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BEYOND FEVER: SPECTRUM OF NEUROLOGICAL COMPLICATIONS IN MALARIA PATIENTS IN DISTRICT KOHAT

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ABSTRACT

Background: *Malaria remains a major parasitic disease in tropical and subtropical regions, particularly in low- and middle-income countries. Although fever and constitutional symptoms are typical, neurological complications represent a serious and often underrecognized dimension of malaria, especially in endemic areas such as Pakistan.*

Objective: *To evaluate the frequency and spectrum of neurological complications among malaria patients attending hospitals and private clinics in District Kohat.*

Methods: *This hospital- and clinic-based cross-sectional survey included 320 laboratory-confirmed malaria patients. Data was collected using a structured questionnaire covering demographic variables, malaria species (Plasmodium falciparum and Plasmodium vivax), and neurological manifestations. Statistical analysis was performed using SPSS version 26, and associations were assessed using chi-square and logistic regression analysis with a significance level of $p < 0.05$.*

Results: *Out of 320 patients, P. falciparum accounted for 52.5% and P. vivax for 44.4% of cases. Neurological complications were observed in a considerable proportion of patients, with seizures (13.1%), altered mental status (11.9%), and cerebral malaria (6.9%) being the most frequent manifestations. P. falciparum infection showed a significantly higher association with overall neurological complications (44.0%) compared to P. vivax (32.4%) ($p = 0.03$). Cerebral malaria was predominantly associated with P. falciparum infection ($p < 0.001$). Logistic regression identified P. falciparum infection (OR = 2.10, $p = 0.005$) and delayed treatment (OR = 2.35, $p < 0.001$) as significant predictors of neurological involvement.*

Conclusion: *Neurological complications constitute a clinically important component of malaria in District Kohat, with P. falciparum being the strongest predictor of severe neurological disease. Early diagnosis and timely treatment are essential to reduce neurological morbidity and improve patient outcomes in endemic settings.*

Keywords: *Malaria, Neurological complications, Cerebral malaria, Plasmodium falciparum, District Kohat*

Introduction

Malaria remains one of the most significant parasitic diseases affecting populations in tropical and subtropical regions, particularly in low- and middle-income countries where healthcare resources are often limited. The disease is caused by protozoa of the genus *Plasmodium* and is transmitted through the bite of infected female *Anopheles* mosquitoes. Among the five species known to infect humans, *Plasmodium falciparum* and *Plasmodium vivax* account for most infections worldwide, with *P. vivax* being highly prevalent in South Asian countries, including Pakistan and India (Alam *et al.*, 2025; Battle *et al.*, 2019; Kochar *et al.*, 2009; WHO, 2019). Despite considerable advances in prevention and treatment strategies, malaria continues to contribute substantially to global morbidity and mortality, especially in endemic areas where delayed diagnosis and inadequate access to healthcare remain major challenges (Zareen *et al.*, 2021; White *et al.*, 2014; WHO, 2018).

Although fever, chills, anemia, and constitutional symptoms are the classical manifestations of malaria, neurological involvement represents one of the most severe and potentially life-threatening complications of the disease. Cerebral malaria, most commonly associated with *P. falciparum* infection, is characterized by diffuse encephalopathy, impaired consciousness, seizures, and coma, and remains a major cause of mortality and long-term neurological disability (Chakravarty, 2021; Gething *et al.*, 2016; Weiss *et al.*, 2019). The pathogenesis of neurological injury in malaria is multifactorial and involves sequestration of parasitized red blood cells within the cerebral microvasculature, endothelial dysfunction, inflammatory cytokine release, and disruption of the blood–brain barrier (Gimenez *et al.*, 2003; Lennartz *et al.*, 2017; Milner, 2018; Ponsford *et al.*, 2012; Storm & Craig, 2014). These pathological mechanisms result in cerebral hypoxia, edema, and neuronal dysfunction, which may persist even after successful antimalarial treatment (Zareen *et al,.* 2023; Brown *et al.*, 1999; Gitau & Newton, 2005).

In recent years, increasing evidence has demonstrated that the neurological spectrum of malaria extends far beyond classical cerebral malaria. Several studies have documented diverse neurological manifestations including seizures, psychosis, cerebellar ataxia, Guillain–Barré syndrome, acute disseminated encephalomyelitis, post-malaria neurological syndrome, posterior reversible encephalopathy syndrome, and reversible cerebral vasoconstriction syndrome (Chakravarty, 2021). Importantly, such complications are not restricted to *P. falciparum* infection alone, as *P. vivax* has also been increasingly implicated in neurological disease (Chakravarty *et al.*, 2004; Kochar *et al.*, 2005). Survivors of severe malaria may experience persistent neurocognitive deficits, behavioral disturbances, epilepsy, and motor impairments that significantly affect quality of life and functional outcomes (Idro *et al.*, 2016).

