Sir,

The anti-convulsant drug carbamazepine may induce generalized exanthema or, less frequently, severe cutaneous eruptions, such as hypersensitivity syndrome or toxic epidermal necrolysis. The development of psoriasiform eruptions and the exacerbation of pre-existing psoriasis have been recognized as common adverse reactions to beta-blockers, lithium and antimalarials (1). Additional causative drugs include angiotensin converting enzyme inhibitor, indomethacin, progesterone, and lipid-lowering drugs, such as gemfibrozil (2). Drug-induced alopecia presents as a diffuse, non-scarring loss of hair that is often reversible upon discontinuation of the drug. The drugs associated with telogen effluvium include beta-blockers (3), angiotensin converting enzyme, oral contraceptives, antithyroid medications, anticoagulants and anticonvulsant (4). We report here a case of a 52-year-old woman who experienced diffuse hair loss and a palmoplantar psoriasiform eruption apparently due to carbamazepine.

CASE REPORT

A 52-year-old woman was referred to our dermatology clinic because of diffuse hair loss and psoriasiform eruptions on her hands and feet. She had no past or family history of psoriasis, alopecia, or other skin disorders. There was no evidence of dermatitis, allergic reaction, or other causes for hair loss and psoriasiform eruption. She was diagnosed with an acoustic schwannoma 2 months previously and had been taking carbamazepine (200 mg, 3 times/day) for 3 months due to severe facial pain. The psoriasiform eruptions on her palms and soles started one month after administration of carbamazepine, and remarkable hair loss occurred one month after the appearance of the eruptions. Physical examination showed thick hyperkeratotic plaques on the palmoplantar areas and diffuse hair loss on the scalp (Fig. 1). A hair-pull test demonstrated that hairs could be extracted easily. Since carbamazepine was suspected as one of the inciting agents, the dosage of carbamazepine was reduced to 200 mg once/day. The psoriasiform eruptions began to improve one week after tapering the drug without any treatment. The palmoplantar lesions also improved (Fig. 2) and the hair loss began to stop 4 weeks after decreasing the dose. Two months later, she underwent surgical operation for the acoustic schwannoma. The eruptions cleared completely and spontaneous hair regrowth was shown after discontinuing medication.

DISCUSSION

A causative relationship between a drug and associated adverse reactions is based primarily on experience and observation. The following three factors are important in supporting the diagnosis of a drug reaction: first, the adverse reaction occurs after drug administration; secondly, as long as the patient continues to take the drug, the adverse reaction resists conventional treat-
ment; and thirdly, the adverse reaction improves within days of ceasing the drug. Above all, the rechallenge test more definitely supports the presence of a drug reaction, but a drug rechallenge is difficult to perform in practice (5). Since the suspected drug reactions in our patient improved immediately after the tapering of medication, carbamazepine should be considered a possible causative agent of hair loss and palmoplantar psoriasiform eruptions.

Although many new anticonvulsants have been introduced recently, carbamazepine has been widely used for many years in the treatment of epilepsy and other neurological diseases. Carbamazepine-induced hair loss has been reported as occurring at various rates. Phillans & Woods (4) reported 177 cases of carbamazepine-induced alopecia. A double-blind, multi-centre trial, the aim of which was to compare carbamazepine and valproic acid, showed that hair loss or changes in hair texture were seen in 6% of the patients treated with carbamazepine (6). Kohno et al. (7) reported that the frequency of carbamazepine-induced hair loss was below 2%. They suggested carbamazepine could induce two patterns of hair loss: anagen effluvium, in which the hair loss starts within one week, and telogen effluvium, in which it starts after 2–3 months. The type of hair loss in our patient seemed to be telogen effluvium because the hair loss started 2 months after initial administration of the medication.

It is estimated that about 3% of patients receiving carbamazepine will develop a cutaneous adverse reaction, including erythematous, morbilliform, or urticarial rashes. Recently, human herpes virus-6 has been reported to have a potential role in carbamazepine-induced hypersensitivity syndrome (8). Case reports of psoriasiform eruptions induced by this drug are limited (2, 9). By contrast, Smith et al. (10) reported that carbamazepine may be a therapeutic option for the treatment of HIV-positive psoriatic patients. The mechanisms of carbamazepine to induce or clear psoriasiform eruption are not known yet. Carbamazepine inhibits the uptake of noradrenaline and blocks cyclic AMP-mediated calcium influx (11). Carbamazepine or its metabolites have been suggested to play a role in the clearing of psoriasis through an immune-modulating effect. We also think that the mechanism is associated with adrenergic inflammation or the release of neuropeptides, which influence psoriasiform eruption. Physicians should recognize that this drug might produce psoriasiform eruptions or aggravate pre-existing psoriasis. Moreover, this is the first report of this type of case, in which carbamazepine induced psoriasiform eruptions and hair loss at the same time. We report that carbamazepine, which has been widely used, might induce hair loss and psoriasiform eruption concomitantly.

The authors declare no conflicts of interest.

REFERENCES