Stroke as a neurological complication of malaria in a young adult: a case report

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ABSTRACT

Background: Stroke is a rare but serious complication of malaria, especially with *Plasmodium falciparum* infection, as a result of cerebral vessel obstruction by the parasitized red blood cells. Prompt recognition in malaria patients presenting with neurological symptoms is essential for effective management.

Case Presentation: A 32-year-old male with no prior cardiovascular risk factors developed acute neurological symptoms after treatment for confirmed malaria. Brain imaging showed multifocal ischemic infarcts. Comprehensive investigations excluded common causes of stroke, including hypertension, diabetes, coagulopathies, and cardiac sources of embolism, as well as vasculitis. The close timing between malaria infection and strokes, along with the absence of alternative explanations, supported a diagnosis of malaria-associated ischemic stroke. The patient continued antimalarial therapy and received supportive care, including physiotherapy. Over subsequent weeks, neurological deficits gradually improved with rehabilitation.

Conclusion: Malaria-induced stroke, though uncommon, should be considered in young patients presenting with acute neurological deficits in endemic areas. Early diagnosis and integrated management of both malaria and stroke can improve patient outcomes and reduce disability. Increased awareness among clinicians is important to identify this rare but treatable complication.

Keywords: Malaria-induced, ischemic stroke, severe malaria, Plasmodium falciparum, case report.

Type of Article: CASE REPORT **Specialty:** Urology

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Introduction

Malaria is one of the rare infectious causes of stroke worldwide and remains a significant global health challenge, with *Plasmodium falciparum* causing the most severe forms of the disease. According to WHO's latest *World malaria report*, there were an estimated 263 million cases and 597 000 malaria deaths worldwide in 2023 [1]. Neurological complications such as cerebral malaria are well known; however, strokes associated with malaria are rare and not fully understood. The underlying mechanisms include microvascular obstruction, inflammation, and endothelial dysfunction, which may result in ischemic or hemorrhagic strokes [2]. Current standard care for severe malaria emphasizes prompt administration of effective antimalarial drugs, supportive treatment, and management of complications [3]. Stroke management in these patients generally follows established ischemic stroke protocols, including neurological support and preventative measures [4].

This case report presents a young, previously healthy adult who experienced an ischemic stroke shortly after a severe malaria infection, without traditional stroke risk factors. It draws attention to the importance of recognizing this uncommon but serious complication in malaria patients. The report highlights the need to incorporate neurological evaluation and stroke management into malaria treatment strategies, particularly in endemic areas. Early detection and comprehensive care can improve outcomes and reduce long-term disability.

Case Presentation

A 32-year-old male with no significant past medical history, recently diagnosed with malaria and a urinary tract infection at a dispensary, presented with complaints of dizziness, blurred vision, and fever lasting three days. One day prior, he had been treated with antimalarial therapy (artemether-lumefantrine) and antibiotics. Initial vital signs recorded at the dispensary showed blood pressure

(BP) of 113/77 mmHg, pulse rate 84 beats per minute, and normal blood sugar levels. The patient reportedly improved initially but experienced intermittent ear ringing.

Prior to his arrival at our facility, the patient had breakfast that morning and went out to attend to his daily activities. Later, his mother received a call from a good samaritan reporting that he suddenly experienced dizziness, one episode of vomiting, headache, and slurred speech. He was then taken to the local dispensary, where his blood pressure was elevated at 149/94 mmHg, and he was diclofenac for pain relief. Subsequently, he developed excessive drooling and became unable to drink. Approximately ten minutes after the drooling began, he lost consciousness. There was no history of trauma. Due to the development of right-sided weakness and numbness, along with elevated blood pressure, he was referred to our higher-level facility, although no hypertensive emergency was documented at the time.

On arrival, the patient was unconscious with a Glasgow Coma Scale (GCS) score of 7/15. He was snoring with excessive drooling and frothing at the mouth, no pallor, jaundice, clubbing, edema, nor lymphadenopathy. Vital signs were stable. Neurologic examination showed pupils equal and reactive to light without facial asymmetry. No signs of meningeal irritation. Motor examination revealed right-sided hemiparesis with decreased muscle strength. Muscle tone on the right side was reduced but not completely flaccid. Deep tendon reflexes on the right side were diminished, and Babinski's sign was positive, indicating an upper motor neuron lesion. The left side had normal tone and reflexes. No clonus was elicited. Cranial nerve testing and Sensory testing were limited due to reduced consciousness. Gait, balance, and coordination could not be assessed due to mental status. The rest of the systemic examination were unremarkable.

