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Complications due to Hydrocephalus in Tuberculosis Meningitis

Arunangshu Dev, Bhavya Kumari
Student, Student
Department of Biotechnology
Ramaiah University of Applied Sciences, Bangalore, India

1. ABSTRACT

Hydrocephalus refers to the accumulation of fluid in the brain's ventricles, which can cause the ventricles to expand in size and exert pressure on the brain. This excess cerebrospinal fluid can lead to damage to the brain tissues and cause various issues with brain function. Normally, cerebrospinal fluid flows through the ventricles and helps bathe the brain and spinal column. However, when there is an excessive amount of this fluid in hydrocephalus, it can disrupt this process and lead to complications. Although hydrocephalus can affect individuals of any age, it is more common in infants and adults over 60 years old. Surgical intervention is often necessary to manage this condition and restore normal levels of cerebrospinal fluid in the brain. Additionally, other therapies may be required to manage symptoms and complications resulting from hydrocephalus.

2. INTRODUCTION

Tuberculosis meningitis (TBM) is a type of meningitis caused by the bacterium Mycobacterium tuberculosis, which is the same bacterium that causes tuberculosis (TB). TBM occurs when the bacteria spread from other parts of the body, such as the lungs, to the membranes that cover the brain and spinal cord. This can lead to inflammation and damage to the membranes, which can result in symptoms such as headache, fever, and stiffness in the neck. TBM is a serious condition that requires prompt diagnosis and treatment to prevent complications such as hydrocephalus and permanent brain damage. Tuberculous meningitis (TBM) is a condition that can lead to hydrocephalus, a known complication where fluid accumulates in the brain's ventricles. Typically, hydrocephalus is observed in patients who have had TBM for four to six weeks. However, in some cases, hydrocephalus may also develop at a later stage of the disease. This complication can result in increased pressure on the brain and may require medical intervention to prevent further damage to the brain tissues. TBM can affect up to 85% of children with the disease. It tends to be more severe in children than in adults and can take the form of either communicating or obstructive types, although the former is more commonly observed. The Vellore grading system, which categorizes patients with TBM and hydrocephalus into four grades from best (grade I) to worst (grade IV), has been validated by various researchers. The management of hydrocephalus in TBM may involve medical therapies, such as dehydrating agents and steroids, particularly for patients with communicating hydrocephalus or those in good grades.

3. REVIEW METHOD

Initially I searched with keywords like Hydrocephalus due to Tuberculosis, Management of TBM and Subsequently the paper that matched with the topic were fully reviewed and their finding duly noted.

4. Tuberculosis Meningitis

Tuberculosis (TB) is a widespread infectious disease affecting over 2 billion people globally, which accounts for one-third of the world's population. Out of these, 10% of people with TB develop clinical symptoms, and the disease claims the lives of around 1.4 million people annually. Tuberculous meningitis (TBM) is a rare form of extrapulmonary TB that occurs in 1-5% of the approximately 10 million cases of TB worldwide. While TB is a less common cause of meningitis in developed countries such as the United States and Europe, it is still the most prevalent cause of chronic meningitis in endemic areas worldwide, particularly in people who are co-infected with HIV. The

bacteria responsible for TB, Mycobacterium tuberculosis, enter the body via inhalation of droplets, and the localized infection then spreads to the lungs and regional lymph nodes. In some cases, the bacteria can reach the central nervous system (CNS) and cause three distinct forms of CNS TB: tuberculous meningitis, intracranial tuberculoma, and spinal tuberculous arachnoiditis. Patients who are co-infected with HIV-1 have a mortality rate of up to 50% from TBM. Unfortunately, studying the pathogenesis of TBM is challenging due to the lack of experimental models that accurately mimic all aspects of the human disease. Additionally, the diagnostic process for TBM is often delayed due to the lengthy and insensitive culture techniques required for disease confirmation.

Current antibiotic regimens for treating TBM are based on those used to treat pulmonary tuberculosis, but these may not be entirely effective in treating TBM. The blood-brain barrier can hinder the delivery of drugs to the cerebrospinal fluid, resulting in suboptimal drug levels in the affected areas. Moreover, the efficacy of adjunctive anti-inflammatory, host-directed therapies, such as corticosteroids, aspirin, and thalidomide, has not been extensively studied.

