JNI Jurnal Neuroanestesi Indonesia

Case Report

Multiple Large Cerebral Infactions in Tuberculous Meningitis: A Rare Case

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Received: April 12, 2025; Accepted: October 15, 2025; Publish: October 21, 2025

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Abstract

Cerebral infarction is well known as a consequence arising from tuberculous meningitis (TBM), which generally involves the small and medium-sized intracranial arteries. These infarcts are usually located in regions termed the "TB zone," perfused by the medial striate and thalamo-perforating arteries, and in the "ischemic zone," supplied by the lateral striate, anterior choroidal, and thalamogeniculate arteries. In contrast, the involvement of larger arteries is an uncommon feature of tuberculous vasculitis. We report the case of a 24-year-old man with TBM and pulmonary tuberculosis, without HIV infection, who developed loss of consciousness after undergoing a ventriculoperitoneal (VP) shunt procedure. Neuroimaging with computed tomography (CT) revealed extensive cerebral edema accompanied by massive infarctions involving the cerebellum, cerebral hemispheres, and brainstem. These findings illustrate an unusual presentation of multiple large cerebral infarctions associated with TBM. Such extensive infarcts represent severe complications that can be resulted in profound neurological deficits. This case underscores the importance of early recognition and management of TBM-related complications. Prompt initiation of antituberculosis therapy is essential to reduce the risk of fatal outcomes. Moreover, further investigations are warranted to establish more effective therapeutic approach and optimize patient prognosis.

Keywords: Tuberculous meningitis, brain infarction, large artery, treatment, VP shunt

J. neuroanestesi Indones 2025;14(2): 94–100

Introduction

Although significant progress has been made in both the diagnosis and management of tuberculosis (TB), the disease continues to pose a major global health challenge, causing high levels of illness and death, especially in low- and middle-income nations. While the lungs are most frequently affected, TB can spread via the bloodstream and affect other organs, including the central nervous system (CNS). Hematogenous spread can be resulted in tuberculous meningitis (TBM), a serious manifestation that accounts for roughly 1–2% of the total tuberculosis burden and approximately 10–15% of extrapulmonary presentations.[2,3] The complications of TBM span hydrocephalus, tuberculoma formation, and cerebrovascular events. Ischemic stroke is reported in 6–47% of patients and is typically linked to vasculitis, arterial thrombosis, and proliferative vascular changes , leading to irreversible brain injury and poor clinical outcomes . While involvement of small- and medium-caliber intracranial vessels is well recognized in TBM, large-artery disease is distinctly uncommon.[2,4,5] Here, we report a patient with TBM who developed multiple, large cerebral infarctions as an unusual complication that underscores the severity of TBM-associated cerebrovascular involvement.

Case

doi: https://doi.org/10.24244/jni.v14i3.688

ISSN (Print): 2088-9674 ISSN (Online): 2460-2302

JNI is accredited as Sinta 2 Journal: https://sinta.kemdikbud.go.id/journals/profile/796

How to cite: Kustila E, et al, "Multiple Large Cerebral Infarctions in Tuberculous Meningitis: A Rare Case".

History

A 24-year-old man presented to the emergency unit of a medical facility located in Bandung with fever and altered consciousness. He experienced impaired awareness and communication difficulties for seven days, preceded by a three-week history of fever and headache. He had a medical history tuberculosis of the lung three years earlier and had completed treatment.

Physical examination

On clinical assessment, the patient's Glasgow Coma Scale (GCS) was 12 (E3M5V4). Vital assessment indicated BP 130/80 mmHg, HR 70 bpm, RR 20/min, and temperature 37.5 °C. Physical examination identified abdominal distension, while neurological evaluation demonstrated neck stiffness and weakness on the left side of the body.

