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Case Report

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Neurotuberculosis in a paediatric patient: a case report

Neurotuberculose em paciente pediátrico: relato de caso

Neurotuberculosis en un paciente pediátrico: reporte de caso

Isadora Durieux Lopes Destri[1,2]
Mariá Lessa Silva[1,3]
Isabela Flebbe Strapazzon[1,4]
Lis de Souza Martinelli[1,5]
Priscyla Rocha Silva[6,7]
Júlia Souza Vescovi[6,8]
Marcos Paulo Guchert[9,10]
Sonia Maria de Faria[9,11]
Emanuela Rocha Carvalho[12]

[1] Acadêmica Universidade Federal de Santa Catarina, Medicina – Florianópolis-SC, Brazil.
[2] **ORCID:** https://orcid.org/0000-0001-9373-5484
[3] **ORCID:** **https://orcid.org/0000-0002-3997-2734**
[4] **ORCID:** https://orcid.org/0009-0004-0241-8213
[5] **ORCID:** https://orcid.org/0009-0006-7329-7679
[6] Hospital Universitário Professor Polydoro Ernani de São Thiago Universidade Federal de Santa Catarina, Residência Radiologia – Florianópolis-SC, Brazil.
[7] **ORCID**: https://orcid.org/0000-0001-6909-5026
[8] **ORCID**: https://orcid.org/0000-0002-6253-2294
[9] Hospital Infantil Joana de Gusmão, Infectologista Pediatra – Florianópolis-SC, Brazil.
[10] **ORCID**: https://orcid.org/0000-0002-5913-7381
[11] **ORCID**: https://orcid.org/0000-0001-5866-8386
[12] Hospital Infantil Joana de Gusmão, Infectologista Pediatra – Florianópolis-SC, Brasil; Docente Universidade Federal de Santa Catarina, Departamento Pediatria – Florianópolis-SC, Brazil; Universidade Federal de Santa Catarina, Doutoranda na Pós-Graduação em Ciências Médicas – Florianópolis-SC, Brazil. **ORCID**: https://orcid.org/0000-0002-2193-0528

Corresponding author:
Emanuela Rocha Carvalho[12]
E-mail: emanuela.carvalho@ufsc.br

ABSTRACT

Introduction: Central nervous system tuberculosis is a severe form of extrapulmonary involvement, with high risk of death and other unfavorable outcomes, particularly in children. Health professionals must be prepared to recognize symptoms and maintain a high degree of suspicion. **Objective:** To report a case of a pediatric patient with central nervous system tuberculosis, documenting clinical manifestations, complementary tests, diagnosis, treatment, and main outcomes. **Case description:** A previously healthy 10-year-old male presented with a two-month history of right parieto-occipital headache, associated with somnolence, blurred vision, nausea, vomiting, fever, and generalized tonic-clonic seizure. Neuroimaging revealed multiple ring-enhancing lesions in the right cerebellar hemisphere, associated with vasogenic edema and mass effect. Cerebrospinal fluid analysis showed 85 cells/mm³ with lymphocytic predominance, glucose 31 mg/dL, protein 120 mg/dL, and a rapid molecular test detecting traces of M. tuberculosis DNA. Treatment was initiated with rifampicin, isoniazid, pyrazinamide, and ethambutol, along with corticosteroid therapy. The patient showed progressive clinical improvement and maintained adherence throughout the treatment. **Discussion:** The prognosis of central nervous system tuberculosis is generally unfavorable, with high mortality and permanent sequelae rates. However, with appropriate treatment, complete clinical recovery may occur. This highlights the importance of surveillance for central nervous system tuberculosis in children, particularly in regions with high tuberculosis prevalence, in order to reduce diagnostic delays and improve patient outcomes.
Keywords: Central Nervous System Tuberculosis. Extrapulmonary Tuberculosis. Early Diagnosis. Child.

