CASE REPORT

Unusual fundus examination findings in tuberculous meningitis

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SUMMARY

Vision impairment is one of the devastating complications of central nervous system tuberculosis (CNS TB). Optic atrophy is a sequelae of various forms of CNS TB which, ultimately, is responsible for vision impairment. It is usually the secondary optic atrophy which occurs in CNS TB. In this case report, we present two cases of CNS tuberculosis developing primary optic atrophy, which is infrequently reported. The relevant pathophysiology and causes of vision impairment in CNS TB are also discussed.

BACKGROUND

Central nervous system tuberculosis (CNS TB) is the most important and serious manifestation of extrapulmonary TB. It leads to a number of complications, vision impairment being one of them, associated with significant morbidity. The incidence of vision impairment in CNS TB varies from 27 to 72% in different studies.¹⁻⁴ Most of these patients ultimately develop optic atrophy which is usually secondary. However, primary optic atrophy may also occur in CNS TB, albeit a rare occurrence. Here, we have discussed two cases of CNS TB developing primary optic atrophy along with relevant pathophysiology. It is rarely reported in the literature.

CASE PRESENTATION

Case 1

A 27-year-old lady, presented to us with a history of headache, fever and vomiting for the last 6 months. The patient also complained of decreased vision in both the eyes, since 2 months, which was acute in onset, progressed rapidly so that, after 10 days, the patient was neither able to see nor able to perceive light. Since then the visual deficit was static until admission. There was no history of seizure, altered sensorium or loss of consciousness. There was no history of tuberculosis or tuberculosis contact in the past.

On examination, her vital parameters were normal. Signs of meningeal irritation were present in the form of neck rigidity and positive Kernig's sign. Higher mental functions were normal. In cranial nerve examination, perception of light was absent in both the eyes. Fundus examination revealed chalky white optic disc on right and pale white optic disc on left side with distinct disc margins. There was diffuse arteriolar attenuation. There was no evidence of any choroid tubercles. Arcuate nerve fibre atrophy was seen in supra-tentorial and infra-tentorial areas (right>left). Fundus findings were suggestive of bilateral primary

optic atrophy- (figure 1). There was bilateral sixth cranial nerve palsy. Other extraocular movements were normal. Rest of the cranial nerves were normal. Motor, sensory system examination and deep tendon reflexes were normal with downgoing plantars. Her sensory, cerebellar examination was normal.

Case 2

A 24-year-old lady presented with a history of headache, fever, vomiting for 5–6 months around 5.5 years ago for which she was evaluated and diagnosed as tuberculous meningitis (TBM). She was started on antitubercular therapy. After 3–4 months, she complained of decreased vision in both eyes. It was insidious in onset, slowly progressive for next 1 month and then it became static. The patient was able to identify faces of persons up to a distance of 4 m approximately and not beyond that distance. However, she did not consult any doctor for that and presented to us after 5 years. She had a history of pulmonary tuberculosis 8 years ago for which she received antitubercular therapy for 6 months.

On examination, patient's vital parameters were normal. Her higher mental functions were normal. The vision in right eye was 6/60 and left eye was 6/36. Fundus examination revealed bilateral primary optic atrophy involving right eye more than left eye (figure 3). Rest of the cranial nerve examination as well as motor, sensory and other neurological examinations were normal.

INVESTIGATIONS

Case 1

Routine blood investigations (including CBC, kidney and liver function test and x-ray of the chest) were normal. Erythrocyte sedimentation rate was 40 mm at 1 h. Her cerebrospinal fluid (CSF) examination revealed 85 cells/mm³, all lymphocytes; proteins were elevated (92 mg/dl); sugar was moderately decreased (CSF=40 mg/dl, blood=106 mg/dl). Acid-fast bacilli (AFB) and Gram staining were negative. MRI of the brain revealed abnormal lepto-meningeal enhancement along the basal cistern and in the region of cavernous sinus, along with non-communicating hydrocephalus with transpendymal ooze (figure 1). Optical coherence tomography (OCT) was performed in this patient. It revealed decreased retinal nerve fibre layer thickness bilaterally (right>left) especially in the superior and inferior quadrant (figure 2).

The patient was started on antitubercular therapy and steroids.

To cite: Verma R, Chaudhari TS. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2012-008138

Figure 1 MRI of the brain and fundus photograph of case 1. (A) Axial section showing intense meningeal enhancement along the basal cistern. (B and C) Axial and coronal sections showing meningeal enhancement in the region of cavernous sinus (arrowheads) and hydrocephalus. (D) Fundus photograph.

