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Acute band keratopathy formation after penetrating keratoplasty: A case report

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ABSTRACT

A 53-year-old male was referred from another eye hospital for right eye corneal perforation secondary to infective keratitis. A penetrating keratoplasty was performed for tectonic purposes. Forty eight hours after penetrating keratoplasty, he developed a band keratopathy located at the interpalpebral area of the corneal button. Despite stopping topical and systemic ciprofloxacin which could have contributed to this, the band keratopathy became more severe. Surgical debridement of the band keratopathy was done and there was no recurrence after that. This case of acute band keratopathy is an uncommon condition that develops within days to weeks of a particular insult to the eye. Various offending medications have been implicated, and treatment options include chelation therapy, surgical debridement and penetrating keratoplasty. Recurrence is common despite treatment and the cessation of possible offending medications. As such, it is prudent to be aware of and recognise the early symptoms and signs of this potentially sight-threatening condition.

1. Introduction

Calcific band keratopathy is a common feature of various systemic and eye disorders^[1]. The development of the calcific band usually takes weeks to months, resulting in chalky white deposits in the interpalpebral area of the superficial cornea layers^[2]. Acute band keratopathy has been reported to occur as early as within days to weeks of a particular insult to the eye such as intraocular surgery, intracameral injections, or instillation of certain topical eye preparations^[3–8]. We would like to report a case of acute band keratopathy developing within 48 h of tectonic penetrating keratoplasty.

2. Case report

A 53-year-old man was referred from a secondary tier healthcare facility with the diagnosis of right perforated corneal ulcer. On initial presentation, he had complained of right eye pain, redness and blurring of vision for five days' duration preceded by a foreign body contact onto that eye. He only sought medical treatment after experiencing increasing pain and tearing

of the right eye. He was previously under regular eye follow-up for past history of recurrent neurotrophic ulcer in the right eye secondary to an old chemical injury (traditional massage oil) to that eye. Premorbid best correction visual acuity in the right eye was only 2/60 due to corneal scarring.

On slit-lamp examination, his vision was light perception in the right eye while the left eye was 6/6. There was a central corneal perforation of about 1.0 mm in the middle of a large corneal ulcer of 7.5 mm × 7.0 mm in the right eye, with iris plugging and iridolenticulocorneal touch. There was a slow leak of aqueous noted. The anterior chamber was flat centrally and very shallow in the other quadrants. There was no view of the posterior segment, but a gentle B-scan ultrasonography showed a flat retina with clear vitreous. Examination of the left eye was unremarkable. Corneal scraping and conjunctival swab for Gram stain and cultures yielded no organisms.

A bandage contact lens was applied while awaiting corneal graft for tectonic penetrating keratoplasty. His medications included topical ciprofloxacin hourly, topical ceftazidime 5% hourly, oral acyclovir 800 mg twice daily, oral doxycycline 100 mg twice daily and intravenous ciprofloxacin 200 mg twice daily.

He underwent right eye tectonic penetrating keratoplasty with the use of a viscoelastic (DiscoVisc[®], Alcon Labs, USA) containing 1.65% sodium hyaluronate and 4% chondroitin sulphate. Intraoperatively, an intracapsular cataract extraction and anterior vitrectomy was done as the lens was strongly adhered to the site

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of corneal perforation. Surgery was otherwise uneventful and subconjunctival gentamicin/dexamethasone combination was given at the end of surgery. The 8.5 mm-diameter corneal button was clear centrally at the end of surgery, with only minimal oedema peripherally. Corneal graft was obtained from an Eye Bank in the USA from a 45-year-old Caucasian male who died due to multiple blunt force injuries from a motor vehicle accident. Donor had no chronic medical illness. The cornea evaluation showed moderate sloughing of epithelium, mild stromal oedema and a few stress lines in the endothelium. Endothelial cell count was 2967/mm² and blood screening for infectious diseases were negative. Post-operatively, all his medications were continued except for intravenous ciprofloxacin.