RESUMO

Introdução: A tuberculose do sistema nervoso central é uma forma grave de acometimento extrapulmonar, com alto potencial de evoluir para óbito e desfechos desfavoráveis, sobretudo em crianças. Profissionais da saúde devem estar aptos a reconhecer sintomas e manter alto grau de suspeição. **Objetivo:** Relatar caso de paciente pediátrico com tuberculose do sistema nervoso central, documentando manifestações clínicas, exames complementares, diagnóstico, manejo e desfechos principais. **Descrição do caso:** Paciente masculino, 10 anos, previamente hígido, com história de cefaleia parieto-occipital direita há 2 meses, associado a sonolência, turvamento visual, náuseas, vômitos, febre e episódio convulsivo tônico-clônico generalizado. Durante a investigação, apresentou neuroimagem de múltiplas lesões com realce anelar no hemisfério cerebelar direito, associadas a edema vasogênico com efeito de massa, e exame de líquor apresentando celularidade de 85 células/mm3 com predomínio linfocítico, glicose 31 mg/dL, proteínas 120 mg/dL, além de teste rápido molecular com traços de DNA de M. tuberculosis. Iniciou-se esquema com rifampicina, isoniazida, pirazinamida e etambutol e corticoide. O paciente evoluiu com melhora progressiva do quadro e manteve adesão durante todo o tratamento. **Discussão:** O prognóstico de casos de tuberculose de sistema nervoso central tende a ser desfavorável, com altas taxas de mortalidade e sequelas permanentes. No entanto, com tratamento adequado, pode-se evoluir com melhora clínica completa, de modo que se reforça a importância da vigilância para tuberculose do sistema nervoso central em crianças, especialmente em regiões com alta prevalência de tuberculose, a fim de reduzir atrasos diagnósticos e melhorar o prognóstico dos pacientes.
Palavras-chave: Tuberculose do Sistema Nervoso Central. Tuberculose Extrapulmonar. Diagnóstico Precoce. Criança.

RESUMEN

Introducción: La tuberculosis del sistema nervioso central es una forma grave de afectación extrapulmonar, con un alto potencial de muerte y pronóstico desfavorable, especialmente en niños. Los profesionales sanitarios deben ser capaces de reconocer los síntomas y mantener un alto grado de sospecha. **Objetivo:** Informar de un caso pediátrico con tuberculosis del sistema nervioso central, documentando las manifestaciones clínicas, las exploraciones complementarias, el diagnóstico, el tratamiento y los principales resultados. **Descripción del caso:** Un paciente masculino de 10 años, previamente sano, presentó antecedentes de cefalea parietooccipital derecha de 2 meses de evolución, asociada a somnolencia, visión borrosa, náuseas, vómitos, fiebre y un episodio convulsivo tónico-clónico generalizado. Durante la investigación, las neuroimágenes mostraron múltiples lesiones con realce en anillo en el hemisferio cerebeloso derecho, asociadas a edema vasogénico con efecto de masa. El análisis de líquido cefalorraquídeo mostró una celularidad de 85 células/mm³ con predominio linfocítico, glucosa 31 mg/dL, proteínas 120 mg/dL, además de una prueba molecular rápida con trazas de ADN de M. tuberculosis. Se inició un régimen con rifampicina, isoniazida, pirazinamida, etambutol y corticosteroides. El paciente presentó mejoría progresiva y mantuvo la adherencia al tratamiento. **Discusión:** El pronóstico de la tuberculosis del sistema nervioso central tiende a ser desfavorable, con altas tasas de mortalidad y secuelas permanentes. Sin embargo, con un tratamiento adecuado, es posible una mejoría clínica completa, lo que refuerza la importancia de la vigilancia de la tuberculosis del sistema nervioso central en niños, especialmente en regiones con alta prevalencia de tuberculosis, para reducir los retrasos diagnósticos y mejorar el pronóstico de los pacientes.
Palabras clave: Tuberculosis del sistema nervioso central. Tuberculosis extrapulmonar. Diagnóstico precoz. Niños.

INTRODUCTION

Even today, tuberculosis (TB) represents a significant global public health problem, being the tenth leading cause of death worldwide and the leading cause of death from a single infectious agent. Deaths in the pediatric population account for approximately 15% of the total.[1]

Mortality rates from untreated TB can reach 14.9% in children aged 5 to 14 years, and 43.6% in children under 5 years of age. Despite its severity, many cases of childhood TB are underreported due to diagnostic difficulties. With timely diagnosis and treatment, mortality can be reduced to as low as 0.9%.[2,3] Although pulmonary disease is the most common form of presentation, the pediatric population may present with extrapulmonary or disseminated disease in about 20-30% of cases.[4]

Central nervous system tuberculosis (CNS TB) is a less frequent, but serious, form of extrapulmonary involvement, with high mortality rates and neurological sequelae. It occurs in up to 5 to 10% of cases, with children under 5 years of age at higher risk.[4-6] Its often nonspecific symptomatology contributes to diagnostic and therapeutic delays, leading to a high risk of death and other unfavorable outcomes.[6]

Thus, raising awareness among healthcare professionals to recognize possible signs and symptoms of CNS TB is essential to improve the prognosis of patients affected by the disease. Therefore, this article aims to describe a case of CNS TB in a pediatric patient, documenting clinical manifestations, complementary examinations, diagnosis, management, and main outcomes.

