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, uric 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clearing of the psoriasis was observed; thus cyclosporine was gradually reduced (0.5 mg/kg every 15 days) and finally withdrawn in July 1994.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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Milker’s Nodule – A Report of 15 Cases in the County of North Jutland

Sir,
Pseudo-cowpox is contagious and is caused by a parapox virus. Lesions are found mainly on the teats of infected cows. In man, cutaneous lesions called milker’s nodules are seen primarily on the hands of dairy farmers (1). Such cases are rare in Denmark, but in 1993 and 1994 we have seen 15 patients. Three patients developed an erythema multiforme-like eruption (Fig. 1). Treatment was mostly curettage followed by cautery. The skin eruption then faded after 1–2 weeks. The lesions of 9 of the 15 patients and 3 lesions from the teats of cows were examined histologically. Seven human and 2 cow teat lesions were also examined by electron microscopy (EM). The lesions of one of the 9 patients and one of the 3 cow teats were in the papular stage. There was vacuolation of cells in the upper third of the epidermis of all lesions. This is consistent with a viral infection. Intracytoplasmatic inclusions were also present. Two of the 9 patients had lesions in the nodular stage, in which fngershaped projections were seen in the epidermis. The dermis contained many newly formed, dilated capillaries and a dense mononuclear infiltrate. Foci of epidermal cells with vacuolation were present. In 6 of 9 patients, lesions were in the ulcerated stage. The epidermis of these lesions was necrotic, with scale-crust and ballooning intraepidermal vesiculation.

EM was carried out on both paraffin-embedded material and on tissue fixed in glutaraldehyde. Parapox virus had a characteristic oval shape and measured approximately 260 × 160 nm. The protein coat had inner and outer layers and a central electron-dense DNA core (Fig. 2). Viral particles showed a remarkable resistance to improper preparation technique.

Fig. 2. Mature viral particles show the morphological characteristics of parapox virus (×80,000).

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