Residência RP Pediátrica
Publicação Oficial da Sociedade Brasileira de Pediatria
ISSN-Online: 2236-6814

Submitted on: 07/16/2023
Approved on: 09/03/2023

CASE REPORT

Neurotuberculosis in childhood: a case report

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Keywords:
Child,
Pediatrics,
Tuberculosis,
Central nervous system infections.

Abstract

Objective: To report the case of a pediatric patient diagnosed with neurotuberculosis, a rare manifestation of the disease, relating it to the literature. **Case report:** Male patient, 10 months old, started with chronic cough associated with fever, lymphadenopathy, in addition to right hemibody paresis and episodes of convulsive crises. In the anamnesis, there was evidence of home contact with a patient with pulmonary tuberculosis who had not been adequately treated. Tuberculin test was performed, with negative result. Cerebrospinal fluid analysis showed negative rapid test for tuberculosis, hypoglycorrhachia, increased proteins and cellularity at the expense of lymphocytes and negative bacterioscopy. The association of computed tomography and magnetic resonance imaging findings with clinical data, then, indicated the possibility of neurotuberculosis with complicated vasculitis and cerebral infarction. Diagnostic confirmation was only possible after a week of hospitalization, with the positive result of the rapid molecular test for *Mycobacterium tuberculosis* in a sample of gastric lavage. It was decided, then, to start the scheme rifampicin 15 mg/Kg, isoniazid 10 mg/Kg, pyrazinamide 35 mg/Kg and dexamethasone 0.3 mg/Kg. After 20 days, the patient was in good general condition and was discharged for outpatient follow-up. **Conclusion:** *Neurotuberculosis is a rare and challenging clinical presentation in the pediatrics practice. The fast diagnosis and the early treatment impact directly in the child morbimortality. Therefore, it must be considered as a differential diagnosis in various neurological presentations.*

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DOI: 10.25060/residpediatr-2024.v14n3-1135

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INTRODUCTION

Tuberculosis (TB) is an infectious disease of high incidence, morbidity, and mortality in its multidrug-resistant form.[1,2] The World Health Organization (WHO) estimated that 10.4 million new cases of the disease were diagnosed worldwide in 2016, 6.9% in children.[1]

The most common findings of TB in children are lung involvement and intrathoracic adenopathy, present in 60-80% of cases.[1,3] Pediatric patients have a greater tendency to present extrapulmonary involvement than adults.[1] The main symptoms are fatigue, loss of appetite, weight loss, sweating, and nocturnal fever.[1,4] Lymph node enlargement, cough, wheezing, dyspnea, hepatosplenomegaly, erythema nodosum, and arthralgia may also be present.[1]

Central nervous system (CNS) involvement can cause a wide array of clinical signs and symptoms, including vomiting, nuchal rigidity, hemiparesis, cranial nerve palsy, focal deficits, seizures, limb dystonia, and hydrocephalus.[2,5]

This article describes the case of an infant with central nervous system tuberculosis (CNS-TB), a rare, difficult-to-diagnose form of the disease. It examines the diagnostic particularities, evolution, and treatment, thus contributing to resolving similar cases.

METHOD

This descriptive observational study was designed to summarize and analyze the evidence in the literature on pediatric CNS-TB, focusing on case reports.

CASE REPORT

A 10-month-old male was admitted to the emergency department with a history of coughing and fever for 29 days, right-sided paresis, and seizures characterized by a vacant stare and movements of the face and right arm. The patient was previously healthy and had been hospitalized for wheezing. He received all vaccines recommended in the immunization schedule published by the Brazilian Ministry of Health. Seven months before admission, he was in contact with an individual with tuberculosis who had not been adequately treated.

On admission, he was hypoactive and febrile. Palpation of the neck revealed bilateral, mobile, painless lymph nodes of fibro-elastic consistency measuring 1-2 centimeters. On neurological examination, he presented central right facial paralysis on the right, with left conjugate gaze palsy, reduced right side movement, hyperreflexia (3+/4), and clonus on the right. The head circumference was in the 50th percentile for his age, and he had a pointy anterior fontanelle.

Empirical antibiotic therapy was started with oxacillin 200 mg/kg/day and ceftriaxone 100 mg/kg/day for suspicion of meningitis. Phenobarbital was added to control the seizures.

The purified protein derivative (PPD) skin test was negative. CSF was analyzed with PCR and the Mycobacterium tuberculosis rapid test, and both were negative. Hypoglycorrhachia (22 mg/dL), increased proteins (84 mg/dL), and cell counts (195/mm3, with 75% lymphocytes, 19% neutrophils, and 6% monocytes) were identified, and bacterioscopy was negative.

