ORIGINAL ARTICLE

Prevalence and factors associated with seizures in tuberculous meningitis

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ABSTRACT

Introduction: Tuberculous meningitis (TBM) is a severe manifestation of extrapulmonary tuberculosis that can lead to debilitating neurological complications. Seizures in TBM pose diagnostic and therapeutic challenges and are associated with adverse outcomes and prolonged hospitalisation. This study aims to determine the prevalence, risk factors, and outcomes associated with seizures in patients with TBM patients.

Materials and Methods: A retrospective observational study was conducted on 96 adult patients diagnosed with TBM at a tertiary hospital in Malaysia. Patients with the diagnosis of tuberculous meningitis were included and classified into the seizures and non-seizures groups. Clinical, laboratory, radiological, and treatment-related variables were analysed. Antiseizure medication use and neurological outcomes were also assessed.

Results: Seizures occurred in 30.2% (n=29) of patients; generalized seizures were the predominant type. Patients with seizures were more likely to present with altered behaviour (48.3% vs 31.3%) and focal neurological deficits (24.1% vs 14.9%). Patients with seizures were more likely to be on antiseizure medications, particularly phenytoin, valproate and levetiracetam (p<0.05). Lower Glasgow Coma Scale scores on admission were more common among seizure patients (17.2%) compared to non seizure group (7.5%). Patients with seizures had higher rates of mortality (27.6% vs. 13.4%) and poor functional outcomes compared to those without seizures.

Conclusion: Seizures are common in TBM and are associated with worse clinical outcomes. Early clinical signs such as altered behaviour and focal deficits may help identify high-risk TBM patients with seizures. Seizures in TBM are associated with worse neurological outcomes. The common antiseizure therapy initiated for treatment include phenytoin, valproate and levetiracetam. Further prospective studies are needed to refine risk stratification and optimize management.

KEYWORDS:
tuberculosis, tuberculous meningitis, seizures, associated factors

INTRODUCTION

Tuberculosis (TB) remains a leading cause of infectious death worldwide, with an estimated 10.8 million people affected by TB and an estimated 1.09 million deaths among HIV-negative people in 2023, according to the World Health Organization (WHO).[1] TBM accounts for approximately 1% of all TB cases[2] but contributes disproportionately to TB-related neurological complications and deaths.[3,4] The burden of TB in Asia countries is high, contributing more than 50% of global cases.[5] In Southeast Asia (SEA), TB continues to be a major public health threat, compounded by challenges in early diagnosis, limited access to healthcare, and overlapping burdens of HIV.[6]

Tuberculous meningitis (TBM) often results in significant neurological impairment and long-term consequences.[7] Seizures results from meningeal inflammation, cerebral infarction, tuberculomas, and hydrocephalus.[8] Prevalence rates of seizures in TBM range from 17% to 93%, with differences attributed to patient demographics and study methodologies.[9] Misra et al. identified seizures in 34% of TBM patients[8], while Song et al. observed an incidence of 20%.[10] The presence of seizures was associated with poorer neurological outcomes.[8,11] TBM patients with seizures were reported to have poorer functional outcomes at 12-months follow-up.[10]

The risk factors associated with seizures have been documented in patients with tuberculous meningitis (TBM). Dharmana et al. identified cerebral vasculitic infarcts as an independent predictor of seizures in TBM.[11] Cortical involvement and epileptiform discharges were significant independent risk factors for recurrent seizures.[10,12] The associated factors for the development of epilepsy in central nervous system TB include young age, early onset of seizures, refractory seizures, tuberculomas, cortical involvement, epileptiform discharges, and residual brain lesions.[13] Abdulaziz et al. observed early-onset seizures in TBM were associated with meningeal irritation and cerebral oedema, while late-onset seizures were linked to cerebral infarction, hydrocephalus, tuberculomas, and paradoxical inflammatory responses.[9]

