Case Report

Tropical turmoil: A battle with malaria and severe dengue co-infection.

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Abstract

A 38-year-old male soldier from a tropical forest region in India presented with persistent high-grade fever, jaundice, altered sensorium, and acute kidney injury. Initially diagnosed and managed as severe dengue based on a positive NS1 antigen test, he showed clinical deterioration, prompting further evaluation that confirmed *Plasmodium. falciparum* infection on peripheral smear. The clinical course was complicated by acute liver injury, thrombocytopaenia, and encephalopathy. He was treated with intravenous artesunate, broad-spectrum antibiotics, N-acetylcysteine, and supportive care. The patient made a full recovery with normalisation of hepatic, renal, and neurological functions. This case highlights the importance of considering dual infections in endemic regions, especially when clinical progression is atypical, and emphasises the need for comprehensive diagnostic workup and timely, multidisciplinary management to improve outcomes.

Keywords*: Dengue fever, Plasmodium falciparum, malaria, co-infection, acute liver injury, acute kidney injury, encephalopathy, tropical infections*

Introduction

Co-infections with dengue fever and malaria pose a significant clinical challenge, especially in tropical regions where both diseases are endemic. Malaria, primarily caused by *P. falciparum* and *P. vivax*, and dengue, caused by the dengue virus (DENV), are transmitted by mosquitoes and often present with overlapping symptoms, complicating diagnosis and treatment.

Globally, malaria remains a major public health issue, with an estimated 249 million cases and 608,000 deaths reported in 2022, according to the World Health Organization's World Malaria Report 2023.[1] *P. falciparum* continues to be responsible for the majority of severe cases and mortality, particularly in sub-Saharan Africa, while *P. vivax* maintains a wider geographic distribution, especially in Asia and Latin America. According to the World Malaria Report 2023, India reported approximately 1.3 million malaria cases in 2022, representing a continued declining trend from previous years, with deaths remaining low at around 83 reported fatalities.[1] *P. vivax* accounts for approximately 50% of cases and *P. falciparum* for about 48% of cases in the country.[1] The states of Odisha, Jharkhand, Chhattisgarh, and Madhya Pradesh

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contribute to nearly 70% of India's malaria burden, highlighting the geographic concentration of the disease within specific endemic regions.[3]

Dengue affects over 390 million people globally each year, with approximately 96 million symptomatic cases reported annually according to recent WHO estimates.[2] The case fatality rate for severe dengue can exceed 20% if untreated, but drops to less than 1% with appropriate clinical management.[2] India reported 233,251 dengue cases in 2022 with 303 deaths, showing fluctuating trends compared to previous years, with states like West Bengal, Kerala, and Tamil Nadu contributing significantly to the national burden.[3]

Co-infections with malaria and dengue, though relatively rare, are increasingly recognized in regions such as Southeast Asia, South America, and Africa. Studies report co-infection rates ranging from 1% to 30%, depending on the endemicity of both diseases.[4-6] The clinical course of co-infections may be more severe than mono-infections, with higher risks of complications such as acute liver failure, acute kidney injury, and thrombocytopaenia, leading to increased morbidity and mortality[5-7] especially in areas where both diseases are endemic, such as parts of northeastern India and West Bengal.[8-10]

This case report presents a rare instance of mixed infection with *P. falciparum* malaria and severe dengue fever in a G6PD-deficient patient, highlighting the clinical challenges in diagnosis, treatment, and management.

Case History

A 38-year-old male soldier, with no prior medical comorbidities, presented with a history of persistent high-grade fever of more than 1 week duration . While at work in a densely forested area of Bastar, Chhattisgarh, India, he developed fever. He took leave and travelled back to his village in Ramanagara, Karnataka. He initially visited a peripheral private clinic, where a dengue NS1 antigen card test was performed and returned positive and conservative outpatient management was started; no additional investigation records were available. Despite this, his fever persisted, and he developed progressive jaundice and reduced urine output. One day before admission, he developed altered sensorium and was therefore referred to a higher-level centre with suspected severe dengue for further evaluation and management.

