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Acute Pancreatitis Complicated by Subacute Intestinal Obstruction and Acute Chest Syndrome in a Patient with Sickle Cell Disease: A Case Report

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ABSTRACT

Background

Sickle cell disease (SCD) is a hereditary hemoglobinopathy associated with chronic haemolysis, vaso-occlusion, endothelial dysfunction, and multiorgan complications. Acute abdominal pain in SCD is diagnostically challenging because vaso-occlusive crisis may mimic pancreatitis, bowel obstruction, mesenteric ischemia, or hepatobiliary disease. Acute pancreatitis and intestinal obstruction are uncommon complications, while acute chest syndrome (ACS) remains a major cause of morbidity and mortality. Their simultaneous occurrence during a single admission is rare and requires prompt multidisciplinary recognition and management.

Case Report

A 26-year-old male with known SCD and beta-thalassemia trait presented with acute abdominal pain associated with vomiting, loose stools and fever. The abdominal pain was preceded by alcohol intake. Examination revealed abdominal tenderness and sluggish bowel sounds. Laboratory investigations showed anemia, leukocytosis, thrombocytopenia, deranged liver function tests and markedly elevated serum amylase and lipase. Erect abdominal radiography showed multiple air-fluid levels suggestive of subacute intestinal obstruction or ileus. Contrast-enhanced computed tomography revealed auto-splenectomy, mildly dilated bowel loops without definite mechanical obstruction. He was managed conservatively with bowel rest surgical monitoring and supportive care. He subsequently developed signs consistent with ACS. Intensive care management included oxygen supplementation, blood transfusion, broad-spectrum antibiotics and supportive management. With appropriate intensive care his haemodynamic parameters improved and he was discharged in stable condition.

Conclusion

Persistent abdominal pain in SCD should not be solely attributed to vaso-occlusive crisis. Early abdominal imaging, pancreatic enzyme estimation, respiratory assessment and multidisciplinary conservative management is essential for detecting overlapping pancreatitis, functional intestinal obstruction and ACS in cases of SCD.

Keywords: Acute Chest Syndrome, Anemia, Sickle Cell, Intestinal Obstruction, Pancreatitis, Beta-Thalassemia.

INTRODUCTION

Sickle cell disease (SCD) is a hereditary hemoglobinopathy caused by structurally abnormal hemoglobin S, resulting in chronic haemolysis, recurrent vaso-occlusion, endothelial dysfunction, and progressive multiorgan injury. Although traditionally considered most prevalent in sub-Saharan Africa, the Middle East, the Mediterranean basin, and parts of the Americas, SCD also represents an important public health problem in India, where the sickle gene is widely distributed among tribal populations, with reported heterozygote prevalence varying from 1% to 40% across communities.[1] The clinical course is heterogeneous and influenced by genotype, fetal hemoglobin levels, co-inherited thalassemia, infection, dehydration, and access to preventive care. In adults with SCD, acute abdominal pain is a frequent reason for emergency presentation and hospitalization, but it poses a diagnostic challenge because vaso-occlusive abdominal crisis may mimic several surgical and medical emergencies. Distinguishing uncomplicated abdominal vaso-occlusion from pancreatitis, hepatobiliary disease, splenic complications, mesenteric ischemia, or intestinal obstruction is essential, as delayed recognition may increase morbidity.[2]

Acute pancreatitis is an uncommon but increasingly recognized cause of abdominal pain in patients with SCD.

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Its pathogenesis may be multifactorial, including pigment gallstone disease secondary to chronic haemolysis, biliary obstruction, and microvascular occlusion of the pancreatic circulation during vaso-occlusive crisis. More recently, systematic review data have reinforced that the overlap between abdominal vaso-occlusive pain and pancreatitis can obscure the diagnosis, particularly when symptoms are attributed solely to a pain crisis. Early measurement of serum amylase and lipase, appropriate imaging, correction of dehydration and hypoxia, analgesia, bowel rest, and careful evaluation for gallstone disease are therefore central to management.[3]

Acute chest syndrome (ACS) is among the most serious acute complications of SCD and remains a major contributor to hospitalization, intensive care admission, and mortality. It is generally defined by the presence of a new pulmonary infiltrate on chest imaging accompanied by fever and/or respiratory symptoms such as cough, dyspnoea, tachypnoea, hypoxemia, or chest pain. ACS may arise due to infection, pulmonary infarction, fat embolism, atelectasis, or hypoventilation secondary to pain and opioid therapy.[4]

