

ORIGINAL ARTICLE

Clinical Spectrum and Presenting Features of Presumptive Tuberculous Meningitis in Children

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Introduction: Tuberculous meningitis (TBM) is a severe childhood tuberculosis that causes serious neurological problems and high death rates in low-resource settings. It is hard to diagnose early because symptoms are nonspecific and tests have low sensitivity with scarce CSF samples. Delays in diagnosis are common and lead to worse outcomes. **Objective:** This study aimed to describe the demographic, clinical, CSF, and imaging profiles and severity patterns in children with presumptive TBM. **Methods & Materials:** This descriptive cross-sectional study was carried out at Dhaka Medical College Hospital over one year, involving 96 children aged 1 month to 12 years with suspected TBM. The assessments encompassed clinical examination, GCS scoring, CSF cytology and biochemistry, brain MRI, and chest X-ray. **Results:** Children under 5 years made up 67.7%, with a mean age of 3.82 years, mostly male (1.5:1) and from rural areas (72.9%). Fever was universal (100%), followed by irritability (55.2%), altered consciousness (44.8%), and convulsions (42.7%). GCS indicated moderate (46.9%) and severe (17.7%) impairment. Severity was related to convulsions ($p=0.042$), altered consciousness ($p=0.001$), focal deficits ($p=0.008$), and neck rigidity ($p=0.045$). CSF showed lymphocytic dominance, high protein (87.8 mg/dl), and low glucose (51.2 mg/dl). MRI findings included basal enhancement (53.7%), infarcts (14.9%), and hydrocephalus (10.4%). Chest X-ray was normal in 71.9%. Contact history was noted in 17.7%. Only 30.2% were probable TBM, with 69.8% possible. **Conclusion:** Young males from rural families are at the highest risk. Persistent fever, altered consciousness, and seizures should alert clinicians in endemic areas. The prominence of TBM cases underscores the urgent need for better rapid diagnostics and targeted actions to cut mortality and prevent disabilities.

Keywords: Tuberculous meningitis; Children; Clinical presentation; Cerebrospinal fluid; Neuroimaging; Glasgow Coma Scale; Presumptive diagnosis; Bangladesh.

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INTRODUCTION

Tuberculous meningitis (TBM) is one of the most serious forms of tuberculosis in children with considerable morbidity and mortality worldwide. This condition is especially dangerous for small children, and late diagnosis and treatment often result in serious neurological complications, long-term disability, or death.^[1,2] When Mycobacterium tuberculosis invades the meninges and cerebrospinal fluid surrounding the brain and spinal cord, it initiates a profound inflammatory cascade that can lead to irreversible neurological damage.^[3] The most frequent clinical symptoms include fever, headache, vomiting, irritability, changes in behavior, and poor weight gain, which can at first be difficult to distinguish from other common childhood illnesses.^[4] As the disease progresses, neurological manifestations like altered consciousness, seizures, focal deficits, cranial nerve palsies (often sixth nerve), and raised intracranial pressure emerge.^[5]

The worldwide impact of tuberculosis remains profoundly significant. Every year, 10 million people fall ill with tuberculosis. It is the most common cause of death worldwide among infectious agents. Approximately 90% of cases occur in 30 high-burden countries, and an estimated 1.7 billion people are infected globally with M. tuberculosis and are at risk of developing active disease. It affects different age groups, with children representing about 11% of cases in 2018.^[6] In 2015, there were an estimated 10.4 million new cases of TB worldwide, of which about 1 million were children. In Bangladesh, the estimated incidence rate was 225 per 100,000 population, and the estimated TB-attributed deaths were 45 per 100,000 population in the same year.^[7] Global frameworks have been developed to combat TB and articulate international efforts. The Sustainable Development Goals (SDGs) set ambitious targets, including an 80% reduction in the incidence rate of TB (compared with the 2015 baseline) by 2030 and a 90% reduction in the absolute

