CASE REPORT

Subdural Tubercular Empyema in an Immunocompetent Patient: A Rare Primary Presentation of CNS Tuberculosis

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Abstract

Tuberculosis (TB) remains a potentially-curable serious health problem in most of the developing world including Pakistan. Subdural Tubercular Empyema is a very rare presentation of CNS tuberculosis. A 25-year-old gentleman presented with a 25-day history of low-grade intermittent fever and generalized headache. On examination, he had a temperature of 99.6°F with equally round and reactive to light pupils with normal extraocular muscle movements. However, there were signs of papilledema on fundoscopic examination. Signs' of neck rigidity (Kernig and Brudzinski) were negative. There was no focal neurological deficit. CSF analysis showed TLC 660 cells/uL with 100% lymphocytes, RBC 0 cells/uL, Protein 73 mg/dl, and Glucose 43 mg/dl with no organisms on Giemsa staining but positive AFB stain microscopy. GeneXpert-PCR for MTB was also positive on CSF with no drug resistance to rifampicin detected. His brain MRI scan showed left subdural empyema. Workup for the immunosuppressive condition was negative. He was diagnosed as having Subdural Tubercular Empyema and started on Anti-tubercular therapy. On follow-up at 4 weeks, he was asymptomatic and tolerating ATT without any adverse effects.

Keywords: *Subdural Tubercular Empyema, GeneXpert-PCR for MTB, Anti-tubercular therapy.*

INTRODUCTION

Tuberculosis (TB) remains a potentially-curable serious health problem in most of the developing world including Pakistan. Subdural Tubercular Empyema is a very rare presentation of Central Nervous System (CNS) tuberculosis with only a few case reports documented in existing literature [1, 2]. Subdural empyema is an accumulation of pus in the subdural space and is usually pyogenic. With the increase in cases of HIV/AIDS and other immunosuppressive conditions, there has been a resurgence in cases of tuberculosis, especially in atypical sites [2, 3]. To decrease mortality and morbidity caused by CNS Tuberculosis, timely diagnosis and early initiation of treatment are crucial. Diagnosis of Subdural Tubercular empyema may be challenging because of variable non-specific features clinically which may be of only mild intensity in the initial course of the disease [4, 5]. Difficulties in getting an adequate sample of CSF, pus, or granulation tissue to diagnose tuberculosis also pose a hurdle in timely diagnosis [4].

CASE PRESENTATION

A previously healthy 25-year-old gentleman presented with a 25-day history of low-grade intermittent fever documented 100°F along with a generalized headache of moderate intensity aggravated by bending forward. There was no history of oral or genital ulcers, respiratory infections, sinusitis, skin rashes, joint pains, or previous psychiatric or neurological manifestations. A shopkeeper by profession, he was unmarried and did not smoke or use illicit drugs. There was no history or contact/family history of tuberculosis. On examination, he had a temperature of 99.6°F with normal blood pressure and pulse rate. On neurological examination, pupils were equally round and reactive to light with normal extraocular muscle movements. However, there were signs of papilledema on fundoscopic examination. Signs' of neck rigidity (Kernig and Brudzinski) were negative. There was no focal sensory, motor, or cerebellar neurological deficit. Respiratory, precordial, and abdominal examinations were normal. On investigation, CBC demonstrated normal TLC, and ESR was raised at 73 mm/hour. His RFTs, LFTs, and urinalysis were normal. He was admitted on lines of encephalitis and a lumbar puncture for CSF analysis was done.

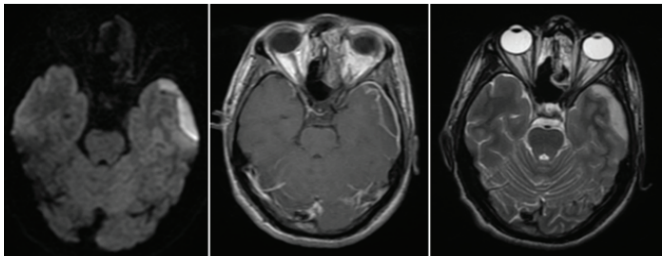

Fig. (1): MRI Scan Brain (with FLAIR) at the time of presentation.

