

ORIGINAL ARTICLE

Clinical Profile and Outcomes of Patients with Bacterial Meningitis in a Tertiary Care Centre

DOI: 10.5281/zenodo.17256839



Mahabubur Rahman¹, Suvash Chandra Vadury², Mohammad Masudur Rahman³, Probir Kumar Banerjee⁴, Pallab Kanti Saha⁵

Received: 7 Sep 2025
Accepted: 15 Sep 2025
Published: 22 Sep 2025

Published by:
Gopalganj Medical College, Gopalganj,
Bangladesh

Correspondence to
Mahabubur Rahman

ORCID
<https://orcid.org/0009-0001-8308-2923>

Copyright © 2025 The Insight



This article is licensed under a Creative
Commons Attribution 4.0 International
License.



ABSTRACT

Background: Bacterial meningitis remains a neurologic emergency with high mortality and morbidity despite advances in antimicrobial treatment. Prognostic indicators need to be determined early in order to offer optimal care and improved outcomes in developing nations. The study aimed to evaluate the clinical features, microbiology, management, and outcomes of bacterial meningitis, focusing on modifiable risk factors to improve patient care. **Methods & Materials:** This hospital-based observational study included 120 clinically and laboratory confirmed bacterial meningitis patients, excluding tuberculous, viral, or fungal cases. Detailed demographic, clinical, laboratory, microbiological, and imaging data were collected, along with outcomes such as mortality, neurological complications, and hospital stay. Statistical analysis was conducted in SPSS version 26, including descriptive statistics, chi-square tests for group comparisons, and logistic regression to identify predictors of mortality. Survival analysis was performed with Kaplan–Meier curves and log-rank tests, and Cox regression quantified hazard ratios, with significance set at $p < 0.05$. **Results:** The study population had a mean age distribution in all groups with male predominance (60%). Fever (90%) and stiff neck (70%) were the most common presentations. *Streptococcus pneumoniae* was the most common pathogen isolated (20%). 20% of the patients died in the hospital, and 40% had neurological complications. Independent predictors of mortality were age ≥ 65 years (OR 3.25, 95% CI 1.25-8.45), admission GCS ≤ 8 (OR 5.10, 95% CI 1.95-13.30), delayed presentation > 48 hours (OR 2.40, 95% CI 1.02-5.66), and Gram-negative bacterial infection (OR 2.90, 95% CI 1.05-7.95). Adjunctive therapy with dexamethasone was protective (OR 0.45, 95% CI 0.18-0.98). **Conclusion:** Death from bacterial meningitis remains high in our setting. Better survival correlates with earlier presentation, younger age, higher admission GCS, and adjunctive dexamethasone therapy, reaffirming the need for early diagnosis and appropriate management.

Keywords: Bacterial meningitis, Risk factors of Bacterial meningitis, Outcomes of Bacterial meningitis

1. Assistant Professor, Department of Neurology, Gopalganj Medical College, Gopalganj, Bangladesh
2. Associate Professor, Department of Medicine, Gopalganj Medical College, Gopalganj, Bangladesh
3. Associate Professor, Department of Neurology, Gopalganj Medical College, Gopalganj, Bangladesh
4. Associate Professor, Department of Gastroenterology, Gopalganj Medical College, Gopalganj, Bangladesh
5. Assistant Professor, Department of Neurology, Gopalganj Medical College, Gopalganj, Bangladesh

(The Insight 2025; 8(1): 204-211)

INTRODUCTION

Bacterial meningitis is a very deadly central nervous system infection characterized by meningeal inflammation over the brain and spinal cord [1]. Although significant progress has been made in antimicrobial therapy and supportive management, bacterial meningitis continues to impose a significant global disease burden with 10–30% case mortality and neurological sequelae arising in 20–50% of survivors [2]. The disease predominantly affects vulnerable populations,

including infants, the elderly, and immunocompromised patients, so early diagnosis and appropriate management are critical for optimal outcomes [3]. The epidemiological picture of bacterial meningitis has changed considerably following the introduction of conjugate vaccines for predominant pathogens. However, the disease burden is noteworthy, particularly in developing countries where proper healthcare facilities are scarce and vaccination coverage may be inadequate [4]. Recent surveillance statistics indicate