number of TB deaths by the same year.^[8] Extrapulmonary tuberculosis accounts for about 0.8 million cases worldwide, and TBM is the most dangerous type.^[9] The incidence and prevalence of childhood tuberculosis are high in developing countries, especially in Southeast Asia, where TBM continues to be the commonest cause of TB-related death.^[5] In this part of the world, childhood TB accounts for an estimated 10–20% of all TB cases and 8–20% of TB mortality, with extrapulmonary disease making up a quarter of pediatric cases.^[10] Children differ from adults in the natural history and clinical manifestations of tuberculosis, with a higher incidence of disseminated forms and a higher risk of progression to severe disease.^[11] 1–5% of all new cases of tuberculosis are attributed to TBM, but it causes death or significant disability in more than half of the affected patients.^[12]

There are a number of difficulties in diagnosing TBM in children. Many patients are diagnosed late, as initial signs are nonspecific and there is a lack of rapid, sensitive diagnostic tests.^[5] Standard diagnostic tests for M. tuberculosis have poor sensitivity and are associated with delayed diagnosis and treatment, which is strongly associated with poor outcomes.^[13] Confirmation of TBM is particularly difficult in young children because of the paucibacillary nature of the disease and small volumes of cerebrospinal fluid (CSF) available for diagnostic evaluation.^[14] This diagnostic gap is mainly due to the lack of highly sensitive, rapid, and accessible diagnostic tests.^[15] The cheapest and most widely available test is CSF smear microscopy with Ziehl-Neelsen staining for acid-fast bacilli, but this has poor sensitivity, often $\leq 15\%$ in routine care without expert microscopists.^[12,16] In some settings, sensitivity can be as high as 50%, but this is still suboptimal.^[14] CSF culture is more sensitive (30–60%) but takes at least two weeks to yield results, which is too slow to be clinically useful and is not readily available in most low-income, TB-endemic settings.^[12]

Early diagnosis and prompt treatment of presumptive TBM in children rely on recognizing clinical features, cerebrospinal fluid (CSF) findings, and neuro-imaging, especially when microbiological confirmation is challenging. This study aims to describe the clinical spectrum and presentation to improve early recognition and management of this life-threatening condition.

OBJECTIVE

General Objective:

To describe the clinical spectrum and presenting features of presumptive TBM in children.

Specific Objectives:

1. To evaluate the demographic characteristics (age, gender, residence, socioeconomic level) of children suspected of having TBM.
2. To assess the prevalence of clinical symptoms and signs, as well as their correlation with the severity of the disease.
3. To observe the CSF and neuroimaging results and classify cases as either possible or probable TBM, ensuring a clear understanding of the findings and helping guide further steps.

METHODS & MATERIALS

This cross-sectional study was carried out over one year at the Department of Paediatrics, Dhaka Medical College & Hospital (DMCH), Dhaka, Bangladesh. It included 96 children aged 1 month to 12 years presenting with clinical signs suggestive of tuberculous meningitis (TBM). Exclusion criteria encompassed children with confirmed alternative diagnoses, those already receiving anti-tuberculous treatment, immunocompromised patients (excluding HIV), and guardians who did not give consent. Each child underwent a comprehensive clinical assessment, including demographic information (age, gender, residence, socioeconomic status), symptom history (fever, irritability, altered consciousness, convulsions, vomiting, headache, weight loss, night sweats), and contact with TB patients. Neurological status was graded using the Glasgow Coma Scale (GCS), categorizing impairment as mild^[13–15], moderate^[9–12], or severe^[3–8], alongside physical examinations for focal deficits, neck rigidity, bulging fontanelle, and BCG scar presence. Cerebrospinal fluid (CSF) was collected via lumbar puncture for analysis of physical appearance, total white blood cell (WBC) and lymphocyte counts, and biochemistry parameters such as protein, glucose, and ADA. Neuroimaging, primarily brain MRI in 67 patients, aimed to detect abnormalities like basal meningeal enhancement, infarcts, hydrocephalus, and tuberculomas; all 96 patients also underwent chest X-rays to identify pulmonary signs like patchy opacities or hilar lymphadenopathy. Cases were classified as "probable TBM," which was identified based on microbiological confirmation or strong clinical evidence, or "possible TBM" (suggestive but lacking definitive microbiological proof) based on combined clinical, laboratory, and imaging data. Data analysis included appropriate statistical methods: continuous variables were expressed as mean $\pm$ SD, categorical data as frequencies and percentages, and chi-square tests examined links between clinical features and disease severity ($p < 0.05$). The study received ethical clearance from the Institutional Review Board of Dhaka Medical College. Guardians provided written informed consent in both English and Bangla, with confidentiality maintained and ethical standards upheld throughout.

