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ASSOCIATION BETWEEN CLINICAL SEVERITY, NEUROIMAGING FINDINGS, AND NEUROLOGICAL RECOVERY IN PATIENTS WITH TUBERCULOUS MENINGITIS

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ABSTRACT

Objective: The aim was to check the correlation between clinical severity, the neuroimaging appearance, and neurological improvement, and to determine the important prognostic factors affecting the outcome in patients with tuberculous meningitis.
Methods: An observational study was carried out at Lady Reading Hospital Peshawar from 23 November, 2024 to 23 April, 2025. Through consecutive sampling 245 patients were included who have been diagnosed with tuberculous meningitis. Neurological examination and Glasgow Coma Scale (GCS) were used to assess clinical severity. Computed tomography (CT) and magnetic resonance imaging (MRI) were used for neuroimaging. A complete recovery or a poor outcome were classified as neurological recovery. The data were analyzed with a statistical software and the chi square test and logistic regression were used to look for associations and predictors.
Results: Out of 245 patients the majority presented with moderate to severe clinical disease. Basal meningeal enhancement hydrocephalus and cerebral infarction were the most common imaging findings. There was a significant relationship between the clinical severity and the extensive radiological abnormalities, $p < 0.001$. In most of patients with severe disease, there was poor neurological recovery. Hydrocephalus and cerebral infarction were the only independent predictors of poor outcome on regression analysis.
Conclusions: The severity of clinical symptoms and the neuroimaging signs were closely associated with neurological recovery in TB meningitis. Prognostic assessment of both parameters at the beginning was helpful in determining management decisions and was more accurate.

Keywords: Tuberculous meningitis, Clinical severity, Neuroimaging, Neurological recovery, Hydrocephalus, Cerebral infarction.

INTRODUCTION

Infection from Mycobacterium tuberculosis is most severe when it results in “tuberculous meningitis”. Despite the incredible progress made in diagnostic imaging, antimicrobial therapy and critical care management, the disease remains associated with high mortality and permanent neurological disability. The central nervous system is involved as a result of the spread to it from a primary focus by hematogenous route. If a subependymal or subpial tubercle ruptures into the subarachnoid space, a strong inflammatory reaction occurs which leads to the formation of thick basal exudates, vasulitis and progressive neuronal injury, causing hydrocephalus. Delay in diagnosis is common because early symptoms are not specific and may be confused with a variety of infectious and inflammatory diseases. As a result, neurological deterioration is frequently confirmed prior to beginning treatment.[1]

Despite the remarkable progress made in controlling TB meningitis in recent years, a significant burden of TB meningitis persists in low and middle income countries where TB is an endemic disease. The impact of poor outcomes is due to high population density, limited healthcare resources, delayed referral systems, and limited access to advanced neuroimaging. Pakistan is still one of the countries with high prevalence of TB. Neurologists,

neurosurgeons, radiologists and physicians in the fields of emergency and critical care are thus faced with significant challenges. In the early stages of the disease there is often a lack of specificity in the diagnosis, such as constitutional symptoms (headache, fever, vomiting, altered mental status), as it can be shared by many neurological diseases. Progressive neurological deterioration is thus seen in many patients prior to a correct diagnosis being reached.[2]

Several pathological changes follow the inflammation of the meninges, which affect the course of the disease. Exudative material is deposited around the basal cisterns and the cranial nerves, becoming thickened. Obstruction of the pathways that the cerebrospinal fluid flows through is created and either communicating or obstructive hydrocephalus may follow. Thrombosis of the deep perforating vessels and major cerebral arteries causing infarction are observed in cerebral vasculitis. Tuberculomas are another complication which can occur throughout the course of the disease due to ongoing inflammatory activity. These pathological changes are associated with typical neuroimaging abnormalities which offer important clues to the severity of the disease and prognosis. It is therefore considered an important part of a thorough evaluation of the patient.

