ISSN NO. 2320-5407

Journal homepage: http://www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH

RESEARCH ARTICLE

Unusual presentation of Plasmodium vivax malaria mimicking acute myocardial infarction – a case report

Dr. Indraneel Dasgupta, Dr. Jayita Das, Dr. Govardhan Desai Reddy, Dr. Indranil Mitra
Department of Emergency Medicine, Peerless Hospitex Hospital and Research Centre Limited, Kolkata, India.

Manuscript Info

Manuscript History:

Received: 12 November 2014
Final Accepted: 22 December 2014
Published Online: January 2015

Key words:

Myocardial infarction; *Plasmodium vivax* Malaria; ECG.

Corresponding Author

Dr. Indraneel Dasgupta

Abstract

Malaria caused by *Plasmodium vivax* is usually an uncomplicated disease that runs a benign course. We came across an unusual presentation of a patient with *Plasmodium vivax* malaria mimicking acute myocardial infarction in the Emergency Department of Peerless Hospital and B C Roy Research Centre. Though conduction abnormalities and non-specific ST-T segment changes are recognized manifestations of malaria, especially *P. falciparum* type, ST elevation mimicking MI has never been reported in *Plasmodium vivax* Malaria. In our knowledge and subsequent literature search this is probably the first case report of this unusual presentation.

INTRODUCTION

Malaria has been a leading public health problem despite several malaria control strategies. According to World Health Organization (December 2013) approximately 207 million cases of malaria and 627,000 deaths were reported in 2012 **[1],** though the actual figures are much higher. The infection is common in tropical and sub-tropical areas and more than 50% of malaria cases are caused by *P. vivax,* **[2],** especially in South East Asia and India. Once considered benign, the number of complicated *P. vivax* malaria is increasing over the past few years. Usual complications reported from various parts of the world include common gastrointestinal symptoms, **[2-5],** severe anaemia, thrombocytopenia, pulmonary complications, and renal failure. **[6-8].** However, cardiac complications in vivax malaria have been seldom been reported. We report a 55 year old male with *P. vivax* malaria who presented with ST elevation and chest pain mimicking acute myocardial infarction. In our knowledge and subsequent literature search this is probably the first case report of this unusual presentation.

Case History:

We report the case of a 55 year old previously healthy male who presented to our Emergency Department (ED) with a history of severe retrosternal chest pain and slurring of speech 3 hours prior to his arrival. He was taken to a local hospital where ECG showed features of anterolateral STEMI and he was transferred to our hospital for further management. Patient had a recent history of *P. vivax* malaria for which he completed a course of Chloroquine 7 days ago and had been afebrile since then. He also had a similar episode of chest pain 3 days ago.

At the time of arrival to ED, patient was afebrile, conscious, oriented but restless with slurred speech and increased response time. Other findings included a SpO2 of 97 % in room air, BP 94/50 mm of Hg, heart rate of 70/minute and random blood sugar 120mg /dl. The rest of his systemic examination was normal, and there were no remarkable findings. However, Electrocardiogram revealed > 2 mm ST segment elevation in leads V2 to V6 suggestive of acute anterolateral myocardial infarction (AMI) **(Figure I),** but bedside Troponin card test was negative.

