IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 6, Issue 4 (May.- Jun. 2013), PP 53-55
www.iosrjournals.org

Myocarditis In Course Of P.Falciparum And P.Vivax Infection:Case Report And Review Of Cardiac Complications In Malaria

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Abstract: *Plasmodium infection is usually complicated by cerebral malaria, hemolysis, acute kidney injury and respiratory distress. Myocardial involvement is a rare complication of plasmodium infection. We have reported a case of plasmodium infection (p. falciparum and p. vivax) complicated by myocarditis.*

I. Introduction

Malaria still remains one of the major health problems especially in developing countries like India. If not recognized promptly, *P. falciparum* malaria can in fact retain a high case-fatality rate. Cerebral malaria remains the most common clinical presentation and cause of death. In contrast, myocardial failure and cardiac arrhythmias have been rarely reported in course of severe malaria despite the well-known sequestration of parasitized erythrocytes in the myocardial vessels and the potential cardiac toxicity of antimalarial drugs. However, myocardial involvement has been observed in presented case of severe malaria due to infection with two species of plasmodia falciparum and vivax.

II. Case Report

A 42 year lady was admitted to ICCU with complaint of breathlessness, chest pain, headache, sweating since 3 days. She was also having complaint of high grade fever with chills and rigor since last 7 days. On admission she was having complaint of palpitation, nausea , vomiting. She was conscious and oriented. She was not having any history of cardiovascular or any systemic illness.

Clinical examination revealed tachycardia , hypotension(systolic 80 mmHg), tachypnea, jaundice. Her peripheral oxygen saturation was 90% on room air.

On systemic examination signs of pulmonary congestion and tachycardia on auscultation were present with normal Neurological examination.

Laboratory investigation:

Haemoglobin	14.1	Troponin I	4.09 ng/ml
Total count	6510	CPK-MB	64 IU/L
Platelet count	24400	HIV	Non-Reactive
Peripheral smear	Ring form of P. Falciparum and trophozoites, schizontes of P. Vivax PI 2%	HCV	Non-Reactive
S. Crt	3.40	MYCOPLASMA Ab	Negative
Urea	128	S. Leptosira(MAT)	Negative
SGPT	59	ANA	Negative
T. Bilirubin	4.2	S. TSH	3.2
PT(INR)	14.9(1.09)	Sputum for AFB	Negative
Aptt	22	ESR	42

Chest x-ray suggestive of pulmonary congestion. ECG was suggestive of nonspecific ST-T changes, ventricular bigeminy. Echocardiography revealed generalised hypokinesia, Mild MR and Mild TR with L V dysfunction was noted.

The patient was treated with inotropic support and non-invasive ventilatory support was given. Patient was treated with artesunate and clindamycin as per NVBDCP guidelines. After 48 hours of admission repeat peripheral smear negative for plasmodial parasite.

After seven days of admission patient was improved haemodynamically and inotropic support was gradually withdrawn and follow up investigations were performed found to be normal.

III. Discussion

In presented case patient initially admitted to intensive coronary care unit as cardiac condition, pt had not suffered previously from any cardiovascular disease. Patient's peripheral smear was showing plasmodium falciparum and vivax infection. Patient was treated with artesunate along with clindamycin parentarally according to standard national guidelines.

After successful treatment patient improved hemodynamicaliy, inotropic support was withdrawn gradually. On 10th day of admission patient's follow up echocardiography, ECG and cardiac biomarkers were performed. and ECG was showing sinus rhythm , cardiac biomarkers were within normal range.

Patient was suffered from myocarditis and other possible etiologic agent for myocarditis were excluded .Patient was improved after treating falciparum and vivax infection.

According to the WHO criteria, severe *P. falciparum* malaria in adults is defined by one or more of the following: impaired consciousness with unarousable coma, jaundice, progressive renal impairment, Metabolic acidosis, hypoglycaemia, respiratory distress. The pathogenetic mechanism is believed to consist mainly of impaired tissue perfusion resulting in hypoxemia and metabolic acidosis.

Primary cardiac involvement is thought to be rare and myocardial function preserved even in severe disease[2-5,6]. Haemodynamic changes have been found to be compatible with systemic and pulmonary vasodilation, and increased pulmonary vascular permeability to be the cause of pulmonary oedema[6].

In complicated *P. falciparum* malaria cases compared to uncomplicated case[09], of a significant increase in the level of N-terminal pro brain natriuretic peptide (NT pro BNP, a sensitive marker of impaired left ventricular function), myoglobin and creatine kinase muscle-brain(CK-MB) (both markers of myocardial injury and necrosis) was observed even in patients who did not display significant ECG abnormalities. In one study cardiac biomarkers were not that much significantly elevated[7] but Electrocardiography revealed nonspecific abnormalities suggesting that the electrophysiology of cardiac myocites can be altered before myocytolysis occurs. Therefore, re-evaluation of current perspectives on the pathophysiology of myocardial dysfunction in course of severe malaria appears to be necessary.

Toxic effects due to cytokines such as the tumor necrosis factor (TNF), have been claimed to play an important role[10-12]. Plasmodial glycosyl phosphatidyl inositol (GPI)—either free or linked to surface antigens exerts direct effect on cardiac myocytes[13]. More recently, such an effect was determined as an up regulation of apoptotic genes and myocardial damage marker (NT pro BNP)[08], suggesting that GPI might induce myocyte apoptosis and therefore be one cause of malaria myocarditis.

In summary, at the present state of knowledge myocardial damage appears to retain a multifactorial pathogenesis, being probably the result of mechanical (microcirculatory obstruction), metabolic (systemic acidosis and related tissue hypoxygenation), and humoral mechanisms. However, cardiac side effects related to therapy should also be considered. Quinine may evoke arrhythmias, angina, and hypotension, potentially causing circulatory failure and/or cardiac arrest. However, these effects are rare and generally occur when the drug is injected rapidly: noticeably, cardiovascular collapse is generally an effect of acute toxicity manifesting when infusion is initiated. None of these side effects could be ascertained in our case.

To date, there is no direct evidence for significant cardiovascular effects of artesunate noted[16] although a case of limited myocardial necrosis occurring just after completion of antimalarial treatment with artemether/lumefantrine was recently reported in an experimentally infected healthy volunteer[14-15], raising an issue of differential diagnosis between acute coronary syndrome and myocarditis[16]. Our experience suggests that in course of severe malaria the frequency of primary cardiac complications may be underestimated, especially in adult patients with cardiovascular risk factors (i.e. ,obesity, smoking, diabetes, hypertension, advanced age), but also in case of unknown or silent underlying cardiomyopathy.

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