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Case report

Tuberculous limbic encephalitis: A case report

Encéphalite limbique tuberculeuse : à propos d'une observation

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1. Introduction

Tuberculosis is the one of the most common infectious etiologies of encephalitis in adults [1]. However, limbic encephalitis (LE) is an unusual clinical manifestation of the tuberculosis which is a clinical situation never described previously in the literature to our knowledge. We report herein the first case of tuberculous LE in a 53-year-old woman who initially presented psychiatric disorders and cognitive disorders.

2. Case report

A 53-year-old woman was admitted to our department for behavioral type of irritability and aggression associated with temporospatial disorientation and weight loss that occurred 45 days before consultation. It was noted in her past medical history that she had a total thyroidectomy for follicular adenoma 3 years ago with replacement therapy with Levothyroxine, and 1 year ago cholecystectomy for gallstones. On admission, neurological examination found a euphoric woman, cooperative with fluent language. She had a temporospatial disorientation and cognitive disorders especially free recall deficit and learning with a Mini-Mental State Examination (MMSE) of Folstein of 11/30 which is pathological. The brain scan showed a hypodense lesion in the right medial temporal lobe. The brain magnetic resonance imaging (MRI) showed on FLAIR sequences a bilateral basifrontal and mesiotemporal hyperintensities interesting the right insula, associated with nodular contrast enhancement of these same lesions and an enhancement and nodular leptomeningeal thickening of the external part of the right temporal lobe on gadolinium enhanced T1-weigthed images (Fig. 1). The study of cerebrospinal fluid (CSF) revealed meningitis with 29 white blood cells of which 80% were lymphocytes, a protein level of 1.37 g/L, low chloride of 108 mmol/L and a ratio CSF glucose/serum glucose of 0.15. The cultures were negative as well as the search of mycobacterium tuberculosis (MTB) in the CSF. The systemic immunological test (antinuclear antibodies, anti-dsDNA, anti-Sm, anti-SSA, anti-SSB, antiphospholipid, ANCA) and research of antineuronal antibodies (anti-NMDAR antibodies, anti-VGKC, anti-AMPA, anti-Hu, anti-Yo, anti-CV2, anti-Ma2, antiamphiphysine) were negative. The dosage of the angiotensin-converting enzyme was normal. The histological study of the accessory salivary gland biopsy was negative. The polymerase-chain-reaction of the herpes simplex virus (HSV-PCR) was negative. The polymerase-chain-reaction of the mycobacterium tuberculosis (MTB-PCR) and quantification of adenosine deaminase (ADA) were not performed due to limited access to these biological investigations in our institution. Serological tests for syphilis, Lyme disease, human immunodeficiency virus (HIV), hepatitis C and hepatitis B were also negative. The cervico-thoraco-abdominopelvic CT scan was normal. At the end of these analyses, the diagnosis

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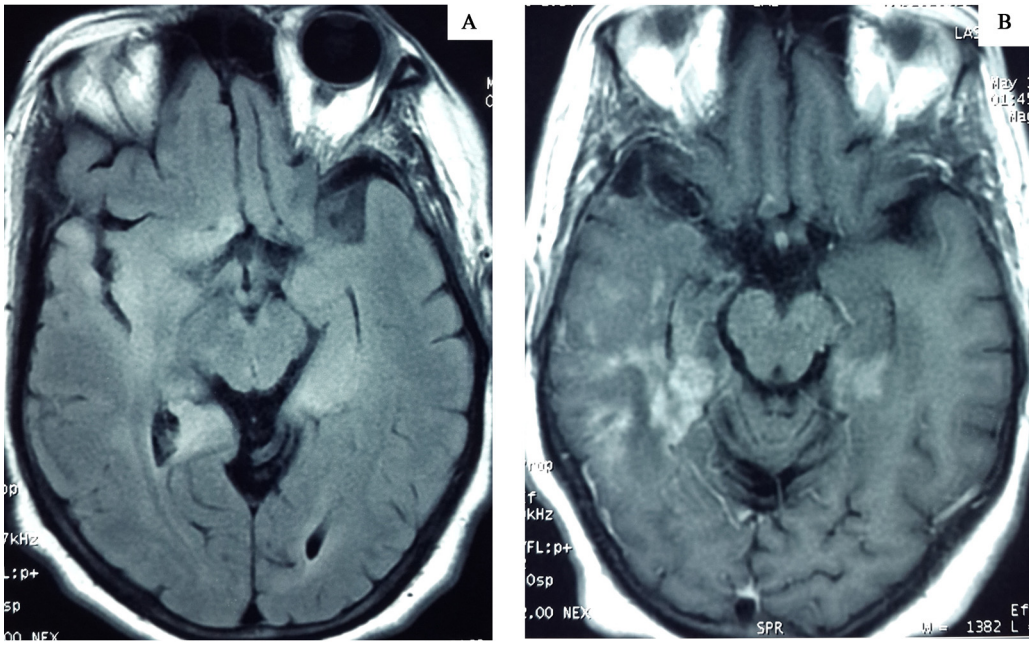

