

## **Research Article**

### **Clinical, Hematological and Coagulation Profile in Malaria**

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**Abstract:** The Objective of the study was to determine the morbidity and mortality in patients with a diagnosis of malaria with altered hematological and coagulation parameters. 100 patients of Malaria confirmed by PS.MPQBC or Antigen Assay underwent detailed clinical history, through physical examinations and investigated with haematological and coagulation parameters. This was followed by monitoring the outcome of patients with respect to morbidity and mortality. Of the 100 patients 14 patients had severe anemia (Hb % <6gm %) and all of these patients were falciparum, mixed infection and one case was Pl. Vivax infection. Thrombocytopenia was observed in 63% of the patients and severe thrombocytopenia (<50,000 cumm) was seen in 5% of the patients. PT and APTT was increased in 21% and 31% of the cases respectively. BT was increased in 5% of the cases. Increased BT is associated with high mortality. Severe anemia is a poor prognostic factor and adverse outcome. Thrombocytopenia, increased in PT, apt does not have any correlation to mortality. Mixed infection behaves like falciparum malaria.

**Keywords:** Malaria; Plasmodium falciparum; Severe Anemia; Thrombocytopenia

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#### **INTRODUCTION**

Malaria is a protozoan disease caused by Plasmodium species (*P. falciparum*, *P. Vivax*, *P. ovale*, *P. malaria*, *P. knowlesi*) which is transmitted by the bite of infected female *Anopheles* mosquitoes occurs through the tropics and sub tropics at altitudes below 1500 metres [1]. It is found all over the world from 40 degree south to 60 degree north [2]. Malaria is a major health problem in India with mortality due to malaria annually in India is about 200,000 [3]. In the last few decades efforts has been made to produce an effective malarial vaccine. These are still at developmental stages[4]. Even with all these efforts the malaria affects almost all the organs of the body. But one of the chief components affected is blood. So, this work puts in an effort to correlate the changes in blood.

#### **MATERIALS AND METHODS**

We undertook a prospective study comprising 100 patients with fever who were proven to have malaria either by MPQBC. Peripheral smear and following Antigen detection sero diagnostic test.

- a. Histidine rich protein 2 for Plasmodium falciparum.
- b. pLDH for any Plasmodium species

After a detailed history and clinical examination these people underwent complete hemogram. BT. PT. APTT. Renal function test and chest x-ray then the patients were treated with anti-malarials and other supportive treatment.

#### **RESULTS**

All the diagnosed cases were aged between 20-70 years with the predominant age group affected 20-40 years, the majority of the population was from rural areas (59%) than the urban areas (41%). The total number of *P. falciparum*, *P. vivax*, mixed infections were 50%, 40% and 10% respectively.

All the patients presented with fever was present in 94% of the patients and chills and rigors were present in 77% of the patients easy fatigability was seen in 44% of the cases most of whom had severe anemia (<6gm/dl). Nausea and vomiting was seen in 21% of the cases. Altered sensorium was seen in 10% of the patients. Only patients with falciparum and mixed infection had these complaints. A typical presentation is very common in the form of abdominal pain arthralgia which was subsided after starting anti malarial therapy.

**Table-1: Prevalence of symptoms Plasmodium species**

Symptoms	<i>P. falciparum</i>	<i>P. vivax</i>	Mixed	Total
Fever	94%	92.5%	100%	94%
Chills and rigor	78%	77.5%	60%	77%
Easy fatigability	46%	40%	50%	44%
Headache	36%	30%	20%	32%
Nausea and vomiting	22%	15%	40%	21%
Cough	4%	0%	0%	2%
Altered Sensorium	14%	0%	30%	10%

**Showing incidence of clinical features**

Splenomegaly is an important sign in malaria which was observed in 53% of the patients, but absence of this does not rule out malaria. 69% patients had Hb% less than 19 gm %. Among the patients who had less than 10gm percentage Hb%.

- 30 patients had Hb 8%-10% gm (mild anemia)
- 25 patients had Hb 6%-8% gm (moderate anemia)

- 14 patients had Hb<6gm% (severe anemia)

Anemia was present in 69% of patients which was more common with *P. falciparum* infection and among them 40 patients had splenomegaly i.e. 58% of the patients with anemia had splenomegaly. Of the total 100 cases 63 patients had thrombocytopenia. 66.6% of the patients with thrombocytopenia had splenomegaly.

