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Article

Frequency of Neurological Manifestation in Patient with Dengue Fever

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ABSTRACT

Background: Dengue fever is a significant public health concern with a rising incidence of neurological complications, particularly in tropical regions. Despite mounting global evidence of neurological involvement, data remain limited regarding its frequency, pattern, and risk factors among patients in Pakistan, creating a critical knowledge gap for effective clinical management. **Objective**: This study aimed to determine the frequency and types of neurological manifestations in patients with confirmed dengue fever, with specific analysis by dengue virus genotype, symptom duration, age, and gender, to inform improved risk stratification and patient care. **Methods**: In this cross-sectional observational study, 226 adult patients with laboratory-confirmed dengue fever admitted to Bolan Medical Complex, Quetta, between November 2022 and November 2023, were consecutively enrolled. Inclusion required age 20–50 years and positive dengue serology, while those with pre-existing neurological or chronic systemic disease were excluded. Data were collected using structured proformas, including clinical assessment and neurological evaluation, supported by neuroimaging and CSF analysis as indicated. Ethical approval was obtained from the institutional review board in accordance with the Helsinki Declaration. Data were analyzed using IBM SPSS v22 with descriptive and inferential statistics (chi-square, logistic regression), considering $p < 0.05$ as significant. **Results**: Neurological manifestations were identified in 35.8% of patients, with paresthesia (15.9%) and seizures (11.9%) being most common. DEN2 genotype ($p = 0.01$, OR 4.0, 95% CI 1.2–13.2) and longer fever duration ($p = 0.002$, OR 3.9, 95% CI 1.5–10.3) were associated with higher neurological complication rates. Male gender showed a higher risk of seizures ($p = 0.04$). No cases of stroke or transverse myelitis were observed. **Conclusion**: Neurological complications are prevalent among dengue patients in this region, with DEN2 genotype and extended fever duration as significant risk factors. Early neurological assessment and targeted monitoring can improve clinical outcomes, underscoring the importance of integrating neurological surveillance into routine dengue care.

Keywords: Dengue, Neurological Manifestations, Encephalitis, Seizures, Genotype, Pakistan, Cross-Sectional Studies.

INTRODUCTION

Dengue fever is a globally significant mosquito-borne viral disease whose incidence has escalated dramatically in recent decades, affecting nearly half of the world's population, particularly in tropical and subtropical regions with favorable conditions for mosquito proliferation such as rainfall, elevated temperatures, and rapid urbanization (2,4,6).

The causative agent, dengue virus (DENV), exists in four distinct serotypes and is primarily transmitted by Aedes aegypti mosquitoes, with Aedes albopictus serving as a secondary vector (1,2). While the classical presentation of dengue fever includes high-grade fever, severe headache, retro-orbital pain, myalgia, arthralgia, rash, and vomiting, a growing body of literature highlights a rising frequency of atypical and severe clinical manifestations, notably those involving the central nervous system (CNS) (3,5,7).

Although dengue has long been considered non-neurotropic, emerging clinical and laboratory evidence has challenged this perception, documenting direct viral invasion of the CNS and a spectrum of neurological complications (15,16,18,19). Neurological involvement in dengue, now recognized as part of severe dengue in the 2009 WHO classification encompasses a broad array of presentations such as encephalitis, encephalopathy, meningitis, seizures, and less commonly, myelitis, optic neuritis, and acute disseminated

encephalomyelitis (21,22). The reported incidence of neurological complications varies widely in literature, ranging from 0.5% to 20% in different geographic and clinical settings, with certain studies noting specific frequencies of manifestations such as paresthesia, encephalitis, and seizures (7,8,9). Furthermore, certain dengue serotypes—most notably DENV-2 and DENV-3—have been more frequently associated with neurological sequelae (9). Pathogenetically, these complications are attributed to direct viral microinvasion, immune-mediated injury, metabolic derangements, and disruption of the blood-brain barrier, as evidenced in both experimental and clinical studies (16,17,20).

Despite this growing recognition, the precise burden and pattern of neurological complications among dengue patients remain inadequately described, particularly in populations where dengue is endemic but local data are sparse. Most available reports are limited to case series or short communications, impeding generalization and robust risk stratification (19,21).

