CASE REPORT

Diagnosis of Tubercular Brain Abscess Through Ocular Manifestation

Smita Anand, Kumar Rajiv, Ranjan Ashis

Department of ophthalmology, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India

ABSTRACT

Central nervous system tuberculosis is a severe form of extra-pulmonary tuberculosis. It mainly presents as meningitis or tuberculoma. Tubercular brain abscess (TBA) is a rare manifestation of tuberculosis in an immunocompetent patient. We report a case of TBA who presented to us due to the defective vision and headache.

Keywords: Brain abscess, papilledema, tuberculosis

INTRODUCTION

Tuberculosis remains a major global problem and a public health issue of considerable magnitude. Tubercular brain abscess (TBA) is a rare manifestation of intracranial tuberculosis, the usual presentation being tuberculoma or meningitis.[1] Prevalence of tuberculosis in India is 2.3/million.[2] TBA has been reported in 4% to 7.5% of patients with central nervous system (CNS) tuberculosis without human immunodeficiency virus (HIV) infection compared with 20% in HIV-positive patients.[1,3,4] The rarity and importance of identifying this entity prompted this case report. We report a case of multiple TBA in an immunocompetent young female who presented to us due to defective vision. A Aremu *et al*., reported a case of isolated TBA in an immunocompetent Nigerian adult with good outcome.[5]

CASE REPORT

A 20-year-old female (housewife) presented to our department complaining of defective vision and headache of 4 months duration. Other symptoms were dizziness, vomiting, and intermittent low-grade fever no history of tuberculosis in other part of the body, there was no history of any contact with a tuberculosis patient.

She was thin built, conscious, well oriented with time, place, person, no meningeal sign, no cranial nerve involvement. Her gait was normal. The muscle bulk was normal, nofasciculation, the plantar response was normal. She was anemic. Respiratory, cardiovascular system, abdominal examination were within normal limits. On ocular examination, her best-corrected visual acuity (BCVA) in right and left eyes were 20/200 and 20/120 respectively. Her anterior segment was within the normal limit in both eyes. Fundus examination showed sign of chronic papilledema as severe disc elevation, no hemorrhage, no cotton wool spots. We advised the computerized tomography (CT) scan, which showed multiple hypodense lesions with ring enhancement in the left tempro-parietal region, suggestive of TBA. Cerebrospinal fluid report showed protein 140 mg/dl, glucose 74 mg/dl, cells 110 mm^3 with neutrophilicleucocytosis. Enzyme-linked immunosorbent assay for HIV was negative. Chest X-ray was normal. Blood investigations showed white blood cell 13,800, neutrophills 66%, lymphocytes 32%, eosinophils 2%, hemoglobin 10 g%, and erythrocyte sedimentation rate 23 mm in 1^{st} h. Patient was referred to our neuro-surgery department. Pus was aspirated by borehole technique. Acid-fast bacilli were seen in direct smear, culture and polymerase chain reaction test.[6] Anti tubercular treatment (Directly Observed

Access this article online	
Quick Response Code	Website: www.nigerianjournalofophthalmology.com
	DOI: 10.4103/0189-9171.164497

Address for correspondence
Dr. Smita Anand, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India. E-mail: smita.anand2001@gmail.com

Treatment Cat II) was started, which includes 10 months course of antituberculous drug isoniazid, rifampicin, pyrazinamide, ethambutol for 2 months in intensive phase and with isoniazid and rifampicin for 8 months in continuation phase. Dose of H (isoniazid 5 mg/kg body weight), R (rifampicin 10 mg/kg body weight), Z (pyrazinamide - 25 mg/kg body weight), E (ethambutol 15 mg/kg body weight). She showed improvement in all symptoms including vision after 3 month of treatment as fever, giddiness, vomiting was absent, she gained weight from 40 kg to50 kg, and papilledema was improving. Her BCVA in both eyes improved to 20/30.after 3 months of treatment.

DISCUSSION

Tubercular brain abscess represents an unusual manifestation of tuberculosis of CNS and probably is the result of an altered host response to invasion by tubercular bacilli. It is characterized by an encapsulated collection of pus, containing viable tubercle bacilli without evidence of tubercular granuloma. The incidence of multiple TBA is more common in immunocompromised person as they are unable to mount a granulomatous inflammatory response. Diagnosis of TBA is done mainly on the basis of Acid Fast bacilli (AFB) demonstrated in the pus by culture. Multiple TBA presents as sub-acutein 3rd or 4th decade of life. These are mainly supra-tentorial and rarely in the cerebellum. These may present with the focal neurological deficit and papilledema. In 1978, Whitner proposed the following criteria for establishing the diagnosis of TBA. (1) Macroscopic evidence of a true abscess formation within the brain as confirmed during surgery or autopsy. (2) Histological proof of the presence of inflammatory cells in abscess wall, (3) demonstration of AFB in pus or abscess wall.[1] Mohanty *et al*., presented stereotactic CT-guided aspiration as a useful alternative modality in the management of tubercular abscesses in selected cases.[7] A single case of cerebellar abscess was reported by Oshinowo and Schoenan.[8] Tang *et al*.,[9] reported a case of multiple tubercular abscesses in a child who had tuberculous meningitis.

CONCLUSION

We should carefully examine and investigate the all cases of papilledema, and hence that death from fatal disease like TBA can be prevented.

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How to cite this article: Anand S, Rajiv K, Ashis R. Diagnosis of tubercular brain abscess through ocular manifestation. Niger J Ophthalmol 2015;23:22-3.

Source of Support: Nil, **Conflict of Interest:** None declared