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CASE REPORT

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# Splenic infarction in a paediatric patient with *Plasmodium vivax* malaria from Ethiopia: a case report

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## Abstract

**Background** Splenic infarction is an uncommon but serious side effect of *Plasmodium vivax* malaria, especially in young patients. Prompt diagnosis and effective treatment are essential to avoid serious consequences. Though there are few report of splenic infarction following *P. vivax* from different endemic country, PubMed and Google-based literature search found that it was the first case report of this type from Ethiopia.

**Case presentation** The patient was an 11-year-old girl, from Wolaita Sodo, Ethiopia, who had a high-grade fever, chills, rigors, headache, vomiting, and abdominal pain in the left upper quadrant. Upon examination, hepatomegaly, splenomegaly, and extreme pallor were found. Laboratory tests revealed acute kidney injury (creatinine 1.63 mg/dL), acute liver injury (AST 323 U/L, ALT 129 U/L), and severe anaemia (haemoglobin 3.4 g/dL, haematocrit 10.2%). A peripheral blood smear showed a trophozoite stage of *P. vivax* and was negative for *Plasmodium falciparum*. An abdominal ultrasound revealed hepatosplenomegaly along with a wedge-shaped, multifocal, hypoechoic splenic region that was consistent with an infarction.

**Management and outcome** The patient had blood transfusions, NSAIDs for pain, and intravenous artesunate as treatment. Primaquine was used in radical therapy. After three days, her abdominal pain had considerably subsided and she became afebrile. Complete symptom relief, normalized abdominal ultrasound findings, and better laboratory results—including normal haemoglobin and liver enzymes—were all observed at the two-month follow-up.

**Conclusion** This case underscores the importance of considering splenic infarction in paediatric patients with *P. vivax* malaria presenting with abdominal pain. Early recognition through imaging and laboratory investigations, along with prompt antimalarial therapy, is critical for favourable outcomes.

**Keywords** *Plasmodium vivax*, Splenic infarction, Ethiopia, Pediatric malaria

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## Background

Malaria is still a major global health issue, especially in tropical and subtropical areas. It is estimated that 627,000 people died from malaria worldwide in 2020, out of an estimated 241 million who were infected with the disease. Humans contract these protozoans from infected *Anopheles* mosquito vectors [1–3]. *Plasmodium vivax* is the second most important parasite of human malaria widely perceived as causing mild and self-limited illness [4]. Almost all severe forms of malaria are caused by *Plasmodium falciparum*, but recently there have been reports of serious complications such as myocardial infarction, severe anaemia, respiratory distress, splenic complications, shock, and multiple organ dysfunction also develop with *P. vivax* infection among children from endemic regions such as Indonesia, India and Brazil [5–9].

Anaemia, leucopenia, and thrombocytopenia are known haematological changes that occur in malaria [10]. Splenic complications identified in cases of malaria are splenic infarction, spontaneous splenic rupture, hyperreactive malarial syndrome, hypersplenism, ectopic spleen and splenic torsion, and splenic cysts. Splenic infarction is not usually noted and is likely underdiagnosed in many cases of complicated malaria [9, 11–13].

Blood filtering and pathogen removal depend on the spleen. The sequestration of parasitized red blood cells in malaria results in splenic enlargement and dysfunction. Splenic infarction may develop from the congestion that results from underlying vascular dysfunction. Children who are dehydrated, anaemic, or co-infected with various infections are at risk for splenic infarction [14, 15]. Splenic infarction patients frequently experience fever, splenomegaly, and abdominal pain. Children's symptoms can be ambiguous and could be confused with those of other conditions like appendicitis or gastroenteritis. Because they often reveal splenic necrosis, imaging tests—particularly CT and ultrasound scans—are essential for the diagnosis [15, 16].

In their assessment of severe sequelae linked to *P. vivax* malaria, Rizvi et al. [17] pointed out that splenic infarction was underreported but that, if not identified promptly, could result in serious morbidity. The necessity of doctors being more cognizant of this possible consequence was underlined.

This report describes the case of a young girl presenting with splenic infarction in acute vivax malaria.

