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RESEARCH ARTICLE

COMPARATIVE STUDY OF CLINICAL AND LABORATORY PROFILE IN SPECIES SPECIFIC MALARIAL INFECTION: OBSERVATIONS FROM A TERTIARY CARE HOSPITAL FROM AN ENDEMIC REGION OF INDIA

S Abraham[1], G Bhandary[1], R Antony[1], M Pais[1], S Suresh[2], P L Palatty[3], RP Jakribettu[4], MS Baliga[1]

[1]*Father Muller Research Centre, Kankanady, Mangalore 575002, Karnataka, India*
[2]*Department of Community Medicine, Father Muller Medical College Hospital, Kankanady, Mangalore 575002, Karnataka, India*
[3]*Department of Pharmacology,Father Muller Medical College Hospital, Kankanady, Mangalore 575002, Karnataka, India*
[4]*Department of Microbiology,Father Muller Medical College Hospital, Kankanady, Mangalore 575002, Karnataka, India*

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Abstract: Background: Malaria continues to be an issue of public importance in Indian subcontinent. This study was conducted to observe the difference in hematological and hepato-renal profile in malaria patients affected by *Plasmodium falciparum, Plasmodium vivax* and both the parasites admitted in the tertiary care hospital. As far as the authors are aware of this is the first study that attempted at understanding the clinical and laboratory profile to ascertain the difference in the clinic-pathogenesis from a malaria endemic region of India. **Methods:** This was a retrospective observational study conducted at a tertiary care hospital between January 2016 to July 2017. All patients aged above 18 years diagnosed with malaria were included in the study. The demographic, clinical and laboratory parameters were entered in Microsoft excel and subjected to statistical analysis. **Results:** A total of 262 patients were diagnosed with malaria during the study period. Majority of the patients were male (208, 79.39%) and patients of age between 18-30 years accounted for 82 (31.3%). Among the 262 patients, 93 (35.5%) were diagnosed with *Plasmodium vivax*, 85 (32.4%) mixed (*P.vivax* and *P. falciparum*) and 84 (32%) *Plasmodium falciparum* infection. In the present study, anemia, leucopenia and thrombocytopenia were seen among hematological parameters. The liver enzymes, total bilirubin were raised and total protein, S.albumin were reduced among the hepatic profile and raised Blood urea, S.creatinine and dyselectrolytemia were seen among the renal profile in the malaria patients. **Conclusion:** Malaria is known to cause heamato-hepato-renal dysfunction. In Mixed malaria, anemia and deranged hepato-renal parameters; in falciparum patients, lymphocytopenia, thrombocytopenia, and raised ALT; and in vivax patients, deranged granulocytes and various RBC volume were seen.

KEYWORDS: malaria, hematology, hepatic, renal, vivax, falciparum, mixed malaria

Corresponding Author :
Dr. Ramakrishna Pai Jakribettu,
Professor and Head/ Dean Academics
Department of Microbiology , Andaman and Nicobar Islands Institute of Medical Sciences, Port Blair, A&N Islands, India – 744101.

INTRODUCTION:

Malaria, a protozoan infection, is one of the acute febrile illnesses endemics in tropical region. It is caused by the parasite of the genus *Plasmodium* and transmitted by the bite of the infected female *Anopheles* mosquito. The causative agents are *Plasmodium falciparum, P.vivax, P. malariae* and *P.ovale.* The fifth species, *P.knowlesi*, is an emerging zoonotic malarial parasite in Malaysia [1]. In 2018, it was estimated that around 228 million cases of malaria occurred globally [2]. Among the 6 WHO regions, South East Asia region reported the second highest cases with 3.4%, while the African region leading with 93%. In South East Region reported 50% of *P. falciparum* and 53% of *P.vivax*, worldwide cases[2]. In India, *P.vivax* contributes for the majority of the cases, *P.falciparum* reported to cause more morbidity and mortality [3].

As per the WHO estimate, 405,000 deaths occur globally due to malaria [2]. The prompt diagnosis and early treatment is the only way to prevent morbidity and mortality from malaria. The diagnosis is done by microscopic examination of peripheral blood, quantitative buff coat (QBC) or by enzyme immunoassay to detect Parasite Lactate Dehydrogenase (pLDH) enzyme and Pf Histidine-rich–protein-2 (PfHRP-2) to identify *P.vivax* and *P.falciparum*, respectively[4]. These diagnostic tests can help to prevent complications due to delay in treatment. The major organ systems affected by malaria are mainly reticulo-endothelial system, hepatic, renal and cerebral.