Laboratory Examination

Laboratory evaluation revealed mild anemia (hemoglobin, 11.0 g/dL), leukocytosis (16.8 × 10^3/μL), low serum sodium and potassium level: 134 mmol/L and 3.4 mmol/L, respectively. Chest radiography revealed findings consistent with active pulmonary tuberculosis, while electrocardiography showed sinus rhythm. The

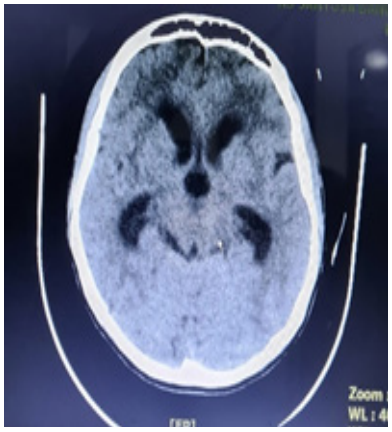

Figure 1

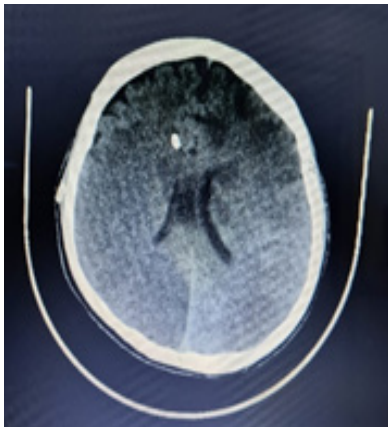

Figure 2a

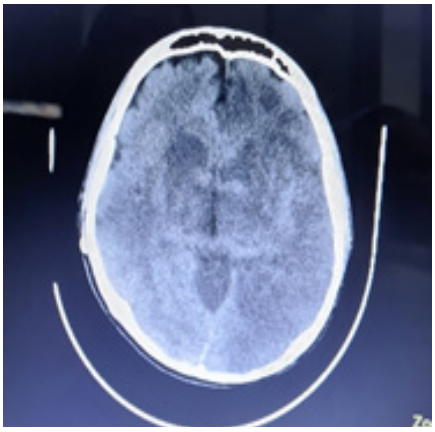

Figure 2b

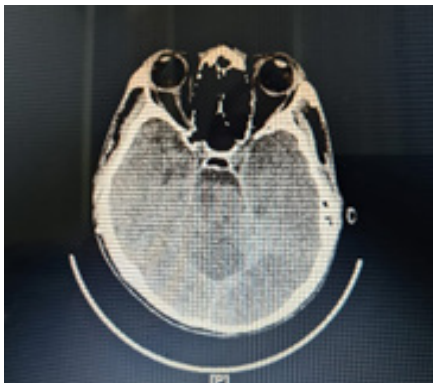

Figure 2c

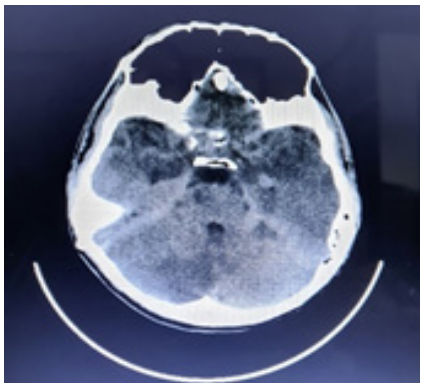

Figure 2d

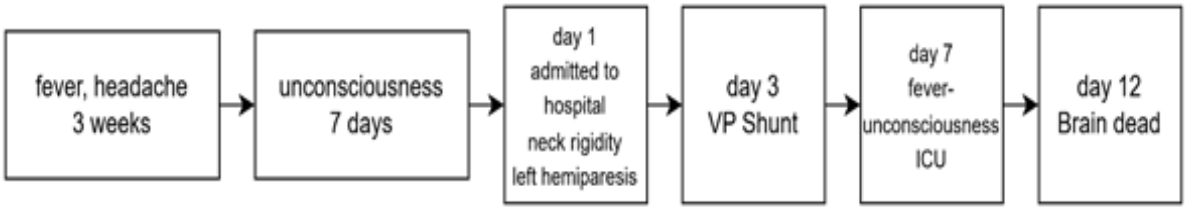

Figure 3. History of Clinical Timeline

Table 1. Hematology and Biochemistry Tests

Parameter	Result	Normal Reference
Hemoglobin	11.0 g/dL	13.2-17.3
Leucocyte	18.68 Th/uL	3.80-10.60
Haematocrit	34%	40-52
Thrombocyte	354 Th/uL	150-450
MCV	56.3 fL	80.0-97.0
MCH	18.5 pq	27.0-31.0
MCHC	32.8 g/dL	32.80-10.60
Neutrophil	89%	50-70
Lymphocyte	2%	20-40
Monocyte	9%	02-Aug
Eosinophil	0	01-Apr
Basophil	0	0-1
Lymphocyte absolute	0.37 Th/uL	
Absolute Neutrophyl Count	16.59 Th/uL	1.5-7.0
Neutrophyl limfosit Ratio	44.05.00	
Glucose at Random	99 mg/dL	55-180
AST	23 U/dL	< 40
ALT	29 U/dL	< 41
Ureum	43 mg/dL	Oct-50
Creatinin	0.66 mg/dL	0.67-1.17
Sodium	124 mmol/L	135-153
Potassium	3.6 mmol/L	3.5-5.3
Magnesium	1.79 mg/dL	1.59-2.56
Chloride	101 mmol/L	98-109
Anti-HIV	non-reactive	non-reactive
Blood Gas Analyze		
pH	7.422	7.350-7.450
pCO2	44.9 mmHg	35.0-45.0
pO2	79.0 mmHg	80.0-105.0
Bicarbonate	29.4 mmol/L	22.0-26.0
Spinal Fluid		
Cell count	4 Cell/uL	
Nonne Test	Negative	Negative
Pandy test	Positive	Positive
Glucose	49 mg/dL	45-70
Total Protein	55.6 mg/dL	15.0-45.0
LDH	79 U/L	

ALT – alanine aminotransferase; AST – aspartate aminotransferase; MCV – average red cell volume (mean corpuscular volume); MCH – average hemoglobin content per red blood cell (mean corpuscular hemoglobin); MCHC – concentration of hemoglobin within red blood cells (mean corpuscular hemoglobin concentration); LDH – lactate dehydrogenase. Arterial blood gas (ABG) parameters included the measurement of pH, partial pressure of carbon dioxide (pCO_2), and partial pressure of oxygen ($pO_{2)}$

