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Neurological Complications of Tuberculosis in Quetta, Pakistan: Clinical Profile, Outcomes, and Mortality Predictors from a Prospective Cohort Study

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ABSTRACT

Background: Neurotuberculosis remains a severe manifestation of tuberculosis with high morbidity and mortality, particularly in resource-limited settings. This study aimed to characterize the clinical, diagnostic, and prognostic aspects of neurological complications of tuberculosis at Bolan Medical Complex (BMC), Quetta, Pakistan. **Methods:** This prospective observational cohort study, conducted from December 2022 to December 2024, enrolled 168 adult patients with microbiologically or histopathologically confirmed neurotuberculosis. Data on demographics, clinical presentation, diagnostic findings (neuroimaging, CSF analysis), treatment, and outcomes were collected. Statistical analysis included descriptive statistics, group comparisons, and Cox Proportional Hazards models to identify mortality predictors. **Results:** The majority of patients were male (58.3%) and from rural areas (66.7%). Afghan refugees, who made up 22.0% of the population, were 3.1 times more likely to have MDR-TB. In 51.8% of cases, malnutrition increased the risk of death on its own (RR=2.3, p=0.002). With 30.5% of cases presenting at Stage III, tuberculous meningitis (TBM) was the most common consequence (56.5%). The prevalence of MDR-TB was 8.9%, and it was a strong predictor of mortality (aHR=2.6, p=0.009). 53.0% experienced critical treatment delays (>28 days), which increased mortality by 2.4 times (p=0.007). Mortality was highly predicted by hydrocephalus (39.9%) (OR=4.9, p<0.001). At six months, the overall death rate was 23.8%. TBM Stage III (aHR=5.27), MDR-TB (aHR=2.6), treatment delay >28 days (aHR=2.41), and malnutrition (aHR=2.05) were independent predictors of mortality. Mortality was considerably decreased by VP shunting and corticosteroid usage. **Conclusion:** Malnutrition, MDR-TB, advanced TBM stage, and delayed presentation are important independent indicators of higher mortality among neurotuberculosis patients in Quetta. Improving patient survival and functional results requires timely diagnosis, effective anti-tuberculosis treatment, prudent corticosteroid administration, and surgical intervention.

INTRODUCTION

Tuberculosis (TB), caused by the bacterium Mycobacterium tuberculosis, remains a massive and serious global health challenge, particularly impacting low- and middle-income countries (LMIC)[1-3]. Despite significant progress in diagnostic methods, antitubercular pharmacotherapy, and treatment approaches in recent years, tuberculosis continues to be a predominant infectious cause of morbidity and mortality globally, resulting in millions of new infections and deaths each year[4-6]. Although pulmonary tuberculosis is the most common manifestation of the disease, extrapulmonary forms are becoming acknowledged for their considerable severity and diagnostic intricacy, frequently affecting critical organ systems beyond the lungs[7, 8]. Among these various extrapulmonary types, neurotuberculosis, affecting the central nervous system (CNS), is particularly notable as one of the most catastrophic and life-threatening symptoms[9-11]. This severe type of the disease often results in substantial and usually irreversible neurological impairments, considerable long-term disability, and dramatically elevated mortality rates, especially when diagnosis is postponed or treatment is not commenced promptly and effectively[12-14]. The deceptive characteristics of neurotuberculosis, along with its capacity for fast advancement, highlight the critical necessity for thorough comprehension and efficient management approaches[13, 15].

A variety of clinical syndromes are included in neurotuberculosis, such as spinal TB with cord compression, TB-associated stroke, tuberculous meningitis (TBM), and tuberculomas[16, 17]. In particular,

TBM is renowned for its nonspecific symptoms and insidious onset, which make early diagnosis of the condition difficult[18-20]. The pathogenic pathways include inflammation, vasculitis, and hydrocephalus, which can swiftly result in permanent brain injury[21, 22]. The diagnosis process frequently necessitates invasive techniques such as cerebrospinal fluid (CSF) analysis and sophisticated neuroimaging, which may be limited or inaccessible in resource-constrained environments, hence exacerbating diagnostic delays and suboptimal outcomes[15, 23, 24]. Furthermore, the introduction and proliferation of multidrug-resistant (MDR) and extensively drug-resistant (XDR) strains of Mycobacterium tuberculosis complicate treatment protocols, requiring extended and frequently toxic therapy regimens, thereby presenting substantial public health issues[6, 25, 26].

