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# CLINICAL CASE REPORTS

## Splenic Infarction in a Child with Sickle Cell Trait

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### ABSTRACT

Sickle cell trait is a benign condition, though rare complications were reported, often triggered by well-established trigger factors. We report a case of a seven-year-old male child with sickle cell trait, who presented to the Emergency Department with progressively worsening abdominal pain over the previous three days, radiating to the left shoulder, which began during a trip to high altitude mountains. On admission, the child had signs of peritonitis, as diffuse tenderness and general tympany. The ultrasound revealed splenomegaly with a peripheral triangular area, lacking blood flow, suggestive of infarction. He was admitted to the hospital for hydration and pain management, obtaining symptoms control. Further investigation revealed a recent asymptomatic Epstein-Barr virus infection, which may have been an additional precipitating factor alongside the high altitude. This case highlights the relevance of knowing the portability of HbS for achieving a better control of the risk factors that may cause complications in the long run.

**Keywords:** pediatrics; sickle cell trait; splenic infarction

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## INTRODUTION

Sickle cell syndromes encompass a group of qualitative hemoglobinopathies characterized by the presence of a haemoglobin variant, HbS, which results from a mutation in the gene responsible for encoding the β-globin chains.[1-3] The disease manifests only when two mutated genes are present, either in homozygosity or in combination with other hemoglobinopathies.[1,3]

Sickle cell trait is defined by the presence of only one abnormal β-globin allele.[2] It is more prevalent in regions where malaria is endemic, as it provides protection against severe forms of the infectious disease.[1-4] Due to migratory phenomena, its current distribution is all over the world.[2,3] It is considered a benign condition that typically does not require specific medical care or regular follow-up, except in the context of family planning, where partner screening is essential to prevent more severe forms of haemoglobinopathy.[2,3] In most cases, carriers are asymptomatic, with a life expectancy and quality of life comparable to individuals without the mutation.[2,3,5]

However, certain metabolic and environmental conditions can promote HbS polymerization, potentially leading to complications.[2,3,5] Hypoxia, often associated with high altitudes (typically above 1,100 meters) or intense physical exertion, alters the solubility of HbS, leading to polymerization and subsequent sickling of red blood cells.[2,6] These sickled erythrocytes exhibit reduced deformability, promoting thrombosis and resulting in tissue infarction, commonly in the spleen.[2,5] It is known that the percentage of HbS can influence the prevalence of complications, with increased risk observed when levels exceed 35%.[6]

Urogenital complications, such as haematuria, progression to chronic kidney disease, and renal medullary carcinoma, are well known in this population.[2] Another well-documented complication is the risk of vision loss following traumatic hyphaemia.[2] There is also a slightly increased risk of sudden death and rhabdomyolysis associated with extreme physical exertion in individuals with sickle cell trait.[2,4,7] In addition to splenic infarction, it has been suggested that sickle cell trait may be connected with other complications.[2,4]

## CASE PRESENTATION

This case is about a seven year old Portuguese male, previously healthy, known to be HbS carrier (40.7% in the last test). There was no relevant family history.

He presented to the Pediatric Emergency Department with progressively worsening abdominal pain in the previous three days, radiating to the left shoulder. He also reported anorexia and a single episode of vomiting. The symptoms began suddenly during a car trip to a high mountain in the country side, while ascending in altitude, without associated physical exertion. He denied trauma, fever, bowel habit changes, or recent infections.

Upon admission, he was in discomfort, afebrile, with age-appropriate vital signs. In the physical examination it was noticed pale skin, tympany in the abdomen with diffuse tenderness on palpation, signs of peritonitis, and a palpable spleen 1 cm below the left costal margin.

The laboratory tests (**Table 1**) revealed haemoglobin 11.2 g/dL, reticulocyte count 90,000/µL, platelet count 314,000/µL, and high LDH (601 U/L).

**Table 1 -** Laboratory evaluation (on the admission date)

*Reference value (RV); Mean Corpuscular Volume (MCV); Mean Corpuscular Haemoglobin (MCH); White Blood Cells (WBC); Erythrocyte Sedimentation Rate (ESR); Aspartate Aminotransferase (AST); Alanine Aminotransferase (ALT); Lactate Dehydrogenase (LDH); C-reactive Protein (CRP); Thyroid Stimulating Hormone (TSH); Free Thyroxine (FT4)*

| | | | |
|---|---|---|---|
| **Haemoglobin** | 11.2g/dL (RV: 11.0 – 14.0) | **ESR** | 9mm (RV: < 13) |
| **MCV** | 76.7fL (RV: 72.0 – 86.6) | **AST** | 49U/L (RV: < 44) |
| **HGM** | 26.7pg (RV: 26.0 – 34.0) | **ALT** | 75U/L (RV: < 37) |
| **WBC** | 11,8x$10^9$/L (RV: 4.5 – 13.0) | **Total Bilirubin** | 0.62mg/dL (RV: ≤ 1.20) |
| **Lymphocytes** | 3,9 x$10^9$/L (RV: 1.0 – 7.8) | **Direct Bilirubin** | 0.16mg/dL (RV: ≤ 0.20) |
| **Platelets** | 314 x$10^9$/L (RV: 140 – 440) | **LDH** | 601U/L (RV: 120 - 300) |
| **Reticulocytes** | 90 x$10^9$/L (RV: 50-100) | **Creatinine** | 0.47mg/dL (RV: 0.40 - 0.60) |
| **INR** | 1.0 (RV: < 1.2) | **Urea** | 23.7mg/dL (RV: 15.0 - 36.0) |
| **APTT** | 23.8s (RV: 23.0 - 31.9) | **CRP** | 1.52mg/dL (RV: < 0.50) |
| **Fibrinogen** | 3.2g/L (RV: 1.8 - 3.5) | **TSH** | 3.25mUI/L (RV: 0.60 - 4.84) |
| **D-dimers** | 2261mcg/L (RV: < 500) | **FT4** | 1.37ng/dL (RV: 0.97 - 1.67) |