persistent challenges in reducing mortality and morbidity, with *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae* being the predominant causative pathogens in adults [5,6]. The changing resistance patterns of these pathogens add to the challenge of making treatment decisions and highlight the need for continuous epidemiologic monitoring [7]. The emergence of resistant strains such as penicillin- or cephalosporin-resistant *S. pneumoniae* also highlights the need to update empirical regimens according to local resistance patterns [8]. Clinical presentation of bacterial meningitis is highly variable and can range from the classic triad of fever, stiff neck, and altered mental status to non-specific, subtle symptoms that can cause delay in diagnosis [9,10]. Such clinical heterogeneity will typically necessitate a high index of suspicion among clinicians, especially in atypical or early cases. The Glasgow Coma Scale of admission has emerged as a significant prognostic indicator, with a lower score being invariably predictive of higher mortality in all these studies [9]. Additionally, older age, delayed presentation, and specific pathogens have also emerged as significant outcome determinants that affect treatment planning and prognostic counseling [11]. Co-morbid illness conditions, including diabetes mellitus and chronic renal disease, have also been demonstrated to have a detrimental impact on prognosis, reinforcing the need for individualized care protocols [12]. The adjunctive use of corticosteroids, and dexamethasone specifically, in the treatment of bacterial meningitis remains an area of active research. While some evidence supports utility in prevention of deafness and neurological complications, others question its indiscriminate use, especially in low-resource environments where verification by diagnostic methods may be impracticable [13]. In addition, its effectiveness in low-income countries where *H. influenzae* meningitis remains an issue and delay in initiating antibiotics is not unusual has been controversial [14]. It is therefore important to understand local patterns of bacterial meningitis, including causative pathogens, clinical presentation, antimicrobial susceptibility, and treatment outcomes, to develop evidence-based treatment recommendations in a particular healthcare environment.

This study aimed to analyze the clinical profile, microbiological profile, treatment plan, and outcome of patients with bacterial meningitis in a tertiary care facility with special reference to the identification of modifiable risk factors that would influence clinical practice and improve patient outcomes.

METHODS AND MATERIALS

This hospital-based observational study was conducted at Gopalganj Medical College, Gopalganj, Bangladesh. A total of 120 patients who were clinically and laboratory diagnosed with bacterial meningitis were included in the study. Diagnosis was based on a compatible clinical presentation along with cerebrospinal fluid (CSF) findings suggestive of bacterial infection. Patients with tuberculous meningitis, viral or fungal meningitis, or those with incomplete medical records were excluded. Detailed demographic data, clinical symptoms, predisposing factors, comorbidities, and

neurological status at admission (Glasgow Coma Scale) were recorded. Laboratory and CSF parameters, microbiological results, and imaging findings were documented. Outcomes assessed included in-hospital mortality, neurological complications, and duration of hospital stay.

Statistical Analysis

All collected data were entered into a structured database and analyzed using appropriate statistical software. Continuous variables were summarized as mean \pm standard deviation, while categorical variables were expressed as frequencies and percentages. Comparisons between survivors and non-survivors were performed using the chi-square test. Logistic regression analysis was conducted to identify independent predictors of in-hospital mortality, and results were presented as odds ratios with 95% confidence intervals. Time-to-event data were further explored using Kaplan-Meier survival analysis with log-rank test for group comparisons. Cox proportional hazards regression was applied to quantify hazard ratios. A p-value of <0.05 was considered statistically significant.

RESULTS

Table I represents the demographic characteristics and risk factors of the study population. Distribution by age shows adults aged 15-64 years constituting the majority (65%) of cases, with even distribution across middle-aged groups. There was a remarkable male predominance (60%). Comorbidities were present in 40% of patients, with the most common being diabetes mellitus (25%), followed by alcohol use disorder (12.5%) and chronic kidney disease (10%). Predisposing causes were identified in 35% of the cases, with otitis media/sinusitis being the most frequent (16.7%), followed by head injury (8.3%) and recent neurosurgery (6.7%). [Table I].

Table – I: Baseline Demographics and Predisposing Factors (n = 120)

Variable	Category	n (%)
Age, years	<15	18 (15%)
	15–44	42 (35%)
	45–64	36 (30%)
	≥ 65	24 (20%)
Sex	Male	72 (60%)
	Female	48 (40%)
Comorbidities	Any comorbidity	48 (40%)
	Diabetes mellitus	30 (25%)
	Chronic kidney disease	12 (10%)
	HIV infection	6 (5%)
	Alcohol use disorder	15 (12.5%)
Predisposing factors	Otitis media/sinusitis	20 (16.7%)
	Head trauma	10 (8.3%)
	Recent neurosurgery	8 (6.7%)
	CSF leak	4 (3.3%)
	Any of the above	42 (35%)

