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# Study of acute kidney injury in malaria at a tertiary care hospital in northern India

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## Abstract

**Introduction:** Malaria remains one of the major public health problems in the tropics. Acute kidney injury in malaria is characterized by abrupt deterioration in kidney function which clinically manifests as a reversible acute increase in nitrogen waste products, measured by blood urea nitrogen and serum creatinine levels with or without reduced urine output over the course of hours to weeks. AKI has various epidemiological and clinical patterns in countries with different climates and socioeconomic features and, therefore, needs improved individualized approaches in these different regions. AKI is a major cause of mortality in malaria.

**Methods:** The present study was a prospective observational study conducted on 100 adult patients of tropical malaria reported between October 2017 to December 2020 in medicine department at Pt. B. D. Sharma PGIMS Rohtak Haryana. A detailed history and clinical examination was done in all subjects included in the study. Patients who fulfilled case definition criteria were evaluated for AKI as per definitions of KDIGO classification on day of admission and then subsequently on day 3, 7 and 14 along with laboratory investigations i.e. serum creatinine, blood urea, urine output and eGFR.

**Results:** The present study, a total of 100 patients of malaria were included out of which 64 cases due to p. vivax , 28 cases due to p. falciparum and remaining 8 cases due to both. 46 patients had AKI among which 33 were due to p. vivax , 11 due to p. falciparum and remaining 2 due to mixed infections. Among AKI cases, 16 cases were in AKI stage I, 10 cases were in AKI stage II and remaining 20 cases were in AKI stage III. 11 cases required urgent hemodialysis, rest improved with conservative management. 2 patients were died within one month.

**Conclusion:** Early referral of malarial AKI patients to dialysis facility unit and early institution of Haemo-dialysis in complicated malaria is effective in decreasing mortality and is associated with rapid recovery.

**Keywords:** Acute kidney injury, Malaria, Plasmodium vivax, Plasmodium falciparum, Hyperbilirubinemia.

**Introduction**

Malaria remains one of the major public health problems in the tropics. It affects 103 endemic countries with a population of approximately 2.5 billion, causing 1-3 million deaths per year.1 Plasmodium falciparum followed by Plasmodium vivax infections are associated with most cases of complicated malaria. The overall prevalence of acute kidney injury in falciparum malaria is less than 1%, but can go up to 60% in patients with heavy parasitemia.2, Every year during and after the rainy season an epidemic of acute febrile illness is witnessed in northern India, but the relative contribution of various etiological agents remains unknown.

Acute kidney injury in malaria is characterized by abrupt deterioration in kidney function which clinically manifests as a reversible acute increase in nitrogen waste products, measured by blood urea nitrogen and serum creatinine levels with or without reduced urine output over the course of hours to weeks. These can be either related to direct involvement of the kidneys and urinary tract via tubulointerstitial toxicity and injury to glomerular endothelium secondary to immune-complex deposition or activation of complement or indirect consequence of systemic effects of infection i.e., hemolysis, rhabdomyolysis, hypovolemic shock, septic shock and immune complex deposition in glomeruli.3

AKI is usually seen by the end of first week and is non-oliguric in 50-75% of cases. Because of hemolysis and hypercatabolism, hyperkalemia is an early feature. A host of other electrolyte abnormalities i.e. hyponatremia, hyperkalemia, hypokalemia, respiratory alkalosis, hypocalcemia and hypophosphatemia even in the absence of renal failure have also been described.4 Although mild hemolysis may occur glucose-6-phosphate dehydrogenase deficient patients due to heavy parasitemia per se, a variety of infections and drugs such as primaquine and aspirin can induce severe hemolysis in such patients with even mild parasitemia3. AKI has various epidemiological and clinical patterns in countries with different climates and socioeconomic features and, therefore, needs improved individualized approaches in these different regions. AKI is a major cause of mortality in malaria.