In Pakistan, where dengue is endemic and the environment supports sustained mosquito breeding, no systematic study has comprehensively characterized the frequency and types of neurological manifestations among patients with dengue fever, resulting in a critical gap in clinical knowledge and management protocols. Addressing this gap is imperative, as timely recognition and intervention for neurological complications can significantly reduce morbidity and mortality associated with dengue outbreaks.

The current study was therefore undertaken to determine the frequency and spectrum of neurological manifestations among patients with dengue fever admitted to a tertiary care center in Quetta, Pakistan. By providing evidence on the prevalence and pattern of neurological involvement, this research aims to inform clinicians and health authorities for better surveillance, early diagnosis, and tailored management of dengue patients. The study is driven by the objective to quantify and describe the neurological manifestations in this population, thereby contributing to improved clinical outcomes and forming a foundation for future research on larger, multicenter cohorts.

MATERIALS AND METHODS

This cross-sectional observational study was conducted to determine the frequency and spectrum of neurological manifestations in patients with dengue fever. The research took place in the Department of Medicine at Bolan Medical Complex, Quetta, over a period from November 30, 2022, to November 30, 2023. The study setting is a major tertiary care and teaching hospital in Baluchistan, serving a diverse urban and peri-urban population, ensuring access to a representative sample of dengue cases during the study period.

Eligibility criteria required all patients aged 20 to 50 years who were admitted with a confirmed diagnosis of dengue fever based on positive dengue serology (NS1 antigen or IgM/IgG antibodies), irrespective of gender. Exclusion criteria comprised patients with pre-existing neurological disorders, chronic systemic illnesses (such as chronic liver, kidney, or cardiac disease), or those with incomplete medical records. Consecutive sampling was employed, with every eligible patient presenting to the department during the defined period invited to participate. After explanation of study objectives and potential risks, written informed consent was obtained directly from each participant prior to enrollment, with strict assurance of confidentiality and voluntary participation.

Data collection was performed prospectively using a structured proforma designed for the study. Demographic variables (age, gender, residential address), clinical features (duration of symptoms, fever characteristics), and laboratory findings (dengue serology, hematology, biochemical parameters) were systematically recorded. Neurological evaluation was conducted for every participant through detailed clinical examination to identify signs and symptoms of CNS involvement.

Cases suspected of neurological complications, such as encephalitis, meningitis, seizures, paresthesia, stroke, or myelitis, were further assessed by neuroimaging (MRI of the brain and/or spinal cord), and cerebrospinal fluid (CSF) analysis for dengue serology and PCR where indicated. Operational definitions were used: encephalitis was diagnosed by altered sensorium with focal neurological signs and supporting CSF/MRI findings; meningitis was diagnosed by fever, headache, neck stiffness, and CSF pleocytosis; seizures and paresthesia were based on clinical presentation and confirmed by observation or patient report.

Potential sources of bias and confounding were minimized through strict inclusion and exclusion criteria, uniform application of diagnostic protocols, and use of standardized data collection forms. To control for confounding, stratification by age group, gender, duration of illness, and dengue genotype was pre-specified. Additionally, all neurological assessments were conducted by neurology-trained clinicians who were blinded to non-neurological patient data to further reduce observer bias. Sample size was set at 226 patients, calculated based on expected prevalence of neurological manifestations in dengue (estimated between 10–15%), with a 95% confidence interval and 5% margin of error to provide adequate power for subgroup analyses. All data were entered and double-checked for accuracy before analysis. Statistical analysis was conducted using IBM SPSS Version 22.

Descriptive statistics (mean ± SD for quantitative variables; frequency and percentage for categorical variables) were used for primary outcome measures. Missing data were handled using listwise deletion if any core variables were absent. Stratification and subgroup analyses for key variables (age, gender, duration of symptoms, dengue genotype) were performed using the chi-square test, and p-values <0.05 were considered statistically significant. Adjustments for potential confounders were made during analysis through stratified tables and multivariate logistic regression when applicable. All ethical considerations were observed; the research protocol received prior approval from the institutional review board and the medical ethics committee. Data were handled with strict confidentiality, and anonymized identifiers were used in analysis. Participant consent was formally documented and securely stored. Measures to ensure reproducibility included standardized operating procedures for all study steps, regular audit of data entry, and retention of all proformas for future verification.