Malaria testing via thick and thin films confirmed *Plasmodium falciparum* infection with a parasite density of 11%. This level of hyperparasitemia and the presence of neurological deficit satisfy the WHO diagnostic criteria for severe malaria. Hepatitis serology panel showed non-reactive results, syphilis and HIV serology tests were non-reactive, and dengue rapid tests were all negative. Additionally, an autoimmune panel was performed and showed no evidence of systemic lupus erythematosus (SLE) or other causes of vasculitis and acquired thrombophilia (Table 1). These results helped exclude infectious and autoimmune etiologies in the differential diagnosis. Lipid profile and fasting blood glucose were also normal, ruling out common metabolic risk factors for stroke. Blood cultures showed no growth.

Table 1. *Initial laboratory tests.*

Laboratory parameters	Patient's values	Normal range
Leukocyte count, x 109 per l	13.15	4.00-10.00
Neutrophil count, x 109 per l	10.14	2.00-7.00
Lymphocyte count, x 109 per l	2.29	0.80-4.00
Hemoglobin, g/dl	15.0	13.0-18.8
Platelet count, x 109 per l	266	150-350
Random blood glucose, mmol/l	6.9	4-10
Erythrocyte sedimentation rate (ESR), mm/hour	0.00	0-7.8
C-reactive protein (CRP), mg/l	27.9	0-10
Creatinine, mmol/l	48.0	62-121
Blood urea nitrogen, BUN, mmol/l	3.58	1.8-8.3
Aspartate Aminotransferase (AST), IU/l	48.8	5.0-41.0
Alanine Aminotransferase (ALT), IU/l	56.0	5.0-41.0
Serum Sodium, mmol/l	141	135-145
Serum Potassium, mmol/l	3.8	3.5-5.5
Serum Chloride, mmol/l	103	98-108
D-dimer	463	0.00-500
Prothrombin time (PT), Seconds	20.7	11-15
Internal Normalized Ration (INR)	1.83	0-1.6
Activated Partial Thromboplastin Time (aPTT)	30.1	25.8-38.8
C- ANCA, AU/ml	5.37	16-20
P-ANCA, AU/ml	1.18	< 20.0
Anti-nuclear antibodies (ANA)	Negative	-
Anti-phospholipid antibodies	Negative	-
Rheumatoid Factor	Negative	-

Radiographic imaging included a non-contrast brain CT scan, which was normal. However, further evaluation with non-contrast brain MRI, showed right cerebellar, medulla, and pontine acute multiple ischemic infarcts without hemorrhage (Figure 1). Echocardiography demonstrated normal cardiac structure and function without any evidence of atrial thrombus or patent foramen ovale. An electrocardiogram showed a normal sinus rhythm without arrhythmias.

These findings excluded common causes of stroke, supporting a diagnosis of malaria-associated ischemic stroke as a neurological complication of *P. falciparum* infection in this young adult patient.

The switch from oral artemether-lumefantrine to IV artesunate was necessitated by the patient's transition from uncomplicated to severe malaria; therefore, the patient was started on IV artesunate in accordance with WHO protocols. Enoxaparin was administered at a prophylactic dose initially, as CT scan results were inconclusive. Prophylactic enoxaparin was started due to the patient's deranged coagulation profile (Table 1) and high risk of venous thromboembolism.

Once an ischemic stroke was confirmed via brain MRI, antiplatelet therapy with aspirin daily was initiated, and the enoxaparin was discontinued. Supportive stroke care included airway protection, seizure prophylaxis with Levetiracetam, vitamin B6, and vitamin B12.

This combined approach targets both the malaria infection and ischemic stroke, optimizing neurologic recovery and reducing mortality risk.

