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Intraventricular Hemorrhage, a Rare Complication of a Cerebral Tuberculoma: Study of a Case in the Neurology Department of the University Hospital of Conakry

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Abstract

Intraventricular haemorrhage (IVH) is an extremely rare and poorly described complication of central nervous system Tuberculosis (CNS-TB). In this study, we report the case of a 42-year-old man who presented with a weakness of the left hemibody with diffuse headache, altered consciousness associated with fever. No notion of contagion was noted. Brain computed tomography (CT) showed a hematoma in the occipital horns of the lateral ventricles with peri-lesional oedema of the right hemisphere; after injection of contrast product (CP), there were ring-shaped contrasts in the occipital horn of the lateral ventricle and the right thalamus. Angioscan showed no aneurysms or vascular anomalies. Chest X-ray showed micronodular opacities less than 1.5 cm in size in relation to miliary. Xpert MTB/RIF of sputum and cerebrospinal fluid (CSF) showed *Mycobacterium tuberculosis* (MT). The patient's course was favourable under antihypertensive and antituberculous treatment. **Conclusion**: This case illustrates the diagnostic difficulties of CNS-TB due to the polymorphism of the clinical and radiological presentation of CNS-TB.

Keywords

Intraventricular Haemorrhage, Tuberculosis, Tuberculoma

1. Introduction

It is estimated that one third of the world's population is infected with TB [1].

While the most common manifestation of TB is pulmonary, CNS-TB is more difficult to diagnose and inherently has a worse outcome than pulmonary TB, especially if treatment is delayed [2]. It occurs in 1% - 5% of all TB patients. It is a major cause of death and disability in developing countries due to difficulties in diagnosis and access to treatment [3]

About 10% of immunocompetent TB patients manifest CNS involvement. The most common form is tuberculous meningitis (about 70% - 80%), followed by intracranial tuberculoma, and spinal arachnoiditis (rare) [4]. Tuberculoma may still account for up to 50% of intracranial mass lesions in undeveloped countries. It is a granuloma (*i.e.*, a focal aggregate of activated macrophages) formed by the inflammatory response to MT infection [5] [6].

Intracranial haemorrhage (ICH) is an extremely rare complication of CNS-TB [7]. ICH centred in the cerebral lobes, basal ganglia, cerebellum, ventricle and suprasellar region and subarachnoid haemorrhage secondary to CNS-TB have been reported [7]. A case of periventricular haemorrhage has also been reported [8]. Hemorrhage within tuberculoma is even rarer, with only 2 cases published [9] [10]. In this study, we report a case of intraventricular haemorrhage revealing a CNS tuberculoma associated with miliary tuberculosis.

2. Patient and Observation

Our case was a 42-year-old man who was admitted to the department for diffuse headache, fever, and motor deficit of the left hemibody evolving for 14 days. There were no reports of contagious or autoimmune diseases or cardiovascular comorbidities.

Clinical examination: the patient had a fever of 39˚C, a blood pressure of 180/100mmhg, a heart rate of 95 beats per minute. The neurological examination showed temporo-spatial disorientation with a Glasgow score of 13/15, hemiparesis and abolition of osteotendinous reflexes in the left hemisphere, there were no meningeal signs. Cardiac auscultation was normal. At the pulmonary level there were crackling rales in both lung areas.

Diagnostic approach: The patient underwent a CT scan which showed spontaneous hyperdensity in the occipital horns of the lateral ventricles with peri-lesional oedema of the right hemisphere (**Figure 1**); after injection of CP there were ring-shaped contrasts in the occipital horn of the lateral ventricle and in the right thalamus and subfalcoral involvement (**Figure 2**). Chest X-ray showed micronodular opacities less than 1.5 mm in size in both lung fields in relation to miliary (**Figure 3**). Angioscan showed no aneurysms or vascular anomalies. The electrocardiogram was normal. The haemogram showed a microcytic anaemia with a haemoglobin level of 11 g/l (VGM = 75 fl), a hyperleukocytosis (16,000/mm^3) with a lymphocytic predominance (81%) (**Table 1**). An acceleration of erythrocyte sedimentation rate (45 mm at the first hour and 65 mn at the second hour) and an elevated C-reactive protein were noticed. There was no coagulation disorder, nor hepatic or renal impairment. The retroviral and hepatic serologies and the parasitology of the stools were negative. Given the cha-