As Malaria mainly affects the reticulo-endothelial system, the pathophysiological changes seen are mainly in hematological and biochemical parameters among the infected individuals. The parasite infects the RBCs and also damages the endothelium, causing anemia, thrombocytopenia and even disseminated intravascular coagulation[5]. Various cytokines like TNF-α and Interferon-α, has been observed to cause increase in selectins and other chemo-attractant chemicals from the leucocytes leading to hematological changes. *Plasmodium* also infects the liver parenchyma causing blockage in liver sinuses, congestion and inflammation. The inflammation in hepatocytes releases the liver enzymes into the serum, causing raised liver enzymes mainly transaminases and alkaline phosphatase [6]. The lysis of infected and non-infected RBCs, especially in *P.falciparum* and liver damage causes increase in unconjugated bilirubin and jaundice [6]. Similarly, acute renal failure is other complication seen in both *P.falciparum* (80.9%) and *P.vivax* (11.7%) [7].The factors responsible for development of ARF may be volume depletion, hypotension, jaundice, heavy parasitemia leading to intravascular hemolysis, and DIC [7].As far as the authors are aware of this is a first of its kind study in the literature, where clinical, hematological and hepato-renal profile of patients diagnosed with malaria i.e., vivax, falciparum and mixed are compared with age and gender matched controls.

MATERIALS AND METHODS:

Study setting: This was a retrospective observational study and was conducted at the Clinical Microbiology and Medicine department of a tertiary care teaching hospital , after approval from the Institutional ethics committee.

Inclusion criteria: A total of 262 patients above 18 years of age, who were diagnosed with malaria by peripheral blood smear stained with Geimsa stain or QBC test from January 2016 to July 2017, were included in the study.

Exclusion criteria: The patients diagnosed with other febrile illness like dengue, leptospirosis, typhoid, tuberculosis and patients with history of systemic illness or medication which affect renal and hepatic dysfunction were excluded from the study. For the control group, seventy age and gender matched individuals, without any illness, who had visited hospital for the health checkup, were included in the study. The demographic, clinical and all the laboratory parameters were collected from the medical records of patients and included in the study.

Statistical analysis: The data collected were entered in Microsoft Excel and statistical analysis was done.

The mean, standard deviation were done for all the parameters and compared with control cases, using ANOVA /Kruskal wallis test. The p value < 0.05 were considered as significant.

RESULTS:

A total of 262 malaria patients were diagnosed with malaria during the study period. Majority of the patients were male (208, 79.39%) and patients of age between 18-30 years accounted for 82 (31.3%). Almost equal distribution was observed among the three types of infection. Ninety three (35.5%) patients were infected with *Plasmodium vivax*, 85 (32.4%) mixed (*P.vivax* and *P.falciparum*) malaria and 84 (32%) *P.falciparum*. Seventy age and gender matched healthy individuals were included as control. The gender and age wise distribution of the study population is presented in the **Table 1**. All patients presented with fever with chills. The second most common presentation was vomiting (94, 35.9%) followed by headache (77, 29.4%) (**Table 1**).

Table 1. Demographic details of the study population.

Parameters	Categories	Vivax (n=93)	Mixed (n=85)	Falciparum (n=84)	Total (n=262)
Gender	Female	26 (27.96)	17(20.00)	11(13.10)	54(20.61)
	Male	67(72.04)	68(80.00)	73(86.90)	208(79.39)
Age wise (in years)	18 - 30	31(33.33)	37(43.53)	14(16.67)	82(31.30)
	31 - 45	27(29.03)	21(24.71)	26(30.95)	74(28.24)
	46 - 60	20(21.51)	20(23.53)	33(39.29)	73(27.86)
	>61	15(16.13)	7(8.24)	11(13.10)	33(12.60)
Symptoms	Fever	93(100)	85(100)	84(100)	262(100)
	Vomiting	26(27.96)	33(38.82)	35(41.67)	94(35.87)
	Headache	13(13.98)	36(42.35)	28(33.33)	77(29.38)
	Generalized weakness and body ache	14(15.06)	35(41.18)	8(9.52)	57(21.75)
	Cough	8(8.6)	22(25.88)	17(20.24)	47(17.93)
	Abdominal Pain	2(2.15)	11(12.94)	7(8.33)	20(7.63)

