International Journal of Current Advanced Research
ISSN: O: 2319-6475, ISSN: P: 2319-6505, Impact Factor: 6.614
Available Online at www.journalijcar.org
Volume 8; Issue 09 (E); September 2019; Page No.20061-20062
DOI: http://dx.doi.org/10.24327/ijcar.2019.3908.20062

Case Report

PLASMODIUM VIVAX INDUCED DIC PRESENTED WITH PANCYTOPENIA

Lokesh Bhalke, Manoj Gupta and Hemant Nargawe

201 Navya Regency Padmawati Colony Geeta Bhawan Indore

A R T I C L E I N F O

Article History:

Received 12th June, 2019
Received in revised form 23rd July, 2019
Accepted 7th August, 2019
Published online 28th September, 2019

Key words:

DIC LDH

A B S T R A C T

Disseminated intravascular coagulation is very rare complication of malaria. Even in malaria it is commonly associated with falciparum malaria. In Todays scenario the typical presentation of malaria with fever, chills and rigor is not present in every patient. Here we present a case of vivax malaria presented without fever and landed up into DIC.

INTRODUCTION

Malaria is caused by plasmodium which transmit through bite by female anopheles mosquito. Despite of govt efforts and public awareness the number of patients presented as complicated malaria is still high. According to the World Malaria Report 2017, in the year 2016, India accounted for 6% of all malaria cases in the world, 6% of the deaths, and 51% of the global *P. vivax* cases. The Report estimates the total cases in India at 1.31 million (0.94-1.83 million) and deaths at 23990.Disseminated intravascular coagulation (DIC)is a major complication of malaria besides those of renal failure, pulmonary oedema, cerebral malaria and anaemia. One of the complications is pancytopenia, which can occur in vivax infection secondary to microangiopathic haemolytic anaemia. Disseminated intravascular coagulation (DIC) is seen in <5% of patients with severe *Plasmodium falciparum* malaria. Here we report a case of vivax malaria induced DIC presented as pancytopenia.

CASE REPORT

A 17 years old female R/o Rajaram Nagar Indore came to MY Hospital Indore on 22/04/2019 with complains of Easy fatigability and generalised weakness since 4 months and yellow discoloration of sclera and urine since 3 months. Patient had history of low grade fever not associated with chills, rigor, rashes, cough, cold, burning micturition prior to this symptoms. Fever subsided within 3 days after taking some medication (no documents available). No significant past or family history was there.

Corresponding author:* **Lokesh Bhalke
201 Navya Regency Padmawati Colony Geeta Bhawan Indore

On examination patient was severely pale and icteric. Pulse was 114/min and BP was 100/70 mmHg. Liver and spleen was not palpable. Rest all physical examination was normal. Her initial blood test were remarkable for anaemia and thrombocytopenia, hyperbilirubinemia with Hb of 2 gm/dl, WBCs of 6000/mm3, Platelet of 15000/mm3, reticulocyte of 1%, bilirubin of 9.2 mg/dl (direct-2.9mg/dl). MCV was raised 104 fl. Peripheral smear shows schistocyteand serum B12 level was low 188 and ferritin was 389 ng/ml.HIV, HbsAg, anti HCV, anti HAV IgM negative. So initially we started IV Vit B12 and blood transfusion. Next day patient became drowsy and dyspnoeic, showing SpO2 76% with room air. Her chest was bilaterally clear so possibility of hypoxic encephalopathy was kept due to anaemia and patient was intubated. In afternoon patient start to bleed from oral mucosa and nose for that platelet transfusion was given and PT INR, APTT, FDP D-DIMER, LDH was sent. Her LDH was 1822U/L, FDP was 40 µg/ml, and D-Dimer was >15000 ngFEU/ml, INR was 1.4 and APTT was 36 sec. Then we have done malaria card test which comes out positive for VIVAX and to rule out the hemolytic anaemia coombs test, G6PD, Hb electrophoresis was sent. Hbelectrophoresis, G6PD was normal and Coombs test was negative. Immature platelet fraction was 24.7%. IV artesunate and clindamycin was started and patient improved within next 3-4 days. Her platelet, WBC, Hb started to rise and bilirubin decreased.

