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Recurrent tuberculous cerebellar abscess: A case study and review of the literature

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ABSTRACT

INTRODUCTION AND IMPORTANCE: Tuberculous cerebellar abscess is a rare form of extra-pulmonary tuberculosis. The outcome is often favorable with well-managed treatment; however, they can continue to develop. We share in this article our experience on the management of this rare pathologie.
CASE PRESENTATION: A 10-year-old boy with a medical history of tuberculous meningitis after 3 months of tuberculosis treatment. He presented to the hospital with acute obstructive hydrocephalus due to a large tuberculous cerebellar abscess. A puncture of the abscess was initially performed, followed by placement of a ventriculoperiotoneal shunt, which resulted in some clinical improvement. However, the child subsequently presented with neurological deterioration due to the massive enlargement of the tuberculous abscess despite adequate antituberculosis chemotherapy. The initiation of corticosteroid therapy associated with a readjustment of the dose of anti-tuberculosis drugs and a repeated puncture ultimately led to clinical improvement.
CLINICAL DISCUSSION: Tuberculous brain abscess is an extra-pulmonary location of tuberculosis rarely seen in immunocompetent children. The treatment consists of surgery associated with antituberculosis chemotherapy and rigorous clinico-radiological monitoring. Recurrence is possible despite well-conducted treatment. Additional corticosteroid therapy is necessary with readjustment of the anti-tuberculosis treatment for an effective cure.
CONCLUSION: Rarely, the tuberculous abscess of the cerebellum continues to evolve despite proper treatment. This pattern does not necessarily mean treatment failure. Close clinical and imaging monitoring is crucial in the management of these cases.

1. Introduction

Tuberculous abscess is a collection of pus containing tubercle bacilli surrounded by a dense vascular capsule [1]. This lesion is very rarely located in the cerebellum [2], and has been found in immunocompromised as well as immunocompetent subjects [3]. Among immunocompetent patients, the pediatric population is one of the most frequently affected [4]. Regression of the abscess is a sign of wee conducteur treatment [1]. However, tuberculous abscesses are often resistant to anti-tuberculosis treatment and may continue to develop, requiring surgery. However the majority of solid tuberculomas respond well to anti-tuberculosis therapy alone [5]. We report the case of a 10 year old boy presenting with a recurrent tuberculous cerebellar abscess treated at the Fann university hospital. This case report has been reported in line with the SCARE 2020 Criteria [6].

2. Case presentation

The patient is a 10-year-old, male, right-handed, student and of Diola ethnicity. He had been diagnosed with tuberculous meningitis in a pediatrics, and undergone antituberculosis treatment since 3 months, with good outcome. He then presented with a very severe headaches, associated with vomiting and impaired consciousness. He had previously presented with fever and night sweats. He had an up-to-date vaccination status, with no reported tuberculosis contagion, no surgical history. The neurological examination revealed: a Glasgow coma Scale of 13/15, the pupils were reactive, a statico-kinetic cerebellar syndrome, a meningeal syndrome with a positive kernig sign. The general examination revealed: a normal temper-

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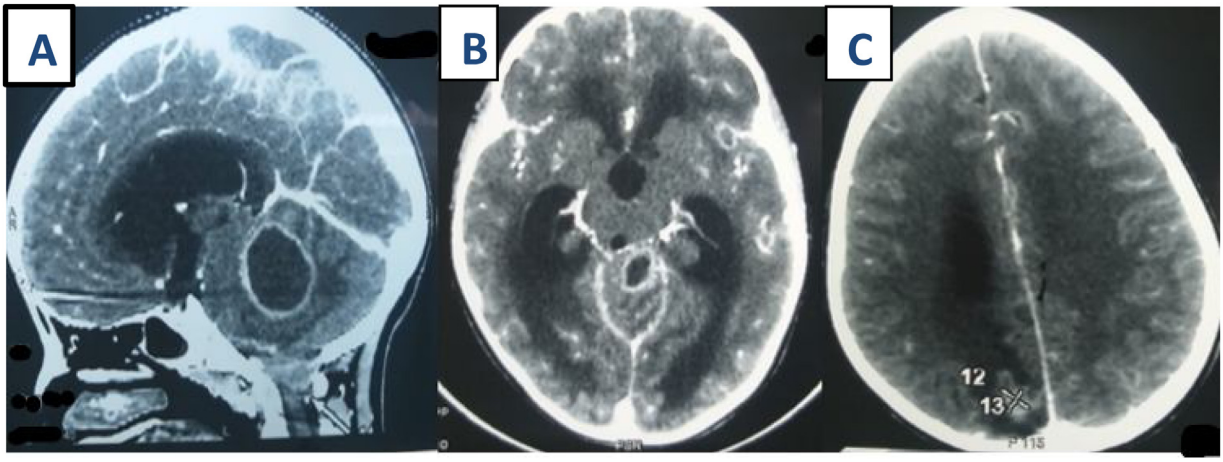

