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A CASE REPORT OF EXPOSURE KERATITIS WITH NEUROTROPHIC COMPONENT

Ophthalmology

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ABSTRACT

Damage to the cornea owing to dryness brought on by partial or insufficient eyelid closure, which results in the loss or insufficiency of the tear film, is known as exposure keratopathy. Reduced corneal sensitivity and inadequate corneal repair are two degenerative symptoms of neurotrophic keratitis. This condition reduces reflex tearing and makes the cornea more prone to injury. Melting, perforation, ulceration, and infection can all result from epithelial disintegration. Typically, milder disease is minor and easy to treat. If untreated, it may develop into an emergency that is challenging to treat.

KEYWORDS

exposure keratitis, neurotrophic keratitis, amniotic membrane, tarsorrhaphy

INTRODUCTION

Exposure keratopathy can result from inadequate or incomplete eyelid closure or less frequent blinking, which reduces the lubrication of the ocular surface. Facial nerve palsy, severe proptosis, lagophthalmos, critical unconscious patients in ICU with incomplete eye closure, herpetic keratitis are some of the causes for exposure keratopathy. If left untreated exposure keratopathy can lead to ulceration, microbial keratitis, and corneal scarring making a subject corneal blind. Exposure keratopathy can be due to decreased corneal sensations which can lead to neuropathic keratitis. Decreased or absent corneal sensations may lead to corneal epithelial sloughing leading to corneal thinning, ulceration, melting and perforation. Therefore, Early diagnosis and management along with close monitoring are essential for restoring patient's vision and outcome.

Case Report

40 year/Male came with complaints of both eyes redness and diminution of vision associated with photophobia since last 1 month. Patient was apparently alright one month back after which he experienced gradual progressive painless diminution of vision associated with whitish opacity seen in both eyes since last 1 month. patient had history of both eyes mild redness and photophobia. Patient had history of putting E/D Vigamox 6 times a day and lubricating drops 6 times a day prescribed by some local practitioner for last 15 days. Patient had a history incomplete closure of both eyes.

There were no history of discharge/watering/foreign body sensations/itching/burning sensation. There was no history of trauma or ocular suregry. Uncontrolled DM since last 5 years noncompliant to treatment. Patient had past history of Pulmonary TB 20 years back for which treatment completed for 1 year. Patient was using spectacle for distance vision since last 30 years.

On examination, No facial asymmetry, Right Eye vision was 6/24 with refraction of -2.0 D Sphere, Reduced Blink Rate 9/min, Tear Break Up Time 10 sec Lagophthalmos was present Circumcorneal congestion, Corneal infiltration with multiple epithelial defects in the inferior half of the cornea, Corneal sensation reduced. left eye vision was 6/24 with refraction of -1.75 D sphere, Tear Break Up Time was 11 sec.

Lagophthalmos was present. Circumcorneal congestion with corneal infiltration in inferior one third of the cornea with reduced corneal sensations, rest anterior segment and fundus examination was normal for both eyes. Sac was patent in both eyes. Extraocular movement were full and free in both the eyes. CBC, RFT, LFT were within normal limits. Random blood sugar level was 326 mg/ml, HbA1C was 10.2 %. Both eyes corneal scraping with Gram and KOH Mount were shown presence of Epithelial and pus cells with few yeast cells in left eye scraping.

