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Case Report

Miliary brain tuberculomas and tuberculous meningitis presenting with stroke ☆

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ABSTRACT

Even though it is an uncommon presentation of tuberculosis, tuberculous meningitis is one of the most deadly manifestations. We report a case of a 6-year-old female who presented to the emergency room for left hemiparesis. Cerebral CT and MRI showed a right ischemic stroke with severe leptomeningitis in the medial cranial fossa. Numerous miliary tuberculomas were demonstrated, as well as a moderate hydrocephalus. Lumbar puncture revealed meningitis, and the mycobacterium tuberculosis polymerase chain reaction from CSF was positive. Pulmonary micronodules on chest CT were suggestive of tuberculosis. The clinical and radiological features, as well as the management approaches of this unusual disease complex, are addressed.

Introduction

Tuberculosis (TB), an infectious condition caused by Mycobacterium tuberculosis, remains a serious public health concern and ranks among the top 10 global causes of death.

In recent years, the global incidence of TB has continued to rise slowly, mainly because of the impaired immune system of AIDS (acquired immunodeficiency syndrome) patients in many developing countries and the rise of multidrug-resistant TB [1].

Even though it is an uncommon presentation of tuberculosis, tuberculous meningitis is one of the most deadly manifestations. In addition, cerebral infarction is an exacerbating factor in this context.

In people with tuberculous meningitis, cerebral infarctions are events whose severity and frequency cannot be underestimated. Their poor prognosis is partly the result of ill-defined management, combining anti-tuberculosis therapy and early corticosteroid administration [1,2].

Case report

We report the case of a 6-year-old female with no relevant past medical history. Immunizations were up to date, includ-

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ing BCG. Her parents reported night sweats and cough over the last month with a weight loss of 5 kg. Close contacts were not sick.

Two days before, her mother noticed weakness and clumsiness in her left hand and leg and behavioral changes consisting of disorientation and agitation. Her Glasgow Coma scale (GCS) score was 13/15 on physical examination. She presented with left hemiparesis with no other neurological deficits. She also showed tachypnea with subcostal retraction.

Cranial CT revealed a large right frontoparietal area of subacute hypointensity, suggesting an ischemic injury. MRI showed a right stroke involving frontal and parietal lobes, coincident with severe leptomeningitis in the medial cranial fossa impeding the flow of the medial cerebral artery. In addition, numerous supra- and infratentorial enhancing nodules were demonstrated, along with moderate hydrocephalus (Fig. 1). Chest CT scan showed miliary mottling tuberculosis (Fig. 2).

A lumbar puncture was performed, and cerebrospinal fluid (CSF) analysis documented normal protein and glucose with lymphocytic pleocytosis (25 cells/μL). Mycobacterium tuberculosis polymerase chain reaction from CFS was positive. The HIV test was negative.

According to the World Health Organization protocol, she received a standard 4-drug regimen for 2 months with corticosteroids followed by rifampicin and isoniazid for ten months. Ventricular drainage was unnecessary.

She progressed favorably and made a full recovery.

Discussion

Tuberculosis typically affects the lungs (pulmonary TB) but can also spread to other sites (extrapulmonary TB) through lymphatic or hematogenous dissemination. The central nervous system (CNS) is involved in 2-5% of cases of TB infection and up to 15% of AIDS-related TB cases. CNS tuberculosis is responsible for an elevated mortality rate and possible severe neurological complications and sequelae [1].

The common presentations of central nervous system TB are tuberculomas and meningitis [2]. Tuberculoma could be found in any portion of the intracranial space. They may be solitary or multiple. Tuberculoma on nonenhanced CT scans might be hyperdense, isodense, or mixed density. It may show a ring-shaped enhancement pattern or, more rarely, an ir-