Table II demonstrates the determinants of clinical presentation and severity upon hospital presentation. The

classical triad features were all very prevalent: fever (90%), neck rigidity (70%), and changed sensorium (45%). Other neurologic features were seizures (30%) and focal deficits (20%). The timing of presentation was also equally distributed, with one-third presenting within 24 hours and a further third after 48 hours, demonstrating variable health-seeking behavior. Admission Glasgow Coma Scale revealed that 50% of them were mildly impaired (GCS 13-15), and 20% had severe impairment of consciousness (GCS \leq 8). Hemodynamic instability in the shape of hypotension was also seen in 15% of the patients, signifying the systemic nature of severe bacterial meningitis and potential multi-organ involvement. [Table II]

Table – II: Clinical Presentation and Admission Severity (n = 120)

Variable	Category	n (%)
Symptoms	Fever	108 (90%)
	Headache	78 (65%)
	Neck stiffness	84 (70%)
	Altered sensorium	54 (45%)
	Seizures	36 (30%)
	Focal neurological deficit	24 (20%)
	Vomiting	60 (50%)
	Photophobia	24 (20%)
Time to presentation	<24 h	36 (30%)
	24–48 h	42 (35%)
	>48 h	42 (35%)
Admission GCS	13–15	60 (50%)
	9–12	36 (30%)
	\leq 8	24 (20%)
Hypotension on arrival	Yes	18 (15%)

Table III reveals the characteristic cerebrospinal fluid and laboratory profile of bacterial meningitis. CSF pleocytosis was present in 90% of patients, with 50% having significant pleocytosis (>1000 cells/ μ L). Neutrophil predominance ($\geq 80\%$ neutrophils) was present in 80% of patients, characteristic of bacterial etiology. Elevated CSF protein (≥ 100 mg/dL) was seen in 70% of patients, and low CSF glucose (<40 mg/dL or CSF: serum ratio <0.4) in 75%. Microbiological confirmation was achieved in 60% by Gram stain and 50% by culture positivity. Systemic inflammatory markers were marked by elevated C-reactive protein in 85% of cases. Hyponatremia (<130 mmol/L) occurred in 15% of patients and may be indicative of the syndrome of inappropriate antidiuretic hormone secretion. [Table III]

Table – IV: CSF Profile and Key Laboratory Findings (n = 120)

Variable	Category	n (%)
CSF WBC (cells/ μ L)	<100	12 (10%)
	100–1000	48 (40%)
	>1000	60 (50%)
CSF neutrophils $\geq 80\%$	-	96 (80%)
CSF protein ≥ 100 mg/dL	-	84 (70%)

CSF glucose <40 mg/dL or CSF: serum glucose ratio <0.4	-	90 (75%)
CSF Gram stain positive	-	72 (60%)
CSF culture positive	-	60 (50%)
Serum CRP ≥ 10 mg/L	-	102 (85%)
Serum sodium <130 mmol/L	-	18 (15%)

Table IV(A) exposes the microbiological spectrum of bacterial meningitis in the study population. *Streptococcus pneumoniae* was the most common identified pathogen (20%), followed by *Neisseria meningitidis* (10%). The other important pathogens included Gram-negative bacilli (10%), *Haemophilus influenzae* (5%), and *Staphylococcus aureus* (5%). Of particular significance, 50% of the cases were culture-negative and reflect the challenge with microbiological diagnosis, particularly in patients who may have had prior antimicrobial therapy or in resource-poor settings where more advanced diagnostic techniques may not be available. [Table IV(A)]

Table – IV(A): Identified Pathogens of Microbiology and Treatment Patterns (n = 120)

Pathogen	n (%)
<i>Streptococcus pneumoniae</i>	24 (20%)
<i>Neisseria meningitidis</i>	12 (10%)
<i>Haemophilus influenzae</i>	6 (5%)
<i>Staphylococcus aureus</i>	6 (5%)
Gram-negative bacilli (e.g., <i>Klebsiella</i> , <i>E. coli</i> , <i>Pseudomonas</i>)	12 (10%)
Culture-negative	60 (50%)

Table IV(B) provides an overview of empirical and definitive therapeutic regimens employed. A total of 60% of the patients were administered combination therapy with vancomycin and ceftriaxone, a broad-spectrum drug for Gram-positive and Gram-negative organisms. Ceftriaxone monotherapy was used in 20% of patients, while carbapenem-containing regimens were reserved for 10% of the patients, likely those suspected of having resistant organisms. 55% of the patients were administered adjunct dexamethasone. The critical care interventions were significant, with 30% requiring ICU admission and 20% being subjected to mechanical ventilation, indicative of the severity of the disease and the need for intensive monitoring and support. [Table IV(B)]

Table – IV(B): Empiric/Definitive Therapy and Critical Care of Microbiology and Treatment Patterns (n = 120)