Fig. 1. Cerebral CT A sagittal reconstruction, B and C axial section: showing a tuberculous cerebellar abscess with onset of engagement, responsible for hydrocephalus. Frontal and occipital tuberculomas.

ature at 37.5 °C, a close of weight, initially at 25 kg he fall to 25 kg, a height of 146 cm, BMI 11.72 kg / m^2, TA 12/8 mm Hg, pulse at 95, respiratory fréquence at 22. The systemic examination was normal as well as the chest x-ray. The initial Genxpert were negative. The intra-dermal reaction to tuberciline (IDRT) result was 15 mm. The brain scan showed a large left cerebellar fluid lesion, surrounded by a hyperdense ring, responsible for acute triventricular hydrocephalus (HCP) and the onset of left tonsillar herniation A similar but smaller lesion was located on the right frontal and a small right occipital tissue lesion (Fig. 1). These lesions were presumed to be tuberculosis abscesses which developed despite ongoing anti-tuberculosis treatment and which did not respond to this treatment. A diagnosis of tuberculous abscess complicating tuberculous meningitis was made on the basis of the above results, however the bacteriological examination was negative. Hepatic and renal function tests were normal. A lumbar puncture was not performed because of the risk of brain herniation. The surgery was performed by a former intern neurosurgeon. The patient after general anesthesia and nasotracheal intubation was placed in a supine position. The cerebellar tuberculous abscess was drained with clear yellow thick pus. The pyoculture were negative for mycobacterium tuberculosis. Delay from presentation to surgery was one day. The patient clinically improved immediately. The following day, doses of anti-tuberculosis drugs were increased (1/3 added to the initial dose). Corticosteroid therapy was added to the treatment at a dose of 20 mg / d. A week later, after the first puncture, the patient presented again a neurological deterioration. The CT scan showed a recurrence of the same cerebellar lesion and an increase in ventricular dilation despite treatment, the other lesions were unchanged (Fig. 2). A ventriculoperitoneal shunt (DVP) was immediately placed resulting in clinical improvement and a Genxpert®, performed on the cerebrospinal fluid retrieved during the surgery, showed the presence of a strain of mycobacterium tuberculosis sensitive to rifampicin. The cytochemistry of the clear CSF showed: Proteinorachia =1.20 g / l, glucorachia at 0.32 g / l, cytology = 18 lymphocytes / mm3. The blood sedimentation rate was 85 mm / h, the level of serum C-reactive protein was normal. Three days later after this second intervention, he presented once again with disturbances of consciousness. The CT scan showed at this time persistence of the abscess with onset of cerebellar herniation despite well-conducted treatment and smaller lateral ventricles than before, indicating that DVP was working (Fig. 3). A third puncture was performed, combined with corticosteroid therapy, and the patient state of consciousness improved after this procedure. Despite this well-conducted treatment 17 day later, he again presented with disturbances of consciousness, anisocoria.

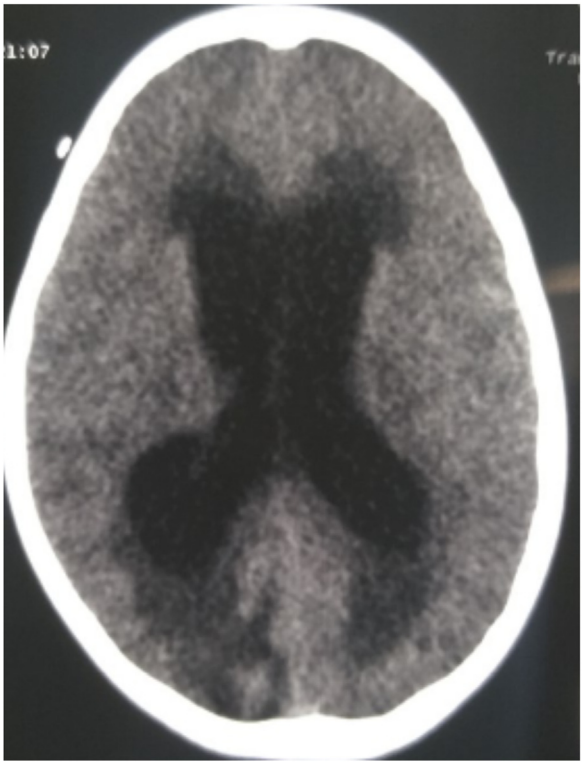

Fig. 2. Cerebral CT scan axial section showing active obstructive hydrocephalus.