patient was confirmed to be HIV-negative (Table 1). A non-contrast brain computed tomography (CT) scan revealed ventricular enlargement indicative of acute hydrocephalus, evidenced by the dilation of the frontal horns of the lateral ventricles relative to the skull diameter (Figure 1). The patient was promptly referred for neurosurgical management, and a ventriculoperitoneal (VP) shunt was inserted. Cerebrospinal fluid (CSF) analysis showed a white blood cell count of 4 cells/µL, a glucose level of 49 mg/dL with a corresponding serum glucose of 99 mg/dL (CSF/serum ratio 0.49), a total protein concentration of 55.6 mg/dL, and a positive Pandy test, while the Ziehl–Neelsen stain yielded negative results (Table 1).

With a clinical suspicion of abdominal tuberculosis leading to obstructive ileus, the digestive surgery team recommended fasting prior to surgery. The patient received intravenous treatment consisting of dexamethasone 10 mg given three times daily, pantoprazole 40 mg once daily, paracetamol 1 g three times daily, and ceftriaxone 2 g once daily. Antituberculosis therapy was started but was discontinued after two days because of concerns related to ileus. Following the ventriculoperitoneal (VP) shunt procedure, the patient's consciousness improved, and abdominal distension subsided. Physical examination showed that the level of consciousness improved, with a GCS score of 14. Abdominal assessment showed a soft, non-rigid abdomen with normal intestinal sounds. Nevertheless, four days later, he experienced a sudden decline in consciousness (E1M4V1) accompanied by fever. Follow-up non-contrast brain CT revealed extensive infarctions involving both cerebellar hemispheres (Figure 2d), bilateral cerebral hemispheres (Figures 2a and 2b), and the brainstem (Figure 2c), accompanied by widespread cerebral edema.