RESULT

This cross-sectional study involved 96 patients at the Department of Pediatrics, Dhaka Medical College & Hospital (DMCH), Dhaka, Bangladesh. Participants were children aged 1 month to 12 years exhibiting clinical signs suggestive of tuberculous meningitis (TBM). The results of this study show the demographic profile, clinical examination, GCS scoring, CSF cytology and biochemistry, brain MRI, and chest X-ray.

Table 1 shows the demographic profile of the cases. The maximum number of cases were in the age group of 1 month to < 5 years (67.5%). The mean age was 3.82 ± 3.42 years, with a range of 2 months to 11 years. Males were 1.5 times as many as females (60% vs 40%). The maximum monthly family income of patients was in the range of Tk.10,000 – Tk. 20,000, followed by the range of Tk. 20,001 – Tk. 30,000 (20.0%) and Tk. < 10,000 (7.5%). Residence (living area) of the cases. Most patients came from rural Areas (72.5% vs 27.5%).

Table I: Distribution of study cases by demographic profile (n=96)

Demographic Variable	Frequency	Percentage (%)
Age Group		
1 month to <5 years	65	67.7
5 years to 12 years	31	32.3
Mean $\pm$ SD		3.82 $\pm$ 3.42 years
Range		2 months – 11 years
Gender		
Male	58	60.4
Female	38	39.6
Male: Female Ratio	1.5:1	
Residence		
Urban	26	27.1
Rural	70	72.9
Monthly Family Income (Tk)		
<10,000	7	7.3
10,000-20,000	67	69.8
20,001-30,000	19	19.8
>30,000	3	3.1

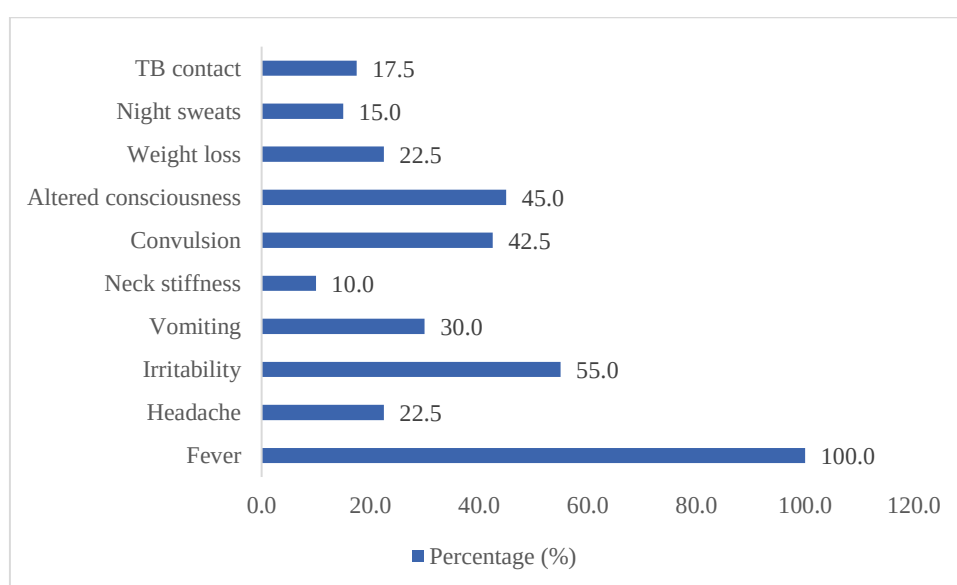

Figure 1: Symptoms of Suspected Tuberculous Meningitis Patients (n=96)