The patient responded well to the treatment regimen. Repeat blood smears, performed after the completion of the artesunate course, confirmed the clearance of parasitemia**.** Neurological function gradually improved, with partial recovery observed in both motor and cognitive abilities. The patient was discharged two weeks later with a Glasgow Coma Scale (GCS) score of 11/15. Close monitoring for potential complications and ongoing neurorehabilitation remain in place.

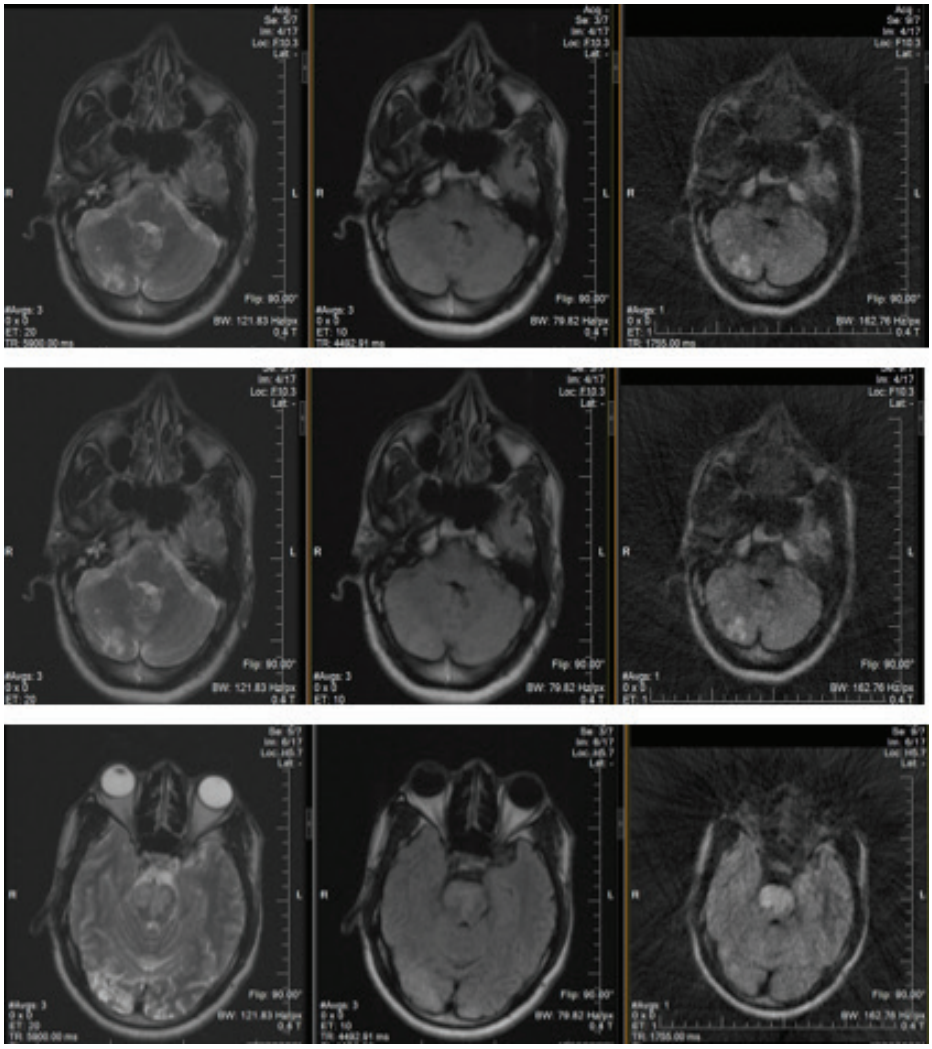

Figure 1. *MRI of the brain showing multiple T2/FLAIR hyperintensities with restricted diffusion involving the right cerebellar hemisphere, medulla, and pons, consistent with acute multiple ischemic infarcts consistent with a small vessel/lacunar distribution. The absence of watershed (border-zone) infarcts effectively ruled out systemic hypotension or low-flow states as the primary cause of the stroke.*