The abdominal ultrasound (**Figure 1**) showed a slightly enlarged spleen (11.5 cm bipolar diameter) with heterogeneous echotexture in the upper third, where a well-defined, hypoechoic, triangular peripheral area measuring approximately 28 mm in its greatest dimension was observed. There was no vascular flow within this area, suggesting splenic infarction.

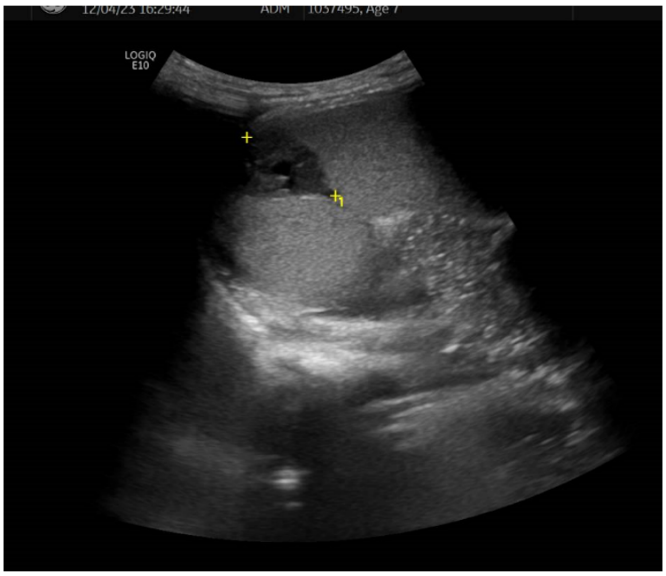


**Figure 1 -** Abdominal ultrasound showing findings consistent with splenic infarction

The patient was admitted for supportive therapy (intravenous fluids, rest, and pain management) and further studies. A rapid symptomatic improvement was observed.

Further investigation identified an asymptomatic acute/recent Epstein-Barr virus (EBV) infection (negative EBV nuclear antigen [EBNA] IgG, positive viral capsid antigen [VCA] IgG and IgM). The patient was not immune to CMV. The peripheral blood smear showed activated lymphocytes and a pleomorphic lymphocyte population.

The diagnosis of splenic infarction was established in a patient with sickle cell trait, probably triggered by altitude exposure and recent mononucleosis. He was discharged two days later, fully asymptomatic with unchanged spleen palpation findings. Preventive guidance was provided regarding avoidance of trigger risk factors, and he was referred to pediatric haematology follow-up.

About one month after the episode, a repeated abdominal ultrasound confirmed a significant reduction in the infarcted area and resolution of the splenomegaly. Clinically, he remained asymptomatic. Repeated EBV serology supported the hypothesis of recent infection (EBNA IgG positive).

## DISCUSSION AND CONCLUSIONS

This child is a carrier of sickle cell trait with an Hb S level >35%, which increases the risk of complications related to blood circulation and tissue oxygenation.

He was brought to an area with an altitude significantly higher than the place he was used to live (1,990 meters above sea level), exposing him to hypoxic conditions. Additionally, the patient had a recent asymptomatic EBV infection. The literature describes several cases of splenic infarction associated with EBV infection.[8,9] Although the exact mechanism is not yet well understood, proposed explanations include splenic hypercellularity and a transient hypercoagulable state.[8,9]

Splenic infarction is typically self-limited, with complete resolution in most cases following supportive therapy.[2,9]

Although sickle cell trait is considered a benign condition, carriers may develop complications, particularly when exposed to certain precipitating factors.[1,3] Identifying these factors and providing appropriate counselling is therefore of critical relevance, both to help avoiding risk exposures and to educate patients and caregivers about symptoms that warrant timely medical evaluation. Such guidance can significantly reduce the morbidity and mortality associated with this condition.[2]

Preventive measures should include maintaining adequate hydration, engaging in moderate and progressive physical activity tailored to individual fitness levels combined with rest periods.[2,5] Patients should also be instructed to recognize early signs and symptoms of complications and to stop activity immediately if they occur.[2,5] In high-altitude settings, a gradual acclimatisation should be encouraged to reduce complications.[2]

It is important to highlight that preconception genetic counselling in essential in these individuals once they plan family life. Their partner should be studied and a medical review should happen before the pregnancy. Fertility techniques may be needed to prevent more severe forms of haemoglobinopathy if the parents have relevant abnormalities.[2]

This case highlights the importance of recognizing and understanding the interaction between genetic, infectious, and environmental factors in managing patients with sickle cell trait.

## ETHICAL CONSIDERATIONS

The authors declare no conflicts of interest. Informed consent was obtained from the child's legal guardian, and confidentiality of the patient's data was ensured.

## AUTHORSHIP

Beatriz Henriques - Investigation; Data curation, Writing –

original draft
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