**Table-2: Clinical profiles of malaria**

	Pallor	Icterus	Pedal edema	Splenomegaly	Hepatomegaly	CNS Involvement
<i>P. falciparum</i>	90%	24%	12%	62%	24%	16%
<i>P. vivax</i>	62.5%	25%	25%	35%	5%	
Mixed	90%	30%	10%	80%	50%	30%
Total	79%	16%	8%	53%	19%	11%

Leucocytosis was predominant in present study. Monocytosis was observed in 15% of the patients which was a good predictor of effectiveness of antimalarial therapy. PCV was <20 in 14% of the patients with predominantly *P. falciparum* infection (20%) and 10% with mixed infection, thus indicating the degree of anemia and higher rate of destruction of RBC's

associated with falciparum malaria. PT AND APTT was increased in 21% & 13% of the cases respectively which was predominantly in falciparum and mixed infections, but this does not result in spontaneous bleeding manifestation which is an indicator of poor prognosis.

**Table-3: Hematological profiles of malaria**

	<i>P. falciparum</i>	<i>P. vivax</i>	Mixed	Total
Lymphocytosis	60%	37.5%	30%	48%
Lymphopenia	20%		10%	11%
Neutrophilia	34%	25%	40%	31%
Neutrocytopenia	24%	12.5%	10%	18%
Eosiniphilia		10%	10%	5%
Monocytosis	16%	15%	10%	15%

**DISCUSSION**

In our study the male to female ratio was 3:76:1 and compared to Bhakshi *et al.*[5], the males were affected were more in our study [5]. The incidence of malaria was more in men than in women due to the working

pattern i.e men are exposed to mosquito bites outdoors whereas females are less exposed. Our study follows the age pyramid where the base is formed by young people and apex by the older age who constitute lesser percentage of the population

**Table-4: Prevalence of incidence of malaria**

Age	Malhotra <i>et al.</i> [6]	Present Study
20-30	36%	46 %
31-49	60%	44%
>50	4%	10%

Fever was the predominant complaint in our study i.e is 94% of our patients presented with the fever of 77% of the patients had chills and rigors. In the study conducted by Mehta et al fever was presented in 100% of patients and 96% in the studies conducted by Malhotra *et al.* [6] and naval hospital respectively. It was also noted that 44% of our patients in our study had easily fatigability as their presenting complaint. There was no mention of regarding in any other studies. Vomiting was observed in 43.3% in the patients of our study conducted by Mehta and et al [8] it was also seen in 21% and 23% patients in the study conducted by the naval hospital cough and breathlessness was presenting complaint in 4.47% of the patients by study of Mehta *et al.* and the symptoms were noticed in 2% of the patients in our study [8]. The higher incidence of these symptoms may due to higher number of falciparum cases compared to other areas where vivax malaria was predominant. It also signifies that the number of complicated malaria cases were more in our area than in other studies. The number of patients presenting were altered in sensorium was seen in 50% of the cases in the study conducted in the study by Malhotra where as in our study it was only 10%. The higher incidence in their study was due to fact that their study was conducted in patients with complicated malaria only [6].

Pallor was presented in 79% of the patients in the study carried out by Malhotra [6], it was noticed 79% in our study. The incidence of pallor was more in the patients with falciparum and mixed infection which was 90%. Pallor was present in vivax malaria in our study. It co-relates with the study by Sharma [10]. Leteures was noted in 16% of the patients in our study where as it was seen in 25% of patients by Malhotra and it was seen in 46% by Nand Splenomegaly was seen in 53% of the patients in the present study where as rates were observed in a study by Murthy where the percentage of the patients with splenomegaly was 50% high incidence of splenomegaly was noted in a study conducted by Ram it was 88.75% in their study comparatively high incidence of 60% was also observed by Nand. Hepatomegaly was noted in 19% of the patients in the present study. Studies by Ram and Murthy [13] have shown an higher incidence of hepatomegaly in their work. It might be due to the fact that their study mainly concentrated on the subject such as malarial hepatitis and jaundice in malaria. The incidence in these studies was 79.5% and 91% in Ram and Murthy respectively where as in another study by Nand the incidence was 13.3% which was comparable to our study.