**Material and methods**

The present study was a prospective observational study conducted on 100 adult patients aged more than 18 years of malaria diagnosed between October 2017 to December 2020 in medicine department at Pt. B. D. Sharma, PGIMS Rohtak, Haryana. Patients diagnosed with malaria on basis of peripheral blood film included in this study. Patients aged less than 18 years or more than 75 years, patients having nosocomial infections, chronic infections, fever due to non infectious etiologies were excluded from the study. Patients of chronic kidney disease, acute kidney injury secondary to non infectious etiologies, urosepsis, lower respiratory tract infections, hematological malignancies, immunocompromised or immunosuppressed individuals and pregnant females were also excluded from the study. All patients were evaluated by a set of routine blood and urine investigations, peripheral blood smears for malaria, chest radiograph, abdominal ultrasonogram and arterial blood gas. KDIGO guidelines were used for AKI diagnosis and

classification.5 Malaria was considered in patients who had clinically and laboratory diagnostic features such as PBF positive or malaria antigen by card . The patients positive for more than one tropical infection by specific investigation were considered as having mixed infections. All patients were evaluated for AKI on day of admission and then subsequently on day 3, 7 and 14 with laboratory investigations i.e. serum creatinine, blood urea, urine output and eGFR6.

**Statistical analysis**

AKI was considered as explanatory variable. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Categorical outcomes were compared between study groups using Chi square test/student's t test. The trend of laboratory values from admission to final follow up, at different time intervals was assessed by comparing the mean values, using one-way repeated measures ANOVA. Data were analyzed and statistically evaluated using SPSS 22.0 software.7

**Results**

The present study included 100 adult patients of malaria. The mean age was 36.9 ± 13.01 years. Minimum age was 18 years and maximum was 62 years in the study population. Among the study population 64% people were males. Out of 100 patients, 64 cases were due to p. vivax , 28 cases due to p. falciparum and remaining 8 cases due to mixed infections. The baseline characteristics of study patients and malaria with acute kidney injury are depicted in table (1&2). 46 cases had AKI and among these, 33 were due to p. vivax , 11 due to p. falciparum and remaining 2 due to mixed infections. Among AKI cases, 16 cases were in AKI stage I, 10 cases were in AKI stage II and remaining 20 cases were in AKI stage III. (as given in table 3 & 4)

Out of 46 malaria patients having acute kidney injury, 82.6% had headache, 30.4% had dysnea, 60.9% had jaundice, 65.2% had deranged liver functions and half of patients had thrombocytopenia. 71.7 % patient had presented with shock and 5% patients had hypoglycemia. 4 patients presented with neurological manifestations. Out of these 4 patients, 2 due to p.vivax and half due to p. falciparum. 3 patients required ventilator support. 22(47.8%) patients had oligouria. 11 cases(24%) required renal replacement therapy, rest cases improved with conservative management like antimalarial drugs, fluids, ionotropic support etc. 2 patients did not respond and died during early course. (as given in table 4&5) Mortality in AKI with malaria was 5%.

Two patients who had died, one presented with plasmodium falciparum positive malaria, altered sensorium with shock, multiple organ dysfunctions, anuria and dysnea. Patient was put on mechanical ventilation and one session of continuous renal replacement therapy for 50 hours was given. Patient did not respond and died after two weeks. Second patient presented with plasmodium vivax positive malaria, multiple organ dysfunctions and oligouria. 5 sessions of intermittent hemodialysis was given to patient. At last, patient had pulmonary odema and shock and resulting in mortality

In 3 patients out of 11 patients having renal replacement therapy, acute kidney injury converted into chronic kidney disease. Now these 3 patients on nephrology clinic follow up and are being managing conservatively. 2 patients had recovered from acute kidney injury. 4 patients lost to follow up. Baseline and following parameter of malaria with acute kidney

injury patients requiring dialysis therapy are depicted in table 6.

The mean duration of hospital stay in days for people with AKI was 10.75 ± 5.58 and 6.88± 0.76 for people without AKI. Difference in Age**,** SpO2, Temperature, Hemoglobin, total leucocyte count, neutrophils, lymphocytes, absolute platelets count, blood urea, Serum Creatinine, corrected Serum Calcium, Serum Uric Acid, Serum Albumin, Serum bilirubin, SGOT, SGPT, Urine Output, eGFR and hospital stay was statistically significantly between AKI and Non AKI groups (P value <0.05).

