CAPTURING TUBERCULAR MENINGITIS PROGRESSION IN CHILDREN WITH MULTI-SLICED COMPUTED TOMOGRAPHY (MSCT) SCAN: A PROSPECTIVE CASE REPORT

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ABSTRACT

Background: Tubercular meningitis in children poses a diagnostic challenge, either for its detection and follow-up. Neuroimaging is essential in the diagnosis process and early identification of complication or sequelae. Computed tomography (CT) scan is widely available in secondary and tertiary hospitals in Indonesia, which would improve the management of paediatric tubercular meningitis case.

Objective: To describe the clinical progression of a child with tubercular meningitis from detection to sequelae at Bethesda Hospital, Yogyakarta.

Case description: A 7-year-old boy was admitted to the emergency unit with acute headache, vomiting, and prolonged fever. Tuberculosis transmission indicated from the mother, which on TB treatment. Non-contrast multi-sliced computed tomography (MSCT) scan was performed on the first-day admission, on the 8th day of admission, before and after VP-shunt procedure for life-threatening hydrocephalus and the follow-up. There was an obvious disease progression shown by CT scan from the early stage, the onset of hydrocephalus, deteriorating hydrocephalus, and post-VP-shunt procedure. The periventricular infarct extended over time, concordant with worsening symptoms.

Conclusion: Paediatric tubercular meningitis diagnosis relies on accurate neuroimaging studies to identify the specific sign. In the disease progression, the sign as early as slight hyperdensity should lead to meningitis diagnosis, and earlier treatment of hydrocephalus might prevent further debilitating sequelae or even fatal complications of tubercular meningitis.

Keywords: meningeal tuberculosis, child, multidetector computed tomography, hydrocephalus
INTRODUCTION
Tuberculosis (TB) is one of the deadliest infections in Indonesia and accounts for 11,993 death from an estimated 842,000 total cases up to March 2020. Around 11% of 2019 cases were extrapulmonary TB, and 2 out of 10,000 children were affected with TB.1 The diagnosis of pulmonary TB in children is quite difficult with general or minimal symptoms, so many children were brought to medical care in the late stages of severe condition.2 Tubercular meningitis is one type of TB infection in the central nervous system, which responsible for the highest mortality and debilitating sequelae in paediatric TB cases. Initial meningeval involvement usually manifests as fever, headache, and malaise in the first one to two weeks. If not recognised early or untreated, it might lead to more severe symptoms such as loss of consciousness, seizure, and even death.3 Current guideline in Indonesia suggests the examination of cerebrospinal fluid, and brain CT scan or MRI when the sign of increased intracranial pressure appears. Further confirmation of TB infection, such as chest X-ray, culture test, and acid-fast bacilli (AFB) test of cerebrospinal fluid or gastric lavage, although those tests might yield false-negative results.4 Neuroimaging, such as multi-sliced computed tomography (MSCT) scan, plays an essential role in early detection of tubercular meningitis, and identification of complication.5,6 This case report narrated an example of neuroimaging utilisation in tubercular meningitis affected a 7-year-old male child admitted to Bethesda Hospital, Yogyakarta.

CASE DESCRIPTION
A 7 years old male child was presented to emergency unit Bethesda Hospital with sudden headache and vomiting and had 3 weeks history of persistent fever. He lived with her mother, who was diagnosed with pulmonary TB (positive AFB) one year earlier. The patient did not receive any prophylaxis treatment. He was immediately admitted under paediatrician supervision.

On the first day of admission (11th February 2019), there was nuchal rigidity and abducens nerve paralysis, on which the first head MSCT was ordered. There was minimal hyperdensity on the right temporal region (Figure 1), which suggest an early sign of meningitis.

![Figure 1. The axial brain MSCT scan result on 11th February 2019 showing a slight hyperdense area in the right temporal region.](image-url)
The second MSCT (Figure 2) was ordered on 19th February 2019 showing obstructive hydrocephalus and multiple glialosis in the thalamus, basal ganglia and bilateral internal capsule, which is a sign of thrombosis caused by tubercular granuloma. The patient was discharged with a treatment regimen for extra-pulmonary TB.

On 30th May 2019 he returned to the emergency unit with vomiting, acute headache, spastic seizure and clonus. The parents admitted that they discontinue TB treatment without physician consultation. The head MSCT scan shows blurred parenchymal differentiation, obscured cortical sulci and gyri, narrowing Sylvian fissure and periventricular region, and symmetrical enlargement of the ventricular system (Figure 3). Evan’s index of ventricle dilation is higher than 30%. Therefore, these findings suggested worsening obstructive hydrocephalus with periventricular hypoxia and parenchymal inflammation. The patient underwent further intracranial pressure relief with a ventriculoperitoneal (VP) shunt procedure.

Figure 2. The axial non-contrast brain MSCT scan result on 19th February 2019 showing the dilating ventricular system (a) and signs of gliosis and tubercular granuloma in multiple sites (b, c).

Figure 3. The axial non-contrast brain MSCT scan result on 30th May 2019 showing the dilating ventricular system (a, b, c).
Post-VP shunt imaging was performed on 10th June 2019, showing soft tissue and bone defect in the right parietal region, a hypodense lesion on both thalamus (similar to the CSF density), dilation of both lateral, third and fourth ventricle with periventricular oedema. The VP shunt end was attached inside the right lateral ventricle (Figure 4). The patient was discharged after the symptoms resided.