Case 2

As per discharge summary, when she was diagnosed as TBM, CSF examination revealed 135 cells/mm³, 90% lymphocytes, protein=113 mg/dl, sugar=46 mg/dl (blood sugar=109 mg/dl). AFB staining was negative. x-Ray of the chest revealed fibrocavitary lesion in the right upper zone. CT of the head performed 1 year back revealed multiple calcified nodules, a few of them showing conglomeration in bilateral frontoparietal and left frontal region and gliotic areas in left frontal region with ex-vacuo-dilation of left frontal horn suggestive of chronic infarct. Fourth ventricle was normal. Routine investigation performed during current admission did not reveal any significant abnormality.

OUTCOME AND FOLLOW-UP

The first patient was followed up at 2 months, but there was no improvement in visual acuity.

In the second patient, as the severity of visual complaints was static for the last 5 months and the patient had already received antitubercular therapy 5 years earlier, she was given only symptomatic treatment. There was no improvement in visual acuity at follow up of 2 months.

DISCUSSION

Tuberculosis is one of the major global public health problems in both developing as well as developed countries. More recently, there has been resurging trend in the magnitude of this problem especially in developed countries.⁵ Central nervous system (CNS) involvement is one of the most serious forms of extrapulmonary tuberculosis. It can manifest in a variety of ways such as TBM, TBM with miliary tuberculosis, tuberculous

encephalopathy, vasculopathy and abscess.⁵ Vision impairment is one of the most devastating sequelae associated with CNS TB causing significant morbidity. It may result from optochiasmatic arachnoiditis, compression of optic nerve by tuberculoma, optic nerve granuloma, optic neuritis associated with antitubercular therapy (ethambutol, sometimes isoniazid), secondary to hydrocephalus and raised intracranial tension, bilateral occipital infarcts due to vasculitis and chorioretinitis.^{2–7} Fundus examination may reveal papilloedema, optic atrophy and choroid tubercles, findings sometimes suggestive of neuroretinitis.^{2–5} As aforementioned, the incidence of vision impairment varies from 27 to 72%.^{1–4} The spectrum of visual impairment may range from minimal impairment to complete vision loss. Sinha *et al*¹ concluded that papilloedema, markedly raised CSF protein (> 1 g/l), cranial nerve palsies and optochiasmatic arachnoiditis on MRI are predictors of visual deterioration in TBM. Aaron *et al*⁸ reported that young women having high CSF protein values are at risk of developing optochiasmatic arachnoiditis.

Optic atrophy is one of the sequelae of TBM. In most patients, it is of secondary type, occurring as a result of hydrocephalus with raised intracranial tension, optochiasmatic arachnoiditis, chiasmal compression due to dilated third ventricle or tuberculomas.^{1–5} In such cases, disc margins are blurred. However, sometimes primary optic atrophy, though uncommon, may be seen in TBM.

In this case report, we present two cases of TBM who developed primary optic atrophy. The basal exudates in TBM are prominent in the interpeduncular fossa, sometimes around the chiasma, known as optochiasmatic arachnoiditis. Sometimes, these exudates may occlude the blood vessels supplying the optic nerve or may be affected by tuberculous vasculitis.^{7–9} Such ischaemic involvement of the optic nerve causes destruction of

Figure 2 Optical coherence tomography (OCT) findings in case 1.

optic nerve fibres leading to optic atrophy. In such circumstances, the disc margins will be distinct as in primary optic atrophy. In the first patient, there was a case of probable TBM, as per consensus TBM diagnosis criteria,¹⁰ though there was evidence of communicating hydrocephalus, the patient developed primary optic atrophy with distinct disc margins. This can be attributed to the fact that optic atrophy probably developed as a result of ischaemic insult before the hydrocephalus became severe enough to cause papilloedema. Our second patient also developed primary optic atrophy as a sequelae to TBM.

The purpose of presenting these two cases is to highlight the unusual sequelae of TBM in the form of primary optic atrophy which is infrequently reported in the literature and thus make treating-physicians aware of it.

Figure 3 Fundus photograph of case 2.

Learning points

- ▶ Central nervous system tuberculosis (CNS TB) is one of the most dreaded forms of extrapulmonary tuberculosis.
- ▶ Vision impairment is the devastating complication of CNS TB.
- ▶ Optic atrophy is a sequelae of various forms of CNS TB, and both primary or secondary optic atrophy can occur.
- ▶ Primary optic atrophy may result from ischaemic insult to the retrobulbar part of optic nerve, as a result of tuberculous vasculitis of vasa nervosum or occlusion of arteries supplying the optic nerve by basal exudates.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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