He presented to a tertiary care centre on day 12 of illness; at admission he was febrile, disoriented, and agitated. On examination, he had icterus and hepatosplenomegaly. Laboratory findings indicated multi-organ involvement: serum creatinine 3.58 mg/dL (acute kidney injury) and total bilirubin 25.6 mg/dL with disproportionately elevated direct bilirubin 21.6 mg/dL. On fever work-up, the dengue rapid diagnostic test was negative. However, a malaria rapid card test was positive, and peripheral smear confirmed *P. falciparum* (Figure 1). Other investigations in the fever panel, including scrub typhus IgM, Leptospira IgM, Weil–Felix, and Widal, were negative. Procalcitonin was markedly elevated at 200 ng/mL, suggesting severe bacterial sepsis or a profound inflammatory response, although no primary infectious focus was identified, and blood cultures showed no growth. A dengue NS1 ELISA was performed for confirmation and was positive, confirming recent dengue virus infection and establishing co-infection with falciparum malaria. This likely accounted for the more severe and prolonged clinical course. (Table 1).

Bed side USG showed bilateral pleural effusion and minimal pleural effusion. Altered mental status could reflect cerebral malaria or dengue encephalopathy; given significant hepatic dysfunction, hepatic encephalopathy was also considered.

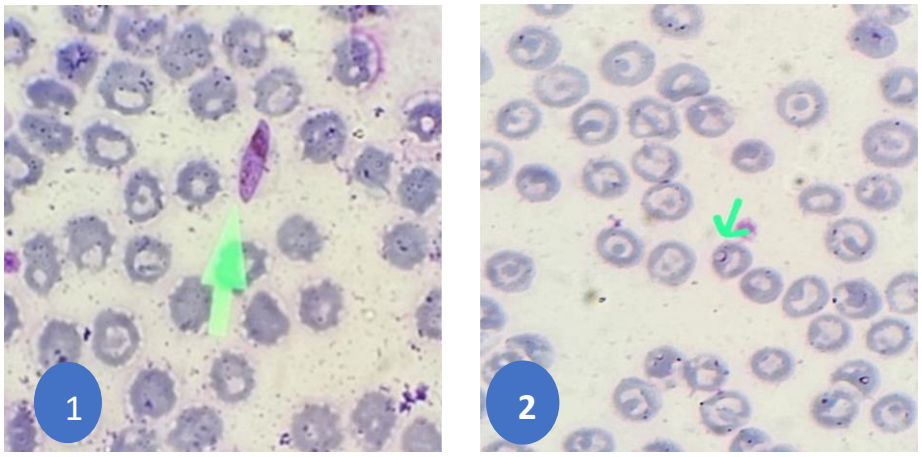

Figure 1: Peripheral blood smear of patient showing 1) *P falciparum* gametocyte and 2) ring forms

Table 1: Investigations during course of hospitalisation

Day of illness	Day12 (On Admission)	Day 15	Day17	Day 18	Day 21	Day 25	Day 28
	5.9.24	**8.9.24**	**10.9.24**	**11.9.24**	**14.9.24**	**18.9.24**	**21.9.24**
Hb	10.4	7.4	7.6	6.7	6.9	6.8	7.1
TLC	6000	6650	6200	7920	7200	6645	6300
Platelet	80000	70000	150000	133000	136000	236000	408000
Bilirubin (Total/Dir)	25/21	13/11	5.16/4.4	7.31/2.85	6.44/2.8	3.28/2.2	2.64/1.12
AST/ALT	99/42	56/33	87/83	44/57	46/57	32/41	30/44
BUN/Creat	142/5.83	119/5.39	71/3.66	34/2.59	18/1.4	17/1.4	12/0.81
Na/K	146/4.94	147/4.4	140/4.58	139/4.65	138/4.25	142/3.92	136/4.87

Hb Haemoglobin **TLC** Total leucocyte count **AST** Aspartate Aminotransferases
ALT Alanine Aminotransferase **Creat** creatinine **NA/K** sodium/potassium **BUN** Blood urea Nitrogen