The clinical severity at presentation has been traditionally assessed using a set of standardised neurological severity scales. Clinical signs that are strongly associated with severity of the disease include a focal neurological deficit, involvement of the cranial nerves, seizures and evidence of raised ICP. High clinical grades have been correlated with higher death rate, longer hospital stays, and ongoing neurological deficits throughout. But, there has been some clinical variability within the patients that have apparently similar clinical grades. Others may show significant neurological improvement, and others suffer from permanent neurological damage despite comparable therapeutic interventions. This variation implies that further prognostic factors need to be found in order to better stratify patients and tailor patient management.[4]

Magnetic resonance imaging and computed tomography have revolutionised the way patients with suspected tuberculous meningitis are evaluated. Basal meningeal enhancement, hydrocephalus cerebral infarction, tuberculomas, cerebral edema and cranial nerve enhancement can be shown with high accuracy. MRI is more sensitive for detecting early inflammatory changes and small ischemic lesions in general. Computed tomography is widely available and is useful in emergency situations especially in resource-limited institutions and allows rapid evaluation. Radiological assessment is thus a complement to clinical assessment to show structural abnormalities that are not visible during physical examination alone.[5]

Several factors interact after treatment and affect neurological recovery. Appropriate early treatment with anti-tuberculous drugs and appropriate early corticosteroid treatment, and early neurosurgical intervention for hydrocephalus have been shown to improve outcome. However, it is important to note that sometimes a complete neurological recovery is not possible as by the time treatment begins, irreversible neuronal damage already exists. Many survivors suffer from functional disability, cognitive impairment, motor weakness, visual deficits, loss of hearing, and epilepsy. Thus, a significant number of survivors of acute illness require long term rehabilitation.[6]

A number of studies have focused on the various aspects of tuberculous meningitis such as diagnosis, treatment, and radiology. There have also been associations of individual neuroimaging abnormalities with clinical outcomes. Findings have been reported varying, however, owing to the difference in study population, disease stages, imaging, outcome measures, and healthcare settings. Comparing the results of the studies is, however, problematical. Moreover, evidence from high-income countries is not always applicable to populations with varying levels of TB burden, health system availability and characteristics.

There remains limited evidence in the literature from Pakistan relating the synergistic effect of the clinical severity of tuberculous meningitis with neuroimaging findings and neurological outcome. The studies available in the literature have concentrated mainly on the diagnostic accuracy or treatment outcomes or single radiological observation. The assessment of neuroimaging and clinical grading of the condition and subsequent recovery of neurological function has not been fully investigated. Mechanisms of these imaging abnormalities within the local population are therefore not completely understood and the prognostic significance of these findings not clearly established. This deficit hampers evidence based clinical decision making and will decrease the chance for identification of patients with a higher risk for poor neurological outcomes at an early stage.

Improved knowledge of the link between neurological presentation, structural brain abnormalities and subsequent recovery could help to improve patient management. Reliable prognostic markers might help identify high-risk patients earlier, so as to maximize the use of intensive care resources, time neurosurgical interventions, and counsel patients and families about outcomes. Clinical and radiological data can also be integrated to facilitate the development of standardised management protocols that can be implemented in healthcare institutions in Pakistan where the burden of disease is high and resources limited.

Thus, the simultaneous assessment of clinical severity and neuroimaging outcomes along with neurological outcome in patients with tuberculous meningitis in Pakistani population remains unknown. The literature has not yet completely determined what radiological abnormalities are markers of increased clinical severity, and what imaging features are

predictive of neurological recovery post-treatment. More data from local tertiary care centers is needed to further validate the prognostic evaluation and enhance evidence based clinical practice.
The aim of the present study was to evaluate the correlation between clinical severity and the neuroimaging parameters with neurological recovery in patients with tuberculous meningitis treated at a tertiary care hospital. The study aimed to determine the relationships of baseline clinical severity and typical neuroimaging features with neurologic outcomes after standard management and to obtain local data that could facilitate the prognosis and care of these patients.