CASE DESCRIPTION

This is a report of a 10-year-old male patient, initially referred from a hospital in the interior of the state for evaluation by the institution's oncology service in September 2022, due to suspected posterior fossa (PF) brain tumor. In his personal medical history, he had an up-to-date vaccination schedule for his age group according to the National Immunization Program and no associated comorbidities.

During the two months preceding the referral, the patient sought medical attention several times for daily, intense headaches in the right parieto-occipital region, associated with general malaise, prostration, drowsiness, blurred vision, loss of appetite, and vomiting preceded by nausea. He did not respond to simple analgesia. He also presented with daily afternoon and night fever, associated with intense sweating. Eventually, he was referred to a tertiary pediatric hospital in the interior of the state due to signs of intracranial hypertension (ICH) observed on cranial computed tomography (CT) scan. The image showed dilation of the ventricular system associated with hypoattenuation of the periventricular cerebral parenchyma, suggesting hydrocephalus with hypertensive signs, and apparent effacement of cerebral sulci, suggesting edema. For this reason, during hospitalization, surgery was performed to insert an external ventricular drain (EVD), later transformed into a ventriculoperitoneal shunt (VPS). The patient remained hospitalized in the Intensive Care Unit (ICU) for 5 days after the procedure.

The possibility of a PF brain tumor was raised by the originating hospital, and the patient was referred to the oncology service at the tertiary hospital in Florianópolis. In the week preceding the transfer, the patient also presented with a tonic-clonic seizure, lasting approximately five minutes, and began with transient episodes of left hemiparesthesia and asymmetry of facial expression.

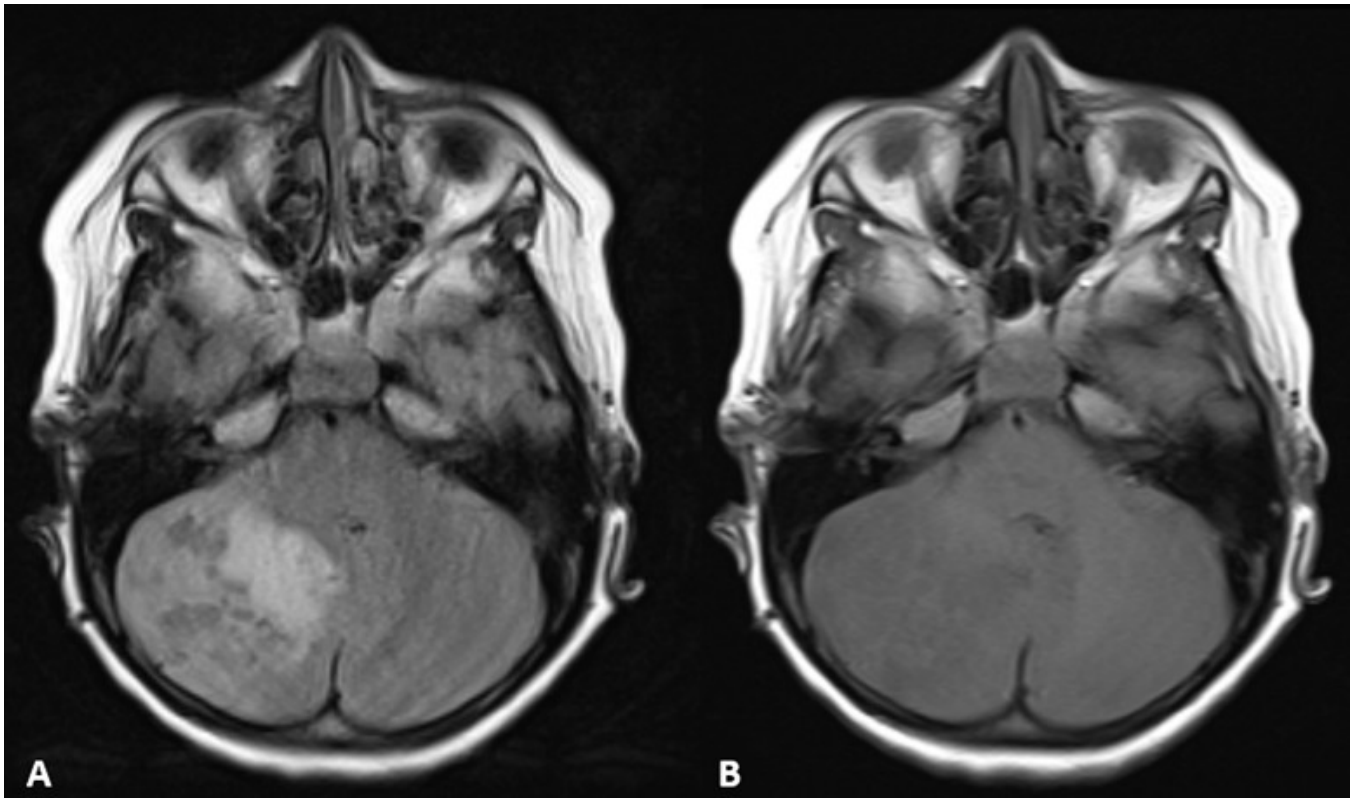
At the time of admission to the referral center, the patient presented on physical examination in good general condition and with a good overall impression, communicative, slightly pale (+/4) and slightly tachypneic. Cardiac auscultation was unremarkable; pulmonary auscultation revealed reduced vesicular murmurs on the left with fine crackles at the base. Abdomen diffusely painful on deep palpation without other abnormalities. Extremities well perfused, pulses strong and symmetrical, and capillary refill time of 3 seconds, lower limbs (LL) with cold and symmetrical edema up to the ankles, calves free. On neurological examination, facial expression was asymmetrical and