Subacute intestinal obstruction or ileus in SCD is less frequently reported than painful crisis or ACS but is clinically significant because it can resemble a surgical abdomen. Proposed mechanisms include sickling within the mesenteric and splanchnic microvasculature, bowel wall ischemia, autonomic dysmotility, opioid-related bowel hypomotility, electrolyte disturbances, and systemic inflammatory stress. Many authors have reported increased occurrence of ileus among patients with SCD and suggested that impaired bowel perfusion may contribute to intestinal dysmotility in this population. In clinical practice, the distinction between mechanical obstruction, ischemic bowel injury, pancreatitis-associated ileus, and functional obstruction related to vaso-occlusion is often difficult. This distinction is particularly important because many patients improve with conservative measures, hydration, decompression, correction of precipitating factors, transfusion support where appropriate, and close surgical observation, whereas delayed recognition of bowel ischemia can be catastrophic.[5]

The simultaneous occurrence of acute pancreatitis, subacute intestinal obstruction, and ACS during a single hospitalization in a patient with SCD is rare and diagnostically demanding. Existing literature largely describes these complications as isolated entities or as pairwise associations, while reports documenting their temporal coexistence and clinical interaction remain limited. This creates a knowledge gap regarding early recognition, pathophysiological linkage, monitoring priorities, and sequencing of multidisciplinary management when abdominal and pulmonary complications evolve together. We present the case of a 26-year-old male with known SCD and beta-thalassemia trait who developed acute pancreatitis complicated by subacute intestinal obstruction and ACS during the same admission at a tertiary care center.

CASE REPORT

A 26-year-old male, a known case of sickle cell disease with beta-thalassemia trait, presented to the casualty department of a tertiary care teaching hospital with acute abdominal pain of approximately five hours' duration. The pain was gradual in onset, progressive, diffuse, and non-radiating, and was associated with vomiting, loose stools, and fever. There was no history of abdominal trauma. He had a past history of blood transfusion approximately two to three years earlier and no similar illness was reported among family members. A history of alcohol intake preceding the current episode was elicited. At presentation, he appeared distressed and febrile.

During admission, the abdominal findings showed mild tenderness, guarding, abdominal distension and sluggish bowel sounds. Baseline laboratory evaluation revealed anemia as well as leukocytosis and thrombocytopenia. Additioally mildly elevated inflammatory markers and deranged liver function tests were also present. Renal function was preserved. Dengue and hepatitis B screening were negative. Markedly elevated serum amylase and lipase supported the diagnosis of acute pancreatitis. Peripheral blood smear examination showed normocytic normochromic anemia with mild anisopoikilocytosis and leukocytosis initially. A subsequent smear demonstrated dimorphic anemia with microcytic hypochromic and macrocytic red cells, macro-ovalocytes, elliptocytes, target cells, nucleated red blood cells, and persistent leukocytosis, with no malarial parasite identified (Table 1).

Contrast-enhanced computed tomography of the abdomen and pelvis showed a normal-sized liver with reduced attenuation and no focal lesion. The gallbladder was distended but the common bile duct was normal. The pancreas was normal in size and attenuation without focal lesion, calcification, or pancreatic duct dilatation. The spleen was small, shrunken, lobulated, and diffusely calcified with residual low-attenuation splenic parenchymal areas, consistent with auto-splenectomy secondary to sickle cell disease. Mildly dilated bowel loops were present without a definite transition point, obstructing lesion, mural thickening, pneumatosis, or free intraperitoneal air, suggesting ileus or functional bowel dilatation rather than definite mechanical obstruction. A small left renal upper calyceal calculus measuring approximately 2.5 mm was also noted. Erect abdominal radiography demonstrated multiple air-fluid levels which could be correlated clinically with subacute intestinal obstruction.

The patient was initially managed with intravenous fluids, analgesics, antiemetics, proton pump inhibitor therapy, thiamine supplementation, and bowel rest. Surgical consultation was obtained because of clinical and radiological suspicion of subacute intestinal obstruction. Per rectal examination revealed semisolid stool stained with blood, with normal sphincter tone and no external anorectal pathology. Conservative management was continued, including nil per oral status, intravenous fluids, antispasmodic support, and enema as advised. Urology consultation was also sought in view of haematuria, and urine examination, urine culture, input-output monitoring, renal function monitoring, and catheterization if required were advised.