Coma seizures or altered sensorium was observed among 10% of the patients in our study. It was noted only in patients with pl.falciparum or mixed infection had these symptoms. The study by Malhotra also had similar observation where the CNS involvement was

noted in 12.5% of the patients this signifies that cerebral malaria can be caused only by pl.falciparum.

In the present study the percentage of falciparum malaria was 50% and the incidence of vivax and mixed infection was 40% and 10% respectively. In a study by Rajanasthein [14] the prevalence of falciparum was 76.2% where as vivax malaria was just 23.8%. In a study by Reddy *et al.* [15] there was high incidence of vivax malaria i.e 61.2% and falciparum being 36.8%. In another study conducted by Bhakshi *et al.* [5] the incidence of falciparum, vivax and mixed infection was 60%, 35% and 5% respectively. From these observations we can conclude that incidence of particular species varies with geographical area, the area where we have conducted the study is known to be endemic for falciparum and hence the higher incidence is noted in my study.

Anemia was present in 69% of the patients in our study, the incidence of severe anemia (hb > 6gm%) was seen in 14% and it was comparable to study done by Mehta et al with incidence of 18% [9]. The overall incidence of anemia was higher in studies conducted by Sharma et al where the incidence was 86.7%. The higher incidence could be explained by the fact that their study involved with cases of falciparum malaria only. If we consider only falciparum cases even our study showed an incidence of 80%. Out of the 69 patients who had anemia only 49 patients had splenomegaly this indicates that there are other factors other than splenic sequestration which could lead anemia.

Leucocytosis was seen 11% of the total patients in our study. Similar observations were made in a study conducted by Sharma SK *et al.* [10] where the incidence of leucocytosis was 13.3%. In our study 14% of the patients with falciparum malaria had leucocytosis and it was comparable to the study by Sharma SK *et al.* [10]. All the patients who had leucocytosis had neutrophilia which indicates superadded bacterial infection.

Monocytosis was observed in 15% of patients in our study. It was observed N.K.D. Hakin *et al.* [19] in their study that monocytosis in patients especially those on antimalarial therapy may be an indicative of an anti malarial effect by monocytes, thus monocytosis may enhance predisposition to a favorable outcome. Eosinophilia was observed in 5% of the cases in our study. Thrombocytopenia was present in 53% of the cases in the present study. In a study by Sharma SK *et al.* [10] observed that 70% of the patients had thrombocytopenia. Thrombocytopenia was present in 80% of the cases with falciparum malaria in our study. In our study 57.6% of the patients with vivax malaria had thrombocytopenia.

In our study only 54 patients out of 63 had splenomegaly. It can be observed that only 66.6% of the patients with thrombocytopenia had splenomegaly. So hereby we can conclude that splenic sequestration is not only the cause of thrombocytopenia other causes such as immune mediated platelet destruction also play a role.

Increased ESR was seen in 56% of the patients in total number of cases. In patients with *Pl. falciparum* malaria elevated ESR was seen in 72% cases. This was comparable to the study by Bakshi *et al.* [5].

In our study 57% of the patients had normocytic normochromic blood picture. It was comparable to a study by Sen *et al* where half the patients had normocytic normochromic blood picture [19]. In our study 25% of the patients had microcytic hypochromic blood picture which was comparable to a study by a same *et al* [24] in their study also had microcytic hypochromic blood picture in 20% of the cases. In our study prevalence of dimorphic anemia was seen in 18% of the cases similar results were also observed by Sen where the prevalence of dimorphic anemia was 20% in their study.

