Hemiparesis in Pediatric Tuberculous Meningitis: A Case Report

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Abstract: Tuberculous meningitis (TBM) can occur as the sole manifestation of tuberculosis (TB) or concurrent with pulmonary or other extrapulmonary sites of infection. It causes high mortality and morbidity. Patients with TBM develop typical symptoms and signs of meningitis. Cranial nerve palsies, hemiparesis, paraparesis, and seizures are common and should raise the possibility of MTB as the etiology of meningitis. We present a case of TBM in an 11-year-old who was admitted to High Care Unit Siloam Dhirga Surya Hospital, Medan, Indonesia with a decreased level of consciousness, sudden weakness of a right extremity, headache, nuchal rigidity, and history of fever and cough. Chest X-ray showed miliary TB and head CT scan showed hydrocephalus. Laboratory results were leukocytosis, hyponatremia, and positive TB IGRA. The patient was then treated with normal saline infusion, anti TB regimen, an antibiotic, and oral corticosteroid. With regular admission of the anti-tuberculosis drug, oral corticosteroid, and physiotherapist, the patient showed improvement in his motoric function. The clinical symptom of TBM may appear as hemiparesis without a seizure. Proper treatment of tuberculous meningitis may lead to a better outcome.

1 INTRODUCTION

Indonesia has one of the highest burden of tuberculosis (TB) globally (WHO, 2018). It is estimated that childhood TB constitutes 10 to 20% of all TB cases in high burden countries, accounting for 8 to 20% of TB-related deaths. Approximately, 25% of pediatric TB cases are extrapulmonary, with tuberculous meningitis (TBM) being the most severe form. Worldwide, tuberculous meningitis (TBM) accounts for the majority of the deaths due to TB (Israni, et al., 2016).

Tuberculous meningitis may present at any age but is less common at the extremes of life. The peak incidence is in children between 2 and 4 years of age. Most early symptoms relate to underlying pulmonary TB present in most infants who develop TBM as a complication of primary infection (Chin, 2014). The most commonly recorded signs and symptoms of TBM were an altered level of consciousness (90.1%), meningism (77.2%), fever (68.2%), and loss of appetite (61.4%). Focal neurological signs included unilaterally non-reactive pupils (13.6%), other cranial nerve palsies (22.7%), limb paresis (27.3%), and aphasia (18.2%) (Rohlwink, et al., 2016).

2 CASE PRESENTATION

An 11-year-old boy was admitted to the High Care Unit (HCU) Siloam Dhirga Surya Hospital with a decreased level of consciousness, headache, and sudden weakness on the right extremities. The patient had been hospitalized before with fever and cough. There was a history of fever and cough for two weeks and weight loss in one month. No history of close contact with active TB patient, but his father was suffering from chronic cough without receiving any medication. Physical examination revealed somnolence (GCS 14), slurred communication, and normal temperature. On neurological examination, there was nuchal rigidity, clonus on the right extremities, positive Babinski sign, and no muscle contraction in the right extremities. No presence of seizure. The chest was clear bilaterally.
Laboratory test showed leukocytosis (11,820/mm³) with elevated neutrophil (74.9%) and Erythrocyte Sedimentation Rate (ESR). The patient was hyponatremic (127 meq/L) and had positive TB IGRA.

Chest X-ray showed miliary TB (Figure 1), and the CT of the brain showed mild hydrocephalus which suggests meningitis (figure 2 and figure 3). Tuberculin test was negative (0 mm). The lumbar puncture could not be performed technically.

The patient was then treated with four anti-tuberculosis drugs (isoniazid, rifampicin, pyrazinamide, and ethambutol), oral corticosteroid, mannitol infusion, and normal saline infusion. Antibiotic meropenem was added to the treatment. Previously before admitted to HCU, the patient had been given ceftazidime as an antibiotic. The patient showed improvement after 3 days in the HCU. His consciousness began to improve, no headache and nuchal rigidity and maintained a stable of hemodynamics. The patient still had right hemiparesis. He was moved to the ward and was discharged with the anti-tuberculosis regimen and oral corticosteroid.

The patient was then scheduled for having physiotherapist regularly twice a week. With regular administration of the anti-tuberculosis drug, oral corticosteroid, and physiotherapist, the patient showed an improvement in his motoric function. Two weeks after being discharged from the hospital, he was able to walk by himself.

