Vitrectomy for tuberculosis

Case Report

Successful Visual Outcome Following Vitrectomy for Complication of Ocular Tuberculosis

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Abstract

A 24-year-old Chinese gentleman presented with two weeks history of sudden floaters in his right temporal visual field associated with blurring of vision. This ex-smoker also reported chronic, bloody cough for two years and recent pleuritic chest pain. Examination revealed a thin patient with right eye visual acuity of 6/18 associated with optic nerve dysfunction, optic disc swelling and macula star, retinal vasculitis and retinitis. Despite anti-tuberculosis medication and corticosteroids, he developed neovascularisation. Subsequent vitreous haemorrhage necessitated trans pars plana vitrectomy, membrane peeling, endolaser and silicone oil injection. Final visual acuity was 6/9 with quiescent retinopathy.

Keywords: Ocular tuberculosis, vitreous haemorrhage, epiretinal membrane, vitrectomy, silicone oil

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Introduction

Tuberculosis is one of the most rapidly spreading communicable diseases worldwide made worse by migration from third world countries and the emergence of diseases requiring immunomodulation or immunosuppression (1,2). While systemic tuberculosis is readily detectable with standard tests, ocular tuberculosis needs a high index of clinical suspicion to diagnose, as it may be the only organ involved. The eye has limited tissue available for biopsy, hence in cases of ocular tuberculosis, collaborative high clinical suspicion and standard tests are crucial (3).

Ocular tuberculosis often results in good visual outcome but may have high ocular morbidity particularly in highly prevalent areas (4) and immunosuppressed patients (5). We report a healthy young gentleman with presumed ocular tuberculosis who failed to respond to initial treatment with antituberculous drugs. He subsequently required surgical intervention and had a good visual outcome.

Case Report

A 24-year-old Chinese gentleman complained of sudden onset of floaters affecting his right outer visual field for two weeks. Three days later, his vision dropped in that eye. There was no redness, photopsia or pain. There was no recent travel abroad and he denied any high-risk behaviour. He reported a history of cough with bloody sputum for 2 years with recent intermittent pleuritic chest pain. There were no other constitutional symptoms and no history of contact with tuberculosis (TB) patient. He was a former smoker of 20 cigarettes a day but ceased a month ago.

Examination revealed a thin young man with stable vital signs. Systemic examination was unremarkable with no lymphadenopathy. His vision at presentation was 6/18 ph 6/12 N 18 OD and 6/9 ph 6/6 N 5 OS. There was right relative afferent pupillary defect with good red reflex bilaterally. The anterior segment was quiet. The intraocular pressure was 18 mmHg OU.

Examination of the posterior segment showed old pigmented anterior vitreous cells of 2 + in the right eye. Funduscopy of the right eye revealed an area of retinitis and vasculitis with flame shaped and preretinal haemorhages at the superonasal arcade. The optic disc was swollen and hyperaemic with a macular star (Fig. 1). Sheathing of the vessels at the periphery was also noted.

The left vitreous was quiet. The left optic disc was also hyperaemic but not swollen. There was a cluster of dot haemorrhages at the mid-periphery temporal to the arcades. The macula was normal.

Fundus fluorescein angiogram of the right eye revealed arterial occlusion distal to the vasculitis area with staining of diseased vessels.

Blood investigations revealed raised total white cell count with neutrophilia. Other blood tests like renal profile, random blood glucose and liver functions, VDRL, retroviral screening and hepatitis screening were all negative. The chest X-Ray and MRI of the thorax and brain was also normal. Mantoux test read 15 mm of induration.

He was started on a trial of oral rifampicin 300 mg daily for 2 weeks and oral prednisolone 60 mg daily. His vision improved to 6/18 ph 6/9 with improvement of the other ocular lesions. The respiratory team commenced multi-drug anti-TB regime of isoniazid, rifampicin and pyrizinamide.

After swapping the treatment regime for some traditional Chinese medication, he presented 3 weeks later with sudden drop in vision of the right eye for 3 days. With a vision of counting finger, the right eye had suffered a dense vitreous haemorrhage (VH). Recommencement of the anti TB treatment allowed the VH to resolve slowly.

Unfortunately, a month later, he developed a dense subhyaloid haemorrhage at the macula. The decision for surgical intervention was made and he underwent 20G trans pars plana vitrectomy, membrane peeling, endolaser and silicone oil injection. Post-operatively, he continued to improve leaving a macular pucker with visual acuity of 6/36.

A second surgery to remove the silicone oil and epiretinal membrane peeling was done 3 months later.

A subsequent posterior subcapsular cataract developed for which cataract surgery was performed. His final visual acuity after 18 months of therapy was 6/9 with stable and quiescent retinopathy (Fig. 2).

Discussion

Tuberculosis is an emerging global endemic caused by a chronic infection by *Mycobacterioun tuberculosis* affecting nearly one third of the world's population and reaching an annual incidence of 8.7 million patients (6). With a mortality rate of 0.4 per 100,000 population, TB has been regarded as the single most common cause of mortality from any infectious disease contributing an estimated 1.87 million deaths per year worldwide (6).

