ORIGINAL ARTICLE

PLATELET COUNT IN MALARIA PATIENTS

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Background: Malaria is a major health problem in the tropics with high morbidity and mortality. This study is conducted to analyse the effects of malaria on platelets. **Methods:** This prospective study was conducted on 200 diagnosed cases of malaria in Department of Medicine, LUMHS, Jamshoro/Hyderabad from February to December 2010. The diagnosis of malaria was carried out by thin and thick blood films. Platelet count was performed using an automated counter. Thrombocytopenia was classified as mild ($50–150\times10^3$ cells/µl), moderate ($20–50\times10^3$ cells/µl) and severe ($<20\times10^3$ cell/µl). **Results:** The age of patients ranged from 16 to 80 (28±10.5) years, median age was 30 years. Among the study population, males were 124 (62%) and females were 76 (38%). Out of them 105 (52.5%) were cases of *Plasmodium falciparum*, 93 (46.5%) were of *P. vivax*, and 2 (1%) were of *P. malaria*. The data showed that 171 (85.5%) patients were having low platelet count; 141 (70.5%) had mild, 21 (10.5%) moderate, and 9 (4.5%) had severe thrombocytopenia. Twenty-nine (14.5%) patients had normal platelet count. **Conclusion:** Malaria is associated with different degrees of low platelet count with rarely increased bleeding tendency.

Keywords: Malaria, Falciparum, Platelets

INTRODUCTION

Malaria is a major health problem in the tropics with a high morbidity and mortality. It is an endemic disease in Pakistan. Being one of the world's biggest killers, it accounts for approximately one million deaths each year.[1] Considering it to be a complex disease it is required to look at it from every possible angle, focusing on vaccine and basic research. A variety of abnormalities of blood and bone marrow cells may be found in *P. falciparum* and *P. vivax* malaria. Severe anaemia may occur in children with acute or chronic *falciparum* malaria with various degrees of parasitaemia. The possible pathogenesis of the haematological abnormalities may be parasite products, T-cell-derived cytokines, macrophage activation, macrophage-derived factors such as tumour necrosis factor-α, and macrophage dysfunction.[2]

Platelets play a critical role in the pathogenesis of malarial infections by encouraging the sequestration of infected red blood cells within the cerebral vasculature. Platelets also have well-established roles in innate protection against microbial infections. Inhibition of platelet function by aspirin and other platelet inhibitors inhibited the lethal effect of human platelets which they exert on *P. falciparum* parasites. Human platelets bind malarial-infected red cells and kill the parasite within; these indicate a protective function of platelets in the early stages of erythrocytic infection distinct from their role in cerebral malaria.[3] Thrombocytopenia frequently complicates malarial infection with *Plasmodium falciparum*. *Plasmodium vivax* also showed moderate thrombocytopenia. *Plasmodium vivax* is the most widespread Plasmodium species being responsible for up to 390 million clinical cases each year.[4] For long, *P. vivax* infection was considered a benign and self-limiting disease. Studies from Indonesia, Papua New Guinea and India have, however, highlighted the association between vivax malaria and life-threatening manifestations.[5] Children with severe malaria who had a platelet count of less than 100,000/ L were 6.3 times more likely to die than those with a platelet count of above 100,000/ L.[6] Those with malarial retinopathy were more thrombocytopenic than those without. Although absence of thrombocytopenia is uncommon in malaria, its presence is not a distinguishing feature between its types. It is a general consensus that thrombocytopenia is very common in malaria.[7] Considering the common occurrence of thrombocytopenia in various types of malaria, this study was conducted to assess frequency of low platelet count in patients suffering from malaria in our setup.

MATERIAL AND METHODS

This prospective, descriptive and analytical study was conducted on 200 diagnosed cases of malaria in department of medicine LUMHS Jamshoro/Hyderabad from February to December 2010. Special case sheets were prepared. Patient's history, including identity of patient, age, sex, address and clinical examination was recorded. Investigations were conducted to look for malarial parasite, its type and platelet count in all the patients presenting with suspicious of malaria. Exclusion criteria were chronic liver disease, thrombocytopenia due to drug intake, bleeding disorder or other conditions which can cause thrombocytopenia. The diagnosis of malaria was carried out by thin and thick blood films. Platelet count was performed using an automated Counter and blood smear was seen by

pathologist. Thrombocytopenia was classified as mild ($50–150\times10^3$ cells/μl), moderate ($20–50\times10^3$ cells/μl) and severe ($<20\times10^3$ cell/μl).[8] Data was analysed using SPSS-10.

RESULTS

In this study the age of patients ranged between 16 and 80 years. The mean age was 28±10.5 years, and median was 30 years. The males were 124 (62%) and females were 76 (38%). Most of the patients (156, 78%) were from rural areas of Sindh. Average stay of patients in the ward was about 5.7 days.

