Cerebral abscesses of tuberculosis origin: study of 8 cases at the University Hospital of Conakry

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Abstract

Introduction: Tuberculosis of the central nervous system (CNS) remains endemic in developing countries with high morbidity and mortality despite advances in imaging and treatment. The objective of our work was to describe the diagnostic and therapeutic difficulties of this pathology in the context of a country with limited health resources.

Materials and methods: We retrospectively studied 8 cerebral tuberculosis abscess files, collected in the archives of the neurosurgery department of the Conakry University Hospital over a 5-year period (January 2013–December 2017). The diagnosis was made by scanning and by isolation of acid-fast bacilli (AFB) by direct examination of pus.

Results: Mean age was 33 years (9–56 years), sex, 5H 5 men/3F 3 women. History of pulmonary TB, 3 cases; TB contagious, 5 cases; TST ITR positive, 5 cases; associated visceral TB, 5 cases; HIV positive serology, 2 cases. The clinic was dominated by altered consciousness, 7 cases; focal signs, 7 cases; and fever, 5 cases. The site was hemispherical in 7 cases and one case in the posterior brain fossa. Treatment was medico-surgical in all patients. The evolution was favorable in 3 cases, the neurological sequelae in 3 cases, 2 cases of death, and 2 cases of recurrence.

Conclusion: Cerebral tuberculous abscesses constitute a medico-surgical emergency. Despite their rarity, morbidity and mortality remains high in the context of developing countries due to diagnostic and therapeutic delays.

Keywords: Abscess, Tuberculosis, CNS, Neurosurgery, Conakry

Introduction

Tuberculosis, caused by Mycobacterium tuberculosis, remains a hot topic around the world. It is a major public health problem in developing countries, where it remains common [1, 2]. It is on the rise in developed countries, especially since the AIDS pandemic [3]. Cerebral intraparenchymal tuberculosis accounts for 10–30% of intracranial expansive processes in developing countries [4–6] where it remains endemic and associated with high morbidity and mortality despite advances in imaging and treatment. Despite their low frequency (1% of extrapulmonary tuberculosis cases) [7], they occupy a special place because of their poor prognosis, mainly due to the delay in treatment. This parenchymal localization is essentially confined to tuberculomas. Abscesses are exceptional [8].

We report our experience in order to describe the diagnostic and therapeutic difficulties of this pathology in the context of a country with limited health resources.

Materials and methods

We retrospectively studied 8 cerebral tuberculosis abscess files, collected in the archives of the neurosurgery department of the Conakry University Hospital over a 5-year period (January 2013–December 2017). The diagnosis was made by scanning and by isolation of acid-fast bacilli (AFB) by direct examination of pus.
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All our patients benefited from a complete clinical examination, a standard check-up (blood count, C-reactive protein, HIV serology, tuberculin intradermal reaction, examination of sputum or gastric tubing, and urine for acid-fast bacilli), cerebral CT scan, chest X-ray, and direct examination of pus with Ziehl-Neelsen staining for mycobacteria after puncture of the abscess. All patients received quadruple therapy (isoniazid, rifampicin, ethambutol, and pyrazinamide) for 3 months, combined with corticosteroid therapy for the first 15 days (prednisolone) and then dual therapy (isoniazid and rifampicin) for 18 months. Surgical treatment was carried out in all cases. The operating technique was a freehand evacuating puncture; an excision of the hull was associated in case of recurrence.

The evolution was judged good when there was clinical recovery without sequelae, average when there were neuropsychic or motor sequelae, and poor in case of death.

Our study essentially concerned patients whose direct examination of pus isolated acid-fast bacilli (AFB) in pus. The data collected were analyzed using the SPSS version 21.0 software.

Results

Epidemiologic characteristics

The average age of our patients was 33 with extremes of 9 and 56. The study concerned 5 men and 3 women. Three patients had a history of pulmonary tuberculosis, and the notion of tuberculosis contagion was found in 5 cases. Intradermal tuberculin reaction was positive in 5 cases. Pulmonary tuberculosis was associated in 4 cases and urogenital in 1 case. The HIV serology was positive in 2 cases.

Clinical manifestations

Clinically, the presentation of symptomatology was variable, dominated by disturbances of consciousness, focal neurological signs, and fever. The general patients’ characteristics are summarized in Table 1. The time to diagnosis ranged from 3 weeks to 4 months.

Biological data

Biology showed hyperleukocytosis in 4 cases and positive CRP in 5 cases. Direct examination of pus isolated numerous acid-fast bacilli (greater than 10/fields) in all cases.

Medical imaging data

Chest X-rays, performed in all patients, showed abnormalities in 5 patients with a miliary appearance in 2 cases, an active tuberculosis appearance in 2 cases, and stigmas of a tuberculous past in 1 case.