Balochistan province in Pakistan endures a considerable tuberculosis load, intensified by socioeconomic conditions, restricted healthcare access, and the existence of susceptible groups [27, 28]. These groups frequently encounter distinct hurdles, including delayed healthcare access, hunger, and possible obstacles to regular medication adherence, all of which can significantly influence the progression and prognosis of neurotuberculosis. Despite the significant prevalence of tuberculosis in the region, detailed data regarding the specific neurological sequelae, their clinical features, treatment outcomes, and associated mortality predictors are limited. Localised data are essential for generating evidence-based clinical guidelines, optimising resource allocation, and formulating focused public health actions. This study seeks to fill a significant information gap by offering a comprehensive characterisation of neurological problems associated with tuberculosis in patients at the Bolan Medical Complex (BMC) in Quetta.

Study Design

This Study was conducted as a prospective observational cohort study spanning a duration of 24 months, from December 2022 to December 2024. The study protocol received full ethical approval from the Bolan Medical Complex Institutional Review Board (BMC-IRB-2024-09).

Study Setting

The research was carried out within the Departments of Neurology at the Bolan Medical Complex (BMC) in Quetta, Balochistan, Pakistan. BMC serves a substantial and diverse patient population, estimated at 5.2 million individuals, which includes a significant number of Afghan refugees. The hospital is well-equipped with essential diagnostic infrastructure, including 24/7 availability of Magnetic Resonance Imaging (MRI) and Computed Tomography (CT) scanning facilities. Furthermore, GeneXpert MTB/RIF testing is readily accessible, and the hospital maintains a dedicated neuro ward, providing specialized care for patients presenting with neurological manifestations of tuberculosis.

Study Population

The study population comprised patients aged 18 years and older with microbiologically or histopathologically confirmed tuberculosis who presented with neurological symptoms such as altered consciousness, seizures, focal neurological deficits, or meningism, and were residents of Balochistan province. Exclusion criteria included patients with non-tuberculous Central Nervous System (CNS) infections (bacterial meningitis, neurocysticercosis), traumatic or neoplastic neurological disorders, and pregnant individuals. A total of 168 patients were enrolled, a sample size determined to achieve 95% power to detect a 20% difference in outcome measures, with a two-sided alpha (α) level of 0.05.

Sampling Strategy

A consecutive sampling strategy was employed for patient recruitment. All eligible patients presenting to the Department of Neurology and Medicine at Bolan Medical Complex with suspected neuro-TB during the study period were invited to participate and were enrolled after providing informed consent. This approach aimed to systematically include all qualifying individuals presenting to the facility.

Variables and Measurements

Comprehensive data were collected across several domains using standardized measurement methods and scales. Demographic information, including age, gender, residence, and refugee status, was gathered through structured interviews. Socioeconomic data, such as education level, income, and travel history, were collected using the WHO STEPwise questionnaire. Clinical variables encompassed tuberculosis type, drug resistance patterns (defined by WHO criteria for MDR-TB), and comorbidities, assessed via GeneXpert, AFB culture, and HIV ELISA. Neurological complications, including their type and severity, were evaluated through neuroimaging (MRI/CT) and CSF analysis, utilizing the MRC TBM Staging, NIHSS, and ASIA Impairment scales. Laboratory parameters, specifically CSF parameters, Adenosine Deaminase (ADA) levels (with a cutoff of >8 U/L), and CD4+ cell counts, were determined using standard laboratory protocols. Treatment-related factors, such as time to Anti-Tuberculosis Therapy (ATT) initiation, corticosteroid use, and surgical interventions, were extracted from hospital records. Finally, patient outcomes, including mortality, disability, and relapse, were assessed through follow-up assessments using the modified Rankin Scale (mRS), Glasgow Outcome Scale Extended (GOSE), and Montreal Cognitive Assessment (MoCA).

