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LETTERS TO THE EDITOR

Treatment of Multiple Basal Cell Carcinomas in the Scalp with Imiquimod 5% Cream

Sir,

Treatment of multiple basal cell carcinomas (BCCs) in the scalp can be most difficult from both therapeutic and cosmetic points of view. Imiquimod 5% cream (Aldara; 3M Pharmaceuticals, St Paul, MN, USA) has demonstrated both antiviral and antitumour activity in animal studies (1) and was found to be effective in the treatment of solitary BCCs in humans (2). Imiquimod is an immune response modifier promoting a Th1- or cell-mediated immune response, which is important for tumor control, via induction of cytokines such as interferon-α and via enhanced Langerhans cell migration (3).

CASE REPORT

A 36-year-old otherwise healthy Caucasian male presented with a 2-year history of multiple small pigmented papules in the left parieto-occipital region of the scalp. Biopsies showed histology consistent with nodular BCCs. He had no family history of multiple BCCs and did not show any sign of BCCs elsewhere on his body. He had been treated topically with Bezniers liquid (which contains chloral hydrate, acetic acid and ether) for alopecia areata in 1970. He had also received grenz ray treatment at 2 or 3 university hospitals in Denmark. The records for this treatment could not, however, be traced. Data were found relating to 21 exposures to grenz rays of 55 Gy in total over the period 1981–85. The relatively well-defined areas of multiple BCCs indicated that grenz rays were the major cause of the tumours. The patient had used minoxidil liniment for androgenetic alopecia in 1988–89.

Three areas of clustered BCCs above and behind the left ear and on the back of the head (Fig. 1) were treated with 3–4 weekly applications of 0.5–1 g imiquimod 5% cream. The treatment was initiated on June 25 and completed on November 29, 5 weeks after clinically successful clearance (Fig. 2). The patient received a total of 49 treatments over a period of 17 weeks. He had a 4-week rest period in August and a 1-week rest in October due to erythema, erosion and crusting at the treatment site. The patient reported no systemic side-effects. He experienced no irritation at the treatment site during the last 5 weeks of the treatment. Hair growth was not affected.

There were 7 persisting, pigmented, small (2–3 mm in diameter) macules and papules in the treatment areas when the use of imiquimod cream was stopped. Three of them were excised, showing nodular BCCs. The remaining 4 lesions appeared clinically as benign pigment naevi and were not removed. Two biopsies were taken blindly from the treated areas above and behind the left ear at the same time. They showed solar elastosis and non-specific perivascular lymphocytic infiltrate. No new lesions were seen at 5-month follow-up.

DISCUSSION

Treatment of multiple BCCs on the scalp with imiquimod 5% cream was very successful from a clinical point of view. The treatment was well tolerated. Hair growth was not affected in the treated area. It would have been difficult to obtain cosmetically acceptable results on such large areas with other treatment modalities, such as surgery or cryotherapy. Three small nodular BCCs did not respond to the treatment and had to be removed surgically. It can be speculated that continuous application of imiquimod might have eliminated them

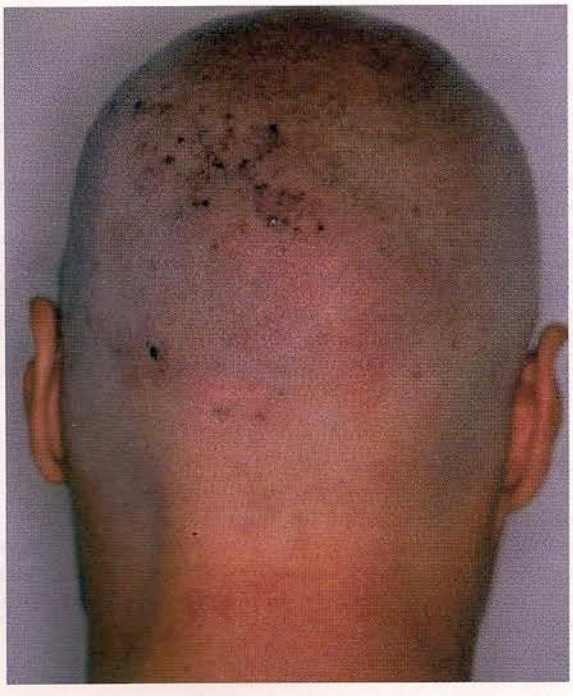

Fig. 1. Multiple BCCs in a parieto-occipital region of the scalp before treatment.