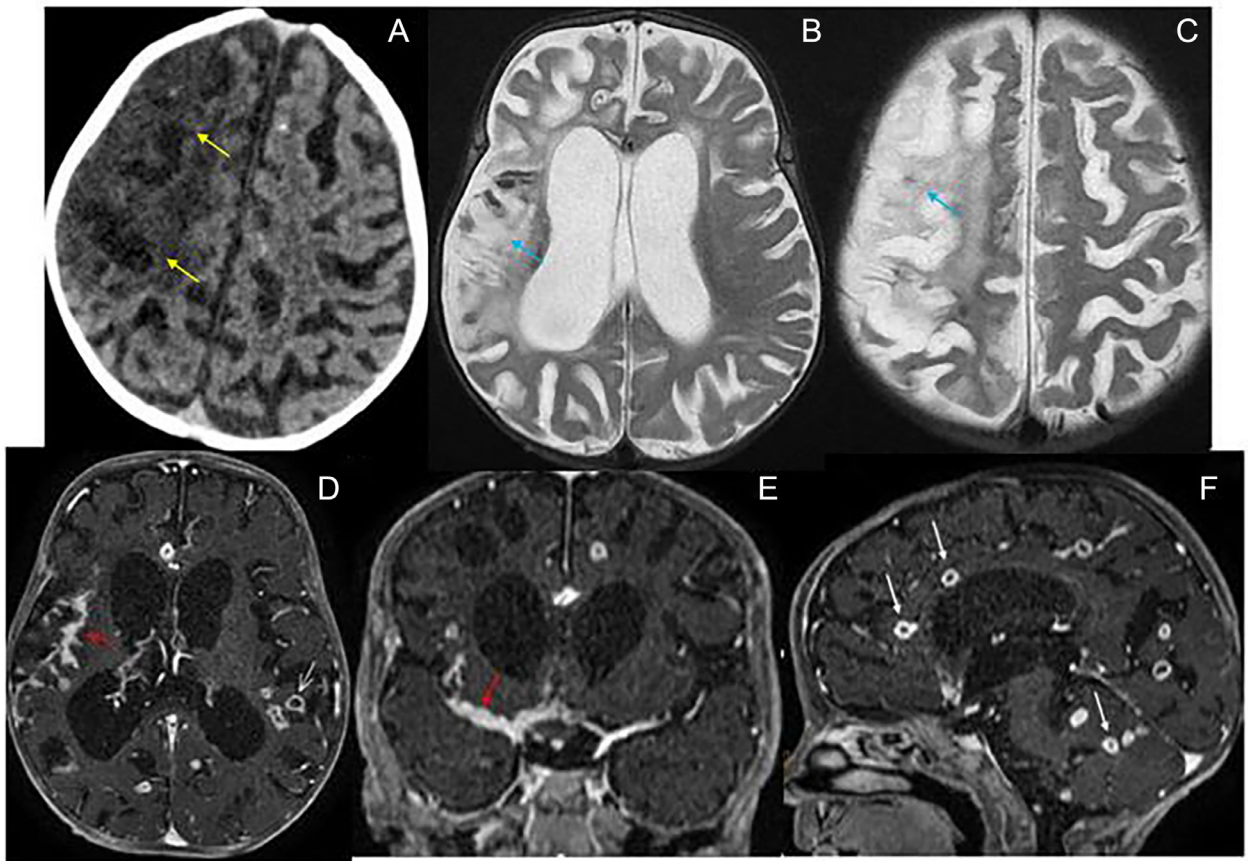

Fig. 1 – Radiological images of the central nervous system. Cranial CT (A) and MRI (B–F). (A) Loss of gray-white matter differentiation in the territory of the right middle cerebral artery (yellow arrow). (B, C) Right stroke involving frontal and parietal lobes (blue arrows) and Ventriculomegaly. (D, E) Severe leptomeningitis in medial cranial fossa occluding the flow of the medial cerebral artery (Red arrow). (F) Multiple nodular or ring-like tuberculomes with a miliary pattern (white arrows).

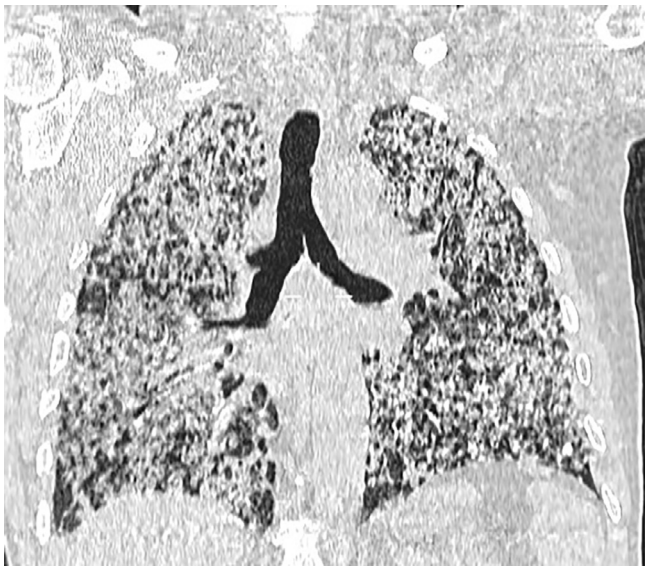

Fig. 2 – Chest computed tomography scan showing Miliary mottling consisting of little nodules uniformly sized and distributed. Abbreviations: TB, Tuberculosis; CNS, Central nervous system; GCS, Glasgow coma scale; CSF, Cerebrospinal fluid; AIDS, acquired immunodeficiency syndrome; BCG, Bacillus Calmette–Guérin vaccine; HIV, Human immunodeficiency virus; MRI, Magnetic resonance imaging; CT, Computed tomography.