MATERIALS AND METHODS

Study Design

An association between clinical severity, neuro imaging findings and neurological outcome was assessed in patients with tuberculous meningitis in a hospital based observational analytical study. The study was conducted for a tenure of 11 months starting from 23rd January 2025 to 23rd December 2025. Data was collected in a prospective manner from eligible patients that met the a priori selection criteria. Comprehensive and consistent methodology was used throughout the study to allow for uniformity of patient recruitment clinical assessment neuroimaging evaluation and outcome measurement.

Study Setting

This study was carried out in Department of Neurosurgery along with Department of Neurology & Department of Radiology, Lady Reading Hospital Peshawar. Lady Reading Hospital is a tertiary care teaching hospital which receives referrals from Khyber Pakhtunkhwa and adjoining areas. It has a large number of patients with infectious diseases involving the central nervous system and offers state-of-the-art neuro imaging facilities (computed tomography and magnetic resonance imaging). All the patients admitted to the ED outpatient clinics and in cases of inpatient services they were screened for eligibility during the study period.

Study Population

The study population comprised of those patients who presented with clinical signs and symptoms of tuberculous meningitis, CSF analysis, neuro-imaging results and supportive laboratory investigations, according to the institutional diagnostic criteria. To reduce selection bias, a consecutive sampling was used. Enrollment of all eligible patients seen during the study period was considered, until the required number of patients was reached.

Sample Size

There were 300 patients sampled in total. The sample size was deemed sufficient to allow for an evaluation of the association between clinical severity neuroimaging findings and neurological recovery with adequate statistical power. Eligible patients were recruited consecutively until the number of patients (n = 300) was reached.

Inclusion Criteria

In the study, patients, regardless of gender, over the age of 18 years with a diagnosis of tuberculous meningitis were included. The diagnosis was confirmed based on compatible clinical features, CSF analysis suggestive neuro imaging findings and microbiological/molecular evidence if available. Patients were included who had a computed tomography magnetic resonance imaging or both administered during admission. Patients who had completed neurological follow up during hospitalisation and received conventional treatment for tuberculosis were included for final analysis.

Exclusion Criteria

Patients less than 18 years old were not included in the study. Bacterial viral or fungal meningitis patients were excluded. Those who had previously suffered from neurological diseases that might have a similar effect on neurological recovery (stroke, neurodegenerative diseases, brain tumour, epilepsy or traumatic brain injury) were not included. Patients who had poor clinical records, inadequate neuroimaging studies or who refused to provide informed consent were also excluded. Those who transferred to another hospital prior to treatment completion or were lost to follow up were not included in the final analysis.

Data Collection Procedure

After obtaining written informed consent eligible patients were enrolled consecutively. Structured data collection proforma was used to collect the demographic data such as age, gender, residence etc and relevant medical information. The clinical history was detailed, with particular attention paid to the duration of symptoms, fever, headache, vomiting, and altered level of consciousness, seizures, cranial nerve deficits, and findings of meningeal irritation, or focal neurological deficits. Experienced clinicians performed general physical examination and a comprehensive neurological examination, following departmental protocols.
The clinical presentation was evaluated according to the staging system designed by the British Medical Research Council (BMRC) for the diagnosis of tuberculous meningitis. Patients were classified as stage I or stage II or stage III based on the level of consciousness, presence of focal neurological deficits and neurological status at admission. Basic

laboratory investigations such as complete blood count, erythrocyte sedimentation rate, serum biochemical profile, cerebrospinal fluid examination and microbiological investigations were documented, if available.

Neuro-imaging was performed with computed tomography or magnetic resonance imaging or both in all enrolled patients based on clinical and/or available indication. The consultant radiologists (not involved in the neuroimaging studies) were blinded to the neurological recovery status. Standardized reporting criteria were used to document radiological findings such as basal meningeal enhancement, hydrocephalus, cerebral infarction, tuberculoma, cerebral edema, ventriculitis and other intracranial abnormalities.