strength was symmetrically reduced in the LL, without gait impairment.

The patient was admitted for investigation, this being day 1 (D1) of hospital admission. Laboratory tests were requested, which revealed microcytic and hypochromic anemia (hemoglobin 10.1 g/dL; MCV 72 fL; MCH 25 pg); elevated inflammatory markers (CRP 8.9 mg/L; ESR 20 mm/h); hyponatremia and hypokalemia (Na 129 mEq/L; K 3.0 mEq/L); alterations in the coagulation profile (PT 17.09 seconds; INR 1.36; aPTT 39 seconds); slightly altered liver function (AST 66 IU/L; ALT 43 IU/L). The other parameters were unchanged.

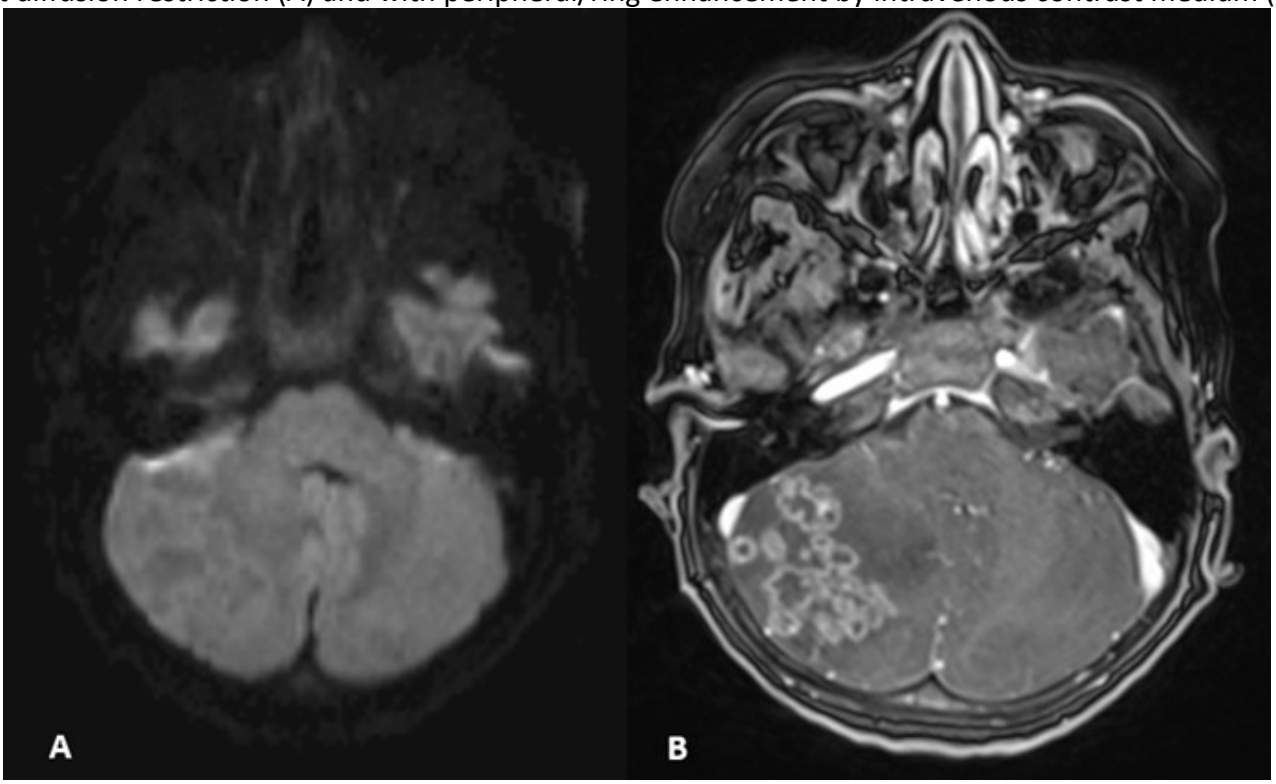
In addition to laboratory follow-up, on D5, a neuroaxial magnetic resonance imaging (MRI) scan was performed (Figures 1, 2, and 3), which showed multiple cystic lesions with ring enhancement, intra-axial and supra- and infratentorial leptomeningeal, in greater number and of larger dimensions in the right cerebellar hemisphere, causing vasogenic edema in the adjacent brain parenchyma, with consequent local mass effect, in addition to occipital and frontal (subcortical) lesions of a similar pattern and without diffusion restriction. No spinal cord involvement was evident. Based on the findings, the hypotheses of leptomeningeal neuroglial tumor or inflammatory/infectious disease, mainly granulomatous, were raised.