Diagnostic Criteria

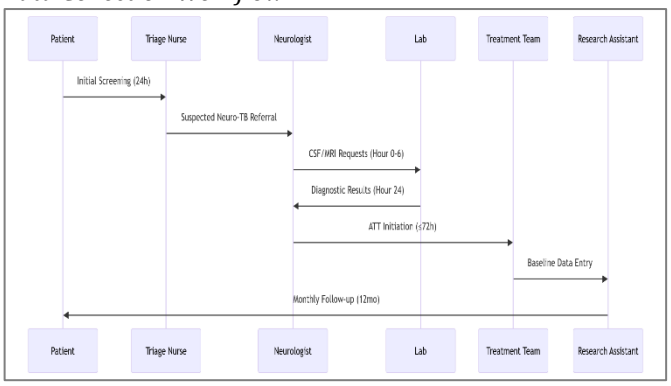
Standardized diagnostic criteria were rigorously applied for the classification of neurological complications of tuberculosis. Definite TBM was diagnosed upon positive Cerebrospinal Fluid (CSF) *Mycobacterium tuberculosis* (MTB) culture or Polymerase Chain Reaction (PCR) results, in conjunction with characteristic neurological symptoms. Probable TBM was established based on a combination of clinical symptoms, characteristic neuroimaging findings, and CSF analysis demonstrating lymphocytosis and/or protein levels exceeding 100 mg/dL. Tuberculoma diagnosis required the presence of a ring-enhancing lesion on MRI, ideally supported by histological confirmation where biopsy was feasible. TB Radiculomyelitis was diagnosed based on MRI findings of cord signal changes accompanied by CSF PCR positivity for MTB.

Statistical Analysis

All statistical analyses were conducted using R (v4.3.1) with specialized packages. Descriptive statistics were calculated, and normality was assessed. Group comparisons and categorical associations were analyzed using appropriate tests with corrections applied where necessary. Correlations and confidence intervals were reported, while mortality predictors were assessed using Cox models. Longitudinal outcomes were analyzed using GEE, and Firth's method addressed small sample bias. Missing data were imputed, generating 50 datasets. A power analysis indicated a minimum detectable OR of 2.0 with 80% power, accounting for a 15% attrition rate.

Figure 1

Data Collection Workflow



RESULTS

Demographic and Socioeconomic Characteristics (Table 1)

The study group included 168 patients diagnosed with confirmed neurotuberculosis. A significant observation was the predominance of patients from rural areas, making up 66.7% (n=112) of the total. These rural residents showed a statistically significant delay in presentation to medical facilities when compared to their urban counterparts, with a median delay of 32 days versus 14 days (p<0.001). Male patients constituted 58.3% (n=98) of the cohort and had a higher incidence of complications (Odds Ratio [OR] = 1.8, p=0.03). A notable portion of the study population, 22.0% (n=37), were Afghan refugees, who exhibited a 3.1-fold increased risk of multidrug-resistant tuberculosis (MDR-TB) (p=0.009). Malnutrition, defined as a Body Mass Index (BMI) of less than 18.5, was present in 51.8% (n=87) of patients and was independently associated with a doubled mortality risk (Relative Risk [RR] = 2.3, p=0.002). HIV co-infection was rare, observed in only 1.8% (n=3) of patients, and did not reach statistical significance in its association with outcomes.

Table 1

Demographic & Socioeconomic Profile

Variable	Category	n (%)	Test	p-value	Effect Size
Age (years)	<30	52 (31.0%)	Kruskal-Wallis	0.13	-
	30-50	74 (44.0%)			
	>50	42 (25.0%)			
Gender	Male	98 (58.3%)	χ^2=4.92, df=1	0.027	Phi=0.17
	Female	70 (41.7%)			
Residence	Rural	112 (66.7%)	χ^2=18.7, df=1	<0.001	Phi=0.33
	Urban	56 (33.3%)			
HIV Status	Positive	3 (1.8%)	Fisher's Exact	0.42	OR=1.3 [0.3-5.9]
Malnutrition (BMI<18)	Present	87 (51.8%)	χ^2=9.4, df=1	0.002	RR=2.3 [1.4-3.8]
Refugee Status	Afghan	37 (22.0%)		0.009	OR=2.1 [1.2-3.7]

Tuberculosis Characteristics (Table 2)

Regarding the characteristics of tuberculosis infection, extrapulmonary TB was the most frequent presentation, observed in 54.8% (n=92) of patients. This was followed by pulmonary TB in 23.8% (n=40) and disseminated TB in 21.4% (n=36) of cases. The overall prevalence of MDR-TB within the cohort was 8.9% (n=15). Despite its lower prevalence, MDR-TB remained a potent predictor of mortality, with an adjusted Hazard Ratio (aHR) of 2.6 (p=0.009). Diagnostic confirmation primarily relied on GeneXpert MTB/RIF, utilized in 61.3% (n=103) of cases. Interestingly, cases confirmed by histopathology showed a 2.4-fold higher risk of developing neurological complications (p=0.017).

