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TUBERCULOUS MENINGITIS (TBM) BEING MISDIAGNOSED AS NEUROCYSTICERCOSIS: A CASE REPORT

General Medicine

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ABSTRACT

Tuberculosis remains a leading cause of morbidity and mortality in the developing world and tuberculosis of the central nervous system (CNS) is the most serious complication of this disease.1 Symptoms and radiological features are nonspecific, leading sometimes to misdiagnosis. The rapid diagnosis of TBM is fundamental to a favourable clinical outcome. Here we present a case which was initially misdiagnosed as disseminated neurocysticercosis due to similar imaging findings and later found it to be Tuberculous meningitis.

KEYWORDS

Tuberculous Meningitis, Misdiagnosis, Neurocysticercosis, Case-report

INTRODUCTION:

Tuberculosis remains a leading cause of morbidity and mortality in the developing world. Cerebral tuberculoma is uncommon but still is a life threatening form of tuberculosis (TB) due to the haematogenous spread of Mycobacterium Tuberculosis and CNS involvement is thought to occur in 2-5% of patients with tuberculosis.

Cerebral Tuberculosis and neurocysticercosis, which often mimic each other and diagnosis is difficult due to similar clinical symptoms and imaging findings.[2]

Here we hope to highlight the ambiguous manifestation of tuberculous meningitis and to enhance clinician awareness regarding the significance of considering these various presentations to aid in timely diagnosis by describing a case which was initially thought to be neurocysticercosis and was later diagnosed to be tuberculous meningitis.

CASE HISTORY:

A 18year old female was admitted to Mayo hospital, Mayo institute of medical sciences, Barabanki, Uttar Pradesh on 06/07/2021 with the complaints of headache, fever since 2weeks; vomiting, generalized weakness since 1week; altered sensorium since 2days.

On examination:

Blood Pressure: 126/80 mmHg,
Heart Rate: 102bpm,
Temperature: 101°F,
Respiration Rate:22/min,
SPO2: 95% on room air
Random Blood Sugar: 116mg/dl.

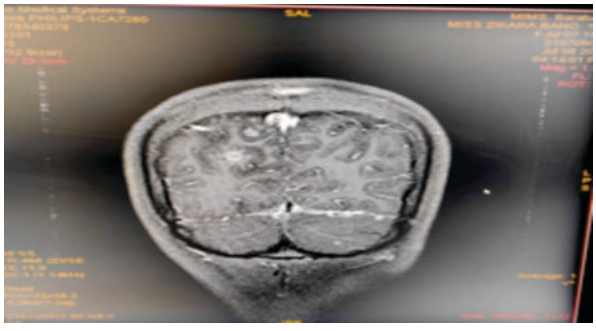
Patient was conscious, drowsy, oriented. Meningeal signs (Neck rigidity & Kernig's sign was positive). Power of all four limbs decreased (3/5), Tone of all four limbs increased. Superficial reflexes were normal, Deep tendon reflexes were exaggerated, Bilateral Babinski was positive.

Initially, the patient was hospitalised and given conservative treatment.

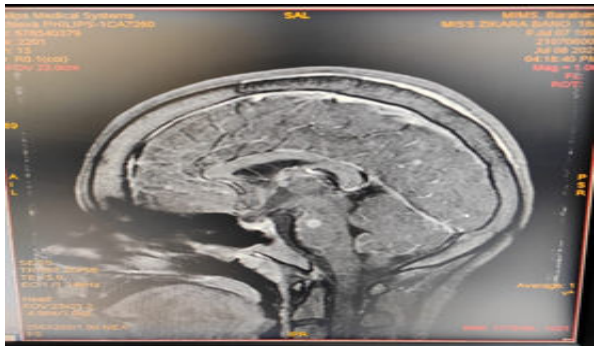
Lab investigations showed:

Total Leucocyte Count: 13200 cells/mm^3
Hemoglobin: 12.8 g/dl
Platelet count: 4.07lakh/mm3
Dengue card Test– Negative
Malaria card Test- Negative
Widal Test- Negative
Liver function tests and kidney function tests done were within normal range.
Sr. Sodium: 136.5mg/dl,
Sr. Potassium: 3.25mg/dl

Initially, an MRI of the brain suggested ?Inflammatory Granuloma, which on further evaluation with CEMRI BRAIN which showed Multiple T2 & FLAIR Hyperintense ring enhancing intra-axial lesions involving the supra and infra tentorial brain parenchyma. (Pic.1 & 2).Bilateral frontoparietal lobes showing Starry-sky appearance and impression given was Disseminated Neurocysticercosis.[3,4]



Pic.1



Pic.2

Based on the above report, patient was started with Albendazole and Steroids, but there was no improvement noted. However, because meningeal signs were present, we performed a CSF analysis, which revealed:
CSF Protein: 102mg/dl,
CSF TLC: 90cells/microL,
Lymphocytes 95%,
CSF Glucose 31mg/dl (RBS was 110mg/dl),
CSF ADA was 11.40u/L (Positive).