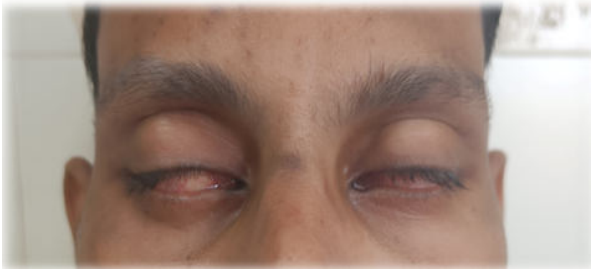


Figure 1 Lagophthalmos

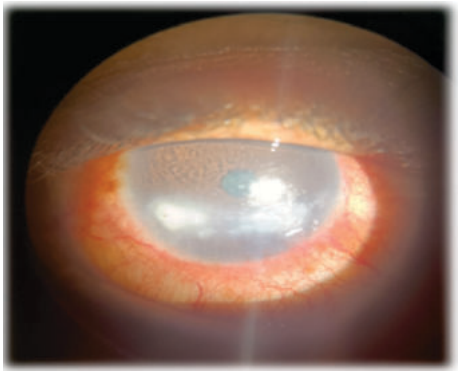


Figure 2 Right eye

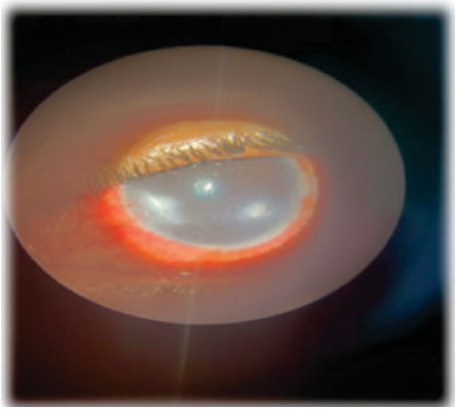


Figure3 Left eye

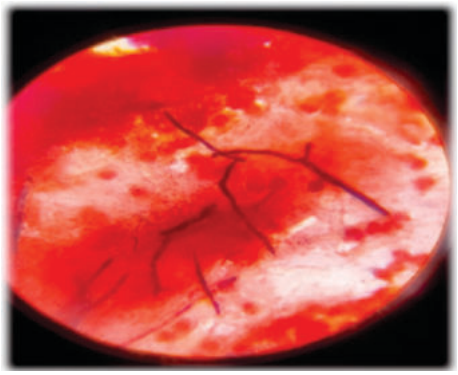
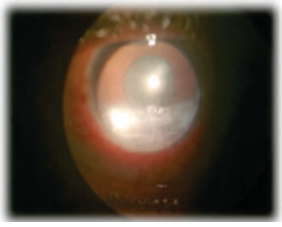


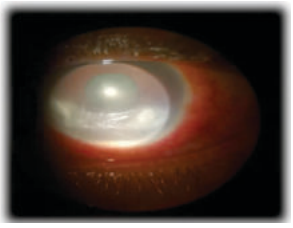
Figure 4 Left eye corneal scraping Gram staining showing few yeast cells

Patient was immediately started on treatment with Diabetic diet, Both eyes started on topical Fortified Amphotericin B E/D 1 hourly, Moxifloxacin E/D 6 times a day, Carboxymethylcellulose E/D 6 times a day, Atropine E/D twice a day, Lacryl PF eye ointment twice a day. Systemic treatment started with IV inj. Taxim (1gm) BD X 5 days, IV Inj. Metronidazole (100 ml) BD X 5 days, IV Inj. Gentamicin (80mg) BD X 5 days, Inj. insulin PI 10 units TDS, Inj. LI 10 units BD.

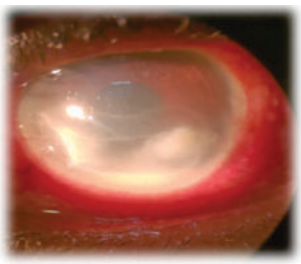
On Day 3 of treatment left eye showed corneal infiltrates with progressive thinning along with streak hypopyon, Fortified Amphotericin B eye drops were stopped suspecting drug toxicity. Treatment continued with chlorocol eye drops, Lubricants. Repeat corneal scraping done after 48 hours, Epithelial cells and pus cells noted. To prevent further thinning in Left eye multilayered AMG grafting with Bandage Contact Lens placement was planned.



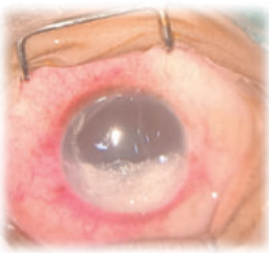
Left eye Day 1



Left eye Day 3



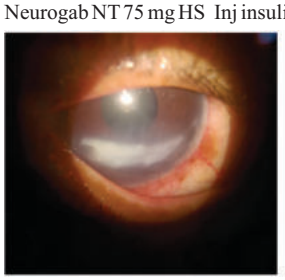
Left eye Day 5



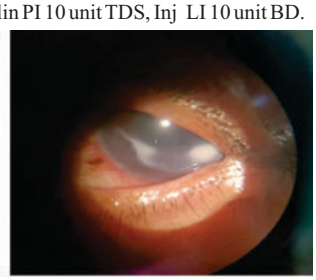
Left eye on Day7 with AMG grafting

So the final treatment for the patient was for both eyes, Wedge Lateral Paramedian tarsorrhaphy was done and topically continued on Lacryl PF eye ointment 4/d, Chloro applicaps 4/d, Carboxymethylcellulose eye drops 6/d, Chlorocol eye drop 6/d.

Systemic treatment continued as Multivitamins vit A, B complex, Vit c, Tab calcium Tab Doxycycline (100mg) BD x 10 days, Tab Neurogab NT 75 mg HS Inj insulin PI 10 unit TDS, Inj LI 10 unit BD.



Right Eye Day 15



Left eye Day 15

DISCUSSION

Cornea is one of the highly developed and essential structure of the eye. It forms major optical part of the eyeball whose function depends mainly on corneal transparency of the cornea. Tear film is one the factor which helps in maintaining in corneal transparency by lubricating and nourishing it. Tear film consists of three main layer namely, mucin the innermost, middle layer formed by aqueous layer while outermost, lipid layer. Tear film instability occurs when there is disturbance in function of these layers.