Standard anti-tuberculous treatment was followed in all the patients as per institutional treatment plan. Adjunctive corticosteroid therapy was used as needed. In patients with clinically significant hydrocephalus, neurosurgical procedures such as ventriculoperitoneal shunting or external ventricular drainage were carried out in keeping with neurosurgical advice. All patients received multidisciplinary treatment by neurologists, neurosurgeons, infectious disease specialists and radiologists throughout their stay.

The Glasgow Outcome Scale (GOS) was used to evaluate neurological recovery at discharge. Outcomes were classified as favourable neurological recovery (good recovery and moderate disability) or unfavourable neurological recovery (severe disability, vegetative state or death). To minimise assessment bias, outcome assessment was carried out by clinicians not involved in interpretation of neuro-imaging findings.

Study Variables

Clinical severity at admission and the neuro-imaging results were the major independent variables. Severity of the clinical picture was classified based on the British Medical Research Council staging. Basal meningeal enhancement, hydrocephalus, cerebral infarction, tuberculoma, cerebral edema, ventriculitis and other associated intracranial abnormalities were the neuroimaging variables. The main outcome measure was neurological function as measured by the Glasgow Outcome Scale on discharge. Other details such as age, gender, duration of symptoms, laboratory results, and treatment information were also collected for descriptive analysis.

Ethical Considerations

Institutional Research and Ethics Committee of Lady Reading Hospital Peshawar, gave the ethical clearance for the study prior to the data collection. The study was done on the basis of ethical principles of the Declaration of Helsinki. All participants or their legal representatives provided written informed consent in patients who were not conscious or could not consent. Patient information was kept confidential throughout the study, with unique identification numbers that did not include personal identifiers. To ensure data anonymity, study data was only available to the principal investigator and research team. Patients were informed that their participation in the study was completely voluntary, and that there would be no consequences for not participating in the study for the quality of medical care received.

Statistical Analysis

The data were entered and analyzed with the use of Statistical Package for the Social Sciences (SPSS) version 26.0. Continuous variables such as age and duration of symptoms were summarized as mean and standard deviation or median and interquartile range, depending on the distribution of the data. Frequencies and percentages were used to present categorical variables such as gender, clinical severity stages, neuroimaging findings, treatment modalities and neurological outcomes. The Chi square test or Fisher exact test was used for assessing associations between categorical variables, as appropriate. For comparison of continuous variables based on data normality, independent sample t test or Mann Whitney U test was used. To determine independent predictor of poor neurologic outcome after adjusting for potential confounding factors, multivariable logistic regression analysis was carried out. Odds ratios with 95 percent confidence limits were adjusted. All analyses were statistically significant at a value < 0.05.

RESULTS

Baseline Demographic and Clinical Characteristics

The study included 245 patients diagnosed with tuberculous meningitis at Lady Reading Hospital Peshawar during the study period. The mean age of patients was 34.6 ± 12.8 years. Male patients were slightly more predominant compared to females. Most patients presented with advanced clinical severity at the time of admission. Fever headache and altered level of consciousness were the most frequent presenting symptoms. The majority of patients were classified as having moderate to severe disease according to clinical grading at presentation. Delayed hospital presentation was commonly observed among the cohort.

Table 1: Baseline Demographic and Clinical Characteristics of Patients

Variable	Value
Total patients	245

Mean age (years)	34.6 ± 12.8
Male	138 (56.3%)
Female	107 (43.7%)
Mean duration of symptoms before admission	9.2 ± 4.6 days
Most common symptom	Headache (82%)
Severe clinical grade at presentation	101 (41.2%)

Clinical Severity Distribution and Neurological Status

Clinical severity assessment revealed variation in neurological impairment at presentation. A significant proportion of patients were found in moderate and severe categories indicating delayed diagnosis and progression of disease before hospital admission. Patients with severe clinical grade demonstrated lower Glasgow Coma Scale scores and higher incidence of focal neurological deficits including cranial nerve palsies and hemiparesis. Seizure activity was also more frequent among severe cases compared to mild disease category.

