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Case presentation

Neurobrucellosis in a pediatric patient. Case report

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ABSTRACT

Brucellosis, a zoonosis with a diverse clinical presentation, can lead to severe complications such as neurobrucellosis. We describe the case of a 4-year-old girl with fever, conjugate gaze palsy, and rigidity, symptoms initially interpreted as seizures. Persistent fever, abdominal pain, and a history of consuming unpasteurized dairy products suggested neurobrucellosis. Cerebrospinal fluid analysis showed findings consistent with acute meningoencephalitis, with subsequent serological confirmation. Specific treatment with rifampin and sulfaprim led to progressive clinical improvement, with resolution of fever and neurological recovery. This case highlights the nonspecific initial presentation, the key value of the epidemiological history, and the need for a multidisciplinary approach for the diagnosis and management of this severe complication.



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Introduction

Brucellosis, also called Malta fever or undulant fever, is a zoonotic disease caused by bacteria of the genus *Brucella*, which is transmitted to humans through contact with infected animals, consumption of unpasteurized dairy products, or inhalation. (1,2) Its initial clinical presentation includes intermittent fever, sweating, myalgia, and general malaise, but it can also present with mild or gastrointestinal symptoms that delay diagnosis. Serious complications include central nervous system involvement, so confirmation depends on serological and bacteriological studies. (3,4)

Therefore, given the limited number of reports on this condition, the objective of the present work was to describe the clinical findings, complementary studies, therapeutic behavior and evolution of a case of neurobrucellosis in a pediatric patient.

Clinical case

We present the case of a 4-year-old girl from a rural area of Sancti Spíritus, who was admitted to the hospital with a fever of 38°C, conjugate gaze palsy, generalized rigidity, and sphincter relaxation, initially interpreted as a seizure. Physical examination revealed a distended abdomen with diffuse tenderness in the mesogastrium, without guarding or palpable masses, and occasional disorientation, without other focal neurological findings or a clear postictal period.

In the emergency department, cerebrospinal fluid (CSF) analysis was performed, which was clear with 4 cells x 10⁶/L and negative for Pandy. For the first two days, the patient had a fever between 38–38.5 °C and abdominal pain. Laboratory studies showed a hematocrit of 0.33 L/L, leukocytosis of 29 x 10⁹/L (78% segmented neutrophils, 20% lymphocytes, 2% band neutrophils), and an erythrocyte sedimentation rate (ESR) of 114 mm/h, suggesting an infectious process without an apparent focus. Empirical treatment with ceftazidime (100 mg/kg/day) was initiated; abdominal ultrasound revealed no abnormalities.



The following day, the patient began to exhibit irritability. A neurology consultation was requested, revealing nuchal rigidity, while the rest of the nervous system examination was normal. Fundoscopic examination showed no evidence of papilledema. An urgent non-contrast computed tomography (CT) scan of the head revealed mild signs of cerebral edema, peripheral effacement of the subarachnoid spaces, and narrowing of the frontal horns of the lateral ventricles (Figure 1).

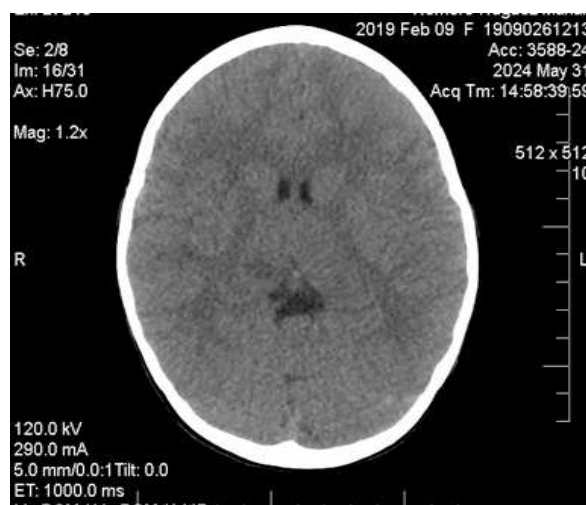


Fig. 1. Simple CT scan of the skull showing signs of cerebral edema

Source: Image taken by the author.

A second cerebrospinal fluid (CSF) analysis showed a cloudy appearance, with pleocytosis ($110 \times 10^6/L$), a positive Pandy sign, elevated protein (20 g/L), and glucose of 3.6 mmol/L, findings consistent with acute meningoencephalitis with cerebral predominance. The patient presented with a fever of 39 °C, alternating between drowsiness and irritability, and was therefore transferred to the intensive care unit. Treatment was initiated there with meropenem (40 mg/kg/8 h) and vancomycin (60 mg/kg/day), in addition to 20% mannitol (0.25 g/kg/dose) for the management of cerebral edema. Fundoscopic examination revealed a hyperemic optic disc with blurred margins, without hemorrhages or exudates.

After 72 hours of intensive treatment, the patient showed general improvement, without headache or vomiting, although fever and epigastric pain persisted.

Gastroenterology evaluation suggested gastroduodenitis of probable parasitic origin, and albendazole (5 mg/kg/day) was prescribed. A white blood cell count showed $13.5 \times 10^9/L$ leukocytes (70% segmented neutrophils, 30% lymphocytes) and an erythrocyte sedimentation rate (ESR) of 65 mm/h.

One week after admission, the patient presented with repetitive speech and drowsiness. A repeat CSF analysis showed 65 cells/ μL . Blood culture was negative, but acute phase reactants remained elevated: leukocytes $13.9 \times 10^9/L$ (78% segmented neutrophils, 21% lymphocytes, 1% band neutrophils) and an erythrocyte sedimentation rate (ESR) of 118 mm/h. Given these findings, a healthcare-associated infection was considered, and amikacin (15 mg/kg/day) was added to the ongoing antimicrobial regimen of meropenem and vancomycin.

