University Journal of Surgery and Surgical Specialities

ISSN 2455-2860 2021, Vol. 7(4)

Case Report – Ocular Tuberculosis associated with miliary and leptomeningeal tuberculosis

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Abstract

A 17 year old immunocompetent young man developed pain, photophobia and defective vision in the Right eye. MRI chest showed features of miliary tuberculosis while MRI brain showed multiple ring enhancing lesions suggestive of leptomeningeal tuberculosis for which he was started on Anti-tubercular therapy. After an initial misdiagnosis of Horner's syndrome, patient presented 4 months later with visual acuity of 1/60 in the right eye and 6/6 in the left. Right eye showed keratic precipitates, shallow anterior chamber, iris neovascularisation, posterior synechiae and complicated cataract with intraocular pressure of 4 mm Hg. He was diagnosed withRight eye granulomatous pan-uveitis of probable tubercular origin and advised to continue anti-tubercular therapy and started on oral and topical steroids. Despite all efforts, after 1 year, vision in the right eye had worsened to hand motions, intraocular pressure was still 5 mm Hg and subsequently it progressed to phthisis bulbi.

Key words

ocular tuberculosis; granulomatous pan-uveitis; miliary tuberculosis; leptomeningeal tuberculosis

Introduction

Tuberculosis (TB) is an airborne infectious disease caused by Mycobacterium tuberculosis and associated with formation of granulomatous infection that has disseminated by haematogenous spread from the lungs. TB is still one of the leading causes of death worldwide, with an estimated annual incidence of 10.4 million and mortality of 1.4 million.[1]Poor socioeconomic factors, immunosuppression and general debility are risk factors.[2]The extra-pulmonary involvement can present as gastrointestinal, central nervous system, ocular, genitourinary or skin which may or may not be associated with pulmonary tuberculosis. Intraocular tuberculosis is most likely due to post-primary infection from the haematogenous spread.[3]We report a case of ocular tuberculosis with miliary and leptomeningeal involvement in an immunocompetent young man.

Case Report

A 17 year old male patient presented with pain, photophobia and defective vision in the Right eye of 4 months duration. He gave history of sudden onset stiffness of left toes progressing toweakness of left lower limb and blurring of vision, irritation and photophobia in the right eye 4 months back.He had consulted a local hospital where,in view of clinical features of left lower limb monoparesis and extensor plantar reflex, MRI imaging was done. MRIimaging of chest (Figure 1) showed miliary mottling with centrilobular markings and fluffy infiltrates suggestive of miliary tuberculosis while MRI brain (Figure 2)showed multiple ring enhancing lesions with leptomeningeal spread, focal cerebritis, and involvement of the paracentral lobule suggestive of leptomeningeal tuberculosis for which he was started on anti-tubercular therapy. Ophthalmologist opinion was taken at the same hospital and a diagnosis of Right Horner's syndrome was made in view of ptosis and miosis.

Figure 1. MRI Chest - miliary mottling with centrilobular markings and fluffy infiltrates (arrows) suggestive of miliary tuberculosis.

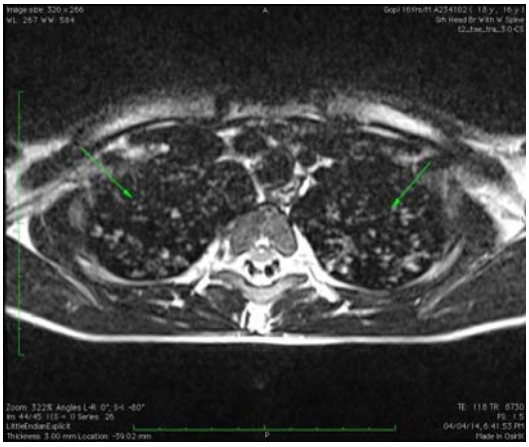
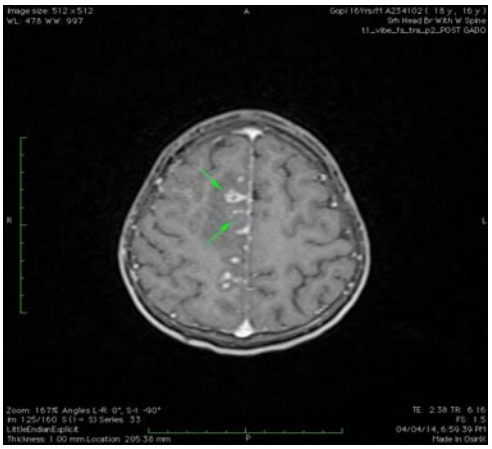


Figure 2. MRI Brain- multiple ring enhancing lesions (arrows) with leptomeningeal spread, focal cerebritis, involvement of the paracentral lobule suggestive of leptomeningeal tuberculosis



Visual acuity at presentation was 1/60 in the right eye and 6/6 in the left. Anterior segment evaluation of the right eye revealed small to medium keratic precipitates, centrally shallow anterior chamber, neovascularisation of iris, posterior synechiae and complicated cataract. Figure 3.Fundus examination was not possible in view of hazy media. Left eye was within normal limits. The intraocular pressure measured using Goldmann Applanation tonometry was 4 mm Hg in the right and 12 mm Hg in the left. Ultrasound B-scan of the right eye showed few dot echoes and no evidence of retinochoroidal complex thickening and retina attached.Figure 4. Ultrasound biomicroscopy of the right eye showed very shallow anterior chamber, large intumescent cataractous lens, cyclitic membrane behind lens and ciliary body atrophy. Blood counts were within normal limits and patient was found to be negative for HIV and syphilis.