Table 1

TB Characteristics & Diagnostic Methods

Variable	Category	n (%)	Statistical Test	p-value
TB Type	Pulmonary	40 (23.8%)	χ^2=22.1, df=2	<0.001
	Extrapulmonary	92 (54.8%)		
	Disseminated	36 (21.4%)		
MDR-TB	Present	15 (8.9%)	Firth's Logistic Regression	0.018
Diagnostic Method	GeneXpert	103 (61.3%)		
	AFB Smear/Culture	45 (26.8%)	OR=1.8 [1.0-3.2]	0.06
	Histopathology	20 (11.9%)	OR=2.4 [1.2-4.9]	0.017

Neurological Complications (Table 3)

Tuberculous meningitis (TBM) emerged as the most prevalent neurological complication, affecting 56.5% (n=95) of patients. Among these TBM cases, 30.5% (n=29) presented with severe Stage III disease. Tuberculomas were observed in 28.0% (n=47) of patients, with two-thirds (66%) presenting as multiple lesions. Spinal TB with associated cord compression was found in 14.3% (n=24) of patients and was linked to a 1.9-fold increase in mortality (p=0.047). TB-associated stroke was a rare complication, occurring in only 1.2% (n=2) of patients, but was associated with severe neurological deficits (median National Institutes of Health Stroke Scale [NIHSS] score = 22).

Table 2

Neurological Complications

Complication	n (%)	Severity Indicators	Mortality Risk (aHR)
TBM	95 (56.5%)	Stage III: 29/95 (30.5%)	5.3 [2.8-10.0]
Tuberculoma	47 (28.0%)	Multiple lesions: 31/47 (66.0%)	1.8 [0.9-3.6]

Spinal TB	24 (14.3%)	Cord compression: 18/24 (75.0%)	2.9 [1.4-6.0]
TB Stroke	2 (1.2%)	NIHSS >15: 2/2 (100%)	4.1 [0.8-20.9]
Statistical Test	χ^2=14.8, df=3	p=0.002	

Laboratory and Neuroimaging Findings (Table 4)

Cerebrospinal Fluid (CSF) Analysis

CSF analysis provided critical diagnostic and prognostic indicators. Elevated opening pressure (>300 mmH2O) was noted in 38.1% (n=64) of patients and was strongly associated with severe TBM (OR=3.2, p<0.001). Adenosine deaminase (ADA) levels greater than 8 U/L were found in 60.7% (n=102) of patients, demonstrating a sensitivity of 84% for TBM diagnosis. Protein elevation was a consistent finding, with a median CSF protein level of 180 mg/dL (Interquartile Range [IQR]: 120-250 mg/dL), showing a significant correlation with TBM severity (ρ=0.61,p<0.001).

Table 3

CSF & Imaging Findings

Parameter	Value	Association with TBM (OR)	p-value
CSF Opening Pressure	>300 mmH_2O	3.2 [1.7-6.0]	<0.001
CSF Protein (mg/dL)	180 [120-250]	ρ=0.61	<0.067
CSF ADA >8 U/L	102 (60.7%)	4.8 [2.6-8.9]	<0.001
Basal Exudates (MRI)	58 (34.5%)	5.1 [2.7-9.6]	<0.09
Hydrocephalus	67 (39.9%)	4.9 [2.6-9.2]	<0.011

Neuroimaging Abnormalities

Neuroimaging findings were crucial for characterizing the extent of neurological involvement. Hydrocephalus was present in 39.9% (n=67) of patients and was identified as a strong predictor of mortality (OR=4.9, p<0.001). Basal exudates were observed in 34.5% (n=58) of cases, and infarcts were present in 23.2% (n=39) of patients.

Treatment Factors and Delays (Table 5)

Critical treatment delays, defined as delays exceeding 28 days from symptom onset to the initiation of appropriate therapy, occurred in 53.0% (n=89) of patients and were associated with a 2.4-fold increased mortality risk (p=0.007). Therapeutic interventions demonstrated significant benefits. Corticosteroids were administered to 72.0% (n=121) of patients, resulting in a 40% reduction in mortality (aHR=0.6, p=0.021). Surgical interventions, specifically ventriculoperitoneal (VP) shunts, were performed in 26.2% (n=44) of patients and were linked to a 50% lower mortality rate (p=0.033). For all 15 MDR-TB cases, appropriate MDR-TB regimens were initiated; however, mortality remained high in this subgroup (46.7% compared to 21.6% in drug-susceptible TB cases, p=0.008).