Figure 1 illustrates the clinical symptom profile of the patients at the time of presentation. Fever was the universally observed presenting symptom, reported in all 96 cases (100%). Irritability was the second most common complaint, identified in 55.2% (n=53) of the children, followed closely by altered consciousness and convulsions, which were present in 44.8% (n=43) and 42.7% (n=41) of cases, respectively. Vomiting was documented in 30.2% (n=29) of the patients, while headache and weight loss were each noted in 22.9% (n=22). A history of contact with a known tuberculosis patient was obtained in only 17.7% (n=17) of cases, and night sweats were the least common symptom, occurring in 14.6% (n=14) of the children.

Table II presents the findings from the physical and neurological exams of the study participants. On average, the

recorded temperature was $99.8 \pm 0.86^{\circ}\text{F}$, indicating that most patients experienced a mild fever. The Glasgow Coma Scale (GCS) assessment revealed that 35.4% (n=34) had mild impairment (GCS 13-15), 46.9% (n=45) showed moderate impairment (GCS 9-12), and 17.7% (n=17) faced severe neurological challenges (GCS 3-8). Signs of focal neurological issues such as hemiparesis and aphasia were seen in 39.6% (n=38), and neck stiffness, a sign of meningeal irritation, was observed in 17.7% (n=17). A bulging fontanelle, which can indicate increased pressure inside the skull, was noted in 10.4% (n=10). Most importantly, 77.1% (n=74) of the patients had a BCG vaccination scar, highlighting good vaccination coverage, although it didn't fully prevent TB meningitis.

Table II: Clinical Signs on Examination (n=96)

Clinical Signs	Frequency	Percentage (%)
Temperature (Mean $\pm$ SD)		99.8 $\pm$ 0.86°F
Glasgow Coma Scale (GCS)		
GCS 13-15	34	35.4
GCS 9-12	45	46.9
GCS 3-8	17	17.7
Neck rigidity	17	17.7
Focal neurological deficits	38	39.6
Bulged fontanelle	10	10.4
BCG mark present	74	77.1

Table 3: Distribution by Severity (GCS) and Its Correlation with Clinical Features (n=96)

Clinical Feature	GCS 13-15 (n=34)	GCS 9-12 (n=45)	GCS 3-8 (n=17)	p-value
Convulsion	10 (29.4%)	20 (44.4%)	11 (64.7%)	0.042*
Altered consciousness	8 (23.5%)	22 (48.9%)	13 (76.5%)	0.001*
Focal neurological deficit	8 (23.5%)	18 (40.0%)	12 (70.6%)	0.008*
Neck rigidity	3 (8.8%)	8 (17.8%)	6 (35.3%)	0.045*

*Significant at $p < 0.05$ (Chi-square test).

Table III shows a significant correlation between disease severity (by GCS score) and neurological features. As severity worsened from GCS 13-15 to 3-8, patients with convulsions increased from 29.4% to 64.7% ($p=0.042$), altered consciousness from 23.5% to 76.5% ($p=0.001$), focal deficits

from 23.5% to 70.6% ($p=0.008$), and neck rigidity from 8.8% to 35.3% ($p=0.045$). The significance ($p < 0.05$) indicates a dose-response relationship between impairment and clinical signs.

