WILDERNESS & ENVIRONMENTAL MEDICINE, ▮, ▮▮▮–▮▮▮ (2017)

CASE REPORT

A Case of Autosplenectomy in Sickle Cell Trait Following an Exposure to High Altitude

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A 24-year-old man presented with acute abdominal pain upon ascent to moderate altitude (3500 m). An immediate evaluation revealed a splenic infarct, and he was evacuated to sea level. Upon recovery, he was sent back to 3500 m without detailed etiological evaluation, whereupon he experienced recurrent episodes of left-side subcostal pain. Imaging suggested autosplenectomy, and workup revealed a negative thrombophilia profile but was positive for sickle cell trait (SCT). Individuals with SCT can be asymptomatic until exposure to severe hypoxia, upon which they can manifest clinically as sickle cell syndrome. We discuss the rare presentation of autosplenectomy in a patient with previously undiagnosed SCT on exposure to high altitude.

Keywords: splenic infarct, sickle crisis, sickling syndrome

Background

Sickle cell trait (SCT) is common in the Indian population, with prevalence up to 13%.[1,2] It is a benign condition that goes undiagnosed in the majority of cases unless neonatal screening is done, but neonatal screening is not routine in the Indian subcontinent. SCT is a carrier condition in which the heterozygosity will rarely lead to clinical manifestations. The importance of SCT detection lies in the coinheritance of other hemoglobinopathies and concern of homozygous state in the next generation if the individual's spouse is also a carrier. Rarely, SCT can present with a vaso-occlusive phenomenon or features of sickle cell disease when the patient is exposed to severe hypoxia-related conditions like high altitude.[3] We discuss the rare presentation of autosplenectomy in a case of SCT on exposure to high altitude and suggest screening for SCT in cases presenting with splenic infarction at high altitude.

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Submitted for publication November 2016.
Accepted for publication August 2017.

Case presentation

A 24-year-old apparently healthy central police force member with no known comorbidities became symptomatic on ascent to moderate altitude (3500 m) as part of military service. He developed high-grade fever, severe pain in the left upper quadrant, vomiting, and weakness 1 day after exposure to high altitude. He had no other associated symptoms suggestive of any other high-altitude illnesses. He gave no history of similar symptoms in the past. Medical and family histories were noncontributory. Military history was noncontributory because he had never served in high-altitude areas before this exposure, and he did not have any similar symptoms on physical exertion during low-altitude training activities. He was a nonsmoker and did not consume alcohol. He was managed as a case of acute mountain sickness with no improvement in his condition.

On initial evaluation, the patient was of average build (height 170 cm, weight 64 kg, and body mass index 22.1 $kg \cdot m^{-2}$). All vital signs were within normal limits except for mild tachycardia (pulse rate 104 $beats \cdot min^{-1}$). Local examination revealed no abnormality. Systemic examination revealed tenderness in left subcostal and left lumbar region with rigidity and decreased bowel sounds. No other abnormality was detected.

Table 1. Laboratory evaluation of the patient

Investigation	*High altitude (at first presentation)*	*Sea level (at time of SCT diagnosis)*
Hb ($g \cdot dL^{-1}$)	11.4	16.9
TLC ($count \cdot uL^{-1}$)	19,900	9500
Platelet ($count \cdot uL^{-1}$)	490,000	521,000
Bilirubin ($mg \cdot dL^{-1}$)	1.3	1.1
AST ($IU \cdot L^{-1}$)	14	16.3
ALT ($IU \cdot L^{-1}$)	23	18.6
ALP ($IU \cdot L^{-1}$)	121	101.9
Urea ($mg \cdot dL^{-1}$)	26	10.1
Creatinine ($mg \cdot dL^{-1}$)	0.9	0.8

SCT, sickle cell trait; Hb, hemoglobin; TLC, total leukocyte count; AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase.

The patient's biochemical and hematological tests done at high altitude were within normal limits except for mild leukocytosis (Table 1). Ultrasonographic findings revealed diffusely hyperechoic splenic parenchyma measuring 12.6x3 cm with Doppler showing no flow. A large supracapsular collection measuring 5x9.4x12.9 cm of approximately 320 mL containing tiny echogenic foci was noted. (Figure 1). He was diagnosed with a case of splenic infarction that was confirmed by abdominal contrast-enhanced computed tomography. He was taken to sea level after conservative management with analgesics. He was further evaluated at a nonmilitary tertiary care center at sea level. His evaluation for thrombophilic states (protein C, protein S, antiphospholipid antibody syndrome workup, factor V Leiden mutation analysis, and prothrombin gene mutation analysis) was unremarkable. Evaluation for any malignancy by radiological evaluation and biomarkers was negative. Doppler evaluation of splenic vessels was normal with no splenic cavernomas, thrombosis, or reversal of flow pattern.

After receiving therapy, he recovered well and his pain subsided. After descent to sea level with no

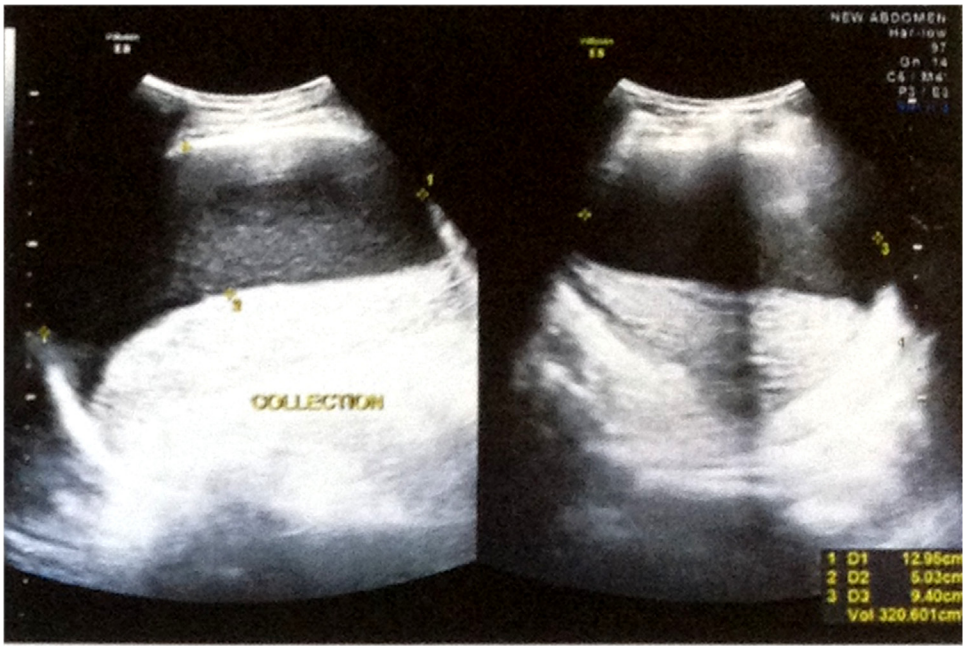

Figure 1. (Left) Ultrasonographic findings revealed diffusely hyperechoic splenic parenchyma measuring 12.6 × 3 cm in size with Doppler showing no flow. (Right) A large supracapsular collection approximately 320 mL noted with tiny echogenic foci within measuring 5 × 9.4 × 12.9 cm.

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