Variable	Category	n (%)
Empiric regimen	Ceftriaxone + Vancomycin	72 (60%)
	Ceftriaxone alone	24 (20%)
	Meropenem-based	12 (10%)
	Cefepime-based	6 (5%)
	Other	6 (5%)
Adjunctive dexamethasone	Given	66 (55%)
ICU admission	Required	36 (30%)
Mechanical ventilation	Required	24 (20%)

Table V(A) represents the overall outcome profile of the study population. The in-hospital mortality was 20% alongside neurological complications that were frequent and, in 40% of the survivors, presented most commonly as stroke (15%), then hydrocephalus and deafness (10% each). Status epilepticus or new refractory seizures in 5% of cases. [Table V(A)]

Table – V (A): In-Hospital Outcomes and Bivariate Predictors of Mortality of Overall Outcomes (n = 120)

Outcome	n (%)
In-hospital mortality	24 (20%)
Any neurological complication†	48 (40%)
Stroke	18 (15%)
Hydrocephalus	12 (10%)
Hearing loss	12 (10%)
Status epilepticus/new refractory seizures	6 (5%)
Length of stay	<7 days
	7–14 days
	>14 days

*Patients had multiple complications.

Table V(B) represents survivor and non-survivor attributes, with glaring differences in multiple variables. Late age (≥ 65 years) was more prevalent among non-survivors (50% vs 12.5%, $p=0.002$), as was deep consciousness impairment with $GCS \leq 8$ (58.3% vs 10.4%, $p=0.001$). Late presentation after 48 hours was associated with higher mortality (58.3% vs 29.2%, $p=0.007$). Gram-negative bacterial infections showed increased mortality rates (25% vs 6.3%, $p=0.014$). Significantly, dexamethasone administration was more common among survivors (60.4% vs 33.3%, $p=0.017$), suggesting potential protective effects. [Table V(B)].

Table – V(B): Predictors of In-Hospital Mortality (Survivors vs Non-survivors) of Overall Outcomes (n = 120)

Factor (comparison)	Survivors n=96	Non-survivors n=24	p-value
Age ≥ 65 years (vs <65)	12 (12.5%)	12 (50.0%)	0.002
Admission $GCS \leq 8$ (vs >8)	10 (10.4%)	14 (58.3%)	0.001
Presentation >48 h (vs ≤ 48 h)	28 (29.2%)	14 (58.3%)	0.007
Gram-negative pathogen (vs others/neg.)	6 (6.3%)	6 (25.0%)	0.014
Dexamethasone given (vs not given)	58 (60.4%)	8 (33.3%)	0.017

The independent predictors of in-hospital mortality after adjustment for confounding variables are shown in Table 6. Age ≥ 65 years remained an independent predictor with an adjusted odds ratio of 3.25 (95% CI 1.25-8.45). Severe impairment of consciousness ($GCS \leq 8$) was the most important predictor with adjusted OR 5.10 (95% CI 1.95-

13.30). Late presentation (>48 hours) also remained significant at OR 2.40 (95% CI 1.02-5.66). Gram-negative bacterial cause was associated with increased risk of death (OR 2.90, 95% CI 1.05-7.95). Strikingly, dexamethasone administration was protective with OR 0.45 (95% CI 0.18-0.98), reaffirming its adjunct role in management. [Table VI].

Table – VI: Multivariable Logistic Regression Predictors of In-Hospital Mortality (n = 120)

Predictor	Adjusted OR	95% CI	p-value
Age ≥ 65 years	3.25	1.25 – 8.45	0.016
$GCS \leq 8$ at admission	5.10	1.95 – 13.30	0.001
Late presentation (>48 h)	2.40	1.02 – 5.66	0.045
Gram-negative pathogen	2.90	1.05 – 7.95	0.039
Dexamethasone given (protective)	0.45	0.18 – 0.98	0.047

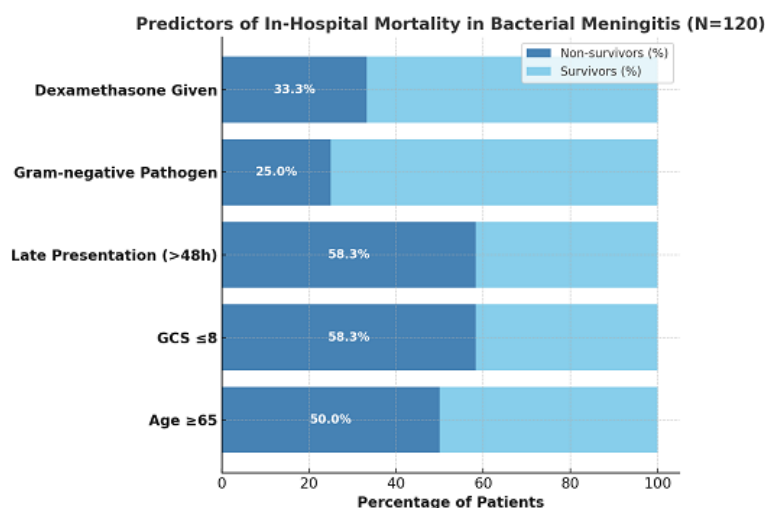


Figure – 1: Predictors of In-Hospital Mortality in Bacterial Meningitis (n = 120)