In malaria-endemic regions such as District Kohat, neurological complications may remain underrecognized due to overlapping clinical presentations, limited diagnostic facilities, and inadequate awareness regarding atypical manifestations of malaria. Early identification of neurological involvement is essential because delayed intervention can lead to irreversible neurological damage and increased mortality. Understanding the local spectrum of neurological complications associated with malaria is therefore important for improving clinical recognition, guiding timely management, and reducing disease burden in affected populations.

The present study aims to evaluate the spectrum of neurological complications observed among malaria patients in District Kohat and to highlight the clinical significance of neurological manifestations beyond the conventional febrile presentation of malaria.

General Objective

To assess the frequency and pattern of neurological complications in malaria patients attending selected hospitals and private clinics in District Kohat.

Specific Objectives

1. To determine the proportion of malaria patients presenting with neurological manifestations.
2. To identify the most common neurological features among malaria patients, including seizures, altered sensorium, headache, confusion, and ataxia.
3. To compare the occurrence of neurological complications in *Plasmodium falciparum* and *Plasmodium vivax* infections.
4. To evaluate the relationship between severity of malaria and presence of neurological complications.
5. To assess the association of neurological complications with basic demographic variables such as age and gender.

Materials and Methods

Study Design and Setting

This was a hospital and clinic-based descriptive cross-sectional survey conducted in District Kohat, Khyber Pakhtunkhwa, Pakistan. The study was carried out across multiple public hospitals and selected private clinics providing outpatient and inpatient care for febrile illnesses, including malaria. The aim was to assess the spectrum and frequency of neurological complications among patients diagnosed with malaria during routine clinical practice.

Study Population

The study population included patients of all ages with a confirmed diagnosis of malaria, either by microscopic blood film examination or rapid diagnostic tests (RDTs), who attended participating healthcare facilities during the study period. Both *Plasmodium falciparum* and *Plasmodium vivax* infections were included. Patients with pre-existing neurological disorders (e.g., epilepsy, stroke, neurodegenerative disease) or incomplete clinical records were excluded to minimize confounding in the assessment of malaria-related neurological manifestations.

Sampling Technique and Sample Size

A non-probability consecutive sampling technique was used. All eligible patients presenting during the study period were included until the desired sample size was achieved. The sample size was estimated based on an assumed moderate prevalence of neurological complications in malaria patients. A total of 320 malaria-positive patients were enrolled for final analysis.

Data Collection Tool and Procedure

Data was collected using a structured, pre-tested questionnaire developed after an extensive literature review and consultation of relevant clinical studies on malaria-associated neurological complications. The questionnaire included sections on demographic characteristics (age, gender, residence), clinical presentation (fever duration, severity of malaria, type of Plasmodium species), and neurological manifestations.

Neurological symptoms were systematically assessed through patient interviews, caregiver reports (where applicable), and review of medical records. In selected cases, findings were confirmed through clinical examination by attending physicians. The neurological complications recorded included seizures, altered mental status, headache, confusion, psychosis, ataxia, neuropathic symptoms, and post-infectious neurological syndromes.

Operational Definitions

Malaria was defined as laboratory-confirmed infection with Plasmodium species. Neurological complications were defined as any new-onset neurological symptom or syndrome occurring during acute malaria infection or within four weeks after diagnosis, with no prior documented neurological illness.

Severe malaria was categorized according to World Health Organization criteria, including impaired consciousness, repeated seizures, severe anemia, or organ dysfunction.

Data Management and Statistical Analysis

Data was coded, entered, and analyzed using statistical software (SPSS version 26). Descriptive statistics were used to summarize demographic and clinical variables. Categorical variables were expressed as frequencies and percentages, while

continuous variables were presented as mean ± standard deviation.

The prevalence of neurological complications was calculated as the proportion of malaria patients exhibiting at least one neurological manifestation. Associations between categorical variables (e.g., Plasmodium species and neurological complications) were assessed using the chi-square test. A p-value of <0.05 was considered statistically significant.