A chest computed tomography (CT) scan showed mediastinal lymph node enlargement with calcifications and necrotizing consolidation extending to the lingula. A head CT scan revealed meningeal enhancement in the basal cisterns, the Sylvian fissure on the left, and a slight dilation of the lateral ventricles. A head magnetic resonance imaging (MRI) scan showed areas of extensive ischemic infarction in the territory of the left middle cerebral artery, associated with leptomeningeal enhancement involving the basal cisterns, probably related to inflammation or infection (Figures 1 and 2).

The association of imaging and clinical findings indicated a possibility of CNS-TB with complicated vasculitis and cerebral infarction. A head magnetic resonance angiography (MRA) scan revealed irregular contours with a reduction in the caliber of the left carotid siphon associated with signs of occlusion of the proximal portions of the ipsilateral middle cerebral artery. These findings reinforced the idea of vasculitis due to the intense parietal contrast enhancement of these vessels. Ultrasound examination of the neck revealed multiple heterogeneous lymph nodes bilaterally with calcifications.

Laboratory diagnostic confirmation was only possible after one week of hospitalization, with a positive result from the rapid molecular test for Mycobacterium tuberculosis, sensitive to rifampicin, performed on a gastric lavage sample.

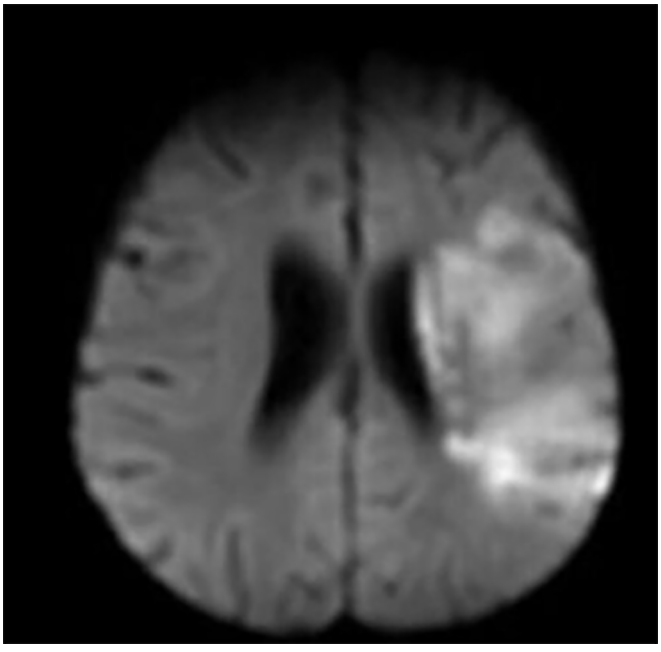


Figure 1. Head MRI scan. Extensive areas of restricted diffusion in the territory of the left middle cerebral artery, characterized by a high signal in the DWI images and a corresponding low signal in the ADC map, consistent with recent ischemic injury.

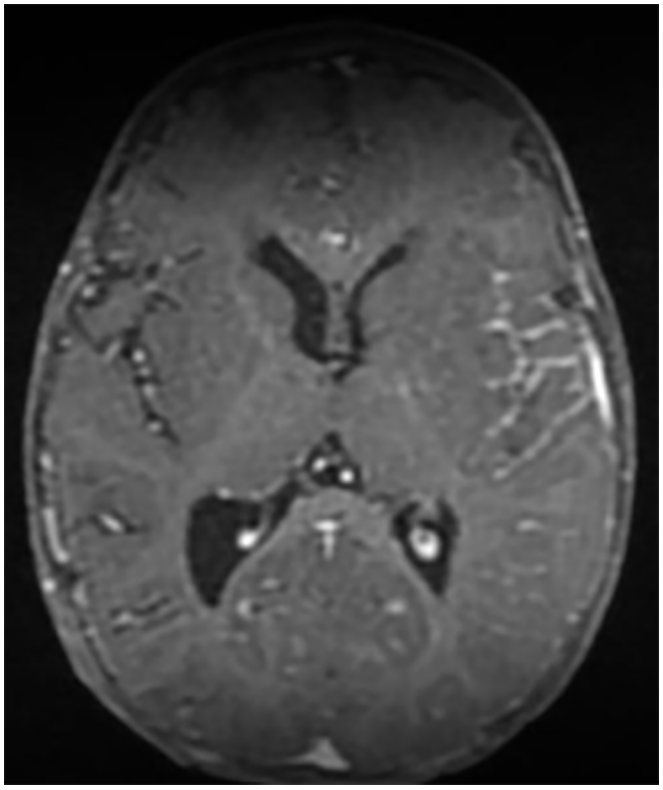


Figure 2. Head MRI scan. Multiple sites with leptomeningeal enhancement in the left frontal, parietal, and temporal regions and the ipsilateral basal cisterns are consistent with infection (meningitis), given the clinical context.