In the brain scan performed in all patients, the image was typical of a cockade of spontaneous hypodensity contrasting in a ring pattern and surrounded by a large hypodense halo of edema. The localization was hemispherical in 7 cases including 4 frontal cases (Fig. 1), 2 temporal cases, 1 multiple case (Fig. 2), and in the

| Table 1 General patients’ characteristics |
|------------------------------------------|
| **Case no. 1** | **Case no. 2** | **Case no. 3** | **Case no. 4** | **Case no. 5** | **Case no. 6** | **Case no. 7** | **Case no. 8** |
| Age/sex | 9 years, male | 37 years, male | 50 years, female | 41 years, male | 24 years, female | 31 years, male | 15 years, male | 59 years, female |
| History of tuberculosis | Yes | No | No | No | Yes | No | No | Yes |
| Notion of contagion | Yes | No | Yes | No | Yes | No | Yes | Yes |
| ITR | Positive | Positive | Negative | Positive | Negative | Positive | Positive | Negative |
| Extracerebral tuberculosis | No | Yes, urogenital | Yes, pulmonary | No | Yes, pulmonary | No | Yes, pulmonary | No |
| HIV serology | Negative | Negative | Negative | Negative | Positive | Negative | Negative | Positive |
| Clinic | Fever, headache, vomiting, confusion | Headache, vomiting, slimmimg, focal sign, confusion | Fever, vomiting, slimmimg, focal sign, coma | Fever, headache, focal sign, confusion | Fever, headache, focal sign, coma | Headache, focal sign, convulsions, coma | Fever, focal sign, convulsions, coma |
| Antituberculosis treatment (duration) | 21 months | 21 months | 21 months | 21 months | 2 months | 21 months | 21 months | 1 month |
| Surgery | Puncture, exeresis | Puncture | Puncture | Puncture | Puncture | Puncture | Puncture | Puncture, exeresis |
| Evolution | Favorable | Favorable | Average (hemiparesis) | Average (epilepsy) | Death | Average (epilepsy) | Favorable | Death |

*ITR* intradermal tuberculin reaction, *HIV* human immunodeficiency virus
posterior fossa in 1 case (Fig. 3). Hydrocephalus was associated in 1 case and subdural empyema in 1 case (Fig. 4).

**Treatment**
The treatment was medico-surgical in all our patients. The medical treatment consisted of quadruple therapy (isoniazid 300 mg/day, rifampicin 600 mg/day, ethambutol 1800 mg/day, and pyrazinamide 1200 mg/day) for 3 months, combined with corticosteroid therapy for the first 15 days (prednisolone 2 mg/kg/day) then dual therapy (isoniazid and rifampicin) for 18 months.

The time of surgery varied from 24 h to 2 weeks. For 6 cases, it was performed within the first 24 h following hospitalization and for 2 cases after failure of 2 weeks of trial treatment (Adiazine® 4 g/d Malocide® 1 mg/kg/d) for suspicion of cerebral toxoplasmosis in the field of HIV. It consisted of a trepano-puncture in all patients, which was justified on criteria of size and clinical tolerance, notably for consciousness disorders associated with a compressive collection.

**Evolution**
The evolution was favorable in 3 cases, average in 3 cases (1 case of hemiparesis and 2 cases of epilepsy), and
bad in 2 cases (2 deaths: 1 case of respiratory distress and 1 case of septic shock). Recurrence was noted in 2 cases, requiring surgical removal.

Discussion
CNS tuberculosis is common in developing countries. It has been on the rise in industrialized countries since the advent of HIV 1 infection [4]. CNS involvement can take three forms of unequal frequency: tubercular meningitis, tuberculomas, and, exceptionally, tubercular abscesses [9]. Cerebral abscess is a rare lesion in tuberculosis, occurring in 4–8% of non-HIV patients and 20% of AIDS patients [10]. The abscess is often single, sometimes multilocular and accompanied by greater perilesional edema [11]. It most often occurs in the corticocortical regions and in the central gray nuclei, but also in the cerebellum. Genuine cerebral subdural tuberculous empyema has been more rarely reported [12, 13], encountered in a patient in our series.

These brain abscesses, like tuberculomas, are probably secondary to hematogenous dissemination from a primitive site, most often pulmonary, sometimes acute, contemporary with the abscess [14] as was the case in 4 patients in our series, sometimes old scarring or chronic [15], encountered in 3 patients in our series and 18 patients out of 122 in Kilani et al. series on adult central nervous system tuberculosis [11], and sometimes digestive, lymph node, or urinary [16] as reported in a patient in our series. On the other hand, the low frequency of abscesses compared to tuberculomas remains unexplained. Lecuit et al. hypothesize the role of CD4 deficiency as an etiopathogenic factor in the lower frequency of granulomas, which are controls for the host response to the presence of the pathogen in HIV-infected patients [17].