3 DISCUSSION

Neuro-tuberculosis is the most serious complication of TB in children. Among the various forms of neuro-tuberculosis, TBM remains the most severe and the most common in developing countries (Torok, 2015). Tuberculous meningitis continues to be an important cause of morbidity (especially neurologic impairment) in children from resource-poor countries (Israni, et al., 2016).

Owing to the suboptimal performance of diagnostic tests of TBM, the diagnosis in children relies on a thorough assessment of all the evidence derived from a careful evaluation of medical history, clinical examination, and relevant investigations. Approximately 60% of children with TBM have radiological evidence of pulmonary TB (Toorn & Solomon, 2014).

Tuberculous meningitis is a subacute meningitis illness, which presents in various stages. According to the British Medical Council Staging System, tuberculous meningitis can be divided into 3 stages. The first stage consists of nonspecific symptoms of low-grade fever, headache, irritability, drowsiness, malaise, vomiting, photophobia, listlessness, and poor weight gain/weight loss. The second stage shows a sign of meningeal irritation with or without
slight clouding of consciousness with focal neurological signs such as cranial nerve palsies or hemiparesis. In advanced clinical stages, TBM presents severe clouding of consciousness or delirium, convulsions, and serious neurological signs such as hemiplegia, paraplegia, involuntary movement (Israni, et al., 2016).

Tuberculin skin test (Mantoux test) may be nonreactive in 50% cases of CNS TB. Hence, it is helpful in supporting the diagnosis of TBM when positive, but an isolated positive Mantoux cannot be used to label a case of TBM, as false positive/false negative reactions are commonly known (Aulakh & Chopra, 2018).

Tuberculous meningitis usually presents with a Cerebrospinal fluid (CSF) of 10–500 cells/μL that are polymorphs initially and lymphocytes later. A low glucose <40 mg/dL (rarely<20 mg/dL) or a CSF/plasma glucose ratio <50% or a high-protein content (400-5000 mg/dL) is suggestive of the diagnosis of TBM. The CSF lactate levels are usually raised to 5–10 mmol/L (normal range, 1.2–2.1 mmol/L). Ziehl–Neelsen (ZN) staining for the smear examination has a sensitivity of approximately 50%, whereas a bacterial culture has a sensitivity of 60% to 70% (Aulakh & Chopra, 2018). CT scanning and MRI of the brain may reveal hydrocephalus, basilar meningeal enhancement, infarcts, edema, and tuberculomas (Toorn & Solomons, 2014).

This patient presented with a decreased level of consciousness, headache, nuchal rigidity, positive Babinski sign, right hemiparesis. TB IGRA was positive in this patient. He previously complained of cough and fever for two weeks and weight loss for 1 month. Chest X-ray also showed miliary tuberculosis, and the CT of the Brain showed mild hydrocephalus which suggests meningitis. From the clinical examination, diagnostic tests, this patient can be categorized into stage 2 of TBM.

WHO recommends a 12-month treatment plan (2HRZE/10RH) for children with suspected or confirmed TBM (Toorn & Solomons, 2014). We gave anti-tuberculosis drugs to this patient according to the WHO guideline. Meropenem was given due to the possibility of bacterial meningitis which is evidently suggested by leukocytosis and increased neutrophil count. Hyponatremia occurs in up to 85% of children with TBM and is thought to be secondary to either syndrome of inappropriate antidiuretic hormone or cerebral salt wasting. We also found hyponatremia in this patient which was treated with normal saline fluid. Corticosteroid oral was given to reduce the risk of death and neurological deficit (Toorn & Solomons, 2014).

The patient showed clinical improvement in his motoric function after being given an anti-tuberculosis drug regimen, oral corticosteroid, and physiotherapist.

4 CONCLUSION

Patients with TBM develop typical symptoms and signs of meningitis including headache, fever, and stiff neck, although meningeal signs may be absent in the early stage. The duration of symptoms before presentation ranges from several days to several months. In particular, in resource-limited settings, TBM cases may present in advanced clinical stages, with GCS scores of 10 or less. Cranial nerve palsies, hemiparesis, paraparesis, and seizures are common and should raise the possibility of tuberculous meningitis as the etiology of meningitis (Chin, 2014).

This case shows that clinical symptoms of tuberculous meningitis can appear as hemiparesis without a seizure. Proper treatment of tuberculous meningitis may lead to a better outcome.

REFERENCES