Figure 1 – Brain magnetic resonance imaging. Axial images, T2-weighted FLAIR (A) and T1-weighted (B), showing multiple intra-axial cystic lesions in the right cerebellar hemisphere, with vasogenic edema in the adjacent brain parenchyma, resulting in a local mass effect



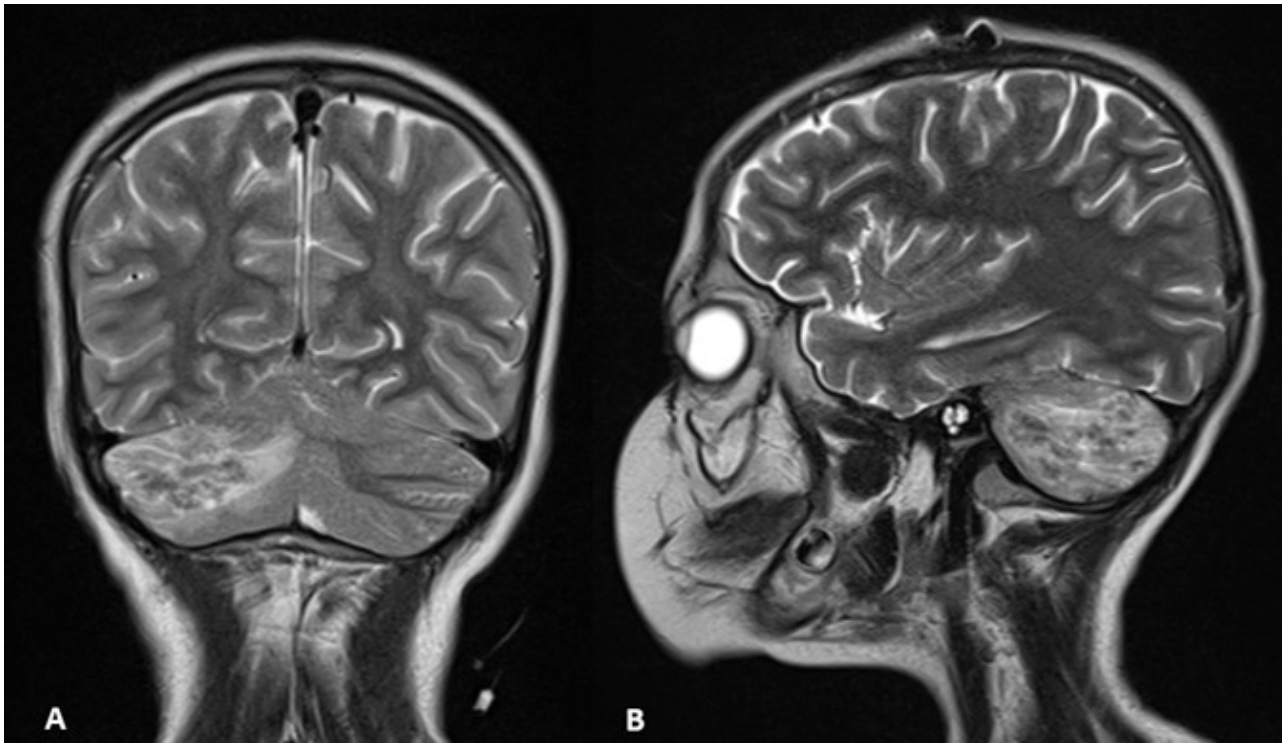
Source: medical records.

