Florence Nightingale

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Case Report

A case of *Plasmodium falciparum* malaria presenting with splenic infarction

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ABSTRACT

Malaria is a parasitic infectious disease caused by *Plasmodium* species, which starts with acute paroxysmal fever attacks, especially in tropical and subtropical regions. Although malaria caused by *Plasmodium vivax (P. vivax)* is mostly seen in our country, *Plasmodium falciparum* (*P. falciparum)* malaria is rarely seen. *P. falciparum* malaria can cause fatal complications and requires immediate treatment. In this case report, a case of *P. falciparum* malaria presenting with splenic infarction detected in a tourist patient is presented.

Keywords: Malaria, *Plasmodium falciparum*, splenic infarction.

Malaria is a significant infectious disease that affects the entire world, mainly tropical and subtropical countries.[1] There are four species of *Plasmodium* (*Plasmodium vivax*, *P. ovale*, *P. falciparum*, and *P. malariae*) that cause infection in humans. In our country and worldwide, *P. vivax* is the most commonly detected species. In our country, following the elimination of *P. vivax*, *P. falciparum* acquired from abroad has become the most commonly encountered malaria parasite in recent years. The disease caused by *P. falciparum* is more severe than other species, with a higher risk of fatality, and untreated *P. falciparum* malaria is always fatal. Early diagnosis and appropriate treatment are the most crucial factors in reducing complications and preventing deaths. The majority of *P. falciparum* cases detected in our country belong to citizens traveling and working abroad.[2-4] In this case report, a malaria case with splenic infarction detected in a tourist patient was presented to increase awareness about *P. falciparum* and to review the relevant literature.

CASE REPORT

A 43-year-old male patient, a British citizen, was brought to our emergency department with complaints of high fever, chills, weakness, loss of appetite, drowsiness, dizziness, and pain on the left side of the abdomen and back. In the patient's history, it was revealed that he had been working in Africa for three months and had been taking chloroquine prophylaxis. His symptoms have been progressively worsening for a week. At the onset of the complaints while in Africa, a malaria antigen test was performed and came back negative. An abdominal computed tomography (CT) scan conducted at an external center revealed splenic infarction (Figure 1). During the physical examination of the patient, there was a fever of 39°C, scleral icterus, and splenomegaly. The laboratory results showed leukocytes at 5,000/mm^3, hemoglobin (Hb) at 13.5 g/dL, platelets at 17.8/mm^3, alanine aminotransferase (ALT) at 54 U/L, aspartate aminotransferase (AST) at 65 U/L, lactate dehydrogenase (LDH) at 599 U/L, total bilirubin at 4.66 mg/dL, direct bilirubin at 3.21 mg/dL. In the urine, bilirubin was present in a significant amount (+++) and urobilinogen was also

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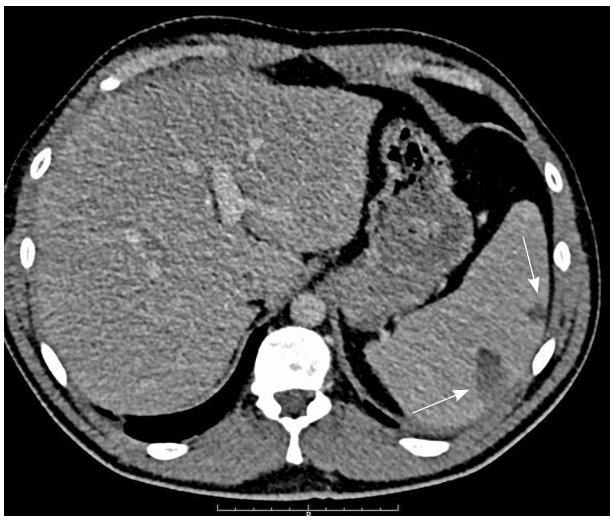

Figure 1. Splenic infarct.

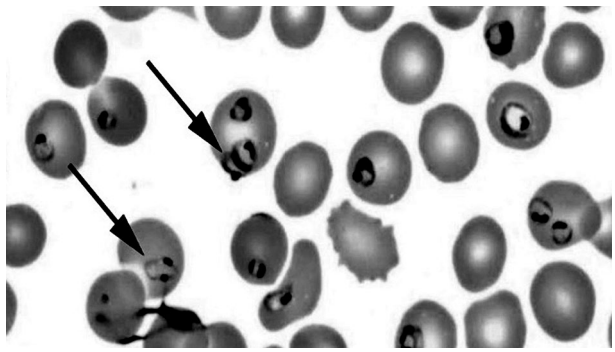

Figure 2. *P. falciparum* young trophozoite (Giemsa staining, ×100).

increased (+++). International normalized ratio (INR) and prothrombin time (PT) were within the normal range. In the peripheral blood smear, *P. falciparum* trophozoites were observed (Figure 2). The patient was started on intravenous (IV) artesunate, artemether/lumefantrine, and prednisolone 40 mg IV treatment. Abdominal ultrasonography (USG) was performed on the patient with ongoing abdominal pain. The findings revealed hepatic steatosis (fatty liver), a spleen size of 15 cm, subcapsular infarct areas in the spleen reaching a maximum size of 5 cm, free fluid in both perinephric areas and sludge in the lumen of the gallbladder. On the seventh day of hospitalization, the patient experienced a cough, difficulty breathing, and chest pain. D-dimer level was found to be 3078 ng/mL, while troponin I was within the normal range. Enoxaparin sodium treatment was initiated. A pulmonary angiography CT scan was performed on the patient. In the right lung upper lobe, there were ground-glass opacities in the perihilar area, and in the right lung lower lobe, subsegmental linear atelectasis was observed at the basal region. In the left lung lower lobe, atelectasis was seen in subpleural areas. Levofloxacin IV treatment was initiated for the patient with sparse crackles on lung auscultation and free fluid in the perinephric area. Following the treatment, the patient's symptoms improved. The patient, with a platelet count of 302,000/mm^3, LDH level of 193 U/L, ALT level of 37 U/L, Hb level of 11.8 g/dL, and a total bilirubin level of 0.64 mg/dL, was discharged from the hospital.