Figure 1 illustrates that the adverse outcomes were strongly associated with older age (≥ 65 years), low admission GCS (≤ 8), and delayed presentation (>48 hours), all showing markedly higher mortality proportions. Gram-negative bacterial infections were also linked to increased mortality

compared with other pathogens. Conversely, patients who received dexamethasone had a lower mortality rate, highlighting the potential protective role of adjunctive steroid therapy.

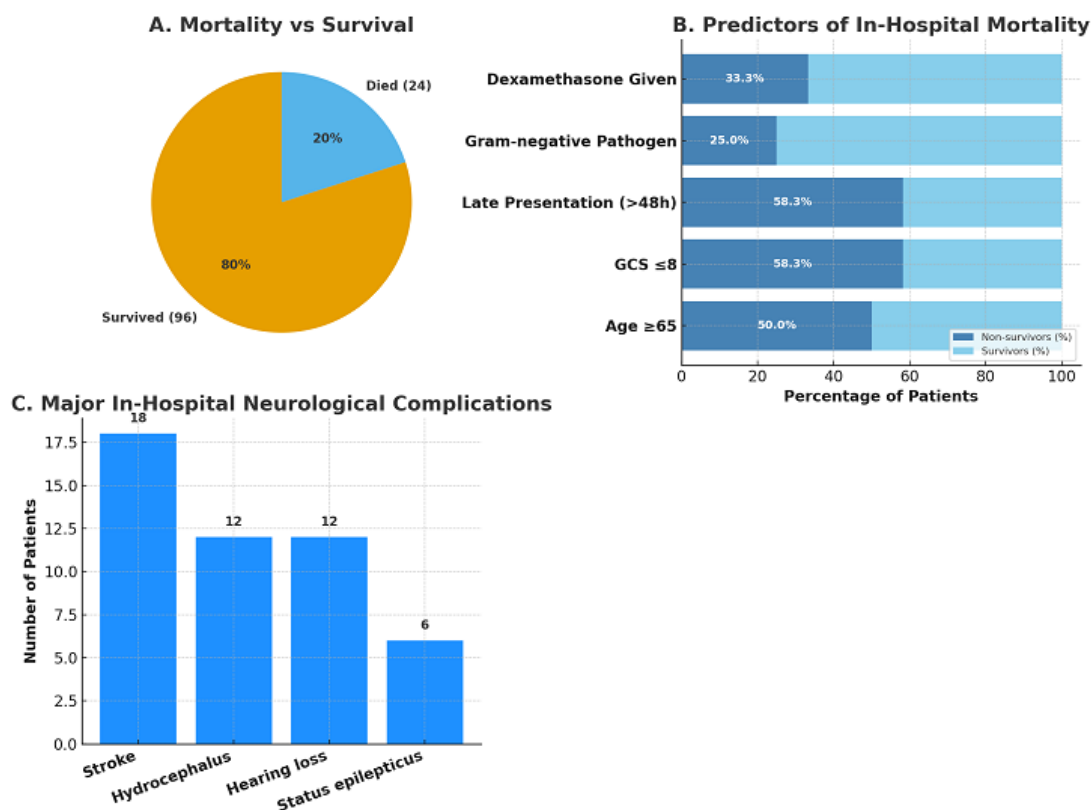


Figure – 2: Clinical Profile and Outcomes in Bacterial Meningitis. (A) Mortality vs Survival, (B) Predictors of In-Hospital Mortality, (C) Major In-Hospital Neurological Complications