## Case presentation

An 11-year-old girl from Wolaita Sodo, Ethiopia, presented with a four-day history of high-grade, intermittent fever accompanied by chills, rigors, headache, and two episodes of non-bloody, non-bilious vomiting. She also reported left upper quadrant abdominal pain that had been present for two days and worsened significantly over the past 24 h. There was no history of jaundice, dark-colored urine, diarrhoea, or abdominal trauma.

On examination, her vital signs were stable: temperature 38.2 °C, pulse rate 80 beats/min, blood pressure 110/60 mmHg, and respiratory rate 14 breaths/min. She weighed 31 kg, measured 143 cm in height, and had a BMI of 15.5 kg/m$^2$ (within the normal range on the Z-score). She appeared pale but was anicteric. Abdominal examination revealed a tender spleen palpable 6 cm below the left costal margin and a soft, non-tender liver palpable 4 cm below the right costal margin. Other systemic examinations were unremarkable.

Laboratory studies showed severe anaemia (haemoglobin 3.4 g/dL, haematocrit 10.2%), acute liver injury (AST 323 U/L, ALT 129 U/L), and acute kidney injury (creatinine 1.63 mg/dL). Peripheral blood smear confirmed the trophozoite stage of *P. vivax* and was negative for *P. falciparum* (Table 1).

Abdominal ultrasound revealed hepatomegaly with a homogenous echo pattern and splenomegaly (16.3 cm) with peripheral, multifocal, wedge-shaped hypoechoic areas, the largest measuring 4.2 × 2.6 cm, indicative of splenic infarction (Fig. 1). Doppler imaging showed no blood flow in these areas.

The patient was treated with intravenous artesunate and NSAIDs for pain relief. She received two whole blood transfusions at 20 mL/kg each and was initiated on radical therapy with primaquine. Her condition improved steadily; she became afebrile three days after starting anti-malarial treatment, and the abdominal pain gradually subsided.

At discharge, on the tenth day of admission, follow-up investigations showed significant improvement, with haemoglobin levels rising to 11.6 g/dL and normalization of liver and kidney function tests. The patient was closely monitored for complications, including splenic rupture, abscess formation, and sepsis.

At a two-month follow-up, the patient was asymptomatic. Abdominal pain had completely resolved, the spleen and liver were no longer palpable, and repeat ultrasound findings were normal.

**Table 1** Relevant investigations of patient

| S. No | Investigation done | | Parameters | Normal reference value for her age | On admission (Day 1) | On Discharge (Day 10) |
|---|---|---|---|---|---|---|
| 1 | CBC | | WBC | 4–10.5 $\mu m^3$ | 4500 cells/mm3 | 4600 cells/$mm^3$ |
| | | | Lymphocytes | 25–33% | 32% | 31% |
| | | | Granulocytes | 54–62% | 56.3% | 52% |
| | | | Haemoglobin | 12.5–16.1 g/dl | 3.4 g/dl | 11.6 g/dl |
| | | | Haematocrit | 36–47% | 10.2% | 39.8% |
| | | | Platelet | 150–450 × $10^3$ µL | 436,000 | 400 × $10^3$ µL |
| 2 | Peripheral blood film | | Thin and thick blood smear | No hemo parasite | Trophozoite stage of *P. vivax* | |
| 3 | Serum bilirubin | | Total | < 1 mg/dl | 0.8 mg/dl | |
| | | | Direct | < 1 mg/dl | 0.2 | |
| 4 | Liver enzyme test | | AST | 10-40U/L | 323U/L | 39 U/L |
| | | | ALT | 5-45U/L | 129U/L | 30 U/L |
| 5 | | Serum electrolyte | Potassium | 3.5–5.5 meq/l | 3.5 meq/l | |
| | | | Sodium | 135-145 meq/l | 139 meq/l | |
| | | | Calcium total | 8.8–10.8 mg/dL | 10 mg/dl | |
| 6 | | Renal function test | Creatinine | 0.31–0.88 mg/dl | 1.63 mg/dl | |
| | | | BUN | 7-18 mg/dl | 14 | |
| 7 | | Urine analysis | Blood | Negative | + 3 | Negative |
| | | | Protein | Negative | Negative | Negative |

## Discussion

A young patient with splenic infarction—a rare but dangerous side effect of *P. vivax* malaria—is depicted in the case study. *Plasmodium vivax* was once believed to be a benign species that mostly caused mild infections, but it has recently been connected to severe side effects such splenic infarction, acute kidney injury (AKI), and anaemia, particularly in endemic areas like Brazil, Indonesia, and India [4–6].

