

Plasmodium vivax malaria with thrombocytopenia presenting as Subarachnoid Hemorrhage in an elderly male

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Abstract

It is of common belief that plasmodium vivax causes benign malaria. But there are lots of evidence now available to suggest that it can cause complicated malaria, even cerebral malaria like plasmodium falciparum. We present a case of an elderly male who presented to us with drowsiness and later diagnosed as having subarachnoid hemorrhage (SAH) due to severe thrombocytopenia caused by plasmodium vivax infection.

Key words: Plasmodium vivax, Falciparum, Thrombocytopenia, Subarachnoid hemorrhage (SAH).

Introduction

Plasmodium vivax malaria is endemic in India and is usually uncomplicated. Though it is associated with mild hematological abnormalities, severe thrombocytopenia is rare. Although severe thrombocytopenia is commonly reported to be associated with *Plasmodium falciparum* infection and has been reported to occur in patients coinfecting with both *P. falciparum* and *P. vivax*, its occurrence has been rarely reported in cases of *P. vivax* malaria. But the current scenario has been changing with vivax malaria. Complicated vivax malaria is in rise in endemic areas now which are supported by a large number of data. Severe thrombocytopenia is multi factorial in vivax malaria and can manifest as catastrophic bleeding diathesis.

Case Report

A 72 year old male presented to us with complaint of headache and mild drowsiness since 2 hours, there was history of fever with chills and rigors since last 2 days for which patient took over the counter medication. There was no history of convulsions, cough or urinary problem. She was non diabetic and non hypertensive.

On examination pulse rate was 110/min regular, BP - 120/70 right arm supine position. There were no rashes. On CNS examination: patient was stuporous, moving all four limbs, pupil normal size reacting to light, planters were bilateral extensor. CVS and RS examination were normal. Per abdomen examination revealed grade I splenomegaly. Investigation revealed Hb- 12gm %, TLC and DLC within normal range. Peripheral smear showed ring form of plasmodium vivax and rapid malarial card test confirmed plasmodium vivax infection. Absolute platelet count were 11,000 /cumm, plasma glucose was 120 mg, KFT and LFT were within normal limits. Dengue and leptospira serology were negative. A non contrast CT brain revealed extensive subarachnoid hemorrhage in bilateral cerebral hemisphere with cerebral edema.



Fig.1 NCCT showing subarachnoid hemorrhage

Manuscript received: 14th Dec 2015
Reviewed: 24th Dec 2015
Author Corrected: 03rd Jan 2016
Accepted for Publication: 15th Jan 2016

In view of above findings a diagnosis was plasmodium vivax malaria with thrombocytopenia with subarachnoid hemorrhage kept patient was treated with Symptomatic and supportive treatment with Chloroquine. Patient also received multiple platelet transfusions till platelet count was maintained above 1 lakh/ mm³. After complication of parenteral chloroquine patient was put on primaquine 15 mg for 15 days. One week after treatment patient showed signs of improvement. Neurologically patient was alert, coherent and was able to walk. Platelet count remained stable at 3.5 lakh /cumm and patient discharged on request.

Discussion

Complicated malaria is usually associated with plasmodium falciparum infection but there is now growing evidence suggesting that p. vivax can also cause severe complicated malaria [1]. P. vivax usually infect young red blood cell and high parasite burden are not a feature of p. vivax infection but it is the higher cytokine production that usually causes severe malaria. [23]. Studies have shown that inflammatory markers like c-reactive protein, tumor necrosis factor alpha, interferon gamma, Interlukin 10, are increased in p.vivax infection as compared to p. falciparum infection. P .vivax can cause sequestration related complication like cerebral malaria, renal failure, liver dysfunction and ARDS. It can also cause non sequestration related complications like anemia and thrombocytopenia [4, 5].

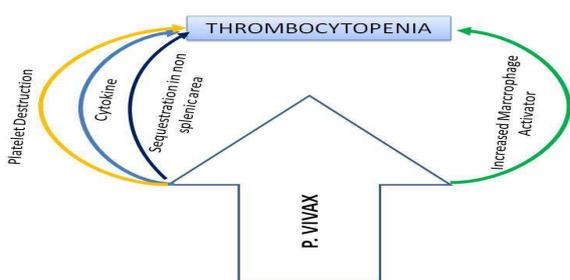


Fig 2: Mechanisms for thrombocytopenia in P. vivax malaria. (2)

How to cite this article?

Kadam N, Acharya S, Shukla S, Gupta A, Vaspute S. Plasmodium vivax malaria with thrombocytopenia presenting as Subarachnoid Hemorrhage in an elderly male. *Int J Med Res Rev* 2016;4(1): 129-130. doi: 10.17511/ijmrr.2016.i01.021.

Cerebral complications are usually common in p falciparum. Subarachnoid hemorrhage has been described in patient with cerebral malaria. Subarachnoid hemorrhage usually is caused by rupture of small vessels which get plugged by dead cells and associated thrombocytopenia and, or DIC (disseminated intra vascular coagulation). Plasmodium vivax infection is usually known to cause benign malaria. It should be taken seriously as far as complications are concerned.

Conclusion

To conclude, contrary to the presumption that usually vivax malaria is uncomplicated, the treating physician should be alert in treating cases of vivax malaria as it can very well present with catastrophic complications. So it is recommended that all possible complications should be sought for through appropriate investigations to prevent such type of serious consequences.

Funding: Nil, **Conflict of interest:** None.

Permission of IRB: Yes

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