Unfortunately, the tuberculin intradermal reaction is of limited value: negative, it does not eliminate the diagnosis, given the possibility of anergy; positive, it is not a certainty. Finally, it is rarely phlyctenular. In our series, it was positive in 5 of the cases and only positive in 1 in 7 cases in the series of Mazodier et al. on neuromeningeal tuberculosis [18].

The clinical signs found in the literature are similar to those due to pyogenic abscesses [9, 17]. They include focal deficits in 7 cases in our series, headache in 6 cases in our series, vomiting in 4 cases in our series, fever in 5 cases in our series, convulsions in 3 cases in our series, and altered consciousness in 7 cases in our series. The delayed diagnosis would explain these disorders of consciousness. The patients being admitted most often at a very advanced stage. The localizations are also not very specific. They are more frequent in the supra-tentorial stage 7 cases in our series, rare in the posterior fossa 1 case in our series and sometimes multiple 1 case in our study, or sometimes may be associated with another tubercular disease of the central nervous system [19].

The progress made with the contribution of CT scans has made it possible to differentiate tuberculoma and abscess, but few radiological signs make it possible to separate a tuberculous abscess from a pyogenous abscess. Both lesions have a hypodense center surrounded by a high-contrast crown, and significant peripheral edema. Only a thick annular opacification would suggest a tuberculous origin [20].

In CNS tuberculosis, cerebral CT is still the first-line examination to be performed, especially since it is more easily performed, especially in the following situations of the emergency. MRI, a more sensitive examination, should be reserved when CT lesions are not suggestive or when vascular lesions, brain stem localizations, or associated spinal injury are suspected. However, the high cost of this examination and its unavailability in several centers in our country limit its use [11].

The rarity of this form of tuberculosis can lead to lack of knowledge. In the presence of an expansive cerebral process, whether it appears as an avascular mass or surrounded by a ring of increased vascularity, glial tumors, infarction, tuberculoma, pyogenous, or tuberculous abscess cannot be differentiated during imaging
investigations with certainty [14]. Tuberculous etiology is evoked if there is also active visceral tuberculosis, encountered 5 times out of 8 times in our series. The presence of epithelioid and gigantocellular follicles is suggestive of a tubercular process but may be totally absent, and other agents may initiate them [14]. That is, without the demonstration of acid-fast bacilli in situ and their isolation in culture, the diagnosis of cerebral tuberculosis can be omitted. Progression under antituberculosis treatment is not discriminatory diagnostic evidence. Indeed, several observations report the development of tuberculous abscesses under well-conducted specific treatment [21, 22]. In our series, diagnostic confirmation was obtained in all cases by isolation of acid-fast bacilli in pus to direct examination after Ziehl-Neelsen coloration, but the culture was not carried out due to the inadequacy of our technical facilities which did not allow sterile pus to be cultured on direct examination, which could explain the relatively low frequency of our series.

The treatment of tuberculous abscesses is medical-surgical, and in all cases it combines a diagnostic and curative surgical procedure with conventional antituberculosis antibiotic therapy [23]. The medical treatment is based on a quadruple therapy combining isoniazid, rifampicin, ethambutol, and pyrazinamide for 2 to 4 months, followed by dual therapy (isoniazid and rifampicin) for 12 to 18 months [24]. Initiated in all patients in our series, it preceded surgical treatment 5 out of 8 cases in patients with visceral tuberculosis. It was associated in all cases in our series with 15 days of corticosteroid therapy which is comparable to the series of Mazodier et al. who combined corticosteroid therapy with antituberculosis treatment in the 7 patients in their study [18]. Corticotherapy would also find its place alongside antituberculosis antibiotics during CNS tuberculosis. One study shows that it reduces mortality and neurological sequelae in patients with a medium severity picture (confusion, focal signs) [18]; other authors show a benefit in severe cases (coma) [25, 26]. Finally, corticosteroid therapy is believed to reduce the volume of hydrocephalus [26].

Surgically, the treatment of the abscess itself involves two possible techniques: puncture(s)—aspiration(s) with or without stereotactic guidance and excision by craniotomy [9]. The choice of procedure will depend on the general condition of the patient and the size and location of the abscess. In our series, all abscesses were treated by unguided puncture—aspiration using the Cushing’s trocar due to the lack of stereotaxis in our technical platform. The direct approach for excision was performed in 2 patients in our series after recurrence.

