

1 P.Vivax Malaria: A Benign Disease with Emerging Complications

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6 **Abstract**

7 Background: Malaria is an endemic disease in India. It is a mosquito-borne infectious disease
8 caused by genus Plasmodium. Complications usually occur with P.falciparum, its occurrence
9 has been rarely reported in cases of P. vivax malaria. We observe 107 patients with P.vivax
10 malaria who developed complications in the form of Thrombocytopenia, Acute kidney injury,
11 hyperbilirubinemia cerebral malaria, a rare presentation in P.vivax malaria. Objectives: As
12 the burden of P.vivax malaria is progressively increasing in community this study has been
13 carried out to find out various complications in P.vivax malaria which is considered as a
14 benign entity. Methods: We prospectively enrolled 150 patients hospitalized in C.U.Shah
15 hospital of P.vivax infection on initial microscopy with complications over a two year period.
16 Hematological, biochemical, serological, radiological investigations are performed to identify
17 complications.

18

19 **Index terms**— P.vivax malaria, complications of P.vivax, thrombocytopenia.

20 **1 Introduction**

21 Malaria is a mosquito-borne parasitic disease. In India it is mainly caused by P.vivax and P.falciparum. Complicated
22 malaria characterized by serious organ failures or abnormalities in the patient's blood or metabolism, usually
23 occurs in P.falciparum malaria. Manifestations of severe malaria include cerebral malaria, severe anemia,
24 hemoglobinuria, AR-DS, thrombocytopenia, cardiova-scular collapse and shock, acute kidney injury, metabolic
25 acidosis and hypoglycemia.

26 In contrast to falciparum malaria, vivax malaria is rarely associated with serious complication. Scattered cases
27 of P.vivax causing severe malaria have been reported in the last 30 years.

28 Manifestations of malaria vary from asymptomatic infection to severe malaria. The essential pathologic feature
29 of severe malaria is sequestration of erythrocytes, which contain mature forms of the parasite in the deep vascular
30 beds of vital organs and rosette formation, thus producing organ dysfunction.

31 P.vivax may no longer be a paradigm for uncomplicated malaria. Presence of thrombocytopenia in acute febrile
32 travelers returning from tropical areas has become highly sensitive marker for malaria diagnosis (D' ??cremont
33 et al.2002). The sensitivity of thrombocytopenia together with the acute febrile illness was 100% for malaria
34 diagnosis, with specificity of 70%, a positive predictive value of 86% & a negative predictive value of 100%
35 ??Patel et al 2004).

36 Since the beginning of the 1970s, there have been reports proposing that malaria associated thrombocytopenia
37 is quite similar in P.vivax and P.falciparum infections (Beale et al 1972). Most of the data were published in late
38 1990s because of an availability of affordable automated machines capable of performing complete blood count
39 (CBC). ??). The TROPHOZOIT form followed by RING form is predominantly seen in the PBF study. The
40 least common form of the parasite is GAMATOZYTE. While most of the time one or more types of the form of
41 parasites are found together in the peripheral blood smear study.

4 DISCUSSION

42 2 II.

43 3 Material & Methods

44 IV.

45 4 Discussion

46 ? Organ dysfunction is characteristic of *P.falciparum* malaria & unusual in *P.vivax* infection. Severe complicated
47 malaria is a well-recognized feature of *P.falciparum* malaria. Although a few cases with *P.vivax* have been reported
48 in literature. Any patient infected with *P. vivax* who exhibits severe malaria is presumed to be suffering from
49 mixed infection [5]. However, that may not be always true. As evident from the present report, *P.vivax* infection
50 can also present with complications. ? Clinical data indicates that *P.vivax* can cause both sequestrations related
51 and non-sequestration related complications of severe malaria [4]. The exact pathogenetic mechanism however
52 remains elusive. Sachdev and Mohan [6] studied the clinicolaboratory profile of patients with *P.vivax* cerebral
53 malaria. Focal neurological signs were observed in one patient. Recently a case of cerebral vivax malaria that
54 presented with status epilepticus has been described [7]. ? *P.vivax* malaria without any complication has been
55 reported many times, even remains silent [8]. It may be presented occasionally with mild anemia or febrile
56 illness. However, none of them had any evidence of thrombocytopenia, AKI and recovered without any sequel
57 [8,9]. ? However almost all type of complications have been found in this study, but more common one is
58 thrombocytopenia. ? There are reports of thrombocytopenia occurring as a manifest of *P.vivax* malaria in
59 adults. The mechanism of thrombocytopenia (figure ??) in malaria is not clearly known?

60 1. Decreased thrombopoiesis, although this hypothesis was later ruled out [9,10] 2. Thrombocytopenia is a
61 result of peripheral destruction in which immune complexes generated by malarial antigens lead to sequestration
62 of the injured platelets by macrophages in the spleen, although this mechanism has not been systematically
63 evaluated in *P.vivax* malaria [1,11] . 3. An inverse relationship between elevated parasite levels and decreased
64 platelet counts observation consistently has been reported for *P.vivax* infection [12].