Given the significant potential for seizures to arise from TBM-related comorbidities and to adversely affect treatment and recovery, a focused investigation into their frequency, risk

This article was accepted: 08 November 2025
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Table I: Demographic of the study participants

Variables	All N=96 (%)	Seizure N=29 (%) N=67 (%)	No Seizure N=67 (%)
Age, years			
18-40	29 (30.2)	11 (37.9)	18 (26.9)
41-60	29 (30.2)	8 (27.6)	21 (31.3)
>60	38 (39.6)	10 (34.5)	28 (41.8)
Sex			
Male	52 (54.0)	12 (41.0)	40 (60.0)
Female	44 (46.0)	17 (59.0)	27 (40.0)
Race			
Malay	54 (56.3)	15 (51.7)	39 (58.2)
Chinese	27 (28.1)	10 (34.5)	17 (25.4)
Others	9 (9.4)	2 (6.9)	7 (10.4)
Indian	6 (6.3)	2 (6.9)	4 (6.0)
Smoking			
Yes	11 (11.5)	4 (13.8)	7 (10.4)
No	85 (88.5)	25 (86.2)	60 (89.6)
Diabetes mellitus			
Yes	22 (22.9)	7 (24.1)	15 (22.4)
No	74 (77.1)	22 (75.9)	52 (77.6)
Cancer			
Yes	7 (7.3)	2 (6.9)	5 (7.5)
No	89 (92.7)	27 (93.1)	62 (92.5)
Chronic kidney disease			
Yes	6 (6.3)	1 (3.4)	5 (7.5)
No	90 (93.8)	28 (96.6)	62 (92.5)
Hematological disorders			
Yes	3 (3.1)	0 (0.0)	3 (4.5)
No	93 (96.9)	29 (100)	64 (95.5)
Human immunodeficiency virus disease			
Yes	6 (6.3)	2 (6.9)	4 (6.0)
No	90 (93.8)	27 (93.1)	63 (94.0)
Autoimmune disorder			
Yes	4 (4.2)	1 (3.4)	3 (4.5)
No	92 (95.8)	28 (96.6)	64 (95.5)
Clinical			
Duration of seizure (days)			
0-5	38 (39.6)	16 (55.2)	22 (32.8)
6-9	32 (33.3)	8 (27.6)	24 (35.8)
>9	26 (27.1)	5 (17.2)	21 (31.3)
Seizure type			
Generalised	20 (20.8)	19 (65.5)	1 (1.5)
Focal	10 (10.4)	10 (34.5)	0 (0.0)
No seizure	66 (68.8)	0 (0.0)	66 (98.5)
Frequency of seizure (months)			
0	76 (79.2)	10 (34.5)	66 (98.5)
1	12 (12.5)	12 (41.4)	0 (0.0)
2	4 (4.2)	3 (10.3)	1 (1.5)
>2	4 (4.2)	4 (13.8)	0 (0.0)
Focal deficit			
Yes	17 (17.7)	7 (24.1)	10 (14.9)
No	79 (82.3)	22 (75.9)	57(85.1)
Cranial nerve palsy			
Yes	6 (6.3)	3 (10.3)	3 (4.5)
No	90 (93.8)	26 (89.7)	64 (95.5)
Fever			
Yes	54 (56.3)	14 (48.3)	40 (59.7)
No	42 (43.8)	15 (51.7)	27 (40.3)
Headache			
Yes	46 (47.9)	15 (51.7)	31 (46.3)
No	50 (52.1)	14 (48.3)	36 (53.7)
Vomiting			
Yes	28 (29.2)	7 (24.1)	21 (31.3)
No	68 (70.8)	22 (75.9)	46 (68.7)
Altered behaviour			
Yes	35 (36.5)	14 (48.3)	21 (31.3)
No	61 (63.5)	15 (51.7)	46 (68.7)
GCS			
13-15	46 (47.9)	12 (41.4)	34 (50.7)
9-12	40 (41.7)	12 (41.4)	28 (41.8)
3-8	10 (10.4)	5 (17.2)	5 (7.5)

