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Case Report

Severe Tuberculous Meningitis with Fatal Consequences in a Pediatric Patient: A Case Report

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Abstract:

Background: Tuberculous meningitis is a critical public health issue in underdeveloped nations due to its high morbidity and death. Tuberculous meningitis with non-specific symptoms of the central nervous system in children needs to be assessed with great vigilance as delay in diagnosis results in poor prognosis.

Case Report: We present a case of 20 months old female child with symptoms of sensory alteration, aberrant movements, and fever. Lumbar puncture findings indicated cell count of 1121/mm3, reduced glucose level, and elevated proteins. Ventriculomegaly with diffuse hypodenseimages in the frontal and paraventricular regions with areas of ischemia and hydrocephalus were evident in the MRI. Neuroinfection was indicated with images compatible with obstructive hydrocephalus of early evolution suggestive of tuberculous meningitis. The patient was started on 2HRZE/10 hr regimen. However, the patient was observed with significant neurological sequel and presented multiple comorbidities despite supportive measures.

Conclusion : Children are more susceptible to tuberculous meningitis than adults and result in death and disability, if not diagnosed early. Hence, the physicians must always be on the alert for tuberculous meningitis, especially in the areas where tuberculosis is endemic

Keywords: Tuberculous meningitis, obstructive hydrocephalus, motor deficit, panarteritis, thrombosis.

1. Introduction:

The most severe form of pediatric tuberculosis (TB), tuberculous meningitis (TM), has a significant fatality and morbidity rate [1]. Children, their families, and healthcare systems must deal with serious long-term implications of the neurological dysfunction caused by TBM, hence prevention and early diagnosis are crucial [2]. 12% of the estimated worldwide TB burden is accounted for by children under the age of 15 [3]. High risk groups for TB and TB-related mortality include people under the age of five and people living with HIV [2, 4]. This places TB as one of the top 10 causes of under-5 death. Disseminated TB, especially TBM, is worrisome for children under the age of two. TBM is devastating without treatment [1], and undetected cases or those diagnosed late probably contribute significantly to juvenile TB mortality worldwide. This case presentation intends to draw attention to the typical TBM presentation, imaging results, and diagnostic difficulties.

2. Case Presentation

A 20-month-old female child, who had previously been healthy, presented to the emergency room with a five-day history of persistent vomiting, a fever measuring 38.5, and brief clonic movements of the upper and lower extremities, followed by a period of lethargy without regaining her regular level of consciousness. Examining the mucous membranes revealed paleness, varying times of irritation and sluggishness, symptoms of dehydration, cervical lateralization to the right, stiffness in the neck, isochoric pupils, photoreactive, retroauricular adenopathies, and retained muscle tone. There were reflexes, ventilated lung fields, normal phonetic heart sounds, no peritoneal irritation in the belly, and limbs that were still mobile but had lost strength, scoring a 4/5 on the muscle strength scale. The preliminary findings indicated normal leukocytes, hypochromic microcytic anaemia, thrombocytosis, increased c-reactive protein, hyperglycemia, normal serum electrolytes, and a normal general urinalysis in the biometry. The diagnosis of neuroinfection was made given the symptoms of sensory alteration, aberrant movements, and

fever. During lumbar puncture, xanthochromic fluid with a cloudy appearance, a cell count of 1121/mm3, primarily monocytic, a glucose level of 26 mg/dl, and high proteins of 1181 g/dl were found. CSF was negative for AFB, CSF gram without microorganisms, and the CSF culture was negative for bacterial growth. Pandy's reaction was positive,

Using a standard skull tomography, it was evident that the patient had ventriculomegaly with the presence of diffuse hypodense images in the frontal and paraventricular regions, as well as areas of ischemia and hydrocephalus (Fig.1). Further investigations indicated non-reactive HIV test, negative toxoplasma, cytomegalovirus and rubella, immunoglobulin dosage and normal compliment. An assessment by the neurology department indicated a neuroinfection with images compatible with obstructive hydrocephalus of early evolution suggestive of TBM and MRI was requested.

In multidisciplinary criteria, pediatrics, neurology, and infectious diseases indicated a diagnosis of TBM based on epidemiological criteria and imaging. The 2HRZE/10 hr regimen was started with prednisolone at 2mg/kg/day for 4 weeks. During her hospital stay, the patient was observed with significant neurological sequel, received phenytoin as anticonvulsant support, presented multiple comorbidities despite supportive measures, including placement of ventricular peritoneal shunt valve and antireflux technique, with torpid evolution.