65 ? Fajardo and Tallent [9] in 1974 demonstrated *P.vivax* within platelets by electron microscopy and suggested
66 a direct lytic effect of the parasite on the platelets. Both non-immunological destruction [13] as well as immune
67 mechanisms involving specific platelet-associated IgG antibodies that bind directly to the malarial antigen in the
68 platelets has been recently reported to play a role in the lysis and the development of thrombocytopenia [14]. ?
69 Oxidative stress damage of thrombocytes has also been responsible based on the finding of low levels of platelet
70 superoxide-dismutase and glutathione peroxidase activity and high platelet lipid peroxidation level in malaria
71 patients, when compared to those of health subjects [15]. ? Malaria may cause anemia and hyperbilirubinemia
72 because of the loss of red blood cells. Intravascular hemolysis & DIC in *P.vivax* malaria can cause ARF, which
73 occurs more in *P.falciparum* malaria but we found 8% cases in *P.vivax* infection [10] . Renal ischemia is the
74 dominant pathogenic mechanism that results in acute tubular necrosis. The prognosis of ARF in *P.vivax* malaria
75 is favorable.

76 ? *P.vivax* may no longer be a paradigm for uncomplicated malaria. It has been observed that the burden
77 of complicated *P.vivax* malaria is progressively increasing. ? Complications are common in the form of
78 Thrombocytopenia(71.33%), Anemia (10%), hyperbilirubinemia(8.67%), Acute renal failure(8%) and cerebral
79 malaria(1.33%) in their respective order. As far as the thrombocytopenia is concerned, it is having favorable
80 prognosis & most of them were recovered with only antimalarial treatment so routine use of platelet transfusion
81 is not recommended in a case of thrombocytopenia. ? *P.vivax* now a days emerging as one of the cause of isolated
82 thrombocytopenia. It is a challenge to differentiate *P.vivax* from *falciparum* malaria and Dengue fever.

83 FIGURE 3

84 No.of pts. 1 2 3 4 5 6

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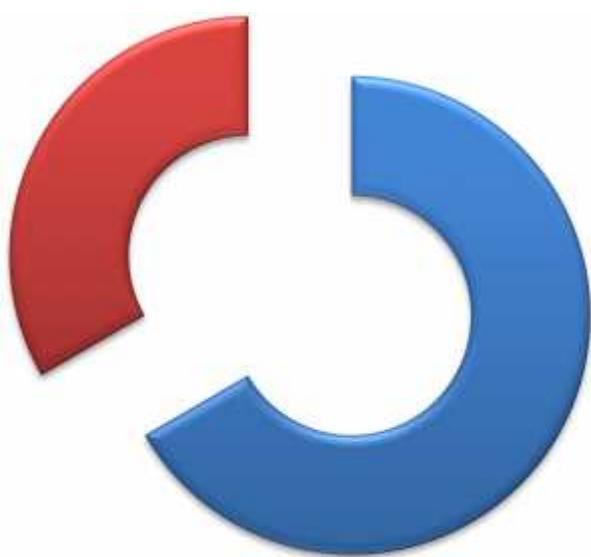
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Figure 1:



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Figure 2: Figure 1 :ConclusionFigure 2 :



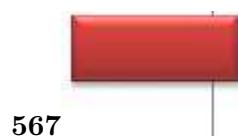
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Figure 3: Figure 3 :



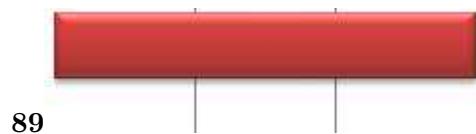
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Figure 4: Figure 4 :



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Figure 5: Figure 5 :Figure 6 :Figure 7 :



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Figure 6: Figure 8 :Figure 9 :

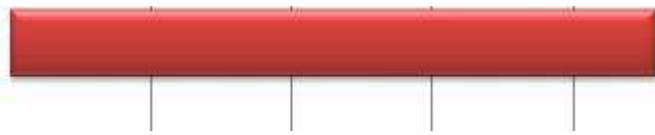


Figure 7:



Figure 8:

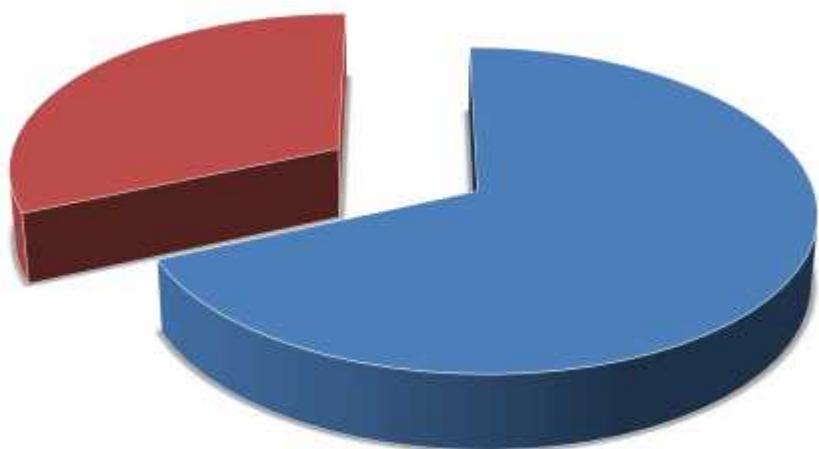


Figure 9:

4 DISCUSSION

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