**How to Cite**:
Agrawal, S., Chaursiya, O. S., & Ravanagomagan, R. (2022). Hepatic dysfunction, renal dysfunction and thrombocytopenia in children admitted with malaria in tertiary care centre in Bundelkhand region - An observational study. *International Journal of Health Sciences*, *6*(S7), 1941–1948. https://doi.org/10.53730/ijhs.v6nS7.11733

# Hepatic dysfunction, renal dysfunction and thrombocytopenia in children admitted with malaria in tertiary care centre in Bundelkhand region - An observational study

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***Abstract*---**Vivax malaria is long considered to have a benign course. It is known for multiple relapses and falciparum infection was associated with complicated malaria. However in the past few years there is a changing trend in the clinical manifestations of vivax malaria namely severe or complicated disease; sometimes even causing death. Objective: To study the hepatic dysfunction, renal dysfunction and incidence of thrombocytopenia in malaria in childhood in Bundelkhand region. Design: Observational study in a Pediatric ward of tertiary care hospital in Jhansi Material and Methods: 100 patients between the age groups of 1 to 15 yrs, whose peripheral smear were positive for malarial parasite have been included in the study group. A detailed history and clinical examination was followed by investigations like hemogram, platelet counts, general blood picture, reticulocyte count, serum bilirubin, SGOT, SGPT, serum alkaline phosphatase, blood urea, serum creatinine. Results: Raised level of SGPT, SGOT and total serum bilirubin seen were 31%, 30%, 53% respectively. In our study 11(11%) of cases had raised level of blood urea or serum creatinine or both. 88 cases (88%) had platelet count < 1.5 lac/$mm^3$. P.falciparum cases who had platelets counts <1.5 was 47(47%) and rest were P.vivax positive cases which was 41(41%).Conclusion: liver is commonly affected in malaria and hepatic dysfunction ranges from mild elevation to the


*Manuscript submitted: 9 April 2022, Manuscript revised: 18 June 2022, Accepted for publication: 27 July 2022*

range of acute hepatitis. There was no significant difference in incidence of thrombocytopenia between vivax and falciparum cases. Mild renal impairement was present in a number of cases and dehydration also plays a role in the genesis of renal impairement in children with malaria.

***Keywords*---**vivax, falciparum, thrombocytopenia, liver dysfunction, renal dysfunction.

## Introduction

Vivax malaria is long considered to have a benign course. It is known for multiple relapses and falciparum infection was associated with complicated malaria. However in the past few years there is a changing trend in the clinical manifestations of vivax malaria namely severe or complicated disease; sometimes even causing death.

Hepatic dysfunction is usual in severe malaria. Jaundice is common as there reduction in clotting factor synthesis, metabolic clearance of drugs, biliary excretion, and failure of gluconeogenesis which contributes to lactic acidosis and hypoglycaemia.

The activity of a broad range of cytochrome p450 mixed function oxidase is reduced including cyp3a4 which is responsible for quinine 3 hydroxylation, principle route of quinine metabolic clearance ***[Pukrittayakamee e1 al, 1997]*** clearance is reduced in proportion of disease severity.

Jaundice in malaria has hemolytic, hepatic, and cholestasis components. Cholestasis jaundice may persist well into the recovery period.

Liver damage may result from an alteration in vascular flow through the organ as the parasitized RBCs adhere to endothelial cells, blocking sinusoids and obstructing intrahepatic blood flow.

Intravascular hemolysis of parasitized and nonparasitized RBCs has been considered an important factor in the causation of mild to moderate jaundice, in which bilirubin is predominantly unconjugated. However, hemolysis is never the sole cause of severe jaundice nor the conjugated hyperbilirubinemia and increases in the serum levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are seen in many patients.

In severe malaria renal microvasculature obstruction conseqent upon sequestration in the kidney is considered to be responsible. Impaired perfusion may be compounded by reduced erythrocyte deformability and the release of significant amount of hemoglobin and cellular debris during hemolysis.

Massive hemolysis compounds the insult in black water fever complicating severe malaria.

Thrombocytopenia is common among people indigenous to the tropics, and non-immune subjects infected by Plasmodium falciparum or P. vivax.

Malaria-related thrombocytopenia may result from either a decrease in platelet production or an increased platelet turnover due to different mechanisms of destruction.

There are converging arguments in favor of immune mediated destruction, platelet activation, removal by the reticuloendothelial system or consumption in different steps of coagulation.

**Material And Methods**

The present study was conducted in the Department of pediatrics with active collaboration of the Department of pathology. Children of age groups between 1 to 15 yrs ,who were admitted to pediatrics wards of this hospital during the outbreak of malaria in July-Sept 2019 and 2020 in whom a diagnosis of malaria were confirmed by the presence of malaria parasite in the blood were studied.