Table II: Laboratory Parameters

Variables	All N=96 (%)	Seizure N=29 (%)	No Seizure N=67 (%)
Laboratory			
Hb (g/dL)			
Hb <13	59 (61.5)	19 (65.5)	40 (59.7)
Hb 13-17	36 (37.5)	9 (31.0)	27 (40.3)
Hb >17	1 (1.0)	1 (3.4)	0 (0.0)
Total white cell ($x10^9$)			
TWC 0-3	2 (2.1)	0 (0.0)	2 (3.0)
TWC 4-10	59 (61.5)	18 (62.1)	41 (61.2)
TWC >10	31 (32.3)	11 (37.9)	20 (29.9)
Platelet ($x10^9$)			
Platelet 0-149	8 (8.3)	3 (10.3)	5 (7.5)
Platelet 150-410	73 (76.0)	21 (72.4)	52 (77.6)
Platelet >410	15 (15.6)	5 (17.2)	10 (14.9)
Sodium (mmol/L)			
Sodium 0-135	48 (50.0)	12 (41.4)	36 (53.7)
Sodium 135-145	39 (40.6)	14 (48.3)	25 (37.3)
Sodium >145	9 (9.4)	3 (10.3)	6 (9.0)
Creatinine (µmol/l)			
Creatinine 0-63	42 (43.8)	15 (51.7)	27 (40.3)
Creatinine 64-104	37 (38.5)	7 (24.1)	30 (44.8)
Creatinine >104	17 (17.7)	7 (24.1)	10 (14.9)
Total Protein (g/l)			
Total protein 0-63	32 (33.3)	10 (34.5)	22 (32.8)
Total protein 64-83	54 (56.3)	15 (51.7)	39 (58.2)
Total protein >83	10 (10.4)	4 (13.8)	6 (9.0)
Alkaline phosphatase (ALP) U/L			
ALP 0-39	5 (5.2)	3 (10.3)	2 (3.0)
ALP 40-150	77 (80.2)	20 (69.0)	57 (85.1)
ALP >150	14 (14.6)	6 (20.7)	8 (11.9)
Alanine transaminase (ALT) U/L			
ALT 0-55	77 (80.2)	23 (79.3)	54 (80.6)
ALT >55	19 (19.8)	6 (20.7)	13 (19.4)
CSF culture			
Yes	3 (3.1)	1 (3.4)	2 (3.0)
No	92 (95.8)	28 (96.9)	64 (95.5)
NA	1 (1.0)	0 (0.0)	1 (1.5)
CSF cell count			
High	22 (22.9)	7 (24.1)	15 (22.4)
Low	73 (76.0)	22 (75.9)	51 (76.1)
NA	1 (1.0)	0 (0.0)	1 (1.5)
CSF TB PCR			
positive	17 (17.7)	8 (27.6)	9 (12.4)
negative	79 (82.3)	21 (72.4)	58 (86.6)
CSF geneXpert			
positive	5 (5.2)	3 (10.3)	2 (3.0)
negative	31 (32.3)	13 (44.8)	18 (26.9)
NA	60 (62.5)	13 (44.8)	47 (70.1)
AFB identified from other sources			
Yes	12 (12.5)	5 (17.2)	7 (10.4)
No	84 (87.5)	24 (82.8)	60 (89.6)
EEG findings			
Generalised	19 (20.7)	12 (42.9)	7 (10.9)
Focal	4 (4.3)	3 (10.7)	1 (1.6)
NA	69 (75)	13 (46.4)	56 (87.5)

CSF cerebrospinal fluid, AFB acid fast bacilli, EEG electroencephalogram, TB PCR tuberculous polymerase chain reaction, FEME full examination microscopic examination, NA not available