racteristics and location of the haematoma, a lumbar puncture with analysis of the cerebrospinal fluid (CSF) was carried out on the patient and revealed a haematic fluid, a protein level of 3.6 g/l, high hypercellularity (1007/mm^3) with a lymphocytic predominance (89%), and a hypoglucorrhachia (0.25 g/l). The results of the lumbar puncture analysis are shown in Table 2. Xpert MTB/RIF of CSF and sputum showed MT, no rifampicin resistance was detected. Direct examination and culture of the CSF did not reveal any germs.

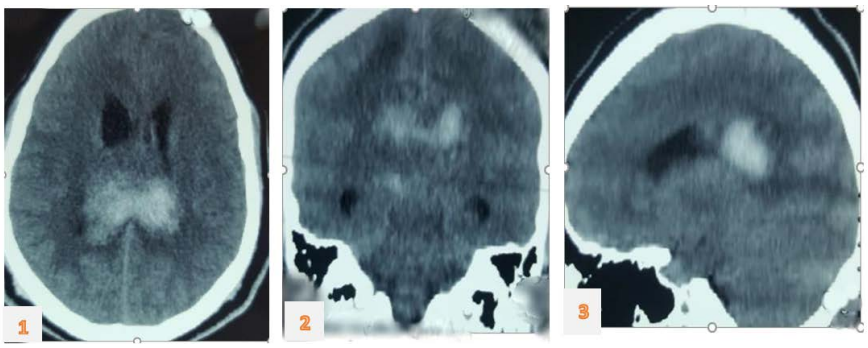

Figure 1. Brain CT without CP injection: spontaneous hyper density at the occipital horns of the lateral ventricles with peri-lesional oedema of the right hemisphere.

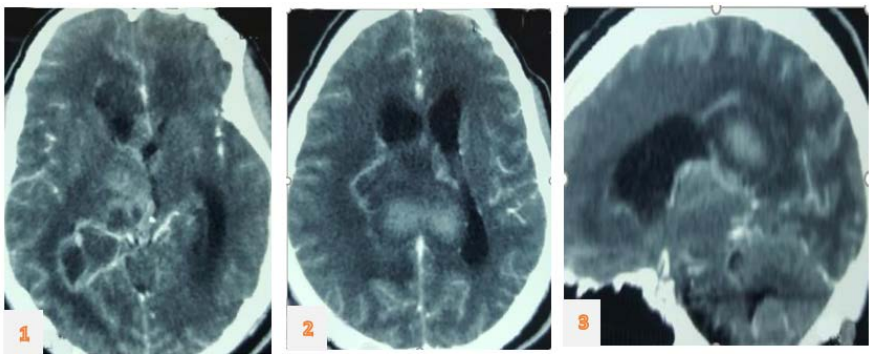

Figure 2. After injection of CP: Multiple annular contrasts in the right occipital horn and the right thalamus.

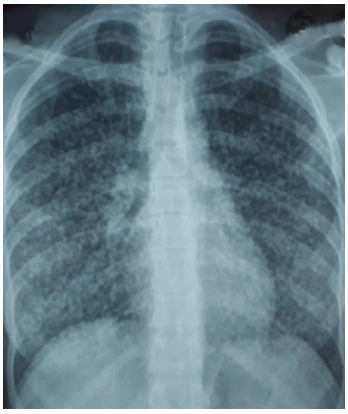

Figure 3. Chest X-ray: Micronodular opacities less than 1.5 centimetres in diameter disseminated in both lung areas: miliary tuberculosis.

Table 1. Results of the patient's haemogram.