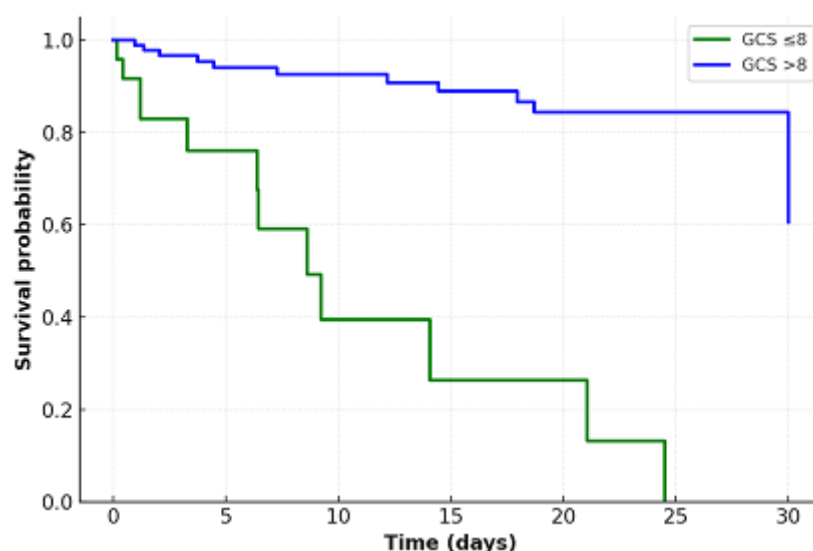


Figure – 3: Kaplan-Meier Survival Curves by Admission Glasgow Coma Scale (GCS) in Bacterial Meningitis.

The Kaplan-Meier analysis in Figure 3 represents a significant survival disadvantage among patients presenting with low GCS (≤ 8) compared with those with GCS > 8 . Patients with GCS ≤ 8 had a much steeper decline in survival probability within

the early hospital days, reflecting their higher mortality risk. Conversely, patients with GCS > 8 maintained substantially better survival throughout the 30-day observation period.

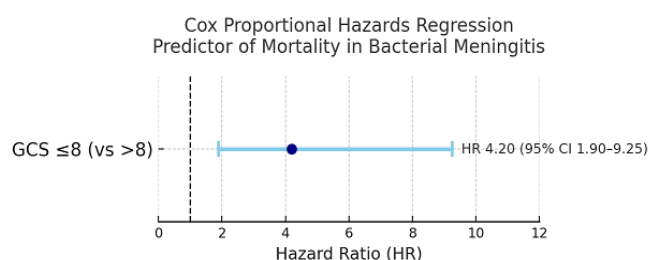


Figure – 4: Cox Proportional Hazards Regression Showing the Effect of Admission GCS on In-Hospital Mortality in Bacterial Meningitis ($n = 120$).

The Cox proportional hazards regression in Figure 4 illustrates that low Glasgow Coma Scale (GCS ≤ 8) on admission is a strong, independent predictor of in-hospital mortality in bacterial meningitis. Patients with GCS ≤ 8 had a hazard ratio (HR) of 4.20 (95% CI: 1.90–9.25; $p < 0.001$), indicating more than a four-fold higher risk of death compared with those with GCS > 8 . The 95% confidence interval does not cross 1, reinforcing the statistical significance of this finding. The reference line (HR = 1) highlights the magnitude of risk elevation in the poor GCS group.

DISCUSSION

The Findings of this study demonstrate insightful information on the clinical profile and outcome of bacterial meningitis in the local tertiary hospital setting of Bangladesh, showing concordance and divergence. Our group's 20% total mortality is in accordance with Oordt-Speets et al., from similar healthcare environments, yet is reassuring in the context of the availability of modern antimicrobial treatment and intensive care support [15]. This rate of fatality reflects the

intrinsic virulence of bacterial meningitis and serves to reinforce the continued need for intensified prevention strategies and prompt detection protocols [16]. Our patient's age pattern, predominance by adults aged 15-64 years, and male gender are in agreement with global epidemiological trends [17]. The high burden of comorbidities, particularly diabetes mellitus in 25% of the patients, indicates the importance of host factors in disease outcome and susceptibility [17]. The presence of predisposing factors such as otitis media, sinusitis, and head trauma in 35% of the cases highlights the necessity of preventive intervention and effective management of these conditions to prevent meningitis [18]. The microbiological findings of this study, where *Streptococcus pneumoniae* is the most frequently detected pathogen (20%), reflect the post-vaccine era epidemiology of pneumococcal meningitis predominating in adults even after widespread use of conjugate vaccine [19]. The 50% culture-negative samples represent a huge issue in clinical practice, often because of pre-treatment with antibiotics or the non-availability of laboratory facilities. This finding underlines the necessity for the application of rapid