regular or nodular area of inhomogeneous enhancement on contrast-enhanced CT. On T2-weighted or FLAIR images, non-enhanced MR scans reveal a mixed, primarily low signal intensity lesion with a central zone of high signal intensity and surrounding high signal intensity edema. Like contrast-enhanced CT, postcontrast MR images typically exhibit a pattern of ring-shaped enhancement [3,4]. Intracranial tuberculomas may rupture into the subarachnoid space, leading to the emergence of tuberculous meningitis [2].

During the early stages of the disease, non-contrast MRI studies usually show little or no evidence of any meningeal abnormality. However, with disease progression, swelling of the affected subarachnoid spaces occurs with associated mild shortening of T1 and T2 relaxation times compared with normal CSF. In addition, postcontrast T1W images show abnormal meningeal enhancement, especially in the basal cisterns.

Noncontrast MRI tests generally show little or no indication of any meningeal abnormalities in the early stages of the illness. Swelling of the afflicted subarachnoid areas develops as the disease progresses, accompanied by an average shortening of T1 and T2 relaxation durations in contrast to normal CSF. T1-weighted postcontrast pictures reveal unusual meningeal enhancement, particularly in the basal cisterns. The sites frequently implicated are the pontine cistern, the interpeduncular fossa, and the suprasellar and perimesencephalic cisterns. The sulci over the convexities and the Sylvian fissures are also involved. It is unusual to see cerebellar meningeal and tentorial involvement [3]. Other infective meningitis, inflammatory disorders like sarcoidosis, carcinomatous meningitis, or rheumatoid arthritis may have a similar pattern of meningeal enhancement [4].

The main complications of TB include cerebral stroke and hydrocephalus [1]. Hydrocephalus in TBM may be broadly classified into 2 categories: communicating hydrocephalus, which is widespread and is caused by an obstruction of the basal cisterns by inflammatory exudates, and obstructive hydrocephalus, which is less frequent and is caused by either an entrapment of a part of the ventricle by granulomatous ependymitis or a focal parenchymal lesion causing mass effect [3].

It has been reported that the incidence of stroke is about 13%- 57% in TB patients, which can cause poor clinical outcomes. Mortality is approximately 3 times higher in patients with TB who have had a stroke than in those who have not. Only 20% of these events are symptomatic, with the most common presentation being monoplegia or proportional hemiplegia [5].

Their main localizations are in the middle cerebral artery territory and its perforators and the thalamic perforating arteries at the level of the basal ganglia and the anterior cerebral areas. These territories, also called the "TB zone," correspond to the caudate nucleus, the lenticular nucleus, the anterolateral thalamus, and the knee of the internal capsule. Lesions are rarely found at the cortical or brainstem level.

Several mechanisms could then lead to cerebral infarction. First, the early events observed at diagnosis would result in vasospasm. At the same time, the lesions occurring later, sometimes after initiation of antituberculosis treatment, would be the consequence of an infiltration of the adventitia and then of intimal and adventitial cell proliferation with the formation of granulomas with caseous necrosis, leading to parietal lesions for which the term "cerebral vasculitis" is sometimes used.

Tuberculosis treatment is unfortunately not very effective in preventing cerebral infarction. In a cohort of children treated for neuromeningeal tuberculosis, 22% of new infarctions were noted after 1 month of treatment. Therefore, the use of high-dose corticosteroids is recommended for the treatment of neuromeningeal tuberculosis. Corticosteroid therapy reduces mortality at nine months by 30% by reducing the inflammatory reaction and the synthesis of cytokines by microglial cells. The introduction of early corticosteroid therapy would prevent the onset of infarction. However, it did not show any efficacy on morbidity or functional recovery.

The systematic prescription of aspirin at an antiaggregation dose is not yet recommended for preventing cerebral infarction complicating tuberculosis [6].

Conclusion

In conclusion, CNS involvement in tuberculosis is more frequent in pediatric patients, and earlier diagnosis and treatment could avoid a bad prognosis. A high index of suspicion is needed, especially in meningitis/meningoencephalitis and focal CNS symptoms. TB should be considered in infants with unexplained stroke [7]. Early diagnosis and timely treatment

are significantly critical in improving the prognosis of these patients [1].

Human and animal rights

The authors declare that the work described did not involve experimentation on patients, subjects, or animals.

Patient consent

The parent of the patient has been informed and has given their consent to the publication of this case report.

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