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Cerebral Complications in *Plasmodium vivax* Malaria

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Plasmodium vivax **has been increasingly reported to cause various serious manifestations of malaria including thrombocytopenia, cerebral malaria, acute renal, hepatic and pulmonary dysfunctions with reports of high morbidity and mortality by cerebral involvement. To evaluate the degree of severe complications in *Plasmodium vivax* such as cerebral malaria, hepatic dysfunction, renal complications and pulmonary complications in *Plasmodium vivax* infection. The study group included patients presenting with fever and associated symptoms characteristic of malarial infection.Diagnosis of malaria was made on the basis of clinical features,peripheral blood smear examination, Q.B.C and Rapid antigen detection test. Other laboratory investigations were also taken into consideration. Out of 1,737 patients positive for *Plasmodium vivax* infection, a total of 65 patients had atypical symptoms.32 patients presented with cerebral symptoms ,Hepatic funtions were affected in 31 patients. Renal dysfunction was observed in 1 patient and gastrointestinal complications in 1 patient..No patient in our study group presented with pulmonary symptoms. In cases of febrile illness with features of malaria, thrombocytopenia or neurological dysfunction not only *Plasmodium falciparum* but also *Plasmodium vivax* should be considered as differential diagnosis. Early detection,prompt management and adequate antimalarial treatment can reduce significant morbidity and mortality associated with severe complications of *Plasmodium vivax* infection.**

Key words: Cerebral complications, *Plasmodium falciparum*, malaria.

Malaria is a protozoal disease caused by infection with parasites of the genus *Plasmodium* and transmitted to man by certain species of infected female Anopheline mosquito[1] The clinical features of malaria vary from mild to severe and complicated according to the species of the parasite present, the patient's states of immunity, the intensity of the infection and also the presence of concomitant conditions such as malnutrition or other diseases.[2]

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Plasmodium vivax infection is considered to produce relatively benign disease without severe complications. *Plasmodium vivax* has been increasingly reported to cause various serious manifestations of malaria including thrombocytopenia, cerebral malaria, acute renal, hepatic and pulmonary dysfunctions with reports of high morbidity and mortality by cerebral involvement.[3] With increasing reports of drug resistance, this indeed is a cause for concern. The underlying mechanisms of severe manifestations are not well understood.

Prompt and effective treatment and case management should be the same as for severe and complicated falciparum malaria. There are reports of high morbidity and mortality by cerebral involvement.[4]The present study was done to

evaluate the degree of severe complications in *Plasmodium vivax* infection, such as cerebral malaria, hepatic dysfunction, renal complications and pulmonary complications in *Plasmodium vivax* infection.

MATERIALS AND METHODS

The study was conducted in the Department of Microbiology, JawaharLal Nehru Medical College and Hospital from January 2007 to September 2011.The study group included patients presenting with fever and associated symptoms characteristic of malarial infection in patients attending out patient departments and admitted in hospital wards.Diagnosis of malaria were made by :Clinical features,Examination of peripheral blood smears,examination of Quantitative Buffy Coat (QBC),Rapid malaria antigen detection test (Diagnos Malaria Stix,, Biomed industries) Other laboratory investigations perfomed were haemogram ,liver function tests,renal function tests,Cerebrospinal fluid-Biochemicals/ Culture and sensitivity.

RESULTS AND DISCUSSION

Out of 1,737 patients positive for *Plasmodium vivax* infection, a total of 65(3.74%) patients had atypical symptoms .A total of 150 (8.63%)patients were diagnosed positive by M.P. smear,1533(88.25%) patients by Q.B.C and 54(3.10%) patients by R.D.T. Almost all patients(90%-100%) had complaints of fever,chills

Table 1. Various clinical features in patients of *P. vivax* Malraia

S. No.	Symptoms	No of cases
1.	Fever	1737
2.	Chills and Rigors	1563
3.	Sweating	1702
4.	Headache	1720
5.	Altered sensorium	32
6.	Low Urinary output	01
7.	Diarrhoea	01
8.	Abdominal pain	00
9.	Yellowish discolouration of urine	08

