

Neuromeningeal Tuberculosis in Immunocompromised Patients: A Case Report and Review of the Literature

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Abstract

Case Report

Neuromeningeal tuberculosis (NM-TB) is one of the most severe extrapulmonary forms of tuberculosis, particularly in immunocompromised patients, where it is characterized by an atypical clinical presentation, difficult diagnosis, and a poor prognosis. We report the case of an immunocompromised patient with complicated neuromeningeal TB, and discuss the diagnostic and therapeutic features in light of recent data in the literature.

Keywords: Neuromeningeal tuberculosis, immunosuppression, tuberculous meningitis, brain MRI, literature review.

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INTRODUCTION

Tuberculosis (TB) remains a major public health problem, responsible for more than 10 million cases and 1.3 million deaths per year according to the WHO. Neuromeningeal involvement, which represents 5 to 10% of extrapulmonary locations, is the most feared due to its severity and frequent sequelae. In immunocompromised individuals, particularly HIV-infected patients, transplant recipients, or those on immunosuppressants, the incidence is higher, symptoms are often atypical, and mortality is increased.

We report the case of an immunocompromised patient with complicated neuromeningeal TB, illustrating the diagnostic difficulties and the complexity of management.

CLINICAL OBSERVATION

A 45-year-old patient with HIV at the AIDS stage, with a high viral load and a collapsed CD4 count, was admitted for headaches that had been developing for three weeks, associated with moderate fever, vomiting and a progressive alteration in consciousness.

He presented a clinical picture suggestive of complicated tuberculous meningitis, associating a fever at 38.5°C, marked meningeal stiffness, partial paralysis of the left VI and an alteration of consciousness (Glasgow score at 10/15). The biological tests highlighted a significant inflammatory syndrome (CRP: 85 mg/L, ESR: 70 mm/h) and hyponatremia at 126 mmol/L. The analysis of the cerebrospinal fluid showed a clear fluid, hyperproteinorachia (1.2 g/L), hypoglycorachia and lymphocytic pleocytosis (250 cells/mm³). The search for AFB was negative but the GeneXpert MTB/RIF PCR confirmed the presence of *Mycobacterium tuberculosis* sensitive to rifampicin.

Brain MRI revealed diffuse basilar meningeal enhancement, the presence of multiple intraparenchymal tuberculomas, communicating hydrocephalus and bilateral capsulothalamic infarcts. The diagnosis of complicated tuberculous meningitis was thus retained.

The patient was placed on quadruple anti-tuberculosis treatment combining Isoniazid, rifampicin, pyrazinamide and ethambutol, supplemented by adjuvant corticosteroid therapy with dexamethasone (0.4 mg/kg/day) and the placement of a ventriculoperitoneal shunt for the management of hydrocephalus.

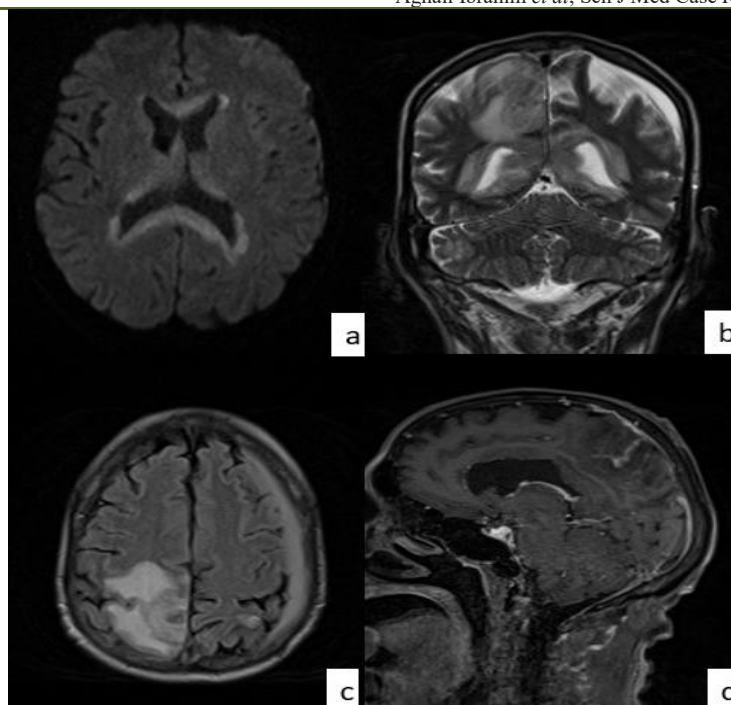


Figure a: Axial section in Diffusion sequence: Showing a restriction of water movements in the periventricular region in relation to septic ventriculitis

Figure b: Coronal section in T2 sequence: Showing hyper signals in the area affecting the right paracentral lobule and in the periventricular region

Fig c: Axial section in FLAIR sequence: Showing a hyper intense lesion of the right paracentral lobule

Fig. d: T1 sequence section after injection of PDC: Showing leptomeningeal contrast uptake at the level of the lesion of the right paracentral lobule

DISCUSSION

Neuromeningeal TB develops by hematogenous dissemination from a pulmonary or lymphatic focus. In immunocompromised patients, progression is more rapid and symptoms are often atypical. [1]

Biological diagnosis: typical CSF but low sensitivity of AFB (<20%). Molecular tests (GeneXpert MTB/RIF, Ultra) have become essential. [2]

Contribution of imaging: MRI is the examination of choice, finding basilar meningeal enhancement, tuberculomas, hydrocephalus and ischemic infarctions. [4] Treatment: prolonged antituberculosis quadruple therapy (9-12 months), adjuvant corticosteroid therapy, treatment of complications. The prognosis remains poor, with high mortality and frequent sequelae. [5]

CONCLUSION

Neuromeningeal tuberculosis remains a diagnostic and therapeutic emergency. Immunosuppression complicates its clinical expression and worsens its prognosis. MRI imaging and rapid molecular tests allow for earlier diagnosis. Early, multidisciplinary management remains crucial.

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