Discussion

Nervous system involvement in malaria primarily presents as cerebral malaria or other neurological symptoms [5]. Stroke, whether hemorrhagic or ischemic, is an uncommon but recognized complication [2,3]. Malaria-induced stroke in young patients is a rare but serious complication that results from multifactorial pathophysiological mechanisms [6]. The primary mechanism involves mechanical obstruction of cerebral vessels by parasitized red blood cells, particularly in infections caused by *Plasmodium falciparum*, leading to local ischemia and infarction [7]. In addition to this mechanical blockage, inflammatory responses induced by the parasite contribute to endothelial damage and a systemic hypercoagulable state, further increasing the risk of cerebral thrombosis and stroke [4,5]. These processes impair cerebral blood flow and trigger neurological deficits, as seen in ischemic stroke presentations. The clinical recognition of malaria-associated stroke is challenging due to symptom overlap with cerebral malaria and other neurological complications of malaria, underscoring the need for heightened diagnostic vigilance in endemic areas. When evaluating a patient with stroke, infectious and tropical diseases should be considered in the differential diagnosis, and neuroimaging plays a critical role in making an accurate diagnosis [8-10]. Management includes prompt initiation of effective antimalarial therapy, supportive care, and antithrombotic treatment, while carefully balancing risks such as thrombocytopenia and hemorrhage [9,10]. Early diagnosis and comprehensive treatment are critical to improving outcomes and reducing the risk of long-term neurological impairment in young patients suffering from malaria-induced stroke.

The patient's GCS of 7, right-sided flaccid hemiparesis with diminished reflexes, positive Babinski sign, and sensory deficit contribute to an approximate score of 15 points, reflecting severity. This classification helps guide clinical treatment decisions and prognosis. A more comprehensive assessment of visual fields, language, dysarthria, and neglect could refine this score further.

Conclusion

Malaria-induced stroke, although rare, should be suspected in patients from endemic regions, even in the absence of traditional stroke risk factors. The pathogenesis is multifactorial, primarily involving mechanical obstruction of cerebral vessels by parasitized red blood cells and an accompanying inflammatory and hypercoagulable state. Early diagnosis and prompt antimalarial treatment, along with supportive care, are essential to improving outcomes and minimizing long-term neurological deficits. This case provides significant insight by demonstrating that malaria-associated stroke can occur in adults with high parasitemia, manifesting as a multiple-territory ischemic stroke that may not be initially detectable on CT scan.

What is new?

This case report highlights a young, otherwise healthy 32-year-old male developing an ischemic stroke shortly after severe malaria infection. It emphasizes the association between malaria and stroke in adult patients without traditional risk factors, expanding awareness of this potential complication. The report underscores the importance of considering malaria as a differential diagnosis in young stroke patients in endemic areas and discusses the management implications, encouraging early diagnosis to improve outcomes.

List of abbreviations

C-ANCA	Cytoplasmic Anti-Neutrophil Cytoplasmic Antibodies
CT	Computed Tomography
DVT	Deep Venous Thrombosis
ECG	Electrocardiogram
ESR	Erythrocyte Sedimentation Rate
FBC	Full Blood Count
ICU	Intensive Care Unit
IV	Intravenous
MRI	Magnetic Resonance Imaging
P-ANCA	Perinuclear Anti-Neutrophil Cytoplasmic Antibodies,
PCR	Polymerase Chain Reaction

Conflict of Interests

The authors declare that there is no conflict of interest regarding the publication of this article.

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Consent for publication

Written informed consent was obtained from the next kin of the patient (since the patient is unable to provide consent for publication)

Ethical Approval

Ethical approval is not required at our institution to publish an anonymous case report.

Take-home message

In malaria-endemic regions, clinicians should maintain a high index of suspicion for stroke in young patients presenting with acute neurological symptoms following malaria infection, as early recognition and prompt treatment are critical to improving outcomes and preventing long-term neurological disability.

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Summary of case

1	Patient (gender, age)	32 years, Male
2	Final diagnosis	Cerebral malaria with multifocal ischemic stroke
3	Symptoms	dizziness, vomiting, headache, slurred speech, drooling, right-sided weakness and numbness
4	Clinical investigations	Laboratory tests, immunological assays and radiographic tests
5	Medications	Artesunate, aspirin, vitamin B6, vitamin B 12, enoxaparin, levetiracetam
6	Clinical procedure	None
7	Specialty	Neurology