Hence these factors can be the predictors of AKI among patients diagnosed with malaria. (as given in table 7)

**Discussion**

The present study reveals that plasmodium vivax (64%) was most common causative agent among malarial patient. 28 % cases were due to plasmodium falciparum and 8% cases due to mixed infection. Similar findings were reported by Trivedi T et al who concluded that plasmodium vivax was the major parasite type (52.54%), followed by P. falciparum (33.75%), and mixed malarial infection (13.69%).8 Plasmodium vivax is not a benign disease any mor., Nair jj et al. showed that plasmodium vivax more common than plasmodium falciparum but aktar cm et al. suggested that plasmodium falciparum more common than plasmodium vivax.9,10 Incidence of AKI in malaria in our study group was 46%. 23.9% of these patients required renal replacement therapy. P.vivax was found to be the cause of severe AKI. Men were affected more in our study as compared to women, similar to observations of other studies.11,12 Our study showed 4.3% mortality in malarial AKI, which was far less than other studies which reported 15-45% mortality.13 The overall mortality rate among those with kidney injury ranges from 15 to 50% in different series.14,15 Low mortality in our study is due to prompt diagnosis, less resistance to anti malarial drugs, timely HD, exchange transfusion, and supportive therapy.16

Our study showed 33% of patients having AKI with P.vivax similar to some other studies.11,17 AKI was seen in early second week and was oliguric in half of patients in present study. The etiology of AKI was usually multifactorial due to hyperbilirubinemia, intravascular hemolysis, hyperparasitemia, volume depletion, hypoxia, shock, pigment nephropathy, DIC, and sepsis.2,14,15,16,18,19 Precise mechanism of renal failure in malaria is not clearly known. Several hypotheses including mechanical obstruction by infected erythrocytes, immune mediated glomerular pathology, fluid loss due to multiple mechanisms and alterations in the renal microcirculation, etc have been proposed.4

Jaundice was present in 28(60.9%) out of 46 patients of AKI in present study, pointing towards multiple organ dysfunctions. Studies have shown that jaundice can be associated with AKI in 75% patients.19 Similar findings are noted in our study. The incidence of jaundice in falciparum malaria has been reported to be between 2 to 57% and associated with 40% mortality.13,20 AKI associated with jaundice had high mortality in comparison with non jaundiced AKI patients due to toxicity to tubular cells with more risk of development of acute tubular necrosis. Indirect hyperbilirubinemia from RBC destruction is the most common form of jaundice reported in malaria.21 Although some patients with falciparum malaria may have direct hyperbilirubinemia and elevated hepatic enzymes.22 In our study, 70% patients had jaundice as

a presenting symptom, which is in accordance to the findings reported in other studies.

There is no direct pathogenic linkage between vivax malaria and AKI, but the associated conditions such as heavy parasitemia, hypercatabolic state, volume depletion, hyperbilirubinemia, intravascular hemolysis, sepsis, and DIC can contribute to AKI. Hence, despite the association, cause and effect relationships remain doubtful.18,19,23 Hyperbilirubinemia may contribute to reduction in total peripheral vascular resistance and in renal blood flow due to left ventricular dysfunction. More studies are required to address factors related to AKI in vivax malaria. Acute cortical necrosis (ACN) has rarely been reported in patients with AKI due to malaria.24 The possible pathogenetic factors in patients with ACN were the renal damage through renal hypoperfusion or endothelial injury through release of various circulating substances (intravascular hemolysis, sepsis, and pancreatitis). It should be suspected in patients with AKI who have a prolonged phase of oligoanuria.24

Many patients with complicated malaria require ICU care. All P.vivax malaria is now increasingly associated with severe disease and high case fatality. A recent retrospective study also concluded that anemia, hepato-renal dysfunctions were equally frequent in vivax malaria and it can no longer be considered as benign infection.25

The mean duration of hospital stay in day in people with AKI in present study was 10.75 ± 5.58 days and 6.88 ± 0.76 days for people without AKI and difference was statistically significant (P value <0.001). The positive predictors of AKI such as hypotension, dehydration, jaundice and underlying sepsis from this study were comparable to the studies conducted by Saravu K et al., Basu G et al. and Nair JJ et al. 9,26,27

**Conclusion**

Malaria is an important cause of AKI in Asia and particularly in tropical areas. Haemodialysis is an effective treatment for malarial AKI. Early referral of malarial AKI patients to dialysis facility unit and early institution of haemodialysis in complicated malaria may further reduce mortality and enhance recovery function. From this present study it can be concluded that the most common cause of AKI was Plasmodium vivax. It is necessary to increase public awareness, provide clinical education and training about tropical illnesses and form specialized renal teams to treat severe AKI patients.

