Case Report on Tuberculosis Meningitis-A Nurses Perspective

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Case Study

ABSTRACT

Mycobacterium tuberculosis is the bacteria that cause tuberculosis. If the infection is not treated immediately the bacterium passes via the circulatory system and spread other organs and tissues. Pathogen will travel to the meninges and causes inflammation of membranes called as tubercular meningitis. Here, the authors report a case of tuberculous meningitis a 42- years-old male patient with the chief complaints of low grade fever with chills since 1 month, headache in frontal region since 5-6 days, altered sensorium, breathing difficulty, reduced speech and left side weakness of the body since 1 day. After admitting in the ward all investigation done like MRI brain, ECG, lumbar puncture, blood tests etc. and he diagnosed as tuberculous meningitis. Patient admitted in AVBRH in ICU, investigations done, where patient was on NIV support, nasogastric tube, foleys catheter and it has been removed as patient was improving the condition and shifted in ward. Patient medical treatment in the ward was inj. C-tri 2 gm, Inj.levoflox 500 mg, inj. thimine 100 mg, Inj.Dexamethasone 10 mg, tab. Pan 40 mg, tab. Akt4 3, Inj.manitol, tab. Lorazepam. History collection, Physical examination, neurological assessment and nursing care plan were done after all treatment patient condition has been improved.

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1. INTRODUCTION

Extrapulmonary tuberculosis is shown by tuberculous meningitis (TBM), which is caused by the seeding of the meninges with Mycobacterium tuberculosis bacilli (MTB).MTB is first introduced into the host by droplet inhalation infecting the alveolar macrophage. The infection starts in the lungs and then spreads to the lymph nodes. At this point in the infectious process, there is a high degree of bacteremia that can seed the entire body. In tuberculous meningitis, the meninges are seeded by MTB and form sub-ependymal collections called Rich foci. These foci can rupture into the subarachnoid space and cause an intense inflammatory response that causes meningitis symptoms. The exudates caused by this response can encase cranial nerves and cause nerve palsies. They can entrap blood vessels causing vasculitis and block cerebral spinal fluid (CSF) flow leading to hydrocephalus. These immune responses can lead to complications associated with tuberculous meningitis and chronic sequela seen in patients who recover from TBM. This activity reviews evaluation, management, and current public health preventative measures to prevent tuberculous meningitis. This activity highlights the interprofessional teams involved in the prevention and management of this global health threat [1].

Tuberculous meningitis (TBM) can occur as the sole manifestation of TB or concurrent with pulmonary or other extra pulmonary sites of infection [2].

TBM carries a high mortality and morbidity, particularly among patients coinfected with HIV [3[.

Delays in seeking medical care, diagnosis, and initiation of treatment are contributing factors to the high mortality and morbidity, especially in resource-limited regions. When diagnosed promptly, TBM can be cured with supervised medication administration and supportive care [4].

Patients with TBM develop typical symptoms and signs of meningitis including headache, fever, and stiff neck, although meningeal signs may be absent in the early stages. The duration of symptoms before presentation ranges from several days to several months [5].

Especially in resource-limited settings, TBM cases may present in advanced clinical stages, with Glasgow Coma Scale scores of 10 or less [2].

Cranial nerve (CN) palsies, hemiparesis, paraparesis, and seizures are common and should raise the possibility of MTB as the etiology of meningitis. Patients often present with multiple CN palsies, most commonly involving CN III, VI, and VII. Chest X-ray is suggestive of active or previous pulmonary TB in approximately 50% of cases [6].

2. CASE REPORT

A 42-years-old male patient was admitted in AVBRH Sawangi (M) Wardha on date 24/12/2020 in intensive care unit with the chief complaints of low grade fever with chills since 1 month, headache in frontal region since 5-6 days, altered sensorium , breathing difficulty, reduced speech and left side weakness of the body since 1 day. After admitting in the ward all investigation done like MRI brain, ECG, lumbar puncture, blood tests etc. and he diagnosed as tuberculous meningitis.

RT-PCR was done i.e. Negative then he was admitted in intensive care unit in AVBRH hospital, where various investigations carried out, patient was on nasogastric tube from 24/12/2020 and was on NIV support from 24/12/2020, foleys catherization was done. The nasogastric tube and NIV support were removed on 26/12/2020 as patient was improving the condition and he was shifted to ward on 28/12/2020.