Table IV: CSF Findings in Presumptive TBM Patients (n=96)

CSF Parameter	Category / Value	RESULT
Physical Appearance	Clear	72 (75.0%)
	Hazy	22 (22.9%)
	Cob-web	2 (2.1%)
Cytology (Mean $\pm$ SD)	WBC (/cumm)	49.6 $\pm$ 62.1
	Lymphocytes (/cumm)	29.1 $\pm$ 36.5
Biochemistry (Mean $\pm$ SD)	Protein (mg/dl)	87.8 $\pm$ 78.1
	Glucose (mg/dl)	51.2 $\pm$ 19.0
	ADA (U/L)	3.87 $\pm$ 3.21

Table IV shows that the CSF analysis showed features of tuberculous meningitis: most fluids were clear (75%), with elevated proteins (mean 87.8 mg/dl) and low glucose (mean 51.2 mg/dl). Cytology revealed lymphocytic predominance (mean 29.1 cells/cumm), characteristic of TBM. CSF findings

included clarity in 75%, hazy in 22.9%, and cob-web in 2.1%. The total WBC count averaged 49.6 $\pm$ 62.1 cells/cumm, with lymphocytes as the main component. Biochemically, proteins were elevated and glucose low, supporting TBM diagnosis. The CSF ADA level averaged 3.87 $\pm$ 3.21 U/L.

Table V: Imaging and Case Classification (n=96)

Variable	Category	Frequency (n)	Percentage (%)
MRI Brain (n=67)	Normal	22	32.8
	Basal Meningeal Enhancement	36	53.7
	Infarct	10	14.9
	Hydrocephalus	7	10.4
	Tuberculoma	2	3
Chest X-ray (n=96)	Normal	69	71.9
	Patchy Opacity	17	17.7
	Hilar Lymphadenopathy / Other	10	10.4
TBM Classification	Possible TBM	67	69.8
	Probable TBM	29	30.2

Table V shows neuroimaging and clinical classification. MRI on 67 patients revealed basal meningeal enhancement (53.7%), infarcts (14.9%), hydrocephalus (10.4%), and tuberculomas (3.0%). No abnormality was seen in 32.8%. Chest X-ray in 96 patients was mostly normal (71.9%), with patchy opacity

(17.7%) and hilar lymphadenopathy (10.4%). Overall, 69.8% (n=67) had 'Possible TBM' and 30.2% (n=29) 'Probable TBM,' highlighting diagnostic challenges due to often lacking microbiological confirmation, requiring high suspicion and a multi-domain scoring approach.

DISCUSSION

Tuberculous meningitis (TBM) is a severe form of tuberculosis in children, with high risks of death and neurological

disability. Early diagnosis and treatment are crucial, but diagnosing pediatric TBM is difficult due to its nonspecific symptoms, low bacterial presence, and limited test

sensitivity.^[5,14] This study observed 96 children aged 1 month to 12 years at Dhaka Medical College Hospital over a year, providing insights into demographics, clinical signs, CSF, neuroimaging, and disease classification to aid clinicians in resource-limited settings.

The study shows that tuberculous meningitis (TBM) mostly affects children under five, with 67.7% of cases. This aligns with the understanding that TBM disproportionately impacts young children, whose immune systems and blood-brain barrier are still developing.^[4] The mean age of 3.82 ± 3.42 years matches previous high-burden studies, indicating children under five bear the greatest burden.¹⁷ The male predominance in our study (male-to-female ratio of 1.5:1) aligns with previous South Asian studies.^[18,19] Reasons for this disparity are unclear, but may include differences in healthcare-seeking behavior, biological susceptibility, and gender-based exposure variations. Hormonal differences might influence immune responses to *Mycobacterium tuberculosis*, but more research is needed.^[20] Social factors, like unequal healthcare access, could also contribute. Most patients (72.9%) lived in rural areas, reflecting Bangladesh's socio-demographic context where overcrowding, poor ventilation, and limited healthcare facilitate transmission.^[6] Rural residents have a significant relationship with delayed diagnosis, limited diagnostic facilities, and higher TB risk.^[21] Socioeconomic data show 77.1% of families earn less than 20,000 Taka monthly, highlighting poverty's role in TB burden through malnutrition, overcrowding, and reduced healthcare access.^[7]