Parameters	Patient	Normal
Erythrocyte count		
RBC	3.4 millions/ml	4.6 - 6
Haemoglobin	11 g/dl	13 - 18
MCV	75 fl	85 - 95
MCHC	30 pg	28 - 32
Leukocyte count		
White Blood Cells	16,000/mm^3	4000 - 10,000
Lymphocytes	81%	20 - 40
Neutrophils	17%	45 - 70
Eosinophils	00%	1 - 5
Basophils	00%	0 - 3
Monocytes	2%	2 - 8

Table 2. Results of cerebrospinal fluid analysis.

Parameters	Patient	Normal
Macroscopic appearance	Haematic	Clear
Microscopic appearance		
Cellularity		<5
Red blood cells	3100/mm^3	
White blood cells	1007/mm^3	
Lymphocytes	89	
Neutrophils	8	
Proteins	3.6 g/l	<0.45 g/l
Glucose	0.25 g/l (1.05 g/l)	1/2 of the glycemia
Germs	**Absent**	**Absent**
Xpert MTB/ RIF	**Positive**	**Negative**

Therapeutic approaches: Antihypertensive treatment was instituted (Nicardipine with electric syringe pump), anti-tuberculosis treatment included Rifampicin, Isoniazid, Ethambutol and Pyrazinamide. Cerebral oedema was treated with prednisolone 1 mg/kg/day. The patient was treated with paracetamol for headache and fever.

Course and follow-up: The patient's evolution was marked by a disappearance of the fever and headache after one week of treatment. Physiotherapy allowed a complete recovery of the motor Weakness.

At one month, the patient showed improvement in inflammatory and biological markers. He was recommended to continue the anti-tuberculosis treatment for 12 months. Brain imaging at 5 months showed disappearance of the tuberculomas and discrete ventricular dilatation (**Figure 4**).

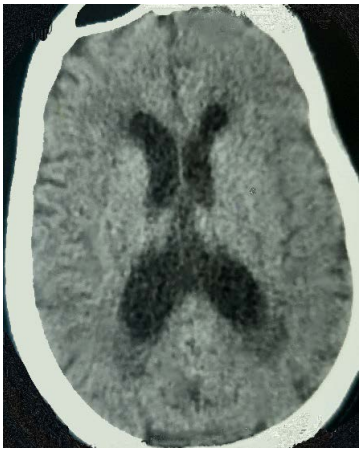

Figure 4. Brain CT after 5 months showed disappearance of the tuberculomas and discrete ventricular dilatation.

3. Discussion

CNS-TB is rare and represents the most severe form of systemic TB due to severe complications, high mortality and disabling sequelae [2]. This is the first documented case of intracerebral haemorrhage in sub-Saharan Africa secondary to CNS-TB. Only few cases have been published worldwide to date [7] [8] [9] [10]. Our case was a young man with no history or notion of tuberculosis infection; CNS-TB is known to occur in all age groups, but 60% - 70% of patients are younger than 20 years [8].

Tuberculomas occur when tubercles in the brain parenchyma enlarge without rupturing into the subarachnoid space. They usually occur in the absence of tuberculous meningitis, but may occur as a consequence of tuberculous meningitis, due to the extension of the infection through the CSF into the adjacent parenchyma via the cortical veins or Virchow-Robin spaces [11]. As tuberculomas are avascular granulomatous formations consisting of mixed epithelioid and giant cells with a predominance of lymphocytes around a central zone of caseous necrosis, intra-tuberculoma haemorrhage is extremely rare [7]. Indeed, CNS-TB often leads to vasculitis and an inflammatory reaction of the intracranial vessels [12]. Studies have shown that aneurysms and venulitis adjacent to tuberculomas can rupture and lead to intracranial haemorrhage [13]. In the absence of aneurysmal lesions on angioscan, we believe that our patient's HIV could be related to rupture of the inflamed vessels. One of the mechanisms of HIV that has also been suggested is invasion of the adjacent vessel wall by the tuberculoma leading to aneurysm formation or venous dilatation and fibrinoid necrosis resulting in vascular rupture [7]. In addition, the passage of MT into the brain parenchyma by the haematogenous route through the vessels of the choroid plexuses and their subsequent colonisation in the periventricular zone could explain the localisation of tuberculomas. The destruction of the choroid plexus vessels would lead to intraventricular haemorrhage.