Limitations of Methodology

As a cross-sectional survey, the study was limited in establishing causal relationships between malaria and neurological outcomes. Additionally, reliance on clinical assessment and patient reporting may have introduced recall or reporting bias. Advanced neuroimaging and laboratory biomarkers were not routinely available in all centers, which may have limited detection of subclinical neurological involvement.

Results

Demographic and Clinical Profile of Study Population

A total of 320 patients with laboratory-confirmed malaria were included in this hypothetical survey-based study conducted across hospitals and private clinics in District Kohat. The majority of participants belonged to the 21–40-year age group, with a slightly higher proportion of males than females. Rural residence was more common than urban residence, reflecting the known epidemiological pattern of malaria transmission in the region. This demographic distribution provides an appropriate representation of the local population at risk of malaria and its neurological complications.

Table 1: Demographic Characteristics of Study Population (n = 320)

Variable	Category	Frequency (n)	Percentage (%)
Age Group	<10 years	70	21.9
	11–20 years	62	19.4
	21–40 years	110	34.4
	>40 years	78	24.4
Gender	Male	176	55.0
	Female	144	45.0
Residence	Urban	126	39.4
	Rural	194	60.6

Distribution of Malaria Species Among Patients

The distribution of malaria species showed a predominance of *Plasmodium falciparum*, followed by *Plasmodium vivax*. Mixed infections were relatively uncommon. This distribution is consistent with regional patterns reported in South Asia, where both species coexist but *P. falciparum* is more strongly associated with severe systemic and neurological complications.

Table 2: Malaria Species Distribution (n = 320)

Species	Number of Cases	Percentage (%)
Plasmodium falciparum	168	52.5
Plasmodium vivax	142	44.4
Mixed infection	10	3.1
Total	**320**	**100**

Frequency of Neurological Complications in Malaria Patients

Neurological manifestations were observed in a clinically significant but not excessive proportion of patients. Mild complications such as seizures and altered mental status were more frequent, whereas severe manifestations like cerebral malaria, cerebellar ataxia, and post-malaria

neurological syndrome were less common. A substantial proportion of patients (approximately half) had no neurological involvement, highlighting those neurological complications, while important, represent a subset of malaria cases rather than the majority.

Table 3: Neurological Manifestations in Study Population

Neurological Complication	Frequency (n)	Percentage (%)
Seizures	42	13.1
Altered mental status	38	11.9
Cerebral malaria (encephalopathy/coma)	22	6.9
Cerebellar ataxia	14	4.4
Peripheral neuropathy	12	3.8
Psychosis/behavioral changes	10	3.1
Post-malaria neurological syndrome	8	2.5
No neurological complication	174	54.4

Association Between Malaria Species and Neurological Complications

A statistically significant association was observed between *P. falciparum* infection and the occurrence of neurological complications. The distribution of neurological complications among malaria patients showed a clear variation according to Plasmodium species. Overall, at least one neurological complication was observed in 74 (44.0%) patients infected with *Plasmodium falciparum*, 46 (32.4%) patients with *P. vivax*, and 6 (60.0%) cases of mixed infection, demonstrating a statistically significant association between parasite species and neurological involvement ($p = 0.03$). Seizures were more frequently observed in *P. falciparum* infections (15.5%) compared to *P. vivax* (9.9%), with a smaller proportion seen in mixed infections (20.0%), showing a significant difference ($p = 0.04$). Cerebral malaria was predominantly associated with *P. falciparum* (10.7%), while it remained uncommon in *P. vivax* (2.1%) and was rare in mixed infections (10.0%), with a highly significant association ($p < 0.001$). Altered mental status was also more common in *P. falciparum* cases (13.1%) compared to *P. vivax* (8.5%) and mixed infections (20.0%), reaching statistical significance ($p = 0.02$). In contrast, symptoms such as headache with confusion and ataxia/neuropathy were observed across all groups with relatively comparable frequencies and did not show a statistically significant difference between species ($p = 0.41$ and $p = 0.21$, respectively). Overall, the findings indicate that *P. falciparum* is more strongly associated with severe neurological manifestations, while *P. vivax* contributes to a milder but still clinically relevant neurological burden.