A biopsy was performed on a neck lymph node, which found necrotizing granulomatous lymphadenitis. A qualitative PCR test detected Mycobacterium tuberculosis.

The decision was then made to suspend antibiotics and start treatment for CNS-TB with rifampicin 15 mg/kg, isoniazid 10 mg/kg, pyrazinamide 35 mg/kg, and dexamethasone 0.3 mg/kg. After 20 days of in-hospital treatment, the patient was in good general condition, afebrile, and with persistent right hemiparesis. She was discharged and referred to outpatient follow-up and homecare. The family was referred to a hospital in their hometown.

DISCUSSION

Children's immature immune system makes them more likely to develop severe diseases, including CNS-TB.[1] Approximately 5-10% of TB cases involve the CNS.[3,4] Of these, 70-80% consist of tuberculous meningitis, with a mortality rate of 5-43% and permanent neurological impairment in 13-17% of cases.[3] Sequelae are more common in children and occur in 14-52% of CNS-TB cases.[6,7]

Few reports have described a presentation similar to our patient's. Perez-Alvarez et al. reported a case of CNS-TB in a 5-year-old with signs and symptoms similar to the ones described in this case, including cerebral infarction seen on head MRI scans and hemiparesis on physical examination.[8,9] Another case involving a 9-month-old described focal and generalized seizures secondary to CNS involvement by TB.[7]

The disease is transmitted by inhaling aerosols from the coughing, speaking, or sneezing of people with active TB.[10] Up to 90% of people do not become ill after primary infection and develop the latent form of the disease.[11] Progression to active TB relies on the interaction between the pathogen and the host's immune system.[11]

The main international consensus published by the WHO on childhood TB indicates that diagnosis requires taking the patient's history, with particular attention to situations of contact with individuals with TB, and verification of symptoms consistent with the disease.[1,2] Thorough clinical examination, chest X-rays, confirmatory workup, specific investigation of the organ involved, and HIV testing are also advised.[1,3,11]

Microbiological diagnosis occurs in only 15-50% of pediatric cases. It is difficult to obtain good samples, particularly in children under seven.[1,4] In addition, most cases are not bacilliferous in this age group.[3] Semi-invasive techniques, such as gastric aspiration or sputum induction, may be necessary to obtain a sample, as in our patient's case.[1] The tests used are the microscopic analysis, rapid molecular test, and culture, the gold standard due to its high sensitivity and specificity.[1,4]

Imaging aids diagnosis and must be performed based on clinical suspicion.[3,11] Chest X-rays and CT scans are particularly valuable in immunosuppressed patients.[5,11] The most common findings in tuberculous meningitis are increased cell count with a predominance of lymphocytes, high protein, and low glucose.[3,10] The CSF may be normal in the first 48 hours, and a predominance of polymorphonuclear cells may be seen in the first ten days.[3] The disease detection rate by CSF bacilloscopy is low. Tuberculous meningitis is a paucibacillary disease. Therefore, a negative result does not rule out the presence of disease.[3,13]

The most common findings in imaging tests, such as CT and head MRI, are hydrocephalus, basal meningeal thickening, and cerebral parenchymal infarctions.[5,6,12,13]

Treatment is divided into two phases. The intensive phase aims to decrease the bacilli population and contagion rapidly. The continuation phase aims to eliminate latent or persistent bacilli and decrease the chances of recurrence.[11] Treatment is usually delivered in an outpatient setting, preferably with the patient under direct observation.[3,11] Hospitalization is necessary in cases of tuberculous meningitis, drug intolerance, complications, or social vulnerability.[10] The basic treatment regimen for all forms, except tuberculous meningitis and osteoarticular tuberculosis, lasts six months.[1,3,11]

In the first two months of treatment (intensive phase), the Brazilian Ministry of Health recommends a protocol with rifampicin (R), isoniazid (H), pyrazinamide (Z), and ethambutol (E), followed by another four months of rifampicin and isoniazid (continuation phase).[7,11] Tuberculous meningitis

and osteoarticular tuberculosis require longer treatment, with a total duration of 12 months, with the RHZE scheme for two months, followed by the RH scheme for another ten months.[1,10] In these cases, the American Academy of Pediatrics recommends initial treatment with RHZ, combined with an aminoglycoside or ethionamide for two months instead of ethambutol, and then 7-10 months of RH.[1] In patients with tuberculous meningitis, adding corticosteroids to the regimen for at least four weeks demonstrated a higher survival rate and better intellectual performance.[1,3,13]

CONCLUSION

CNS-TB is a rare and challenging condition in pediatric practice. Rapid diagnosis and early management substantially impact morbidity and mortality in children. CNS-TB must be considered in the differential diagnosis of various neurological conditions.

The authors have no conflicts of interest to declare. This study was produced without funding.

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