With markedly elevated pancreatic enzymes and a preceding history of alcohol intake, the working diagnosis was refined to acute pancreatitis, likely alcohol-associated, occurring in the background of sickle cell-beta thalassemia and functional bowel obstruction/subacute intestinal obstruction. The patient continued to have leukocytosis and developed worsening anemia during hospitalization, with hemoglobin progressively falling to 7.6 g/dL, then 6.6 g/dL, and subsequently 6.3 g/dL. Liver function tests also showed transient worsening of hyperbilirubinemia, with total bilirubin rising markedly before later improvement. In view of sickle cell crisis with significant anemia, hydroxyurea and folic acid were added, and packed red blood cell transfusion was administered under monitoring after pre-transfusion medication.

During hospitalization, the patient developed chest pain, back pain, tachypnoea, tachycardia, and oxygen desaturation, which were clinically suggestive of acute chest syndrome in the setting of sickle cell disease. He was managed in intensive care with oxygen supplementation, intravenous fluids,

analgesia, packed red blood cell transfusion, anticoagulation, and broad-spectrum antibiotics, which were escalated because of persistent fever and suspected pneumonia or sepsis. Blood and urine cultures were sent. With supportive and conservative management his respiratory status, fever and bowel symptoms gradually improved and post-transfusion hemoglobin also improved (Table 2). He was able to tolerate oral feeds and was continued on hydroxyurea, folic acid, supportive medication, and monitoring. At discharge, he had no fresh complaints, was afebrile and hemodynamically stable, with blood pressure of 120/80 mmHg, pulse rate of 94/min, respiratory rate of 12/min, and oxygen saturation of 96% on room air. Systemic examination was unremarkable. The final clinical diagnoses were subacute intestinal obstruction, sickle cell disease/sickle cell crisis, acute pancreatitis likely secondary to alcohol intake, and acute chest syndrome. He was discharged in stable condition with advice for regular follow-up. At the time of discharge, he was prescribed hydroxyurea 500 mg once daily, folic acid 5 mg once daily and B-complex supplementation. Counselling regarding strict avoidance of alcohol and other precipitating factors for sickle cell crisis was done.

Parameter	Result	Reference Range
CBC (Blood Routine)		
Haemoglobin (Hb)	**9.2**	13–17 g/dL (Male)
WBC	18,900	—
Polymorphs	61%	40–75%
Lymphocytes	36%	20–45%
Eosinophils	01%	1–6%
Monocytes	02%	2–8%
Basophils	00%	0–1%
Platelet Count	**87,000**	1.5–4.5 L
Inflammatory Markers		
CRP	**8.01**	0–6 mg/L
ESR	2 mm/hr	0–15 mm/hr
Electrolytes		
Sodium	135.2	136–145 mEq/L
Potassium	4.49	3.5–5.1 mEq/L
Chloride	101.9	98–107 mEq/L
Liver Function Tests		
Serum Bilirubin (Total)	**2.2**	0–2 mg/dL
Serum Bilirubin (Direct)	**0.5**	0–0.2 mg/dL
Total Protein	**5.6**	6–7.8 g/dL
Albumin	3.7	3.5–5.2 g/dL
SGPT (ALT)	35	5–35 U/L
SGOT (AST)	**117**	5–45 U/L
Alkaline Phosphatase	117	80–306 U/L
Renal Function Tests		
Blood Urea	25	15–40 mg/dL
Serum Creatinine	0.7	0.4–1.3 mg/dL
Other		
Blood Group	B Positive	—
HBsAg	Negative	—
Dengue IgM ELISA	Negative	—
Serum Amylase	**438 U/L**	Normal range
Serum Lipase	**1126 U/L**	Normal range

Table 1: Laboratory Investigations at Admission

Parameter	On Admission	Day 3	Day 5	Day 6	Day 8
Hb (g/dL)	9.2	**7.6**	**6.6**	**6.3**	8.6
WBC	18,900	**25,500**	**28,900**	**27,600**	**29,300**
Platelets	**87,000**	2.70 L	3.13 L	3.06 L	1.96 L
T. Bilirubin	—	—	**3.8**	—	—

Table 2: Serial Haematological Parameters During Hospitalization

DISCUSSION

This case underscores the diagnostic complexity of a patient presenting with acute abdominal pain in sickle cell disease (SCD). Colah R et al emphasized that the sickle gene is widely distributed in Indian tribal populations.[6] There is heterozygote prevalence reported from 1% to 40%, underscoring the relevance of keeping a high index of suspicion for SCD-related complications in Indian clinical practice. Ebert EC et al similarly described the broad range of gastrointestinal and hepatic manifestations of SCD, including abdominal pain syndromes that may mimic surgical emergencies.[7] In comparison with these reports, the present case is notable not merely for abdominal vaso-occlusive pain but for the coexistence of pancreatitis, subacute intestinal obstruction /ileus, and ACS in a single clinical course.

