SEVERE LEFT VENTRICULAR DYSFUNCTION IN FALCIPARUM MALARIA: A CASE REPORT AND REVIEW OF LITERATURE ON CARDIAC INVOLVEMENT IN MALARIA

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INTRODUCTION

Malaria is an important cause of death in both children and adults, especially in tropical countries like India[1]. Nearly one third cases of malaria are reported from South Asia where majority (65%) of them are from India alone [1,2]. If undiagnosed and untreated promptly, falciparum malaria can be an important cause for high case fatality rate. Cerebral malaria is one of the commonest causes of death in children[1]. Although involvement of myocardium and cardiac failure is rare, few cases have been reported in literature. We report a 10-year old male child diagnosed as falciparum malaria with severe left ventricular dysfunction.

CASE SCENARIO

A 10 year old male child, a resident of Bihar (malaria endemic zone) was referred to our tertiary care hospital (All India Institute of Medical Sciences, New Delhi, India) with complaints of high grade fever associated with chills and rigors for 10 days. He had history of non projectile, non bilious vomiting two days back and two episodes of loose watery stool on the day of admission. There was no history of cough, rapid breathing, haematuria, bleeding from any sites, jaundice, altered sensorium, joint pains, haematuria or oliguria. Two days prior to admission, he had received packed red cell transfusion outside in view of anemia. On admission, child presented with shock, tachypnea, tachycardia and severe pallor but was conscious and responding well to commands. He had hepatomegaly with no palpable spleen. On investigation, he had anemia (Hb-7.5 gm/dl), thrombocytopenia (Platelet count: 10,000), deranged renal parameters (urea/creatinine: 64/1.4), prolonged prothombin time (12/23), no dyselectrolytemia, normal liver function tests (bilirubin -0.5, SGOT-38, SGPT-29, ALP-156). Malarial antigen test was positive for falciparum malaria. Malarial parasite quantitative buffy count (MP-QBC) was positive for P. falciparum with parasite count of 120,000/μL. Child received fluid boluses and ionotropic support (dopamine infusion). Child had clinical features suggestive of heart failure and 2D- echocardiography was done which was suggestive of severe left ventricular dysfunction (ejection fraction: 20%), global hypokinesia with collapsing IVC. Child was treated with IV artesunate and Clindamycin for 7 days. He was also started on milrinone infusion for 3 days. Broad spectrum antimicrobials were also added with the possibility of associated sepsis. He required one packed red cell transfusion and multiple platelet concentrate transfusions. Child also was put on non invasive CPAP for 3 days to reduce the work of breathing. DIC profile was positive. Chest X-ray was normal and cultures were sterile. Serial P. falciparum parasite count was done which showed improvement serially and was zero at day 8 of therapy. Repeat ECHO showed improvement of left ventricular ejection fraction (LVEF-40%).

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Table 1: Values of various laboratory parameters

Parameters	Reference values	Day 1	Day 2	Day 4	Day 8
Hemoglobin (g/dL)	12.5–16.9	7.5	11.3	8	10.3
PLT	110–330	10,000	18,000	15,000	95,000
WBC	3.5–11.0	4900	6000	6500	6000
Creatinine (mg/dL)	<1.3	1.4	0.7	0.6	0.5
Urea (mg/dl)		64	41	24	25
Glucose (mg/dL)	60–126	74	128	102	109
Sodium (mmol/L)	135–145	134	137	131	134
Calcium (mg/dL)	8.5–10.5		7.1	7.1	8.7
Potassium(mmol/L)	3.3–5.0	4	4.5	3.5	4
Chloride (mmol/L)	95–110	116	115	107	112
Phosphate			1.7	2.8	4.7
AST (lUlL)	<37		95	38	52
ALT (lUlL)	<53		41	29	38
Total bilirubin (mg/dL)	0.3–1.5		0.5	0.4	0.5
Alkaline Phosphatase			156	181	202
Creatine phosphokinase(lUlL)	<200			58	
LDH				487	
CPK MB				5	
PT (seconds)	11.0–13.5	12/23	11/14.6		
Transfusion (PRBC-1, PRP-2)		1,2	2	2	No

DISCUSSION

Falciparum malaria is one of the commonest cause for causing severe malaria. Severe malaria is characterized by involvement of one ore more of the following: Severe anemia, impaired consciousness or unarousable coma, metabolic acidosis, jaundice, acute kidney injury, increased lactate, hypoglycemia, respiratory distress with pulmonary edema and with hyperparasitemia.[1] Cardiac failure in malaria is probably due to impaired tissue perfusion resulting in hypoxemia and metabolic acidosis. Cardiac involvement is rarely noticed and left ventricular function is preserved even in severe disease.[1, 3-5] If involved, has a high case fatality rate (Table 2). Table 2 below depicts cases of P. falciparum malaria causing cardiac dysfunction. Case fatality rate of more than 60% has been noticed.

Table 2: Comparision of cases with cardiac involvement and its outcome

References	Cases	Diagnosis	Outcomes
Kumar et al (6)	2	Acute heart failure ventricular fibrillation	Cure Death
Costenaro et al (7)	1	Myocarditis	Death
Wennicke et al (8)	1	Acute heart failure	Death
Wichmann et al (9)	2	Myocarditis Unknown	Death Death
Tripathy et al. (10)	1	Myocarditis	N/A
Mohsen et al (11)	1	Myocarditis	cure
Present case	1	Acute Left Heart failure	Cure

In the past, the antimalarial quinine was thought to be cardiotoxic which caused arrhythmias and hypotension, but evidence available suggests that at therapeutic doses, it has no adverse effects on cardiac conduction or function.[3, 12]

In a recent study done by Kathrin Wennicke et al in vitro and in mice suggest that the Glycosylphosphatidylinositol (GPI) purified from Plasmodium falciparum has a important role in the pathogenesis of malaria and could be responsible for cardiomyocyte apoptosis.[8] Severe and complicated malaria has been associated with myocardial injury, reversible global hypokinesia and diffuse myocardial necrosis diagnosed by raised troponin-T, echocardiography and autopsy findings.[13] Circulating concentrations of cardiac proteins have been demonstrated in patients with both complicated and uncomplicated P. falciparum malaria.[14] In a study in Ghana, where cases were diagnosed clinically and based on positive blood smears, regardless of the severity of the infection, myocardial damage demonstrated by cardiac troponin T (cTnT) was rare.[5] In our case also cardiac enzymes (CPK-MB,

LDH) were normal despite the severity in Left ventricular dysfunction, suggesting the poor sensitivity of cardiac enzymes in malaria induced heart failure. Mocumbi et al studied 47 children and concluded that myocardial damage and dysfunction are rare in severe and complicated Plasmodium falciparum malaria.[15] Tripathy et al. found one case of myocarditis in a series of 1682 severe malaria children.[10] In a case reported from UK by Mohsen et al in a 30-year old female with Worst EF of 35% having myocarditis was discharged subsequently.[11] Franzen et al had reported one patient with global ventricular hypokinesia.[16] In a recent case series of two patients, mortality rate of 50% was demonstrated by Kumar et al[6]. Yacoub et al demonstrated that children with severe malaria and metabolic acidosis have evidence of intravascular volume depletion and associated cardiac dysfunction.[17]

Cardiac involvement in malaria requires early diagnosis and treatment measures to reduce the case fatality rate. Clinical diagnosis added by echocardiography helps in diagnosis. Use of fluid therapy, ionotropes, blood components and Artemisinin Combination Therapy (ACT) helps in a favorable outcome.

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