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A Case Presentation of Severe Plasmodium Falciparum Malaria in an 8-Year-Old Boy

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Abstract

Malaria is a life-threatening disease caused by Plasmodium species, with Plasmodium falciparum being the most virulent form. Cerebral malaria is a severe complication that can lead to various neurological manifestations. We report the case of an 8-year-old boy who presented with an unusual symptom of cerebral malaria—spastic gait. Diagnostic workup included a positive malaria rapid diagnostic test (RDT) and peripheral blood smear (PBS), confirming P.falciparum infection. Brain MRI revealed high signal intensities in the bilateral globus pallidus, consistent with cerebral involvement. The patient received intravenous Artesunate and a combination of antimalarial medications including Coartem, Primaquine, Quinine, and Clindamycin. He showed significant clinical improvement following treatment. This case highlights a rare neurological presentation of cerebral malaria and emphasizes the importance of early diagnosis and prompt treatment. Additionally, it underscores the value of neuroimaging, particularly MRI, in confirming cerebral involvement in atypical clinical presentations.

Key Words: Malaria, Neuromalaria, Parasite infection, Plasmodium falciparum.

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1- INTRODUCTION

Malaria is a global parasitic infection with various types of symptoms caused by plasmodium species (1,2). Plasmodium falciparum is accountable for more severe illness and deaths (3). One of the most onerous presentations of plasmodium falciparum is cerebral malaria (CM) or neuromalaria (4, 5).

Neuromalaria attributed to coma followed by seizure which is more prevalent in children than adults (2,6). Due to mortality of CM and its neurological sequels, immediate diagnosis is resuscitative (5).

In this research, we present a child with spastic gait, different manifestation of cerebral malaria, due to plasmodium falciparum.

2- CASE PRESENTATION

An 8-year-old boy from Torbat-e Jam, Iran, with no known past medical history, was admitted with a five-day history of high-grade fever with nocturnal spikes, chills, headache, and periumbilical abdominal pain. His growth, developmental milestones, and level of consciousness prior to admission were completely normal.

On the fifth day of illness, he developed impaired balance and a spastic gait. Notably, the patient had traveled to Pakistan with his father two weeks prior to admission without receiving any chemoprophylaxis.

Upon presentation, his vital signs were stable except for tachycardia (PR: 155 bpm). He was alert (GCS: 15/15), and physical examination revealed splenomegaly. Neurological assessment showed no cranial nerve deficits or signs of meningeal irritation, but motor examination demonstrated generalized rigidity and patellar reflexes +3.

On the third day of hospitalization, he experienced a generalized tonic-clonic seizure progressing to status epilepticus, with an altered level of consciousness (GCS: 6/15). He was transferred to the intensive care unit (ICU). Brain CT scan showed cerebral edema. Cerebrospinal fluid (CSF) sampling was not performed due to lack of parental consent. During his ICU course, the patient developed anemia, thrombocytopenia, hematuria, and elevated lactate dehydrogenase (LDH) levels, for which he received packed red blood cell and platelet transfusions. Initial laboratory investigations are summarized in Table 1.

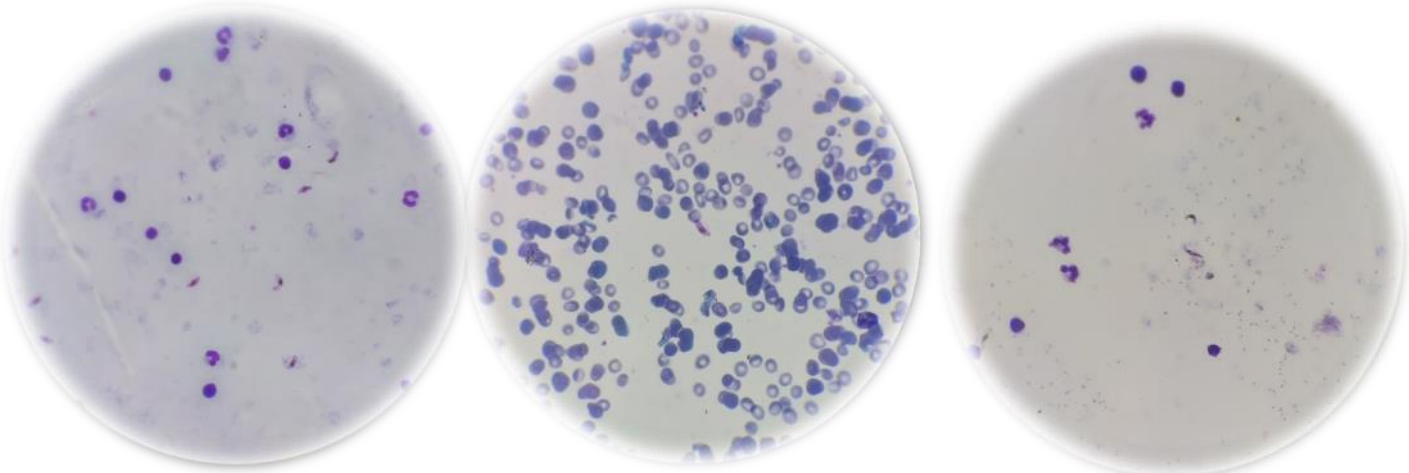
Due to his history and all clinical and laboratory symptoms, we suspected cerebral malaria. We performed a malaria rapid diagnostic test (RDT) along with thick and thin peripheral blood smears (PBS). The RDT, which detects malaria antigens in a person’s blood, was positive and Plasmodium falciparum was seen in PBS (Figure 1).