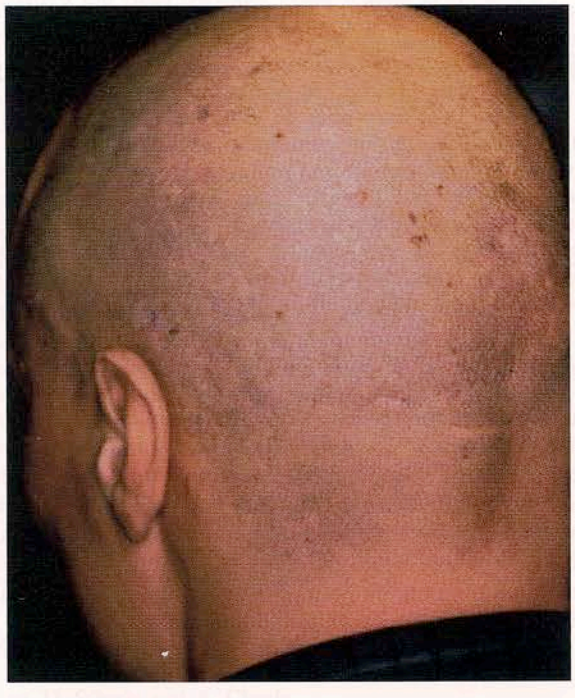

Fig. 2. Good clinical response after a 17-week treatment with imiquimod 5% cream.

eventually. The long-term effect of the treatment is not known. In a study on solitary BCCs, imiquimod gave complete histological clearance in all patients who were treated at least 3 times a week (2). In our patient, blind biopsies did not show BCCs and no new tumors were seen 5 months after completing treatment. It is likely, however, that new BCCs will develop due to the suspected underlying heavy dosing of grenz rays.

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Psoriasiform Eruption and Anticonvulsant Drugs

Sir,

The development of a psoriasiform eruption or the exacerbation of pre-existing psoriasis caused by intake of drugs is well known. Numerous drugs have been implicated as culprits: β-adrenergic blocking agents, lithium, antimalarials, non-steroidal anti-inflammatory drugs (NSAIDs) and photosensitizers (1). We report on a patient in whom the association between the anticonvulsant drugs carbamazepine and sodium valproate caused an exacerbation of psoriasis. Withdrawal of the drugs resulted in dramatic improvement of the skin lesions, pointing to a causal relationship.

CASE REPORT

A 25-year-old woman presented with a psoriasiform eruption of 2 months' duration. She suffered from epileptic seizures following herpes meningoencephalitis at the age of 8 years, for which she had been taking the anticonvulsant drugs carbamazepine and sodium valproate for the last 2 years. She had had psoriasis but was lesion-free for the last 6 years. Two years prior to examination she developed an erythematous scaly eruption in the intertriginous areas which was not alleviated by topical steroids and antifungal agents. The rash became generalized in the last 2 months. Skin biopsy was compatible with psoriasis. A drug-induced exacerbation of the psoriasis was suspected. The anticonvulsants carbamazepine and sodium valproate were substituted by other chemically unrelated medication and the skin condition improved dramatically within 14 days.

DISCUSSION

The relationship between the suspected drugs and the worsening of the skin eruption in our case is based on circumstantial evidence: lack of response to topical steroids while taking carbamazepine and sodium valproate, and subsidence of the lesions upon withdrawal of these drugs. The exacerbation of our patient's skin condition could have been induced by one or both of these antiepileptics, but more definitive evidence by rechallenge was, of course, out of the question. The literature on this subject is not consistent, with only 2 reports (2, 3) implicating the drugs in the induction or aggravation of psoriasis: Smith et al. (4) found carbamazepine beneficial in a human immunodeficiency virus-positive patient with psoriatic erythroderma, and Marron (5) found no improvement with carbamazepine in 14 psoriatic patients.

It is possible that valproic acid or carbamazepine affected the immune status of our patient in such a way as to aggravate her psoriasis, perhaps by acting as superantigens. While the mechanism of drug-induced or exacerbated skin eruptions remains to be delineated, the case reported here and the steadily growing list of drugs found to aggravate psoriasis strongly suggest that patients with psoriasis should be closely watched for the possible effect of systemic drugs on their dermatosis.

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