Figure 2 – Brain magnetic resonance imaging. Axial, diffusion-weighted (A) and post-contrast T1-weighted (B) images showing multiple intra-axial cystic lesions in the right cerebellar hemisphere, without diffusion restriction (A) and with peripheral/ring enhancement by intravenous contrast medium (B)



Source: medical records.

Figure 3 - Brain magnetic resonance imaging. Coronal (A) and sagittal (B) T2-weighted images showing multiple intra-axial and leptomeningeal cystic lesions in the cerebellar hemispheres

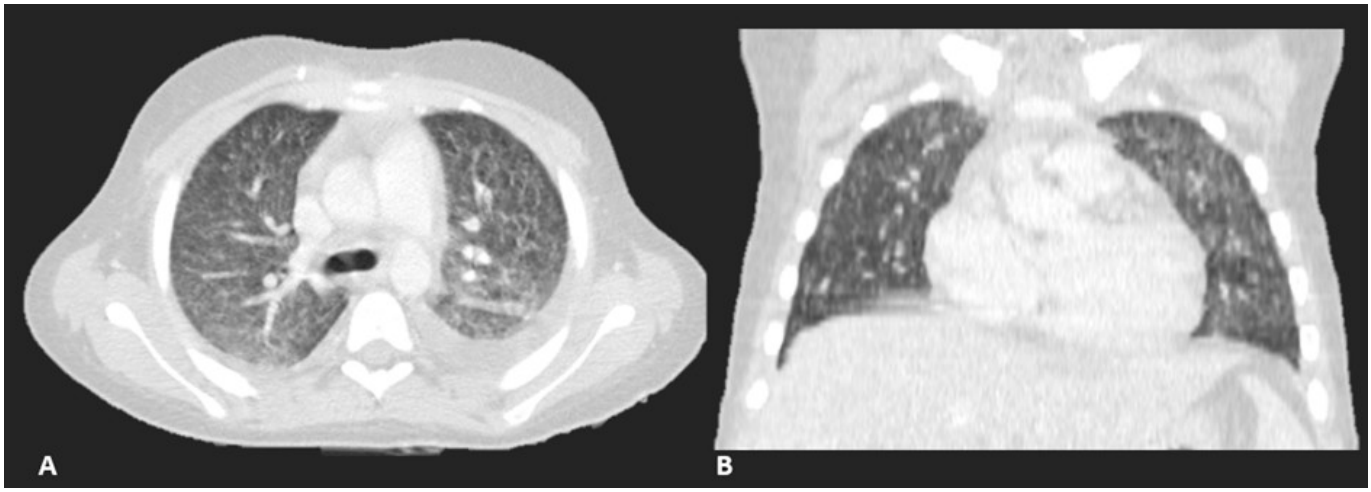


Source: medical records.

Considering the images, TB was suggested as the main etiological diagnosis. Since it was likely non-neoplastic etiology, the patient was transferred to the care of the neurology and infectious disease teams.

For diagnostic clarification purposes, on D6, a chest CT scan was requested (Figure 4), which showed findings suggestive of TB.

Figure 4 - Chest CT scan, axial (A) and coronal (B) views, lung window, demonstrates diffuse micronodules in both lungs. Ground-glass opacities are also noted in the lung bases and alveolar consolidation in the left lung base. Bilateral pleural effusion is also present.



Source: medical records.