**Limitation of study**: As this study was done at tertiary care level, the representation of more sick patients are likely in study group. Hence the finding cannot be generalized.

Table 1: Baseline and follow up Haematological and Renal parameters in malaria patients(N=100)

| Parameter | Baseline | Day 3 follow up | Day 7 follow up | Day 14 follow up | P* value |
|---|---|---|---|---|---|
| Hemoglobin (g/dl) | 12.48 ± 2.2 | 13.42 ± 15 | 12.98 ± 1.7 | 12.74 ± 1.4 | 0.08 |
| Total Leucocytes Count | 6928 ± 3789.33 | 6591 ± 2178.83 | 6254 ± 1569.05 | 6498 ± 1096 | 0.09 |
| Absolute Platelets Count | 68910 ± 58408.19 | 100580 ± 44514.25 | 158990 ± 32417.57 | 267200 ± 46233.49 | <0.001 |
| Blood urea(mg/dl) | 45.59 ± 57.84 | 42.49 ± 42.55 | 30.37 ± 25 | 19.42 ± 14.3 | <0.001 |
| Serum creatinine (mg/dl) | 1.52 ± 1.38 | 1.45 ± 1.26 | 1.07 ± 0.89 | 0.89 ± 0.55 | <0.001 |
| Corrected Serum Calcium (mg/dl) | 9.39 ± 0.53 | 9.24 ± 0.36 | 9.26 ± 0.33 | 9.27 ± 0.26 | 0.19 |
| Serum phosphate (mg/dl) | 3.5 ± 0.38 | 3.4 ± 0.42 | 3.6 ± 0.41 | 3.4 ± 0.38 | 0.12 |
| Serum Uric Acid (mg/dl) | 3.8 ± 1.09 | 3.52 ± 1.14 | 3.39 ± 0.75 | 2.84 ± 0.71 | 0.10 |
| Serum Protein (g/dl) | 7.49 ± 0.29 | 7.37 ± 0.25 | 7.45 ± 0.25 | 7.48 ± 0.32 | 0.14 |
| Serum Albumin (g/dl) | 3.21 ± 0.24 | 3.41 ± 0.22 | 3.59 ± 0.17 | 3.59 ± 0.21 | 0.18 |
| Serum bilirubin (mg/dl) | 6.5 ± 3.1 | 5.5 ±2.1 | 4.5 ± 1.5 | 3.5 ± 1.1 | 0.05 |
| SGOT( u/L) | 89 ± 35 | 80 ± 45 | 74 ± 30 | 69 ± 37 | 0.24 |
| SGPT (U/L) | 96 ± 32 | 87 ± 22 | 67 ± 36 | 73 ± 36 | 0.12 |
| Serum sodium (meq/l) | 143.35 ± 2.99 | 141.45 ± 2.14 | 137.22 ± 2.84 | 140.79 ± 3.39 | 0.12 |
| Serum Potassium (meq/l) | 3.23 ± 0.31 | 3.71 ± 0.34 | 3.61 ± 0.37 | 3.79 ± 0.31 | 0.16 |
| Urine Output (ml) | 1017.3 ± 443.89 | 1128.8 ± 425.58 | 1501.5 ± 335.48 | 1784.5 ± 307.95 | <0.001 |
| eGFR (ml/min/1.73m²) | 80.66 ± 43.22 | 82.53 ± 43.64 | 90.94 ± 43.61 | 97.94 ± 40.22 | <0.001 |

