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Imaging of Cerebral Tuberculomas

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Abstract **Case Report**

Intracranial tuberculomas represent one of the most serious localisations of tuberculosis. The most intra-parenchymal sites are the grey matter (GM)/white matter (WM) junction, the periventricular regions and the posterior cerebral fossa (a frequent site of lesions, especially for children). They may also be deeper, involving the basal ganglia, protuberance and cerebellum. In the early phase, the tuberculoma presents a discrete high signal in T1 sequence and low intense signal in T2 sequence with nodular enhancement. Non-caseating tuberculomas appear in low signal in T1 sequence and high signal in T2 sequence compare to the brain parenchyma and are intensely and homogeneously enhanced after injection of contrast in T1 sequence. Caseating tuberculomas with a solid centre appear low- or iso signal in both T1 and T2 sequences and are often associated with perilesional oedema. Caseating tuberculomas with a necrotic centre appear in low signal in T1 sequence and high signal in T2 sequence and are enhanced in the periphery after injection of contrast medium.

Keywords: Intracranial tuberculoma, Caseating tuberculoma, MRI sequences (T1, T2), Neurotuberculosis, Brain parenchyma.

INTRODUCTION

Intracranial tuberculomas are one of the most serious localisations of tuberculosis. Extra- pulmonary involvement of the central nervous system is the second most frequent site after tuberculous meningitis [1]. Cerebral-meningeal involvement occurs mainly via the haematogenous route from a primary site, most often in the lungs. The tuberculoma is a mass of granulomatous tuberculous tissue that has been contained and limited by the host's immune defences. It presents as an expansive intracranial lesion. The diagnosis is based on a combination of anamnestic, clinico-biological and radiological evidence. Confirmation is histological. The purpose of this article is to present cerebral tuberculoma at its clinical and radiological form (MRI and spectroscopy) and to provide an update on this infectious pathology based on this observation [1, 3].

CASE PRESENTATION

Child aged 1 year and 4 months, father with tuberculosis, presented with splenomegaly and multiple cervical adenopathies. A cerebral CT scan showed parenchymatous cerebral formations.

Cerebral MRI showed an intra-parenchymal lesion centred on the head of the caudate nucleus, opposite the frontal horn of the right VL, oval, well limited, with regular contours, heterogeneous signal: Isosignal T1, low signal T2 and Flair with areas of diffusion restriction, enhanced after injection of Gadolinium.

There is a small central area and a peripheral border with T1 highsignal and T2* empty signal, with peri-injury oedema.

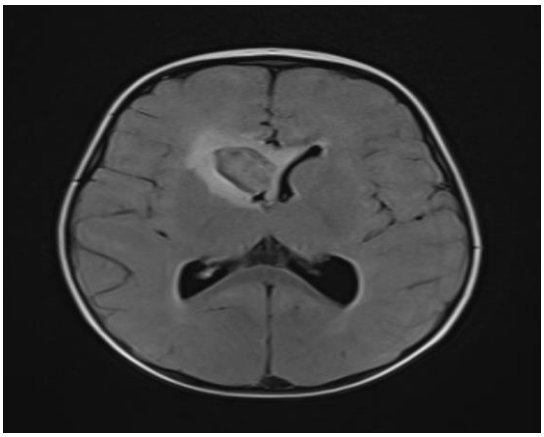

Figure 1: Flair axial sequence: lesion of the head of the caudate nucleus in discrete lowsignal