Following the imaging studies with an MRI and we found high signal intensities in the Globus pallidus, confirming the couse of spastic gait and muscular spasms. Cardiovascular consultation was conducted and found to be completely normal.

He was treated with seven days of continuous intravascular Artesunate and show significant. Thirty hours after initiating treatment, his GCS improved to 11/15 and after two days it was 14/15. The clinical course of the patient is summarized in Figure 2. Due to the persistence of Plasmodium in the PBS, we treated the patient with Coartem in three doses and Primaquine in one dose on the last day of Coartem. Quinine and Clindamycin were added as a third line of therapy due to the severe nature of the disease and treatment failure. additionally, he was treated with Baclofen and Clonazepam as anti-spastic drugs and Levetiracetam as an anti-convulsive medication.

Table-1. The results of laboratory tests in the days of hospitalization.

Parameters	04.02.03	06.02.03	07.02.03	08.02.03	09.02.03	10.02.03	11.02.03	12.02.03	13.02.03
WBC(G/l)	4.20	4.90	6.50	7.10	4.90	4.60	5.70	6.10	6.40
RBC(G/l)	3.84	1.73	2.86	3.79	3.41	3.19	3.10	3.41	3.45
Hb(g/dl)	10.2	4.5	7.8	10.7	9.5	8.8	8.5	9.1	9.3
PLT(G/l)	54	32	90	114	78	68	95	156	206
CRP(mg/l)	91.5		139	116		28		5.9	
Urea(mg/dl)	39	115	151	140		69	36	33	24
Cr(mg/dl)	0.54	1.60	1.70	1.53		0.85	0.61	0.60	0.66
AST(U/L)	33.8	250		151	87	48		37	
ALT(U/L)	36	84		66	53	36		36	
ALP(U/L)	238	165		124					
PT/INR		15/1.26		14/1.13					
PTT		30		28					
Bili(T,D)(mg/dl)				0.7-0.3					
Urine analysis	WBC0-1 RBC0-1 Blood neg	WBC 2 RBC 20 Blood ++	WBC 2 RBC 30 Blood ++++						
Ammonia(µg/dl)		468	466	144	111				
Lactate(mg/dl)		140	89	19	9				
Wright	neg								
2ME	neg								
Blood culture			neg						



A ***B*** ***C***

Figure-1. Pripheral blood smear: ***A:*** Falciparum gametocye in thick smear, ***B:*** Falciparum gametocye in thick smear, ***C:*** Falciparum trophozoite in thick smear.

3- DISCUSSION

Malaria is a parasitic disease transmitted by the female Anopheles mosquito and remains one of the most complex and life-threatening illnesses in children (2). The causative agent, Plasmodium, includes five species: P. vivax, P. falciparum, P. ovale, P. malariae, and P. knowlesi. Among these, P. falciparum is responsible for the most severe and neurological forms of the disease, whereas P. vivax is also associated

with complications such as cerebellar ataxia and seizures (7, 8).

The clinical manifestations of malaria are diverse and may include hypoglycemia, hyponatremia, anemia, thrombocytopenia, acute respiratory distress syndrome (ARDS), central nervous system (CNS) involvement, metabolic acidosis, and multiorgan dysfunction (5). Neurological involvement, often referred to as CM, varies in presentation among children and adolescents. Cerebral malaria is defined as a diffuse encephalopathy characterized by seizures and loss of consciousness, without other identifiable causes, in the presence of P. falciparum on peripheral blood smear (3,8). Although the reasons remain unclear, children are more prone to severe illness; however, pediatric mortality rates tend to be lower compared to adults (7,8). Moreover, neurological and behavioral complications are more predictable in children. Common early presentations include fever, seizures, and coma, while long-term sequelae may involve paresis, ataxia, blindness, deafness, epilepsy, and cognitive deficits following discharge from hospital care (3,6,9).The early recognition of motor symptoms—especially in children with recent travel to endemic areas—should prompt clinicians to consider cerebral malaria even before altered consciousness develops. While these signs are not pathognomonic, they offer a critical diagnostic window before the onset of coma.