Repeated ANOVA test*

Table 2: Baseline and follow up Haematological and Renal parameters in malaria with acute kidney injury patients (N=46)

| Parameter | Baseline | Day 3 follow up | Day 7 follow up | Day 14 follow up | P* value |
|---|---|---|---|---|---|
| Hemoglobin (g/dl) | 11.33 ± 2.1 | 12.12 ± 1.9 | 12.56 ± 1.0 | 11.04 ± 1.7 | 0.07 |
| Total Leucocytes Count | 6945 ± 3756 | 6541 ± 2179 | 6234 ± 1560 | 6445 ± 996 | 0.08 |
| Absolute Platelets Count | 68945 ± 58458.19 | 104580 ± 44594.25 | 138990 ± 37617.57 | 297200 ± 49233.49 | <0.001 |
| Blood urea(mg/dl) | 84.59 ± 57.84 | 73.49 ± 42.55 | 40.36 ± 25 | 39.42 ± 14.3 | <0.001 |
| Serum creatinine (mg/dl) | 1.72 ± 1.38 | 1.45 ± 1.29 | 1.11 ± 0.89 | 0.99 ± 0.55 | <0.001 |
| Corrected Serum Calcium (mg/dl) | 9.51 ± 0.53 | 9.34 ± 0.36 | 9.23 ± 0.33 | 9.45 ± 0.26 | 0.17 |
| Serum phosphate (mg/dl) | 3.7 ± 0.38 | 3.8 ± 0.42 | 3.5 ± 0.41 | 3.4 ± 0.39 | 0.13 |
| Serum Uric Acid (mg/dl) | 3.9 ± 1.09 | 3.72 ± 1.14 | 3.49 ± 0.75 | 3.24 ± 0.71 | 0.09 |
| Serum Protein (g/dl) | 7.32 ± 0.29 | 7.12 ± 0.25 | 7.44 ± 0.25 | 7.45 ± 0.32 | 0.15 |
| Serum Albumin (g/dl) | 3.11 ± 0.24 | 3.31 ± 0.22 | 3.59 ± 0.17 | 3.49 ± 0.21 | 0.17 |
| Serum bilirubin (mg/dl) | 7.5 ± 3.1 | 6.5 ±2.1 | 4.9 ± 1.5 | 3.7 ± 1.1 | 0.04 |
| SGOT( u/L) | 99 ± 35 | 85 ± 45 | 79 ± 30 | 70 ± 37 | 0.05 |
| SGPT (U/L) | 95 ± 32 | 88 ± 22 | 77 ± 36 | 66 ± 36 | 0.05 |
| Serum sodium (meq/l) | 145.35 ± 2.99 | 140.45 ± 2.14 | 138.22 ± 2.84 | 141.79 ± 3.39 | 0.15 |
| Serum Potassium (meq/l) | 3.33 ± 0.31 | 3.41 ± 0.34 | 3.71 ± 0.37 | 3.89 ± 0.31 | 0.17 |
| Urine Output (ml) | 997.3 ± 443.89 | 988.8 ± 425.58 | 1101.5 ± 335.48 | 1584.5 ± 307.95 | <0.001 |
| eGFR (ml/min/1.73m$^2$) | 80.66 ± 43.22 | 82.53 ± 43.64 | 90.94 ± 43.61 | 97.94 ± 40.22 | <0.001 |

Repeated ANOVA test*

Table 3: Causes of acute kidney injury in Malaria (N=100)

| Diagnosis | Total n-100 | AKI | |
|---|---|---|---|
| | | AKI n-46 | NO AKI n-54 |
| • Malaria(vivax) | 64 | 33 (51.56%) | 31 (48.44%) |
| • Malaria (falciparum) | 28 | 11 (39.29%) | 17 (60.71%) |
| • Malaria (vivax+ falciparum) | 8 | 2 (25%) | 6 (75%) |

Table 4: Distribution of patients in relations to diagnosis and AKI staging(N=46)

| Diagnosis | AKI STAGE | | | Dialysis required | |
|---|---|---|---|---|---|
| | I | II | III | Yes | No |
| Malaria(vivax) =33 | 12 | 6 | 15 | 8 | 25 |
| Malaria (falciparum) =11 | 3 | 3 | 5 | 3 | 8 |
| Malaria (vivax+ falciparum) =2 | 1 | 1 | 0 | 0 | 2 |
| **Total (N=46)** | 16 | 10 | 20 | 11 | 35 |