Fig. 1. Brain MRI showing on FLAIR sequences (A) bilateral basifrontal and mesiotemporal hyperintensities interesting the right insula, associated with nodular contrast enhancement of these same lesions and an enhancement and nodular leptomeningeal thickening of the external part of the right temporal lobe on gadolinium enhanced T1-weigthed images (B).
IRM cérébrale montrant sur les séquences FLAIR (A) des hypersignaux en basifrontal et mésiotemporal bilatéral intéressant l'insula droit, avec un rehaussement nodulaire de ces mêmes lésions et un épaississement et rehaussement leptoméningé nodulaire temporal externe droit sur les séquences T1 après injection de gadolinium (B).

of tuberculous meningitis complicated with limbic encephalitis was considered according to the diagnostic criteria of the consensus case definitions for tuberculous meningitis [2] (Table 1). Antituberculosis chemotherapy was started for nine months associated with adjuvant oral corticosteroid therapy for eight weeks. Three weeks after the onset of antituberculosis chemotherapy, the patient had improved her cognitive status with a MMSE at 25/30 indicating a good clinical response to treatment although biological and radiological control were not performed at this time. After 3 months, the patient was in complete clinical remission with a normal CSF study.

3. Discussion

LE is histologically defined as an inflammation – degeneration of limbic structures [3]. It is clinically characterized by an acute or subacute onset of short-term memory disorders, psychiatric disorders, confusion and temporal lobe epilepsy [4]. The brain MRI plays an important role in the diagnosis of LE when it's positive by highlighting on T2 and FLAIR sequences hyperintensities in the limbic regions such as the internal part of the temporal lobe, hippocampus and amygdala, cingulate gyrus, fornix and hypothalamus [5].

Herein, we describe the case of LE associated with tuberculous meningitis which is an unusual clinical situation. The usual infectious causes of LE and commonly described in the literature are herpes simplex virus and syphilis [5,6]. However, tuberculosis is a rare clinical entity as etiology of LE. To date, no cases of tuberculous LE have been reported in the literature to our knowledge. Our observation is the first case to be reported in the literature on tuberculous meningitis with LE as the first manifestation. The diagnosis of neurotuberculosis was considered based on diagnostic criteria of the consensus case definitions for tuberculous meningitis [2] with a good clinical response under antituberculosis chemotherapy and after ruling out other etiologies with similar clinical pictures (syphilis, HSV, paraneoplastic causes, sarcoidosis, lupus, Wegener).

The clinical presentation of the present case was marked by psychiatric disorders and cognitive disorders that are the usual manifestations of LE. In comparison with the clinical picture of other infectious causes reported of LE, there are no particular clinical differences. However, the radiological findings of the present case are different from those of other infectious causes by the presence of nodular lesions in the limbic structures associated with an enhancement and nodular leptomeningeal thickening which are characteristic radiologic manifestations of the neurotuberculosis after ruling out sarcoidosis and Wegener.

An early diagnosis of neurotuberculosis and an appropriate antituberculosis chemotherapy associated with an oral corticosteroid therapy lead to rapid and remarkable clinical improvement.

4. Conclusion

In conclusion, our observation shows herein the importance of considering tuberculosis among etiologies of LE, which

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