The subjects were divided into four groups, namely, healthy individuals as control; patients diagnosed as vivax malaria; falciparum malaria and mixed malaria. The comparison of the hematological parameters with control group revealed that there was significant leucopenia, lymphocytopenia, eosinopenia, thrombocytopenia, neutrophilia, monocytosis, among each malarias sub group patients. No significant difference was seen with hemoglobin, packed cell volume and RBC parameters (**Table 2**). When compared among the malaria patients, lowest hemoglobin, lymphocytes, platelet counts and raised monocytes, MCHC was seen in mixed malaria; lowest total leucocyte count was seen in falciparum group; raised neutrophils, MCV, MCH and lowest eosinophil, PCV in vivax group.

Table 2. Comparison of haematological parameters among the study population

Blood Parameters	Group	Mean± Std. Dev	Median(IQR)	Control vs All	ANOVA Kruskall wallis test
Haemoglobin (g%)	Control	12.88±1.71	13(11.88-13.9)		0.973
	Vivax	12.97±1.86	13.1(11.83-14.18)		
	Falciparum	12.97±2.22	13.1(11.7-14.4)		
	Mixed	12.84±2.23	13.2(11.8-14.2)		
Total leucocyte count (/mm^3)	Control	8452.86±2030.12	8000(6800-9700)		0.0001
	Vivax	6133.72±1979.46	5950(4700-7525)	< 0.001	
	Falciparum	5729.49±2250.37	5450(4300-6600)	< 0.001	
	Mixed	6071.6±2187.25	5900(4500-7200)	< 0.001	
Neutrophil (%)	Control	55.77±10.47	56(49.25-62.75)		0.0001
	Vivax	68.76±11.88	71(60-78)	< 0.001	
	Falciparum	68.45±11.76	70.5(62.75-77)	< 0.001	

	Mixed	67.54±11.68	69(62-74)	< 0.0001	
Lymphocyte (%)	Control	36.34±9.07	38(38(28.75-43)		< 0.0001
	Vivax	21.22±11.59	19(13-28)	< 0.0001	
	Falciparum	21.87±12.24	19(13.75-27)	< 0.0001	
	Mixed	20.31±9.97	18(13.5-26)	< 0.0001	
Eosinophil (%)	Control	5.99±4.21	5(3-8)		< 0.0001
	Vivax	1.77±2.71	1(1-1)	< 0.0001	
	Falciparum	1.95±2.39	1(1-2)	< 0.0001	
	Mixed	1.88±1.89	1(1-2)	< 0.0001	
Monocyte (%)	Control	1.9±0.42	2(2-2)		0.0001
	Vivax	8.66±4.07	10(6-12)	< 0.0001	
	Falciparum	8.47±4.24	10(5-12)	< 0.0001	
	Mixed	9.27±3.86	10(6.5-12)	< 0.0001	
Platelet (/mm^3)	Control	244557.14±65190.39	246500(191750-292500)		< 0.0001
	Vivax	100707.87±52019.49	88000(61000-134500)	< 0.0001	
	Falciparum	109273.81±85783.61	92500(55750-126000)	< 0.0001	
	Mixed	92009.64±48659.97	80000(54000-128000)	< 0.0001	
PCV	Control	40.1±4.05	40.5(36.88-43)		0.37
	Vivax	38.67±5.92	38.8(34.9-43)		
	Falciparum	38.71±6.81	39(35.4-43.3)		
	Mixed	38.69±5.82	39.3(35.55-42.65)		
MCV	Control	83.8±5.67	83.6(80.7-88.7)		0.44
	Vivax	86.07±6.88	86.5(81.9-90)		
	Falciparum	84.85±7.63	84.9(81.15-88.9)		
	Mixed	84.78±7.63	85.55(81.4-88.33)		
MCH	Control	27.88±2.1	28(26.8-29.1)		0.50
	Vivax	28.71±2.63	29(27.1-30)		
	Falciparum	28.31±3.04	28.5(26.65-29.95)		
	Mixed	28.43±2.73	28.7(27.08-30.23)		
MCHC	Control	33.33±0.87	33.3(32.9-33.8)		0.18
	Vivax	33.43±0.71	33.5(33.1-33.9)		
	Falciparum	33.64±1.09	34.1(33.4-34.3)		
	Mixed	33.75±0.63	33.9(33.45-34.2)		

The liver function parameters were deranged in malaria group compared to control. The liver enzymes (AST, ALT), total bilirubin were raised and total protein, S.albumin were reduced significantly in malaria groups compared to control. When compared among the malaria subgroups, highest AST, AST/ALT ratio and reduced total proteins, S. albumin, Albumin/ globulin ratio was seen in mixed malaria group; ALT was highest in falciparum group; highest total bilirubin, S. globulin seen in Vivax group (**Table 3**).