DISCUSSION

In our country, an average of 200-250 malaria cases are reported annually from abroad, and approximately 75% of these cases are due to *P. falciparum* malaria.[5-11] Clinical manifestations of *P. falciparum* malaria include chills, shivering, fever, and sweating. However, it has a more severe course compared to other malaria infections and represents the group with the highest mortality rate.[6-11] Early diagnosis and administration of appropriate and effective medication are critical factors in increasing the success of *P. falciparum* malaria treatment. Furthermore, the patient's follow-up should be conducted under hospital conditions, ensuring the regular intake of medication and monitoring for possible complications.[6] In the presented case, the patient was kept under observation in the hospital during the treatment period, and blood values were checked at regular intervals. Follow-up using abdominal USG was performed to monitor the infarct area and other complications. The patient, who presented with jaundice and thrombocytopenia, has been started on prednisolone treatment. During the treatment, due to an increase in D-dimer levels, pneumonia, and atelectasis, as well as the development of fluid in the perinephric area, IV levofloxacin has been initiated.

During malaria attacks, the breakdown of red blood cells leads to anemia, and the release of pigment from the fragmented red blood cells causes an increase in the blood, resulting in jaundice.

Additionally, the deposition of these pigment-laden cells in the reticuloendothelial system leads to the development of hepatosplenomegaly.[7] Patients may exhibit physical examination findings such as hepatomegaly, splenomegaly, abdominal tenderness, and changes in bowel habits. Splenomegaly is an indication of increased red blood cell destruction and serves as a measure of the disease's duration. In studies conducted in our country, Mert et al.[8] reported splenomegaly in 91% of malaria cases and hepatomegaly in 55% of cases, İnan et al.[9] observed splenomegaly in 72% of cases, and Gül et al.[12] found splenomegaly in 67% of cases and hepatomegaly in 46% of cases. In our case, splenomegaly was detected.

Splenic infarction can occur during the course of infectious diseases. For example, Alkan-Çeviker et al.[13] reported a case of splenic infarction during the course of brucellosis. With the case presented here, it is once again emphasized that splenic infarction can also be seen in association with malaria.

Treatment for *P. falciparum* malaria should be initiated urgently. The World Health Organization recommends artemisinin-based combination therapies for the treatment of falciparum malaria.[10] The goal of artemisinin-based combination therapies is to provide effective antimalarial treatment by taking advantage of the different half-lives of the compounds used.[14] Artemisinin is recommended due to its rapid clearance of parasitic load in the blood and its effectiveness against sexual forms of the parasite. For uncomplicated falciparum malaria, artemether-lumefantrine, artesunate-mefloquine, artesunate-amodiaquine, dihydroartemisinin-piperaquine, and artesunate-sulfadoxine-pyrimethamine are the first-choice treatment options.[10] In the treatment of severe malaria, the administration of parenteral therapy with artemisinin derivatives (artesunate/artemether) or quinine/quinidine is recommended initially, followed by the continuation of treatment with artemisinin-based combinations orally.[10,15,16] If a patient presents with symptoms such as coma (cerebral malaria), metabolic acidosis, severe anemia, thrombocytopenia, hypoglycemia, acute kidney failure or acute pulmonary edema, splenic infarction, spontaneous bleeding, or coagulopathy, severe malaria should be suspected, and parenteral therapy should be initiated.[10] Due to the patient's limited oral intake and the presence of symptoms suggesting severe *P. falciparum* malaria, a combination of IV and oral treatments was chosen. The patient has been started on IV artesunate obtained from the Malaria Control Center in Antalya, as well as artemether and lumefantrine tablets. With fluid resuscitation and steroid treatment, the patient's thrombocytopenia has improved.

Abnormal laboratory findings in malaria vary according to the severity of hemolysis. Anemia, thrombocytopenia, and neutropenia are commonly observed. In *P. falciparum* malaria, Hb, hematocrit, and haptoglobin decrease, while LDH increases. In cases of acute kidney failure, there is an increase in creatinine levels, and significant elevations in transaminases and bilirubin levels indicate disease complications.[1,7] In our case, in line with the literature, we observed an increase in anemia, thrombocytopenia, AST, ALT, LDH, and bilirubin levels.[17-19]

The simple, effective, and rapidly resulting method used in the diagnosis of malaria is Giemsa staining. In this method, a thin smear and a thick drop are made from a finger-prick blood sample, and after staining with Giemsa, the different stages of the parasite's development are examined. If the initial examination does not lead to a definitive diagnosis, it is recommended to repeat the examination for three consecutive days.[7] In our case, the diagnosis of *P. falciparum* was confirmed by the presence of the parasite in the thin smear and thick drop.

In travel-related fatal malaria cases, risk factors have been reported to include inappropriate or lack of chemoprophylaxis usage, advanced age, delayed healthcare seeking, incorrect treatment, delayed diagnosis, infection with *P. falciparum*, and lack of immunity.[19]

In conclusion, early diagnosis and appropriate treatment are the most crucial factors that positively influence the clinical course of malaria caused by *P. falciparum*. Especially in cases of *P. falciparum*, spontaneous splenic rupture can lead to fatal complications.

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