The CT scan again showed an increase in the size of the tuberculosis abscess and signs of engagement, the others were unchanged. A fourth procedure was performed immediately (associated with corticosteroid therapy and an increase in the dose of isoniazid), and this procedure is followed by an improvement in the clinical condition of the child. Rigorous monitoring has always been carried out. A detailed investigation of the immune status did not reveal any abnormalities. The serology was normal, as was the cardiac ultrasound. Re-evaluation of the patient 5 months later after the start of treatment and 2 months after increasing the doses, showed weight gain. Neurologically he was conscious without complaints, able to walk with support. The control CT scan was requested (Fig. 4) and showed regression of the lesion. Re-evaluation of the patient 5 months later after the start of treatment and 2 months after increasing the doses, showed weight gain. Neurologically he was conscious without complaints, able to walk with support.

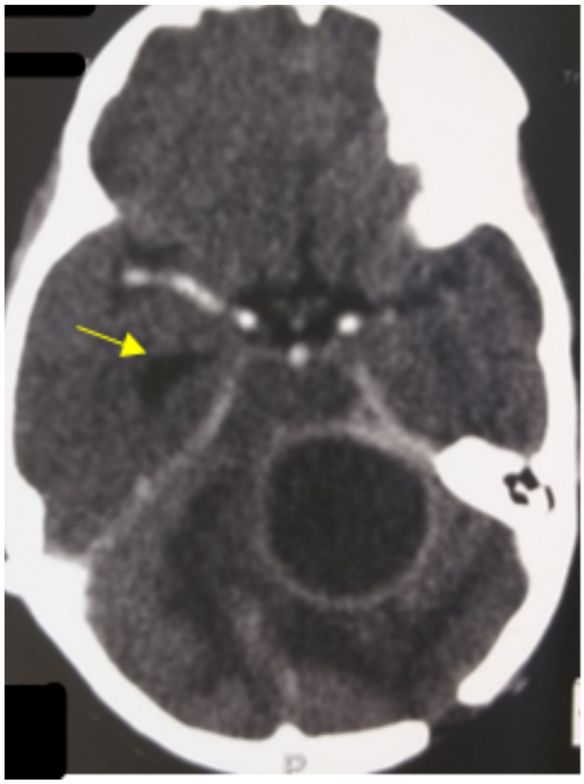

Fig. 3. Brain CT scan showing recurrence of the cerebellar abscess and smaller ventricles after the shunt (yellow arrow).

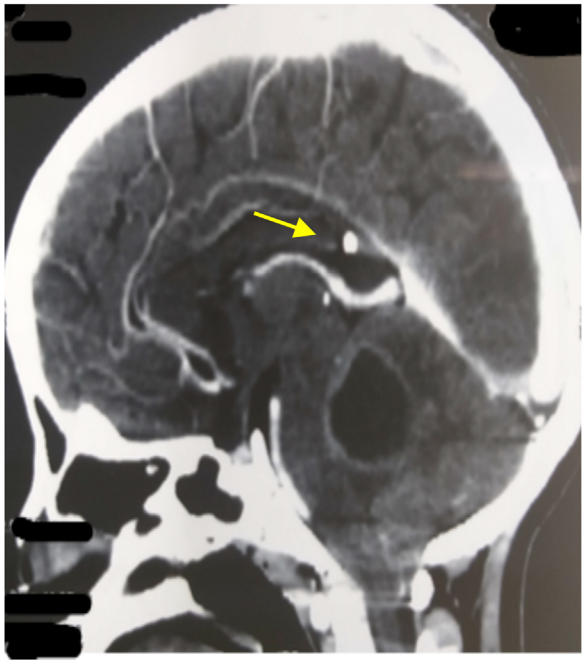

Fig. 4. Axial cut brain CT showing recurrence of the abscess and the shunt in place (yellow arrow).