Clinically, our patient presented with left hemiparesis with fever, altered con-

sciousness. Many authors have reported that patients with tuberculoma usually present with headache, seizures, focal neurological deficit, and features of elevated intracranial pressure. Subtentorial tuberculomas may present with brainstem involvement, cerebellar symptoms and cranial nerve palsies [14]. The diagnosis of tuberculoma has been made on the basis of periventricular CT lesions with annular contrast (cocardial appearance). This entity poses a problem of differential diagnosis with brain tumours, primary lymphoma of the central nervous system, pyogenic abscess, fungal infections, neurocysticercosis and toxoplasmosis [15]. The presence of a miliary tuberculosis and the detection of acid-fast bacilli (AFB) in CSF and sputum helped to make the diagnosis of cerebral tuberculoma. Until recently, a definitive diagnosis could only be made after growth and identification of the MT in a culture, which could take 4 - 8 weeks and could give a false negative in 15% - 75% of cases [16]. Currently, polymerase chain reaction (PCR) in CSF allows the diagnosis of CNS-TB with higher sensitivity than microscopy and cultures [16].

This patient was treated according to the British Infection Society guidelines, which state that the initial treatment regimen for CNS-TB is a combination of isoniazid, rifampicin and pyrazinamide plus ethambutol (or streptomycin or fluoroquinolone) for 2 - 3 months, followed by isoniazid and rifampicin for up to 12 months of consolidation therapy. If necessary, the total duration of treatment can be extended to 18 months [17]. The use of glucocorticoids such as dexamethasone has been described to inhibit inflammation, reduce cerebral oedema [18].

Honnorat *et al.* report that rarely a paradoxical response with an increase in the size of the lesion or the appearance of new lesions or stationary images can occur while the patient is on a well-conducted medical treatment [19].

When the clinical evolution is successful, the continuation of anti-tuberculosis treatment is justified and a radiological check-up a few weeks later will show a decrease in the volume of the lesions. If the clinical course is poor, it is either a diagnostic error or resistance to treatment [20].

4. Conclusion

The present case is evidence of the polymorphism of the clinical presentation of CNS-TB. In the presence of intracerebral haemorrhage in an infectious setting, CNS infection with MT should be suspected. In the absence of a lumbar puncture, a pulmonary localization of the infection may aid in the diagnosis.

Authors' Contributions

All authors made contributions to the design, analysis and interpretation of the information and gave their final approval to the version to be published.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] World Health Organization (2016) Global Tuberculosis Report 2016. WHO, Geneva.

[2] Wilkinson, R.J., Rohlwink, U., Misra, U.K., *et al.* (2017) Tuberculous Meningitis. *Nature Reviews Neurology*, **13**, 581-598. https://doi.org/10.1038/nrneurol.2017.120

[3] Mayasari, E. and Sufida (2016) Multiple Intracranial Tuberculomas: Diagnosis Difficulties in a Clinical Case. *Procedia Chemistry*, **18**, 199-204. https://doi.org/10.1016/j.proche.2016.01.031

[4] Schaaf, H.S. and Zumla, A. (2009) Tuberculosis: A Comprehensive Clinical Reference. Elsevier, Amsterdam, 313-388.