Table 2: Clinical Severity Distribution and Neurological Findings

Severity Grade	Number of Patients	Mean GCS	Cranial Nerve Palsy	Seizures
Mild	68	14.2 ± 0.8	12 (17.6%)	10 (14.7%)
Moderate	76	11.6 ± 1.4	34 (44.7%)	28 (36.8%)
Severe	101	7.9 ± 2.1	71 (70.3%)	52 (51.4%)

Neuroimaging Findings in Patients with Tuberculous Meningitis

Neuroimaging evaluation revealed multiple pathological findings consistent with tuberculous meningitis. Basal meningeal enhancement was the most frequent radiological abnormality followed by hydrocephalus and cerebral infarction. Magnetic resonance imaging demonstrated higher detection rates of small infarcts and meningeal enhancement compared to computed tomography. Severe clinical cases showed higher frequency of hydrocephalus and cerebral infarction compared to mild and moderate cases.

Table 3: Neuroimaging Findings in Study Population

Imaging Finding	Number of Patients	Percentage
Basal meningeal enhancement	176	71.8%
Hydrocephalus	112	45.7%
Cerebral infarction	98	40.0%
Tuberculoma formation	64	26.1%
Cerebral edema	53	21.6%

Association between Clinical Severity and Neuroimaging Findings

A significant association was observed between clinical severity and neuroimaging abnormalities. Patients with severe clinical grade demonstrated markedly higher incidence of hydrocephalus and cerebral infarction compared to mild cases. Statistical analysis showed strong correlation between worsening clinical status and presence of multiple radiological lesions. The association was found to be statistically significant with p value less than 0.001 indicating strong relationship between disease severity and imaging burden.

Table 4: Association between Clinical Severity and Neuroimaging Findings

Imaging Finding	Mild (n=68)	Moderate (n=76)	Severe (n=101)	p-value
Hydrocephalus	18 (26.4%)	41 (53.9%)	53 (52.4%)	<0.001
Cerebral infarction	12 (17.6%)	34 (44.7%)	52 (51.4%)	<0.001
Basal enhancement	38 (55.8%)	59 (77.6%)	79 (78.2%)	0.002
Tuberculoma	10 (14.7%)	21 (27.6%)	33 (32.6%)	0.01

Neurological Recovery Outcomes and Predictors of Outcome

Neurological recovery assessment demonstrated variable outcomes among patients. Complete recovery was observed in patients with mild disease and limited radiological involvement. Partial recovery was more common in moderate cases while poor outcomes including severe disability or death were predominantly observed in severe clinical

category. Multivariate statistical analysis identified clinical severity hydrocephalus and cerebral infarction as independent predictors of poor neurological recovery.

Table 5: Neurological Recovery Outcomes and Predictive Analysis

Outcome Category	Mild (n=68)	Moderate (n=76)	Severe (n=101)
Complete recovery	42 (61.8%)	21 (27.6%)	8 (7.9%)
Partial recovery	20 (29.4%)	34 (44.7%)	28 (27.7%)
Poor recovery/death	6 (8.8%)	21 (27.6%)	65 (64.4%)

Multivariate Logistic Regression Analysis of Poor Neurological Recovery

Multivariate logistic regression analysis was performed to identify independent predictors of poor neurological recovery. Severe clinical grade showed strongest association with poor outcome followed by presence of hydrocephalus and cerebral infarction. Basal meningeal enhancement showed weaker but still significant association. Age and gender were not found to be statistically significant predictors in this model.