In view of the severity of presentation and established co-infection, a multimodal therapeutic approach was undertaken. The patient was initiated on intravenous artesunate, the standard of care for severe falciparum malaria as per WHO[11] and NCVBDC India[12] guidelines. Considering the markedly elevated procalcitonin and possibility of superadded bacterial sepsis, intravenous ceftriaxone was added for broad-spectrum antimicrobial coverage. In light of the significant hepatic involvement, N-acetylcysteine (NAC) infusion was started to mitigate ongoing liver injury. Furthermore, oral doxycycline was administered for possible atypical bacterial infections or rickettsial illnesses, which are endemic in the region and may co-exist. Supportive care, including intravenous fluids, electrolyte correction, and renal function monitoring, was aggressively provided to manage dehydration and AKI

During hospitalisation, the patient showed gradual clinical improvement. His neurological status normalised, indicating reversal of encephalopathy. His renal function improved, and serum bilirubin levels declined progressively. IV Artesunate was given for 4 days till reversal

of encephalopathy and the patient was able to tolerate oral administration. He was then given 3 days of oral artemisinin-based combination therapy (ACT) course as per National Centre for Vector Borne Diseases Control (NCVBDC) guidelines, India[12] (artesunate once daily for 3 days plus a single dose of sulfadoxine-pyrimethamine). On day 2 of ACT, he was given a single low-dose of primaquine 0.25 mg /kg for gametocyte clearance, as per WHO Guidelines[11] (instead of the full 0.75 mg /kg dose as per India NCVBDC Guidelines[12]) in view of absence of immediate G6PD test availability. Within 24 hours, the patient developed falling haemoglobin and rising indirect bilirubin consistent with haemolysis; a subsequent qualitative G6PD test was positive. The patient was continued on conservative management (hydration, monitoring, transfusion as indicated) and there was gradual improvement in bilirubin and stabilisation of haemoglobin. The patient was discharged after a period of close monitoring with stable vital signs, normalised sensorium, and improving renal and hepatic parameters.

The timeline of the patient's illness is shown in Figure 2.

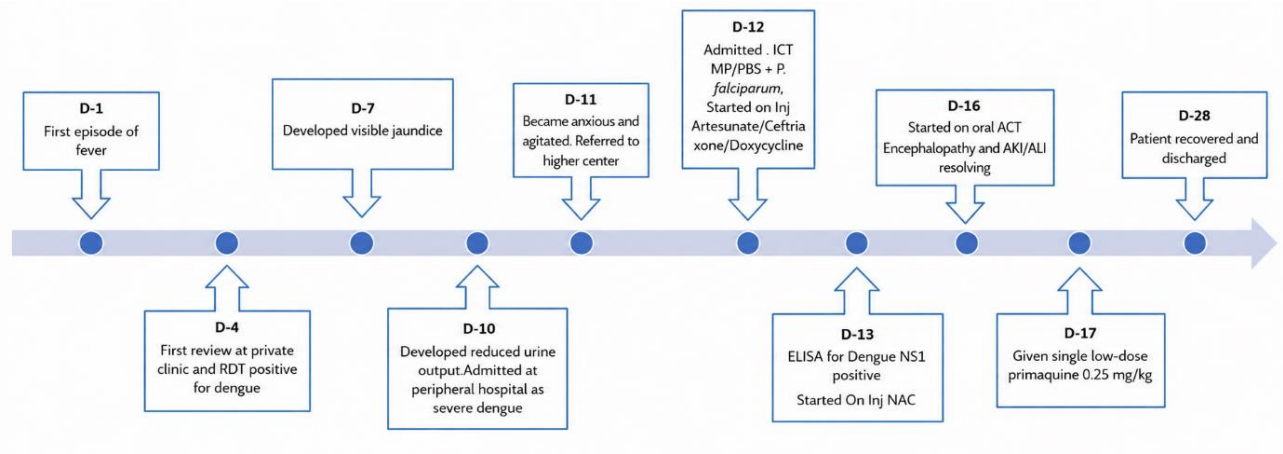

Figure 2: Timeline

Discussion

This case underscores a diagnostic and clinical challenging co-infection with dengue and *P. falciparum* malaria in a previously healthy 38-year-old soldier, complicated by acute liver injury and acute kidney injury. The encephalopathy was likely multifactorial, with differentials including cerebral malaria and dengue encephalopathy, with a possible hepatic contribution. The case highlights how overlapping tropical infections can present with indistinguishable features and require high clinical suspicion, systematic work-up, and tailored, guideline-concordant therapy to achieve favourable outcomes. The clinical course also underscores the importance of assessing G6PD status prior to primaquine; even single low-dose primaquine for gametocyte clearance can precipitate haemolysis in G6PD deficiency when immediate testing is unavailable.

