erythematous plaques on his cheeks and forehead, with sharp borders and no scales. After 10 days these lesions had slightly worsened in redness; a new plaque had developed on the forehead. A skin biopsy was performed. Histologic examination and positive immunofluorescence findings confirmed the clinical diagnosis of DLE. We recommended the patient to avoid direct sun exposure, to use a hat, a broad-spectrum sunscreen and a glucocorticoid cream on facial lesions for 3 weeks. In the meanwhile a complete clearing of the psoriasis was observed; thus cyclosporine was gradually reduced (0.5 mg/kg every 15 days) and finally withdrawn in July 1994. The clinical lesions of DLE improved after topical steroid treatment but worsened in September, after the patient had returned from a 2-week vacation at sea in Tunis. Clinical examination showed erythematous plaques with sharp borders, plaques on the face, the V of the chest and shoulders. A new cycle of topical therapy with topical steroids on these lesions gave good results. Currently the patient is being treated only with topical calcipotriol for his psoriasis; DLE is in good remission.

DISCUSSION

Our patient experienced a DLE during cyclosporine treatment. The onset of DLE skin lesions occurred when the patient was being treated with 3 mg/kg/day of cyclosporine for recalcitrant psoriasis. The lesions of DLE worsened after withdrawal of the drug. The lack of relation between the dosage of cyclosporine and the worsening of DLE led us to exclude a causal relationship between the drug and skin eruption.

Oral cyclosporine has proved effective in some autoimmune disorders, such as pemphigus, pemphigoides, and myasthenia gravis (4). Cyclosporine proved ineffective at the dosage of 5.3 mg/kg/day in a woman with a 10-year history of DLE, unresponsive to many treatments including topical steroids, antimalarials, prednisolone and azathioprine (1). No results were observed in a group of 14 patients affected with DLE who were treated with cyclosporine 3 mg/kg/day during the first month. 2 mg/kg/day and 1 mg/kg/day during the second and third month, respectively (2). Recently Yell & Burge observed no benefits in 2 cases of DLE treated with cyclosporine at the dosage of approximately 4–5 mg/kg/day (3). These authors speculated that the therapeutic failure in their cases could be ascribed to the use of the drug after priming of the immunological response. Our observation of an onset of small lesions of DLE during cyclosporine treatment suggests that cyclosporine is ineffective in the treatment of DLE independently of the duration of the disease; however, definitive conclusions cannot be drawn from a single case.

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