Herpes Zoster-associated Trigeminal Neurotrophic Ulcer

Sir,

Surgical rhizotomy of the dorsal trigeminal nerve root or alcohol injection into the Gasserian ganglion in patients suffering from intractable trigeminal neuralgia may lead to chronic neurotrophic ulcer (1–3). Another known cause of this syndrome is occlusion of the posterior inferior cerebellar artery with subsequent infarction of that part of the brain stem that contains the sensory root of the trigeminal nerve (4).

We have recently seen three female patients with trigeminal trophic syndrome, in two of whom an ophthalmic zoster was the precipitating factor.

CASE REPORTS

Case 1

An 85-year-old otherwise healthy woman was admitted due to a chronic ulceration in the left parietal region of 24 months' duration. The history was unremarkable except for a diagnosis of zoster ophthalmicus affecting the first division of the left trigeminal nerve approximately 12 months before development of the ulceration. Ocular complication was not reported. In spite of initial oral acyclovir, persistent chronic postherpetic pain and paresthesia occurred in the area. At admission, an 8 × 5-cm large superficial ulceration with crustformation and surrounding erythema was observed (Fig. 1A). Cultivations for pathogenic bacteria and fungi were negative. Herpes simplex virus and *Varicella zoster* virus could not be detected in the lesion. Histopathological examination of a biopsy specimen excluded temporal arteritis as the basic cause of the ulceration, and showed ulceration with minimal unspecific inflammation in the dermis. The dermal-epidermal zone was intact in non-ulcerated areas. Direct immunofluorescence test and indirect test for circulating autoantibodies were also negative. Treatment with betamethasone dipropionate cream (Diproderm) once daily for 3–4 weeks resulted in healing of the ulceration leaving a cicatricial alopecia (Fig. 1B).

Case 2

A 84-year-old otherwise healthy woman was admitted due to a chronic ulceration in the right fronto-parietal area of 24 months' duration. The lesion developed approximately 8 months after an episode of ophthalmic zoster affecting the first division of the right trigeminal nerve. Pain and paresthesia had been present constantly in the affected area since the zoster episode. Examination revealed a large area of confluent erosions in the parietal region (Fig. 2A). Relevant cultiva-

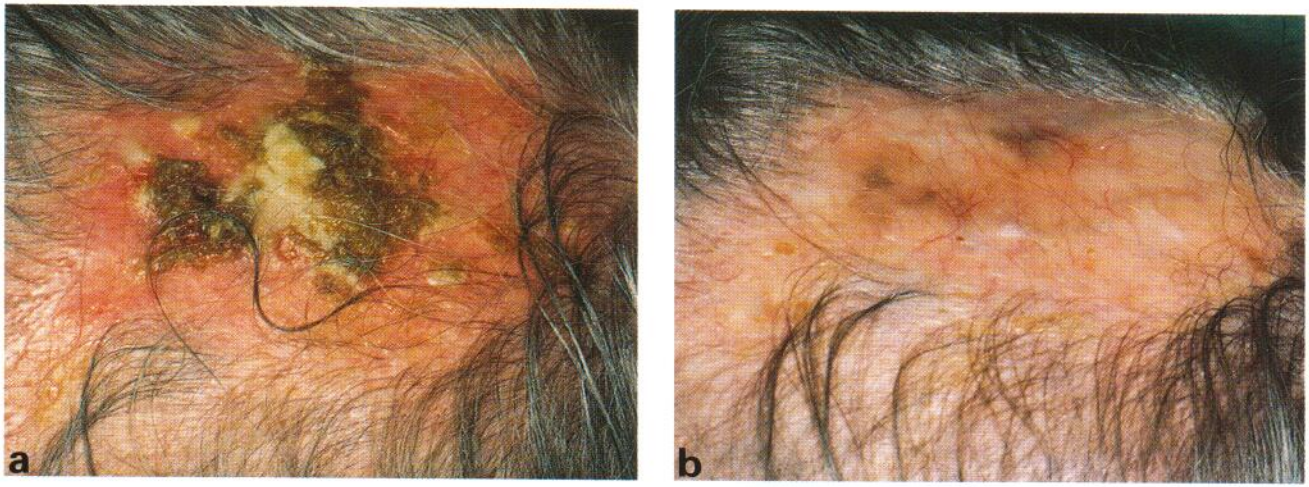

Fig. 1. Scalp ulceration induced by ophthalmic zoster before (*a*) and after (*b*) treatment with betamethasone dipropionate cream.