As CSF analysis picture was suggestive of Tuberculous Meningitis (TBM) , treatment with Albendazole was been stopped and was started Anti Tubercular Therapy (ATT)with Isoniazid, Rifampicin, Pyrazinamide and Ethambutol (HRZE) Inj.Streptomycin 750mg IM OD, Dexamethasone, Glycerol. Fundus examination of patient showed Bilateral Choroidal tubercles with Papilledema. Lateral Rectus involvement in left Eye noted. Patient showed improvement after starting ATT and was able have food orally and perform routine

activities. She was discharged on 15/07/2021. She was being followed up on a regular basis for the same.

On 14/09/21, about 2 months after discharge she came back to Medicine OPD with complaints of difficulty in walking, decreased vision bilaterally, fever - on and off, vomiting and bilateral lower limb pain since couple of weeks. She was admitted and repeat CEMRI Brain was done on 16/09/2021 which showed Conglomerate tuberculomas with suprasellar arachnoiditis. Repeat CSF Analysis on 17/09/2021 showed:
CSF Glucose: 21.82mg/dl (RBS was 98mg/dl),
CSF Protein: 224.5mg/dl,
CSF Lymphocytes: 90%; which showed similar Tuberculous Meningitis picture as before. Treatment for TBM continued with ATT, Streptomycin, and Prednisolone. Patient was discharged on 21/09/21 and was on regular follow up. On 05/10/21, that is about 10 weeks after diagnosis with TBM, patient during regular follow up presented with complaints of pain and decreased hearing in left ear and jaw pain since previous three days. Inj. Streptomycin was been stopped at ten weeks and HRZE continued. After twelve weeks of initial treatment with HRZE, regimen was shifted to HRE. Gradual improvement was seen in patient condition and she recovered.

DISCUSSION:

Tuberculosis (TB) is a major global health problem that continues to affect India's public health. Mycobacterium tuberculosis can infect almost any organ of the body, but when it comes to the brain, the implications are deadly. Tuberculous meningitis (TBM) is the most deadly and crippling type of the disease, with an estimated 1 lakh new cases diagnosed each year around the world.[5] Most people's innate and adaptive immune systems either eliminate or contain M. tuberculosis after inhaling aerosolized M. tuberculosis. Failure of these mechanisms, on the other hand, can result in haematogenous spread of bacteria to distant areas, including the meninges, and rupture of a meningeal tuberculoma can cause tuberculous meningitis. [6] Fever, headaches, recurrent seizures lasting more than 2 to 3 weeks, poor response to conventional antibiotics, and focal neurological symptoms such as hemiplegia, aphasia, and numerous cranial nerve palsies, as well as signs of meningeal irritation, are the most common symptoms. The ensuing neurological pathology is caused by three general processes: adhesion development, obliterative vasculitis, and encephalitis or myelitis.[7] To avoid neurological impairment or possibly death, early diagnosis and consistent therapy are required. However, it is frequently misdiagnosed due to the nonspecific clinical presentation and ambiguous radiological finding.[8]

Differential diagnosis of Tuberculous meningitis 9
Pyogenic meningitis – partially treated
Neurocysticercosis
Intracranial space-occupying lesions of various etiologies
Sarcoidosis
SLE vasculitis
Viral encephalitis
Cryptococcal meningitis
Glioma

The diagnosis of TBM can be made by the clinical features and staging by assessing the severity using the modified British Medical Research Council TBM grading.[10] Laboratory investigations are Lumbar puncture and CSF analysis for glucose, leucocyte count and ADA, CSF culture for MTB,,microscopy using Ziehl – neelsen staining of acid fast bacilli , Nucleic acid amplification tests and radiological investigations like X-ray,CT and MRI can be done.[11]Due to the resemblance in clinical symptoms and typical imaging data, distinguishing between tuberculous meningitis and neurocysticercosis is challenging. In this case, a high CSF ADA level aided in the differential diagnosis.

The most essential aspect of TBM diagnosis is to detect TBM and act swiftly to refer the patient to a centre where they will receive: rapid access to CSF investigation, neuroimaging, prompt ATT treatment, and supportive care.[12] All kinds of CNS TB are treated with four medications for two months (isoniazid, rifampicin, pyrazinamide, and ethambutol), followed by two treatments (isoniazid, rifampicin) for nine to twelve months. Adjuvant corticosteroids (either dexamethasone or prednisolone) are also recommended.

Around 30% of people with TBM have clinical worsening after starting antituberculosis medication, which is known as paradoxical responses and is linked to increased intracerebral inflammation. All potential paradoxical reactions should be investigated with brain imaging, and the findings, treatment, and outcomes should be documented and managed appropriately.14

CONCLUSION:

Tuberculous meningitis is an illness that can be lethal. Delayed presentation, dependence on alternative or traditional medicine, inability to perform or refusal of lumbar puncture, lack of laboratory facilities, and antibiotic unavailability or price are all factors that contribute to severe gaps in diagnosis and treatment. MTB confirmation by culture in the CSF considered as the gold standard worldwide, despite the advances in creating superior and accurate diagnostic techniques. To avoid irreversible impairment or death, early diagnosis and good treatment are imperative.

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