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Case Report

Cerebral tuberculoma and convulsion in infants: A case report ☆

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ABSTRACT

Tuberculosis of the central nervous system is the second most common site after tuberculous meningitis. It represents 0.2% of intracranial expansive lesions in some Western countries compared to 10%-30% in developing countries. We report the case of an infant of 1 year and 2 months old who presented for 15 days with convulsions with asthenia, hypotonia, without fever. The clinical examination and laboratory workup were without abnormalities. His father had ongoing pulmonary tuberculosis, but the infant had no clinical or radiological signs of pulmonary tuberculosis. A brain MRI was showed multiple punctiform brain lesions, suggesting intracerebral tuberculomas in the first place, given the clinical and radiological appearance and the father's history of tuberculosis. The patient was put on anti-convulsant and antibacillary treatment. Through this case, we can see the clinical and radiological polymorphism of cerebral tuberculoma. The diagnosis of certainty remains anatomopathological. The prognosis is poor when it is detected late.

Introduction

Tuberculosis remains a major cause of morbidity and mortality in developing countries [1,2]. With a prevalence of only 1% of all tuberculosis cases, intracranial tuberculomas are one of the most dreadful expressions of tuberculosis [3]. This severe form of intracranial granulomatosis is secondary to the hematogenous spread of Mycobacterium tuberculosis from a primary site (often pulmonary) to the brain parenchyma, ventricle, and meninges [4]. It favors areas with an abundant blood supply [4]. Management has benefited from advances in neuroimaging and stereotaxis as well as the efficacy of current antitubercular treatment regimens [1]. We report the case of multiple intracranial tuberculomas in a 14-month-old toddler.

Case report

A 14-month-old infant came in with a history of seizures, asthenia and hypotonia with no fever, in the last 15 days. He had

Abbreviations: CRP, C-reactive protein; MRI, magnetic resonance imaging; CT scan, computed tomography scan; HIV, human immunodeficiency virus; AIDS, acquired immunodeficiency syndrome.

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no history of delivery complications or neuro-developmental disease. Clinical examination did not reveal any sensory or motor deficit nor cranial nerve damage or signs of meningitis. The blood workup performed in the emergency room showed a mild elevation of inflammatory markers with a CRP = 17 mg/L (Normal: <6 mg/L) and a sedimentation rate = 22 mg/L (Normal: 3-13 mm/H).

Although the father was being treated for pulmonary tuberculosis, the infant had no respiratory symptoms and his chest X-ray was normal. A brain MRI was then ordered. It revealed multiple diffuse punctate lesions of the white and gray matter, predominantly in the posterior cerebral fossa and brainstem (Fig. 1). They appeared as hypointense on T1WI and hyperintense on T2WI, solid enhancing nodules (Fig. 2) surrounded by edema, and containing central calcifications (Fig. 1). There was moderate active hydrocephalus but no leptomeningeal enhancement.

Given the seizures, the father's history of pulmonary tuberculosis and the brain MRI appearance of the lesions, we retained the diagnosis of cerebral tuberculomas. The patient was put on anti-convulsant and anti-bacillary treatment using a combination of isoniazid and rifampicin for 12 months and pyrazinamide and ethambutol for 2 months. We assessed the effectiveness of the treatment through regular clinical and radiological follow-up.

Discussion

Tuberculosis is a major cause of morbidity and mortality in developing countries and particularly in Morocco where the annual incidence is 30,000 new cases/year, all sites included [1,2]. Extra pulmonary localization in the central nervous system is the second most frequent site after tuberculous meningitis. It represents 0.2%-5% of intracranial processes in Western countries compared to 10 to 30% in developing countries [1,5]. However, with the HIV/AIDS pandemic, this entity is emerging in the large urban centers of developed countries [1].

An intracranial tuberculoma is a mass of tuberculous granulomatous tissue that has been contained and limited by the host's immune defenses. It stems through hematogenous spread from a primary remote location, usually in the lungs. Several tubercles form and then fuse to form a lesion that is often lobulated [1,6]. From an etiopathogenic point of view, although the mycobacteria trigger the cell-mediated immune reaction that creates the tuberculome, Koch's Bacili are not present in the specimens. Histological examination of the tuberculome shows a central caseous necrosis surrounded by giant Langerhans epithelial cells, lymphocytes and polynuclear cells [1,7].

