In the treatment of Wilson's disease the newer drugs like Trientine (Syprine 500mg BD half an hour before or 2 hrs after meals) is less toxic chelator can be used in addition to Zinc .Both drugs should not be taken together because trientine will chelate zinc forming ineffective complexes.

Our patient is receiving only Zinc acetate (Galzin). Penicillamine and trientine both can cause worsening of neurologic manifestations. For initial neurologic therapy Tetrathiomolybdate is emerging as the drug of choice because of its rapid control of free copper preserving neurologic function and low toxicity & is being studied in clinical trials. Polyarthritis can be managed with symptomatic treatment & physiotherapy.

CONCLUSION:

The Wilson's disease can present in various forms. Commonly hepatic and neuropsychiatric features are seen but its rare manifestation like poloyarthritis should be remembered in a given case particularly when etiology of arthritis appears to be obscure, more so when it is associated with hepatosplenomegaly. A high index of suspicion is necessary, otherwise a treatable condition can be missed and irreversible damage to liver and brain will occur.

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A rare association between Cardiac Involvement and Plasmodium Falciparum Malarial Infection.

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Abstract:

Falciparum malaria is known to cause multi organ dysfunction. Cardiac function is not affected in malarial infection .We report a case of association between cardiac involvement and *Plasmodium Falciparum* Malarial Infection. This association is rare and author suggest that every case of fever especially in malarial endemic area associated with cardiovascular risk should be thoroughly investigated and treated to prevent complications.

Keywords: Myocardial Infarction, Plasmodium falciparum malaria

Introduction:

Malaria caused by *Plasmodium falciparum* is a major health burden globally, causing an estimated 225 million illness episodes and around 800,000 deaths per year.[1] Cerebral malaria, renal impairment are some of the major complications of *Plasmodium falciparum* malarial infection in comparison to other species. Although Cardiac function is not affected in malarial infection but rarely studies have been done to assess the myocardial pathology associated with this infection. Until now very few case had been reported of *Plasmodium falciparum* Malarial Infection associated with Myocardial Infarction.[2,3] The present case is a rare association between cardiac involvement and Plasmodium falciparum Malarial Infection. A Case of Cardiac complication after experimental human malaria infection has also been reported in past[4] The case therefore emphasizes that every case of fever in malarial endemic area associated with

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cardiovascular risk should be thoroughly investigated and treated to prevent complications.

Case Report:

A 40 year old female presented with fever with rigors of two days duration & Precordial pain radiating to left infra axillary area and left back. On examination the patient was conscious, febrile (101.5°F),respiratory rate of 24 per minute, pulse rate of114 per minute and blood pressure of 100/60 mmHg. Systemic examination showed soft abdomen with no spleenomegaly. His cardiovascular & Respiratory system examination did not show any abnormality. There was no focal neurological deficit. The haematological and biochemical investigations revealed haemoglobin 8.2 gm/dl, total leukocyte count 6850/mm^3, differential leukocyte count with polymorphs88%, lymphocytes 10%, monocytes 1%, eosinophil 1%, Platelet count was 255000/mm^3,blood urea 33.7 mg/dl, serum creatinine 0.73mg/dl, serum Homocysteine was 8.7 µmol/L. She was G6PD non deficient. peripheral smear showed gametocyte of *Plasmodium falciparum and* rapid malarial antigen test was also positive for *Plasmodium falciparum.* Her ECG Showed RBBB.She was treated with injectable artesunate 2.4 mg/kg BD on day 1 and 2.4 mg/kg for next 5 days as per WHO guide lines& intravenous fluids. Chest pain left side radiating to back was persistent for next 36 hours, ECG after 12 hours revealed s/o RBBB with VPc's , ventricular bigemini, runs of VPc's. Troponin T was positive with normal CPK-MB 21 U/L as it was done two days after chest pain. Her lipid profile was normal.. She received clopidogrel (300 mg stat), aspirin (300 mg stat),rosuvastatin (20 mg OD), Enoxaparin 60 mg subcutaneous BD, intravenous nitroglycerine and metaprolol 12.5mg BD. 3rd day ECG showed ST coving in anteroseptal leads. She was diagnosed as *Plasmodium falciparum* malarial fever with Non-Q Anteroseptal wall myocardial infarction with RBBB and was treated accordingly. Her 2 D Echo showed moderate LVEF- 52% and no RWMA. She was discharged on 10th day Coronary angiography done after 25 days revealed normal coronaries.

Discussion:

Malaria, a protozoal disease, caused by genus plasmodium, is prevalent in about 100 countries worldwide. In India about 1.65 million cases were reported (with 943 deaths) during the years 2003 and 2004.[5] Malarial infection is associated with various complications and 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] The present case shows a rare association between acute coronary syndrome – Non Q-MI and Plasmodium Falciparum Malarial infection.Either *falciparum* infection was alone responsible or acted as an aggravating factor for myocardial damage.The possible pathophysiology which is responsible for this rare association is blockage of vessels due to cytoadherance of erythrocytes to capillary endothelium mediated by strain specific erythrocyte membrane adhesive protein and or sequestration of red blood cells interfering with microcirculation of heart.[6] The vasoconstriction due to raised catacholamine in malaria may further aggravate the myocardial damage.

In conclusion, association between cardiac involvement and Plasmodium Falciparum Malarial Infection is rare, still MI should be considered as an important but rare clinical complication of malaria. The author suggest that every case of fever especially in malarial endemic area associated with cardiovascular risk should be thoroughly investigated and treated to prevent complications.

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