On day 1 post-operative review, right eye vision was hand movements with the aid of a +10 Dioptre lens. There was a full epithelial defect over the corneal button with diffuse haziness at its periphery. There were some blood clots within the anterior chamber, but the chamber was otherwise formed and Seidel's test was negative. There were mild suture reactions at 10 and 11 o'clock positions. Intraocular pressure was 11 mmHg. A bandage contact lens was applied. His medications post-operatively include topical ciprofloxacin hourly, topical ceftazidime hourly, oral acyclovir 800 mg twice daily, oral doxycycline 100 mg twice daily, and oral ascorbate 1 g daily. No topical steroid eye drop was started yet in view of the large epithelial defect.

On day 2 post-operatively, we noted the development of a superficial white band located at the interpalpebral area of the corneal button. The grainy opaque lesion became more apparent despite the cessation of topical and intravenous ciprofloxacin which were replaced with topical moxifloxacin. His vision remained as hand movements and anterior chamber depth improved gradually with slow resolution of blood clots within the chamber. Day 3 review revealed a more pronounced horizontal epithelial band which started to encroach onto the recipient cornea at 4 and 8 o'clock positions (Figure 1).

A clinical diagnosis of 'acute band keratopathy' was made. Blood investigations revealed normal renal and liver function tests, and also serum calcium and phosphate that were within normal limits.

Attempts to remove the sub-epithelial band keratopathy via superficial debridement of the epithelium at the slit lamp proved unsuccessful. We proceeded to perform right eye surgical

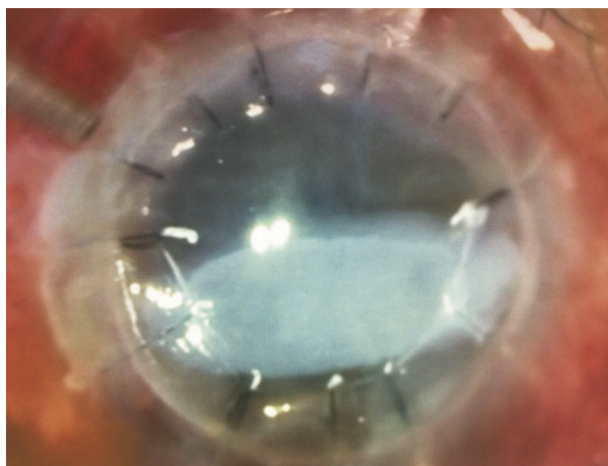


Figure 1. On-table appearance of the linear band keratopathy at day 2 post-penetrating keratoplasty (just prior to surgical debridement).

debridement of the band keratopathy under local anaesthesia in the operating theatre. Intra-operatively, the 'band keratopathy' was noted to be a flaky whitish lesion confined to the interpalpebral fissure and moderately attached to the corneal epithelium. It was removed together with the corneal epithelium with moderate difficulty as the epithelium was quite firmly attached to the underlying Bowman's layer. The corneal epithelium overlying the band keratopathy was structurally rigid and had a grainy texture. There was no stromal infiltration. A bandage contact lens was applied immediately post-operatively.

Post-operatively, corneal epithelial wound healing progressed well without any further development of band keratopathy (Figure 2). The cornea remained clear throughout the remainder of his hospitalisation stay of 9 days. Topical prednisolone acetate 1% thrice daily was started after 4 days of the debridement surgery. The patient was discharged home on day 9 post-debridement with topical eye drops of moxifloxacin 2-hourly, homatropine 2% thrice daily, prednisolone acetate 1% 6-hourly, and oral medications of doxycycline 100 mg twice daily, acyclovir 800 mg daily and ascorbate 1000 mg daily. At weekly reviews until about one month post-operatively, there was no recurrence of the band keratopathy.

