



## RESEARCH ARTICLE

**A STUDY OF ASSOCIATION OF ABO BLOOD GROUPS WITH THE TYPE AND SEVERITY OF  
MALARIA.**

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**Abstract**

**Context:**-This study summarizes the understanding of relationship between ABO blood group and the incidence and complications of malaria in Western Maharashtra

**Aims and Objectives:**-To determine the type of malarial infection in conjunction with a particular blood group and also to study the blood group in which the rates of complications of malaria were higher..

**Materials & Methods:**-This was a prospective study over one year period at a tertiary care centre in Western Maharashtra. Peripheral smear was the main diagnostic procedure. ABO blood group, complete haemogram, renal functions and liver functions of total 160 patients of malarial fever were analysed. All patients of P.vivax were treated with chloroquine and later followed to check for relapse of malaria.

**Results:**-Of the total 160 patients of malaria, 145 patients were positive for P.vivax(90.6%) and 15 patients for P.falciparum(9.4%). B-positive blood group was found in 52 patients(32.5%) of malaria, O-positive blood group in 21 patients(12.65%), B-negative blood group in 13 patients(7.83%), O-negative blood group in 6 patients(3.61%). 55 patients(34.37%) suffered from thrombocytopenia, of which P.vivax constituted 89.1%(n=49) and P.falciparum 10.9%(n=6). All the patients suffering from P.vivax infection were treated with chloroquine and primaquine thus resulting in complete cure.

**Conclusion:**-Although, in our study, patients with B-positive blood group were a bit more susceptible to the chances of malarial infection and O-negative blood group were the least susceptible ones, this difference is not statistically significant thus indicating the need for further genetic studies to analyse this correlation as the ABO groups are genetically controlled. Our study also proves that the P.vivax strains are still susceptible to the chloroquine therapy in Western Maharashtra.

## Introduction:-

Malaria is still a devastating infectious disease of the countries of the developing world with about 1.5-2.7 million deaths annually.<sup>[1]</sup> many papers have been published about the correlation of malaria with ABO blood group with many recent papers contradicting the same.<sup>[2]</sup> Many questions still arise whether a particular blood group can offer resistance to the severity of malarial infection or not. This need has now been met by two recent studies agreeing in their conclusion that blood group O confers resistance to malaria.<sup>[3,4]</sup> We will hereby correlate our findings with these recently published data and also correlate the blood groups of ABO system with the severity of complications. As per the recent data, *P.vivax* is resistant to chloroquine in many parts of the world where malaria is prevalent but we hereby will also describe, in short, the scenario in areas where chloroquine is still the drug of choice with no resistance to it till date.

## Materials & Methods:-

This was a prospective study conducted between June 2011 to November 2012 at the Krishna Institute of Medical Sciences University, Karad, a tertiary health care centre in western Maharashtra, India. EDTA blood samples were taken after informed oral consent and were subjected to thick smear using Giemsa stain and thin smear examination using Wright's stain. Five drops each, of blood samples were dropped on glass slides and grouping sera, Anti A, Anti B, anti A+B and Anti D were added, mixed and observed for reactions, in form of agglutination. Observed reactions were recorded for determining the blood group. The patients were divided in two groups- one group with *P.vivax* malaria and other group with *P.falciparum* malaria. ABO blood group, platelet count, renal function and liver function were noted for each group. Patients were then divided into uncomplicated malaria and complicated malaria. All the patients of *P.vivax* malaria were treated with chloroquine and were later on given radical cure with primaquine as per WHO guidelines and were followed up for possible relapse of malaria which fortunately was reported in none.

## Results:-

160 patients with suspicion of malaria were admitted and clinically examined for malaria symptoms. Blood sample was taken for examining malarial parasites using thick and thin smear. Their ABO blood groups were determined. The patterns of malaria infection were determined among ABO blood groups. 145 patients (Table 1) were positive for *P.vivax* (90.6%) and 15 patients for *P.falciparum* (9.4%). B-positive blood group was found in 52 patients (32.5%) of malaria, A-positive blood group in 35 patients (21.87%), O-positive blood group in 21 patients (12.65%), AB-positive blood group in 15 patients (9.03%), A-negative blood group in 14 patients (8.43%), B-negative blood group in 13 patients (7.83%), O-negative blood group in 6 patients (3.61%), AB-negative blood group in 3 patients (8.33%).