Table III: Types of medications

	All N=96 (%)	Seizure N=29 (%)	No Seizure N=67 (%)
AntiTB			
Ethambutol			
Yes	95 (99)	29 (100)	66 (98.5)
No	1 (1)	0 (0)	1 (1.5)
Rifampicin			
Yes	94 (97.9)	28 (96.6)	66 (98.5)
No	2 (2.1)	1 (3.4)	1 (1.5)
Isoniazid			
Yes	94 (97.9)	28 (96.6)	66 (98.5)
No	2 (2.1)	1 (3.4)	1 (1.5)
Pyrazinamide			
Yes	94 (97.9)	29 (100)	65 (97)
No	2 (2.1)	0 (0)	2 (3)
Moxifloxacin			
Yes	5 (5.2)	2 (6.9)	3 (4.5)
No	91 (94.8)	27 (93.1)	64 (95.5)
Streptomycin			
Yes	7 (7.3)	2 (6.9)	5 (7.5)
No	89 (92.7)	27 (93.1)	62 (92.5)
Carbamazepine			
Yes	1 (1)	0 (0)	1 (1.5)
No	95 (99)	29 (100)	66 (98.5)
Levetiracetam			
Yes	15 (15.6)	13 (44.8)	2 (3)
No	81 (84.4)	16 (55.2)	65 (97)
Phenytoin			
Yes	15(15.6)	14 (48.3)	1 (1.5)
No	81 (84.4)	15 (51.7)	66 (98.5)
Sodium valproate			
Yes	5 (5.2)	4 (13.8)	1 (1.5)
No	91 (94.8)	25 (86.2)	66 (98.5)
Side effects			
Rash			
Yes	7 (7.3)	1 (3.4)	6 (9)
No	89 (92.7)	28 (96.6)	61 (91)
Hepatitis			
Yes	25 (26)	7 (24.1)	18 (26.9)
No	71 (74)	22 (75.9)	49 (73.1)
Neuropathy			
Yes	1 (1)	0 (0)	1 (1.5)
No	95 (99)	29 (100)	66 (98.5)
Optic neuritis			
Yes	1 (1)	0 (0)	1 (1.5)
No	95 (99)	29 (100)	66 (98.5)
Sequelae			
Deceased	17 (17.7)	8 (27.6)	9 (13.4)
Vegetative state	3 (3.1)	2 (6.9)	1 (1.5)
Severe disability	3 (3.1)	1 (3.4)	2 (3)
Moderate disability	12 (12.5)	1 (3.4)	11 (16.4)
Recovery	61 (63.5)	17 (58.6)	44 (65.7)
Intervention			
Yes	25 (26)	9 (31)	16 (23.9)
No	71 (74)	20 (69)	51 (76.1)

Table IV: Association between sociodemographic, clinical factors, laboratory and anti-tuberculosis medication