Table 3. Comparison of Hepatic parameters among the study population

Liver Parameters	Group	Mean± Std. Dev	Median(IQR)	Control vs All	ANOVA Kruskall wallis test
AST (IU/ml)	Control	18.13±8.94	16(14-18)		< 0.0001
	Vivax	52.8±95.14	27(21-44)	0.283	
	Falciparum	57.39±41.02	44(30-67)	0.138	
	Mixed	75.46±115.77	34(23.5-62)	0.014	
ALT (IU/ml)	Control	18.58±10.74	15(11-22)		< 0.0001
	Vivax	50.83±80.38	28(20-50)	0.227	
	Falciparum	60.47±65.14	38(26-68)	0.039	
	Mixed	59.83±83.33	40(23-66)	0.081	
AST/ALT	Control	1.09±0.39	1(0.82-1.43)		0.27
	Vivax	1.06±0.4	1(0.78-1.25)		
	Falciparum	1.17±0.51	1.13(0.84-1.38)		
	Mixed	1.27±0.86	1.04(0.77-1.49)		
Total Bilirubin (mg %)	Control	0.72±0.51	0.6(0.37-0.8)		< 0.0001
	Vivax	1.89±1.63	1.43(0.9-2.31)	0.012	
	Falciparum	1.88±2.03	1.24(0.76-1.82)	0.011	
	Mixed	1.85±1.68	1.32(0.99-2.13)	0.032	

Total Protein (g %)	Control	7.32±0.4	7.34(6.93-7.69)		0.0001
	Vivax	6.8±0.62	6.69(6.42-7.29)	0.001	
	Falciparum	6.61±0.62	6.65(6.34-6.98)	< 0.0001	
	Mixed	6.49±0.67	6.49(5.85-6.98)	< 0.0001	
S. Albumin (g %)	Control	4.36±0.35	4.42(4.03-4.6)		< 0.0001
	Vivax	3.69±0.53	3.74(3.36-4.06)	< 0.0001	
	Falciparum	3.72±0.43	3.8(3.43-4.02)	< 0.0001	
	Mixed	3.49±0.53	3.62(3.14-3.72)	< 0.0001	
Globulin (g %)	Control	2.96±0.51	2.97(2.52-3.31)		0.10
	Vivax	3.11±0.38	3.15(2.79-3.38)		
	Falciparum	2.89±0.52	2.9(2.57-3.11)		
	Mixed	3.00±0.5	3.09(2.82-3.25)		
Albumin / Globulin ratio	Control	1.53±0.4	1.44(1.26-1.84)		0.026
	Vivax	1.21±0.24	1.2(1.05-1.35)	0.059	
	Falciparum	1.4±0.85	1.29(1.15-1.47)	< 0.0001	
	Mixed	1.21±0.37	1.16(1.03-1.33)	0.107	

The renal parameters including electrolytes were compared among the four groups. When compared to control group, there was significantly high S.creatinine, blood urea and lowered electrolytes were observed among malaria groups (**Table 4**). Highest S. creatinine, Blood urea was observed in mixed malaria; lowest S.sodium, and lowest S.chloride in Falciparum group and lowest S.potassium in vivax group. Among 262 malaria patients, twenty-one succumbed (12 mixed and 9 falciparum). No death was reported among the vivax group of patients. Death was reported in elderly patients with range of age 32-94 (average 65.04) years compared to 18-94 (average 40.82) years infected.