Table 5: Clinical manifestations in malaria with acute kidney injury patients (46)

| Clinical manifestations | No. (%) | Clinical manifestations | No. |
|---|---|---|---|
| Headache | 38(82.6%) | Shock | 33(71.7%) |
| Dysnea | 14(30.4%) | Cerebral malaria | 4(8.7%) |
| Jaundice | 28(60.9%) | Ventilator requirement | 3(6.5%) |
| Deranged liver functions | 30(65.2%) | Renal replacement therapy | 11(23.9%) |
| Oligouric | 22(47.8%) | Death | 2(4.3%) |

Table 6: Baseline and follow up Haematological and Renal parameters in malaria with acute kidney injury patients who required renal replacement (N=11)

| Parameter | Baseline | Day 3 follow up | Day 7 follow up | Day 14 follow up | P* value |
|---|---|---|---|---|---|
| Hemoglobin (g/dl) | 9.33 ± 1.1 | 10.11 ± 1.3 | 10.56 ± 1.0 | 10.04 ± 1.7 | 0.05 |
| Total Leucocytes Count | 7745 ± 3756 | 4541 ± 2187 | 4534 ± 1560 | 5545 ± 996 | 0.07 |
| Absolute Platelets Count | 65945 ± 33458.19 | 90580 ± 23594.25 | 120990 ± 32217.57 | 167200 ± 44533.49 | <0.001 |
| Blood urea(mg/dl) | 114.59 ± 57.66 | 93.49 ± 42.53 | 80.36 ± 12.5 | 70.42 ± 13.3 | <0.001 |
| Serum creatinine (mg/dl) | 4.72 ± 1.39 | 3.45 ± 1.19 | 2.11 ± 0.99 | 1.99 ± 0.87 | <0.001 |
| Corrected Serum Calcium (mg/dl) | 8.91 ± 0.53 | 9.22 ± 0.36 | 9.23 ± 0.45 | 9.45 ± 0.45 | 0.08 |
| Serum phosphate (mg/dl) | 5.7 ± 0.38 | 4.8 ± 0.42 | 3.9 ± 0.41 | 3.4 ± 0.33 | 0.10 |
| Serum Uric Acid (mg/dl) | 4.9 ± 1.33 | 4.44 ± 1.22 | 3.89 ± 0.78 | 3.24 ± 0.77 | 0.08 |
| Serum Protein (g/dl) | 7.72 ± 0.77 | 7.33 ± 0.25 | 7.44 ± 0.33 | 7.45 ± 0.67 | 0.14 |
| Serum Albumin (g/dl) | 3.57 ± 0.24 | 3.45 ± 0.22 | 3.33 ± 0.17 | 3.47 ± 0.21 | 0.15 |
| Serum bilirubin (mg/dl) | 8.5 ± 3.7 | 7.0 ±2.1 | 6.9 ± 1.5 | 4.7 ± 1.1 | 0.03 |
| SGOT( u/L) | 111 ± 35 | 95 ± 40 | 89 ± 37 | 71 ± 36 | 0.05 |
| SGPT (U/L) | 105 ± 37 | 98 ± 22 | 87 ± 36 | 69 ± 34 | 0.05 |
| Serum sodium (meq/l) | 133.35 ± 2.99 | 140.45 ± 2.14 | 141.22 ± 2.84 | 138.79 ± 3.39 | 0.14 |
| Serum Potassium (meq/l) | 3.23 ± 0.31 | 3.51 ± 0.35 | 3.81 ± 0.37 | 3.79 ± 0.31 | 0.16 |
| Urine Output (ml) | 597.3 ± 433.89 | 688.8 ± 325.58 | 901.5 ± 235.48 | 1184.5 ± 357.95 | <0.001 |
| eGFR (ml/min/1.73m$^2$) | 40.66 ± 43.22 | 52.53 ± 43.64 | 60.94 ± 43.61 | 70.94 ± 40.22 | <0.001 |