## CONCLUSION

The incidence is higher in males than females with peak in 3<sup>rd</sup> and 4<sup>th</sup> decade. Fever is the presenting complaint in almost all cases. Easy fatigability indicates severe anemia in malaria. A typical presentation is very common in the form of abdominal pain, arthralgia which was subsided after starting anti malarial therapy. Splenomegaly is an important sign in malaria, but absence of this does not rule out malaria. Anemia is the most common hematological abnormality. Thrombocytopenia is very common in malaria, but spontaneous bleeding is rare. The higher incidence of *Pl Falciparum* in this study is because these areas are endemic for *Pl. Falciparum* infections. PT and APTT were prolonged in some cases predominantly in *falciparum* and mixed infections, but this does not result in spontaneous bleeding. BT was prolonged in 5% of the cases, most of them had spontaneous bleeding. It is also indicator of poor prognosis. Severe anemia is poor prognostic factor and it increased the duration of hospital stay and even mortality. Mixed infections behave like *falciparum* malaria but its incidence and severity is less than severe *Pl Falciparum* malaria. In mixed infection, *Pl. Vivax* malaria has a protective role against severity of *Falciparum* malaria. Use of antibiotics along with antimalarial has shown better response in patients with malaria indicating the prevalence of superadded bacterial infection.

## REFERENCES

1. WHO, WHO Expert committee on Malaria Twentieth report. 1998. Geneva. Switzerland 2000

2. Murray CJL, Lopez AD; Evidence based health policy – Lessons from the Global Burden of Disease Study Science 1996; 274:740-743.
3. Chatterjee KD; Subphylum Sporozoa Genus Plasmodium Parasitology in relation to clinical medicine 12<sup>th</sup> edition, 1980;70-100.
4. Park K; Malaria Text book of preventive and social medicine. 17<sup>th</sup>Edn. Page 192-201.
5. Bhakshin; Hematological manifestation of Malaria; Indian Journal of Hematology and blood transfusion, 1997;15-40.
6. Malhotra B; A study of clinical and hematological manifestations of malaria. Indian journal of hematology and blood transfusion, 1997;15:40
7. Naval Hospital; navy medical department; Guide to Malaria Prevention and Control; 2001: chapter 3.
8. Mehta; Clinical pattern of epidemics in Rajasthan; Journal of Physicians of India, 2001;48:211-15.
9. Mehta SR; *Falciparum* Malaria – 210 cases JADI, 2001;2:119-120
10. Sharma SK, Das RK, Das BK, Das PK; Hematological and coagulation profile in *Al. falciparum* malaria; JAPI, 1992;40:581-583.
11. Nand; Renal dysfunction in Malaria. JAPI, 1999; 47(1):103
12. Rom study of jaundice in malaria; JAPI, 2002; 50 – 54.
13. Murthy; Malarial hepatitis- Does such a clinical work exist: Journal of Am and association of physician of india, 1999; 47:1:27.
14. Rajanasthein; Haematological and coagulation studies in Malaria; journal of Medical association of Thailand, 1992;75(supp17):190-194.
15. Reddy DS; A study of *falciparum* malaria in emergency medicine department; Indian journal of haematology, Blood transfusion; 1995; 135(1):38-86.
16. Mehta SR ; *falciparum* malaria -210 cases JADI, 2001; 2:119-120.
17. Sen R, Tewari AD, Sehgal PK, Singh U, Sikka R, Sen J; Clinico-haematological profile in acute and chronic *Plasmodium falciparum* malaria in children. Journal of communicable disease, 1994; 26(1): 31-38.
18. Roy S; haematological profile in patient with acute *falciparum* malaria; JAPI, 2002 poster presentation no. 114.
19. Hakin NKD; Monocytosis in acute malarial infection Nigerian journal of clinical practice, 2002; 5(2):106-108.
20. Ladhani, Brett lowe, Andrew cole, Ken K, Charles JCN; Changes in white blood cell a platelet in children with *falciparum* malaria: relationship to disease outcome. Br. Jr. of haematol, 2002; 119:839-847.

21. Horstmann RD; Malaria induced thrombocytopenia; BLUT 1981; 421(3); 157.
22. Wheatherall; Importance of anaemia in cerebral and un complivated malaria; role of complications dyserythropoiesis and; quarterly journal of medicine 1986; 58; 305-323.
23. Clemens R, Pramoolsinsap C, Lorenz R, Pukrittayakamee S, Bock HL, White NJ; Activation of the coagulation cascade in severe falciparum malaria through the intrinsic pathway. Br J Haematol. 1994; 87(1):100-5.