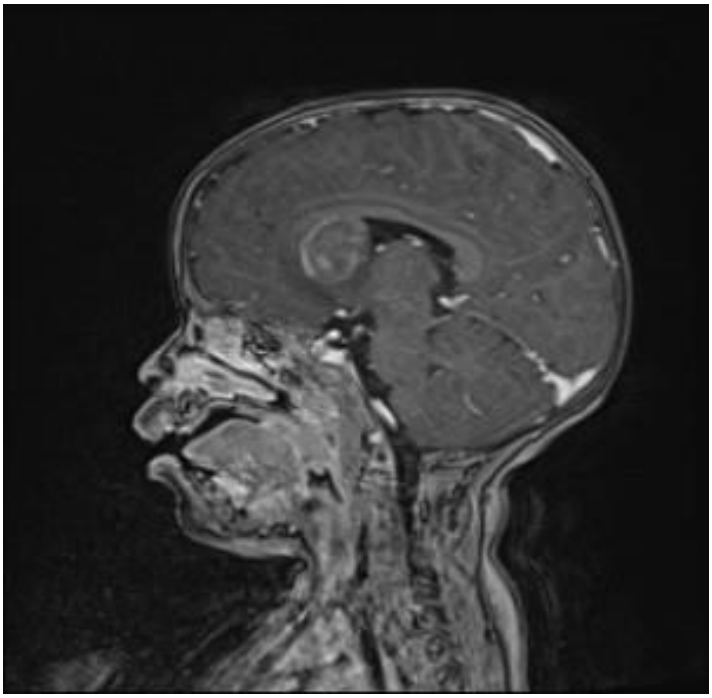

Figure 2: T1EG sagittal sequence with injection showing peripheral contrast

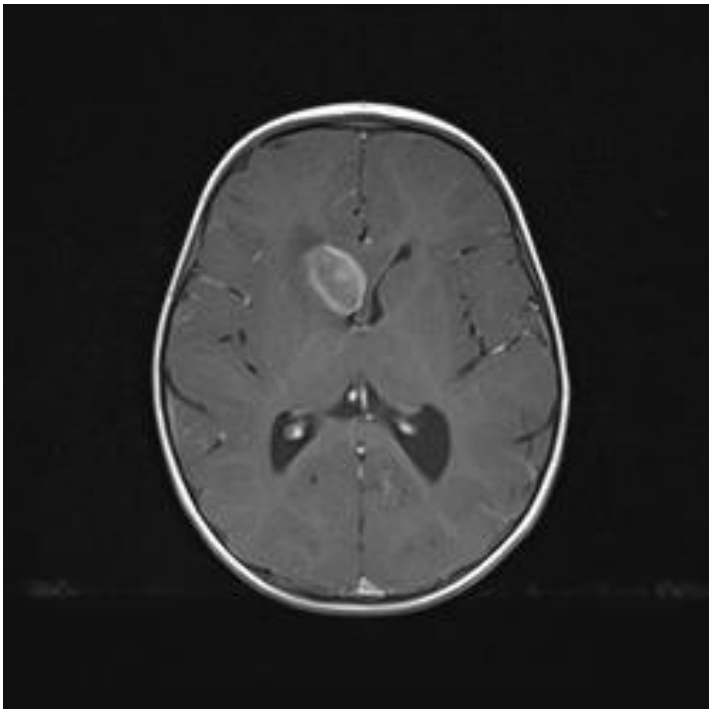

Figure 3: T1EG axial sequence with injection showing peripheral contrast

DISCUSSION

Cerebral tuberculomas represent a particular form of extra-pulmonary tuberculosis, more by its potential severity than by its frequency. It is a mass of tuberculous granulomatous tissue which has been contained and limited by the host's immune defences.

Intracranial tuberculoma results from haematogenous spread from a primary site. It can occur at any age, children and adults. The most common locations are the cerebral hemispheres, cerebellum, brain stem and optic chiasm. Other rarer sites include the quadrigeminal tubercles, corpus callosum, cerebellar tonsils, choroid plexuses, third ventricle, subdural space, mastoid cavities and cavernous sinus [2].

The revealing neurological signs are those of an intracranial expansive process and are directly related to the topography of the lesion. They may include headache, intracranial hypertension, focal neurological

deficits, epileptic seizures or confusion [7]. Diagnostic clues such as active extra-cranial tuberculosis, a history of tuberculosis, tuberculosis infection and general signs such as fever and weight loss may be absent [3].

Biological elements can provide a diagnostic orientation.

The introduction of medical imaging (CT and later MRI) has significantly improved the presumptive diagnosis of tuberculomas, with more cases being diagnosed since the advent of MRI. MRI is more sensitive than CT in detecting small tuberculomas and those located in the brain stem [2], cerebellum and spinal cord. The rounded or oval mass is substantially isointense to the grey matter in all signal weightings [5, 6]. It therefore appears in T1 as a moderate low signal of the white matter, sometimes more marked in the periphery due to the oedematous corona. In the T2 sequence, the tuberculoma is contrasted in 'negative' within the high signal oedematous area. The centre of the lesion has a high signal corresponding to the caseum. The capsule is isosignal or discrete high signal in T1, low-signal in T2 taking on a target appearance [5]. This decrease in T2 signal is thought to be related to the presence of paramagnetic free radicals produced by macrophages and distributed heterogeneously in these caseous granulomas. Those have a discretely more intense signal on proton density than on T2 (probably related to the inflammatory exudate and the high protein content of the caseum). The respective widths of the peripheral zone and the caseous 'core' are variable, and some lesions may appear solid. Another aspect consists of concentric circles representing layers of different ages, some of which are necrotic [6]. The presence of calcifications is rarely detected on MRI in the form of signal-free areas within the tuberculoma. The difficulty with which MRI detects focal calcifications is not a handicap in cases of central nervous system tuberculosis, since calcifications are only detected on CT in 1 to 6% of tuberculomas [5]. After injection of Gadolinium, very intense, more or less voluminous and often irregular nodular contrast is observed. They may be homogeneous or heterogeneous, with central necrosis that may be on iso or low signal [6]. Tuberculomas are solitary in about 2/3 of cases.