Both dengue and malaria remain major public health burdens in India and other tropical countries. India contributes significantly to the southeast asian malaria burden, with co-infections reported increasingly in high-transmission states (e.g., Odisha, Chhattisgarh, Jharkhand). Co-infections of dengue and malaria, though once considered rare, are now increasingly reported, especially during the post-monsoon season when vector activity peaks. In a study from Delhi by Kaushik et al. (2020)[13], the incidence of dengue-malaria co-infection

was found to be 1.3% among febrile patients, and most had severe outcomes, including multiorgan dysfunction.

The patient initially presented with fever and jaundice and a positive dengue NS1 card at a peripheral clinic which led to a presumptive dengue diagnosis. However, no malaria testing or clinical documentation was available from that visit, which contributed to delayed recognition of co-infection. With persistent high-grade fever, encephalopathy, and worsening hepato-renal dysfunction, urgent re-evaluation was undertaken when he reported to a tertiary centre on day 12, where a complete fever work-up and peripheral smear confirmed *P. falciparum* co-infection. This case underscores the need to test for malaria, even when an alternative diagnosis appears confirmed in endemic settings, as dengue and malaria share overlapping features (fever, myalgia, thrombocytopaenia, hepatosplenomegaly) and their co-occurrence can amplify severity. Both infections can disrupt endothelial integrity and coagulation pathways, leading to capillary leak, organ dysfunction, and rarely, thrombosis. Where immediate G6PD testing is unavailable, even a single low-dose primaquine for gametocyte clearance may precipitate haemolysis in individuals with deficiency.

The complications observed in this patient align with findings from similar case reports. A 2017 case report by Sharma et al.[14] described a fatal case of dengue-malaria co-infection complicated by liver failure and cerebral malaria. Similarly, Singh et al. (2016)[15] reported a case of dual infection presenting with thrombocytopaenia and encephalopathy, highlighting the synergistic damage to hepatic and neurological systems caused by both pathogens. In the present case, acute kidney injury (AKI) was likely multifactorial: volume depletion, malarial sequestration, and acute tubular necrosis all contributed. Acute liver injury (ALI), characterised by a bilirubin level of 25.6 mg/dL, likely resulted from dengue-induced hepatocellular injury and malarial sequestration in hepatic sinusoids. The altered mental status was most consistent with hepatic encephalopathy, although cerebral malaria could not be ruled out definitively due to the presence of *P. falciparum.*

The patient was started on intravenous artesunate, which is the drug of choice for severe *P. falciparum* malaria per WHO guidelines[11] and NCVBDC.[12] Empirical antibiotics, including ceftriaxone and doxycycline, were added due to elevated procalcitonin (200 ng/mL) suggesting possible bacterial sepsis. N-acetylcysteine (NAC) was used to manage ALI, given its potential to reduce oxidative stress and improve liver function, a strategy supported by studies in non-paracetamol-induced liver injury (Nabi et al[16]). These interventions were complemented by intravenous fluids and supportive measures.

In conclusion, this case exemplifies the complex interplay between endemic infections, organ dysfunction, and genetic predispositions that affect treatment outcomes. It contributes to the growing body of literature advocating for comprehensive diagnostic workup in undifferentiated tropical fevers, especially in military or high-risk populations. It also reinforces that in areas of overlapping transmission, dengue and malaria co-infection should be actively sought and not considered mutually exclusive. Lessons from this case should guide improved screening protocols, therapeutic strategies, and preventive measures in endemic settings.

Patient perspective: The patient complained of severe fatigue and worry about the severity of his condition. On discharge, he was relieved and thankful for the multidisciplinary management that resulted in his recovery.

Declaration

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Authors' contributions: Management of case and clinical decision making : AS, AR. Preparing of lab slides and providing images:LN, SP Drafting the article, revising it critically for important intellectual content and final approval of the version to be published: AS,LN,SP,AR

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