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Subacute tuberculous meningitis in immunocompetent patients: a diagnostic challenge in the emergency department

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Abstract

Tuberculous meningitis is a rare but severe form of extrapulmonary tuberculosis, often diagnosed late due to its subacute and non-specific clinical symptoms. This report describes a 42-year-old immunocompetent man with tuberculous meningitis, diagnosed in the emergency room, presenting with acute hydrocephalus and typical basal meningeal enhancement on MRI. PCR confirmed the diagnosis of Mycobacterium tuberculosis on gastric aspirate. Early treatment with antituberculosis drugs, corticosteroids, and neurosurgical intervention led to a positive outcome. This case emphasizes the importance of maintaining a high index of suspicion for tuberculous meningitis, even in immunocompetent patients, and highlights the need for early multidisciplinary management to reduce mortality and neurological complications.

Key words: meningitis; hydrocefalus; tuberculosis.

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Introduction

Tuberculosis (TB) remains a leading cause of illness and death worldwide, with more than 10 million new cases estimated by the World Health Organization (WHO) in 2023 and about 1.3 million deaths, mostly in low- and middle-income countries. In Europe, although the incidence is lower, cases are rising among migrant populations, asylum seekers, and immunocompromised individuals.[1] Tuberculous Meningitis (TM) is one of the most serious extrapulmonary forms of Mycobacterium tuberculosis infection, with a mortality rate that can exceed 30% even with proper treatment. The symptoms usually develop slowly, often over 1 to 4 weeks, and include nonspecific signs such as fever, headache, weakness, behavioral changes, and, in advanced cases, focal neurological signs, seizures, and coma. Diagnosis can be delayed, especially in countries with low incidence like Italy, where the disease is not commonly seen in emergency rooms.[2] However, globalization and increased migration from endemic areas make encountering unusual forms of TB in the emergency department more common. The subacute onset and generalized symptoms create a challenging diagnostic situation for emergency physicians, requiring a high level of clinical suspicion.[3-5]

Case Report

A 42-year-old man from Bangladesh, living in Italy for about 6 months, was brought to the ED by the local emergency service after being found in a state of drowsiness and confusion at a reception center where he was staying. At the entrance to the ED the patient had a GCS of 10 (E3, V4, M3), a blood pressure of 120/75 mmHg, a heart rate of 98 bpm sinus, a temperature of38.2°C, and a saturation of 98% in ambient air. Neurological examination showed a soporific patient, awakened to intense verbal stimulus, no apparent focal deficits, intense nuchal rigidity with positive Lasegue, right eyelid ptosis and right to left anisocoria. The blood tests showed lymphopenia (400/mmc) MCV 79, CRP 2.9, and normal coagulation. Chest X-ray was negative due to thickening.

From an anamnestic point of view, the patient presented post-traumatic left leg amputation outcomes, gastro-esophageal reflux disease, previous chronic osteomyelitis of the left stump.

An emergency brain CT scan was performed, revealing acute tetraventricular hydrocephalus, predominantly involving the lateral ventricles, without evidence of focal obstructive lesions.

Subsequent brain MRI with intravenous contrast agent confirmed ventricular dilation and demonstrated diffuse basal leptomeningeal enhancement involving the subarachnoid spaces at the skull base, brainstem, and suprasellar cisterns, with extension along the cranial nerves, particularly the VII–VIII nerve complexes and the trigeminal nerves bilaterally.

These findings were interpreted as highly suggestive of tuberculous meningitis with secondary communicating hydrocephalus due to inflammatory obstruction of cerebrospinal fluid reabsorption at the basal cisterns (Figure 1).

Based on the presence of acute hydrocephalus associated with

altered consciousness, neurosurgical consultation recommended temporary cerebrospinal fluid diversion.

Specifically, a temporary external CSF derivation was placed, and the patient was then admitted to the emergency medicine department. In the following days, the patient underwent repeated CT scans that showed a subdural air layer in the right frontal region, with air bubbles in the temporal horns of the lateral ventricles, without changes in ventricular dilation. For this reason, the external CSF shunt was replaced. It was removed after 13 days, following positive control CT results that included a reduction in the size of the ventricular cavities and the disappearance of air bubbles in the ventricular system. Placement of a permanent ventriculoperitoneal shunt was not necessary.