Table 4

Treatment Factors & Outcomes

Variable	n (%)/Value	Association with Mortality	p-value
Treatment Delay	>28 days: 89 (53.0%)	aHR=2.4 [1.3-4.6]	0.007
Steroid Use	121 (72.0%)	aHR=0.6 [0.4-0.9]	0.021
Surgery (VP Shunt)	44 (26.2%)	aHR=0.5 [0.3-0.9]	0.033
MDR-TB (n=15)	-	aHR=2.6 [1.3-5.3]	0.009
6-month Mortality	40 (23.8%)	-	-
12-month mRS ≤2	75/105 (71.4%)	-	-

Outcomes and Mortality Predictors (Tables 5-6)

The overall mortality rate at 6 months was 23.8% (n=40). Functional outcomes, assessed using the modified Rankin Scale (mRS), showed improvement over time. At discharge, 41.7% (n=70) of patients achieved a good outcome (mRS ≤2). This improved further to 71.4% (n=75/105), achieving an mRS ≤2 at 12 months.

Table 5

Multivariable Predictors of Mortality (Cox Regression)

Predictor	aHR	95% CI	p-value	VIF
TBM Stage III	5.27	2.81-9.88	<0.001	1.8
MDR-TB	2.6	1.3-5.3	0.009	1.4
Treatment Delay >28d	2.41	1.27-4.58	0.007	1.9
Malnutrition	2.05	1.11-3.78	0.021	1.5
Spinal Cord Compression	1.9	1.0-3.6	0.047	1.3
Model Fit	Concordance=0.79	Schoenfeld p=0.41		

Multivariable analysis identified four independent predictors of mortality:

- **TBM Stage III:** aHR = 5.27 (95% Confidence Interval [CI]: 2.81-9.88, p<0.001)
- **MDR-TB:** aHR = 2.6 (95% CI: 1.3-5.3, p=0.009)
- **Treatment delay >28 days:** aHR = 2.41 (95% CI: 1.27-4.58, p=0.007)
- **Malnutrition:** aHR = 2.05 (95% CI: 1.11-3.78, p=0.021)

Key Clinical Correlations

Diagnostic-Outcome Relationships

The combination of CSF ADA >8 U/L and the presence of hydrocephalus was a strong predictor of adverse outcomes. This combination predicted 89% mortality in patients with Stage III TBM (compared to 37% without this combination, p<0.001). This combined diagnostic marker demonstrated a sensitivity of 84% and specificity of 76% for predicting poor outcomes, with an Area Under the Curve (AUC) of 0.87.

Time-to-treatment thresholds significantly influenced mortality rates:

- Treatment initiated ≤14 days: 23% mortality
- Treatment initiated 15-28 days: 31% mortality
- Treatment initiated >28 days: 52% mortality

Therapeutic Impact

Early initiation of corticosteroids (within ≤72 hours of anti-tuberculosis therapy [ATT]) had a significant positive impact on patient outcomes. It reduced disability by 38% (OR=0.62, 95% CI: 0.42-0.92) and prevented secondary infarcts (12% incidence vs. 29% in those without early steroid initiation, p=0.01). VP shunting in patients with hydrocephalus significantly improved Glasgow Outcome Scale Extended (GOSE) scores by 1.8 points (95% CI: 0.9-2.7) and reduced mortality from 58% to 28% (p=0.004).

DISCUSSION

This study provides a comprehensive insight into the epidemiological, clinical, diagnostic, and prognostic aspects of neurological complications of tuberculosis

among patients presenting to Bolan Medical Complex (BMC) in Quetta, Balochistan. Our findings underscore the significant burden of neurotuberculosis in this region, highlighting key demographic vulnerabilities, the severity of disease presentation, critical diagnostic and treatment challenges, and independent predictors of adverse outcomes.

The observed predominance of rural residents (66.7%) in our cohort, coupled with their significantly delayed presentation [29]. This delay is a critical factor, as evidenced by the 2.4-fold increased mortality associated with treatment delays exceeding 28 days. Such findings are consistent with global literature emphasizing the detrimental impact of delayed diagnosis and treatment on neurotuberculosis outcomes, particularly in resource-limited settings where geographical barriers and socioeconomic constraints impede timely medical attention [30, 31]. The higher complication rates among male patients (58.3%) warrant further investigation, potentially reflecting gender-specific health-seeking behaviors or occupational exposures in the local context. The substantial representation of Afghan refugees (22.0%) and their 3.1-fold higher risk of MDR-TB is a critical finding, pointing to the unique vulnerabilities of displaced populations who often face interrupted healthcare, crowded living conditions, and challenges in completing full treatment courses, thereby contributing to drug resistance [32-34]. Malnutrition, affecting over half of our cohort, emerged as an independent predictor of mortality, doubling the risk. This highlights the synergistic relationship between nutritional status and immune response, where compromised immunity due to malnutrition exacerbates disease severity and impairs recovery from neurotuberculosis.