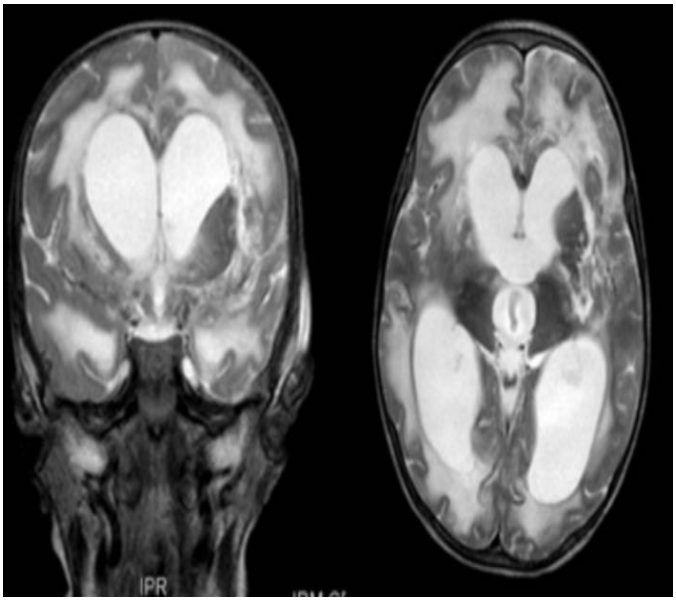

Figure 1: Skull tomography showing evidence of ventriculomegaly with the presence of diffuse hypodense images in the frontal and paraventricular region, with areas of ischemia and hydrocephalus.

3. Discussion

The most severe extrapulmonary form of Mycobacterium tuberculosis infection, TBM, is a critical public health issue in underdeveloped nations due to its high morbidity and death, despite the availability of contemporary treatments. Children with untreated extrapulmonary TB have a 1% to 2% chance of developing meningitis [1]. The risk factors that encourage progression include infancy, malnutrition, measles infection, HIV infection, immunosuppression, and chronic illness. Through hematogenous dissemination, the bacilli enter the central nervous system, form granulomas, and then combine to create Rich's caseous foci, which can rupture the subarachnoid space and cause meningitis [3]. Children are more susceptible than adults to developing TBM from primary TB. Stage III of the illness, a deep coma, and young age are the greatest risk factors for a bad prognosis, including fatality. Cerebrospinal fluid proteins above 100 mg/dl, hypertonia, delayed hydrocephalus care, the existence of strokes, a focal deficit at admission, cranial nerve palsy, and seizures are other factors that have been observed, though not consistently in most series [4]. Inflammatory exudates, one of the most important features in the pathogenesis of meningitis, involves the basal blood vessels of the brain and generates a panarteritis that results in occlusion and thrombosis of the blood vessels [3].

Contact history is important for the diagnosis of TB. Wu and colleagues suggested that household contact has a higher risk of severe TB, with a reported rate of 31% [5]. According to some researchers, the prognosis is associated with CT findings, particularly at the time of diagnosis; nevertheless, some secondary problems that arise after the diagnosis may have a detrimental impact on the prognosis. The most frequent secondary issue in TBM is hydrocephalus, which has a poor prognosis and is linked to persistent impairment [5]. The intracranial findings in our case agreed with previous research.

Mycobacteria found in CSF provide the basis for the conclusive diagnosis of TBM. In adults, the diagnostic yield of culture ranges from 37-87%, whereas in children, it is just 15-20% [6]. When the CSF volume is larger than 5 ml [7], the sample quality is good, the likelihood of isolating bacilli improves. In our case, acid resistant bacillus (ARB) was negative at the initial CSF evaluation, also ARB positivity could not be established in the CSF in serial lumbar punctures. This can be explained by the presence of small number of bacilli and/or technical deficiencies [7]. Due to the spinal canal obstruction, the CSF protein levels in TBM patients are elevated [6] and protein levels of >2g/l suggested a greater possibility of TBM. The patient in our case had CSF protein level of 11.81 g/l, thereby indicating a greater possibility of TBM. The reduced glucose levels (26mg/dl) in our case were similar to the findings of previous studies.

Clinically, the management of TBM typically entails anti-TB medication in addition to symptomatic and supportive care. In children with moderate to severe TBM, Schoeman et al [5] examined the impact of high dosages of predinsolone on intracranial pressure changes, imaging results, and clinical outcomes. It was shown that patients who received steroids had higher survival rates and improved mental abilities. It is debatable whether corticosteroids should be used as adjuvant therapy in TBM. According to the majority of the studies, TBM patients using steroids have better neurological and survival results [7].

4. Conclusion

To minimize death and disability, it is essential that TBM in children is diagnosed early. Children with TBM may have

vague symptoms, and the usual neck stiffness is frequently absent in the early stages of the disease. Hence, healthcare professionals must always be on the alert for TBM, especially in areas where TB is endemic. Therefore, the diagnosis is primarily clinical and is based on a combination of the clinical history, physical examinations, CSF characteristics (typically clear appearance, moderately raised white cell count, lymphocyte predominance, elevated protein level, and hypoglycorrhachia), and neuroimaging that showed basal meningeal enhancement, infarction, hydrocephalus, and/or tuberculomas.

Declarations

Patient's consent

Informed consent was obtained from the patient for the publication of this case.

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Authorship

All authors had access to the data and a role in writing this manuscript.

Declaration of competing interest

None

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