The patient underwent lumbar puncture, which revealed pleocytosis (79 cells/μL) with a lymphocytic predominance, elevated protein levels, and hypoglycorrhachia. Cerebrospinal fluid Ziehl–Neelsen staining and PCR for Mycobacterium tuberculosis were negative, consistent with the known low sensitivity of CSF microbiological tests in tuberculous meningitis. Given the high clinical and neuroradiological suspicion, antituberculosis therapy with four drugs (isoniazid, rifampicin, pyrazinamide, and ethambutol) and high-dose corticosteroids was promptly initiated. Microbiological confirmation was subsequently obtained by PCR positivity for Mycobacterium tuberculosis in a gastric aspirate.

On the sixth day of hospitalization, he underwent HRTC, which showed pulmonary involvement, and gastric aspiration was weakly positive. During therapy for pyrazinamide liver toxicity, antituberculosis treatment (rifampicin, ethambutol, levofloxacin, then moxifloxacin, and linezolid) was adjusted for three weeks, after which isoniazid was resumed. A follow-up MRI performed two months after starting treatment showed significant improvement.

During hospitalization, the patient was treated for hyponatremia suspected to be caused by SIADH. Also, during the episode of fever, blood cultures were taken and tested positive for MRSE, so the patient was treated for septicemia with daptomycin. Because of urinary symptoms, a urine culture was also obtained, which grew E. coli ESBL+, and he was treated with meropenem. For persistent fever despite adequate antibiotic therapy, a diagnosis of IRIS (immune reconstitution inflammatory syndrome) was considered. As a result, prednisone therapy, initiated early during hospitalization, was increased, which led to symptom resolution and a gradual tapering of the steroid.

A follow-up brain MRI performed two months after initiation of therapy showed marked reduction of basal leptomeningeal enhancement and resolution of hydrocephalus, with no evidence of ischemic lesions.

Discussion

The case presented highlights the main challenges in managing tuberculous meningitis in a European emergency-urgency setting. The diagnosis was made based on clinical and radiological findings, with microbiological confirmation limited to gastric aspirate, underscoring the known low sensitivity of CSF tests.[6] The prompt initiation of antituberculosis and corticosteroid therapy proved to be crucial for the outcome, in accordance with WHO and CDC guidelines.

From a neurological perspective, hydrocephalus is the most common and severe complication of tuberculous meningitis, often requiring temporary or permanent CSF diversion. In our case, mul-

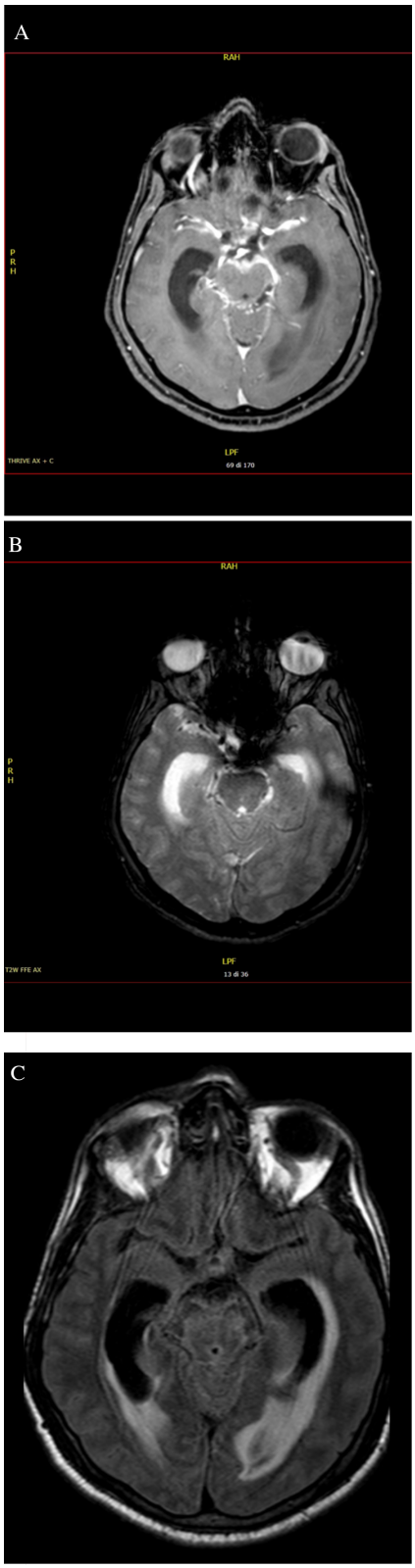

Figure 1. Brain MRI in a patient with tuberculous meningitis. (**A**) Axial T1-weighted post-contrast image demonstrating prominent basal leptomeningeal enhancement, predominantly involving the suprasellar and perimesencephalic cisterns. (**B**) Axial T2-weighted image showing ventricular dilatation consistent with hydrocephalus. (**C**) Axial FLAIR image demonstrating periventricular hyperintensity suggestive of transependymal CSF permeation.

