An Unusual Case of Metastatic Tubercular Panophthalmitis in a 14-Year-Old Boy

To the Editors:

Ocular manifestations in tuberculosis occur either by direct invasion or as a result of hypersensitivity reaction caused by *Mycobacterium tuberculosis*. Primary infection (usually involving cornea/conjunctiva) occurs in the absence of systemic lesion, whereas secondary infection (usually intraocular) results either from hematogenous spread or contagious spread from an adjacent structure. The various ophthalmic manifestations of tuberculosis include choroiditis (most common), anterior vasculitis, vascular lesions, dense vitritis, and papillitis. Panophthalmitis in tuberculosis is a rarely described entity.

A 14-year-old boy presented to the pediatric ophthalmic services of our institute with complaints of severe pain, redness, and decreased vision in the right eye for 3 days. He had been diagnosed as having panuveitis 2 months earlier and was receiving continuous treatment with topical steroids, antibiotics, cycloplegics, and antiglaucoma drugs, with no significant improvement.

On systemic examination, he was thinly built and chest examination revealed bilateral crepts. On ophthalmic examination, there was no light perception in the right eye and the best-corrected visual acuity in the left eye was 6/6. There was circumcorneal congestion, corneal haze, hyphema, and exudates in the anterior chamber (Fig. A). The pupil was semidilated and not reacting to light. There was altered yellow reflex through the pupil and details of the fundus were not clear. Ocular movements were restricted in all gazes. The left eye did not show any other abnormality except absent consensual pupillary reaction. Ultrasonography A and B scan (Fig. B) was done. The right eye revealed multiple low to moderate spikes in vitreous suggestive of exudates (Fig. B). The intraocular pressure was 30 mm Hg in the right eye and 14 mm Hg in the left eye. Computed tomographic scan revealed thickness of uveoscleral coats in the right eye with no apparent calcification.

His blood profile was normal except for leukocytosis and elevated erythrocyte sedimentation rate. Chest x-ray was suggestive of resolving pulmonary Koch's infection. A provisional diagnosis of panophthalmitis was established and topical treatment in the form of steroid antibiotics, antiglaucoma drugs, and systemic antitubercular treatment was continued.

In lieu of absent vision, severe pain, and investigation suggestive of panophthalmitis, enucleation of the right eye was planned. On gross examination of the enucleated eye, the uveoscleral coat was thickened and the vitreous cavity was full of necrotic tissue. Histopathology confirmed the diagnosis of tubercular panophthalmitis (Figs. C and D).

There are two possible contributory factors for ocular involvement in tuberculosis. First, a septic embolus from the pulmonary infection occludes the central retinal artery and embolic fragments are then disseminated peripherally. Second, in patients with bacteremia, the blood-borne organisms permeate the blood–ocular barrier either by direct invasion or by changes in vascular endothelium caused by substrates released during the infection. The choroid is highly vascular and it is thus the most commonly involved structure; in certain circumstances this may progress to panophthalmitis leading to loss of the eye.
Few cases of tubercular panophthalmitis have been reported in the Indian literature.\(^4\) Because the ocular presentation in Kocbs’ infection may be non-specific, the importance of early suspicion of panophthalmitis in children with systemic tuberculosis is to be emphasized so that ocular manifestations, if any, are diagnosed and treated in a timely fashion to preserve useful vision and prevent further morbidity.

REFERENCES