On the 23rd of July 2019, the patient returned again to the hospital with vomiting. The brain MSCT scan showed the slightly reduced hydrocephalus on both lateral, third and fourth ventricle compared to the post-VP shunt MSCT scan. There was also post-encephalitis gliosis in the cerebral cortex of left frontal lobe and both thalamus (Figure 5).

**Figure 4.** The axial non-contrast brain MSCT scan results post-VP-shunt procedure on 10th June 2019 showing the position of VP shunt (b).

**DISCUSSION**

Neuroimaging plays an important role in the diagnosis and follow-up of tubercular meningitis. In the absence of pulmonary TB symptoms or inconclusive microbiological evaluation, neuro-imaging might become an important diagnostic clue in the decision to start extrapulmonary TB treatment. It complements thoracic X-ray to confirm TB diagnosis; usually in the form of miliary TB in children. Brain imaging could detect the earlier tuberculous granuloma, usually on subpial and subependymal surface of the cerebral and spinal cord, and further meningitis sign in subarachnoid space and ventricular system. Furthermore, neuroimaging confirms the presence of complications, such as hydrocephalus caused by parenchymal infarct.

The clinical manifestation of meningitis TB would help in finding abnormalities in imaging studies. Loss of consciousness might suggest increased intracranial pressure or brain herniation. Focal neurologic deficit, such as monoplegia, hemiplegia, and aphasia, would suggest the level of the central nervous system affected by the TB infection. Tremor, choreoathetosis and hemiballismus, which occur more often in children, suggests cerebellar and vascular involvement.
The CT scan of tubercular meningitis might show leptomeningeal and cisterns enhancement, hydrocephalus-related ventriculomegaly, periventricular infarction, and tuberculoma. Meningeal infection is distinguished by a contrast-enhanced lesion on pia mater and arachnoid space, which might extend to the sulci, basal cisterns, and dural folds of the falx cerebri and the tentorium cerebelli. Generally, the CT scan would display basal cistern obliteration by isodense or slight hypodense exudate.

The presence of hydrocephalus and focal infarct is specific findings of tubercular meningitis. In neuroimaging, hydrocephalus is defined as ventricle enlargement with Evans’ ratio over 30%, and/or enlargement of one or two temporal horn over 2 mm. The presence of the fourth ventricle determines the communicating-type hydrocephalus, and otherwise obstructive-type if the fourth ventricle size is normal. Focal infarction is vaguely visible in the basal ganglia, thalamus, and internal capsule. It might be easier to identify when there is secondary ischemia due to vasculitis in the surrounding area. Regardless, MRI provides superior sensitivity in detecting infarction compared to CT scan. Tuberculoma usually visible in contrast-enhanced CT scan, resembling ring capsule with irregular edge and various thickness. Further MRI might identify caseous tuberculoma with central liquefaction.

The patient was presented with the meningeal inflammation signs. However, the fever might not indicate the initial pulmonary TB, as it is too early to develop into meningitis. The fever might indicate the start of extrapulmonary progression, as pulmonary TB symptoms in children is not specific. In this case, there was
a clear progression of meningitis from the first scan on 11th February, to the second scan on 19th February, where the pathognomonic sign visible, such as the early sign of obstructive hydrocephalus and periventricular infarct. The third scan on 30th May showed further deterioration, directly caused by the TB and secondary by the hydrocephalus. This could be explained by very low treatment adherence, which worsened the focal destruction on the periventricular region. Although the VP shunt reduced the intracranial pressure, there were already severe damage resembles post-encephalitis glosis.

The MSCT scan had identified the obstructive hydrocephalus quite early in the disease progression; however, the invasive treatment is delayed until after severe symptoms occurred three months after. Combined with the destruction caused by TB, the increased intracranial pressure left the patients with cerebral damage. Although it is not irreversible, this patient outcome might be favourable when the surgical management offered in the earlier stage of hydrocephalus, thus diminishing the related sequelae.13

Unknown to the physician was the background of medication discontinuation by the parents, which might be related to the side effects of medication or symptoms of deteriorating hydrocephalus. In addition, expanding destruction might be caused by resistant TB, which was not further confirmed in this case. Besides, the attending physician on the second admission did not have complete information on whether the patient received other treatments outside the continuous follow-up from our outpatient clinic. Therefore, we could not exclude the possibilities of aggravating factors from other unknown treatments. Continuous and intensive monitoring of paediatric patients with tuberculous meningitis is important, specifically in the core TB treatment and signs of complication.14

CONCLUSION

Paediatric tubercular meningitis is a diagnostic challenge, which relies on accurate neuroimaging studies to identify the specific sign. It could assist in the treatment decision and procedure follow-up, although the diagnosis of pulmonary tuberculosis is inconclusive. In the disease progression, the sign as early as slight hyperdensity should lead to meningitis diagnosis, and earlier treatment of hydrocephalus might prevent further debilitating sequelae or even fatal complications of tubercular meningitis.

CONFLICT OF INTERESES

The authors declare no conflict of interests regarding this case report.

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