After doing a lumbar puncture, he was started empirically on intravenous acyclovir, ceftriaxone, and vancomycin. CSF analysis showed TLC 660 cells/uL with 100% lymphocytes, RBC 0 cells/uL, Protein 73 mg/dl, and Glucose 43 mg/dl with no organisms on Giemsa staining but positive AFB stain microscopy. GeneXpert-PCR for MTB was also positive on CSF with no drug resistance to rifampicin detected. His blood, urinary, and spinal

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fluid cultures did not grow any organism growth. His brain MRI scan with FLAIR **(Fig. 1)** showed left subdural empyema. Workup for the immunosuppressive condition was done. Serology for HBV, HCV, HIV, and syphilis was negative. His echocardiography, and chest and abdomen CT scan were within normal parameters. He was diagnosed as having Subdural Tubercular Empyema and a neurosurgical consult was taken for craniotomy and drainage but the patient was unwilling to undergo any neurosurgical procedure. He was started on Anti-tubercular therapy consisting of oral rifampicin, isoniazid, pyrazinamide, and ethambutol along with steroids. On follow-up at 4 weeks, he was asymptomatic and tolerating ATT without any adverse effects. A repeat MRI brain with FLAIR at a 4-week follow-up **(Fig. 2)** showed progressive resolution of the subdural empyema.

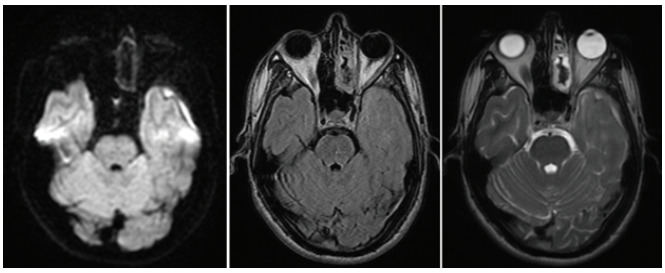

Fig. (2): MRI Scan Brain (with FLAIR) at 4-week follow-up.

DISCUSSION

Tuberculomas and tuberculous meningitis are not uncommon presentations of CNS tuberculosis but subdural tubercular empyema is extremely rare. CNS tuberculosis in almost 1% of patients of tuberculosis and Tuberculous meningitis accounts for up to 80% of cases of CNS tuberculosis [6, 7]. Extrapulmonary tuberculosis including CNS tuberculosis and tuberculosis involving atypical sites is more commonly seen in immunodeficient patients especially in patients with HIV/AIDS [7]. Our patient was previously healthy, HIV-negative, and did not have any evidence of immune deficiency. Indicators for diagnosis of CNS tuberculosis in the cerebrospinal fluid analysis include mononuclear pleocytosis, decreased glucose, and increased protein levels [7, 8]. Identification of the Mycobacterium in CSF by AFB staining, culture, and GeneXpert-PCR MTB help to confirm the diagnosis but may still be challenging [8, 9]. Our patient had 100% lymphocytes with low glucose and high protein on CSF analysis. Furthermore, his CSF was positive for AFB stain and GeneXpert-PCR for MTB with negative gram staining and negative bacterial cultures. However, a neurosurgical aspiration of the empyema to demonstrate AFB could not be done as the patient was not willing to undergo neurosurgical intervention.

Anti-tuberculous therapy (ATT) is the mainstay treatment of CNS tuberculosis [10]. The duration of treatment is 9-12 months with ATT. Treatment of tuberculosis is most efficacious when initiated early and may be started promptly on basis of clinical assessment without laboratory confirmation [10]. Adjunctive corticosteroids help to decrease mortality and morbidity in all but late-stage diseases [10, 11]. Indicators of poor outcomes in CNS tuberculosis patients include co-existing HIV infection, drug-resistant tuberculosis, and a depressed conscious level at presentation [6]. Surgical treatment of subdural tubercular empyema requires individualization according to each case depending on the size of the empyema and the neurological condition of the patient [2]. Our patient did not have focal neurological signs and was unwilling to undergo neurosurgical intervention. He was started on at the 4-week follow-up with a resolution of the empyema on a follow-up MRI scan. Banerjee *et al.* [12] reported full recovery in a 12-year-old child with subdural tubercular empyema following 18 months of treatment with anti-tubercular treatment. Konar *et al.* [13] recommend that prompt diagnosis and immediate intervention is the goal of the management of subdural empyema because a delay in diagnosis may lead to complications such as unconsciousness, cortical venous thrombosis, and infarction which adversely affect the outcome.

CONCLUSION

In conclusion, high clinical suspicion is necessary to diagnose CNS tuberculosis, especially Subdural Tubercular Empyema so that timely diagnosis and adequate treatment may be instituted to improve prognosis in these patients.

CONSENT FOR PUBLICATION

Detailed informed consent was taken from the patient

CONFLICT OF INTEREST

The authors of declare no conflict of interest.

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None.

AUTHOR'S CONTRIBUTION

This study was conceived and designed by NIB, MSAG, and HW. NIB, MBR, and MUJ did the initial literature research. MSAG, HW, and DS did the data collection, assembly, and patient assessment. NIB, MSAG, and MBR were involved in manuscript writing. HW, MUJ, and DS did the final critical review and corrections. NIB is the corresponding author on behalf of all other authors.

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