5. Hydrocephalus due to Tuberculosis Meningitis and It's complication

Hydrocephalus in tuberculous meningitis can occur as either a communicating or obstructive type, with both being caused by inflammatory exudates blocking the subarachnoid spaces or ventricular pathways. The build-up of thick gelatinous exudates can block the subarachnoid spaces in the early stages of the disease, leading to communicating hydrocephalus. In the later stages, scarring of the subarachnoid spaces can cause the same type of hydrocephalus. Inflammation of the choroid plexus and ependyma can also lead to overproduction of cerebrospinal fluid in the acute phase. Obstructive hydrocephalus can develop when exudates or scar tissue block the fourth ventricular outlets or the aqueduct, or when there is a subependymal tuberculoma. The incidence of hydrocephalus is higher in children than adults, and it can occur at any point during the disease, though it is most commonly an early symptom. In paediatric cases of TBM, hydrocephalus is often present at the time of diagnosis.

Hydrocephalus can present as a primary symptom of tuberculous meningitis (TBM) or may develop after starting antituberculosis treatment. The hallmark pathological feature of TBM is the presence of thick gelatinous exudate that primarily affects the basal parts of the brain, encasing and constricting **cranial nerve trunks, optic nerve, optic chiasm, and vessels of the circle of Willis**. This exudate can also block the flow of cerebrospinal fluid (CSF) in the brain, leading to ventriculomegaly. **It can be challenging to differentiate between the two common types of hydrocephalus, communicating and obstructive,** using routine neuroimaging. Progressive hydrocephalus can lead to high intracranial pressure and potentially life-threatening complications, such as deteriorating vision and consciousness. In these cases, surgical CSF diversion procedures, such as ventriculoperitoneal shunt or endoscopic third ventriculostomy, may be necessary and can be life-saving. However, the long-term benefits of CSF diversion are not well understood.

Genetic variations in toll-interleukin-1 receptor domain and leukotriene A4 hydrolase (LTA4H) gene have been found to affect the risk of inflammation in TBM. In children, the cerebellum is a common site for tuberculomas, which can rarely progress to tuberculous encephalopathy, a condition associated with high mortality rates. Optochiasmatic arachnoiditis is more likely to develop in young females with high levels of protein in their cerebrospinal fluid. Spinal TB meningitis can present similarly to transverse myelitis or Guillain-Barre syndrome. Clinicians should seek an extra-neural focus of TB, as it may provide safer and more accessible sites for diagnostic sampling. Diagnostic tests like the cartridge-based nucleic acid amplification test (CBNAAT) and line probe assays have improved the detection of TB and drug-resistant strains. Since TBM is often difficult to diagnose definitively, empirical treatment for at least 9 months is recommended based on clinical judgement. Adjunctive corticosteroids should be given to all TBM patients, even those with HIV infection. The key principle of managing multidrug-resistant TB is to avoid adding a single drug to a failing regimen. Bedaquiline and delamanid have been approved for MDR TB treatment, but the correct combination and duration of most effective second-line drugs require further modifications. Early shunting should be considered for those with hydrocephalus that is unresponsive to medical management. The stage of TBM at which treatment is initiated is the single most important determinant of outcome.

6. Conclusion

Hydrocephalus is a common and potentially life-threatening complication of tuberculosis meningitis. It can occur at any stage of the disease and may present as a primary feature or develop later in the clinical course. There are two common types of hydrocephalus - communicating and obstructive - which can be difficult to differentiate on routine neuroimaging. The long-term benefits of CSF diversion procedures such as ventriculoperitoneal shunts or endoscopic third ventriculostomies are largely unknown, but they can be life-saving in patients with progressive hydrocephalus.

Early detection of hydrocephalus is essential to prevent irreversible brain damage and reduce the risk of mortality in TBM patients. Prompt diagnosis of TBM and initiation of appropriate antituberculosis treatment along with adjunctive corticosteroids can help in reducing the incidence of hydrocephalus. Radiological evaluation of the extra-neural focus of TB should be done to indicate safer and more accessible sites for diagnostic sampling. The use of rapid diagnostic tools such as CBNAAT and line probe assays can help in early detection of TB and rifampicin resistance, which is crucial for effective treatment.

Therefore, healthcare professionals should have a high index of suspicion for hydrocephalus in TBM patients and ensure early detection, appropriate treatment, and timely referral to neurosurgical services, if required.

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