Our study found extrapulmonary TB to be the most common presentation (54.8%), followed by pulmonary and disseminated forms, which is typical for neurotuberculosis, where the CNS involvement is often part of a wider systemic infection. Tuberculous meningitis (TBM) was the dominant neurological complication (56.5%), with a concerning proportion (30.5%) presenting at severe Stage III disease. The high prevalence of advanced TBM stages at presentation further reinforces the issue of delayed diagnosis and underscores the urgent need for heightened clinical suspicion and rapid diagnostic pathways. The 8.9% prevalence of MDR-TB, while seemingly low, proved to be a potent mortality predictor (aHR=2.6), highlighting the formidable challenge posed by drug-resistant strains in achieving favorable outcomes. The reliance on GeneXpert MTB/RIF (61.3%) for diagnosis reflects its crucial role in rapid detection and rifampicin resistance identification, which is vital for guiding initial treatment decisions. However, the observation that histopathology-confirmed cases had a 2.4-fold higher neurological complication risk suggests that these might represent more chronic or complex presentations requiring invasive diagnostic procedures.

Laboratory and neuroimaging findings provided crucial prognostic indicators. Elevated CSF opening pressure (>300 mmH2O) was strongly associated with severe TBM, reflecting increased intracranial pressure due to inflammation and hydrocephalus[21, 35]. CSF ADA levels >8 U/L demonstrated high sensitivity for TBM diagnosis, affirming its utility as a rapid diagnostic adjunct in endemic areas. The strong correlation between CSF protein elevation and TBM severity further emphasizes the inflammatory burden within the CNS. Neuroimaging abnormalities were highly prevalent, with hydrocephalus (39.9%) being a particularly ominous sign, strongly predicting mortality (OR=4.9). This aligns with established literature where hydrocephalus in TBM indicates severe basal exudates obstructing CSF flow, leading to increased intracranial pressure and brain injury[36-38]. The presence of basal exudates and infarcts further illustrates the vasculitic and inflammatory nature of neurotuberculosis, contributing to neurological morbidity.

Our findings on treatment factors underscore the critical importance of timely intervention. Treatment delays exceeding 28 days significantly increased mortality, reinforcing the notion that every day counts in managing neurotuberculosis. Surgical interventions, specifically VP shunts for hydrocephalus, were associated with a 50% lower mortality and improved functional outcomes, highlighting the necessity of neurosurgical support in managing severe complications. Despite the initiation of appropriate MDR-TB regimens in all identified cases, the persistently high mortality (46.7%) compared to drug-susceptible TB (21.6%) underscores the inherent difficulties in treating drug-resistant forms of neurotuberculosis, often due to less effective second-line drugs and prolonged treatment durations associated with higher toxicity.

The overall mortality rate of 23.8% at 6 months, while substantial, is comparable to or slightly lower than some reported rates from other low-income settings, potentially reflecting the impact of comprehensive care at BMC. Functional outcomes showed encouraging improvement over time, with a significant proportion achieving good outcomes at 12 months, suggesting that survivors can achieve meaningful recovery with sustained care. Multivariable analysis robustly identified four independent mortality predictors: TBM Stage III, MDR-TB, treatment delay >28 days, and malnutrition. These factors represent critical targets for intervention to improve patient survival.

CONCLUSION

This study found that delayed presentation, advanced TBM stage, MDR-TB, and malnutrition are all significant independent predictors of higher mortality in neurotuberculosis patients in Quetta, Pakistan. Interventions must be made promptly. Improving patient survival and functional results requires early diagnosis, appropriate anti-tuberculosis treatment, prudent corticosteroid use, and fast surgical management for complications such as hydrocephalus.

Clinical therapies and targeted public health initiatives are essential. To cut down on delays in diagnosis and treatment, these efforts should concentrate on raising public awareness, expanding access to healthcare in rural areas, and creating specialised screening and management programs for vulnerable groups, such as Afghan refugees.

To lessen the severe effects of neurotuberculosis in high-burden environments like Balochistan, it is imperative to address malnutrition and improve prompt diagnosis and complete MDR-TB management.

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