Clinical symptomatology is non-specific and depends on the location, size and number of lesions. General s such as fever or feverishness, asthenia and weight loss in the weeks preceding the neurological signs are inconstant [1,8]. The most frequent symptoms are headaches, signs of intracranial hypertension and sometimes troubles of consciousness and seizures in children. Other clinical manifestations come in the form of fever, meningismus, neurological deficits and coma.

TB skin test is positive in only 1 out of 4 cases while chest X-ray is positive in 1 out of 3 cases [9].

Lepto-meningitis and tuberculoma are the most frequent forms of tuberculosis in the brain [5], with tuberculomas occurring more often in immunocompromised patients [8]. Other neurological manifestations come in the form of hydrocephalus, secondary to either inflammatory obstruction of the basal cisterns or to extrinsic compression on the cerebrospinal fluid outflow tract by tuberculomas [10]. Other complications may occur such as multiple miliary parenchymal lesions, solitary or numerous abscesses and pachymeningitis. Vascular complications come in the form of ischemic infarction or cerebral thrombophlebitis [8].

The search for another tuberculous location must be systematic. Indeed, frequency of a concomitant infection in another location can be as high as 40% in the literature, with pulmonary tuberculosis and spondylodiscitis being the most common [11]. However, the absence of extracerebral tuberculosis should not rule out the diagnosis of tuberculoma [1].

With its rich clinical polymorphism and low specificity of radiological signs, affirming the diagnosis of tuberculosis can be difficult thus delaying treatment.

Cerebral tuberculomas can occur anywhere in the brain. Classically, they are supratentorial in adults and infratentorial in children [8,12]. Intracranial tuberculoma is most often unique (90%) [1,8]. Its radiological appearance is neither constant nor specific, suggesting numerous other inflammatory pathologies (cysticercosis and pyogenic abscesses) or neoplasms (metastases, gliomas or lymphomas) [1,13].

On CT scan, no image is specific for tuberculoma. A hypodense lesion with ring enhancement is typical and can be associated with central calcifications forming a targer sign. On MRI, the appearance of cerebral tuberculoma depends on its stage of evolution and the presence or absence of caseous necrosis, with a typical appearance found only in 34% of cases [8,14]. In the early stages, it appears discretely hyperintense on T1WI and hypointense on T2WI with solid enhancement [8,14]. Non-caseating tuberculoma appears hypointense on T1WI and hyperintense to brain parenchyma on T2WI with solid enhancement. Some caseating tuberculomas have a solid center, appearing hypo- or isointense in both T1WI and T2WI, with surrounding edema. Caseating tuberculomas with a necrotic center appear hypointense on T1WI and hyperintense on T2WI with ring enhancement [8,15].

MR spectroscopy can also be helpful for the differential diagnosis. Tuberculomas are characterized by little to no amino acids and lactates (unlike pyogenic abscesses), and a choline to creatinine ratio greater than 1 (unlike neurocysticercosis) [10].

In our patient, MRI showed multiple diffuse punctiform intraparenchymal lesions surrounded by edema, with solid enhancement, predominantly in the posterior cerebral fossa and the brain stem, suggestive of caseating tuberculomas with solid centers. Other infectious organisms (cysticercosis, cryptococcosis, bilharzia, toxoplasmosis, pyogenic bacterial abscesses, HIV, echinococcosis) have been sought, with the serologies coming back negative, thus allowing us to retain the diagnosis of tuberculomas.