The evolution of cerebral tuberculous abscess after surgical treatment is often favorable under antituberculosis treatment continued for at least 18 months, initially associated with corticosteroid therapy [9]. The fatal evolution in our series (2 cases: 1 case of respiratory distress and 1 case of septic shock) and the sequelae (3 cases: 1 case of hemiparesis and 2 cases of epilepsy) would be related to late surgery due to the delay in diagnosis. In our context, this can be explained by the diagnostic wandering that these patients experience in the upstream structures before their arrival in the referral centers, by the under-equipment of the hospitals, and by the financial difficulties that most of these patients experience.

Conclusion
Cerebral tuberculous abscesses are a medical-surgical emergency in bulky forms with mass effect. Despite their rarity, morbi-mortality remains high in the context of developing countries because of diagnostic and therapeutic delay. There is no clinical or radiological evidence that points with certainty to the diagnosis of cerebral abscess of tuberculosis origin. It is therefore always necessary to think about it in the face of favorable ground and evocative radiological signs; the diagnosis of certainty is not always easy, and any delay in diagnosis leads to serious sequelae and mortality; the treatment remains medico-surgical.

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Authors’ contributions
All the authors meet the 4 criteria of the International Committee of Medical Journal Editors (ICMJE): 1. Substantial contributions to research design or methods or to acquisition, analysis, or interpretation of data; 2. Preliminary writing of the article or its critical review involving significant contribution to the intellectual content; 3. Final approval of the version to be published; 4. Commitment to assume accountability for all aspects of the research by ensuring that the issues related to the accuracy or integrity of any part of the work are examined in a manner that appropriate and resolute.

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References
1. De Castro CC, De Barros NG, De Sousa Campos ZM, Cerri GG. CT scans of cranial tuberculosis. Radiol Clin NorthAm. 1995;33:753–69.
2. Paganini H, Gonzalez F, Santander C, Casimir L, Berberian G, Rosanova MT. Tuberculous meningitis in children: clinical features and outcome in 40 cases. Scand J Infect Dis. 2000;32:41–5.

3. Villoria MF, Fortea F, Moreno S, Munoz L, Manero M, Benito C. MR imaging and CT of central nervous system tuberculosis in the patient with AIDS. Radiol Clin N Am. 1995;33:805–20.

4. Elmanmouna E, Souylem MB, Klebs AS, Memou SS, Soumaré O, Salihy SM. Tuberculose cérébrale : à propos de 21 cas. Neurochirurgie. 2019;65:106–44.

5. Mohanti S, Rao CJ. Tuberculous abscess of the brain. Postgrad Med J. 1978;54:678–9.

6. Rouzaud M, Gouaze A, Degiovanni E. Les formes chirurgicales de la tuberculose cérébrale. A propos de trois observations dont un abcès tuberculeux. Sem Hôpitaux Paris. 1971;47:2063–6.

7. Gavazzi G, Bouchard O, Queyrel V, Bosseray A, Leclercq A, Micoud M. Vascularite et miliaire cérébrale d’origine tuberculeuse: aggravation sous traitement adapté. Rev Med Interne. 1998;19:1.

8. Granier H, Robinet G, Guiavarch M, Garo B, Boles JM, Garre M. Abcès multiple d’origine tuberculeuse chez un immunodéprimé. Med Mal Infect. 1991;21:330–1.

9. Mohammedi I, Veber B, Gachot B, Wolff M, Vachon F. Abcès tuberculeux de la fosse postérieure, chez un patient infecté par le VIH. Réan Urg. 1995;4(3):317–9.

10. Bannister C. A tuberculous brain abscess. Case report. J Neurosurg. 1970;33:203–6.

11. Lecuit M, Rogeaux D, Brinquette F, Gentilini M. Tuberculomes intracérébraux au cours de l’infection par le VIH. Épidémiologie et apport de l’IRM. Presse Med. 1994;23:991–5.

12. Mazodier K, Benoit E, Faure V, Rovry C, Gayet S, Seux V, Donnet A, Brouqui P, Diderot P, Schleinitz N, Kaplanski G, Veit V, Harlé J-R. Tuberculose cérébroméningée chez l’adulte séronégatif pour le VIH : à propos de 7 cas. La Revue de Médecine Interne. 2003;24:78–85.

13. David M, Benda R, Benda P. foyer confluent encéphalique de nécrose suppurée tuberculeuse : vérification bactériologique per-opératoire. Rev Neurol. 1954;90:854–6.

14. Decuns P, Garre H, Pascalis G. Abcès froids miliaires du cerveau, du cervelet et du tronc cérébral chez un tuberculeux traité par antibiotiques. Rev Otoneuroophthalmol. 1956;28:250–5.

15. Dooley DP, Carpenter JL, Rademacher JS. Adjunctive corticosteroid therapy for tuberculosis: a critical reappraisal of literature. Clin Infect Dis. 1997;25:872–87.

16. Coyle PK. Glucocorticoids in central nervous system bacterial infection. Arch Neurol. 1999;56:796–801.

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