



Cerebral Tuberculoma revealed by epilepsy, Successful Removal and Good Evolution (About one case). Tuberculome cérébral révélé par une épilepsie, exérèse chirurgicale complète et bonne évolution (à propos d'un cas).



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Abstract

Epilepsy may be one of clinical presentations of any space occupying lesions of brain so that the later must be taken in consideration in any patient present with new convulsion crises. Tuberculosis has been an important public health problem in developing nations. Tuberculosis of the central nervous system (CNS) is rare; tuberculosis meningitis and tuberculoma are the two most important manifestations of tuberculosis in the CNS. Intracranial tuberculomas may be solitary or multiple. Solitary tuberculomas may be indistinguishable from cranial abscess or primary brain tumor. It is necessary to rule out tuberculoma in patients with intracranial mass lesions especially in endemic area. We present her a case of tuberculoma mimicking a high grade glial tumor on magnetic resonance imaging (MRI) in a 10-year-old child presented with six-month history of epilepsy and two-month history of gradual left hemiparesis. Cranial CTscan and MRI showed a right fronto-parietal peripheral ring-enhanced lesion with central necrosis. There was a strong suspicion of glial tumor. The lesion was totally excised with right fronto-parietal craniotomy. Histological examination of mass revealed a tuberculoma. Epilepsy disappeared just after surgery and the patient treated with antituberculous chemotherapy with good evolution after 10 months of follow up.

Keywords: Epilepsy- Tuberculoma- Glial tumor- Antituberculous therapy.

Résumé

L'épilepsie peut être l'une des présentations cliniques de n'importe quelle lésion cérébrale occupant de l'espace. Cette dernière doit être évoquée chez tout patient présentant des crises convulsives de novo. La tuberculose est l'un des problèmes de santé publique dans les pays en voie de développement. La tuberculose du système nerveux central (SNC) est une affection rare. La méningite tuberculeuse et les tuberculomes en sont les deux principales manifestations. Les tuberculomes intracrâniens peuvent être solitaire ou multiples. Les tuberculomes solitaires posent le problème du diagnostic différentiel avec les abcès intracrâniens et les tumeurs cérébrales primitives. Il est important d'évoquer ce diagnostic devant toute masse intracrânienne notamment dans les régions endémiques. Nous présentons un cas de tuberculome prêtant confusion avec un gliome de haut grade à l'IRM encéphalique, chez un enfant de 10 ans présentant des crises épileptiques évoluant depuis 6 mois, avec une hémiparésie gauche évoluant depuis 2

mois. La TDM et l'IRM cérébrales ont montré une lésion périphérique fronto-pariétale droite se rehaussant à l'injection du produit de contraste avec une nécrose centrale. Il y avait une forte suspicion de tumeur gliale. La lésion a été totalement réséquée avec une craniotomie droite fronto-pariétale. L'étude histologique a révélé un tuberculome. Les crises convulsives ont disparu juste après la chirurgie, et le patient a reçu un traitement anti bacillaire avec une bonne évolution après 10 mois de suivi.

Mots-clés: Epilepsie- Tuberculome- Tumeur gliale- Traitement anti bacillaire.

Introduction

Central nervous system tuberculosis is an infectious disease process that continues to be a prevalent endemic problem in certain world regions including Morocco. In recent times, the incidence of tuberculosis has been on the rise because of the increased incidence of acquired immunodeficiency syndrome [1, 2]. Intracranial tuberculomas are space-occupying masses of granulomatous tissue that result from hematogenous spread from a distant focus of tuberculous infection. Histologically, the mature tuberculomas are composed of a necrotic caseous center surrounded by a capsule that contains fibroblasts, epithelioid cells, Langhans giant cells, and lymphocytes [3]. Cerebral tuberculomas are a rare but well recognized complication of tuberculosis and most cases had associated with tuberculous meningitis which not occurs in our case. Paradoxical development or enlargement of tuberculomas during antituberculous chemotherapy has also been reported, and possibly has an immunological basis [4]. Treatment is with high dose steroids and continuation of antituberculous therapy, often for a prolonged course. Surgery has been used in isolated cases [5]. Tuberculomas should be considered in patients present with focal neurological deficit or signs of raised intracranial pressure (ICP) and newly developing epilepsy. Intracranial tuberculoma might be difficult to diagnose when the patient has no evidence or history of tuberculous infection as in our case [6, 7]. Intracranial tuberculoma can occur in otherwise healthy individuals and should always be considered in the differential diagnosis of solitary intracranial mass lesions especially in endemic area. Radiological diagnosis of a brain tuberculoma is difficult because the imaging presentation is varied and can be non-specific [8, 9].