Although rare, the incidence of ocular tuberculosis varies widely across time, patient populations and geography. The incidence among TB patients was

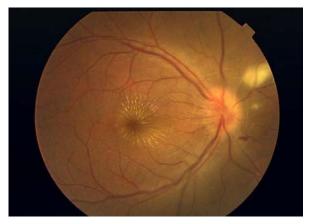


Figure 1: Fundus photograph of the right eye at presentation showing retinitis at the superonasal area and vasculitis. The optic disc is swollen and hyperaemic with a macular star.

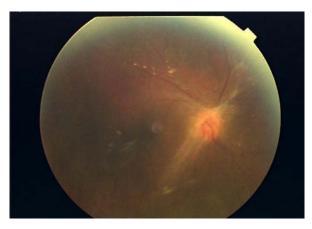


Figure 2: Fundus photograph of the right eye showing gliosis and macular pucker following vitrectomy to remove vitreous haemorrhage.

reported to be between 1.46% and 18% and among uveitic patients, between 0.6% and 10.5% (6).

The bacillus is spread via droplet inhalation into the lungs. Upon reaching the lung alveoli, the bacillus is either destroyed by the resident alveolar macrophage (AM) or survives causing necrosis of the phagocyte (7). The subsequent dissemination of the bacterialaden monocytes/histiocytes to multiple organs via the lymphatics and blood circulation allows seeding of tuberculous foci which may remain dormant in a resistant host. Ocular TB results either from direct pulmonary haematogenous spread or from reactivation of dormant ocular lesions. The latter is thought to be the commoner cause for active ocular lesions and clinical manifestations (7).

This patient represents the commonly seen clinical features of posterior uveitis in ocular TB characterized by retinitis and vasculitis. These lesions are thought to originate either from a direct extension of choroidal granuloma or haematogenous spread (8). The optic disc swelling and macular star are classical features of neuroretinitis which is frequently associated with tuberculous retinal vasculitis and may precede the onset of exudative retinal detachment seen in tubercular multifocal choroiditis (7).

Although a definitive diagnosis can be made from culturing or amplifying DNA material of *M. tuberculosis* from involved tissue (8), there exists impracticality of applying the standard diagnostic techniques to the eye. Therefore, greater reliance has to be placed on detecting evidence of systemic TB from the lungs or any other organ where samples are more readily obtained. Among others, a positive tuberculin test (Mantoux test), positive x-ray findings, positive acid fast bacilli from smears, and positive sputum culture are all collectively used to support the diagnosis of systemic TB (7). At best, a diagnosis of presumed ocular TB should be made in the absence of any positive results.

The other criteria used to diagnose ocular TB in this patient, apart from the corroborative suggestion of systemic tuberculous infection evidenced by a chronic cough with bloody sputum, pleuritic chest pain, malaise and lethargy and a positive Mantoux test of 15 mm, was the fact that he responded poorly to immunosuppression and showed excellent response to a trial of rifampicin therapy. This is especially useful when all other laboratory results was negative, including a normal chest X-Ray and negative polymerase chain reaction(PCR) test, the results of which is reportedly positive in one third of presumed tuberculous retinal vasculitis and panuveitis patients (9). This approach has been described by Abrams et al in arbitrary cases of ocular TB (3).

Systemic antiTB treatment is given in presumed ocular TB as pulmonary and other foci of infection may coexist even in the absence any clinical findings and that the majority of these patients need immunosuppression therapy as well. It is successful in the majority of cases with subsequent resolution of inflammation and improvement in visual acuity (7).

Unlike other causes of retinal vasculitis, tubercular vasculitis causes severe vascular cuffing and infiltrates, extensive peripheral capillary closure and retinal neovascularization. At this stage panretinal photocoagulation (PRP) may stabilize the ischemic drive, the failure of which may result in subsequent vitrous haemorrhage and tractional retinal detachment (TRD) (7, 10). The indication for surgical intervention in this patient was inevitable as he continued to bleed despite initial medical treatment and given that TRD had set in. This case has demonstrated the crucial role of surgical intervention in salvaging vision.

To our knowledge, this is the first report of vitrectomy for ocular TB which resulted in good visual outcome. In a case series involving 12 eyes of 11 patients with subretinal granuloma, Gupta V et al reported worse visual outcome in two eyes which had to be subjected to vitrectomy after failing medical therapy (11). Another report reserved vitrectomy for severe posterior uveitis unresponsive to medical therapy which also resulted in poor final visual outcome (12). We decided to intervene surgically relatively early and this has resulted in a better visual outcome, although medical treatment is still considered the mainstay treatment in this infectious uveitis (13).

Conclusion

Extrapulmonary TB particularly ocular TB is reemerging as a major challenge for physicians worldwide. Ocular TB results from haematogenous seeding from the primary complex or secondary reactivation of a dormant lesion. The diagnosis is made on the basis of clinical features, demonstration of *M. tuberculosis* from intraocular fluids, and corroborative evidence of the disease in other organs. While the mainstay of therapy for ocular TB is anti-TB drugs, sight threatening complications such as VH and macular pucker can be safely and successfully managed by vitrectomy surgery.

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