55 patients (34.37%) suffered from thrombocytopenia (Table 2), of which *P.vivax* constituted 89.1% (n=49) and *P.falciparum* 10.9% (n=6). The incidence of thrombocytopenia is much higher in malaria (n=55) as such, but when compared between *P.vivax* and *P.falciparum*, there is no much statistically significant difference ( $\chi^2=0.770$ ;  $p>0.05$ ). Deranged renal function was found in 52 patients (32.5%) (Table 3), of which *P.vivax* constituted 88.5% (n=46) and *P.falciparum* 11.5% (n=6). its incidence is much higher in malaria (n=55) as such, but when compared between *P.vivax* and *P.falciparum*, there is no much statistically significant difference ( $\chi^2=0.770$ ;  $p>0.05$ ).

Deranged liver functions were found in 82 patients (51.25%) (Table 4), of which *P.vivax* constituted 89% (n=73) and *P.falciparum* 11% (n=9). The incidence of thrombocytopenia is much higher in malaria (n=55) as such, but when compared between *P.vivax* and *P.falciparum*, there is no much statistically significant difference ( $\chi^2=0.770$ ;  $p>0.05$ ). Amongst all the patients of *P.vivax* malaria, the sensitivity to chloroquine was 100% (n=145).

## Discussion:-

Present study highlights the burden of malarial infection in western Maharashtra. Total 160 patients suffered from complicated malaria. Blood group antigens A and B are trisaccharides attached to a variety of glycoproteins and glycolipids on the surface of erythrocytes and these triglycerides are thought to act as receptors for resetting on uninfected erythrocytes and bind to parasite resetting ligands. However, blood group antigens A and B are not expressed in blood group O individuals and so the rosettes formed by blood group O are smaller and easily disrupted as compared to the rosettes formed by erythrocytes of blood group A, B or AB. This is because the A and B antigens are receptors for resetting on uninfected erythrocytes, being bound by a parasite protein called PfEMP1 which is expressed on the surface of infected erythrocytes.

**Singh et al**<sup>[7]</sup> have reported that 'A', 'B' and 'O' groups are equally susceptible to malaria while 'AB' is less susceptible. However, significantly lower frequency of 'A' and 'O' groups were found in *P. falciparum* malaria. Results of our study were also consistent with this study in the form that AB blood group was less susceptible to malarial infection.

**Thakur and Verma**<sup>[8]</sup> did not find correlation with ABO groups and susceptibility to malaria which is consistent with our study.

**Akanbi et al**<sup>[9]</sup> have observed that 'A' group has more parasite density than 'B' and 'O' groups and 'O' group red cells have minimum density. In our study, B group had a bit more (statistically insignificant) parasite density than A-group.

**Barragan et al** have observed that 'A' antigen is the co-receptor for *P. falciparum* rosetting. 'O' group erythrocytes have reduced rosetting in *P. falciparum* malaria. In the present study, though incidence of 'O' group was decreased in malaria cases, we did not find statistical correlation.

**Cserti and Dzik**<sup>[10]</sup> reviewed literature that investigated relationship between *P. falciparum* malaria and ABO groups but did not find a study with adequate sample size. In the mean time, **Gupte et al**<sup>[11]</sup> studied 8028 malaria cases and 11,303 normal controls and found that 'A' blood group is more susceptible to have malaria infection, and risk of cerebral malaria and DIC is more in 'A' group. Our findings in a small scale study showed increased incidence of malaria in B-group rather than A-group. **Chowdheri et al**<sup>[12]</sup> in bulletin of World Health Organisation has mentioned with blood group A were found to have more complications in the form of cerebral malaria, severe anaemia, jaundice and renal failure and this finding is consistent with our study also.

Our study shows a kind of protection for complications in blood group O patients suffering from malaria which is in accordance with **Fischer et al**<sup>[5]</sup> who reported favourable outcomes for blood group O. Our study is also having findings persistent with **UM Jadhav et al**<sup>[6]</sup> who found that thrombocytopenias was common in *P. vivax* malaria and various mechanisms viz. immune-mediated lysis, sequestration in the spleen and a dyspoietic process in the marrow with diminished platelet production have all been postulated. Abnormalities in platelet structure and function have been described as a consequence of malaria, and in rare instances platelets can be invaded by malarial parasites themselves.

Taken together, the study by **Rowe et al**<sup>[3]</sup> with its focus on pathogenic mechanisms, and that by **Fry et al**<sup>[4]</sup> with its focus on genetic mechanisms, provide strong evidence that individuals with non-O blood groups are at increased risk of severe malaria which is consistent with the findings in our study also. We hereby want to conclude that there is no clearcut association of incidence of malaria with any blood group and all the studies done till date have concluded variable results depending on the endemicity of malaria in their regions. Furthermore, the pattern of sensitivity of *P. vivax* is changing and more and more reports of *P. vivax* being resistance to chloroquine are being published and this is due to the misuse of the drug and in some endemics where the mutation has occurred in the genome of *P. vivax*. The data obtained support the hypothesis that malaria parasite rosetting plays a direct role in the pathogenesis of severe malaria and thus provide extra impetus for research. We also want to conclude that in rural areas, where the scope for the use of novel antimalarials is still a dream, in such places chloroquine should be used as the primary drug for the treatment of malaria rather than anything else and once started, it should be given in full complete therapeutic doses to avoid resistance to chloroquine in such areas where only that drug is the lifeline for almost all the patients of malaria.