On D8, cerebrospinal fluid (CSF) was collected for analysis, with the following results: nucleated cells 85/mm3 with a lymphocytic predominance of 62%; red blood cells 5/mm3; glucose 31 mg/dL; protein 120 mg/dL; negative acid-fast bacilli (AFB); detection of traces of *Mycobacterium tuberculosis* DNA through a rapid molecular test (GeneXpert® MTB/RIF Ultra); negative cultures for fungi and *Cryptococcus*. Pleural fluid was also evaluated, with the following results: glucose 85 mg/dL; nucleated cells 230/mm3 (lymphocytes 67%; neutrophils 30%; basophils 1%); protein 2.0 g/dL (albumin 1.0 g/dL; globulin 1.0 g/dL); red blood cells 65/mm3; negative AFB. TRM for *Mycobacterium tuberculosis* was not detected in pleural fluid or pleural biopsies. The HIV serology result was non-reactive.

Thus, disseminated TB with CNS involvement was diagnosed, and on D10, a standardized regimen recommended by the Ministry of Health (MH) was initiated, with RHZE (rifampicin, isoniazid, pyrazinamide, and ethambutol) to be maintained for 2 months, followed by 10 months of RH (rifampicin and isoniazid), totaling 12 months of treatment. Intravenous corticosteroids were also initiated as per MH guidelines for severe CNS TB, later transitioned to oral therapy.

After the introduction of medication, the patient showed progressive improvement in symptoms, remaining afebrile. There was improvement in neurological changes, with only blurred vision remaining, in addition to improvement in laboratory parameters, with only anemia remaining.

As complications, he presented with tonic-clonic seizures, requiring management with anticonvulsant medication (phenytoin), and thrombosis of the right external iliac vein, managed with warfarin. He was discharged from the hospital 16 days after starting treatment with RHZE. At the time of discharge, he was using phenytoin 100mg every 12 hours, warfarin 8.5mg/day, in addition to RHZE, and received referrals to hematology, neurology, and neurosurgery services for outpatient follow-up.

The patient used warfarin for 9 months and phenytoin for 8 months, with subsequent discontinuation without recurrence of thromboembolic events or seizures, and was discharged from the hematology and neurology outpatient clinics. In the latest neurological assessment, the patient presented preserved visual field and oculomotor function, without nystagmus, normal facial expression, no dysmetria, and no meningeal signs. Strength was preserved in the lower limbs and upper limbs, with an atypical gait. However, he presents inattentive and anxious behavior, sometimes heteroaggressive, and has started follow-up with a psychiatrist and psychologist.

The patient was discharged from the infectious disease outpatient clinic after 12 months of treatment with excellent adherence to RHZE, showing good tolerance to the medication. He showed complete improvement of symptoms, remaining without neurological or pulmonary alterations.

DISCUSSION

CNS TB represents a severe form of extrapulmonary TB, with high mortality rates and neurological sequelae, such as motor deficits and cognitive disorders. It shows pleomorphic clinical pictures, and early diagnosis is directly related to the prevention of negative outcomes by providing timely treatment. Thus, high clinical suspicion is fundamental, especially in the pediatric population, in which 5 to 10% of TB cases may present with CNS involvement.[7]

Among the main symptoms of CNS TB are headache, vomiting, and altered mental status. Hydrocephalus, intracranial hypertension, and seizures may also be found,[6] in addition to symptoms compatible with cranial nerve involvement – mainly oculomotor, optic, and abducens nerves – such as paralysis and focal deficits related to the functionality and topography of each nerve.[8] In the case described, all these clinical findings were present, in addition to afternoon fever and night sweats, symptoms highly suggestive of TB. Given this clinical picture, it is imperative that the diagnosis of TB be considered, especially given its high prevalence and importance to public health in Brazil.[5] In the case described, a delay in clinical suspicion was observed, with oncological diagnoses considered rare initially being considered. Diagnostic delay in similar cases represents a failure that can compromise the prognosis.[9]

Regarding laboratory investigation, cerebrospinal fluid analysis stands out, which, when suggestive of CNS TB, shows pleocytosis with lymphocytic predominance, a slight increase in protein levels, and low glucose levels, a pattern compatible with that presented in the clinical case. In addition, because it contains few bacilli, it is expected that it will be difficult to observe *M. tuberculosis* in the CSF by direct method, and molecular techniques should be used to detect genetic material, which have good accuracy for detecting the bacillus in the CSF. In the reported case, the nucleic acid amplification technique was performed with the GeneXpert® MTB/RIF Ultra test. Despite the still nascent literature, studies indicate that this molecular methodology performs adequately for the diagnosis of extrapulmonary TB in paucibacillary tissues – such as CNS TB – and provides information on rifampicin resistance, contributing to better therapeutic management.[10]

