CASE REPORT

COMPLICATED *PLASMODIUM FALCIPARUM* MALARIA INITIALLY PRESENTING AS MYOCARDITIS

Abdul Latif Khattak, Waqar Ali*, Naseer Ahmad Samore, Muhammad Idris***, Muhammad Nadir Khan**, Waseem Pasha***

Combined Military Hospital Sialkot,*Combined Military Hospital Murree, **Armed Forces Institute of Cardiology, Rawalpindi, ***Department of Pathology, Ayub Medical College, Abbottabad, Pakistan

Despite recent advancements in diagnostic and treatment modalities, malaria is still one of the most prevalent human diseases with high mortality and morbidity. We described a case of 45 years old man with Plasmodium falciparum malaria primarily presenting with myocarditis. The possibility of malaria was subsequently considered when he developed fever followed by signs of cerebral involvement. This happens to be a distinctly unusual presentation and we highlighted various features of this case. Thus in hyperendemic areas complicated *Plasomdium falciparum* malaria may present initially with atypical features and high index of suspicion may lead to prompt early aggressive antimalarial therapy and reduce the complications.

Keywords: Plasomdium falciparum, myocarditis, complications malaria

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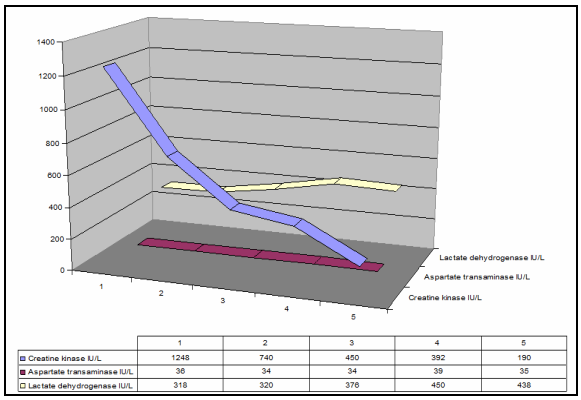
INTRODUCTION

Despite recent advancements in diagnostic and treatment modalities, malaria is still one of the most prevalent human diseases with high mortality and morbidity.[1] About 300–500 million cases occur per year with over one million deaths annually.[2] The incidence of malaria in Pakistan is one case per thousand population.[3,4] According to WHO estimates, over 40 % of the world population is living in malaria endemic areas including Africa, India, Pakistan, Bangladesh and areas of Middle East.[5] We describe a case of *Plasmodium falciparum* malaria primarily presenting with myocarditis. This happens to be a distinctly unusual presentation and we highlighted various features of this case.

CASE REPORT

A 45 years old man was admitted to Coronary Care Unit, Combined Military Hospital, Sialkot with left sided chest pain and palpitations of one hour duration. The pain was non radiating and severe in intensity. There was no vomiting, sweating or shortness of breath. The patient was non-smoker and no other history of addiction. He had no other illness and nor had he previously suffered from hypertension or any other known cardiovascular disease. There was no family history of diabetes mellitus, ischemic heart disease, cerebrovascular accident or hypertension. Patient was of medium built. He was fully conscious, with blood pressure of 140/90 mm/Hg. His pulse was 80 beats per minute and was irregular in rhythm. His temperature at admission was 98.6 ^{o}F. The chest of the patient was clinically clear with normal air entry in both lungs. Heart auscultation revealed normal first and second heart sounds with irregular rhythm. There was no murmur. The neurological inspection did not show any alterations.

The *Plasomdium falciparum* patient's ECG showed atrial ectopia. He was managed with oral Atenolol, Nitoglycerine, Dispirin, Clopidogrel, Alprazolam and intravenous Heparin. The investigations at admission are given in table-1 and 2. On second day of admission he had fever (100.0 $^{\circ}$F) with rigors and chills. He was started on oral chloroquine and blood samples for malarial parasites were requested. Five hours later he developed severe headache, dizziness in addition to his chest pain. On examination he was toxic and had drowsiness (Glasgow Coma Scale 13/15). His temperature rose up to 105.6 $^{\circ}$F and clinically malaria was suspected. Ring forms of Plasmodium falciparum were seen in peripheral blood film (Malarial Parasite Index 0.01%). Nitoglycerine, Dispirin, Clopidogrel and intravenous Heparin were discontinued while dose of oral Atenolol was reduced. Intravenous Quinine infusion supplemented with Potassium chloride was started along with oral doxycycline and intravenous Ranitidine. Cardiac Troponin T was negative (level <0.08 mg/L). His initial Creatinine kinase–Muscle brain (CK-MB) was 96 IU/L and serial cardiac enzymes are graphically depicted in Figue-1. There were no changes in subsequent ECG tracings. Chest radiograph posteroanterior view revealed no abnormality. The fever settled in 24 hours and malarial parasites were not detected in subsequent blood samples. On third day of admission oral Quinine sulphate 600 mg every 8 hours was started for another three days after stopping Quinine infusion. The patient gradually improved and had an uneventful recovery. He was discharged on seventh day of hospitalization.