To manage the increased intracranial pressure, the patient received 20% mannitol at a total daily dose of 500 mL, administered in three divided portions. A paradoxical reaction was suspected, leading to an escalation of dexamethasone therapy to 0.4 mg/kg/day.

Post-surgical management

The patient was subsequently managed in the intensive care unit (ICU), where endotracheal intubation and ventilatory support were initiated. Decompressive surgery was not pursued in view of worsening neurological decline linked to severe cerebral edema. Despite intensive management, the patient's status continued to deteriorate, and brain death was confirmed three days later.

Discussion

In this case, the diagnosis of large-vessel occlusion was made on the basis of the acute loss of consciousness accompanied by rapidly worsening intracranial hypertension, which correlated with the extensive infarctions observed on CT imaging. The diagnosis of tuberculous meningitis (TBM) was considered based on symptoms persisting for over five days, including fever, headache, and a mix of focal as well as generalized neurological impairments. The diagnosis was further supported by cerebrospinal fluid (CSF) findings showing a clear appearance with a CSF-to-blood glucose ratio below 50%, as well as radiological evidence of hydrocephalus and cerebral infarction. Chest radiography revealed active pulmonary tuberculosis, and no alternative etiology was identified. The low sodium level in this patient worsened their state of consciousness. Hyponatremia can cause loss of consciousness due to cerebral edema and impaired neuronal function resulting from disrupted osmotic balance in the brain.

In areas where tuberculosis is highly endemic, such as Indonesia, cases of subacute meningitis should strongly raise suspicion of TB as the underlying etiology, especially if patients exhibit the typical triad of fever, headache, and nuchal rigidity. Focal neurological deficits, frequently encountered in TBM, are believed to be resulted from longstanding vascular inflammation. The inflammatory process in tuberculous meningitis may be resulted in vascular thrombosis and compression of blood vessels within the Virchow–Robin spaces due to thick, gelatinous leptomeningeal exudates, or arise from the systemic hypercoagulable state commonly associated with tuberculosis.[6] In this case, alternative etiologies

of subacute meningitis, including parasitic and fungal infections, were excluded as the patient was immunocompetent. Such infections are more frequently encountered in immunocompromised populations, particularly in individuals with HIV/AIDS. Additional diagnostic evaluations, such as lateral flow assays, India ink staining, and serological testing for toxoplasmosis, were not conducted because of insurance constraints. The patient's previous history tuberculosis of the lung strengthened the suspicion of TBM, suggesting prior exposure to Mycobacterium tuberculosis. Hydrocephalus represents one of the commonest complications in tuberculous meningitis (TBM), affecting around 56% of cases. It typically appears as part of a distinctive neuroradiological triad, alongside basal meningeal enhancement and cerebral infarction.[7] In the present case, the placement of a ventriculoperitoneal shunt (VPS) proved beneficial. This is consistent with a study from China involving 14 patients with TBM-related hydrocephalus, where 92.9% experienced favorable short-term recovery and 57.1% demonstrated good long-term outcomes.[8] Comparable results have been described in both adults with severe TBM[9,10] and in pediatric populations,[11] although some studies have reported different results.[12]