Among the multiple forms, one presentation that is highly suggestive of tuberculosis is a cluster of small annular lesions, often grouped together in a focus. Another suggestive aspect is the coalescence of several tuberculomas forming a multiloculated, cluster-like lesion [4, 5]. For extra-cerebral tuberculomas adherent to the dura mater, tenta or convexity, MRI with injection is more sensitive than CT with injection, thanks to its multiplanar imaging and the absence of bone artefact [4].

Cerebral oedema surrounding the granuloma may be minimal in small lesions, and in general there is less oedema than around a pyogenic abscess of the same size. This peri-lesional oedema is more prominent in the early phases of tuberculoma formation [3, 5] and is reflected by a T2 high signal [6]. The appearance of a tuberculoma on a T2 sequence is therefore much more specific than on a CT scan or on a T1 sequence after injection of gadolinium, particularly in relation to primary tumours. These tumours have a long T2 and their high signal is often indistinguishable from the peripheral oedema. However, it is not pathognomonic, as it can be found in granulomas of other aetiologies and some tumours (lymphoma in particular).

Diffusion and spectroscopic imaging sequences are currently very useful for the early, non- invasive diagnosis of cerebral tuberculomas, especially in patients with no systemic signs of tuberculosis. They also allow specific characterisation of tuberculomas in relation to other infectious lesions and primary and secondary tumour lesions [2, 6].

Surgical treatment depends on both the clinical form and the location in the brain. There are many indications for surgery. A biopsy under stereotactic conditions will be carried out for deep forms that are difficult to access surgically (at the base of the skull or in the brain stem) and for multifocal or abscessed forms, with aspiration under stereotactic conditions after CT scanning. In addition to surgical treatment, medical treatment is always necessary for cystic, compressive forms that cause deficits or hydrocephalus [7].

CONCLUSION

Cerebral tuberculoma remains a severe, highly polymorphic infection that can be life- threatening. Diagnosis has long benefited from the contribution of medical imaging, particularly CT and MRI.

On MRI, the lesion was hypointense in T1 with peripheral enhancement by gadolinium. The T2 appearance depended on the phase of the disease. The definitive diagnosis was made on the basis of clinico-biological data or after therapeutic testing, or confirmed by stereotactic biopsy. Progression with antibacillary therapy was always favourable.

REFERENCE

1. Semlali S, El Kharras A, Mahi M, Hsaini Y, Benameur M, Aziz N, *et al.*, Les aspects en imagerie de la tuberculose du système nerveux central. J Radiol. 2008;89(2):209–220. doi: 10.1016/s0221-0363(08)70396-2.
2. Ech-cherif El kettani N, Jerguigue H, Karouache A, El quessar A, El hassani MR, Chakir N, Boukhrissi N, Jiddane M. No 5 imagerie des tuberculomes encephaliques. Journal de radiologie. 2004 Sep;85(9):15–17.
3. Idris MN, Sokrab TE, Arbab MA, Ahmed AE, El Rasoul H, Ali S, *et al*., Tuberculoma of the brain: a series of 16 cases treated with anti-tuberculosis

drugs. Int J Tuberc Lung Dis. 2017;11(1):91–95.
4. Boulahri T, Taous A, Berri MA, Traib A. Atteinte cérebro-méningée multiple révélant une tuberculose multifocale chez un immunocompétent. Pan African Medical Journal. 2016;25:231. doi: 10.11604/pamj.2016.25.231.11074.
5. Jinkins JR, Gupta R, Chang KH, Rodriguez-Carbajal J. MR imaging of central nervous system tuberculosis. Radiol Clin North Am. 1995 Jul;33(4):771–86.
6. Boukobza M, Tamer I, Guichard JP, Brunereau L, Polivka M, Leibinger F, *et al*., Tuberculose du système nerveux central: aspects IRM et évolution à propos de 12 cas. J Neuroradiol. 1999;26(3):172–81.
7. White AE, Davies KG, Anwar S, Neal JW, Vafidis JA. Cerebral tuberculoma. Br J Clin Pract. 1994;48(4):222–3.