Table 4. Comparison of renal parameters among the study population

Renal Parameters	Group	Mean± Std. Dev	Median(IQR)	Control vs All	ANOVA Kruskall wallis test
Serum Creatinine (mg %)	Control	0.75±0.17	0.76(0.6-0.9)		< 0.0001
	Vivax	1.09±0.51	1.01(0.88-1.18)	0.119	
	Falciparum	1.06±0.37	1.02(0.85-1.16)	0.177	
	Mixed	1.24±1.15	1.02(0.82-1.21)	0.009	
Blood Urea (mg %)	Control	21.32±7.55	21(15-27)		0.036
	Vivax	30.23±19.82	26(18.5-35.5)	0.182	
	Falciparum	29.02±16.21	24(19-31)	0.340	
	Mixed	34.08±24.15	28(20-37)	0.025	
S.Sodium (mEq/ml)	Control	138.87±1.82	139(137-140)		< 0.0001
	Vivax	135.7±3.89	136(134-137.75)	0.001	
	Falciparum	133.08±4.39	134(130-136)	< 0.0001	
	Mixed	134.64±3.35	135(133-137)	< 0.0001	
S.Potassium (mEq/ml)	Control	4.21±0.32	4.2(3.9-4.47)		< 0.0001
	Vivax	3.74±0.43	3.66(3.44-4.04)	< 0.0001	
	Falciparum	3.8±0.54	3.75(3.44-4.26)	< 0.0001	
	Mixed	3.89±0.41	3.8(3.62-4.15)	0.022	
S.Chloride (mEq/ml)	Control	101.52±2.03	101(99.78-103.3)		< 0.0001
	Vivax	96.47±4.44	97.2(93.8-99)	< 0.0001	
	Falciparum	94.32±4.83	95.1(91.45-97.45)	< 0.0001	
	Mixed	96.62±4.45	96.4(93.6-99.2)	< 0.0001	

DISCUSSION:

Malaria is an important differential diagnosis for acute febrile illness in a patient from a malaria-endemic area [8-10]. Our study confirms that hematologic changes due to malaria are common, as was observed in other

studies [9, 11-13]. Although anemia is a common manifestation of malaria [9,11], our study did not reveal a significant difference in hemoglobin or hematocrit across the various forms of malaria parasites, this can possibly be explained by early diagnosis of the disease, prompt treatment and awareness among the general population [14].

Leukopenia was observed among all patients with malaria, with a more profound decreased in the falciparum species, which was consistent with another study [11]. This can be explained by marginal and splenic sequestration of leukocytes [11,12]. Neutrophilia, lymphocytopenia, eosinopenia and monocytosis was a similar feature across all the subgroups, which were observed in other studies as well [12,13,16,17]. Monocytosis is known to be associated with a favorable clinical outcome [16]. There was no significant difference in the red cell indices as seen in a previous study[5]. Thrombocytopenia is a well-documented manifestation of malarial infections[11]. The changes in myeloid series can be explained due by increased cell lysis, accelerated removal of parasitized cells, bone marrow dysfunction and oxidative stress [11,15].

The elevated hepatic enzymes seen especially with falciparum and mixed malaria are evidence of malarial hepatopathy, consistent with other studies [17,18]. Increase in total bilirubin is attributed to both hemolysis of red blood cells and hepatocyte injury [17]. Hypoalbuminemia was common amongst the malarial patients. Albumin is one of the negative phase reactants and is a serological marker of acute inflammation [19].

Electrolyte disturbance, in the form of hyponatremia and hypokalemia, is a common complication in patients with severe malaria and act as an indicator for the severity of disease [20]. The patho-physiology leading to the development of hyponatremia in patients with malaria is not well established. However, an increased secretion of vasopressin (ADH) from the posterior pituitary, either appropriately or inappropriately, has been found to be the cause of hyponatremia in various studies [21].

Patients affected by from *P. falciparum* malaria are reported to have a higher risk of developing hyponatremia[20,22]. Electrolyte disturbance serves as a marker for severe disease and prompt correction of the same is warranted to prevent the associated complications [23, 24]. Acute kidney injury is hypothesized due to hypovolemia; immune mediated glomerular injury and decreased renal perfusion [25]. Our patient population did not have any evidence of gross renal damage probably due to early diagnosis and prompt treatment.

CONCLUSION:

Malaria is known to cause heamato-hepato-renal dysfunction.These parameters vary depending on type of malaria parasite causing infection. In Mixed malaria, anemia and deranged hepato-renal parameters (lowered Total protein, S.albumin, Alb/glob ratio andraised AST, AST/ALT ratio, Blood urea and S. creatinine); in falciparum patients, lymphocytopenia, thrombocytopenia, and raised ALT; and in vivax patients, deranged granulocytes (neutrophilia, eosinopenia, lowered PCV, MCV, MCH) seen. Limitations of the study, firstly this is a retrospective study conducted in patients admitted in a single center, these findings needs confirmation by multi-centric study with larger study population.

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