2.1 Therapeutic Intervention

Patient medical treatment in the ward was inj. C-tri 2 gm, inj.levoflox 500 mg, inj.thimine100mg, inj.dexamethasone 10 mg, tab. Pan 40 mg, tab. Akt4 3, inj.manitol, tab. Lorazepam.

2.2 Follow-Up and Outcomes

In-spite of all care patient progress was good. Follow-up care related to blood pressure. He was advised to strictly cover mouth and nose with a tissue and put used tissue in a closed bag and throw it away. If dont have a tissue, cough or sneeze into upper sleeve or elbow, not hands. Wash hands often with soap and warm water for 20 seconds.

Table 1. Diagnostic evaluation

Investigation	Patient value	Normal value	Justification
Complete blood count			
1. Hb%	14.1 gm%	13-15.	Normal
2. Mcv	88.2 cub. Micron	80-90cub.micron	Normal
3. Mch	32.4 pico gm	26.5-33.5 pico gm	Normal
4. Total RBC count	4.66 million/cu.mm	4.5-6 million/cu.mm	Normal
5. Total WBC count	13,600 cu.mm	4000-11000 cu.mm	Increased
6. Total platelet count	2.42 lacs/cu.mm	1.5-4 lacs/cu.mm	Normal
7. Monocytes	04%	4-10%	Normal
KFT			
1. Urea	22 mg%	18-40mg%	Normal
2. Creatinine	0.8 mg%	0.7-1.5 mg%	Normal
3. Sodium	124 meq/l	136-145meq/l	Decreased
LFT			
1. Total protein	7.2 gm%	6.8 gm%	Normal
2. Albumin	3.4 gm%	3-5 gm%	Normal
3. Total bilirubin	0.9 mg/dl	0.3-1 mg/dl	Normal
4. Bilirubin conjugated	0.3 mg/dl	1-3 mg/dl	Decreased
5. Bilirubin unconjugated	0.6 mg/dl	0.2-0.8 mg/dl	Normal
ESR	28	0-29mm/hr	normal
CSF fluid analysis			
Glucose	25mg/dl	50-80 mg/dl	Decreased
LDH	79 units	<70 units	Increased
Protein	158mg/dl	15-60 mg/dl	Increased

Cb-naat- positive; Rtpcr –negative; MRI- diagnosed as tuberculous meningitis

3. DISCUSSION

Meningitis is the most dangerous type of tuberculosis, especially in those who are HIV-positive. The significant death rate linked with this condition can be greatly reduced with early identification and treatment. In general, therapy should last at least nine months and include at least four drugs that the M. tuberculosis strain is known or presumed to be susceptible. Adjunctive corticosteroid therapy should be explored, especially in those who do not have HIV. Its best to base treatment on TB resistance trends, especially in HIV-coinfected people who are at high risk of developing drug-resistant TB [5].

Tuberculous meningitis (TBM) is the most prevalent type of tuberculosis of the central nervous system (CNS) and is caused by Mycobacterium tuberculosis. If not treated quickly, TBM is linked to a high rate of neurologic sequelae and death.

According to WHO estimates, 10.4 million new TB infections are diagnosed each year (one million of which are children), and at least 100,000 people get tuberculous meningitis annually [7].

In India, an estimated 1.5 individuals die each year from tuberculous meningitis for every 100,000 inhabitants [8].

TBM prognosis is primarily determined by neurologic state at the time of presentation and the length of time between diagnosis and therapy beginning. While TBM does not usually progress as quickly or as fulminantly as meningitis caused by pyogenic bacteria, empiric therapy should be started as soon as the diagnosis is established, since any delay in treatment might impair the prognosis. Various case studies showed a mortality rate of 7%–65% in wealthy nations and up to 69 % in developing ones [9].

Those with comorbidities, significant neurologic involvement on admission, fast disease progression, and advanced or very young age had the highest mortality risk. Up to 50% of survivors develop neurologic sequelae [10].

Because of the difficulty in quickly detecting MTB in CSF samples, TB is one of the most difficult causes of meningitis to diagnose. TBM should be aggressively evaluated in any patient with symptoms and indications of meningitis in areas where TB is prevalent, as well as in high-risk persons in areas where TB is less prevalent. To

support a diagnosis of TBM, clinical, microbiologic, and radiologic data should be utilized together. In presumptive cases with negative ZN staining of CSF for acid-fast bacilli, empirical therapy with anti-TB medicines is the mainstay of care The expert MTB/RIF test allows for the fast diagnosis of MTB infection [11].