Table 1 Day wise investigations

Lab test	22/4/19	24/4/19	26/4/19/	28/4/19	30/4/19
Hb	2	6	5.6	5.6	8
WBC	6000	4900	2800	7200	7700
Platelet	15000	12000	8000	1 lakh	4.7 lakh
Bilirubin	9.2	18.5	5.4	2.1	2.2
Direct	2.9	6.2	2.4	1.5	1.5
SGOT	147	256	16	20	27

SGPT	117	176	63	43	25
Creatinine	0.1	0.2	0.2	0.2	0.3
FDP			40		
D DIMER			>15000		
Coombs			Negative		
Hb electrophoresis			Normal		
G6PD			Normal		

DISCUSSION

Disseminated intravascular coagulation is a condition in which blood clots form throughout the body's small blood vessels. The mechanism of DIC is the uncontrolled generation of thrombin by exposure of the blood to pathologic levels of tissue factor along with suppression of physiologic anticoagulantmechanism. This leads to systemic fibrin depositionin small and midsize vessels. It can compromise the blood supply of many organs like lung, kidney, liver, and brain, with consequent organ failure. The prolonged activation of coagulation results in consumption of clotting factors and platelets, which can lead to systemic bleeding. The release of proinflammatory cytokines such as interleukin 6 and tumor necrosis factor α plays a role in DIC. Common causes of DIC are sepsis, abruptio placentae, abortion, trauma, malignancy etc. Another important cause of DIC is malaria.DIC is seen in <5% of patients with severe *Plasmodium falciparum* malaria. Incidence is even low with plasmodium vivax. The mortality ranges from 30 to >80% depending on the underlyingdisease, the severity of the DIC, and the age of the patient. This DIC can cause microangiopathic haemolytic anaemia and patient can present with bleeding manifestation, jaundice and pancytopenia. Laboratory parameter shows pancytopenia, raised aptt, PT, FDP, D Dimer, bilirubin, LDH and peripheral smear shows schistocyte. The primary treatment of this condition includes treating the underlying cause.

Administration of FFP and platelet concentrates is indicated forpatients with active bleeding or at high risk of bleeding. Low doses of heparin (5–10 U/kg/hour) may be effective in patients with low-grade DIC associated with solid tumor, acute promyelocytic leukemia. In acute DIC, the use of heparin is likely to aggravate bleeding. This case is being reported because of atypical presentation of malaria without any acute febrile episode and DIC is very rare complication of VIVAX malaria and anti-malarial treatment can prevent the further progression of disease.

Reference

1. LaltanpuiiSailo, Debasis Pradhan, Rakesh Nongthombam, and Prithwis Bhattacharyya Disseminated intravascular coagulation in malaria.
2. Yamakawa H, Kiyotaki M, Hattori Y, Obana M, Matsuoka Y, Irimajiri S. A case of Plasmodium vivax malaria complicated with pancytopenia due to hypoplasia of the bone marrow.
3. Mohapatra MK, Padhiary KN, Mishra DP, Sethy G. Atypical manifestations of Plasmodium vivax malaria.
4. Miller LH, Good MF, Milon G. Malaria pathogenesis. Science.
5. Zvulunov A, Tamary H, Gal N. Pancytopenia resulting from hemophagocytosis in malaria. The Pediatric Infectious Disease Journal.
6. Mohapatra S[1], Samantaray JC, Arulselvi S, Ghosh A. Disseminated intravascular coagulation following malaria due to Plasmodium vivax: a thromboelastography-based study.

How to cite this article:

Lokesh Bhalke, Manoj Gupta and Hemant Nargawe (2019) 'Plasmodium Vivax Induced DIC Presented with Pancytopenia', *International Journal of Current Advanced Research,* 08(09), pp.20061-20062. DOI: http://dx.doi.org/10.24327/ijcar.2019.3908.20062

****