Afebrile malaria patient with multisystem involvement and Hepatitis B infection: A case report

Rabindra Ghimire, Kaushal Raj Pandey, Prabhat Adhikari, Ashna Pokhrel, Mora Maximo and Mirela Sam

ABSTRACT

Malaria is caused by obligate intraerythrocytic protozoa of the genus Plasmodium. Humans can be infected with one (or more) of the following five species: P. falciparum, P. vivax, P. ovale, and P. malariae and P. knowlesi. Malaria typically produces a string of recurrent attacks, or paroxysms, each of which has three stages; chill, followed by fever, and then sweating. Although malaria without fever is rare, we present a complicated case of P. ovale malaria without fever associated with Hepatitis B virus infection, pre-excitation pattern ECG and secondary adrenal insufficiency in a young African American adult male who had travelled to Africa 9 months prior to clinical presentation. Our patient did not have any features to characterize severe malaria and the parasitemia was <5%.

KEYWORDS: Malaria, Plasmodium ovale, Preexcitation, Hepatitis B

Malaria is caused by obligate intra-erythrocytic protozoa of the genus Plasmodium. Humans can be infected with one (or more) of the following five species: P. falciparum, P. vivax, P. ovale, and P. malariae and P. knowlesi. Plasmodia are transmitted by the bite of an infected female Anopheles mosquito and these patients commonly present with fever, headache, fatigue and musculoskeletal symptoms.

Diagnosis is made by demonstration of the parasite in peripheral blood smear. The thick and thin smears are prepared for identification of malarial parasite and genotype respectively. Rapid diagnosis of malaria can be done by fluorescence microscopy with light microscope and interference filter or by polymerase chain reaction.

We report a complicated case of P. ovale malaria without fever associated with Hepatitis B virus infection, pre-excitation (WPW pattern), and secondary adrenal insufficiency.

Case Report:

A 23 year old African American man presented to the emergency department with headache and dizziness for one week. He had 8/10 throbbing headaches associated with dizziness, nausea and ringing sensation in the ears and also complained of sweating but denied any fever. He had loose, watery bowel movements 3 times a day for a few days and had vomited once 5 days ago. He denied any past medical history or family history. He was a chronic smoker and smoked 1PPD for 8 years and denied alcohol or drug use. He had travelled to Africa 9 months before presentation and had stayed in Senegal for 1 month though he did not have any illnesses during or after returning from Africa.

On examination: T: 97.6, HR: 115/min, BP: 105/50, no orthostasis, SPO$_2$: 100% in room air and RR: 18/min. Head, neck and throat examinations were normal and respiratory and cardiovascular system examinations were unremarkable except for tachycardia. Abdominal examination revealed no organomegaly and his CNS examination was unremarkable.

Laboratory examination revealed: WBC: 6.4, Hb: 14.4 and Hct: 41.3, Platelets: 43, L: 7.4, M: 9.3, B: 0.1. His serum chemistry was normal except for a creatinine of 1.3 (BUN 14) and albumin of 2.6 (total protein 5.7). A pre-excitation (WPW Pattern) was seen on ECG and head CT and Chest X-ray were normal.

He was admitted to the telemetry unit to monitor for arrhythmia. Peripheral blood smear (PBS) was sent because of thrombocytopenia and mild renal failure and revealed malarial parasites later identified as P. ovale (Pic. 1 and 2).
Malaria continues to be a major health problem worldwide. In 2007 the CDC received reports of 1,505 cases of malaria from New York with all but one of these cases being acquired among person in the United States. 326 cases were reported outside of the United States.

Malaria without fever has been reported in cases of Plasmodium falciparum malaria in non-immune people. Hepatitis B infection associated with asymptomatic malaria has been reported in the Brazilian Amazon. This study was done in P. falciparum and P. vivax infected person with HBV co-infection though not in the P. ovale group. HBV infection leads to increased IFN-gamma levels which are important for plasmodium clearance in the liver, in addition to its early importance for malarial clinical immunity. High levels of IFN gamma, IL6 and TNF alpha are detectable in the blood of malaria patients and in the spleen and liver in the rodents' model of malaria. These inflammatory cytokines are known to suppress HBV replication in HBV transgenic mice. This might explain the low levels of HBV viremia in our patient although human studies are required to confirm this finding.

Cardiac complications after malaria have rarely been reported. In our patient pre-excitation on ECG disappeared after starting antimalarial treatment. Whether WPW pattern and its subsequent disappearance was incidental or caused by malarial infection that improved with treatment could not be determined. Lengthening of the QTc and severe cardiac arrhythmia has been observed, particularly after treatment with halofantrine for chloroquine resistant Plasmodium falciparum malaria. Post-infectious myocarditis can be associated with cardiac events especially in combination with viral infections. A case of likely acute coronary syndrome and possible myocarditis was reported after experimental human malaria infection. To date, except for cardiac arrhythmias that developed after treatment with halofantrine and quinolines, no other arrhythmias has been reported in patients with malaria before treatment.
Transient thrombocytopenia is very common in uncomplicated malaria in semi-immune adults. A person with a platelet count <150 × 10^9/l is 4 times more likely to have asymptomatic malarial infection than one with a count ≥150 × 10^9/l. In an observational study among 131 patients, patients with involvement of more than one organ system was found to have a lower mean platelet count compared to those with single organ involvement.

Conclusions:
Our case highlights the need for further studies to understand the multi-organ involvement in patients without severe malaria as well as early recognition of potential complications to prevent mortality and morbidity in this subgroup of patients.

Acknowledgements
We are thankful to our pathologist Maximo Mora, MD for providing the picture of the malarial parasites from our patient.

Competing Interests
None declared

Author Details
Rabindra Ghimire, MD: Resident, Internal Medicine PGY3, Interfaith Medical Center, Brooklyn, NY. Kaushal Raj Pandey, MD: Resident, Internal Medicine PGY3, Interfaith Medical Center, Brooklyn, NY. Prabhat Adhikari, MD: Resident, Internal Medicine PGY3, Interfaith Medical Center, Brooklyn, NY. Ashna Pokhrel, MBBS: Resident, Internal Medicine, Interfaith Medical Center, Brooklyn, NY. Mora Maximo, MD: Pathologist, Infectious Disease division, Interfaith Medical Center, Brooklyn, NY. Mirela Sam, MD: Chief, Infectious Disease division, Interfaith Medical Center, Brooklyn, NY.

CORRESPONDENCE: Rabindra Ghimire, MD
Department of Medicine, 1545 Atlantic Avenue, Brooklyn, NY-11213
Email: drrabindraghimire@gmail.com

REFERENCES