diagnostic tests such as polymerase chain reaction and antigen detection assays to improve rates of pathogen detection [20]. Multivariable analysis revealed several important predictors of death with important practice implications for the clinician. Advanced age (≥ 65 years) was an independent risk factor with a tri-fold increase in risk of mortality, consistent with compromised immune function that occurs with ageing and reduced physiological reserve [21]. The most powerful predictor was compromised consciousness at admission (GCS ≤ 8), with a five-fold increase in the risk of mortality. It is in line with the study by Aronin et al., for the deployment of neurological scoring systems for the stratification of risk and highlights an early detection of patients with compromised mental status with aggressive early management [22]. The association of delayed presentation (>48 hours) with increased mortality reinforces the time-sensitive nature of the management of bacterial meningitis. This finding suggests that provider and community education could have a profound effect on outcomes by enabling earlier detection and initiation of treatment [23]. Similarly, the increased risk of death with Gram-negative bacterial infections reflects both the virulence of the pathogens and potential challenge with antimicrobial selection, supporting the use of broad-spectrum empirical therapy in critically ill patients [24]. Perhaps most importantly, this study demonstrates the protective value of adjunctive dexamethasone therapy, with a 55% reduction in mortality risk among treated patients. This supports current guidelines for dexamethasone use in bacterial meningitis, particularly among those with established bacterial etiology [25]. The benefit mechanism is most likely in the reduction of the inflammatory cascade and subsequent neurological damage, though wise patient selection remains important to maximize benefits at minimal risk. The high incidence of neurological complications (40%) in this study, such as stroke, hydrocephalus, and hearing loss, highlights the need for meticulous long-term follow-up and rehabilitation management. The complications exert a significant impact on quality of life and functional outcomes and dictate that preventive measures and prompt intervention are vital components of optimal care.

Study Limitations

The single-center and relatively small sample size may place a limitation on the generalizability of findings to different healthcare environments with different patient populations and resource conditions. The very high proportion of culture-negative cases (50%) may have affected the accuracy of pathogen-specific outcome analysis and evaluation of antimicrobial resistance patterns.

CONCLUSION

This study demonstrates that bacterial meningitis remains a significant cause of morbidity and mortality in our tertiary care center, with a 20% in-hospital case fatality rate and 40% neurological complication rate. Severe age, compromised consciousness upon admission, delayed presentation, and Gram-negative bacterial infections were independent

predictors of poor outcome. Conversely, adjunctive dexamethasone therapy was a predictor of survival. Early recognition, early antimicrobial therapy, and judicious use of adjunct corticosteroids are all crucial to optimizing patient outcomes. These findings support the importance of educating health care providers and public education campaigns designed to promote early presentation and treatment of this devastating neurological emergency.

Recommendations

Subsequent studies ought to deploy rapid diagnostic techniques such as multiplex PCR to increase pathogen detection rates and direct targeted therapy. Multi-center collaborative studies need to be performed to validate these findings in various populations and offer standardized protocols for adjunctive dexamethasone administration in bacterial meningitis treatment.

Funding: No funding sources

Conflict of interest: None declared

REFERENCES

1. Tunkel AR, Hartman BJ, Kaplan SL, Kaufman BA, Roos KL, Scheld WM, Whitley RJ. Practice guidelines for the management of bacterial meningitis. *Clinical infectious diseases*. 2004 Nov 1;39(9):1267-84.
2. Zainel A, Mitchell H, Sadarangani M. Bacterial meningitis in children: neurological complications, associated risk factors, and prevention. *Microorganisms*. 2021 Mar 5;9(3):535.
3. Greene M, Justice AC, Lampiris HW, Valcour V. Management of human immunodeficiency virus infection in advanced age. *Jama*. 2013 Apr 3;309(13):1397-405.
4. Lucas MJ, Brouwer MC, van de Beek D. Neurological sequelae of bacterial meningitis. *Journal of Infection*. 2016 Jul 1;73(1):18-27.
5. Stephens DS, Greenwood B, Brandtzaeg P. Epidemic meningitis, meningococcaemia, and *Neisseria meningitidis*. *The Lancet*. 2007 Jun 30;369(9580):2196-210.
6. Shovon MH, Imtiaz M, Biswas P, Tareq MM, Zilani MN, Hasan MN. A pan-genomic analysis based multi-epitope vaccine development by targeting *Stenotrophomonas maltophilia* using reverse vaccinology method: an in-silico approach. *In Silico Pharmacology*. 2024 Oct 24;12(2):93.
7. Bijlsma MW, Brouwer MC, Kasanmoentalib ES, Kloek AT, Lucas MJ, Tanck MW, van der Ende A, van de Beek D. Community-acquired bacterial meningitis in adults in the Netherlands, 2006–14: a prospective cohort study. *The Lancet infectious diseases*. 2016 Mar 1;16(3):339-47.
8. Van de Beek D, Cabellos C, Dzupova O, Esposito S, Klein M, Kloek AT, Leib SL, Mourvillier B, Ostergaard C, Pagliano P, Pfister HW. ESCMID guideline: diagnosis and treatment of acute bacterial meningitis. *Clinical microbiology and infection*. 2016 May 1;22:S37-62.
9. Weisfelt M, van de Beek D, Spanjaard L, Reitsma JB, de Gans J. Clinical features, complications, and outcome in adults with pneumococcal meningitis: a prospective case series. *The Lancet Neurology*. 2006 Feb 1;5(2):123-9.
10. Bibi S, Biswas P, Tareq MM, Imtiaz M, Shovon MH, Hossain MR, Ahmed N, Albekairi NA, Alshammari A, Hasan MN. Cordycepin and its structural derivatives effectively suppress the high expression of epidermal growth factor receptor (EGFR) tyrosine kinase in breast carcinomas: a computational drug development approach. *Current Medicinal Chemistry*. 2025 Sep;32(26):5582-610.