Table 1: Table showing the incidence of P.vivax and P.falciparum in different blood groups of ABO system:-

<b>Blood Group</b>	<b>P.falciparum</b>	<b>P.vivax</b>	<b>Total</b>
<b>A-negative</b>	n=1 (7.1%)	n=13 (92.9%)	<b>14</b> <b>(100.0%)</b>
<b>A-positive</b>	n=14 (40.0%)	n=21 (60.0%)	<b>35</b> <b>(100.0%)</b>
<b>AB-negative</b>	n=0 (0%)	n=3 (100.0%)	<b>3</b> <b>(100.0%)</b>
<b>AB-positive</b>	n=0 (0%)	n=15 (100.0%)	<b>15</b> <b>(100.0%)</b>
<b>B-negative</b>	n=0 (0%)	n=14 (100.0%)	<b>14</b> <b>(100.0%)</b>
<b>B-positive</b>	n=0 (0%)	n=52 (100.0%)	<b>52</b> <b>(100.0%)</b>
<b>O-negative</b>	n=0 (0%)	n=6 (100.0%)	<b>6</b> <b>(100.0%)</b>
<b>O-positive</b>	n=0 (0%)	n=21 (100.0%)	<b>21</b> <b>(100.0%)</b>
<b>Total</b>	<b>n=15</b> <b>(9.4%)</b>	<b>n=145</b> <b>(90.6%)</b>	<b>160</b> <b>(100.0%)</b>

Table 2: Table showing the incidence of thrombocytopenia in P.vivax and P.falciparum:-

<b>Platelet Count</b> <b>(in lakhs)</b>	<b>P.falciparum</b>	<b>P.vivax</b>	<b>Total</b>
<b>&lt;1.5</b>	n=6 10.9%	n=49 89.1%	<b>55</b> <b>100.0%</b>
<b>1.5-4.0</b>	n=8 8.0%	n=92 92.0%	<b>100</b> <b>100.0%</b>
<b>&gt;4.0</b>	n=1 20%	n=4 80.0%	<b>5</b> <b>100.0%</b>
<b>Total</b>	<b>n=15</b> <b>8.8%</b>	<b>n=145</b> <b>91.2%</b>	<b>160</b> <b>100.0%</b>

 $\chi^2=0.770$ ;  $p>0.05$

**Table 3:** Table showing the incidence of deranged renal functions in P.vivax and P.falciparum:-

<u>Serum Creatinine (md/dL)</u>	<u>P.falciparum</u>	<u>P.vivax</u>	<u>Total</u>
<1.6	n=9 (8.4%)	n=99 (91.7%)	<b>108</b> <b>(100.0%)</b>
>1.6	n=6 (11.5%)	n=46 (88.5%)	<b>52</b> <b>(100.0%)</b>
<b>Total</b>	<b>15</b> <b>9.4%</b>	<b>n=144</b> <b>(90.6%)</b>	<b>160</b> <b>(100.0%)</b>
$\chi^2=0.401$ ; $p>0.05$			
<u>Blood Urea (mg/dL)</u>	<u>P.falciparum</u>	<u>P.vivax</u>	<u>Total</u>
<40	10 7.9%	117 92.1%	<b>127</b> <b>100.0%</b>
>40	5 15.2%	28 84.8%	<b>33</b> <b>100.0%</b>
<b>Total</b>	<b>15</b> <b>9.4%</b>	<b>145</b> <b>90.6%</b>	<b>160</b> <b>100.0%</b>
$\chi^2=0.163$ ; $p>0.05$			

**Table 4:** Table showing the incidence of deranged liver functions in P.vivax and P.falciparum:-

<u>Serum Bilirubin (mg/dL)</u>	<u>P.falciparum</u>	<u>P.vivax</u>	<u>Total</u>
0.2-1.0	6 7.7%	72 92.3%	<b>78</b> <b>100.0%</b>
>1.0	9 11.0%	73 89.0%	<b>82</b> <b>100.0%</b>
<b>Total</b>	<b>15</b> <b>9.4%</b>	<b>145</b> <b>90.6%</b>	<b>160</b> <b>100.0%</b>

 $\chi^2=0.507$ ;  $p>0.05$

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