Case report

A 10-year-old child without past medical personal or familial history of tuberculosis or epilepsy presented with six-month history of epilepsy, the crises was generalized tonico-clonic which increased gradually in frequency without respond to sodium valproate (described by error by a generalist without EEG or other investigation) the patient present also by two-month history of functional impotence of left side of the body. There was no prior history of seizures or headaches. Neurological examination revealed left hemiparesis. Hematological examination revealed a raised ESR. There was no chest radiograph abnormality. CT scan and MRI of the brain demonstrated a large ring lesion lying within the right fronto-parietal region with important edema and deviation of the median line. Axial non-contrast CT scan showed a large iso-dense lesion with important edema (fig. 1);

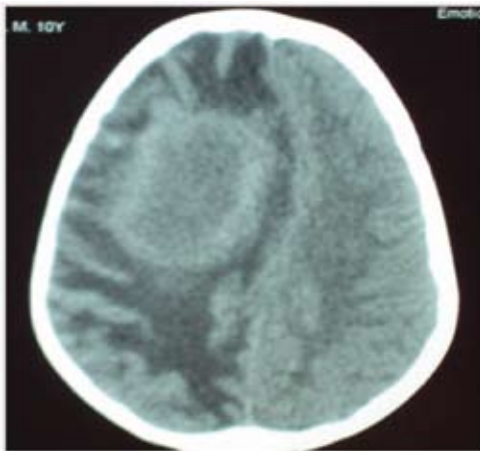


Figure 1: Axial non-contrast CT scan image showed an iso-dense right fronto-parietal lesion.

Contrast-enhanced CT scan images showed peripheral ring-like enhancement of the lesion with a central hypo-intense area (fig. 2).

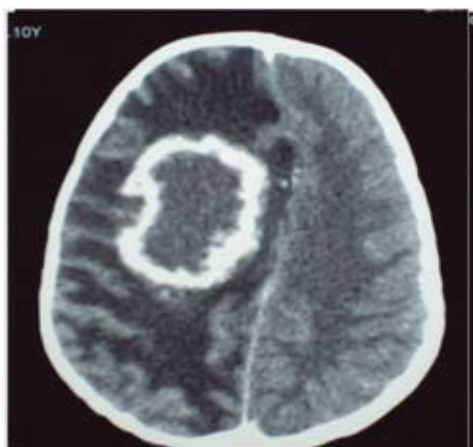


Figure 2: Axial contrast CT scan image showed a peripheral ring-like enhancement of the lesion with a central hypo-dense area.

MRI images showed identical lesions (fig. 3 and 4).

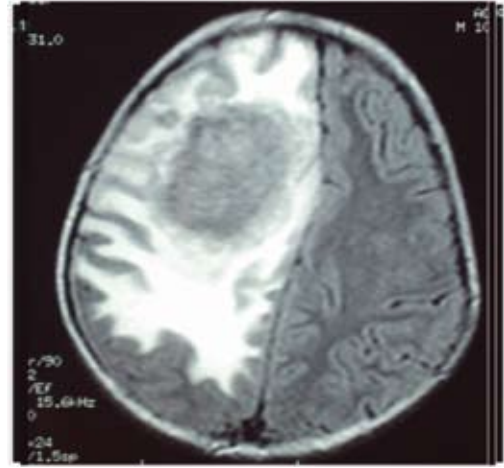


Figure 3: Contrast-enhanced axial T1-weighted images showed a large right fronto-parietal mass with perilesional edema represented by taking the contrast.

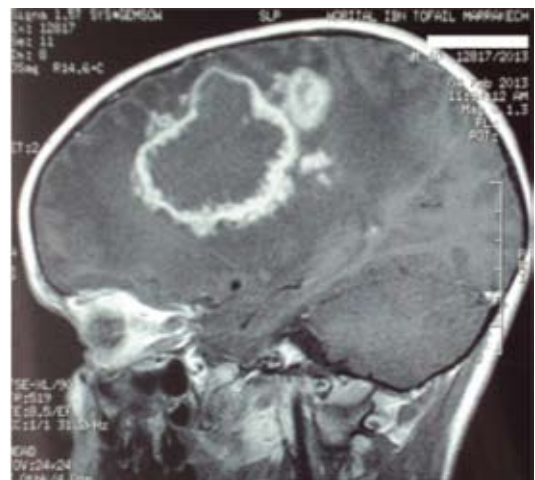


Figure 4: Contrast-enhanced sagittal T1-weighted images showed the same lesion in figure 3.