Mann-Whitney U test						
Variables	**Seizure**	**Mean Rank**	**U**	**Z**	**p-value**	**Effect size**
Gender						
	Yes	54.64	793.5	-1.646	0.05	0.01
	No	45.84				
Smoking	Yes	47.38	939.0	-0.470	0.32	
	No	48.99				
Focal neurological	Yes	45.41	882.0	-1.080	0.14	
	No	49.84				
Cranial nerve palsy	Yes	46.53	914.5	-1.085	0.14	
	No	49.35				
Fever	Yes	52.33	860.5	-1.031	0.15	
	No	46.84				
Headache	Yes	46.67	918.5	-0.489	0.31	
	No	49.29				
Vomiting	Yes	50.91	901.5	-0.709	0.24	
	No	47.46				
Altered behaviour	Yes	42.83	807.0	-1.574	0.06	
	No	50.96				
Alanine transaminase	Yes	48.93	959.0	-0.145	0.44	
	No	48.31				
CSF culture	Yes	48.66	967.0	-0.119	0.45	
	No	48.43				
CSF cell count	Yes	49.09	954.5	-0.186	0.43	
	No	48.25				
AFB from other sources	Yes	50.78	905.5	-0.919	0.18	
	No	47.51				
AntiTB						
Ethambutol	Yes	48	957.0	-0.658	0.26	
	No	48.72				
Rifampicin	Yes	49.16	952.5	-0.613	0.27	
	No	48.22				
Isoniazid	Yes	49.16	952.5	-0.613	0.27	
	No	48.22				
Pyrazinamide	Yes	47.50	942.5	-0.935	0.17	
	No	48.93				
Moxifloxacin	Yes	47.69	948.0	-0.487	0.31	
	No	48.85				
Streptomycin	Yes	48.69	966.0	-0.097	0.46	
	No	48.42				
Clonazepam	Yes	48.28	971.5	0.000	0.50	
	No	48.50				
Carbamazepine	Yes	49	957.0	-0.658	0.26	
	No	48.28				
Lamotrigine	Yes	48.50	971.5	0.000	0.50	
	No	48.50				
Levetiracetam	Yes	34.48	565.0	-5.157	< 0.01	0.02
	No	54.57				
Phenytoin	Yes	32.83	517.0	-5.766	< 0.01	0.03
	No	55.28				
Topiramate	Yes	48.50	971.5	0.000	0.50	
	No	48.50				
Valproate	Yes	44.38	852.0	-2.478	< 0.01	0.02
	No	50.28				
Side effect						
Rashes	Yes	50.34	918.0	-0.948	0.17	
	No	47.70				
Hepatitis	Yes	49.41	945.0	-0.278	0.39	
	No	48.10				
Neuropathy	Yes	49	957.0	-0.658	0.26	
	No	48.28				
Optic neuritis	Yes	49	957.0	-0.658	0.26	
	No	48.28				
Surgery	Yes	46.10	902.0	-0.730	0.23	
	No	49.54				
Independent t -test						
		t-value	df	p value	CI	
Age		0.826	94	0.411	-5.034 – 12.204	
CSF glucose		-0.364	94	0.717	-0.995 – 0.687	
CSF cerebrospinal fluid						

p significant at <0.05. U: Mann-Whitney test; Z: Z value; t: t statistics; df: degree of freedom

Table V: Relationship between medications and seizures

	Seizure	Sex	Levetiracetam	Phenytoin
1. Seizure				
2. Sex	0.169*			
3. Levetiracetam	-0.529**	-0.295**		
4. Phenytoin	-0.592**	-0.065	0.289**	
5. Valproate	-0.254*	0.027	0.157	0.028

*Correlation is significant at the 0.05 level (2-tailed)
**Correlation is significant at the 0.01 level (2-tailed)

factors and clinical impact is essential. Notably, there is a conspicuous lack of comprehensive data on seizure-related complications among TBM patients within the Malaysian context. Seizures in TBM can complicate therapeutic management, prolong hospitalization, increase healthcare utilisation and ultimately worsen patient prognosis. This research aims to determine the prevalence of seizures in TBM to guide early interventions and improve neurological outcomes in patients.

MATERIALS AND METHODS

This retrospective study was conducted at Hospital Canselor Tuanku Muhriz (HCTM), Universiti Kebangsaan Malaysia from 1st January 2018 to 30th June 2025, and approved by the Ethics and Research Committee (JEP-2023-530). Patients aged 18 years and above were included, excluding those with neurodegenerative disorders and other central nervous system infections. An epileptic seizure is defined conceptually as a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity.[14] A diagnosis of TB meningitis was made following the consensus case definition of definite, probable and possible tuberculous meningitis.[15]

The demographic and clinical data were documented. Radiological findings were categorized based on computed tomogram (CT) or magnetic resonance imaging (MRI) reports into the presence or absence of hydrocephalus, infarcts, basal meningitis, or tuberculomas. The Glasgow outcome scale was used to assess the patients' recovery after brain injury. 16 It can be categorised into 1 -good recovery, 2- moderate disability, 3- severe disability , 4- persistent vegetative state, and 5- death.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics for Windows, Version 30. Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR) for nonnormally distributed data. The Mann-Whitney U test was applied to compare qualitative variables. Spearman's correlation was used to analyze the relationship between individual risk factors and the occurrence of seizures. Statistical significance was established at a p-value <0.05.

