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Research Paper

Myocardial Infarction Associated with Plasmodium Falciparum Malarial Infection

Medical Science

KEYWORDS : plasmodium falciparum, myocardial infarction, infection

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ABSTRACT

Cardiac functions are almost well preserved in malarial infection. We report a rare case with fatal complication of myocardial infarction in patient diagnosed of Plasmodium falciparum infection. The authors therefore suggest that the patient who came with symptoms and signs suggestive of malaria along with any signs of left ventricular failure should be evaluated thoroughly for cardiac complication of malaria. This is even more important for people living in malarial endemic areas.

Introduction

Malaria, a protozoal disease, caused by genus plasmodium, is prevalent in about 100 countries worldwide[1-3] and is a major cause of morbidity and mortality especially in sub-Saharan Africa, Southeast Asia, and Latin-America.[2,6] In India about 1.65 million cases were reported (with 943 deaths) during the years 2003 and 2004.[3] Malaria is an endemic disease in the Northeast part of Rajasthan because of canal irrigation and favorable condition for vector.

Cardiac involvement in malaria has not been studied widely. There have been few reports of experimental and postmortem studies indicating myocardial involvement in malaria. [2–7] We investigated the extent of cardiac involvement in malaria in the clinical situation, by analyzing the occurrence of acute myocardial infarction (AMI) in patients with malaria and comparing it with AMI in non-malarial patients.

Case Report

A 45 year old male presented in the emergency with severe chest pain 2 days back and breathlessness. The patient had history of intermittent fever for 5 days for which he was given symptomatic treatment by local doctor. The patient was non diabetic and no history of hyperlipidemia, obesity or any other cardiovascular dysfunction or complication. On examination the patient was conscious, febrile (oral temperature 38.2°C), respiratory rate of 37 per minute, irregular pulse rate of 114 per minute and blood pressure of 70/50 mm Hg. Systemic examination showed soft abdomen with no neurological deficit but with presence of S3 heart sounds and orthopneia.

The electrocardiogram showed features suggestive of recent anterior wall myocardial infarction with qs pattern in anterior chest leads (Figure 1).

Cardiac tropomin are elevated to 40 times and 2-dimensional echocardiography shows Akinesia of antero-apical segments with severe LV systolic dysfunction (LVEF = 30%) (Figure 2).

Biochemistry report showed blood urea 52 IU/L, serum creatinine 1.9 mg%, SGOT/SGPT 65/72 IU and ESR 65 mm/h. The hematological examination showed haemoglobin 8 gm/dL, total leukocyte count 6.05x109/L, differential leukocyte count with polymorphs 68%, lymphocytes 28%, monocytes 10% and myelocytes 4%. Platelet count was 20,000/dl. Peripheral blood smear examination showed marked agglutination with gametocytes and trophozoites of *Plasmodium falciparum* (Figure 2).

Patient was immediately shifted in ICU, kept on vasopresor support, with dobutamine, LMWH, atrovastatin 80 mg, clopidogril 300 mg loading dose, IV artesunate, cefotaxime and other supportive drugs. Patient was not thrombolysed because of late presentation. On second day of admission, patient shows significant improvement with normalization of blood pressure, platelets were increased to 38000/dl, resolution of chest pain. On third day patient condition was too good with platelet count 90,000/dl and all parental drugs are stopped and on 5th day patient was discharged without investigations regarding coronary intervention because of deranged renal function test and non willingness of patient.

Discussion:

Malaria constitutes one of the most important cause of parasitic burden in tropical countries. In India, during 2005 about 1.8 million cases of malaria were reported and out of which 0.79 million cases were due to *Plasmodium falciparum*[3]. Various complications are associated with malarial infection but only few studies have reported and assessed the cardiac dysfunction in it[4,5]. Jain K *et al* have observed statistical significant difference between patients of MI with malaria in comparison without malaria suggesting the possibility of MI in it[2]. This patient presented with fever and hematological complications of malaria with acute anterior wall MI. This reflects that either the *falciparum* infection was alone responsible for myocardial damage or less likely have acted as an aggravating factor to worsen the previously compromised cardiovascular function. Both of these conditions indicate the possible association of *falciparum* infection with MI. It is unlikely that malarial infection was an incidental finding of fever and MI in young person with no risk factor for CAS.

The possible pathophysiology is related to blockage of vessels due to cytoadherance of erythrocytes to capillary endothelium mediated by strain specific erythrocyte membrane adhesive protein[1]. In addition, the sequestration of red blood cells may also

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interfere with microcirculatory flow of heart[1].

Thus to conclude the case highlights that MI is rare but important severe complication of *Plasmodium falciparum* infection. The authors suggest that every case of fever especially if associated with cardiovascular complication should be immediately investigated for malaria so as to prevent this complication. Thus early investigation and prompt treatment of malaria is even more important in endemic areas.

Figure 1 – ECG showing Recent Anterior wall Myocardial Infarction

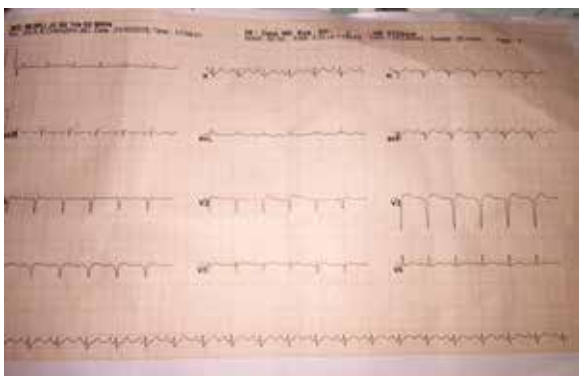
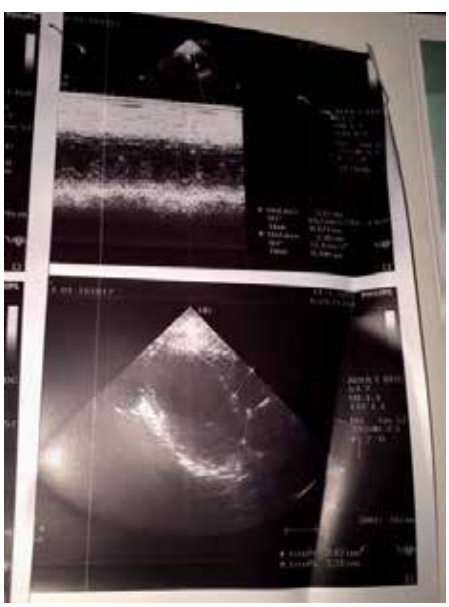


Figure 2- 2D Echo Image showing Anterior wall Akinesia



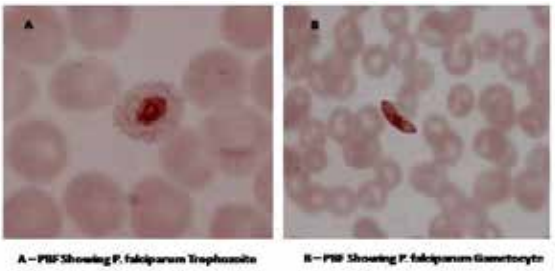


Figure 3 – PBF showing trophozoite and gametocytes

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