There was a strong suspicion of a glial tumor. Surgery was performed using right fronto-parietal craniotomy. The mass was firm, avascular and its removed totally (fig.5)

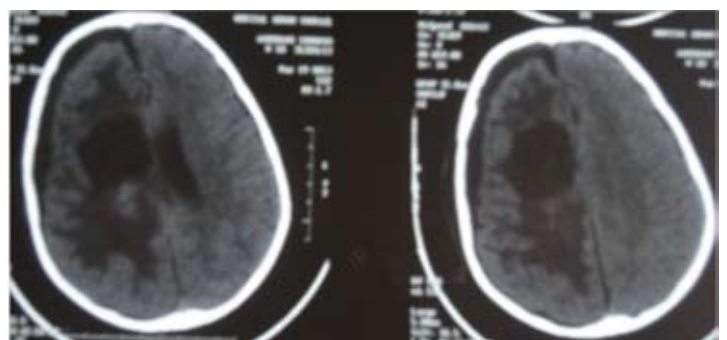


Figure 5: Post surgical CT scan showing a total removal of the cerebral tuberculoma.

The postoperative period pass without particular event. The histological examination confirmed tuberculoma. No bacilli could be obtained from lesion or cerebrospinal fluid (CSF). A serological test for HIV was negative. The patient

was discharged from the hospital and treated for 9 months with anti-TB therapy, including streptomycin, isoniazid, rifampicin, ethambutol, and pyrazinamide. Three months of sodium valproate was continued. The patient was symptom-free at the 10-month follow-up examination.

Discussion

Intracranial tuberculosis results from haematogenous spread of the organism from a distant site of infection [10]. In many developing countries TB is considered endemic. There is also an increasing incidence of TB in western countries due to migration, HIV, poor economic conditions in urban areas, drug-resistant mycobacteria, and the use of immunosuppressive drugs [11, 12]. It is therefore important that radiologists worldwide be familiar with the imaging features of intracranial tuberculosis. Diagnosis may be difficult when the CSF is unhelpful or when lumbar puncture is contraindicated [13]. Tuberculin skin testing is unreliable in endemic areas because of the high incidence of positive results [10]. Tuberculosis may mimic many diseases including neoplasms, e.g. gliomas [11, 12, 14, 15], cerebellopontine angle tumours [15], xanthoastrocytomas [16], metastases [11], lymphoma [11], posterior fossa tumours [10, 12, 17, 18], pineal mimicking pinealoma, pineoblastoma or germ-cell tumour [15]. The differential diagnosis varies according to the stage of development of the TB lesion [19]. Prior to granuloma formation the differential diagnosis includes mycotic disease, astrocytoma, metastases and sarcoidosis [20]. At the caseous stage it is often impossible to differentiate tuberculoma from a malignant tumor by CT [11]. A TB abscess may be indistinguishable from other causes of ring enhancement [21].

According to one study the MRI appearances of intracranial tuberculosis are more specific than the CT appearances [19]. MRI also assists the diagnosis by characterizing the rim, showing it to be layered in some patients [11]. Central hypo intensity on T2-weighted images is considered characteristic of tuberculomas [11], but may be seen in neoplasm that is very cellular with high nucleo-cytoplasmic ratios, e.g. primitive neuroectodermal tumour (PNET). TB abscesses, however, are indistinguishable from pyogenic abscesses on MRI. Sadeghi et al. [22] suggest that it may be possible to differentiate TB abscess from hyperintense tuberculoma on diffusion-weighted imaging (DWI). In our patient, contrary to our expectations, the lesion was avascular, but there was clear evidence of arteritis, at a considerable distance from the lesion, suggesting that this may not be a malignant lesion but a tuberculoma. However in our case the diagnosis was confirmed only by anatomopathological examination and by the response; clinical and radiological to antituberculous therapy. The common presenting symptoms and signs are headache, ICH, seizures, focal neurological signs and papilloedema. Fever may be present [23]. The CSF findings are unremarkable or show a mild non-specific increased protein content. The CSF culture is usually negative. The diagnosis is therefore made on the basis of neuroimaging findings which depend on whether the granuloma is

noncaseating, caseating with a solid center, or caseating with a liquid center [23]. The degree of surrounding edema is variable and is thought to be inversely proportional to the maturity of the lesion.

Conclusion

Despite advances in neuro-imaging techniques, the diagnosis of intracranial tuberculoma without systemic tuberculosis remains a challenge. It is important to make a definite differential diagnosis between tuberculoma and other space occupying lesions. Epilepsy even in a child must raise the suspicion of space occupying lesion including tuberculoma specially if not respond to treatment. Medical treatment is the preferred management method for intracranial tuberculoma, and anti-TB medication should be immediately administered if tuberculoma is suspected. Surgical intervention still has a role in the treatment of this disease.

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