Fourteen days after admission, the patient presented with disorientation and periods of unresponsiveness to questioning. Given the worsening clinical condition and persistent fever, a neurological evaluation attributed the neurological manifestations to a systemic infection. Fundoscopic examination revealed a hyperemic optic disc with blurred borders, without venous pulse, hemorrhages, or exudates. On day 16, the neurological deterioration became more pronounced, with fluctuations between irritability and obtundation. The patient lost the ability to articulate words, although she expressed pain gestures, and developed signs of pyramidal tract dysfunction, such as distal spasticity, hyperreflexia with a polykinetic response, and an increased reflexogenic area.

The patient initially presented with focal motor seizures that subsequently generalized. An electroencephalogram (EEG) was performed, revealing cortical dysfunction. Treatment was initiated with levetiracetam (100 mg suspension, 10 mg/kg/day). A brain MRI revealed diffuse lesions in the subcortical gray matter and in the periventricular and subcortical white matter. These lesions appeared hypointense on T1-weighted images (Figure 2A) and hyperintense on T2-weighted and FLAIR images (Figure 2B), predominantly in the left frontoparietal and right parietal regions, with asymmetry in the left frontal sulci.

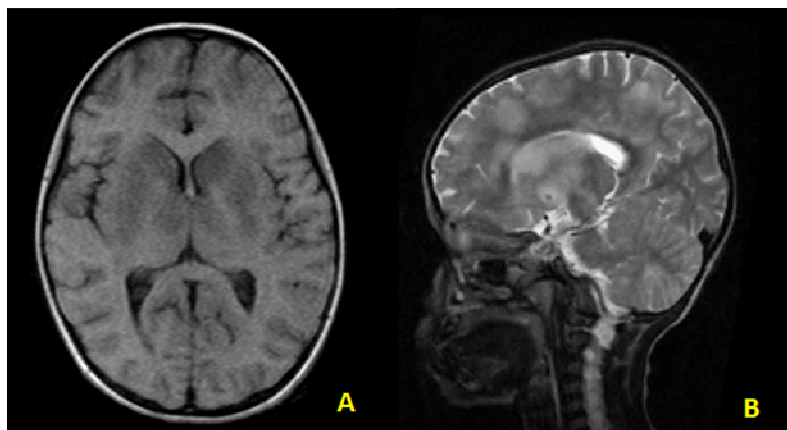


Fig. 2AT1-sequence MRI.

Fig. 2B. T2 sequence MRI sagittal section.

Source: Image taken by the author.

The evolutionary leukogram showed a count of $15.8 \times 10^9/L$, with a distribution of 87% segmented neutrophils (S), 10% lymphocytes (L) and 2% band neutrophils (Stabs), as well as an ESR of 80 mm/h.

The bone marrow scan revealed hyperplastic bone marrow, without infiltration by a tumor process; the virological study of blood and CSF sent to the National Reference Center Pedro Kourí Institute of Tropical Medicine (IPK) was negative.

Given the persistent, prolonged fever, elevated erythrocyte sedimentation rate (ESR), leukocytosis with a left shift, and multisystem clinical deterioration, a group evaluation of the case was performed. The combination of these findings, along with the epidemiological history of unpasteurized milk consumption, led to a suspected diagnosis of brucellosis. The disease was confirmed with positive serological tests for Brucella, and treatment with rifampicin (300 mg capsules) (10 mg/kg) and sulfaprim (480 mg ampoules) (60 mg/kg/day) was prescribed for 10 days. Improvement in clinical manifestations was observed. The patient was discharged with follow-up appointments in pediatrics and neurology.

Informed consent was obtained from the patient's parents before this report was prepared.

Discussion

Neurobrucellosis is a severe and complex manifestation of systemic brucellosis in pediatrics. Its heterogeneous clinical presentation, illustrated in this case by nonspecific initial symptoms, frequently delays diagnosis by mimicking other central nervous system infections. (3,4) Confirmation requires the integration of microbiological and serological methods, according to the established diagnostic standard. (5,6) In this patient, cerebrospinal fluid analysis was key to identifying meningeal involvement, a finding consistent with previous reports. (7,8)

Neuroimaging studies proved crucial in assessing the extent of the brain lesion. Diffuse lesions were observed in the subcortical white and gray matter, with a bilateral frontoparietal pattern that may reflect the anatomical variability of this condition. (9,10) From a therapeutic perspective, the favorable response to the combined rifampicin and sulfaprim regimen validates the efficacy of prolonged antibiotic therapy. However, this case highlighted the critical need for multidisciplinary management, including the control of severe neurological complications such as cerebral edema and seizures, a point corroborated by recent literature. (6,7)

Significant challenges remain, including the underreporting of pediatric cases, which limits the development of specific clinical guidelines. (8,9) This report underscores the importance of epidemiological factors, such as the consumption of unpasteurized dairy products, to guide clinical suspicion.

Conclusions

This case highlighted the diagnostic challenges posed by the nonspecific initial presentation and the complexity of management in a context of significant morbidity. The definitive diagnosis, supported by clinical and epidemiological history, specific serology, and neuroimaging, underscored the importance of a multidisciplinary approach.



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Conflict of interest

The authors declare no conflict of interest.

Authorship contribution

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Methodology, writing, review and editing: Eliecer González Valdéz, Miguel Angel Amaró Garrido, Arkel David Martín-Martínez