Table 6: Multivariate Logistic Regression for Poor Neurological Recovery

Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
Severe clinical grade	4.82	2.91–7.64	<0.001
Hydrocephalus	3.76	2.21–6.12	<0.001
Cerebral infarction	3.41	1.98–5.89	<0.001
Tuberculoma	1.68	0.94–2.91	0.07
Age	1.02	0.99–1.05	0.11
Gender	1.12	0.78–1.63	0.42

DISCUSSION

Tuberculous meningitis is one of the most serious forms of extrapulmonary tuberculosis and has been reported to have a high mortality rate and a high risk of long term neurological disability. The current study showed that there was a high correlation between the clinical severity, neuroimaging findings, and neurological outcome in patients who were treated at a tertiary care center. The observed demographic pattern was of predominance of young adults and male patients, which was consistent with the epidemiological trend reported in similar settings where productive age groups are often affected, with social and environmental exposure factors. The time lag in most cases highlighted the fact that there were ongoing issues in the early identification of the disease and timely referrals, as has been seen in other clinical settings with health access problems leading to late presentations.[11]

In the present cohort, when the clinical severity of the disease was analyzed, a significant number of patients had moderate and severe clinical presentation. This pattern represents a late diagnosis and subsequent progressive intracranial inflammatory damage before being admitted to hospital. These findings are consistent with those of other clinical populations, in which impaired awareness at presentation has been correlated with generalized and vague early symptoms and advanced neurological impairment. Ongoing meningeal inflammation, vascular compromise and elevated ICP have been linked to the progression from mild neurological symptoms to severe consciousness impairment. These pathological processes have been shown to progress silently at early stages leading to delayed diagnosis and poor outcomes.[12]

The present study showed that Glasgow Coma Scale score, cranial nerve involvement and seizure frequency were significantly associated with severe clinical grade. These findings fit with those previously described where neurological deterioration has been directly correlated with the severity of disease at presentation. In other similar analyses increased neurological deficits have been associated with extensive inflammatory exudates at the basal cisterns leading to vascular occlusion and ischemic injury. There is a correlation between clinical grading and neurological damage, which is important for early neurological assessment for prognostication and treatment planning.[13]

Basal meningeal enhancement hydrocephalus and cerebral infarction were most prevalent abnormalities detected by neuroimaging in this study. These findings are similar to those previously reported in which meningeal inflammation and obstruction of CSF flow were deemed to be the most important radiological signs. Almost half of the patients exhibited a picture of impaired CSF absorption (hydrocephalus) reflecting basal exudates. Comparable frequencies have been reported in similar studies where hydrocephalus was seen as being a significant factor in neurological deterioration and surgery. Infarction occurs in a large number of patients, suggesting that the vessels are involved by inflammatory arteritis of the perforating vessels.[14]

In the present study, clinical severity was correlated with radiological burden which was significant. There were more hydrocephalus infarction and multiple intracranial lesions in patients with severe clinical grade. Previous studies in other cohorts have shown that increasing radiological abnormalities were directly correlated with worsening neurological status in the same. Pathologically, this relationship is due to progressive inflammatory exudation which leads to vascular compression ischemia and obstructive hydrocephalus. All these changes together result in worsening of clinical symptoms and poor neurological outcome. The use of clinical and radiological parameters for the better stratification of the disease has been thus emphasised as fundamental for accurate disease stratification.[15]

In this study, neurological recovery outcomes found that patients with mild disease had a higher rate of full recovery, whereas those with severe disease were more likely to have poor outcomes. These findings have been similarly observed in other clinical groups in which early stage disease has been linked to good functional recovery. The degree of irreversible neuronal damage seems to be a key issue in the outcome with early treatment beginning work preventing further progression of vasculitic and necrotic changes. As the disease progresses, the damage to the brain structures will be severe, and even with proper treatment the brain cannot recover.[16]

Severe clinical grade hydrocephalus and cerebral infarction were recognized as independent predictors of poor neurological outcome for the multivariate analysis of the current study. This is consistent with the previous observations in which these factors were known to be strong determinants of prognosis. Raised ICP and progressive neuronal compression is caused by hydrocephalus that can lead to permanent neuronal damage if not properly treated early. Cerebral infarction is due to vascular occlusion and an ischemic event resulting in permanent neurological defi cits. The ability to identify these is a reminder of the importance of early radiological assessment and treatment in high-risk patients.[17]

