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Original Research Article

Investigation of Hepatic Profile in Plasmodium Falciparum Malaria Diagnosed Patients

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Abstract:

Aim: This study aims to show the effect of malaria on the hepatic profile of the patient. Malaria is a serious health hazard around the world. Malaria is one of the dreadful burdens on humankind. Plasmodium falciparum is correlated with hepatic dysfunction.

Materials and Methods: This study includes 100 patients organized in a tertiary care centre in Jharkhand, India for 18 months. Out of 100 cases, 60 patients were male and 40 patients were female. Complete clinical, biochemical, and radiological examinations were carried out for the detection of illness. The age group of patients was between 16-56 years.

Results: According to this study, it was evaluated that in 67% of the cases, there was an increase in total and direct bilirubin, and 45% of patients showed a rise in the level of direct bilirubin.

Conclusion: Acute plasmodium falciparum is strongly associated with liver dysfunction, which can cause a slight increase in hepatic enzymes to the extent of severe hepatitis. Hepatitis with falciparum in subjects has higher chances of complications, multi-organ failure, and poor prognosis.

Keywords: *Plasmodium falciparum, Malaria, Liver dysfunction*

Introduction

Malaria is an infectious disease caused by a plasmodium species and 200-400 million cases are reported a year all over the world and cause 2 million deaths per year [1]. The major number of cases of malaria are caused by falciparum. When female anopheles bite the human the parasites get transmitted into human blood which causes malaria. Malaria infection in humans ranges from without showing any symptoms, to malaria without complications, complicated malaria, and fatal malaria. Plasmodium falciparum has a high level of mortality.

According to many studies carried out by numerous authors, 17%-66% of complication is caused by falciparum [2-8]. The malaria parasite belongs to the genus Plasmodium. Four species of plasmodium infects human namely Plasmodium falciparum, plasmodium vivax, plasmodium malaria, and Plasmodium ovale. Falciparum is the most threatening one among all and high incidence of complications and death. The initial symptom includes severe fever vomiting and headache at 48 hours intervals. In many areas, chloroquine is the only base of treatment. The release of sporozoites in the liver confirms malarial transfer to the human host. These sporozoites develop into schizonts. Schizonts boost the infection by making a huge number of merozoites. Each merozoite has the potential to attack human red blood cells and initiate an asexual cycle of replication.

Clinical presentation of falciparum malaria may differ in individuals according to the load of the parasite in the blood and the immunity of the patient. Jaundice indicates an acute falciparum infection. It is more prevalent in grown people rather than in young ones and maybe with other complications [9]. Occurrence of acute jaundice associated with malaria is increasing in many countries of south-east Asia including India [10-11]. Most cases have only infection with falciparum or infection with both

falciparum and vivax [12, 13]. Malarial hepatitis is a dysfunction of hepatocytes in acute and complicated malaria [14] which also includes a rise in serum bilirubin [15]. Therefore, malarial hepatitis is an element of organ dysfunction and leads to a bad prognosis for falciparum infection. So in this study, it was examined the effect of P falciparum on the liver.

Materials and Methods

This is a prospective study that includes 100 patients which was organized in a tertiary care centre in Jharkhand, India. The duration of the study was 18 months. Malaria was diagnosed by carrying out clinical, biochemical, and radiological examinations.

Inclusion Criteria*:* Subjects of both genders of different ages infected with falciparum malaria associated with jaundice.

Exclusion Criteria: Patients who are not infected with falciparum and those patients who are only infected by vivax infection.

This research was planned to comprise the population study and changes in blood detected in the subjects. Based on relevant investigations patients with hepatic diseases like viral hepatitis, cirrhosis, liver abscess, etc. were excluded from the study. All the clinical examinations were done on the patients.

Patients of plasmodium falciparum malaria with jaundice were examined for a history of headache, fever, vomiting, icterus, pallor, and reduction in passing of urine. The presence of an asexual strain of Plasmodium falciparum with jaundice confirms the diagnosis of malaria. If the parasites are not found in 100 high-power fields, then the slide is taken as negative.

The laboratory investigation includes bleeding time, clotting time, random blood sugar, complete blood tests, and urea. By finding the amount of conjugating and nonconjugating bilirubin, serum protein, and prothrombin time liver function test was examined. To exclude the probability of viral hepatitis blood, work for hepatitis B and C was done.

Based on serum bilirubin levels subjects were divided into following

- Group A bilirubin less than 4mg %
- Group B bilirubin between 4-10mg %
- Group C serum bilirubin more than 10mg %

The size and echo texture of the liver, gallbladder abnormality, and portal hypertension were examined by ultrasound. A hepatic biopsy was done by gunshot method.

Results

A total of 100 patients were included in this study of which 60 (60%) were male and 40 (40%) were females. All the patients were 17-57 years of age. The common symptoms include fever with chills associated with vomiting subsequently stomach pain, headache, and impaired consciousness. The mean period of illness before the patient came to the hospital was 1 week. Icterus was detected in all the patients, pallor in 50 patients (63.4%), 43 (52%) patients had splenomegaly, hepatomegaly, and impaired consciousness in 18 (21%) patients. 35 patients had less than 4mg % of serum bilirubin, 35 patients' bilirubin level was between 4-10mg %, and 30 patients had more than 10 mg% serum bilirubin.