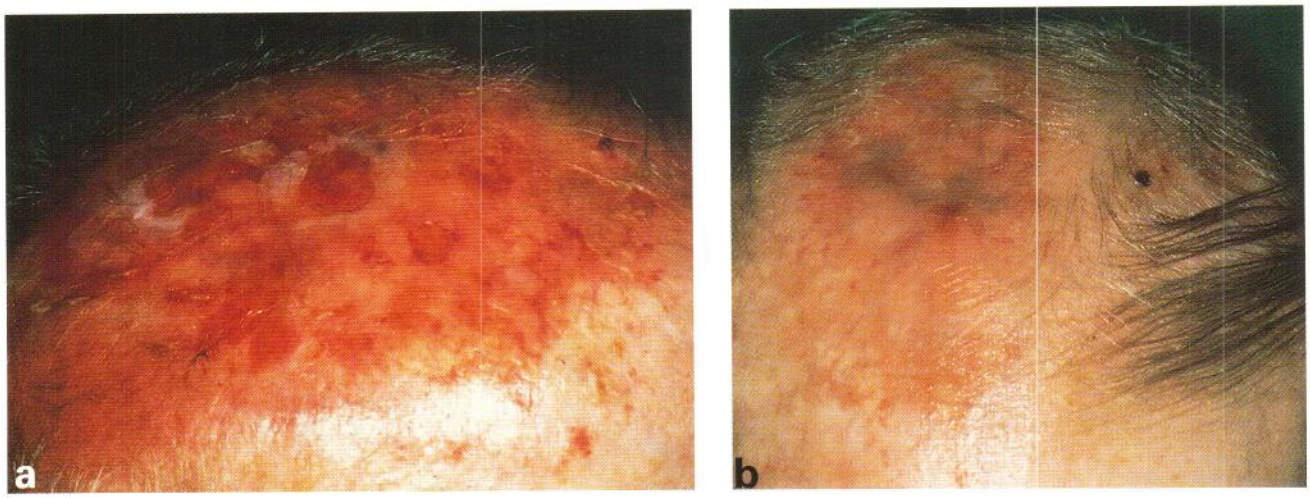

Fig. 2. Scalp ulceration induced by ophthalmic zoster before (*a*) and after (*b*) treatment with betamethasone dipropionate cream.

tions and histopathological examinations did not give clues to a specific diagnosis (see Case 1). Treatment with betamethasone dipropionate cream (Diproderm) once daily for 3–4 weeks resulted in healing of the erosions leaving a cicatricial alopecia (Fig. 2B).

Case 3

A 65-year-old woman was admitted due to a chronic ulceration of 12 months' duration located in the left frontoparietal region. The patient had a resection of an acusticus neurinorma on the left acusticus nerve 2½ years prior to the appearance of the ulceration. As sequelae to the operation, a persistent left facial paresis and facial dysesthesia developed. A 4 × 6-cm large ulceration with crust formation was seen in the left upper half of the forehead and adjacent part of the scalp. Cultivations for pathogenic bacteria and fungi were negative. *Varicella zoster* antigen was not present in the lesion. Ophthalmological evaluation was unremarkable. Histopathological examination showed fibrosis and a minimal unspecific inflammatory infiltrate in the upper dermis. Direct immunofluorescence test did not show the presence of tissue-bound immunoglobulin or C3. In spite of topical application of clobetasol cream (Dermovat) total healing of the lesion was not achieved.

DISCUSSION

To our knowledge, chronic scalp ulceration has not been described as a complication of ophthalmic zoster in immunocompetent persons. Healing of zoster lesions within a few weeks is the rule in otherwise healthy individuals, although scar formation may develop. Patients with ophthalmic zoster often have a severe inflammatory reaction in the trigeminal ganglion that can induce both sensory and motor neuropathy. We suggest that the peripheral neuropathy is the pathogenesis of the observed chronic scalp ulcerations seen in our patients (cases 1 and 2), and that these lesions should be considered analogous to the facial ulceration previously described in patients operated on for severe trigeminal neuralgia. This is supported by the finding of exactly similar face and scalp ulceration in a woman with traumatically induced damage to both the trigeminal and facial nerve during surgery for an acusticus neurinoma (Case 3).

A careful examination of our patients has excluded other specific causes of scalp ulceration including arteritis temporalis, lupus erythematosus and bullous diseases, especially the Brunsting-Perry variant of cicatricial pemphigoid. The possibility of the lesion being at least partly caused by excoriation cannot be excluded although the patients did not support this. The lesion in our patients had clinical and histological similarities with "erosive pustular dermatosis of the scalp" (5). Pye et al. (5) also reported a marked response to potent local steroids. Grattan et al. (6) reported 12 patients with "erosive pustular dermatosis of the scalp" and 6 of these followed shingles in the ophthalmic division of the trigeminal nerve.

Treatment of trigeminal trophic syndrome is unsatisfactory. A plastic surgical approach with use of innervated flaps to cover the defect has been reported (7) as well as transcutaneous electrical stimulation (8). Our 2 patients with zoster associated lesions healed during topical treatment with a potent corticosteroid. The basis of this therapeutical effect is at the moment unknown.

REFERENCES

1. McKenzie KG. Observations on the results of operative treatment of trigeminal neuralgia. Can Med Assoc J 1933; 29: 494–496.
2. Freeman AG. Neurotrophic ulceration of the face with erosion of the ala nasi in vascular disorders of the brain stem. Br J Dermatol 1966; 78: 322–331.
3. Finlay AY. Trigeminal trophic syndrome. Arch Dermatol 1979; 115: 1118.
4. Walton S, Keczkes K. Trigeminal neurotrophic ulceration – a report of four patients. Clin Exp Dermatol 1985; 10: 485–490.
5. Pye RJ, Peachey RDG, Burton JL. Erosive pustular dermatosis of the scalp. Br J Dermatol 1979; 100: 559–566.
6. Grattan CEH, Peachey RD, Boon A. Evidence for a role of local trauma in the pathogenesis of erosive pustular dermatosis of the scalp. Clin Exp Dermatol 1988; 13: 7–10.
7. Tatnall FM, Stearus M, Sarkany I. Trigeminal trophic syndrome. Br J Dermatol 1985; 113 (Suppl 29): 86–87.
8. Westerhof W, Bos JD. Trigeminal trophic syndrome: a successful treatment with transcutaneous electrical stimulation. Br J Dermatol 1983; 108: 601–604.

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