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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