ISSN: 2582-4686 SJIF 2021-3.261, 2022-2.889, 2023-5.384, 2024-6.875 ResearchBib IF: 9.948 / 2024

THE MULTIDISCIPLINARY JOURNAL OF SCIENCE AND TECHNOLOGY

VOLUME-5, ISSUE-2

Tuberculous Meningitis: Pathogenesis, Clinical Features, Diagnosis, and Treatment

Student of master's degree at ASMI **Khushnudbekov Abdulbokiy**
Student of bachelor degree at KUAF **Bakhtiyarova Khonzodabegim**

Annotation

Tuberculous meningitis (TBM) is the most severe form of extrapulmonary tuberculosis, leading to high mortality and neurological complications. It results from the hematogenous spread of Mycobacterium tuberculosis to the meninges, causing inflammation, vasculitis, and hydrocephalus. This article explores the pathogenesis, clinical presentation, diagnostic methods, and treatment of TBM. Cerebrospinal fluid (CSF) analysis, polymerase chain reaction (PCR), and neuroimaging play key roles in early diagnosis. Prompt initiation of anti-tuberculous therapy, along with corticosteroids and symptomatic management, improves survival outcomes.

Key words

Tuberculous Meningitis, pathogenesis, Mycobacterium tuberculosis, cerebrospinal Fluid Analysis, PCR Diagnosis, neuroimagin

Introduction

Tuberculosis (TB) remains a major global health issue, with an increasing burden of extrapulmonary manifestations, including TBM. TBM accounts for approximately 1% of all TB cases but is associated with high morbidity and mortality. The disease progresses through a subacute course, often leading to delayed diagnosis and severe neurological sequelae. Despite advancements in molecular diagnostics, TBM remains challenging to diagnose due to its nonspecific symptoms and the difficulty of detecting M. tuberculosis in CSF. This paper reviews the pathogenesis, clinical features, diagnostic approaches, and management of TBM.

Methods of Investigation

Cerebrospinal Fluid (CSF) Analysis

- Appearance: Xanthochromia due to increased protein levels.
- Cell Count: Lymphocytic pleocytosis (100-1000 cells/μL).
- Protein: Elevated (0.5-3.0 g/L).
- Glucose: Decreased (<2.2 mmol/L), a hallmark finding.
- Chlorides: Reduced levels.

Microbiological and Molecular Testing

- Acid-Fast Bacilli (AFB) Staining: Low sensitivity (~20%).
- Culture (Löwenstein-Jensen Medium): Requires 2-6 weeks for growth.
- PCR for M. tuberculosis: High sensitivity (~80-90%).
- GeneXpert MTB/RIF: Rapid detection with rifampicin resistance screening.

Neuroimaging (CT/MRI)

- Basal meningeal enhancement.
- Hydrocephalus.

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• Infarcts due to vasculitis.
• Tuberculomas (granulomatous lesions).

Ophthalmologic Examination
• Choroid tubercles and optic nerve edema, which may support TBM diagnosis.

Results and Discussion

TBM typically progresses in three stages:

1. Prodromal stage: Nonspecific symptoms such as headache, fever, irritability, and fatigue lasting weeks.
2. Meningitic stage: Severe headache, neck stiffness, photophobia, altered consciousness, and cranial nerve involvement.
3. Advanced stage: Coma, seizures, hemiparesis, and respiratory failure.

Early diagnosis and treatment are crucial. The mainstay of therapy is a four-drug anti-tuberculous regimen:

• Intensive phase (2 months): Isoniazid, rifampicin, pyrazinamide, ethambutol.
• Continuation phase (10+ months): Isoniazid and rifampicin.
• Adjunctive corticosteroids (dexamethasone, prednisolone) reduce brain inflammation and mortality.
• Symptomatic treatment includes anticonvulsants, mannitol for cerebral edema, and ventriculoperitoneal shunting for hydrocephalus.

Without treatment, TBM is universally fatal. With appropriate therapy, mortality rates range from 20-30%, but up to 50% of survivors develop neurological disabilities.

Conclusion

TBM remains a life-threatening condition requiring early recognition and aggressive management. CSF analysis, PCR-based diagnostics, and neuroimaging are essential for timely diagnosis. Despite improved treatment protocols, delayed diagnosis continues to contribute to high mortality and long-term complications. Enhanced awareness and early therapeutic intervention are critical to reducing TBM-related morbidity and mortality. Certainly, here are the references with their respective authors:

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