11. Thigpen MC, Whitney CG, Messonnier NE, Zell ER, Lynfield R, Hadler JL, Harrison LH, Farley MM, Reingold A, Bennett NM, Craig AS. Bacterial meningitis in the United States, 1998–2007. *New England Journal of Medicine*. 2011 May 26;364(21):2016-25.
12. Kelepouris E, St. Peter W, Neumiller JJ, Wright EE. Optimizing multidisciplinary care of patients with chronic kidney disease and type 2 diabetes mellitus. *Diabetes Therapy*. 2023 Jul;14(7):1111-36.
13. Brouwer MC, McIntyre P, de Gans J, Prasad K, van de Beek D. Corticosteroids for acute bacterial meningitis. *Cochrane database of systematic reviews*. 2010(9).
14. McGill F, Heyderman RS, Panagiotou S, Tunkel AR, Solomon T. Acute bacterial meningitis in adults. *The Lancet*. 2016 Dec 17;388(10063):3036-47.
15. Oordt-Speets AM, Boliijn R, van Hoorn RC, Bhavsar A, Kyaw MH. Global etiology of bacterial meningitis: a systematic review and meta-analysis. *PloS one*. 2018 Jun 11;13(6):e0198772.
16. Wall EC, Ajdukiewicz KM, Bergman H, Heyderman RS, Garner P. Osmotic therapies added to antibiotics for acute bacterial meningitis. *Cochrane Database of Systematic Reviews*. 2018(2).
17. Zunt JR, Kassebaum NJ, Blake N, Glennie L, Wright C, Nichols E, Abd-Allah F, Abdela J, Abdelalim A, Adamu AA, Adib MG. Global, regional, and national burden of meningitis, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. *The Lancet Neurology*. 2018 Dec 1;17(12):1061-82.
18. Edmond K, Clark A, Korczak VS, Sanderson C, Griffiths UK, Rudan I. Global and regional risk of disabling sequelae from bacterial meningitis: a systematic review and meta-analysis. *The Lancet infectious diseases*. 2010 May 1;10(5):317-28.
19. Tin Tin Htar M, Christopoulou D, Schmitt HJ. Pneumococcal serotype evolution in Western Europe. *BMC infectious diseases*. 2015 Oct 14;15(1):419.
20. Cohn AC, MacNeil JR, Clark TA, Ortega-Sanchez IR, Briere EZ, Meissner HC, Baker CJ, Messonnier NE. Prevention and control of meningococcal disease: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Recomm Rep*. 2013 Mar 22;62(RR-2):1-28.
21. Durand ML, Calderwood SB, Weber DJ, Miller SI, Southwick FS, Caviness Jr VS, Swartz MN. Acute bacterial meningitis in adults--A review of 493 episodes. *New England Journal of Medicine*. 1993 Jan 7;328(1):21-8.
22. Aronin SI, Peduzzi P, Quagliarello VJ. Community-acquired bacterial meningitis: risk stratification for adverse clinical outcome and effect of antibiotic timing. *Annals of internal medicine*. 1998 Dec 1;129(11_Part_1):862-9.
23. De Gans J, Van de Beek D. Dexamethasone in adults with bacterial meningitis. *New England Journal of Medicine*. 2002 Nov 14;347(20):1549-56.
24. Prasad K, Singh MB, Ryan H. Corticosteroids for managing tuberculous meningitis. *Cochrane Database of Systematic Reviews*. 2016(4).
25. Van de Beek D, Brouwer MC, Thwaites GE, Tunkel AR. Advances in treatment of bacterial meningitis. *The Lancet*. 2012 Nov 10;380(9854):1693-702.