Alanine transaminase levels varied from 33-1239 IU/L and aspartate transaminase levels varied from 29-1460 IU/L. 12 patients had increased levels of serum transaminase three times the normal value. Prothrombin time was not normal in cases with severe malaria associated with high bilirubin but the majority of the patients had normal prothrombin time. 60 (70%) patients were anaemic. 20 patients had thrombocytopenia 3 had leucocytosis. 8 patients died due to cerebral malaria or multi-organ failure.

Table 1: Sonographic findings

USG findings	Number of patients	Percentage
Normal size with normal echogenicity of liver	30	30%
Normal size with decreased echogenicity of liver	5	5%
Hepatomegaly with normal echogenicity of liver	25	25%
Hepatomegaly with decreased echogenicity of liver	20	20%
Increased wall thickness of gall bladder	10	10%
Splenomegaly	5	5%
Ascites	5	5%

As shown in Table 1, 30 patients had normal size with normal echogenicity of the liver, 5 patients had normal size but decreased echogenicity of the liver, 25 patients had hepatomegaly with normal echogenicity of the liver, 20 patients had hepatomegaly with decreased echogenicity of the liver, 10 patients had increased wall thickness of gall bladder, 5 patients had splenomegaly and 5 had ascites.

Table 2: Changes in histopathology in the liver in patients of plasmodium falciparum malaria with jaundice

Changes in histopathology	Number of patients	Percentage
Swollen hepatocytes	35	35%
Pigment deposition	25	25%
Portal infiltration	10	10%
Sinusoidal infiltration	14	14%
Kupferr cell hyperplasia	5	5%
Liver cell necrosis	6	6%
Fatty change	5	5%

As shown in Table 2, swollen hepatocytes were present in 35 (35%) patients, 25 (25%) patients had pigment deposition, Portal infiltration was found in 10 (10%) patients, 14 (14%) patients had sinusoidal infiltration, Kupfer cell hyperplasia was seen in 5 (5%) patients, liver cell necrosis was present in 6 (6%) cases and fatty changes was seen in 5 (5%) patients.

Discussion

Liver dysfunction is commonly associated with falciparum infection and is acknowledged from the beginning. Jaundice in acute falciparum infection is complex; intrahepatic hemolysis of parasitized red blood cells, hemolysis of nonkeratinized RBCs, liver disorder, and hemolysis caused by drugs. The existence of hepatitis E virus or hepatitis A virus causes jaundice in malaria [16].

In many studies, it was evaluated that malarial hepatitis is the chief cause of jaundice [11,17,18]. The dysfunction of hepatic cells in acute and complex malaria is termed malarial hepatitis. Malarial hepatitis is distinguished increase in bilirubin with an increase in the alanine transaminase level thrice the normal unit [10]. The prevalence of jaundice with liver dysfunction has differed which can be due to the condition of the region, studied age group, and coexistent viral hepatitis [14]. Jaundice is more commonly found in adults with around 30-35% unconjugated hyperbilirubinemia as reported by Harris et al [19]. Falciparum infection associated with hepatitis in patients had greater chances of complications with a bad prognosis.

In category C thrombocytopenia and anemia were higher as compared to patients in category A. A study conducted by Kochar et al which shows the same prevalence of thrombocytopenia because of falciparum [20]. Patients with acute falciparum infection and interruption in normal coagulating time cause elevation of prothrombin time. Increased levels of bilirubin to a greater extent cause damage to hepatic cells. The main histopathological change in an infected liver consists of a reticuloendothelial response which is Kupfer cell hyperplasia and the presence of malarial pigments. The existence of Kupfer cells suggests liver damage. The presence of hyperbilirubinemia and a rise in liver enzymes with hepatic cell necrosis in histopathological examination confirms the hepatocytic dysfunction in patients of plasmodium falciparum malaria with jaundice [20].

In severe malaria, hepatic dysfunction is amendable with proper treatment and anti-malarial therapy. The level of bilirubin gets back to normal within 2 days or it may slow down in patients already having renal disorder. After the malaria treatment, there was an improvement in liver function test and serum bilirubin came back to normal level. The size and echogenicity of the liver were normal with mild hepatomegaly. Hepatomegaly was also a common finding in the patients. In all the subject transaminases was three times the normal range.

Conclusion

In this study, it was evaluated that plasmodium falciparum affects the normal functioning of the liver. It involves a mild increase in hepatic enzymes to the extent of severe hepatitis. Malarial hepatopathy can be detected by clinical examination, and ultrasounds and is likely to be detected in patients with high fever, jaundice, and an increase in transaminases. Liver disorder with jaundice is a major occurrence in severe falciparum infection which leads to more complications. Proper diagnosis is required to deal with the increasing number of cases.

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