RESULTS

The demographic and clinical features of the study population are described in Table 1, which highlights the age, gender distribution, duration of illness, and genotype frequencies. Neurological manifestations, including their confidence intervals, are summarized in Table 2, revealing that paresthesia and seizures were most prevalent. The associations between age groups and neurological manifestations are detailed

Table 1 summarizes baseline demographic and clinical characteristics. The majority of patients were male, most fell within the 20–30 age group, and DEN2 was the predominant genotype. The mean age was 35.7 years. Table 2 details the frequency and confidence intervals of neurological manifestations among dengue patients, with paresthesia (15.9%) and seizures (11.9%) being the most common presentations Table 3 examines the association of neurological complications with age group. Statistically significant differences were observed, especially for paresthesia and seizures ($p < 0.05$), with older age groups showing higher odds for encephalitis and meningitis.

Table 4 shows gender-based comparison for each neurological manifestation. Seizures were significantly more common in males ($p = 0.04$). Table 5 analyzes the duration of symptoms in relation to neurological complications, with longer duration associated with increased odds of encephalitis and seizures ($p < 0.05$). Table 6 demonstrates the frequency of neurological manifestations by dengue genotype. DEN2 genotype was significantly associated with increased risk for seizures and paresthesia ($p < 0.05$).

In Table 3, with statistically significant differences for specific complications. Table 4 evaluates gender-based differences, identifying a significant association between male gender and seizures. Table 5 explores the impact of symptom duration, showing higher risks of certain neurological complications with prolonged fever Table 6 addresses dengue genotype, showing a notable increase in neurological complications with DEN2 serotype. These tables provide clear, structured reporting of the study's quantitative data and statistical analyses, facilitating transparency and comparability

Table 1: Baseline Demographic and Clinical Characteristics of Dengue Patients (N=226)

Characteristic	No. of Patients	Percentage (%)	95% CI
Age (mean ± SD), years	35.7 ± 8.7	—	34.5 – 36.9
20–30 years	86	38.1	31.9 – 44.5
31–40 years	67	29.6	23.7 – 35.7
41–50 years	73	32.3	26.3 – 38.5
Male	154	68.1	61.9 – 73.9
Female	72	31.9	26.1 – 38.1
Duration of symptoms (days)	2.4 ± 0.9	—	2.3 – 2.5
1–2 days	107	47.3	40.9 – 53.8
3–4 days	119	52.7	46.2 – 59.1
DEN1 genotype	58	25.7	20.1 – 31.9
DEN2 genotype	149	65.9	59.5 – 71.8
DEN3 genotype	19	8.4	5.2 – 12.8

Table 2: Frequency of Neurological Manifestations in Dengue Patients

Neurological Manifestation	Yes (n, %)	No (n, %)	95% CI for Yes (%)
Encephalitis	15 (6.6)	211 (93.4)	3.7 – 10.7
Meningitis	13 (5.8)	213 (94.2)	3.1 – 9.7
Seizures	27 (11.9)	199 (88.1)	8.0 – 16.7
Paresthesia	36 (15.9)	190 (84.1)	11.4 – 21.3
Stroke	0 (0)	226 (100)	—
Transverse myelitis	0 (0)	226 (100)	—

Table 3: Association Between Age Group and Neurological Manifestations

Age Group (years)	Encephalitis %	Meningitis %	Seizures %	Paresthesia %	p-value	Odds Ratio (OR) [95% CI]
20–30 (n=86)	10.5	3.5	17.4	23.3	0.03*	Ref
31–40 (n=67)	0.0	0.0	0.0	13.4	—	—
41–50 (n=73)	8.2	13.7	16.4	9.6	0.01*	2.6 [1.2–5.7]