Only a few cases of splenic infarction following *P. vivax* infection have been reported in the English literature, and this is the first documented case from Ethiopia. This is likely not due to the rarity of the condition, given that Ethiopia is a malaria-endemic country, but may rather be due to underreporting [10, 18].

Splenic infarction in malaria has a complicated aetiology. The spleen, an essential organ for blood filtration and immunological response, expands and becomes obstructed as a result of immune cell invasion and parasitized red blood cell sequestration. This splenic congestion, which is made worse by vascular dysfunction, dehydration, anaemia, and other co-infections, puts patients at risk for infarction [14, 15].

Severe anaemia (haemoglobin 3.4 g/dL, haematocrit 10.2%) and AKI (creatinine 1.63 mg/dL) were clearly contributing factors in this patient, aggravating splenic hypoxia and vascular stasis.

Abdominal pain can result from a number of malarial consequences, including pancreatitis, hepatitis/hepatomegaly, acalculous cholecystitis, acute surgical abdomen, splenic rupture, splenic infarction, splenic torsion, and acute renal failure [19, 20].

Clinically, splenic infarction is difficult to diagnose since symptoms, such as fever and abdominal pain, are vague and can be mistaken for other illnesses such as appendicitis or gastroenteritis [15]. In this instance, abdominal ultrasonography indicated splenic infarction due to splenomegaly and pain in the left upper quadrant. Imaging tests are still essential for detecting this issue; ultrasound reveals hypoechoic, wedge-shaped lesions that are suggestive of an infarction. The lack of blood flow in the afflicted areas was further verified by Doppler imaging [16].

In this instance, treatment focused on using intravenous artesunate to treat the underlying *P. vivax* malaria, primaquine for radical therapy to avoid relapse, and NSAIDs and blood transfusions to manage symptoms. Over a two-month follow-up period, the youngster reacted nicely, with all symptoms completely resolved and haematological, renal, and hepatic values returning to normal. This highlights the need of early diagnosis and appropriate treatment in achieving optimal outcomes in patients of splenic infarction.

This case confirms findings in the literature that *P. vivax* malaria sequelae are underreported and can