Along with tear film, blinking of the eyelids and eyelid closure are also important factors for corneal functioning. As a defence mechanism, blinking serves to keep a tear layer over the ocular surface, which is essential for epithelium health and optical performance. A clean and moist anterior corneal surface is kept by full blinks. Complete blinks sweep dirt into the inferior marginal tear strip, distributing a cleaner tear film as the upper lid rises[1]. Incomplete blink causes uneven distribution of tear film also leads to evaporation of tear film due to increased contact with external environment. Thereby affecting corneal function.

Exposure keratitis is keratopathy due to prolonged exposure of cornea to the environment[2]. Exposure keratopathy can result from inadequate or incomplete eyelid closure or less frequent blinking, which reduces the lubrication of the ocular surface. Facial nerve palsy, severe proptosis, lagophthalmos, critical unconscious patients in ICU with incomplete eye closure, herpetic keratitis are some of the causes for exposure keratopathy[1].

There have been reports of exposure keratitis rates in critically ill patients ranging from 10% to 55% to 60%[3]. Exposure keratitis is an unpleasant disorder that can result in corneal scarring and irreversible vision loss, even though it typically goes away once the patient is feeling better. In the worst case scenario, exposure keratitis triggers microbial keratitis, which can result in acute perforation, endophthalmitis, and permanent vision loss[4].

An impairment of the trigeminal corneal innervation, which results in a reduction or absence of corneal sensation, is the cause of the uncommon degenerative corneal illness known as neurotrophic keratitis. Numerous ocular and systemic disorders may cause lesions in the trigeminal ophthalmic branch, nucleus in the pons, Gasserian ganglion, nasociliary nerve, or long ciliary nerve of the fifth cranial nerve. Herpes simplex and herpes zoster keratoconjunctivitis are the most frequent causes of corneal anaesthesia, followed by chemical burns, physical traumas, and corneal surgery.1 The trigeminal nerve or ganglion may also be compressed by intracranial space-occupying tumours such neuromas, meningiomas, and aneurysms, which would compromise corneal sensitivity[5].

In severe cases, the cornea will look dry and may ulcerate, leading to perforation. Patients will experience pain or irritation, foreign body sensation, burning, blurring of vision, watering, redness and sensitivity to light.

Regular use of artificial tears and daily application of lubricating ointment are part of standard therapy. Every hour, artificial tears without preservatives can be used. As regular use of eye drops with preservatives might cause inflammation and may disturb the tear film, it is ideal for artificial tears and lubricating drops to be preservative-free[6]. Another option to investigate is punctal plugs[7].

Lagophthalmos is inability to close to both eyes properly. Its etiology is multifactorial . Facial nerve dysfunction is the most frequent cause of paralytic lagophthalmos. The majority of the eyelid muscles are controlled by the facial nerve (CN VII), hence problems with this nerve will result in weaker eyelid closure. Injury, cerebrovascular accidents, Bell's palsy, tumours, infections, immune-mediated causes, and congenital cranial disinnervation disorders are some of the things that might harm it[8]. Following blepharoplasty, post-operative lagophthalmos is frequently observed, with one study claiming post-op lagophthalmos rates as high as 47%[9]. According to one study, up to 23% of people have some degree of nocturnal lagophthalmos, or partial eyelid closure during sleeping, which is rather prevalent in the general population[10]. When the upper and lower eyelids are partially or totally joined, the eye can be closed partially or entirely.

Milder forms of lagophthalmos can be managed with lid taping.

Temporary tarsorrhaphies are applied to the cornea to aid in healing or to provide temporary protection from injury or disease. To permanently shield the cornea from a possibility of long-term harm, permanent tarsorraphies are utilised[11].

In a number of ocular surface conditions, the amniotic membrane, a biologic tissue, has been employed as a corneal and conjunctival graft. It has antiangiogenic, antiscarring, and anti-inflammatory characteristics in addition to being avascular. Instead of serving as a replacement, it serves as a platform for cells to migrate and renew, creating fresh, wholesome tissue. Additionally, the amniotic membrane can be applied as a bandage or biologic patch to treat acute inflammatory conditions[12].

CONCLUSION

Exposure keratitis is preventable cause of corneal blindness. Especially prevalent in patients of lagophthalmos, diabetes, critically ill/ patients admitted in ICU. Initial mainstay of treatment is medical. Removal of toxins such as Preservatives followed by aggressive lubrication essential Corneal sensations to be checked in any poorly healing cornea. Lid defects to be treated first. Early Tarsorrhaphy is necessary when medical therapy fails. Prevention of melting and/ scarring avoid the need for corneal transplant. Early adjuvant Amniotic Membrane Transplantation shortens the time for corneal healing.

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