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Case Report

Tuberculous Meningitis Complicated Without Deep Sylvian Cerebral Infarction: About an Immunocompetent Patient with HIV-AIDS

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Abstract

Introduction: Neuromeningeal tuberculosis is the most common form of extra-pulmonary tuberculosis in developing countries. It is the most serious form of Mycobacterium tuberculosis infection. Vascular complications of tuberculous meningitis involve functional prognosis of patients. Observation: In our daily practice, we frequently encounter cases of cerebral infarction associated with neuromeningeal tuberculosis in the infectious etiological balance. We report the case of an HIV immunocompetent patient hospitalized for neuromeningeal tuberculosis complicated with ischemic stroke. It was a 39-year-old patient, right-handed, admitted to the Neurology department of University Hospital of Cocody in whom the diagnosis of neuromeningeal tuberculosis was retained. She received treatment for tuberculosis in emergency. On the seventh day of hospitalization, she suddenly presented with a massive motor deficit of the left hemibody. The MRI performed was compatible with meningoencephalitis associated with recent deep right Sylvan ischemia foci. The evolution was marked by partial recovery of the left hemiplegia, right retro-bulbar neuritis, and iatrogenic hepatic cytolysis. Comments: Tuberculous meningitis remains today one of the most serious neurological conditions involving the vital and functional prognosis of patients. Association of cerebral infarction and tuberculous meningitis is of variable occurrence. There are reported cases in which cerebral infarction is a complication of tuberculous meningitis. On the other hand, the discovery of tuberculous meningitis can be observed in the assessment of a cerebral infarction in young subjects. Tuberculous meningovascularitis remains a rare condition in immunocompetent patients for HIVAIDS. It results from arterial occlusion by thrombosing and obliterating endarteritis. It represents a medical emergency requiring starting anti-tuberculosis treatment. The evolution depends on the precocity of the diagnosis and the speed of treatment. Conclusion: The sudden appearance of a neurological deficit in àpatient with tuberculous meningitis even under specific treatment should suggest an ischemic stroke, especially if the symptomatology respects an anatomo-functional vascular systematization.

Keywords

Tuberculous Meningitis, Cerebral Infarction, Meningovasculitis, Immunocompetent HIV

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1. Introduction

Neuromeningeal tuberculosis represents a diagnostic and therapeutic emergency [1]. It associates a meningeal syndrome and an encephalitic syndrome. Tuberculous meningovascularitis is one of the vascular complications responsible for ischemic stroke [2, 3]. It is important to know this affection because it can raise questions about the diagnosis and the effectiveness of anti-tuberculous treatment. It is a condition resulting from obliterating arteritis involving the functional prognosis of patients with tuberculous meningitis [4]. Through this observation, the authors highlight the rarity of tuberculous meningovascularitis in HIVimmunocompetent patients and the clinical particularity of the onset mode of this serious and disabling condition.

2. Observation (File 040/22/1)

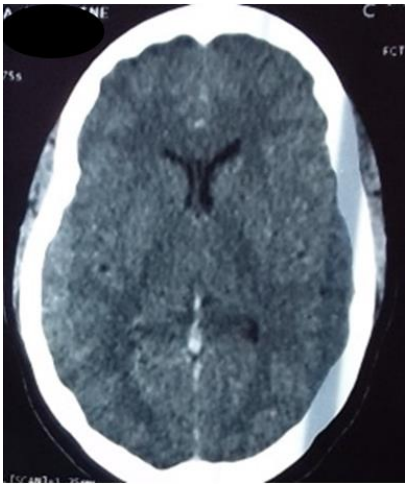

Figure 1. *Appearance of radiological meningitis with contrast enhancement of the leptomeningeal spaces.*

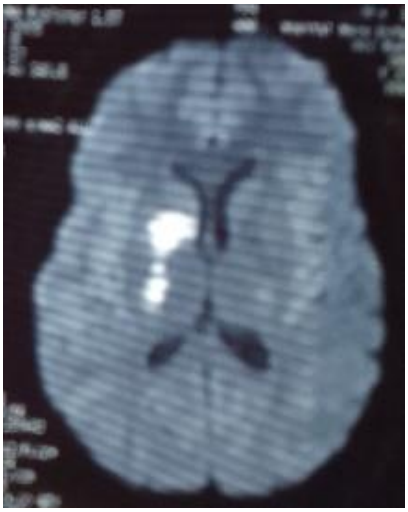

Figure 2. *DWI hyper signals at the level of the perforators of the right middle cerebral artery with damage to the basal ganglia.*