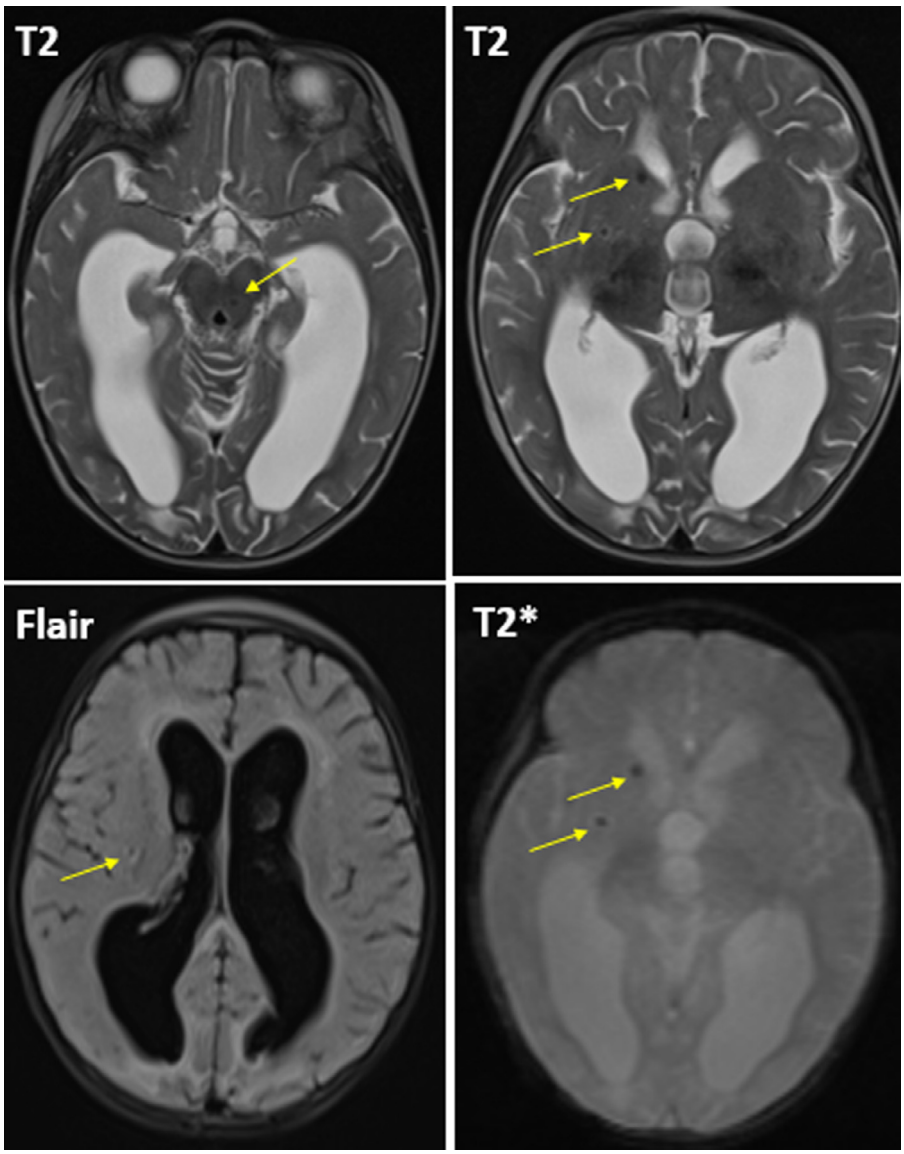

Fig. 1 – Axial sections of a brain MRI in T2, T2 echo gradient and FLAIR sequence showing the presence of multiple punctate subcortical, supratentorial, and infratentorial lesions, more marked on the right at the level of the head of the caudate nucleus, the pallidum, the internal capsule and the mesencephalic tegmentum, in T2 hyposignal and flair, surrounded by a thin border in T2 hypersignal (edema) and containing abnormalities in asignal on the T2 echo-gradient sequence related to central calcifications. There is moderate dilatation of the ventricular system with discrete signs of transependymal resorption.

In endemic areas, some authors advocate for empirical therapy based on a combination of evidence (history, symptoms, laboratory testing and imaging), thus avoiding invasive diagnostic methods, while others believe that a blind treatment may be harmful to patients and prefer to get a histopathological proof through biopsy first [12].

Treatment for cerebral tuberculoma is primarily based on anti-bacillary drugs. Surgery is exceptional, with only a handful of indications like optic tract compression, acute hydrocephalus or tuberculoma of the fourth ventricle [9]. For the treatment of children with cerebral tuberculomas, a 12 months combination of isoniazid (10-20 mg/kg/d, max 500 mg) and rifampicin (10-20 mg/kg/d, max 600 mg) is recommended, and a 2 months combination of pyrazinamide (30-35 mg/ kg/d, max 2 g) and ethambutol (15-20 mg/kg/d, max 1 g) [16]. Considering that cerebral tuberculoma is a "severe form of tuberculosis", this also corresponds to French recommendations. Tuberculoma involution is slow, several months on average, with vasogenic edema disappearing after 6 months. Lesions of about 2.5 cm in size disappear in about 5-8 months. When progression is too slow, it is not uncommon to continue treatment for up to 30 months [9]. Corticosteroids are frequently used

