Extraocular spread following evisceration for rapidly progressive intraocular tuberculosis

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We describe a case of right eye tubercular (TB) panuveitis with corneal involvement where repeated intraocular sampling was unsuccessful. Finally, evisceration and histopathology of ocular tissue confirmed the diagnosis of ocular tuberculosis. A chest X-ray showed signs of pulmonary TB. The patient was lost to follow-up but reported 2 months later with the right eyelid margin nodules with surface ulceration. Both eyelid and pulmonary lesions were resolved with anti-TB therapy.

Key words: Evisceration, extraocular spread, inflammation, ocular tuberculosis, TB panuveitis

Ocular tuberculosis (TB) has myriad clinical manifestations involving all segments of the eye.[1,2] Extraocular TB involving the orbits or ocular adnexa is relatively uncommon and generally occurs in isolation.[3] Herein, we present a case of rapidly progressive TB panuveitis that required evisceration and subsequently presented as eyelid ulceration.

Case Report

A 66-year-old woman presented with watering, redness and painful, progressive, decreased vision for 2 months in her left eye (LE). She was diabetic and hypertensive for 6 years. She had received bilateral sequential cataract surgery in the past with good postoperative outcomes. She was diagnosed elsewhere as pseudophakic bullous keratopathy LE and had used loteprednol etabonate 0.5% eye drops. Her best-corrected visual acuity (BCVA) at presentation was 20/20, N06 in the right eye (RE), and hand movements close to face and projection of rays (PR) accurate in LE. Intraocular pressure was 12 mmHg in RE and digitally normal in LE. RE was unremarkable. LE revealed congested conjunctiva, large epithelial defect measuring 9 × 10 mm, dense stromal edema and inferior large, and irregular pigmented keratic precipitates (KPs) [Fig. 1a]. Anterior chamber showed 3+ flare and cells with a yellow fungal glow. B-scan ultrasonography revealed intraocular lens reverberations, medium echo-spike membranous and dot echoes in the vitreous cavity, inferior shallow choroidal detachment (CD), and choroid thickening (CT) of 1.70 mm [Fig. 1b]. Based on the presence of corneal stromal edema, pigmented KPs, and acute onset, we made a provisional diagnosis of endothelitis with panuveitis of presumed viral etiology.

Anterior chamber (AC) paracentesis and vitreous biopsy were performed. Meanwhile, she was started on oral acyclovir 800 mg 5/day, hourly topical prednisolone acetate 1% eye drops, and cycloplics. The smear/culture reports were negative but aqueous analysis by conventional polymerase chain reaction (PCR) was positive for both HSV1 (gB glycoprotein D gene) and MTB DNA (MPB64 gene). Tuberculin skin test (TST) revealed 16 mm induration. The chest X-ray showed a blunted right costophrenic angle with calcified lymph nodes in the superior mediastinum. Tests for HIV, syphilis, and sarcoidosis were negative. Over the next 48 h, there was rapid worsening, with large multiple corneal infiltrates now visible [Fig. 1c and d]. Corneal scrapings did not reveal any organism on smear/culture. Her BCVA dropped to the perception of light with inaccurate PR, inferior paracentral corneal melt, and iris prolapse was noted [Fig. 1e]. B-scan ultrasonography revealed multiple CDs with CT 2.20 mm. No sub-Tenon’s fluid (STF) was found. After counseling and discussing treatment options, she underwent evisceration and contents sent for microbiology and histopathology evaluation. Postoperative medications were advised and the patient was referred to a pulmonologist for systemic evaluation and treatment. The histopathology report of the eviscerated contents showed multinucleated giant cell granulomas in the retinal fragments [Fig. 2c and d]. Ziehl-Neelsen stain with 20% sulphuric acid revealed acid-fast bacilli in the corneal stroma [Fig. 2a and b]. The patient was lost to follow-up for 2 months and presented with pain, swelling, and discharge of left eyelids for 1 week. Examination revealed lid margin thickening, irregularity, and nodules with surface ulceration [Fig. 1f]. The patient was started on anti-tubercular therapy (ATT) with symptomatic treatment for eyelid lesions. The eyelid lesions resolved a month later and no recurrence was noted after completion of ATT.

Discussion

This case presents with two rarities for intraocular TB that have not been reported earlier – rapidity of corneal progression and extraocular spread following evisceration for rapidly progressive intraocular tuberculosis. Indian J Ophthalmol 2020;68:2583-5.
extraocular spread of infection despite evisceration. Intraocular tuberculosis presenting as panuveitis and endophthalmitis or panophthalmitis[2,4] have been reported but progression to involve cornea or eyelids has not been reported. Similarly, keratitis typically an immune-mediated stromal interstitial keratitis or peripheral ulcerative keratitis, and even progression to corneal melting has been reported,[5] but our case stands out by the rapidity of progression. Rapid progression of corneal disease probably resulted from mycobacterial infiltration of the corneal lamellae which was predominantly noted during a histopathological evaluation.

Our initial clinical impression at presentation was of viral etiology given corneal involvement and presence of pigmented KPs. However, other signs of a viral infection such as raised intraocular pressure or iris atrophy were not seen. The clinical dilemma was further heightened by mixed results from PCR testing of ocular fluids, which could well be due to variability and inherent fallacies in PCR testing.[6] In retrospect, we realized that pigmented mutton fat KPs are also found in TB and other forms of granulomatous inflammation.[7] Another differential diagnosis that could have been considered is infectious endophthalmitis, but history did not reveal any endogenous focus of infection.

Progression of intraocular TB to involve sclera has been described.[7] Eyelid involvement after contiguous extension from cutaneous lesions and orbit have also been noted.[8,9] However, extraocular spread following evisceration for intraocular TB has never been reported. Postoperative infection after evisceration has been reported to be extremely rare.[10] We did not find any evidence of STF before the evisceration. While it is likely that intraocular infection is disseminated to the extraocular tissues, it also raises the possibility of unrelated hematogenous dissemination from the lungs. The occurrence of nodular ulcerative lesions on lid margins not noted in previous visits also raises the suspicion of progressive extraocular/orbital spread. Unfortunately, the patient was not tested for TB bacilli in the sputum despite the presence of radiological changes in the lungs. A limitation
here is a lack of lid biopsy. However, the resolution of both eyelid lesions and pulmonary signs after initiation of ATT confirms their TB etiology.

**Conclusion**

In conclusion, this case highlights a rare but severe form of intraocular tuberculosis with probable extraocular extension. It stresses the need to consider a differential of TB etiology in panuveitis, even with rapidly progressing keratitis, and the importance of postoperative ATT in cases requiring evisceration.

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**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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