TRIGEMINAL TROPHIC SYNDROME FOLLOWING HERPES ZOSTER OPHTHALMICUS
(Case report)

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Summary

A case of trigeminal trophic syndrome presenting with painless ulceration of the scalp following herpes zoster ophthalmicus is presented The pathogenesis of neurotrophic changes in the trigeminal territory is reviewed.

Trophic disturbances are changes appearing in tissues that have been deprived of certain qualities of sensation, namely those of pain, temperature and light touch and they conform sharply to the neuro-anatomic delineations[1]. Trophic lesions in the trigeminal field especially the forehead, scalp and malar region are extremely uncommon[1,2]. Nevertheless neurotrophic changes in the trigeminal area of non-herpetic type have been described following trigeminal neurectomy[2]. Recently a case of trigeminal trophic syndrome following intracranial trigeminal sensory rhizotomy has been described[3]. We are reporting a rare case of trigeminal trophic syndrome following herpes zoster ophthalmicus.

Case Report

A 40 year old sikh gentleman was admitted to the hospital with right sided herpes zoster ophthalmicus. He developed neuroparalytic keratitis for which tarsorrhaphy was done. Since the onset of the illness he had persistent unbearable pain in the right forehead region. There was no other neurological illness. The patient was treated with analgesics, sedatives and systemic corticosteroids and was later put on carbamazepine 600 mg per day. Healing took place in 6 weeks but some areas were felt raw by the patient. Six weeks later patient developed non-healing ulcers on the right side of scalp and forehead accompanied by moderately severe neuralgic pain.

Examination showed dark brownish post-inflammatory hyperpigmentation and characteristic punched out geographical scars in the distribution of the ophthalmic division of the trigeminal nerve on right side. There were four ulcers 0.5–2 cm in size on the forehead and scalp on the affected side. The ulcers were shallow, clean, non-inflammatory and non-tender. Because of tarsorrhaphy right eye could not be fully examined. Partial examination indicated that the keratitis had healed ; but that the corneal reflex remained absent. All the sensations were lost in the territory of supply of ophthalmic

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division of trigeminal nerve on the right side. Masseter, temporalis and orbicularis occuli muscles had normal strength. Other cranial nerves were normal. General physical, systemic and detailed neurological examination showed no other abnormality.

Routine invetigations on blood and urine did not reveal any abnormality. Total and differential proteins, fasting and post prandial blood sugar, blood urea and serum electrolytes were within normal limits. Blood VDRL test was non-reactive.

Discussion

Trophic changes are uncommon on the face possibly because it is instinctively better protected from trauma than other parts of the body and has better blood supply[1]. Nevertheless such changes have been documented following trigeminal sensory neurectomy/rhizotomy[2,3] and leprous trigeminal neuritis[4]. Trophic lesions of the face have been reported in posterior inferior cerebellar artery syndrome and syringobulbia as a sequel to the interruption of trigeminal sensory pathway in the brain stem[1]. In the absence of any demonstrable anaesthesia such changes have been documented in post-encephalitic parkinsonism[5].

Herpes zoster is a disease of varying severity. In its worst form it may have devastating effects on sight and even life. On the other extreme it may be so mild as to pass unnoticed. Ocular involvement complicates approximately 50 percent of herpes zoster ophthalmicus[7]. Chronic skin lesions in the form of typical punched out geographical scars, pigmentation, depigmentation, graying and loss of hair, acne formation, cicatrix production over skin, eyelids, conjunctiva, sclera and cornea are well known. Severe chronic neuralgia complicates 7 percent of the patients[6]. Trophic ulcerations in the region have not been reported.

The exact pathogenesis of the trophic changes and the cause of its rarity on the face are unknown. In addition to the repeated trauma to the anaesthetic area, autonomic and vascular changes are incriminated. Vasospasm and giant cell granulomatous arteritis have been documented radiologically and pathologically in internal and external carotid circulation following herpes zoster ophthalmicus[8,9]. The role of these vascular changes in the development of trophic ulcers is unknown. Carotid arteriography was not carried out in this patient because of the hazard of increased incidencc of vasospasm reported in the post herpetic phase[8].

Prevention of infection, trauma and protective dressings are the mainstay in the management of the disease. The usual course of post-herpetic neurological sequlae in general is one of slow improvement. The lesions do heal gradually, and not much more is known about this because of its rarity.

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