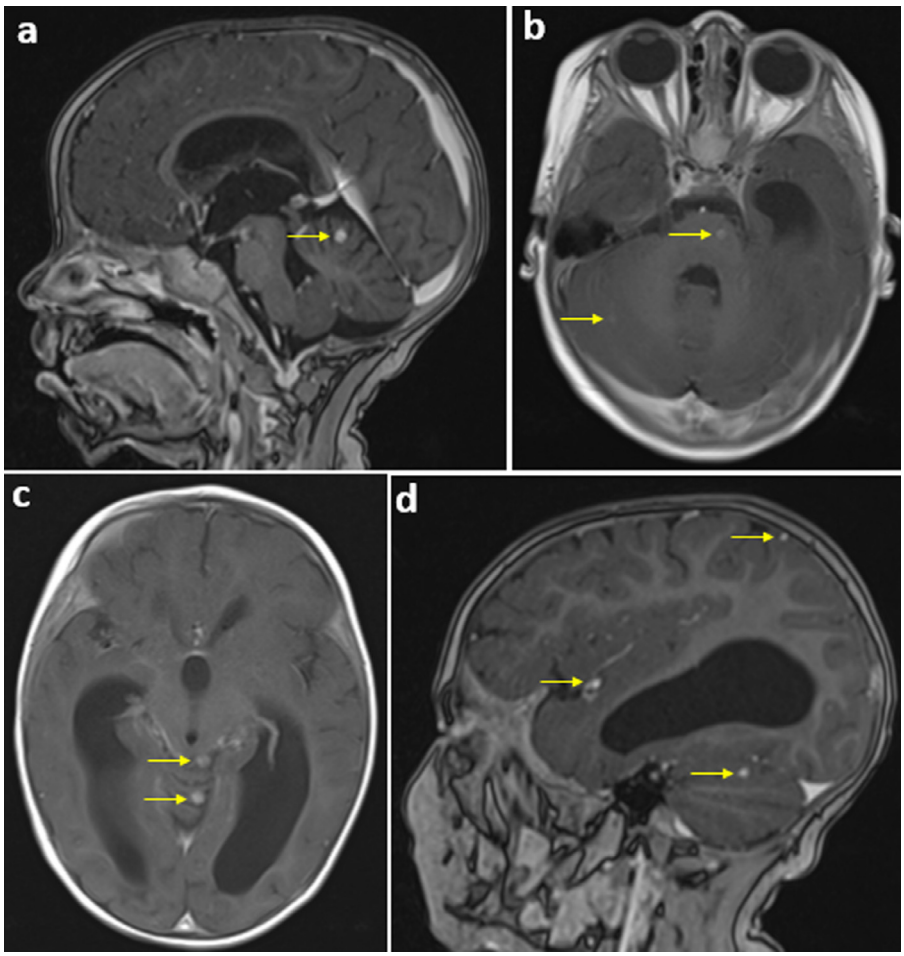

Fig. 2 – Axial and sagittal sections of a brain MRI in T1 sequence after injection of Gadolinium showing intense and homogeneous enhancement of lesions predominantly at the sub tentorial level at the level of the vermis (a and c), the mesencephalic tegmentum (b) and the cerebellum (b and d).

for anti-inflammatory purposes. However, the value of systematic corticosteroid therapy at the beginning of treatment of tuberculomas has not been proven, although it is strongly recommended in the treatment of tuberculous meningitis in children [12,16].

Sequelae of tuberculomas are rare and take the form of calcifications or atrophy of the adjacent parenchymal are, in often asymptomatic patients. Residual epilepsy may persist [9].

Conclusion

Central nervous system tuberculoma remains a clinical challenge because of its rarity. Positive diagnosis is based on a series of presumptive clinical, biological and radiological findings, while diagnosis of certainty is histopathological. MRI is an essential tool for patients with suspected cerebral tuberculoma, especially in tuberculosis-endemic countries. It has a vital role to avoid misdiagnosis and delayed treatment that cause significant mortality and morbidity, thus bettering prognosis of patients with brain tuberculomas who can access to treatment faster.

Ethical approval

Our institution does not require ethical approval to report individual cases or case series.

Patient consent

Written informed consent was obtained from one or more legally authorized representatives for the publication of anonymous patient information in this article.

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