In addition to cerebrospinal fluid alterations, there may be other laboratory changes, the most frequent being: anemia, leukocytosis, elevation of inflammatory markers and hydroelectrolytic disorders, with emphasis on hyponatremia – usually related to aggravating conditions such as salt-wasting brain syndrome and syndrome of inappropriate antidiuretic hormone secretion – and hypocalcemia.[11,12] These findings are not very sensitive or specific for TB and CNS TB.[13]

With respect to the radiological findings of CNS TB, the most common are hydrocephalus, basal meningeal thickening and cerebral infarcts. Parenchymal involvement, manifested by the presence of tuberculomas and brain abscesses, usually results from hematogenous dissemination, justifying the fact that they are frequently found in association with other sites of TB infection.[6] The radiological characteristics of tuberculomas often mimic those of other infectious and non-infectious conditions, such as neurocysticercosis, metastasis, CNS lymphoma, neurotoxoplasmosis, tumors, and pyogenic abscess. Therefore, other diagnostic

methods besides CT – such as MRI – should be used for better diagnostic clarification.[14]

In cases of parenchymal involvement, tuberculomas are the most frequent lesions, and occur due to the coalescence of microgranulomas.[13] In children, the infratentorial regions are the most affected, especially the PF.6 In non-contrast CTs, tuberculomas may present as iso- or hypodense masses, while contrast-enhanced CTs and MRIs demonstrate solid lesions with ring enhancement. The most frequently described neuroimaging findings are like those found in the reported case.[13] The patient also presented signs of non-communicating hydrocephalus, a relatively frequent form of presentation of CNS TB that results from obstructions and mass effect caused by the presence of the tuberculoma.[13]

Because it is a severe form of extrapulmonary involvement, which is associated with high morbidity and mortality and a risk of permanent neurological sequelae, it is imperative that, upon suspicion of a diagnosis of CNS TB, treatment be promptly initiated.[6,15] The Ministry of Health recommends treatment for 12 months, using the RHZE regimen for the first 2 months, followed by 10 months of the RH regimen (2RHZE/10RH regimen).[15] In addition, the use of systemic corticosteroid therapy is strongly recommended, as it has been shown to reduce morbidity and mortality rates and disease recurrence, with the benefit being greater the higher the severity of the case. The importance of adequate treatment with RHZE at the time of implementation of corticosteroid therapy is emphasized, to mitigate the risks of immunosuppression and worsening of the condition.[15]

The prognosis for CNS TB cases tends to be unfavorable, with high mortality rates and permanent neurological sequelae. In children, the main factors associated with a worse prognosis are young age, lower socioeconomic levels, presence of altered mental status and level of consciousness, abnormal postures, hydrocephalus, and basal exudates.[8] However, with appropriate treatment, it is possible to progress to clinical improvement and complete remission of neurological symptoms, as observed in this patient.

Therefore, the importance of surveillance for CNS TB in children is reinforced, especially in the presence of warning signs and in regions with a high prevalence of TB, such as Brazil. Suspicion of CNS TB should be considered in children with neurological symptoms of subacute and protracted evolution, especially when accompanied by systemic manifestations and nonspecific findings in complementary examinations. As observed in this case, differential diagnosis is fundamental, although it may result in delays in diagnostic definition and untimely interventions. Raising awareness among health professionals is essential to reduce delays in diagnosis, enabling the implementation of early treatment and ensuring a better prognosis for patients affected by the disease.

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Scientific Editor:
Fernanda Pinto Mariz
ORCID: https://orcid.org/0000-0002-6981-2352

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E-mail: secretaria@soperj.org.br

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IDL Destri: data collection, conceptualization, investigation, writing - preparation of the original manuscript, writing - revision and editing.

ML Silva: data collection, conceptualization, investigation, methodology, writing - preparation of the original manuscript, writing - revision and editing.

IF Strapazzon: data collection, conceptualization, investigation, methodology, writing - preparation of the original manuscript, writing - revision and editing.

LS Martinelli: data collection, conceptualization, investigation, writing - preparation of the original manuscript, writing - revision and editing.

PR Silva: writing - revision and editing.

JS Vescovi: data collection.

MP Guchert: writing - revision and editing, visualization.

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ER Carvalho: statistical analysis, project management, investigation, supervision.