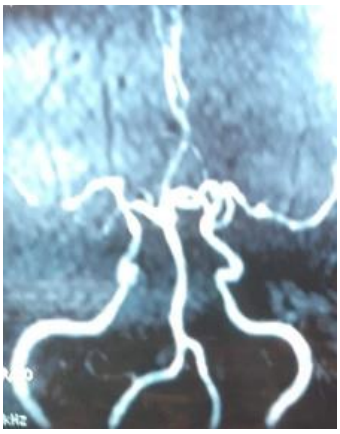

Figure 3. *Irregularity of the proximal portion of the right middle cerebral artery and anterior cerebral artery related to vasculitis.*

Mrs. G. R., a 39-year-old patient with no medical history, was admitted to the Neurology department for febrile generalized tonic-clonic seizures. The clinical examination on admission revealed an infectious syndrome, a meningeal syndrome, right oculomotor paralysis due to damage of the 3rd and 6th pair of cranial nerves and an anamnestic comitial syndrome. The brain scan showed radiological meningitis with contrast enhancement of the leptomeninges without parenchymal lesion or signs of brain involvement. The lumbar puncture revealed lymphocytic meningitis (687 elements/mm^3 with 82% of lymphocytes) with hyperproteinorachia (2.34g/l) and hypoglycorachia (0.11g/l). The presence of acid-alcoholo-fast bacilli and Mycobacterium tuberculosis were detected at the Xpert gene of cerebrospinal fluid (CSF). The diagnosis of neuromeningeal tuberculosis was retained. Our patient received five tablets of RHZE (Rifampicin Isoniazid Pyrazinamide Ethambutol) combined with corticosteroid therapy, antiplatelet and motor physiotherapy. However, during hospitalization, her condition got worse with sudden onset of left hemiplegia associated with confessional syndrome. Brain computed tomography revealed leptomeningitis (Figure 1). Brain MRI confirmed hypothesis of recent ischemia with multiple foci of deep infarction in the territory of the right sylvian artery (Figures 2, 3). The exhaustive etiological assessment of the other causes of cerebral infarction was normal (cardiac ultrasound, ultrasound of the supra-aortic trunks, EKG and lipid assessment are normal). At three weeks of the antituberculous treatment, the evaluation of the efficacy of the treatment made it possible to note apyrexia with total disappearance of the meningeal syndrome, a partial improvement in the cytology of the CSF at 30 lymphocytes and of the proteinorachia at 1.5 g/l associated with normalization of glycorachia. The clinical monitoring made it possible to diagnose retrobulbar optic neuritis with oculalgia and visual blurring associated with ophthalmoplegia of the right eye. In addition, she presented biological abnormalities in

favor of hepatic cytolysis (ASAT 127UI/l, ALAT 226UI/l) iatrogenic linked to anti-tuberculous drugs. After a collegial decision, the anti-tuberculous treatment was readjusted, maintaining five HR (Rifampicin Isoniazid) tablets, and introducing ofloxacin at a dosage of 200 mg every 12 hours. The evolution was favorable with slow recovery of the motor deficit and other neurological symptoms.

3. Discussion

3.1. Epidemiology

In Côte d'Ivoire, as in most African countries, tuberculosis is endemic with a high susceptibility of black people to developing tuberculosis [5]. It is a major public health problem. In Côte d'Ivoire, a study conducted on neurotuberculosis in a hospital environment did not make it possible to estimate the frequency due to the small sampling, the poor technical facilities, and the difficulties of access to expensive microbiological analyses. Worldwide, the incidence of tuberculosis was estimated around twelve million of new cases in 2015, i.e., 30% concerning the central nervous system location. The world's first cases of tuberculous meningovascularitis were observed in the 17th century. We have few data in the literature related to tuberculous meningovacularitis in HIV-AIDS immunocompetent field. It affects both men and women equally. The target age group remains the population of young subjects in full activity. *Hsaini* and al reported four cases of tuberculous meningovascularitis with three men for one woman, all aged between 14 and 53 years old as in our observation [5]. Tuberculous meningovascularitis remains a pathology of the young subject. It can occur both in immunocompromised subjects and in immunocompetent HIV patients. However, its onset in immunocompetent patients is not much listed in literature. It is therefore a rare form of meningovascularitis that can occur in a patient with no vascular risk factor. Recent scientific work has established a possible link between the discovery of a factor called 'growth endothelial vascular factor' which remains high in the vascular involvement of tuberculous meningitis [6]. The clinical presentation remains one of the rare modes of onset of tuberculous meningovascularitis beginning with meningitis with damage to the cranial nerves and complicated by cerebral infarction of the deep territory (MCA, ICA, intracranial ICA and sometimes vertebrobasilar territory) [7].

