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clearing of the psoriasis was observed; thus cyclosporine was gradually reduced (0.5 mg/kg every 15 days) and finally withdrawn in July 1994.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 local 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.

REFERENCES

1. Heule F, van Joost T, Beukers R. Cyclosporine in the treatment of lupus erythematosus. Arch Dermatol 1986; 122: 973–974.
2. Guarneri B, Califano L, Cannavò SP, Vaccaro M. Oral cyclosporine

in the treatment of dermatoses with pathogenesis autoimmune. Rivista di Immunologia ed Immunofarmacologia 1992; 13: 68–69.
3. Yell JA, Burge SM. Cyclosporine and discoid lupus erythematosus. Br J Dermatol 1994; 131: 132–133.
4. Thomson AW, Neild GH. Cyclosporine: use outside transplantation. Promising, but long-term follow-up is essential. BMJ 1991; 302: 4–5.

Accepted May 22, 1995.

Vito Di Lernia and Giuseppe Bisighini
Department of Dermatology, Arcispedale S. Maria Nuova, I-42100 Reggio Emilia, Italy.