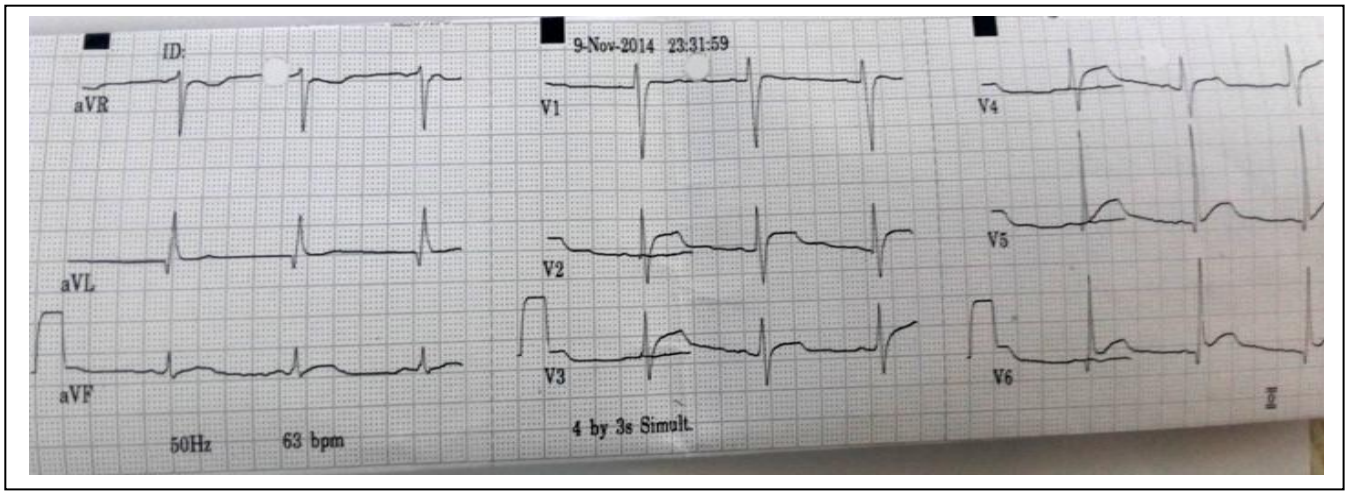

Figure I

A clinical diagnosis of AMI was made in the ED and the patient was promptly seen by a cardiologist. Bedside echocardiogram did not show any regional wall motion abnormality or LV dysfunction (LVEF: 64%). So, the decision of primary angioplasty was withheld as the patient was pain free by that time and the repeat ECG showed resolution of the initial ST elevations **(Figure II)**. Oral statin, aspirin and clopidogrel were commenced and patient was admitted in ICCU for close monitoring.

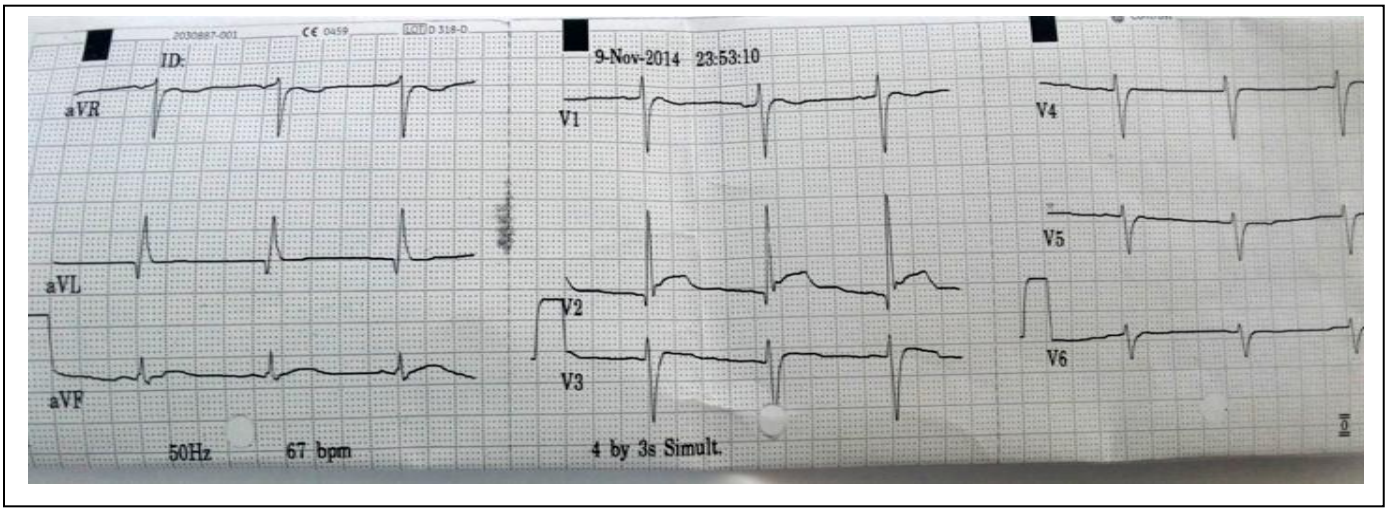

Figure II

The investigations sent from ED revealed the following results: White blood cell count (5,880 cells/ cu mm), Urea (56 mg/dl) and Serum Creatinine (1.23mg/dl), and electrolytes (Na+: 135 mg/dl; K+: 3.6 mmol/L) were normal. Patient was slightly anaemic (Hb: 10.7 g/dl) and thrombocytopenic (platelet 30,000 cells /cu mm). His cardiac biomarkers were normal (CK, 25 U/L; CKMB 5 U/L; Trop T: 10.39 ng/L). Though Dengue (ELISA) NS1, IgM and IgG were non reactive, blood smear revealed the presence of trophozoites of *P. vivax* and antigen test was positive for *P. vivax*. Of note, tests for malaria parasite detection from the ED is not routinely carried out in chest pain patients, but a recent history of malaria might have prompted this. Other investigation revealed high serum CRP of 99.4mg/L and ESR of 62mm/ 1st hr. CT scan of brain was normal.

As no definite cardiac problem was identified after 24 hours of close monitoring in ICCU, the patient was shifted to ward under general medicine where he was treated with antimalarial and discharged after uneventful recovery. His ECG changes fully resolved after 3 days **(Figure III)**.