RESULTS

We screened 152 patients with a total of 96 TBM patients. 29 patients (30.2%) experienced seizures during illness. Most patients were aged over 60 years (39.6%), and 54.2% were male. Comorbidities were evenly distributed between groups, with no statistically significant associations observed ($p > 0.05$) (Table I).

Patients with seizures were more likely to present with altered behaviour (48.3% vs. 31.3%, p=0.06) and focal neurological deficits (24.1% vs. 14.9%, p=0.14), although not significant Generalized seizures were the predominant type in 65.5%.
Lower GCS scores on admission were more common among seizure patients, with 17.2% having a GCS of 3–8 compared to 7.5% in the non-seizure group. (Table I).
No significant differences were found between seizure and non-seizure patients in laboratory, CSF parameters and radiological abnormalities (Table II). Levetiracetam (44.8%) and phenytoin (48.3%) were the most prescribed antiseizure drugs. (Table III)

Mortality was higher in the seizure group compared to the non-seizure group. Additionally, the proportion of patients with severe disability or vegetative state was also higher in the seizure group (10.3% vs. 4.5%). Full recovery was achieved in 58.6% of seizure patients compared to non-seizure patients. Adverse drug reactions did not significantly differ between groups (Table III).

Statistical Associations

Univariate analysis identified several variables associated with seizures (Table IV). Gender showed a significant association with seizures (p=0.05). The patients with seizures in TBM are more likely to be on antiseizure medications such as levetiracetam (p<0.001), phenytoin (p<0.001), and valproate (p=0.007).

Missing Data

Missing values for CSF cell count were imputed using mode values as data were not normally distributed, provided it does not exceed 10%. Missing categorical data for CSF culture and EEG were imputed using the most frequent category to minimise bias and preserve data structure.

Correlation Between Seizure and Medications

Spearman's correlation analysis revealed moderate negative correlation between seizure history and the use of levetiracetam (r=–0.529, p<0.001), phenytoin (r=–0.592, p<0.001) as shown in Table V. Sex demonstrated a very weak positive correlation (r=0.169, p=0.05).

DISCUSSION

Tuberculosis is a challenging problem in a developing country such as Malaysia, where the estimated incidence rate was 234 per 100, 000 population.[17] This study investigated the prevalence, risk factors, and outcomes associated with seizures in patients with TBM in a local tertiary hospital. Seizures occurred in approximately 31.2% , which aligns with seizure rates globally, ranging from 17% to 93% depending on study setting and population.[9] Our findings are consistent with those of Misra et al., with an incidence of 34% among TBM.[8]

Seizure occurrence in TBM patients was associated with altered behaviour, focal neurological deficits, and lower GCS scores on admission. These findings support prior studies indicating that seizures are often markers of more severe central nervous system involvement.[10,11] Seizures following central nervous system infection are related to brain inflammation and subsequent neuronal injury and reactivation of glial cells.[18] Acute symptomatic seizures that occur in the first 2 weeks may be associated with meningeal irritation and cerebral edema. Late seizures are usually due to infarct, hydrocephalus, tuberculoma and paradoxical response.[19]

TBM demonstrate CSF lymphocytosis, raised protein and reduced glucose ratio. This current study found positivity for CSF PCR and CSF GeneXpert was 17.7% and 5.4% respectively. A previous study reported 9.8% with positive CSF tuberculous PCR 20. Studies reported that CSF PCR had a high specificity of 87%-98% and sensitivity between 56% -75%.[21, 22] Advanced diagnostics include urine lipoarabinomannan (LAM), which is a phosphorylated lipopolysaccharide in the Mycobacterial cell wall. Alere TB- LAM in people living with HIV have a sensitivity of 33% (95% CI 9.9- 65.1%) and a specificity of 96% (95% CI: 85.5-99.5%).[23] Using a cut point of > 5.5 mmol/L, CSF lactate was able to diagnose definite/probable TBM with a sensitivity of 67.7% and specificity of 80.3%.[24] The diagnosis of TBM is largely clinical and aided by various investigations. Despite advances in the technological methods in TBM diagnosis, clinical vigilance is of utmost importance in suspected patients presenting with altered behaviour or focal deficits.