The diagnosis can be established through direct blood smear microscopy, and rapid diagnostic tests (RDTs) can aid in early identification of the infection (10, 11).

In our case, neuro-malaria initially presented with fever and constitutional symptoms, followed by the development of spastic gait, loss of consciousness, and seizures during hospitalization. MRI revealed bilateral high signal intensities in the globus pallidus, explaining the patient's scissoring gait. To the best of our knowledge, this is the first reported case of neuro-malaria manifesting in this manner. While the exact pathophysiology remains unclear, basal ganglia involvement is documented. Literature suggests that in pediatric patients, the basal ganglia and thalami are the most frequently affected regions (12).

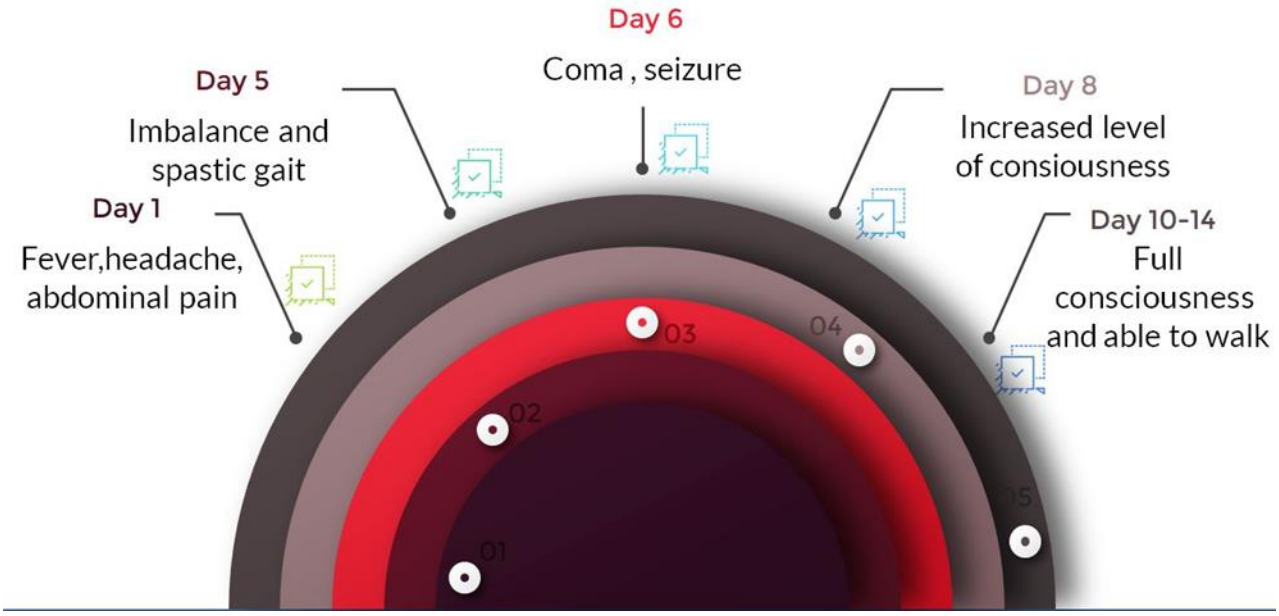


Figure-2. Clinical course of the patient.

In our case, treatment with intravenous artesunate followed by ACT (artemether-lumefantrine), primaquine, and eventually quinine with clindamycin led to significant clinical improvement. Baclofen and clonazepam were also administered to control spasticity, in line with supportive neurological management guidelines (10, 13-15).

Preventive measures are crucial in reducing the incidence of malaria, especially in travelers to endemic areas. Different literatures recommend several strategies, including:

- **Vector Control:** Use of insecticide-treated bed nets (ITNs) and indoor residual spraying (IRS) to reduce mosquito populations.

- **Chemoprophylaxis:** Administration of antimalarial drugs to travelers before, during, and after their stay in endemic areas, tailored to the resistance patterns of the destination.

- **Vaccination:** The RTS,S/AS01 vaccine has shown partial protection against malaria in children and is recommended in areas with moderate to high transmission.

- **Personal Protective Measures:** Wearing long-sleeved clothing, applying insect repellents containing DEET or picaridin, and ensuring accommodations are well-screened or air-conditioned (13).

This case highlights the importance of recognizing early motor abnormalities such as spasticity and imbalance as potential indicators of cerebral involvement in malaria. Prompt diagnosis and aggressive treatment are essential to prevent irreversible neurological damage.

4- CONCLUSION

Cerebral malaria is more common in P.falciparum with different presentations. The most prominent symptoms are seizures and coma. Although we typically expect walking disorders, such as ataxia as post malaria complications, this research revealed a novel first presentation of neuromalaria : spastic or scissor gait.

This result could be a clue for subsequent research to discover its pathophysiology.

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