	1	2	3	4	5
Creatine kinase IU/L	1248	740	450	392	190
Aspartate transaminase IU/L	36	34	34	39	35
Lactate dehydrogenase IU/L	318	320	376	450	438

Figure-1: Serial cardiac enzymes of the case

Table-1: Patients biochemical investigations at admission

Investigations	Patient	Reference Range
Bilirubin	12	upto 17µmol/L
Alanine Transaminase	34	Upto 40 IU/L
Alkaline Phosphatase	118	65–303 IU/L
Urea	5.1	3.3–6.7 mmol/L
Creatinine	80	63–120 µmol/L
Sodium	139	135–148 mmol/L
Potassium	4	3.5–5.0 mmol/L
Plasma Glucose Random	7	3.3–11.1 mmmol/L
Total Cholesterol	4	Desireable <5.2 mmol/L
HDL-Cholesterol	1	Desireable >0.9 mmol/L
LDL-Cholesterol	2.4	Desireable <3.4 mmol/L
Triglyceride	1.2	Desireable <2.3 mmol/L

Table-2: Patients' haematological investigations at admission

Complete Blood Counts		
RBC	5.82	$4.5–5.5\times10^{12}$/L
Haemoglobin	15.5	13.5–17.5 g/dL
Haematocrit	44.6	39–51%
MCV	76.6	77–97 fL
MCH	26.6	27.5–32.5 pg
MCHC	34.8	31.5–34.5 g/dL
WBC	6.9	$4.0–10.0 \times 10^{9}$/L
Neutrophils	68	40–75%
Lymphocytes	28	20–45%
Monocytes	2	2–8%
Eosinophils	2	1–5%
Platelets	94	$150–400\times10^{9}$/L
Coagulation tests		
Prothrombin time		
Patient	16	Seconds
Control	13	Seconds
Activated Partial Thromboplastin Time		
Patient	41	Seconds
Control	33	Seconds
D-Dimers	< 200	< 200 ng/ml

DISCUSSION

Myocarditis is a rare complication of Plasmodium falciparum. Myocarditis was the initial presentation of this case. When the patient developed a febrile spike and started having cerebral symptoms, only then the possibility of malaria was considered which was confirmed on blood film examination. In countries where malaria has been eradicated and only rare sporadic imported cases occur the population is non-immune. In non-immune persons, Plasmodium falciparum parasitaemia may go unrecognized and lead to high morbidity. In such patients cerebral malaria remains the most common clinical presentation and cause of death.[1] This stands in contrast to the fact that myocarditis leading to myocardial failure and cardiac arrhythmias have been rarely reported in course of severe Plasmodium falciparum. In Pakistan malaria is always on top of differential diagnosis of an acute febrile illness because of high index of suspicion due to high prevalence and all around the year transmission of malaria. *Plasomdium falciparum* parasitaemia may be life threatening as reported in Medical Literature from Western hemisphere where malaria is uncommon and rare sporadic imported cases occur.[7] Patients of Plasmodium falciparum malaria have presented with myocarditis. The patients were routinely investigated for other causes initially. *Plasomdium falciparum* was later diagnosed and treatment was started at a late stage and in some death occurred. An important contributory factor in those cases was that in non-immune persons if *Plasomdium falciparum* malaria is not promptly recognized then it has a high mortality rate.[6,8] This contrasts with our case that *Plasomdium falciparum* malaria was diagnosed early and the patient had previous exposure and immunity to Plasomdium falciparum. Furthermore empirical antimalarial therapy was started in this case even before the results of blood film for malaria became available.

The clinical features of myocarditis in *Plasomdium falciparum* malaria are generally those of myocarditis itself. ECG changes are non-specific as in our case and they resolved with treatment. Creatinine kinase was raised in our patient and in one study there is evidence that Creatinine kinase–Muscle brain (CK-MB) is significantly raised in complicated falciparum cases as compared to uncomplicated cases.[9] In our patient Cardiac Troponin T was not elevated. This is the expected finding in accordance with other reports that Cardiac Troponin T was elevated in only 0.6% of patients.[10] Thus in hyperendemic areas complicated *Plasomdium falciparum* malaria may present initially with atypical features and high index of suspicion may lead to prompt early aggressive antimalarial therapy and reduce the complications.

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Address for Correspondence:

Dr Abdul Latif Khattak, Classified Medical Specialist, Combined Military Hospital Murree, Pakistan
Cell: +92-321-5293223
Email: alkhattak@gmail.com