100 patients between the age groups of 1 to 15 yrs ,whose peripheral smear were positive for malaria, parasite have been included in this study group, A detail history was taken and clinical examination was done to rule out any past disease of renal, prerenal, post renal, cardiovascular disorder ,hepatic ,endocrine, and metabolic disorder. A detailed history was taken such as H/O fever, and its details, duration, severity, associated chills and rigor, course, paroxysm and any other variation. H/o vomiting, loose motion, headache, convulsion, abdominal pain, hemoglobinurea, jaundice, bleeding manifestation, any h/o malaria were recorded.

Patients have been examined for dehydration, pallor, hypotension, CNS manifestation, jaundice, edema, respiratory distress, evidence of bleeding, liver size, spleen size, and urine output. Then patients were subjected to, thorough systemic examination and then following investigation were undertaken like hemogram, platelet counts, general blood picture, reticulocyte count, serum bilirubin (total and direct), SGOT, SGPT, serum alkaline phosphatase, blood urea, serum creatinine, urine for routine examination.

About 8 ml of blood was collected by venipuncture from every patient alter correction of any dehydration taking due aseptic precaution.

**Observation**

In our study maximum no. of patient affected was in the 5-10 yrs age group and the least affected group was 1-5 yrs, which was 38% and 25% respectively. 37% of affected children were between the ages of 10-15 yrs,

In our study total P. falciparum affected patient were 58(58%), and P. vivax positive cases were 42(42%). P. falciparum cases were maximum 27(27%) in the age group of 1-5 yrs. P. vivax affected cases were 17%, 15%, 10% in the age groups of (1-5), (5-10), (10-15) yrs respectively(Table 1).

In our study, it was found that malaria can present with a wide range of symptoms. 99% of cases were presented with fever and 1% case without any fever. P. falciparum cases presented with fever were 57(57%), whereas P. vivax cases presented with fever were 42*(42%)*. 'Total 16% of cases were presented with abdominal pain of which 10% were P. falciparum and 6% were P. vivax positive cases in our study. 5% of total cases were presented with diarrhea and 24(24%) of all cases presented with convulsion in which 17(17%) was P. falciparum positive and 7( 7%) cases were P. vivax positive. Jaundice was present in 18(18%) cases at the time of admission in our hospital, in which 8(8%) were P. falciparum positive and 10(10%) cases were P. vivax positive. Bleeding manifestation was present in 19(19%) cases, in which 13(13%) were P. falciparum positive cases and 6% were P. vivax positive cases. 11% of cases were presented with decreased urine output and 5% of total cases presented with cough. Edema was present in 5% of total cases in which 3% were P. vivax and 2% cases were P. falciparum positive.43(43%) cases were presented with pallor in which, 30(30%) were P. falciparum positive cases and 13(13%) were P. vivax positive 2(2%) cases presented with signs suggestive of CHF. Single Case (1%) was presented with black water fever(Table 2).

In our study 92% (92) cases had splenomegaly/ hepatosplenomegaly/ hepatomegaly. Isolated hepatomegaly was present in 9% of total cases, whereas isolated splenomegaly was present in 21% of total patient. 62%(62) of patient had hepatosplenomegaly. Out of 58 were P. falciparum cases, 51 had organomegaly, and 41 cases of total 42 vivax infected cases had organomegaly.

The number of cases whose hemoglobin level were between 6-9.9 gm% were 34 or 34% of total. 20 cases of them were P. falciparum and 14 cases were *P.* vivax positive cases. 24% of patients had hemoglobin level between 10-12gm% and 4% cases have Hb level of >12 gm%. It was observed that 27% of cases who had Hb% <6gm were P. falciparum cases and 11% cases were vivax positive.

In our study 53% (53) of cases have increased level of total serum bilirubin (>1mg%). The number of P. falciparum infected person who had increased level of total serum bilirubin were 29( 29%). 24%(24) of P. vivax positive cases had increased level of total serum bilirubin. Most affected age group was 5-10 yrs in which 22% cases had increased level of serum bilirubin and least affected group was 1-5 yrs in which 13% of patients have increased level of total serum bilirubin.

It was observed that the total number of cases in whom raised level of SGPT, SGOT and total serum bilirubin seen were 31%, 30%, 53% respectively. Highest number of cases who had increased level of liver enzymes and serum bilirubin were seen in P. falciparum cases, which was 20%, 19%, 29% for SGPT, SGOT, and serum bilirubin respectively.

In our study 88 cases (88%) had platelet count < 1.5 lac/$mm^3$. P. falciparum cases who had platelets counts <1.5 was 47(47%) and rest were P. vivax positive cases which was 41(41%). Cases in which platelets counts were less than 50000 was 40(40%) of total cases, where 22(22%) cases were P. falciparum infected cases and 18(18%) cases were P. vivax infected cases. 24(24%) cases had

platelets counts between 0.5-1 lac and, again 24(24%) cases had platelet counts between 1-1.5 lac./$mm^3$ (Table -3).

In our study 11(11%) of cases had raised level of blood urea or serum creatinine or both. Greatest affected group was 10-15 yrs in which 6 cases had increased urea, creatnine level(Table-4). Next group was age group of 5-10 yrs and least affected was 1-5 yrs age group.

