CASE REPORT

Tuberculomas in “Critical” Locations of the Visual Pathway—A Masquerader

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ABSTRACT

Tuberculosis never stops fascinating physicians. The burden of tuberculosis is a major cause of disability. While drugs are often blamed when patients develop new focal deficits on treatment, the deterioration in these patients can have a myriad of causes including the development of new tuberculomas. Recognizing the critically located tuberculomas as potential causes of vision loss is indispensable. Here, we report two patients with strategic tuberculomas leading to vision loss. In one patient, the cause of visual morbidity was compression of the optic pathways; in the other patient, cortical vision loss was noted due to strategic location of tuberculomas.

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Introduction

Although there is a myriad of pathological substrates that can lead on to vision loss in tuberculosis, the disorder never ceases to fascinate physicians. The burden of tuberculosis is still a major cause of disability in developing countries. Often drugs are blamed when patients develop new focal deficits on anti-tubercular therapy (ATT). Recognising the critically located tuberculomas as potential causes of vision loss is indispensable. Here we report two patients with tuberculomas along critical regions of the visual pathway leading to vision loss. Where in the first case the cause of visual morbidity was compression of the optic pathways, in the second case cortical vision loss was noted due to a selective location of tuberculoma.

Case reports

Case 1

A 15-year-old girl presented with painless progressive vision loss of 1-month duration. History of low-grade fever with loss of weight and appetite was present for the last 4 months. On examination the patient could only perceive hand movements from both eyes. Optic atrophy was noted on fundus examination in both eyes.

Gadolinium-enhanced magnetic resonance imaging (Gd-MRI) revealed multiple ring-enhancing lesions in bilateral cerebral and cerebellar regions and similar conglomerate lesions in the suprasellar region, compressing the optic chiasma (Figure 1a). Cerebrospinal fluid (CSF) analysis showed 80 cells/mm³, all lymphocytes, with a protein level of 88 mg/dL (normal range: <45 mg/dL) and a sugar level of 48 mg/dL (normal range: 45–80 mg/dL) (corresponding blood sugar: 120 mg/dL). The Gram stain, India ink, and cryptococcal antigen were negative, but adenosine deaminase (ADA) was 12.0 U/L (normal range: <10 U/L). Polymerase chain reaction (PCR)-based assay for tuberculosis was negative, and pan-viral screening, cryptococcal antigen, cultures, and malignant cytology were negative. The lesions in the brain in the sellar and suprasellar regions were categorised as tuberculomas compressing the optic chiasma, without any other lesions along the visual pathway. Subsequently contrast-enhanced computed tomography (CT) of chest and abdomen showed multiple mediastinal and abdominal lymph nodes. MRI of dorsolumbar spine suggested tubercular involvement of spine at T12 vertebral level (Figure 1b). The patient was initiated on daily anti-tubercular therapy (four-drug regimen: rifampicin, isoniazid,

Figure 1. (1a) Gd-MRI Brain, T1 weighted image showing lesions in sellar and suprasellar cistern (black and white arrows); compressing the optic chiasma (Case 1). (1b) Sagittal T2 weighted images of spine showing wedge collapse of the T12 vertebrae with reduced disc space (grey arrow). (1c) Gd-MRI Brain, T1 weighted images after 6 months of anti-tubercular therapy showing decrease in lesions (black and white arrows).

pyrazinamide, and streptomycin) with steroids based on radiological evidence and raised CSF ADA. On the 1-month follow-up, the patient had regained her vision to 6/60 in the right eye and counting fingers at 3 m in the left eye, with subsequent repeat MRIs showing a decrease in the size of the lesions (**Figure 1c**).

Case 2

A 64-year-old lady was diagnosed with pulmonary tuberculosis approximately 2 months ago when she presented to a physician with history of low-grade fever, cough, and haemoptysis. She was started on daily anti-tubercular therapy based on a positive sputum acid-fast bacilli (AFB) (four-drug regimen: rifampicin, isoniazid, pyrazinamide, and ethambutol). At 1.5 months into the therapy, she presented with subacute-onset, painless loss of vision in both eyes without field defects or dyschromatopsia to the extent of absent perception of light in both eyes. Ethambutol was considered as a culprit; it was stopped. Even after 2 months of stopping ethambutol there was no improvement in vision, and the patient was referred to our neuro-ophthalmology centre.

On examination, the patient had no perception of light in both eyes, with normal fundus examination, normal pupillary reflexes, normal optical coherence tomography (OCT)-measured retinal nerve fibre layer (RNFL), and normal cranial nerve examination. A Gd-MRI of the brain revealed well-defined, rounded, enhancing lesions,

strategically placed in both occipital lobes (**Figure 2**). CSF analysis showed no cells and normal protein and sugar levels. The Gram stain, India ink, and cryptococcal antigen were negative, and the ADA was 6.0 IU/L. Polymerase chain reaction (PCR)-based assay for tuberculosis was negative. The lesions were consistent with tuberculomas, and intensive phase of ATT was continued (without ethambutol) along with intravenous (IV) steroids, following which there was gradual improvement in vision to perception of hand movements bilaterally.

Discussion

Visual impairment in neuro-tuberculosis has been reported to range from 14% to 35% in various studies.^{1,2} A myriad of causes can be attributable: presence of optochiasmatic arachnoiditis (OCA), involvement of ocular structures (chorio-retinitis or retinal vasculitis), raised intracranial pressure, hydrocephalus, and iatrogenic (drug-induced optic neuropathy) are well-known causes of vision loss in tuberculosis. Tuberculomas per se account for 10–20% of space-occupying lesions in developing countries.³ Recognising the selectively located tuberculomas as potential causes of vision loss is indispensable. In both of our cases, despite negative or bland CSF picture, there was evidence of tuberculosis elsewhere, which was a major help in confirming these lesions as tuberculomas apart from radiological diagnosis. In case of doubtful diagnosis of tuberculosis, one can look for evidence of tuberculosis elsewhere, including imaging of the chest and abdomen. These

Figure 2. (a) Gd-MRI of the brain, T1-weighted image showing contrast-enhancing lesions in right calcarine cortex and bilateral occipital lobe cortices (Case 2). (b) T2 FLAIR images with hyperintense lesions in the corresponding regions.

lesions can mimic a drug-induced optic neuropathy or vasculitis and cause significant delay in achieving right diagnosis and subsequent visual morbidity, as the management differs in each of the cases. Prompt diagnosis and management at the right time may help salvaging vision.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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