The universal presence of fever (100%) in our study population emphasizes its role as a key feature of TBM in children, consistent with reports of fever as the most common symptom.^[22,23] Irritability (55.2%), reflecting neurological involvement, is prominent in young children who can't articulate symptoms, aligning with Sharma S et al.'s findings of irritability as an early sign of TBM in infants.^[24] Altered consciousness (44.8%) and seizures (42.7%) are comparable to other high-burden studies, indicating significant neurological morbidity.^[25] The elevated incidence of seizures may be attributed to cortical irritation, edema, and infarcts, with seizures associated with poorer outcomes, thereby necessitating rigorous management. Vomiting (30.2%), headache (22.9%), and weight loss (22.9%) were less common, possibly due to young age making symptoms like headache difficult to assess. Headache is harder to evaluate in pre-verbal children, and this may explain lower prevalence compared to adults, where it's reported in up to 80%.^[24] Weight loss, although underreported, indicates chronic illness and should prompt consideration of tuberculosis in children presenting with prolonged fever and failure to thrive. A contact history with TB was documented in only 17.7%, lower than the 30-50% seen elsewhere, possibly due to under-recognition, stigma, or unawareness among caregivers.^[25]

The Glasgow Coma Scale (GCS) assessment showed that nearly half of the patients (46.9%) had moderate neurological impairment (GCS 9-12), while 17.7% had severe impairment (GCS 3-8) at admission. These findings are concerning, indicating many children present with advanced disease, likely with irreversible neurological damage. The GCS score distribution in our cohort is similar to van Toorn et al., who found about 60% of children with TBM had GCS scores < 13 at presentation in South Africa.^[26] This late presentation highlights diagnostic challenges and limited healthcare access in resource-limited settings. Focal neurological deficits appeared in 39.6% of patients, often indicating cerebral

infarction due to vasculitis of small arteries at the brain's base.^[27] This rate aligns with 30-50% in the literature and emphasizes the need for prompt anti-tuberculous therapy to prevent neurological decline.^[28] The relation between focal deficits and disease severity ($p=0.008$) suggests early detection and treatment could reduce permanent neurological damage. Neck rigidity, a common sign of meningeal irritation, was present in only 17.7%, much lower than the 50-70% in adult TBM reports.^[29] This lower prevalence may be due to pediatric anatomical differences and difficulty eliciting the sign in distressed children. Absence of neck rigidity doesn't exclude TBM, as children often show atypical symptoms, emphasizing the need for high suspicion and early diagnostic testing. A high proportion of children (77.1%) had BCG scars, reflecting Bangladesh's robust vaccination coverage. However, while BCG helps prevent disseminated TB, it offers limited protection against TBM, with effectiveness estimated at 50-70%. Breakthrough cases still occur, especially in high-transmission areas, so clinicians must remain vigilant for TBM despite vaccination.^[30]

Our study reveals a significant link between GCS scores and various neurological features, offering valuable insights into TBM's clinical spectrum. As disease severity increased from mild (GCS 13-15) to moderate (GCS 9-12) and severe (GCS 3-8), there was a proportional rise in the occurrence of convulsions (29.4% to 64.7%, $p=0.042$), altered consciousness (23.5% to 76.5%, $p=0.001$), focal neurological deficits (23.5% to 70.6%, $p=0.008$), and neck rigidity (8.8% to 35.3%, $p=0.045$). These significant associations affirm a direct relationship between clinical symptoms and CNS involvement, validating GCS as a marker of disease severity. The high significance between altered consciousness and severity ($p=0.001$) indicates its potential as the most sensitive indicator of advanced disease. This proves the need for prompt diagnosis and intervention for children with altered consciousness, fever, and other symptoms. The correlation between focal neurological deficits and severity ($p=0.008$) emphasizes TBM's progressive nature and the need for swift treatment to avert lasting neurological damage. Although neck rigidity was also associated with severity ($p=0.045$), its correlation was weaker, likely due to lower prevalence and assessment difficulties in children.