[5] Lucas, S.B. (2008) Part 2, Histopathology. Ormerod P. Part 3, Non-Respiratory Tuberculosis. In: Davies, P.D.O., Barnes, P.F. and Gordon, S.B., Eds., *Clinical Tuberculosis*, 4th Edition, Hodder Arnold, London, 105-188. https://doi.org/10.1201/b13468-10

[6] Al-Kawi, M.Z. (2004) Tuberculosis of the Central Nervous System. In: Madkour, M.M., Ed., *Tuberculosis*, Springer-Verlag, Berlin, 535-543. https://doi.org/10.1007/978-3-642-18937-1_32

[7] Schaller, M.A., Wicke, F., Foerch, C. and Weidauer, S. (2019) Central Nervous System Tuberculosis: Etiology, Clinical Manifestations and Neuroradiological Features. *Clinical Neuroradiology*, **29**, 3-18. https://doi.org/10.1007/s00062-018-0726-9

[8] Liu, Y. and Chen, Y. (2020) Unusual Periventricular Hemorrhage as the Initial Manifestation of Central Nervous System Tuberculosis. *World Neurosurgery*, **142**, 371-374. https://doi.org/10.1016/j.wneu.2020.06.193

[9] Deogaonkar, M., De, R., Sil, K. and Das, S. (2006) Pituitary Tuberculosis Presenting as Pituitary Apoplexy. *International Journal of Infectious Diseases*, **10**, 338-339. https://doi.org/10.1016/j.ijid.2005.05.008

[10] Mittal, P., Dua, S., Saggar, K. and Gupta, K. (2010) Magnetic Resonance Findings in Sellar and Suprasellar Tuberculoma with Hemorrhage. *Surgical Neurology International*, **1**, 73.

[11] Kioumeher, F., Dadsetan, M.R., Rooholamini, S.A., *et al.* (1994) Central Nervous System Tuberculosis: MRI. *Neuroradiology*, **36**, 93-96. https://doi.org/10.1007/BF00588067

[12] Leonard, J.M. and Des Prez, R.M. (1990) Tuberculous Meningitis. *Infectious Disease Clinics of North America*, **4**, 769-787. https://doi.org/10.1016/S0891-5520(20)30376-7

[13] Saraf, R. and Limaye, U. (2013) Ruptured Intracranial Tubercular Infectious Aneurysm Secondary to a Tuberculoma and Its Endovascular Management. *British Journal of Neurosurgery*, **27**, 243-245. https://doi.org/10.3109/02688697.2012.717986

[14] Rajshekhar, V. and Chandy, M.J. (1997) Tuberculomas Presenting as Isolated Intrinsic Brain Stem Masses. *British Journal of Neurosurgery*, **11**, 127-133. https://doi.org/10.1080/02688699746474

[15] Luthra, G., Parihar, A., Nath, K., *et al.* (2007) Comparative Evaluation of Fungal, Tubercular, and Pyogenic Brain Abscesses with Conventional and Diffusion MR Imaging and Proton MR Spectroscopy. *AJNR American Journal of Neuroradiology*, **28**, 1332-1338.

[16] Takahashi, T., Tamura, M., Takasu, T. and Kamei, S. (2013) Current Advancement of the PCR-Based Molecular Diagnosis for Tuberculous Meningitis. *Rinsho Shinkeigaku*, **53**, 1187-1190. https://doi.org/10.5692/clinicalneurol.53.1187

[17] Chen, W., Huang, L., Tang, Q., *et al.* (2020) Progress on Diagnosis and Treatment of Central Nervous System Tuberculosis. *Radiology of Infectious Diseases*, **7**, 160-169. https://doi.org/10.1016/j.jrid.2020.07.005

[18] Prasad, K., Singh, M.B. and Ryan, H. (2016) Corticosteroids for Managing Tuberculous Meningitis. *Cochrane Database of Systematic Reviews*, **4**, 2244-2248. https://doi.org/10.1002/14651858.CD002244.pub4

[19] Honnorat, E., De Broucker, T., Mailles, A. and Stahl, J.P. (2013) Encephalitis Due to *Mycobacterium tuberculosis* in France. *Médecine e Maladies Infectieuses*, **43**, 230-238. https://doi.org/10.1016/j.medmal.2013.05.003

[20] Chaudhary, V., Bano, S. and Garga, U.C. (2017) Central Nervous System Tuberculosis: An Imaging Perspective. *Canadian Association of Radiologists Journal*, **68**, 161-170. https://doi.org/10.1016/j.carj.2016.10.007