3. Discussion

Central nervous system (CNS) tuberculosis abscess is a rare extra-pulmonary location of tuberculosis in which the typical tuberculoid granuloma is not present [1] Immunocompromised patients and malnourished children are at greater risk of CNS tuberculosis and abscesses in particular [7]. Our child was well nourished, HIV negative and showed no signs of immune deficiency. Few cases of tuberculous cerebellar abscess have been described in the literature [2]. Tuberculomas, tuberculosis abscess is a very rarely observed in children, as in our case [5]. In a review of 23 reports of increasing intracranial tuberculomas, 17 reports of tuberculomas developed in patients treated for tuberculous meningitis, such as our case who had tuberculous meningitis [7]. Seizures, neurologic signs, or intracranial hypertension that occur within weeks or months of starting anti-tuberculosis chemotherapy are usually the first clinical manifestations preceding the development of intracranial lesions [5]. Most symptoms of tuberculosis abscess follow a more indolent course over time than other bacterial infections, progressing over a period of 7–90 days [8]. Tuberculosis infection of the CNS is usually secondary to hematogenous dissemination of the tubercle bacillus, from a primary focus, often pulmonary [2]. In our case the pulmonary examination as well as the x-ray were unremarkable. On CT scan the tuberculous abscess usually presents as a hypodense lesion surrounded by an annular contrast enhancement with perilesional edema [1]. Clinically and radiologically, it can be difficult to differentiate a tuberculous abscess from a pyogenic abscess. It could mimic a pyogenic abscess or glioma on imaging studies. On MRI spectroscopy, tuberculous abscesses have a high concentration of lipids and phosphoserine in their walls [9]. MR spectroscopy was not performed in our case. According to Whitener diagnostic criteria for cerebellar tuberculous abscess are based on bacteriological evidence of the tuberculous origin and macroscopic evidence of abscess formation in the brain parenchyma, histological confirmation that the abscess wall is composed of granular tissue vascular with acute and chronic inflammatory cells [9]. Testing for tuberculosis bacilli in body fluids is not always positive. The GeneXpert®, which is a new molecular diagnostic tool, was developed to improve the detection of tuberculosis and decentralize drug resistance testing. The test can be used with sputum samples and also, with varying sensitivity, with other samples including cerebrospinal fluid, lymph node or aspirate tissue, pleural fluid, ascites fluid, urine, dialysis fluid and pus [10] In our patient the Genxpert performed on the CSF had shown the presence of a strain of mycobacterium tuberculosis sensitive to rifampicin. The expansion of existing tuberculomas and the development of new intracranial tuberculomas after the start of treatment is a well-known clinical entity, as in our case where tuberculomas developed after the start of treatment [7]. Neurotuberculosis must be treated with an appropriate therapeutic regimen, in the absence of treatment the consequences are devastating. One of the most important components of treatment is long-term anti-tuberculosis chemotherapy [2]. Treatment options for tuberculosis abscess include: simple puncture, continuous drainage, fractional drainage, repeated aspiration through holes, and total excision of the abscess [11]. Surgical drainage of the tuberculous abscess is necessary to relieve the mass effect caused by a large lesion or to obtain a microbiological diagnosis [1]. This surgical drainage of the abscess through a hole does not appear to be curative and when it is performed these patients should be carefully monitored [11]. In our patient, we performed a puncture of the abscess associated with medical treatment and rigorous monitoring. Corticosteroid therapy tends to reduce the incidence of tuberculomas which paradoxically develop in children treated for tuberculous meningitis [12]. This corticosteroid therapy, frequently used in the acute phase against cerebral edema, should be avoided because it could prevent the penetration of anti-tuberculosis drugs into the abscess and interfere with the encapsulation [13]. Generally, the management of tuberculosis lesions by anti-tuberculosis drugs leads to regression of tuberculous abscesses [1]. However, the tuberculous abscess can increase in size, despite surgical drainage and well-conducted anti-tuberculosis treatment, as in our

case [14]. An enlarged tuberculosis abscess can pose a dilemma for the clinician since it can potentially mean: treatment failure, anti-tuberculosis drug resistance, superinfection, an overlying neoplasm or inflammatory changes [14]. A prior study of the enlargement of the tuberculous lesions of the CNS showed in most cases the enlargement occurred 3 months later after the start of the anti-tuberculosis treatment, as in our case [15]. The majority of cases of enlarged TB lesions in the CNS resolve with a change in anti-TB chemotherapy. However, other cases may require: additional corticosteroid therapy, anti-tuberculosis treatment, or rarely surgical decompression [8,14]. Cerebellar tuberculosis abscesses have been notoriously difficult to treat [1]. The treatment of our patient was difficult. In our patient, clinical improvement was observed after: repeated punctures, corticosteroid therapy and appropriate anti-tuberculosis chemotherapy (increased in doses, in particular isoniazid). The management of a tuberculosis abscess is medical and surgical. Recurrence despite several punctures does not mean the failure of antituberculosis treatment. Careful monitoring is necessary before, during and after surgery, which should be carried out in a neurosurgical department.

4. Conclusion

Rarely, the tuberculous abscess of the cerebellum continues to evolve despite proper treatment. This pattern does not necessarily mean treatment failure. Given the possibility of asymptomatic enlargement of the abscess, close clinical monitoring and imaging is crucial in the management of these cases. Our patient's view of TB treatment is that it is long but effective

Declaration of Competing Interest

The authors claims no conflicts of interests.

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Ethical approval

The study is exempted from ethical approval.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in Chief of this journal on request.

Author contribution

All the authors contributed to this work.

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