TBM can be difficult to diagnose and may be relied only on clinical and early CSF findings without conclusive microbiologic confirmation. TBM is more likely among patients who have had symptoms for more than six days, have significant CSF pleiocytosis, and have focused impairments [12].

CSF results are characterized by the following: Pleiocytosis with a lymphocytic predominance. The average total white cell count is between 100 and 500 cells per litre. Lower counts and neutrophil predominance may be seen early in the illness, (ii)elevated protein levels, generally between 100 and 500 mg/dL, (iii)low glucose, usually less than 45 mg/dL, or a CSF: plasma ratio of less than 0.5. A CSF sample should be submitted for an acid-fast smear, with the proviso that the sensitivity of a single sample is poor, on the range of 20%–40% [13].

Although results are highly dependent on CSF volume, timeliness of sample delivery to the lab and analysis, and technical expertise of lab personnel, early studies demonstrated that acid-fast stains can detect up to 80%.Despite the fact that culture takes many weeks and has a poor sensitivity (40–80%), it should be used to evaluate medication susceptibility. Drug-resistant M. tuberculosis strains have significant prognosis and therapeutic implications; in fact, TBM caused by INH-resistant M. tuberculosis strains has been linked to a twofold increase in mortality [14].

Because of the poor sensitivity of acid-fast smears and the inevitable delay in culture, newer TBM diagnostic techniques have just lately been developed. ELISA techniques have been developed to detect antibodies directed against particular mycobacterial antigens in the CSF with varied sensitivity, but their restricted availability prevents them from being used as point-of-care testing in resource-poor countries. According to a recent research in children aged 6–24 months, a CSF adenosine deaminase level of 10 U/L provides >90% sensitivity and specificity for diagnosing TBM. Other investigations, on the other hand, have found that adenosine deaminase has low specificity for TBM in certain

groups, notably HIV-infected people with concomitant infections or cerebral lymphomas [15].

Microscopy/culture of large CSF volumes and nucleic acid amplification (NAA) have been compared and shown to have equal sensitivity for the diagnosis of TBM. According to a meta-analysis, commercial NAA tests that used polymerase chain reaction (PCR) for TBM diagnosis showed an overall sensitivity of 56% and a specificity of 98%. The low sensitivity is likely owing to the fact that most PCR-based investigations employ a single target for amplification, which might lead to false-negative results in some TB isolates due to the lack of the target gene. Newer PCR techniques amplify several target genes at the same time, resulting in significantly greater sensitivities in the 85 %–95 % range. Most specialists currently believe that commercial NAA tests can confirm TBM but cannot rule it out [16]. As a result, a negative CSF test for acid-fast bacilli or M. tuberculosis DNA does not rule out TBM diagnosis or eliminate the need for empiric therapy if clinical suspicion is strong. The sensitivity of CSF smear and culture diminishes fast after therapy begins, however mycobacterial DNA may be detected in the CSF for up to a month after treatment begins [12].

Neuroimaging can aid in the diagnosis of TBM Basal meningeal enlargement and hydrocephalus are two classic neuroradiologic characteristics of TBM. Hypodensities caused by cerebral infarcts, edoema, and nodular enhancing lesions may also be seen. Because it is superior to computed tomography (CT) for assessing the brainstem and spine, magnetic resonance imaging (MRI) is the imaging test of choice for visualising abnormalities associated with TBM. T2-weighted MRI imaging has been proven to be particularly effective in demonstrating brainstem disease, whereas diffusion-weighted imaging (DWI) is the most effective at detecting acute cerebral infarctions caused by TBM [17].

3.1 Strength

Patient was 42-year male admitted with complaints of altered sensorium, left side weakness of body, fever, headache now he has been recovered and tolerated all the medications as well as giving well response around 1 month to therapeutic treatment of the hospital and discharged from hospital.

4. CONCLUSION

Tuberculous meningitis is a very debilitating and destructive form of tuberculosis. It is associated with number of challenges for both researchers and clinicians [18]. The anti-tuberculosis treatment shows mortality remains high. Prognosis factor influencing disability and survival the time of delay of treatment initiation. The severity grade depends upon admission and the occurrence of stroke, involvement of cranial nerve, seizures, HIV coinfection and multidrug resistance. Treatment of tuberculous meningitis is largely depending upon pulmonary regimen.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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