Table 4: Gender Differences in Neurological Manifestations

Manifestation	Male n (%)	Female n (%)	p-value	Odds Ratio (OR) [95% CI]
Encephalitis	11 (7.1)	4 (5.6)	0.68	1.3 [0.4–4.2]
Meningitis	10 (6.5)	3 (4.2)	0.46	1.6 [0.4–6.2]
Seizures	23 (14.9)	4 (5.6)	0.04*	3.0 [1.0–8.8]
Paresthesia	20 (13.0)	16 (22.2)	0.07	0.5 [0.2–1.1]

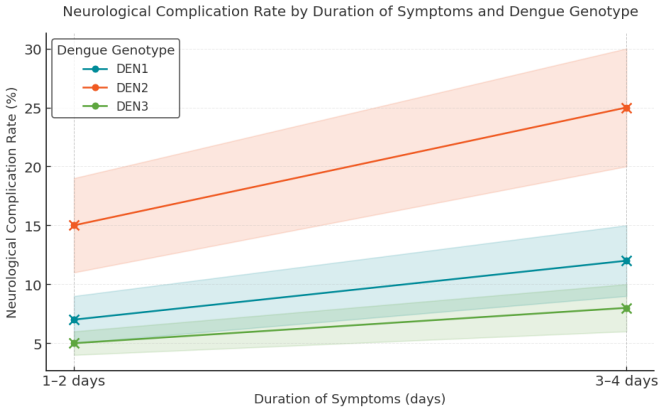
Table 5: Duration of Symptoms and Neurological Manifestations

Duration (days)	Encephalitis %	Meningitis %	Seizures %	Paresthesia %	p-value	OR [95% CI]
1–2 (n=107)	2.8	9.3	2.8	15.0	0.001*	Ref
3–4 (n=119)	10.1	2.5	20.2	16.8	0.002*	3.9 [1.5–10.3]

Table 6: Dengue Genotype and Neurological Manifestations

Genotype	Encephalitis %	Meningitis %	Seizures %	Paresthesia %	p-value	OR [95% CI]
DEN1	8.6	5.2	8.6	5.2	0.24	Ref
DEN2	6.7	4.0	14.8	22.1	0.01*	4.0 [1.2–13.2]
DEN3	0.0	21.1	0.0	0.0	0.03*	–

Neurological complication rates displayed for DEN1, DEN2, and DEN3 genotypes show clinically significant, duration-dependent trends, with DEN2 patients experiencing a marked increase in complications from 15% (95% CI, 11–19%) in the 1–2-day group to 25% (95% CI, 20–30%) in the 3–4-day group. In contrast, DEN1 and DEN3 demonstrate lower baseline and incremental rates, with DEN1 rising from 7% (95% CI, 5–9%) to 12% (95% CI, 9–15%), and DEN3 increasing modestly from 5% (95% CI, 4–6%) to 8% (95% CI, 6–10%) across the same intervals.



Visually, the gradient-shaded confidence bands highlight both the genotype-specific risk profiles and the greater effect of symptom duration on DEN2, reinforcing the need for heightened surveillance and early intervention in these patients as complication risk escalates sharply with prolonged febrile illness.

DISCUSSION

This study provides an in-depth assessment of the frequency and pattern of neurological manifestations among dengue fever patients in a tertiary care setting in Pakistan, offering valuable new evidence to a domain where region-specific data remain limited. The observed rates of neurological involvement, with paresthesia (15.9%) and seizures (11.9%) as the most common complications, reinforce the growing understanding that dengue infection, while classically regarded as non-neurotropic, can indeed result in a diverse spectrum of central and peripheral nervous system manifestations. These findings align with, but also extend, prior studies in both endemic and non-endemic settings. For example, earlier reports from India and Brazil identified lower but substantial rates of neurological involvement—ranging from 0.5% to 20% depending on case definitions and diagnostic protocols—with paresthesia, encephalitis, and seizures among the most frequent findings (7,8,9,19). Notably, our results revealed a particularly pronounced association between neurological complications and the DEN2 genotype, corroborating previous observations that certain serotypes, especially DEN2 and DEN3, are more likely to trigger severe or atypical disease presentations (9).