tidisciplinary management involving infectious disease specialists and neurosurgeons successfully resolved the condition without the need for a permanent ventriculoperitoneal shunt.[7]

The radiological appearance on MRI with contrast shows thickening and enhancement of the basal meninges, caused by an inflammatory exudate rich in cells and caseous material. These deposits are found in the cisterns at the base of the brain, such as the interpeduncular cistern, prepontine cistern, and middle cranial fossa. This inflammation mainly affects the base, while the brain's convexities are less often involved. Another common feature is communicating hydrocephalus, which results from obstruction of the normal Cerebrospinal Fluid (CSF) flow due to exudate buildup in the basal cisterns. This leads to symmetrical ventricular dilation without focal blockages, indicating widespread disruption of CSF reabsorption.[8, 9]

Vasculitis is a recognized complication of tuberculous meningitis and may result in ischemic infarctions involving the basal ganglia, thalamus, or internal capsule, typically appearing as hyperintense lesions on T2- and FLAIR-weighted images with restricted diffusion on DWI during the acute phase. In the present case, no ischemic lesions were identified on MRI; therefore, this aspect is discussed as a general pathophysiological consideration rather than a case-specific finding.

In summary, the typical MRI features of tuberculous meningitis include: i) basal leptomeningeal enhancement; ii) communicating hydrocephalus, and iii) ischemic infarctions of the basal ganglia.

MRI, thanks to its multiplanar sequences and gadolinium sensitivity, is therefore the most useful tool for recognizing the disease, assessing its extent, and identifying its complications, allowing early diagnosis and effective monitoring of the therapeutic response.[10]

The differential diagnosis of basal leptomeningeal enhancement is broad and includes inflammatory, infectious, and neoplastic conditions such as neurosarcoidosis, fungal meningitis (particularly cryptococcosis), carcinomatous meningitis, and other forms of chronic infectious meningitides. Differentiating among these entities can be challenging, especially in the emergency setting, as they may share overlapping clinical and radiological features. In the present case, however, the subacute clinical course, the presence of communicating hydrocephalus, the characteristic basal meningeal involvement on MRI, the patient's epidemiological background, and the subsequent microbiological confirmation collectively supported the diagnosis of tuberculous meningitis over alternative causes.[11]

A further element of interest was pyrazinamide hepatotoxicity, which required modification of the therapeutic regimen with the introduction of fluoroquinolones and linezolid. This underscores the importance of serious biochemical monitoring in patients on antituberculosis therapy. The appearance of IRIS, despite the patient being HIV-negative, required corticosteroid augmentation, demonstrating how this syndrome can also occur in immunocompetent people undergoing immune recovery from severe lymphopenia.[12,13]

Finally, nosocomial infections (MRSE, ESBL+) added to the therapeutic complexity, reflecting the vulnerability of these patients. The case underscores the importance of an integrated and timely approach, combining neurosurgical management, personalized antituberculosis therapy, and complication control, particularly in frail and migrant patients.

Conclusions

The diagnosis of tuberculous meningitis in the emergency department is often delayed because of its subacute and nonspecific clinical signs, which can resemble those of more common viral or bacterial illnesses. The classic meningeal signs (nuchal rigidity, Kernig's, and Brudzinski's signs) are often absent, especially in the early stages.

This condition remains a stealthy medical problem with a severe prognosis if not identified and treated promptly. In the emergency room, it is crucial to keep a high level of suspicion when encountering subacute neurological symptoms, even in patients with normal immune function.

The experience described highlights the need for an early multidisciplinary approach, the importance of neuroimaging in diagnosis, the need to monitor for pharmacological toxicities and infectious complications, and the role of corticosteroids in managing IRIS.

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