Figure 3. Anterior segment photograph of right eye at presentation with complicated cataract

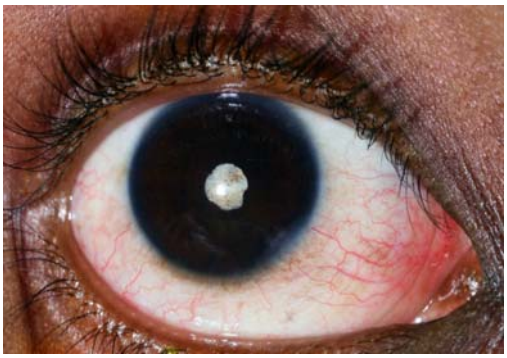
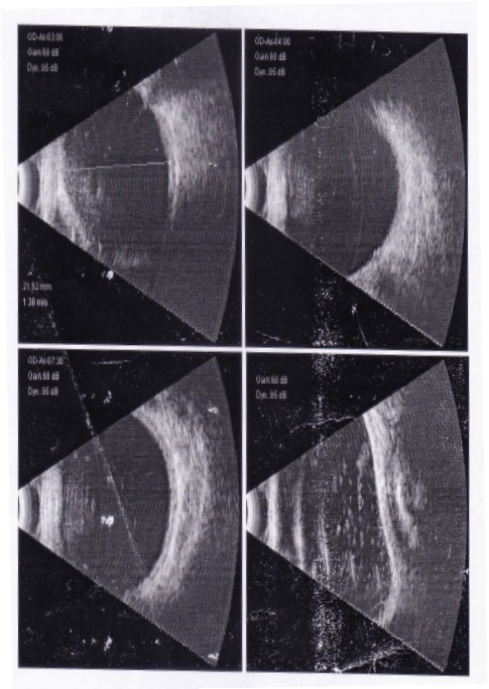


Figure 4. B-scan OD - Incomplete PVD with anterior mild vitritis and disc cupping. Mature cataract Normal axial length and RCS complex No evidence of RD or CD



A diagnosis of Right eye granulomatous pan-uveitis of probable tubercular origin was made and patient was asked to continue anti-tubercular therapy, started on T. Prednisolone acetate 40mg/day for 1 week and tapered and topical Ofloxacin(0.3% w/v)+Prednisolone(1% w/v) eye drops 5 times a day for 1 week and tapered.

Despite all efforts after 1 year, vision in the right eye had worsened to hand motions, intraocular pressure was still 5 mm Hg and subsequently it progressed to phthisis bulbi.

Discussion

The clinical presentations in intraocular tuberculosis areanterior uveitis, intermediate uveitis, posterior or panuveitis,retinitis, vasculitis, neuroretinitis, optic neuropathy, endophthalmitis,and panophthalmitis. Diagnosis is based upon clinical presentation, systemic evaluation and response to treatment. This is however problematic due the wide spectrum of presentations and difficulty in taking biopsy for culture and direct histopathology. Absence of clinically evident pulmonary TB does not rule out the possibility of ocular TB as up to60% of patients with extra-pulmonary TB have no evidence of pulmonary TB and chest X-Rays can be normal in cases of latent TB and early pulmonary TB.[4]Ocular investigation include demonstration of acid-fast bacilli from fluid or tissue which is the Gold standard or positive PCR for *Mycobacterium tuberculosis*. Due to lower inoculum and presence of inhibitors, PCR techniques are more sensitive for pulmonary TB (89%) than non-pulmonary infections (42%).[5,6] Systemic investigations include a positive Mantoux reaction, radiographic features suggestive of healed or active TB or demonstration of AFB in sputum culture or microscopy. Treatment of ocular TB requires control of the infection with multidrug Anti-tubercular therapy as well as the inflammation with topical and oral

corticosteroids. When severe, corticosteroids and supportive management should be given along with ATT to prevent irreversible ocular damage caused by delayed type hypersensitivity reaction.[7]In our case, the initial misdiagnosis as Horner's syndrome and 4 month delay in controlling the inflammation was enough to cause irreversible ciliary body shut down and phthisis bulbi thus reinforcing the need for steroids in ocular TB. Use of corticosteroids alone without concomitant ATT should be avoided as it may promote multiplication of bacilli, leading to panophthalmitis or cause a flare up of systemic tuberculosis by activating a latent infection.[8, 9]

Conclusion

TB is a great masquerade.Due to insensitive laboratory investigations, delayed diagnosis and misdiagnosis is not uncommon.[10] In the Indian setting,a high degree of suspicion must be always present especially in the presence of an extra-ocular focus of TB and/or the absence of other aetiologies. Controlling the infection and inflammation adequately and promptly is crucial in preventing such needless blindness.

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