Our CSF findings align with typical TBM characteristics. Most samples were clear (75%), with 22.9% hazy and 2.1% with cob-web formation, a classic but rare TBM feature.^[31] Cytology showed lymphocytic dominance (mean 29.1 lymphocytes/cumm) and a total WBC count of 49.6 cells/cumm. Early in TBM, polymorphonuclear cells may predominate, mimicking bacterial meningitis.^[32] Biochemically, CSF protein was elevated (mean 87.8 mg/dl), and glucose was reduced (mean 51.2 mg/dl), reflecting inflammation and bacterial activity. The protein rise results from increased permeability and exudation; low glucose reflects mycobacterial metabolism and immune cell consumption, though less pronounced than in bacterial meningitis.^[33] CSF ADA averaged 3.87 U/L, lower than the 5-10 U/L suggested in some adult studies. Factors like lab differences, disease stage, and age may influence ADA levels. While ADA can aid TBM diagnosis, its value in children is uncertain, and no clear cutoff exists.^[34] Our low ADA levels suggest limited diagnostic utility in pediatric cases, warranting further research.

Neuroimaging findings showed basal meningeal enhancement was the most common abnormality on MRI (53.7%), aligning with TBM's preference for basal cisterns where inflammatory exudate accumulates.^[35] The presence in over half of patients

emphasizes MRI's role in diagnosing suspected TBM, especially with inconclusive CSF. Notably, 32.8% had normal MRI findings, showing neuroimaging limitations, as early or mild TBM may not show abnormalities.^[36] Cerebral infarcts occurred in 14.9% of patients, lower than the 20-40% reported elsewhere, possibly due to milder disease severity, as infarcts often develop in advanced vasculitis cases. Hydrocephalus (10.4%) and tuberculomas (3.0%) are known TBM complications, requiring close monitoring and prompt intervention, as hydrocephalus can quickly cause brain herniation.^[37] Chest X-ray was normal in 71.9%, consistent with TBM often occurring without lung involvement.^[38] When abnormal, patchy opacities (17.7%) and hilar lymphadenopathy (10.4%) were most common. The mostly normal chest X-ray highlights that TBM can occur without pulmonary TB, with extrapulmonary spread possible without a primary focus. Patients were classified as possible TBM (69.8%) and probable TBM (30.2%), reflecting diagnostic challenges. The high proportion of possible TBM cases underscores the limitations of current diagnostics and reliance on clinical judgment in resource-limited settings. Lack of microbiological confirmation in most patients raises concerns about delayed diagnosis and unnecessary treatments, but criteria like those from the British Infection Society can standardize diagnosis and aid decision-making.^[39]

LIMITATIONS

This study has several limitations. It was conducted at a single tertiary care center in Bangladesh, limiting generalizability to other settings with different epidemiology. The diagnosis of presumptive TBM was clinical, risking misclassification from other meningitis causes. Its retrospective nature may have led to incomplete data, especially for unrecorded variables. The lack of routine HIV testing is a major limitation, as HIV affects TBM presentation and outcomes; future studies should include systematic testing. The study also did not evaluate long-term outcomes like neurological sequelae and mortality, which are vital for assessing diagnostic and treatment effectiveness. Additionally, the small sample size may have reduced the power to identify certain associations.

CONCLUSIONS

This study found that young male children, rural residents, and those with low socioeconomic status are especially vulnerable to TBM. Symptoms like fever, irritability, altered consciousness, and convulsions should prompt suspicion, especially in endemic areas. Early detection and treatment are vital to prevent neurological damage. CSF and neuroimaging support diagnosis when microbiological confirmation isn't available. A significant number of suspected cases indicates diagnostic challenges, calling for better pediatric diagnostic tools. Clinicians should consider TBM in children with relevant symptoms and recent TB exposure, using CSF analysis and neuroimaging. Developing diagnostic algorithms and investing in rapid tests like molecular assays and biomarkers can improve early detection. Strengthening healthcare systems is key to reducing delays and improving outcomes. Preventive measures, including BCG vaccination and active case finding, are essential to reduce TBM's impact. Comprehensive strategies addressing diagnosis and prevention are crucial to lowering the global burden of pediatric TBM.

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