Repeated ANOVA test*

Table 7: Predictive factors associated with AKI (N=100)

| Parameter | AKI | | *P value |
|---|---|---|---|
| | Present (N=46) | Absent (N=54) | |
| | Mean ± SD | Mean ± SD | |
| Age (Years) | 36.88 ± 13.67 | 32.56 ± 12.09 | *0.008* |
| **Gender** | | | **P value |
| Male | 30 (71.87%) | 32 (66.17%) | 0.569 |
| Female | 16 (28.12%) | 22 (33.82%) | |
| **Diagnosis** | | | **P value |
| Malaria(vivax) | 33 (51.56%) | 31 (48.44%) | <0.001 |
| Malaria (falciparum) | 11 (39.29%) | 17 (60.71%) | |
| Malaria (vivax+ falciparum) | 2 (25%) | 6 (75%) | |
| **PHYSICAL EXAMINATION PARAMETERS** | | | |
| **Parameter** | Mean ± SD | Mean ± SD | *P value |
| Syatolic BP(mm Hg) | 115.88 ± 7.92 | 113.85 ± 5.01 | *0.435* |
| Diastolic BP(mm Hg) | 73.94 ± 6.53 | 68.74 ± 7.25 | *0.036* |
| Pulse rate(per min.) | 84.56 ± 10.05 | 84.06 ± 8.62 | *0.797* |
| SPO2(%) | 96.03 ± 2.1 | 97.47 ± 1.2 | *<0.001* |
| Temperature(°F) | 103.22 ± 0.83 | 102.32 ± 1.1 | *<0.001* |
| **Biochemical parameters** | | | |
| **Parameter** | Mean ± SD | Mean ± SD | *P value |
| Hemoglobin (g/dl) | 12.57 ± 2.8 | 13.91 ± 1.81 | *0.005* |
| Total Leucocytes Count (per mm$^3$) | 9615.63 ± 5322.75 | 5075 ± 1188.42 | *<0.001* |
| Neutrophils(per mm$^3$) | 76.47 ± 10.85 | 60.93 ± 7.62 | *<0.001* |
| Lymphocytes(per mm$^3$) | 19.47 ± 10.16 | 34.1 ± 7.18 | *<0.001* |
| Absolute Platelets Count(per mm$^3$) | 94444.44 ± 59298.68 | 61852.94 ± 56484.38 | *0.014* |

| Parameter | Mean ± SD | Mean ± SD | *P value |
|---|---|---|---|
| Blood urea (mg/dl) | 109.97 ± 66.11 | 15.29 ± 3.17 | *<0.001* |
| Blood sugar (mg/dl) | 97.56 ± 11.6 | 99.22 ± 8.23 | *0.414* |
| Serum creatinine (mg/dl) | 2.99 ± 1.67 | 0.83 ± 0.11 | *<0.001* |
| Corrected Serum Calcium (mg/dl) | 9.65 ± 0.63 | 9.41 ± 0.47 | *0.035* |
| Serum phosphate (mg/dl) | 3.31 ± 0.43 | 3.44 ± 0.35 | *0.109* |
| Serum Uric Acid (mg/dl) | 3.73 ± 1.67 | 3.24 ± 0.62 | *0.036* |
| Serum Protein (g/dl) | 7.52 ± 0.31 | 7.62 ± 0.27 | *0.110* |
| Serum Albumin (g/dl) | 3.49 ± 0.29 | 3.37 ± 0.2 | *0.014* |
| Serum bilirubin (mg/dl) | 13.5 ± 3.1 | 5.5 ± 3.1 | *<0.001* |
| SGOT( u/L) | 134 ± 35 | 76 ± 3.1 | *0.04* |
| SGPT (U/L) | 146 ± 32 | 67 ± 35 | *0.05* |
| Serum sodium (meq/l) | 141.41 ± 3.71 | 141.32 ± 2.61 | *0.898* |
| Serum Potassium (meq/l) | 3.46 ± 0.5 | 3.56 ± 0.16 | *0.142* |
| Urine Output (ml) | 532.19 ± 354.5 | 1473.53 ± 233.48 | *<0.001* |
| eGFR (ml/min/1.73m²) | 32.05 ± 23.31 | 107.94 ± 25.27 | *<0.001* |
| Hospital stay ( days) | 10.75 ± 5.58 | 6.88 ± 0.76 | *<0.001* |

***Student's t- test**

**#Chi square test**

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