

Case report

Nocardia Keratitis following penetrating corneal injury treated with topical ampicillin

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Abstract

Background: Nocardia keratitis following surgical management of penetrating corneal injury is very rare. **Case:** A 38-year-old male presented with a deep stromal corneal infiltration with hypopyon of 10 days duration in his left eye. He had undergone a corneal tear repair on that eye 15 days prior to the onset of keratitis. The infiltrations were along the sutured corneal tear tracts. Before presenting to us, he was treated empirically with hourly topical antifungal and antibiotic for 10 days with poor clinical response. Microbiological investigations revealed the presence of Nocardia in both smear and culture. The infiltrations responded well to fortified 5% ampicillin eye drops. **Conclusion:** Although Nocardia keratitis is very rare following penetrating corneal injuries and clinically can mimic mycotic keratitis, it should also be suspected in cases not responding to standard treatment. Management of Nocardia keratitis requires a good clinical judgement with appropriate microbiological support.

Keywords: Nocardia Keratitis, ampicillin, corneal tear

Introduction

Nocardia are aerobic Gram-positive filamentous bacteria that are partially acid-fast and a rare cause of infectious keratitis (Sridhar et al, 2001). The incidence of Nocardia keratitis varies with different geographical locations. One study in Nepal reported that Nocardia constituted 0.3% of all bacterial isolates from cases of keratitis (Upadhyay et al, 1991) whereas in South India, it ranges from 1.7 % to 4.2 % (Sridhar et al, 1998; Srinivasan et al, 1987). The nature of traumatizing agents reported includes vegetative material, dirt, stone, gravel, and finger nail injury. Nocardia

keratitis may be misdiagnosed clinically because of unfamiliarity or because of its clinical resemblance to mycotic or mycobacterial keratitis (Huang, 1996). Nocardia stromal keratitis following deeper corneal injury post repair is very rarely reported. We herewith report an atypical presentation of Nocardia keratitis following corneal tear repair.

Case

A 38-year-old male presented with history of pain, redness and diminution of vision in his left eye of 10 days duration. He had sustained trauma to his left eye 25 days prior with broken spectacle glass pieces while at work. Following his injury, he went to an ophthalmologist and had the corneal foreign bodies removed and

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the corneal tear repaired. He was discharged after one day with prednisolone acetate 1% and ofloxacin 0.3% combination eye drops six times a day. He was also prescribed oral ciprofloxacin tablets 500 mg twice a day for 5 days and oral analgesic tablets.

However, 15 days following the surgery, pain and redness in left eye increased with a marked reduction in vision. The patient reported back to his physician, who noticed the presence of sutural infiltrations and removed 3 sutures. Topical steroid was stopped and natamycin 5% and gatifloxacin 0.3% eye drops were started on an hourly basis. Since the patient did not respond adequately, he was referred to us. At presentation, his best corrected visual acuity in the right eye was 6/6 and 1/60 in the left eye. Clinical examination of the right eye did not reveal any abnormality. In left eye, he had blepharospasm and mild lid edema. There was both ciliary and diffuse conjunctival congestion. The corneal examination revealed that there had been two different tears. One tear was lamellar, 6 mm long and superotemporal in location with 3 sutures. The second tear was perforating and was 5 mm long, inferotemporal and full-thickness with two interrupted sutures (Fig: 1). There were two deep corneal stromal infiltrations along the corneal tear tracts. One of the infiltrations was in a superotemporal location and measured 2.5 mm x 1.5 mm while the other was in the inferonasal area and measured 3 mm x 3.5 mm. Both the infiltrations had irregular margins with few satellite lesions. The surrounding cornea was edematous. Anterior chamber had a mobile hypopyon of 1.5 mm. Pupil was pharmacologically dilated and lens appeared to be clear. Fundal glow was present with a hazy view.



Figure 1: Left eye showing two deep stromal corneal infiltrations along the corneal tear tracts with hypopyon in anterior chamber (At presentation; after corneal scrapping)

Using standard techniques, corneal scraping was done for microbiological evaluation. Gram's stain showed Gram positive thin beaded branching filaments suggestive of *Nocardia* (Figure 2). He was treated with topical amikacin 2% eye drops hourly and sulphacetamide 20% eye drops every 2 hourly along with atropine 1% drop once a day. Culture report confirmed the presence of small, dry, white colonies suggestive of *Nocardia* species in blood agar after 48 hours (Figure 3). Antibiotic susceptibility was tested on blood agar using the Kirby-Bauer method and the organism was found to be sensitive to amikacin, gentamycin, tobramycin, cefotaxime and moxifloxacin.