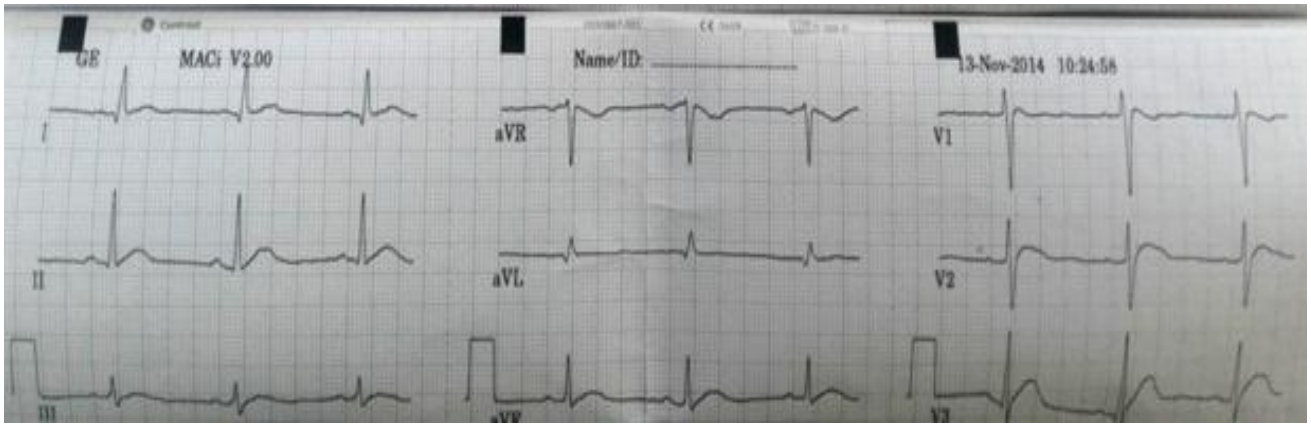

Figure III

Discussion:

Cardiac involvement such as conduction abnormalities and non-specific ST-T segment changes is a recognized manifestation of malaria **[9].** Gunther *et al* (2003) **[10]** studied 161 cases of *P. falciparum* malaria, of which 14.3% of patients had either non-specific T-segment changes or abnormal conduction delay but the cardiac biomarkers were rarely elevated. Though the exact mechanism of cardiac involvement in malaria remains unknown, previous case reports and pathophysiological studies **[11, 12]** have proposed several hypotheses.

Transient myocardial ischemia manifested by ST segment changes may not cause elevation in the cardiac biomarkers. This might be due to coronary microvascular dysfunction characterized by diffuse distal epicardial vessel spasm and microvascular spasm **[13]** giving rise to angina pectoris. Some previous autopsy studies **[11]** suggest myocardial ischemia can be caused by blockage of capillaries by parasites and parasitized red blood cells, known as cytoadherence, where red blood cells adhere to the walls of capillaries and are sequestered to the brain and heart. However, cytoadherence is typically noted by *P. falciparum* malaria and not seen in *P. vivax* malaria due to low parasite density as per the previous studies. Our patient presented with slurred speech and increased response time, which can be correlated with the phenomenon of cytoadherence.

Our patient had severe thrombocytopenia which could cause altered thrombostasis and microvascular thrombosis contributing to micro vascular obstruction **[7].** This could have lead to transient coronary and cerebral ischemic symptoms in our case.

Cytokine-mediated endothelial activation has been found to be associated with complicated *P. vivax* malaria. Some cytokines and *vivax*-specific "malaria toxins" are believed to cause greater organ-specific inflammation, increased alveolar-capillary membrane permeability, capillary leakage, and leukocyte aggregation **[14, 15].** In a study from Pakistan, the authors observed that tumor necrosis factor alpha, interleukin-10, intercellular adhesion molecule-1, and vascular cell adhesion molecule-1 were released in complicated P. *vivax* malaria **[16].** These toxic effects of cytokines, such as tumor necrosis factor alpha, interleukin-10, or other unknown factors might have played a role in causing transient myocardial ischemia in our patient.

CONCLUSION

The clinical presentation of malaria is non-specific and is often misdiagnosed [2]. In the present case, we found evidence of ST elevation mimicking AMI, which improved following symptomatic treatment. Unfortunately, the mechanism of cardiac complications associated with malaria is not well understood and hence there is lack of evidence regarding management protocol. This case report illustrates that diagnosis of malaria based on clinical features alone is difficult and requires a high index of suspicion among clinicians. The key to diagnosing malaria is

to elicit detailed history and request malaria test in anyone who feels unwell or had a recent history of malaria, as in our case.

Physicians should be aware of this rare and unique presentation of *P. vivax* and the fact that aggressive management of AMI such as thrombolytic therapy should only be undertaken only after extensive evaluation of the patient. Further research on the role and effects of mediators and methods to prevent these complications is needed.

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