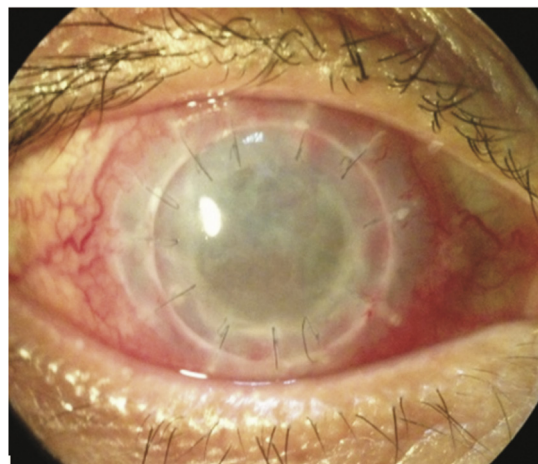


Figure 2. Appearance of the cornea 1 week after surgical debridement of band keratopathy.

3. Discussion

Calcific band keratopathy is an established complication of a large number of inflammatory and degenerative processes in the eye^[1]. It can also be due to systemic disorders which cause elevated calcium levels in the blood, providing a conducive environment for calcium salts to precipitate^[9]. Precipitation can be easily triggered by minor local events such as a change in pH, sudden evaporation or an increase in the local concentration of calcium and phosphate ions. Inflammation, infection, chronic dry eyes, and the use of phosphate-buffered eye solutions can all significantly alter the calcium homeostasis of the eye leading to band keratopathy. In these conditions the corneal opacification is typically present in the interpalpebral area and develops very slowly.

Band keratopathies usually develop gradually over a period of weeks to months. However, reports have emerged highlighting the development of band keratopathy within hours and days of an insult to the eye^[4-8,10]. In our patient, band keratopathy

Table 1

Identified agent and treatment of band keratopathy.

Authors	Year	Identified agent	Treatment
Binder <i>et al.</i> ^[5]	1987	Sodium hyaluronate-chondroitin sulphate (Viscoat)	Superficial debridement & bandage contact lens
Huige <i>et al.</i> ^[10]	1991	Timolol maleate & topical steroids (dexamethasone, fluorometholone or prednisolone)	Superficial debridement (2/8 patients)
Bernaer <i>et al.</i> ^[8]	2006	Sodium hyaluronate artificial tears (Hilo-Comod)	Excimer laser, penetrating keratoplasty
Spitzer <i>et al.</i> ^[7]	2007	Recombinant tissue plasminogen activator	Superficial debridement and sodium EDTA
Moisseiev <i>et al.</i> ^[6]	2013	Recombinant tissue plasminogen activator	Superficial debridement and sodium EDTA

developed within 48 hours after undergoing tectonic penetrating keratoplasty. While our initial impression was that of possible ciprofloxacin precipitation, it was deemed unlikely as the patient was already on systemic and topical ciprofloxacin for almost two weeks prior to surgery without the development of white precipitates.

More rapid development of band keratopathy has been described in silicone oil keratopathy^[3], the use of intracameral viscoelastic substances^[4,5], tissue plasminogen activators (tPA)^[6,7], and instillation of sodium hyaluronate artificial tears^[8], or a combination of topical steroids and a beta-blocking medication^[10] (Table 1).

The only similarity our patient had with any of these offending agents was the use of an intracameral viscoelastic substance. In the prior reports^[4,5], the viscoelastic used was a phosphate-buffered Viscoat (Cilco Inc, Huntington), which was left intracamerally. In our case, DiscoVisc[®] (Alcon Labs, USA) was used to cushion the corneal button during suturing and was removed before the end of surgery. While phosphate buffers are still necessary in today's viscoelastics, they are at a lower concentration than before. Therefore, we cannot conclusively implicate DiscoVisc[®] as being the cause of the acute band keratopathy formation.

While there are many case reports documenting the occurrence of calcific band keratopathy after corneal grafting, the aetiologies were attributed to the use of Viscoat, tPA, or a topical steroid and beta-blocking eye drops. To our knowledge, this was the first case to date that had none of the above medications used.

Another interesting observation in our case was that there was no recurrence of the band keratopathy after surgical debridement was done. This is in contrast to the other cases reported elsewhere which had repeated occurrence of calcific band keratopathy developing despite chelation with ethylenediaminetetraacetic acid (EDTA)^[11] or after repeat corneal grafting^[8].

Conflict of interest statement

The authors report no conflict of interest.

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