**Discussion**

The aim of the study is to study the hepatic dysfunction, renal dysfunction and incidence of thrombocytopenia in malaria in childhood in bundelkhand region.

9% of cases presented with hepatomegaly, 21% with isolated splenomegaly. Overall hepatomegaly/splenomegaly/hepatosplenomegaly was present in 92% of the cases which correlated with study of *Nityanand et al.*

It was observed that 88% of cases had platelet count below 1.5 lac/$mm^3$,64% cases below 1 lac/$mm^3$, 40% cases below 50000/$mm^3$. Platelets were diminished in association with intravascular coagulation. It took 5-7 days after start of treatment before it return to normal. Thrombocytopenia is a common finding in falciparum malaria and may in part be due to sequestration of platelets in capillaries of internal organs. Patients with vivax malaria, a mild infection without severe forms also frequently had thrombocytopenia.

Among the cases of malaria, 53% had elevated level of serum bilirubin out of which 29% were of P. falciparum and 24% of P. vivax. This was in concordance with the study by *Patwari et al(1979)* where serum bilirubin was raised in 26% of cases infected with P.vivax. serum bilirubin was also increased in many other studies by *Mishra et al(1992), Isreal and Soloman(1994) and Arya and Prasad(1996).*

In our study SGPT was raised in 31% cases and SGOT was raised in 30% of cases which is higher than those reported by *Bag et al(1994)* in a small series of 16 cases of falciparum malaria.

Renal impairement was depicted in our study as raised levels of blood urea and serum creatinine in 11% of the cases. *Ahmad et al(2003)* reported impaired renal function in 48% of cases of total malaria out of which 66% accounted for falciparum malaria and 30% for vivax malaria. *Habte et al* reported acute renal failure in 33% of patients of severe malaria. *Weber and boker et al* reported acute renal failure in 29.03% of falciparum malaria cases in one series.

So the incidence of malaria varies from study to study and depends on individual susceptibility. The disturbance in renal microcirculation is responsible for acute renal failure and immunological reaction to parasites accounts for glomerular lesion.

**Conclusion**

It was observed that liver is commonly affected in malaria and hepatic dysfunction ranges from mild elevation to the range of acute hepatitis. There was no significant difference in incidence of thrombocytopenia between vivax and falciparum cases. Mild renal impairement was present in a number of cases and dehydration also plays a role in the genesis of renal impairement in children with malaria.

**Table 1**
**Number of cases and parasite distribution in each age group**

| Age group | 1-5 yrs | 5-10 yrs | 10-15 yrs | total |
|---|---|---|---|---|
| No. of cases | 25% | 38% | 37% | 100% |
| P. vivax | 17% | 15% | 10% | 42% |
| P. falciparum | 8% | 23% | 27% | 58% |

**Table 2**
**Presentation of malaria**

| Symptoms/signs | P.falciparum | P.vivax | Total |
|---|---|---|---|
| Fever | 57 | 42 | 99 |
| Without fever | 1 | 0 | 1 |
| Abdominal pain | 10 | 6 | 16 |
| Diarrhea | 2 | 3 | 5 |
| Convulsion | 17 | 7 | 24 |
| Jaundice | 8 | 10 | 18 |
| Bleeding | 13 | 6 | 19 |
| Oliguria | 5 | 6 | 11 |
| ARI | 2 | 3 | 5 |
| Edema | 2 | 3 | 5 |
| Moderate to severe pallor | 30 | 13 | 43 |

| | | | |
|---|---|---|---|
| CHF | 1 | 1 | 2 |
| Black water fever | 1 | 0 | 1 |

Table 3
Correlation between hepatosplenomegaly, hb%, s.bilirubin, SGOT, SGPT, thrombocytopenia and type of malaria

| Parasite | P.falciparum | P.vivax | Total |
|---|---|---|---|
| Hepatosplenomegaly<br>Hepatomegaly<br>splenomegaly | 32<br>4<br>15 | 30<br>5<br>6 | 62<br>9<br>21 |
| Hb% level<br><6 gm%<br>6-9.9 gm%<br>10-12 gm%<br>>12 gm% | <br>27<br>20<br>16<br>3 | <br>11<br>14<br>8<br>1 | <br>38<br>34<br>24<br>4 |
| S.bilirubin | 29 | 24 | 53 |
| SGOT | 19 | 11 | 30 |
| SGPT | 20 | 11 | 31 |
| Platelet count<br><50000<br>50000-1 lac<br>1-1.5 lacs | <br>22<br>13<br>12 | <br>18<br>11<br>12 | <br>40<br>24<br>24 |

Table 4
Correlation between raised blood urea, raised serum creatinine, and type of malaria

| Age group | 1-5 yrs | 5-10 yrs | 10-15 yrs | Total |
|---|---|---|---|---|
| P. vivax | 0 | 1 | 4 | 5 |
| P. falciparum | 1 | 3 | 2 | 6 |
| Total | 1 | 4 | 6 | 11 |

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