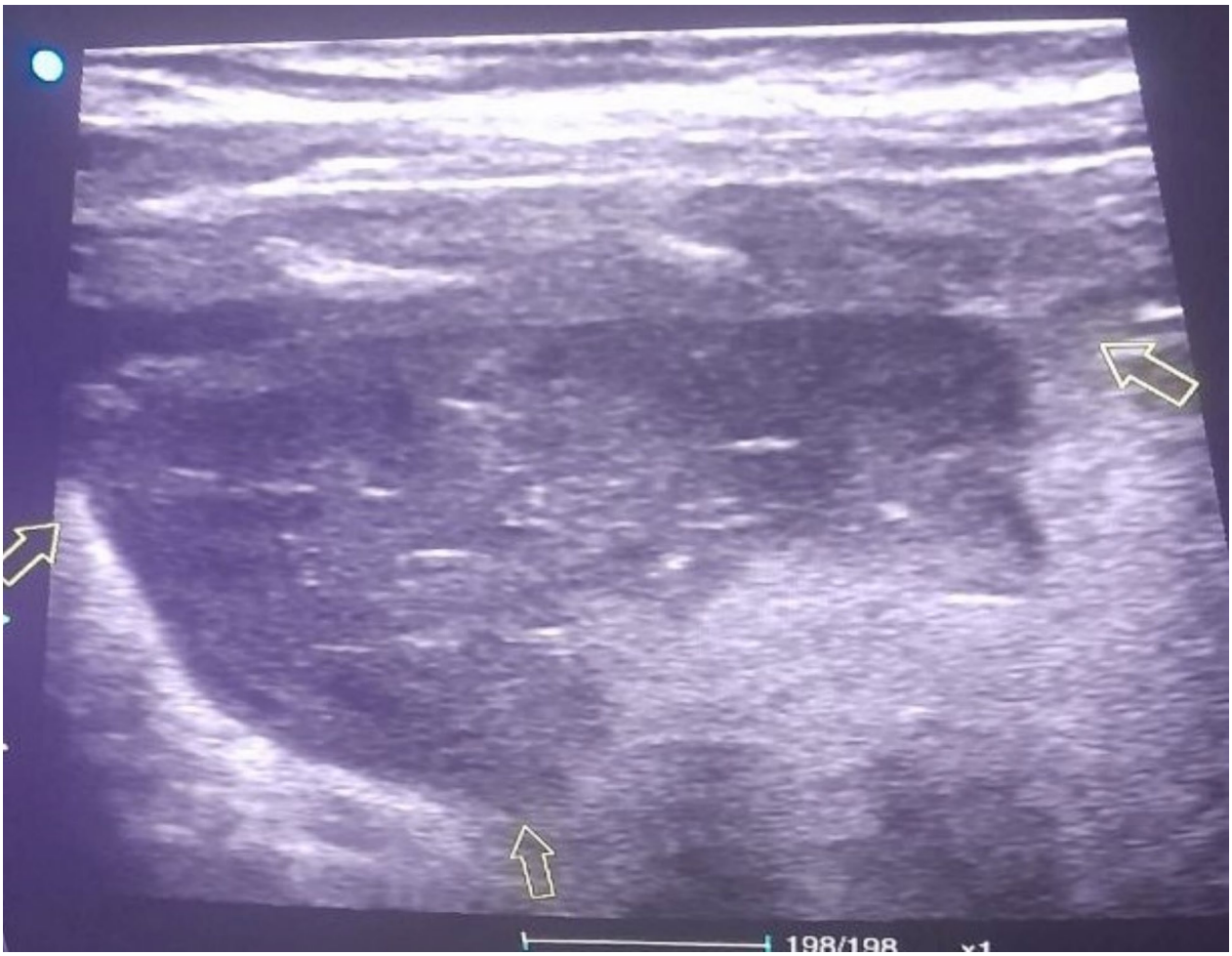


**Fig. 1** Abdominal ultrasound showing splenic infarct

cause severe morbidity if not detected promptly, even though they are rare. As emphasized by Rizvi et al. [17], greater clinician awareness is essential for the timely diagnosis of splenic infarction, especially in endemic areas where *P. vivax* is prevalent.

## Conclusion

As demonstrated by this example, *P. vivax* malaria can no longer be considered consistently benign. Despite their rarity, complications like splenic infarction call for caution, particularly in children who exhibit splenomegaly and abdominal pain. Adequate anti-malarial treatment, early imaging, and careful monitoring are essential for guaranteeing recovery and averting negative consequences.

**Abbreviations**

| | |
|---|---|
| CBC | Complete blood count |
| ALT | Alanine transaminase |
| AST | Aspartate transaminase |
| BUN | Blood urea nitrogen |
| AKI | Acute kidney injury |
| NSAIDS | Non steroidal anti inflammatory drugs |

**Author contributions**

ABT, TTB, and ABS were involved in the conception and design of the study, drafting and revising of the article, and final approval of the version to be submitted and also involved in direct patient management. MAK, MTB, and DDB were involved in the conception and design of the study, drafting and revising the article, and final approval of the version to be submitted.

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**Data availability**

On a valid request, the corresponding author will provide access to the datasets that were gathered and used to conduct this article.

## Declarations

**Ethics approval and consent to participate**
The need for ethical approval was waived.

**Informed consent**
Before preparing the case report, the patient's family provided written informed consent to write the case and be published.

**Competing interest**
The authors declare no competing interests.

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## Publisher's Note



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