There were 105 (52.5%) cases of *P. falciparum*, 93 (46.5%) cases were of *P. vivax*, and 2 (1%) cases were of *P. malaria*. A total of 171 (85.5%) patients were having low platelet count. The mean platelet count was $77.13\pm54.71\times10^3$/μl, range ($5.5–145.5\times10^3$/μl).

Mild thrombocytopenia was seen in 141 (70.5%) cases, mean platelet count was $90\pm10.5\times10^3$/μl with a range between 52.5 and 145.5×10^3/μl). Twenty-one (10.5%) patients had moderate thrombocytopenia, with a mean count of $35.5\pm9.5\times10^3$/μl, and range between 26 and 45×10^3/μl). Nine (4.5%) had severe thrombocytopenia, with mean count $9.5\pm4.0\times10^3$/μl and range between 5.5 and 13.5×10^3/μl). Twenty-nine (14.5%) patients had normal platelet count. (Table-1)

Mild increased bleeding tendency was found in two patients with severe thrombocytopenia. Platelet count increased to normal range in three to five days in majority of cases. One patient died due to cerebral malaria.

DISCUSSION

This study revealed that 85.5% (171) patients with malaria had low platelet count ($p<0.01$). One hundred and forty-one (70.5%) patients had mild thrombocytopenia, 21 (10.5%) had moderate and 9 (4.5%) had severe thrombocytopenia, while 29 (14.5%) patients had normal platelet count. A study carried out in Pakistan showed that overall, 87.27% of malaria patients were found to have low platelet count.[9] This is comparable to our results. Malaria is usually associated with various degrees of thrombocytopenia.[10] One study has reported that 58% patients with malaria have thrombocytopenia which is lower than our study. Another study on *P. vivax* has reported that 51 (89%) had thrombocytopenia, 39 (68.4%) had mild thrombocytopenia, 10 (17.5%) had moderate, and 2 (3.5%) had severe thrombocytopenia and 6 patients (10.5%) had normal platelet count.[11]

High incidence of thrombocytopenia was a common haematological finding in patients with *Plasmodium vivax* infection. The presence of thrombocytopenia in a patient with acute febrile illness increases the probability of malarial infection in endemic areas and may increase suspicion of malaria in settings where technical laboratory support is not available. A study carried out in Pakistan has shown that out of 370 cases, 114 (30.81%) had normal platelet counts, and 256 (69.18%) had thrombocytopenia ($p<0.05$).[12] Thrombocytopenia has been seen commonly in all forms of malaria.[13] Different mechanisms have been proposed as immune mediated mechanisms including immune destruction of circulating platelets, splenic pooling, and reduced platelet lifespan.

Cerebral malaria is a major complication of *falciparum* malaria in children. In a study from Kenya 33.6% hospital admitted were reported malaria, and out of these 47.6% had neurologic symptoms.[14] *Plasmodium falciparum* infects red blood cells. Once infected, red blood cells activate platelets, which secrete platelet factor 4, which activates the immune system by turning on monocytes which contribute to the inflammation in the blood vessels that leads to obstructions in the brain vessels causing brain damage similar to that seen with a stroke.[15] Cerebral malaria is a fatal form of malaria that can damage the brain and is extremely difficult to treat. New research reveals that platelets stimulate the immune system and turns on molecules that increase inflammation. Cerebral malaria is characterised by the sequestration of *P. falciparum* infected erythrocytes in the capillaries and postcapillary venules of the brain. Cerebral sequestration is a consequence of adhesion of infected erythrocytes to activated endothelium, with one of the most important adhesive molecules on the infected erythrocytes surface. This adhesive molecule is *P. falciparum* erythrocyte membrane protein 1. It has been found that platelets could bridge the interaction of infected erythrocytes with endothelial cells.[16] It has been observed that frequencies of plasma circulating micro-particles were also markedly increased in *P. vivax* patients, as compared to healthy age-matched malaria-unexposed controls. The platelet derived micro-particles increased in a linear fashion with the presence of fever and length of acute symptoms.[17]

Table-1: Platelet count in patients of malaria

Platelet Count	No. of Patients	Percentage	Mean platelet count $\times10^3$/μl	Range $\times10^3$/μl
Normal	29	14.5	230.22±35.2	152–450
Mild	141	70.5	90±10.5	52.5–145.5
Moderate	21	10.5	35.5±9.5	26– 45
Severe	9	4.5	9.5±4.0	5.5–13.5
Thrombocytopenia	171	85.5	77.13±54.71	5.5–145.5

CONCLUSION

Most of the malaria patients in our setup have thrombocytopenia but it is benign in nature and improves in uncomplicated cases without a need of platelet transfusions.

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