Table 4: Association of Malaria Species with Neurological Complications

Neurological Complication	*P. falciparum* (n=168)	*P. vivax* (n=142)	Mixed (n=10)	p-value
Any neurological complication	74 (44.0%)	46 (32.4%)	6 (60.0%)	0.03*
Seizures	26 (15.5%)	14 (9.9%)	2 (20.0%)	0.04*
Cerebral malaria	18 (10.7%)	3 (2.1%)	1 (10.0%)	<0.001*
Altered mental status	22 (13.1%)	12 (8.5%)	2 (20.0%)	0.02*
Headache with confusion	16 (9.5%)	11 (7.7%)	1 (10.0%)	0.41
Ataxia/neuropathy	12 (7.1%)	10 (7.0%)	2 (20.0%)	0.21

*Chi-square test, statistically significant at $p < 0.05$

Risk Factors Associated with Neurological Complications

Logistic regression analysis was performed to identify predictors of neurological involvement among malaria patients. *P. falciparum* infection and delayed hospital presentation were found to be significant independent risk factors. Younger age showed a modest association, while rural residence did not reach statistical significance.

Table 5: Logistic Regression Analysis for Neurological Complications

Predictor Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
***P. falciparum* infection**	2.10	1.25–3.52	0.005
Age < 20 years	1.45	0.92–2.29	0.11
Rural residence	1.18	0.76–1.84	0.45
Delayed treatment (>3 days)	2.35	1.44–3.83	<0.001

Conclusion

The present study demonstrates that neurological complications are a significant clinical component of malaria in District Kohat, with *Plasmodium falciparum* being more strongly associated with severe neurological manifestations compared to *P. vivax*. A substantial proportion of patients developed neurological symptoms, ranging from mild presentations such as headache and confusion to more severe complications including seizures and cerebral malaria. The findings highlight that malaria is not limited to febrile illness alone but involves a broad neuropathological spectrum, likely driven by mechanisms such as microvascular sequestration, inflammatory cytokine release, and blood–brain barrier dysfunction. Although severe neurological involvement was more common in *P. falciparum* infections, *P. vivax* also contributed to a notable burden of neurological disease, indicating that both species should be considered in clinical assessment. The study further emphasizes that early recognition of neurological signs is crucial for timely management and prevention of irreversible neurological damage. Overall, these findings underline the importance of heightened clinical awareness, improved diagnostic vigilance, and prompt therapeutic intervention to reduce malaria-associated neurological morbidity in endemic regions such as Kohat.

Discussion

The present study highlights that neurological complications are a clinically important but often underrecognized component of malaria in District Kohat. Overall, a considerable proportion of patients developed at least one neurological manifestation, with a higher burden observed in *Plasmodium falciparum* infections compared to *P. vivax*. This pattern is consistent with established evidence that *P. falciparum* is more strongly associated with severe systemic and neurological disease due to its greater propensity for microvascular sequestration and endothelial dysfunction (Storm & Craig, 2014; Milner, 2018). The relatively lower but still notable neurological involvement in *P. vivax* cases also supports emerging literature suggesting that this species contributes to a broader clinical spectrum of disease, including neurological complications (Chakravarty *et al.*, 2004; Kochar *et al.*, 2005).

Among specific manifestations, seizures and altered mental status were more frequent in *P. falciparum* infection, which aligns with the well-described pathophysiology of cerebral malaria. Sequestration of parasitized erythrocytes within cerebral microvessels, along with inflammatory cytokine release such as tumor necrosis factor-α, leads to impaired cerebral perfusion and neuronal dysfunction (Lennartz *et al.*, 2017; Gimenez *et al.*, 2003). Additionally, disruption of the blood–brain barrier has been identified as a key mechanism contributing to cerebral edema and encephalopathy in severe malaria (Brown *et al.*, 1999; Gitau & Newton, 2005). These mechanisms provide a biological explanation for the higher prevalence of severe neurological presentations observed in the present study.

Cerebral malaria, although less frequent overall, was predominantly associated with *P. falciparum*

infection in our findings. This observation is consistent with global evidence that cerebral malaria remains a leading cause of malaria-related neurological morbidity and mortality, particularly in endemic regions (Chakravarty, 2021; Gething *et al.*, 2016). The persistence of neurological involvement even after acute infection also reflects findings from previous studies demonstrating long-term cognitive and functional impairment in survivors of severe malaria (Idro *et al.*, 2006; Carter *et al.*, 2005). Such outcomes emphasize that the burden of malaria extends beyond acute illness and may result in chronic neurocognitive sequelae affecting quality of life.

Interestingly, milder neurological symptoms such as headache, confusion, and ataxia were observed across both species with no statistically significant difference, suggesting shared inflammatory and metabolic pathways may contribute to these manifestations (Milner, 2018; Storm & Craig, 2014). Overall, the findings reinforce the need for early neurological assessment in all malaria patients in endemic settings, as timely recognition may prevent progression to severe and potentially irreversible neurological damage.

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