Acne Keloidalis of the Scalp in a Renal Transplant Patient Treated with Cyclosporine

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Sir,

Acne keloidalis, also known as folliculitis cheloidalis, acne keloid, and dermatitis papillaris capillitii, is a dermatosis that occurs most commonly on the nape of the neck of young black males. It is characterized by the presence of small, smooth, firm papules, with occasional pustules. In a minority of cases, lesions are more numerous on the vertex or crown. Over time, the papules tend to coalesce into hairless keloid-like protuberant plaques (1). We describe here a Caucasian patient who developed acne keloidalis on the central portion of the scalp. The skin lesions appeared in this unusual location during treatment with cyclosporine.

CASE REPORT

A 60-year-old Caucasian male patient, who had undergone cadaveric renal transplantation in 1993, presented in 2003 at the routine dermatological examination that we perform for each transplant patient twice a year, with multiple skin lesions on the scalp. The lesions had appeared 3 months earlier. His current treatment was cyclosporine 250 mg daily, methylprednisolone 6 mg daily, azathioprine 50 mg daily, atorvastatin 10 mg/day and amlodipine 10 mg/day. On examination, multiple firm reddish papules with a smooth surface, 0.5 cm in diameter, were seen distributed over the central portion of the scalp. Some papules coalesced into a large plaque, with scanty pustular secretions and alopecia (Fig. 1). The eruption increased in size and extended onto the back of the neck over a period of a few weeks. Skin swabs grew *Staphyloccocus aureus* and mycological culture was negative. Testing for human papillomavirus (HPV) was negative. Treatment with topical clobetasol propionate and topical erythromycin 2% lotion was unsuccessful.

An initial punch-biopsy taken from a papule showed a dilated follicular infundibulum and, in the adjacent dermis, a marked granulomatous reaction with a few giant cells. Subsequent punch-biopsy showed a follicular and perifollicular inflammatory infiltrate with plasma cells (polyclonal κ and λ chains), lymphocytes and giant cells. The dermis exhibited a marked fibrotic reaction, with thick eosinophilic hyalinized collagen fibres (Fig. 2). The histological diagnosis was keloid acne.

DISCUSSION

Acne keloidalis most commonly affects young, black males. It occurs generally on the nape of the neck in the region of the hairline and occipital scalp (1–2). Our Caucasian patient developed a severe case of acne keloidalis in an unusual location; the vertex. The disease occurred after several years of chronic immunosuppressive therapy consisting of cyclosporine, azathioprine and corticosteroid.

An association between cyclosporine and the development of acne keloidalis has been described recently by Azurdia et al. (3) and Carnero et al. (4). Both case reports concerned organ transplant recipients treated with cyclosporine. Treatment with topical or systemic antibiotics was, as in our case, completely unsatisfac-

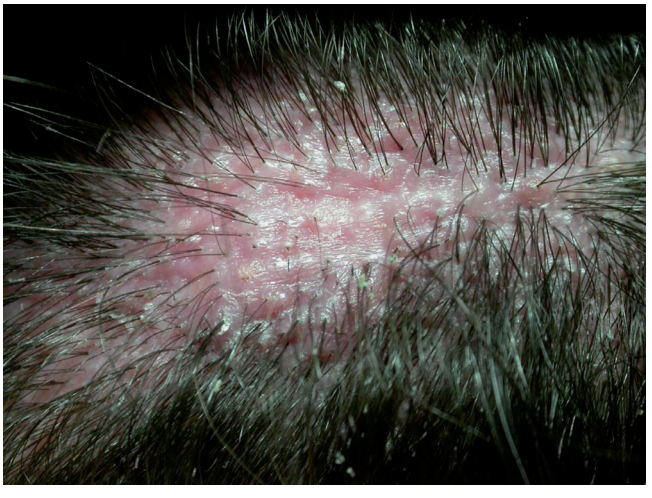

Fig. 1. Numerous firm, reddish papular lesions. Most of them coalesced into a large plaque, with scanty pustules.

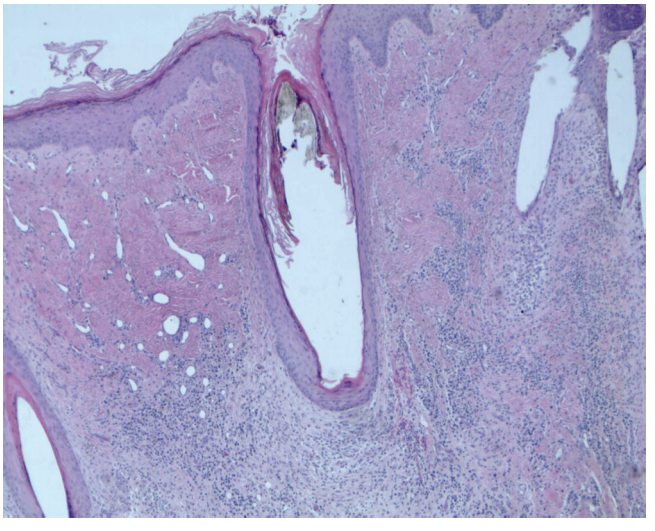

Fig. 2. A dilated follicular infundibulum and, in the adjacent dermis, a marked granulomatous reaction with fibrosis and a few giant cells.

tory. The skin lesions occurred on the nape of the neck. In our case, the uncommon location of the lesions made the diagnosis particularly difficult.

The skin is one of the major sites of accumulation of cyclosporine, and its lipophilic nature influences the concentration of the molecule in the sebaceous glands (5). This may affect the function of the pilo-sebaceous follicles, as it occurs in hypertrichosis, sebaceous hyperplasia and pilar keratosis induced by cyclosporine (6–7).

The increased hair growth may be relevant to the pathogenesis of the acne keloidalis developed in patients treated with cyclosporine. Moreover, a hypothetical initial bacterial infection, whether of the sebaceous gland or the infundibular or isthmian epithelium, may be facilitated by the immunosuppressive action of cyclosporine. The inflammation consequent to an infection or possibly to a direct action of cyclosporine on the sebaceous gland may enable the release of hair shafts into the surrounding dermis. The hairs incite further inflammation, inducing a foreign-body granulomatous reaction, with production of giant cells (1). Cyclosporine has also been documented to induce gingival hyperplasia (8), a hyperplastic response to pseudofolliculitis (9) and hypertrophic scar formation (10). An increased collagen production may play a role in the pathogenesis of these processes. There is evidence that cyclosporine is able to increase collagen synthesis and reduce collagen degradation (10). This effect could enhance the development of a fibrotic reaction of the dermis in a context of chronic granulomatous inflammation. The subsequent fibrosis might distort and occlude the follicular lumen, leading to hair retention and further inflammation and scarring.

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