Figure 2: Gram stain of corneal scraping showing Gram positive thin beaded filaments



Figure 3: Culture of corneal scraping on blood agar showing small, dry, white colonies of *Nocardia*

The patient came for follow up after 1 week with no clinical improvement. Based on the sensitivity report, topical moxifloxacin was substituted for sulphacetamide. After two weeks of topical amikacin therapy, even though there was no disease progression, there was no improvement either. A decision was made to stop amikacin and start topical fortified 5% ampicillin eye drops empirically. After 1 week of topical ampicillin therapy, both the size of the infiltrate and hypopyon decreased. Corneal vascularisation started from the periphery towards the infiltrations (figure 4). Topical moxifloxacin was then stopped and ampicillin was continued. After 2 weeks of topical ampicillin drops, the hypopyon resolved (Figure 5). Topical ampicillin was continued for another 4 weeks four times a day. At two months follow ups, the infiltration completely resolved with scarring. There was deep stromal vascularisation with lipid deposition. Best corrected vision in left eye improved to 6/24 at his last follow up.



Figure 4: Resolving infiltrations and hypopyon with corneal vascularisation (after 3 weeks)



Figure 5: Resolving infiltration with corneal vascularisation. Hypopyon disappeared. (After 4 weeks)



Figure 6: Resolved keratitis with scarring and vascularisation (After 2 months)

Discussion

Nocardia are gram-positive organisms, which in smears appear as extensively branching and thin beaded filaments. They are a relative

rare cause of infectious keratitis. The usual predisposing factors for *Nocardia* keratitis are trauma, surgery, corticosteroid use, and contact lens wear. In a series of 16 cases from India, trauma was the inciting factor in 25% of cases (Sridhar et al, 1998). The nature of traumatizing agents reported in the same case series included mostly vegetative material related to agriculture works. In our case report, trauma with glass particles was the predisposing factor, which is an unusual finding.

Nocardia typically causes a superficial spreading wreath shaped keratitis. Patchy infiltrates, which are predominantly anterior stromal are pathognomonic (Sridhar et al, 1998) whereas cases resembling fungal ulcers are grouped as atypical presentations (Srinivasan et al, 1987). Cases without characteristic diagnostic features of any keratitis are grouped separately as nonspecific (Lalitha P et al, 2007). Our case was unique in that the corneal tear and suture tracks provided a nidus for the *Nocardia* infection. Unlike the common superficial presentation, there was mainly a deep stromal involvement with minimal epithelial involvement. The deep corneal lesions in our case reflect the prolonged duration of infection. It may also be a result of the modified growth pattern of *Nocardia* organisms due to use of corticosteroids. Our patient was on topical steroids post corneal tear repair and probably due to induced local immunosuppression, conditions became favourable for developing *Nocardia* keratitis.

The nocardial corneal infiltrates are usually situated in the midperiphery of the cornea adjacent to the sites of minor corneal trauma or abrasion. *Nocardia* keratitis is rarely reported in literatures following deeper corneal injuries. To our knowledge, only one prior similar report exists (Frilling et al, 1995). Our case is the second of such kind. In both the cases the clinical presentation was intrastromal corneal infiltrates.

Nocardia keratitis has revealed varying drug sensitivities in concordance with previous reported studies. *Nocardia* keratitis usually considered refractory to conventional topical antibiotics, resulting in a protracted clinical course and progressive extension of the disease (Huang, 1996). Trimethoprim-sulfamethoxazole, sulphacetamide and amikacin are reported to be first line drugs in the management of *Nocardia* keratitis (Sridhar et al, 2001). Denk et al (1996) also recommended amikacin in the concentration of 2–2.5% as the treatment of choice in the monotherapy of *Nocardia* keratitis. Bajracharya et al (2012) reported a *Nocardia* keratitis case that even improved with 1.4% amikacin, which is lower than the usual concentration used in other case reports. In our case, after initial Gram stain report, we started with topical sulphacetamide 20% and amikacin 2%. However, the response to the standard treatment for *Nocardia* keratitis was not satisfactory and the patient finally responded well with topical ampicillin drops.

Systemic ampicillin is commonly used as a combination therapy for systemic Nocardiosis. However, topical ampicillin is rarely used for treating *Nocardia* keratitis. Our decision to start topical ampicillin was based on our previous experiences with topical ampicillin for resistant *Nocardia* keratitis. Srinivasan et al (1987) have described four patients of *Nocardia* keratitis that healed successfully with topical 10% sodium sulphacetamide and 10% ampicillin trihydrate.

Conclusion: *Nocardia* keratitis can have atypical presentation especially following deeper corneal injury. A good microbiological support is necessary for the diagnosis and successful management. It may not respond to the conventional antibiotics and antifungals used for the treatment of corneal ulcer. Topical ampicillin can be considered in those cases not responding to the standard therapy.

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