Case Report

Impending hepatic failure due to Plasmodium vivax Malaria: a case report

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Abstract

Plasmodium falciparum is traditionally known for complicated malaria but Plasmodium vivax is now becoming a major culprit in many cases. We are presenting a case report of 16 year male patient presented with high grade fever with chills. He has abnormal liver function tests and coagulation profile. Peripheral blood film showed presence of Plasmodium vivax trophozite.

Key Words: Impending hepatic Failure, Plasmodium Vivax, Quinine

Introduction

Plasmodium falciparum is a known culprit of complicated malaria since long time. Now a days more and more cases of vivax malaria with abnormal features are increasing [1].

Plasmodium vivax malaria with cerebral manifestations, renal failure, circulatory collapse, severe anemia, hemoglobinuria, abnormal bleeding, acute respiratory distress syndrome, jaundice, disseminated intravascular coagulation, gastrointestinal complications and pulmonany edema have been reported [2, 3].

Case Report

A 16 year old male patient presented with high grade fever associated with chills and rigors for 4 days, yellowish discoloration of eyes for two days and two episodes of vomiting for one day.

There was no history of previous jaundice, blood transfusion, or any drug intake. He has no history of alcohol addiction or I. V. drug abuse. He had no previous history of any liver disease. He was anxious and ill looking.

General examination revealed fever, icterus and sinus tachycardia without other sign of liver failure.

Systemic examination revealed tender hepatomegaly and spenomegaly with other system in normal limits. Investigations revealed; Total Serum Bilirubin was 3.82 mg/dl with direct Bilirubin 3.18 mg/dl, SGOT-9890 IU/L, SGPT-9161 IU/L, Alkaline phoshpatase 380 IU/L , Serum albumin 4.7 mg/dl. Prothrombin time was 24.3sec with INR of 2.07.

All serological markers for hepatitis viruses were negative.

Peripheral blood smear showed plasmodium vivax only and malarial antigen test for Plasmodim falciparum was negative.

USG abdomen showed mild hepatomegaly with hepatitis with mild ascites. Patient started improving with intravenous Quinine.

There was marked improvement in liver parameters after 7 days; SGOT- 843IU/L, SGPT-1058IU/L, S.Bilirubin-2.0 mg/dl, Prothrombin Time 19 sec and PBF examination was negative for malaria parasite.

On follow-up after 2 weeks patient was normal with no fever & jaundice.

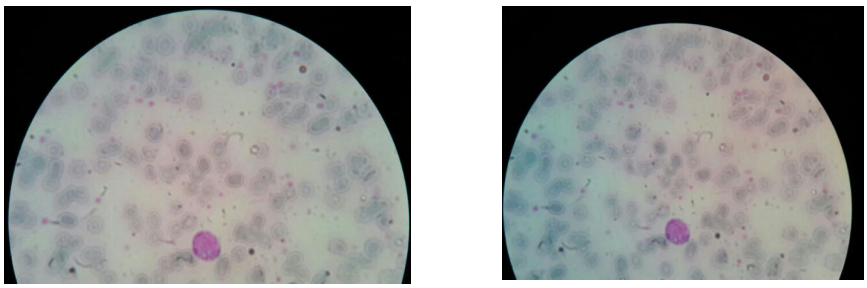

Fig 1: Peripheral smear of patient showing trophozoites of plasmodium vivax

Discussion

Malaria is still a leading cause of morbidity and mortality. According to WHO, 26 million cases of malaria were reported in 2011 resulting in 106820 deaths [4]. The majority of severe and fatal malaria is caused by Plasmodium falciparum [5].

Severe complicated malaria is a well recognised feature of Plasmodium falciparum malaria which usually manifests as cerebral malaria, though occasionally with liver failure, thrombocytopenia and other complications.

Mild hemolytic jaundice is common in malaria.

Hepatic dysfunction in malaria is common, but hepatic encephalopathy is unusual, and most of the time had either isolated infection with P. falciparum or a mixed infection with P. Vivax [6].

Hepatic involvement in malaria has largely shown severe infection with P. Falciparum infection [7].

There have been occasional reports of mixed infection with P. vivax and hepatitis E along with P. falciparum, resulting in malarial hepatitis [8].

P. vivax earlier termed as ‘benign tertian malaria' because of uncomplicated disease and was rarely fatal.

P. vivax had been reported recently with symptoms and signs of severe disease and even deaths [9]. Plasmodium vivax malaria presenting with impending hepatic failure is a rare with one case report in 2013 by Gupta HV and Ruku [10].

Our case presented with symptoms and signs of impending liver failure with deranged liver function tests and coagulation profile along with high grade fever with chills and rigors, without evidence of pre-existing liver disease. More importantly patient improved dramatically with qunine treatment.

Conclusion

In our case patient presented with symptoms and sign of severe acute hepatic impairment along with deranged coagulation and with blood smear positive for trophozoites of vivax.

Patient improved dramatically with antimalarial therapy with subsequent normalization of liver function test.

We suggest patient having impending hepatic failure, physician should be vigilant to rule out malaria because of its excellent prognosis if diagnosed and treated early.

Funding: Nil

Conflict of interest: Nil

Permission from IRB: Yes

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How to cite this article?

Nigam P, Indulkar M, Bansod A. Impending hepatic failure due to Plasmodium vivax Malaria: a case report. *Int J Med Res Rev* 2014;2(2):156-158. doi:10.17511/ijmrr.2014.i02.16.

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