In the recent literature the importance of neuroimaging in predicting outcome is highlighted. Magnetic resonance imaging was more sensitive at detecting early infarcts and subtle meningeal enhancement than computed tomography in the present study. This observation is consistent with the results of other studies that used more sophisticated imaging techniques that gave better diagnostic accuracy. The early detection of radiological abnormalities has been correlated with the better treatment planning and better prognostic stratification. Despite the limited availability of advanced imaging in resource constrained environments, there remains a challenge to optimally assessing the disease and intervening in time.

The high percentage of patients with tuberculoma formation in this study was another important finding. The incidence of tuberculoma was lower than that of other imaging features, but was correlated with more complicated clinical courses. Other groups have experienced similar findings, with tuberculomas associated with longer duration of disease and unpredictable outcomes of treatment. These lesions are granulomatous, indicating that they are a result of chronic inflammation, and can take a long time to be treated. Their presence also makes radiological interpretation difficult, and they can be confused with other space occupying lesions of the brain.[19]

The pattern of neurological recovery noted in this study underscores the need for early intervention. Patients who are treated at mild stages showed significantly better functional outcomes than patients who are treated in advanced stages. Similar results have been observed in other clinical settings where prompt TB treatment resulted in fewer neurological effects. The onset of treatment seems to be an important factor in avoiding irreparable damage to neurons. Inflammatory cascades cannot be fully reversed with appropriate treatment, and there is evidence that they progress with delay in treatment.[20]

The links between hydrocephalus and poor outcome seen in this study are significant in clinical decision making. The same was reported in other studies where there was an association between hydrocephalus and death and disability rates. The pathophysiological process is caused by blockage of the CSF tracts by inflammatory exudates, resulting in the enlargement of the ventricles and increased ICP. If the damage is severe, surgical correction of the condition may be necessary, but that depends on the length of the condition and its severity. Therefore, early diagnosis and treatment of hydrocephalus may lead to better neurological outcome.[21]

Another significant predictor of poor outcome in this study was cerebral infarction. This result is consistent with other studies that reported that vascular complications were important factors in neurological disability. Thinning and narrowing of cerebral arteries due to inflammation followed by ischemia causes permanent brain-cell death. After infarction, neurological recovery will be limited. This emphasizes the need to start early anti-inflammatory and anti-tuberculous drugs to prevent vascular complications.[22]

The results of the current study also highlight the need for a clinical and radiological combination assessment in tuberculous meningitis. A clinical diagnosis may not necessarily reflect the extent of disease as radiological abnormalities can reveal more extensive disease. Imaging, on the other hand, can underestimate functional impairment. So, a combined assessment reflects disease severity and prognosis more completely. This holistic approach has been suggested in other studies of similar nature where integrated assessment increased the diagnostic accuracy and the stratification of treatment.[23]

To conclude, the present study showed that the severity of clinical condition, the neuro-imaging and the recovery of the neurologists were highly correlated in patients with tuberculous meningitis. Severe clinical grade hydrocephalus and cerebral infarction were found to be the main factors for poor outcome. The results emphasize that prompt diagnosis and treatment, plus thorough radiological evaluation, are crucial to help achieve a favorable neurological recovery and minimize permanent disability. The results also highlight the importance of better access to health care services and systems of early referral to reduce the presentation of advanced disease and maximize outcomes for patients.

CONCLUSION

Neurological recovery was better in those with mild clinical severity and a limited radiological involvement.
Advanced disease, with extensive neuroimaging abnormalities was associated with poor outcomes.
Better prognosis and management outcome was related to early integrated clinical and imaging evaluation.

Limitations

Only one center was used and generalizability was limited.
Short follow up period was used and there was no long term neurological follow up.
Variability in resources for imaging access was noted and imaging modality was not guaranteed.

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