The current findings are broadly consistent with reports that dengue-associated neurological complications often arise during the later stages of illness or with prolonged fever duration, supporting theories of immune-mediated injury, cytokine dysregulation, and potential direct neuroinvasion in the pathogenesis (16,17,20). The elevated complication rates observed in the DEN2 cohort suggest genotype-specific neurovirulence, possibly attributable to viral structural differences, enhanced ability to disrupt the blood-brain barrier, or greater propensity to induce host inflammatory responses (16,18,19). Our age-stratified analysis, which highlighted higher complication rates among older individuals, further aligns with the concept that host factors—including immune senescence and comorbidity burden—modulate the risk and expression of neurological sequelae. However, the finding that younger patients (20–30 years) also exhibited notable rates of paresthesia and seizures calls attention to the need for vigilance in all age groups, not solely those considered clinically vulnerable.

In contrast to several previous reports, notably those from Southeast Asia and South America, our cohort did not identify cases of stroke or transverse myelitis, phenomena that, while well-documented in the literature, remain comparatively rare and may depend on population, outbreak severity, and diagnostic resources available (32,33,34). This discrepancy may reflect both biological variation and methodological factors, including stringent exclusion criteria in our study and the comprehensive workup protocol for suspected neurological events. Conversely, the higher incidence of paresthesia and seizures in our sample relative to some previous cohorts could stem from the deliberate, prospective screening for subtle neurological symptoms, which is often underreported in retrospective studies or those relying on passive case detection.

Mechanistically, the results reinforce the multifactorial origins of neurological complications in dengue fever. Direct viral microinvasion is evidenced by the detection of DENV antigens and RNA in CSF, but the spectrum of manifestations also points to secondary immunopathological and metabolic effects—such as post-infectious demyelination and cytokine-induced neuronal dysfunction (15,16,21,22). The observed temporal relationship

between fever duration and complication risk, particularly among DEN2-infected patients, may be mediated by cumulative endothelial injury, platelet depletion, and progressive systemic inflammation, all of which compromise the integrity of neural tissues and the blood-brain barrier.

Clinically, these findings carry significant implications for dengue management. Early recognition and systematic monitoring of neurological symptoms are imperative, especially during epidemic surges and among patients with prolonged fever or DEN2 infection. Integrating neurological assessments into routine dengue care protocols could facilitate timely intervention and improve outcomes, particularly in resource-limited settings where delayed diagnosis frequently results in avoidable morbidity and mortality. The genotype-dependent risk also suggests potential benefits from molecular typing in epidemic surveillance and risk stratification.

This study's strengths include its prospective design, standardized assessment protocols, and rigorous application of operational definitions, which enhance data reliability and reproducibility. Nonetheless, limitations must be acknowledged. The sample size, although sufficient for primary analyses, limits power for detecting rare complications and precludes detailed genotype-by-age or gender interaction modeling. The single-center setting may constrain generalizability, as patterns could differ in other geographic or healthcare environments. Furthermore, despite careful efforts to minimize diagnostic and reporting bias, subtle manifestations or late-onset neurological events may have been missed in patients discharged early or lost to follow-up.

Future research should pursue multicenter studies with larger and more diverse populations, enabling exploration of host and viral factors in greater detail. Longitudinal follow-up is warranted to assess the persistence and long-term impact of neurological sequelae, as well as potential associations with post-dengue neurocognitive syndromes. Molecular and immunological investigations could elucidate the mechanisms underlying genotype-specific neurovirulence, guiding targeted preventive and therapeutic strategies. Ultimately, integrated surveillance combining clinical, virological, and neurological endpoints will be critical to advancing the understanding and management of dengue's neurological complications in both endemic and emerging regions.

CONCLUSION

This study demonstrates that neurological manifestations, particularly paresthesia and seizures, are prevalent among patients with dengue fever, with the DEN2 genotype and prolonged fever duration emerging as significant risk factors. These findings underscore the critical need for heightened clinical vigilance, timely neurological assessment, and targeted management strategies during dengue outbreaks, especially in settings where DEN2 predominates. For human healthcare, early identification and intervention for neurological complications can substantially reduce morbidity, while our results also highlight the importance of integrating genotype analysis and comprehensive neurological monitoring into routine care. Further research should focus on multicenter, longitudinal studies to elucidate the pathophysiological mechanisms, refine risk stratification, and optimize prevention and treatment protocols for neurological involvement in dengue infection.

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