



Tuberculous Meningitis with Obstructive Hydrocephalous with Stroke –A Rare Case Report

KEYWORDS

Tuberculous meningitis (TBM), Hydrocephalous, ventriculo peritoneal shunt (VP shunt)

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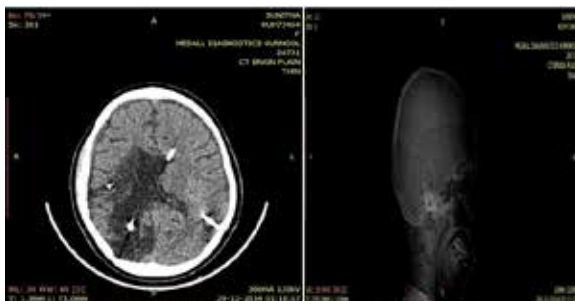
ABSTRACT Tuberculous meningitis is still very common problem in children and adult in india. Mortality and serious long-term sequelae still occur in about 50% of patients with tuberculous meningitis. Distressing sequelae are common such as mental retardation, epilepsy, hypothalamic disturbance, blindness and paraplegia. We here by report a 16 years old girl with TBM, hydrocephalous, mental retardation and stroke

INTRODUCTION

The first clinical description of TBM in the late 18th century was credited to the Scottish physiologist Sir Robert Whytt even before Robert Koch isolated Mycobacterium tuberculosis in 1882. Central nervous system tuberculosis may take several forms including TBM, meningoencephalitis, tuberculoma, tubercular abscess, and rarely myeloid radiculopathy. Of these TBMs, the most common account for 70-80% of neurotuberculosis. Damaging effects to the brain due to formation of granulomatous exudates and perivascularitis of blood vessels supplying the brain. Blindness, deafness, cranial nerve palsies (3rd, 4th, 6th, and 7th) up to 25%, motor deficit is 40% and seizure in 10% of cases is noted. In this paper we describe the complications of TBM observed in these patients.

CASE REPORT

A 16-year-old female brought to the emergency department in a government general hospital, Kurnool, with chief complaints of two episodes of seizures since evening. She was known epileptic patient and had similar episodes of seizures previously. She had a past history of TBM with obstructive hydrocephalus, with right hemiparesis at the age of one year. On examination, she is conscious, coherent, and mentally subnormal. Vitals were within normal limits. All systems were normal except mental subnormality with left hemiparesis.



Bilateral lateral ventricle VP shunt

DISCUSSION

TBM results from the haematogenous spread of bacteria to the central nervous system from the primary focus elsewhere in the body, usually the lungs. A caseous lesion is formed in the cerebral cortex or meninges which ruptures to discharge the tubercle bacilli into the subarachnoid

space, leading to the formation of thick gelatinous exudate that infiltrates the cortico-meningeal blood vessels and causes inflammation and obstruction of vessels around the interpeduncular and pontine cisterns in acute stages and adhesive leptomeningitis in the chronic stages. Clinical features include alteration in consciousness (79%), fever (66%), focal neurological signs (66%), and seizures (53%). TBM with hydrocephalus features headache, vomiting, drowsiness, lethargy, irritability, behavioural abnormality, visual blurring, diplopia, tense fontanelle, distended scalp veins, frontal bossing, macrocephaly, and sun-setting sign in infants, papilloedema, lateral rectus palsy, gait abnormality in older children, and adult.

The strokes associated with tuberculous meningitis are usually ischaemic. The infarcts are often multifocal and haemorrhagic conversion is common. The infarcts are often in areas supplied by the deep penetrating arteries: the basal ganglia, internal capsules, and thalamus, but cortical and subcortical infarcts are not uncommon. Leptomeningeal exudates can cause hydrocephalus by obstructing the flow of CSF from the fourth ventricle, or impairing absorption of CSF by the arachnoid villi. Cranial neuropathies include sixth and third cranial nerves were most frequently affected. Cranial nerve lesions usually result from ischaemia or entrapment of the nerve in the subarachnoid space, but tuberculomas in the cerebellopontine angle or the brain stem can compress cranial nerves. Tuberculomas were uncommon with tuberculous meningitis. Tuberculous brain abscess is a rare

complication, most commonly seen in patients with tuberculous meningitis and concurrent human immunodeficiency virus (HIV) infection. Myelodysplasia is very rare with radicular pain, sensory loss, mixed upper and lower motor neuron signs in the legs, and sphincter dysfunction. Myelodysplasia is caused by leptomeningeal exudates encasing the spinal cord, nerve roots, and blood vessels. A contrast CT scan is the imaging of choice in sick patients, especially children.

Medical management includes steroids, dehydrating agents such as mannitol, diuretics such as furosemide, and acetazolamide to reduce CSF production. Tapering doses of dexamethasone (12 to 16 mg/day) can be given for four to six weeks. The antituberculous therapy (ATT) also probably helps in reducing the inflam-

matory response leading to opening of the CSF pathways. Surgical management of hydrocephalus in patients with TBM included repeated tapping of the ventricles third ventriculostomy (open OR endoscopic) and ventriculo-subarachnoid shunts.

Conclusion

Neurological and systemic complications are common and significant factors contributing to the high rate of mortality and long-term sequelae of tuberculous meningitis, but the best chance of reducing the risk of severe complications is by early diagnosis and treatment of tuberculous meningitis.

REFERENCE

1. Tandon PN. Tuberculous meningitis (cranial and spinal). In: Vinken PJ, Bruyn GW, editors. Handbook of Clinical Neurology. Infections of the Nervous System. vol. 33. Amsterdam: North—Holland; 1978. p. 195-262. | 2. Thwaites GE, Hien TT. Tuberculous meningitis: many questions, too few answers. Lancet Neurol 2005;4:160-70. | 3. Mathew JM, Rajshekhar V, Chandy MJ. Shunt surgery for poor grade patients with tuberculous meningitis and hydrocephalus: Effect of response to external ventricular drainage and other factors on long-term outcome. | 4. Chakraborty AK. Estimating mortality from tuberculous meningitis in a community: Use of available epidemiological parameters in the Indian context. Indian J Tuberc 2000;47:9-5. Singh D, Kumar S. Ventriculoperitoneal shunt in post tubercular hydrocephalus. Ind Pediatr 1996;33:854 | 6. Wadia NH, Dastur DK. Spinal meningitides with radiculo-myelopathy. Part 1. Clinical and radiological features. J Neurol Sci 1969;8:239-60.. | 7. Lamprecht D, Schoeman J, Donald P, Har tzenberg H. | Ventriculoperitoneal shunting in childhood tuberculous meningitis. Br J Neurosurg 2001;15:119-25. |