The manifestation of hemiparesis as part of the long-tract signs was consistent with cerebral infarction, which may have been compounded by new vascular occlusive events despite the administration of ATDs and steroids. Ischemic stroke is a relatively frequent complication of tuberculous meningitis (TBM), occurring in about 20–30% of affected patients. Cerebral infarctions may manifest clinically or remain silent. The presence of infarction in TBM has been associated with up to a threefold higher risk of fatal outcome compared with cases without infarcts.[13] These infarcts are generally attributed to vasculitic changes and/or reactive intimal proliferation, thought to be arisen from direct seeding of tubercle bacilli via the bloodstream. The vessels most often involved lie at the brain base and within the sylvian fissures, areas where exudates tend to accumulate. Exudative basal meningitis promotes vascular compromise through strangulation, vasospasm, luminal narrowing, and periarteritis, ultimately leading to infarction. The so-called "tubercular zone" of the brain—comprising the basal ganglia and internal capsule, which are immersed in basal exudates—is especially vulnerable.[15]

In tuberculosis, ischemic events within the brain predominantly affect the anterior circulation, with frequent involvement of the middle cerebral artery as well as the medial and lateral striate branches.[1] Roughly three-quarters of infarctions occur in the region referred to as the "TB zone," supplied by the medial striate and thalamo-perforating vessels. By comparison, only around 11% are localized in the "ischemic zone," which receives blood flow from the lateral striate, anterior choroidal, and thalamogeniculate arteries. Tuberculosis is well known to affect the small- and medium-caliber intracranial vessels, whereas large arterial involvement represents an uncommon feature of tuberculous vasculitis.[3] Worsening of clinical status despite antituberculosis treatment (ATT) is observed in nearly 30% of patients with tuberculous meningitis and is commonly known as a paradoxical reaction. This condition is marked by the worsening of already existing lesions or the emergence of additional problems in individuals who initially exhibited improvement after beginning therapy.17 Importantly, the diagnosis of a paradoxical response is considered only when such deterioration occurs after at least 10 days of continuous ATD treatment.[18,19]

In the present case, the patient had received antituberculosis therapy (ATDs) for just two days, making the possibility of a paradoxical reaction improbable. Subsequent clinical decline was more likely to be a direct result of tuberculous meningitis itself, which frequently arises during the first month of illness.[20] Corticosteroids and mannitol were administered with the aims to manage inflammation and reduce intracranial pressure. Together, these therapies support anti-TB treatment by improving cerebral perfusion and neurological recovery. However, depite treatment with corticosteroids and mannitol, cerebral edema persisted and was insufficiently controlled in our patient. We describe a rare case

of tuberculous meningitis (TBM) in an HIV-negative patient with pulmonary tuberculosis, complicated by multiple extensive cerebral infarctions. Diagnosis was established based on clinical presentation, cerebrospinal fluid findings, and radiological evidence of the disease. Within ten days, follow-up CT demonstrated extensive infarctions in various regions, likely related to the delayed initiation of antituberculous therapy or severe immunosuppression

Conlucsion

This report illustrates the complexity of tuberculous meningitis (TBM) management, particularly in the presence of uncommon but severe adverse condition, such as large vessel occlusion, multiple ischemic lesions, and hydrocephalus. While TBM diagnosis in endemic regions can often be established clinically, timely initiation of antituberculosis therapy is critical to improving prognosis. Despite appropriate interventions, including corticosteroids and ventriculoperitoneal shunting (VPS), the patient experienced rapid deterioration, underscoring the unpredictable and multifactorial course of TBM.

Conflict of Interest

The authors stated that there are no financial or personal conflicts of interest that could have affected the content of this manuscript.

Author Contribution

E. K. wrote the first complete draft and collected the data, and S. D. contributed to writing and reviewing the manuscript.

List of Abbreviations

ALT – alanine aminotransferase; AST – aspartate aminotransferase; MCV – average red cell volume (mean corpuscular volume); MCH – average hemoglobin content per red blood cell (mean corpuscular hemoglobin); MCHC – concentration of hemoglobin within red blood cells (mean corpuscular hemoglobin concentration); LDH – lactate dehydrogenase. Arterial blood gas (ABG) parameters included the measurement of pH, partial pressure of carbon dioxide (pCO_2), and partial pressure of oxygen (pO_2).

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