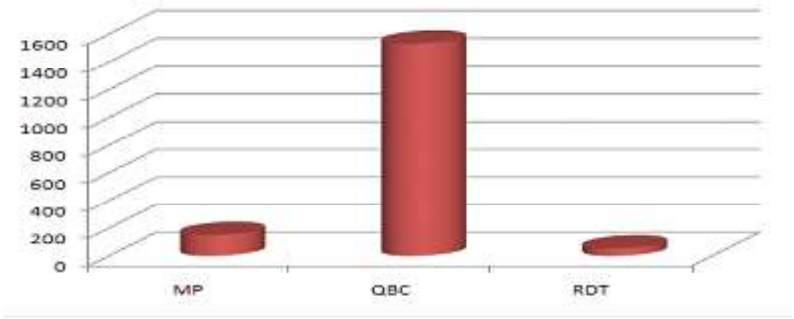

Fig. 1. BAR Diagram showing no of positive cases of *P. vivax* in past 5 years

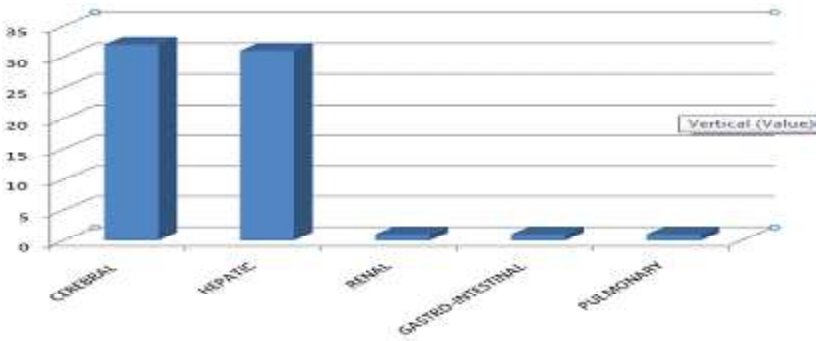

Fig. 2. BAR Diagram showing atypical complications of *P. vivax*

and rigors,sweating and headache,32 patients had altered sensorium,1 patient each with diarrhoea and low urinary output and 8 patients with yellowish discolouration of urine. Out of 65 patients ,32(49.2%) patients presented with cerebral symptoms along with other features of malaria. Hepatic funtions were affected in 31(47.6%) patients. Renal dysfunction was observed in 1(1.53%) patient.1(1.53%) patient presented with gastrointestinal complications.No patient in our study group presented with pulmonary symptoms.Almost all patients with atypical symptoms presented in the last two years.

Exact pathogenesis and organ-specific morbidity caused by *P. vivax* infection remains unrecognized and poorly studied because of a paucity of research in this area. *Plasmodium vivax* is widely believed to be incapable of causing cytoadherence and microvascular sequestration and therefore is unable to cause organ dysfunction. Recent observations have shown evidence of sequestration of parasites in lung vasculature during evaluation of lung injury in *P. vivax* malaria.[5]Cerebral dysfunction in *P. vivax* malaria may occur through generation of nitric oxide.[6] Cytokines and leukotrienes may be responsible for severe anemia and hemostatic complications. [7] Recent microrhematologic research that analyzed malaria severity in P. vivax infection clearly demonstrated enhanced aggregation, erythrocyte clumping, and reduced deformability affecting microcirculation.[8]

A unique case of *P. vivax* infection unexpectedly complicated by seizures and multiple basal ganglia infarcts was reported in Karachi,Pakistan.[6] In two studies conducted in 2008 in Austalia it was found that cerebral symptoms can present with both plasmodium falciparum or vivax malaria.[10,11] In 2009,2 cases of cerebral malaria due to P.Vivax infection have been reported in Jammu district.[12]A study conducted in India has reported 2 cases of cerebral complications with vivax malaria.[9]

The trend of disease with plasmodium vivax malaria is changing.It is increasingly recognized that serious and life threatening complications can occur with vivax malaria. There is an urgent need to re-examine the clinical spectrum and burden of *P. vivax* malaria so that adequate control measures can be implemented against this emerging but neglected disease.

REFERENCES

1. Kochar DK, Saxena V, Singh N, Kochar SK, Kumar SV, Das A, *Plasmodium vivax* malaria. *Emerg Infect Dis* 2005; **11***:* 132-134.
2. Mendis K, Sina BJ, Marchesini P, Carter R: The neglected burden of *Plasmodium vivax* malaria. *Am J Trop Med Hyg* 2001; **64**: 97-106
3. Kaur D, Wasir V, Gulati S, Bagga A, Unusual presentation of *Plasmodium vivax* malaria with severe thrombocytopenia and acute renal failure. *J Trop Pediatr* 2007; **53***:* 210-212.
4. Mohapatra MK, Padhiary KN, Mishra DP, Sethy G, Atypical manifestations of *Plasmodium vivax* malaria. *Indian J Malariol* 2002; **39***:* 18-25.
5. Nicholas MA, Handojo T, Michael CF, Tjitra E, Price RN, Graeme PM, Lung injury in *vivax* malaria: pathological evidence for pulmonary vascular sequestration and posttreatment alveolar inflammation. *JID* 2007; **195***:* 589-596.
6. Beg MA, Khan R, Baig SM, Gulzar Z, Hussain R, Smego RA, Cerebral involvement in benign tertian malaria. *Am J Trop Med Hyg* 2002; **67***:* 230-232.
7. Clark IA, Cowden WB, Why is the pathology of falciparum worse than that of vivax malaria? *Parasitol Today* 1999; **15***:* 458-461.
8. Jayavanth S, Park BC, Microrheologic dysfunctions in blood during malaria. *Indian J Exp Biol* 2007; **45***:* 111-120.
9. Gursharan Singh Narang and Neha Singla.Thrombocytopenia and other complications of *Plasmodium vivax* malaria. *Curr Pediatr Res* 2011; **15**(2): 117-119
10. Genton B, D'Acremont V, Rare L, Baea K, Reeder JC, *et al.,* Plasmodium vivax and mixed infections are associated with severe malaria in children: A prospec-tive cohort study from Papua New Guinea. PLoS Med 2008; 5: e127. doi:10.1371/journal.pmed.0050128
11. Tjitra E, Anstey NM, Sugiarto P, Warikar N, Kenangalem E, *et al.,* Multidrug-resistant plasmodium vivax associated with severe and fatal malaria. A prospective study in Papua, Indonesia. PLoS Med 5: e128. doi:10.1371/journal.pmed.0050128a, 2008.
12. Rekha Harish and Sanjeev Gupta. *Indian J Pediatr* 2009; **76**(5): 551-552.