Acute pancreatitis was a central diagnostic consideration in this patient because serum amylase and lipase were significantly elevated, and the abdominal symptoms were persistent and progressive. Although alcohol intake preceded the episode and may have contributed, SCD itself provides additional mechanisms for pancreatic injury, including microvascular occlusion, ischemia, dehydration, haemolysis-related pigment stone disease, and systemic inflammatory stress. Ahmed S et al reported four cases of acute pancreatitis

during sickle cell vaso-occlusive painful crisis.[8] The authors proposed that pancreatic ischemia causing microvascular occlusion may be the cause of pancreatitis in these cases. Dike CR et al in a systematic review of acute pancreatitis in individuals with SCD reiterated that abdominal pain from pancreatitis may be difficult to distinguish from abdominal vaso-occlusive crisis and that delayed recognition may occur when symptoms are attributed only to sickling pain.[9] The present case is concordant with both studies because biochemical testing was decisive even though contrast-enhanced computed tomography did not show overt pancreatic enlargement or ductal abnormality. This discrepancy reinforces that early pancreatitis may be enzyme-positive before definite structural changes appear on imaging, and that pancreatic enzyme testing should be considered in SCD patients with persistent abdominal pain, vomiting, fever, leukocytosis, or unexplained clinical deterioration.

The development of abdominal distension, guarding, sluggish bowel sounds, multiple air–fluid levels, and mildly dilated bowel loops without a clear transition point favoured functional obstruction or ileus rather than definite mechanical obstruction. Helvaci MR et al. reported increased ileus among patients with SCD.[10] However, the presence of guarding and blood-stained stool on rectal examination justified careful monitoring because bowel ischemia, mesenteric vaso-occlusion, pancreatitis-associated ileus, opioid-related hypomotility, and infectious colitis may overlap clinically. Compared with the case described by Khosla A et al [11] this case further demonstrates that ileus in SCD may occur in the setting of simultaneous pancreatitis and evolving systemic crisis, rather than as an isolated abdominal complication.

However, in this case subsequent onset of chest pain, back pain, tachypnoea, tachycardia, and oxygen desaturation was clinically consistent with ACS. Maitre B et al[12] reported adult ACS episodes and noted that adults may have severe disease, often requiring close monitoring and transfusion support. In the present case, ACS developed after the abdominal presentation, which is clinically important because abdominal pain, splinting, basal atelectasis, dehydration, systemic inflammation, opioid exposure, and immobilization may all precipitate pulmonary sickling. The basal atelectatic changes noted on abdominal CT were an early warning sign, and the later fall in oxygen saturation justified intensive monitoring, oxygen therapy, broad-spectrum antimicrobials, anticoagulation, and packed red cell transfusion. This temporal sequence is similar to prior observations that ACS may develop during hospitalization for a painful or non-pulmonary crisis, and it supports serial respiratory assessment in SCD patients admitted with pancreatitis or abdominal obstruction. The favorable outcome in this case was due to intensive supportive care, repeated reassessment of complications and multidisciplinary management. In the present patient, hemoglobin fell from 9.2 g/dL to 6.3 g/dL before improving after packed red cell transfusion, while abdominal symptoms, fever, and oxygen saturation improved with supportive treatment.

CONCLUSION

Persistent abdominal pain in SCD should not be presumed to be uncomplicated vaso-occlusive crisis and pathologies such as pancreatitis and intestinal obstruction must be actively excluded with imaging and serum enzymes. Bowel dilatation without transition point may be managed conservatively under surgical supervision and any new tachypnoea, chest pain, or desaturation should prompt urgent evaluation for ACS. The simultaneous occurrence of pancreatitis, functional intestinal obstruction and ACS makes this case unusual and clinically instructive especially for tertiary centres managing adult SCD patients.

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