A recent study on risk factors for seizures in TB meningitis reported that vasculitic infarcts were strongly associated with seizure recurrence.[11] Seizures were also significantly associated with advanced disease, cortical involvement and epileptiform pattern. In addition, focal to bilateral seizures, status epilepticus and rifampicin resistance were significantly associated with poor outcome at 6 months.[25] Additional factors contributing to increased risk of epilepsy following TB meningitis include a younger age, recurrent seizures and status epilepticus, tuberculoma, infarction, hippocampal sclerosis and persistent epileptic activity in EEG.[19]

Our correlation analysis demonstrated more frequent use of phenytoin and levetiracetam, particularly in patients with more severe or recurrent seizures. These parenteral antiseizure medications are available in the emergency department setting, where patients are initially treated in the acute setting.

Phenytoin and levetiracetam were the most prescribed medications, which may reflect clinician familiarity, availability, and local treatment protocols. Valproate, though less commonly used, was also an important therapeutic medication. These patterns are aligned with the findings of Song et al. and Ramos et al., who noted a higher likelihood of antiseizure treatment in patients with cortical involvement, refractory seizures, or epileptiform EEG discharges.[10,13]

Seizures were associated with worse neurological outcomes, including higher rates of death, disability, and vegetative state. Our centre reported mortality rates of 18% in TBM patients in a previous study. Dharmana et al., did not find seizures to be an independent predictor of mortality.[11] Our results suggest that seizures are at least a surrogate marker of more severe disease and are linked to poorer functional recovery. This finding supports the need for early identification and aggressive management of seizures in TBM to improve long-term outcomes.

These findings highlight several important clinical implications. First, early recognition of seizure risk based on clinical signs, is critical for timely intervention. Second, the absence of significant associations with routine cerebrospinal fluid or radiological markers suggests that these investigations alone are insufficient for predicting seizure risk, reinforcing the importance of clinical judgment in patient assessment. Finally, the observed pattern of antiseizure medication use in seizure management must be individualised rather than prophylactic. While this supports rational prescribing practices, further research should be carried out to determine if high-risk patients could benefit from earlier initiation of seizure prophylaxis.

This study has several limitations. First, the retrospective design may limit data completeness and introduce selection bias. Second, EEG availability was limited, which may have led to underdiagnosis of subclinical seizures. Third, the sample size may not have been sufficient to detect weaker associations. Finally, the exclusion of GCS from multivariate analysis, while methodologically justified limits the ability to formally model consciousness level as a predictive factor.

Future studies should incorporate prospective designs, larger sample sizes, and standardized EEG monitoring to better elucidate seizure subtypes, temporal patterns, and long-term outcomes. There is also a need to evaluate the role of seizure prophylaxis in high-risk TBM patients and the comparative effectiveness of different antiseizure medications in this population.

CONCLUSION

Seizures are a clinically significant complication of tuberculous meningitis, affecting nearly one-third of patients in this cohort. Clinical features such as altered mental behaviour and focal neurological deficits were more prominent among seizure patients . Seizures were associated with poorer neurological outcomes, including higher rates of death and disability. These findings underscore the importance of early clinical recognition and individualized

management strategies to mitigate seizure-related morbidity in TBM. Future prospective studies are warranted to explore the role of prophylactic anticonvulsants in high-risk patients and to further refine risk stratification tools.

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