Case Report

Neurotrophic Corneal Ulcer Complicating: A Case of Posterior Fossa Bleed

Sneha Batra, Saumya

Rajendra Institute of Medical Sciences, Ranchi, India

Corresponding author: Sneha Batra

Abstract:

Purpose: To report a rare case of neurotrophic corneal ulcer in a 14 year old girl with a history of posterior fossa bleed.

Methods: On examination, patient had a corneal ulcer in the Right eye (visual acuity – counting fingers at 2 feet), diminished corneal sensation, end-gaze nystagmus in all directions of gaze, cerebellar ataxic gait, and residual hemiparesis on the right side of her body. MRI showed haemorrhage in the posterior fossa. Treatment was started on the lines of neurotrophic corneal ulcer.

Results: Ulcer healed completely by the 8th day, and visual acuity improved to 6/24. Corneal sensation remained impaired for 3 months.

Conclusion: Neurotrophic keratitis warrants a full neurological evaluation to locate the exact cause of Vth Nerve dysfunction which could be a result of a life endangering event.

Keywords: neurotrophic keratitis; corneal ulcer; cerebellar stroke; posterior fossa.

Introduction:

Neurotrophic corneal ulcer is defined as keratitis which occurs due to loss of Trigeminal innervation to the cornea resulting in partial or complete anaesthesia[1]. Here we present a case of unilateral corneal ulcer in a 14-year old girl with a history of ipsilateral posterior fossa bleed.

Case Report

A 14-year old girl presented to our outpatient department on 17th July, 2014, with a complaint of sudden onset pain, redness and dimness of vision in the Right eye since 2 days. She had a history of acute onset right sided hemiparesis since three weeks, which had been accompanied by loss of consciousness for 3 days. She was admitted in the Neurosurgery department at our hospital for the same, and diagnosed as a case of stroke due to bleed in the posterior fossa (involving cerebellum and adjoining part of brainstem) (Fig 1 & 2). She also gave a history of inability to close the right eye completely during the initial part of the episode.

Her best corrected visual acuity was recorded as RE – Counting Fingers at 2 metres and LE – 6/6 (Snellen test types). Diffuse light examination showed mild upper lid edema, matted lashes, ciliary congestion and a large stain-positive epithelial defect in the central cornea covering the pupillary area, with horizontal Descemet membrane folds in the Right eye.(Fig 3) Corneal sensation was absent in the same eye; pupillary reaction to light was brisk and well maintained. There was presence of end-gaze nystagmus in all directions of gaze in both eyes. Anterior segment of the left eye and posterior segment examination of both eyes revealed no abnormalities. Extraocular muscle movements were full and not restricted in any gaze in either eye.

Her GCS was 15/15 at the time of presentation, and B.P was recorded as 110/70 mm of Hg. On systemic examination, there was no abnormality noted in

www.ijbamr.com P ISSN: 2250-284X , E ISSN : 2250-2858

cardiovascular, respiratory or gastro-intestinal systems. Neurological examination revealed typical cerebellar ataxic gait (reduced step frequency with a prolonged stance and double limb support duration, but normal range of motion), which was accentuated with tandem gait test. Residual hemiparesis was present in the right half of the body. Also, there was loss of sensation in all the areas supplied by the Right Trigeminal nerve (Ophthalmic, Maxillary and Mandibular areas). There was mild weakness of the muscles supplying right half of the face, but the eyelids could be completely closed at the time of presentation. There was also associated hyperacusis in the right ear. Other tests for cerebellar function were normal. No neurological abnormality was noted in the left half of the body.

Management

Based on the above findings, a diagnosis of neurotrophic corneal ulcer was made (with an added neuroparalytic component as the precipitating factor). The patient was admitted and started on a course of preservative-free antibiotic and lubricant eye drops, and lubricant eye ointment, coupled with daily antiseptic dressing and eye-patching. The ulcer was observed for regression every day, and by the eighth day the lesion showed no evidence of stain positive epithelial defect. Her visual acuity at the time of discharge was RE-6/24 and LE-6/6. She was advised to continue the lubricant eyedrop and eye gel, and d-panthenol eye ointment was added. A search was also made for the cause of the posterior fossa bleed in consultation with the Medicine and Neurosurgery Department. Her MR Angiography at the time of discharge showed no evidence of any aneurysm or AV malformation (Fig4). A hypercoagulable state profile was also done, and no abnormality was found.

Discussion

Primary posterior fossa bleed without any antecedent history of trauma is rare, and is usually associated with hypertensive and degenerative changes[2]. In the younger age group, arteriovenous malformations and blood dyscrasias are more common causes. Location of the hemorrhage (midline vs hemispheric) is important in determining symptoms and clinical course, which is more significant than absolute hematoma size for prognosis. The more lateral the hemorrhage and the smaller the hematoma, the more likely the brainstem structures are spared and the better the prognosis[3]. Development of obstructive hydrocephalus from ventricular compression may lead to increased intracranial pressure and decreased cerebral perfusion pressure.

Brainstem damage by compression from an expanding mass in the posterior fossa is a common and feared complication, and leads to loss of consciousness, hemiplegia, and palsy of multiple cranial nerves depending upon the exact location, among other features. In this patient, it seemed that there was bleed in the right sided fourth ventricle with extension into the right ambient cistern. This caused compressive symptoms on both the ipsilateral cerebellar hemisphere, causing the typical gait, and end-gaze nystagmus; and the pons, causing loss of consciousness, hemiparesis, and cranial nerve involvement (V^{th} and VII^{th}).

The Trigeminal and Facial nerves form the afferent and efferent pathways of the corneal reflex and are crucial to the integrity of the normal Ocular Surface. While paralysis of the Trigeminal nerve causes loss of sensation from the cornea, resulting in intracellular oedema, exfoliation of epithelial cells, impairment of epithelial healing and loss of goblet cells, culminating in epithelial breakdown and persistent

www.ijbamr.com P ISSN: 2250-284X , E ISSN : 2250-2858

ulceration. Loss of acetylcholine, substance P, and growth factors from the epithelium appear to be important[4]. This results in Neurotrophic keratitis. At the same time, paralysis of the Facial nerve causes lagophthalmos, resulting in abnormal blinking and drying of the cornea despite normal tear production, resulting in Neuroparalytic keratitis.

The management in both cases warrants identification and treatment of the cause, adequate protection of the Ocular Surface with preservative-free lubricants, and temporary taping to prevent further exposure and for rapid healing of the keratitis [5].

References:

1. Kanski Jack J, Bowling B. Cornea. 7th ed. Elsevier; 2011.
2. Henry B. Dinsdale. Spontaneous Hemorrhage in the Posterior Fossa: A Study of Primary Cerebellar and Pontine Hemorrhages With Observations on Their Pathogenesis. Arch Neurol.
3. 1964;10(2):200-217.
4. Witsch J, Neugebauer H, Zweckberger K, Jüttler E. Primary cerebellar haemorrhage: complications, treatment and outcome. *Clin Neurol Neurosurg*. Jul 2013;115(7):863-9.
5. Groos Jr EB. Neurotrophic keratitis. In: Krachmer JH, Mannis MJ, Holland EJ (eds.) Cornea: Clinical Diagnosis and Management. Mosby: St Louis, 1997, pp 1340
6. Lambiase A, Rama P, Aloe L, Bonini S. Management of neurotrophic keratopathy. Curr Opin Ophthalmol 1999; **10**: 270–276.