3.2. Clinical Signs

Neuromeningeal tuberculosis is responsible for several neurological manifestations. Its clinical polymorphism makes positive diagnosis difficult. It may be meningitis and/or meningoencephalitis, medullary or radicular involvement, an expanding intracranial or intramedullary lesion. In our clinical case, the involvement of the cranial nerves reflected that of a preferential localization of Koch's bacillus at the base of the skull, very pathognomonic for tuberculous meningitis [6]. We should think about it and evoke a tuberculous origin in immunocompetent or dysimmune subject to HIV. Vascular involvement remains not much described in literature. The neurological signs are related to the arterial territory concerned. Most often the motor deficit that occurs suddenly remains in the foreground. Moreover, this deficit can be associated either with motor aphasia or with a minor hemisphere syndrome depending on whether the location of the lesion is on the left or right hemisphere. In our reported case, Mrs. G.R. presented with massive left hemiplegia associated with confusional syndrome probably related to the involvement of the minor hemisphere.

3.3. Paraclinical Signs

In daily practice, a meningeal syndrome requires the realization of a lumbar puncture with analysis of CSF in emergency. Recommendations stipulate that any clear-liquid meningitis associating hypoglycorachia and hyperproteinorachia must be considered as tuberculous origin until proven otherwise [8]. Treatment is therefore started urgently without waiting for the results of CSF analysis. Nowadays, the improvement of the technical platform allows the detection of Koch's Bacillus by polymerase chain reaction technique correlated with the culture to eliminate false positives and negatives [2]. Brain imaging has revolutionized the diagnosis of neuromeningeal tuberculosis. Cerebral MRI remains the most appropriate examination to detect small areas of ischemia secondary to vasculitis [9]. It is an aid to positive diagnosis but also allows the monitoring of cerebral parenchymal lesions. It is typically cerebral infarction of the middle cerebral artery or its perforators (internal capsule), identical location also observed in our patient [10]. It may also involve the basal ganglia, which explains the involvement of the gray nuclei highlighted on neurological examination in our patient. It is also possible but rare to observe a hemorrhagic infarction or intraventricular hemorrhage by rupture of aneurysm.

3.4. Treatment

Treatment of tuberculous meningovascularitis is identical to that of other cerebral tuberculous involvement. In Côte d'Ivoire, the therapeutic strategy follows international recommendations with the use of anti-tuberculosis drugs according to the therapeutic regimen proposed by the WHO and established as such in our patient [11]. Due to the immunopathological mechanism responsible for either thrombosis or stenosis of the vascular lumen, it is recommended to combine antiplatelet therapy and corticosteroid therapy with antituberculous treatment in inflammatory vascular disorders [10, 11].

3.5. Evolution – Prognosis

Neuromeningeal damage, complicated or not with cerebral infarction is responsible for high morbidity and mortality that is life-threatening. The functional prognosis will depend on the occurrence of neurological sequelae. The disappearance of sudden onset of neurological manifestations should lead to search for a vascular cause. Hence the interest of looking for vascular damage in the classic evolution of tuberculous meningitis in front of the occurrence of a sudden motor deficit. Evolution depends on the rapidity of the diagnosis and the urgent establishment of the specific treatment. Ophthalmological and hepatic examination is necessary for the monitoring of treatment with Ethambutol and Isoniazid, respectively [7]. In our clinical case, we noted the occurrence of toxic retrobulbar optic neuritis and hepatic cytolysis. The functional prognosis will depend on irreversible ischemic parenchymal and vascular complications causing disabling motor sequelae [12, 13].

4. Conclusion

The association of tuberculous meningitis and cerebral infarction produces a picture of tuberculous meningovasculariti. It remains a less frequent but current association in tropicle regions. The vascular complication is possible during tuberculous meningitis even treated early and quickly. It is responsible for severe, heavy and disabling sequelae, hence the therapeutic urgency of appropriate care.

Conflicts of Interest

Authors declare no conflict of interest.

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