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CASE REPORT

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Splenic hematoma following acute pancreatitis, and the role of conservative management: A case report

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Key clinical message: We reported a unique case in which the patient developed splenic hematoma following acute pancreatitis, the condition responded well to conservative management without any surgical intervention.

Abstract: Splenic hematoma following acute pancreatitis is a rare complication that is thought to be due to the distribution of pancreatic exudates to the spleen. We presented a case of a 44-year-old patient with acute pancreatitis who developed a splenic hematoma. He responded well to conservative management and the hematoma was resolved.

KEYWORDS

acute pancreatitis, conservative treatment, splenic artery, splenic hematoma

1 | INTRODUCTION

Acute pancreatitis is inflammation of the pancreas; it is sometimes associated with a systemic inflammatory response that can impair the function of other organs or systems. The wide spectrum of causes includes gall- stones (50%) and alcohol (25%). Rare causes (<5%) include drugs (e.g., valproate, steroids, azathioprine), endoscopic retrograde cholangiopancreatography, hypertriglyceridemia or lipoprotein lipase deficiency, hypercalcemia, pancreas divisum, and some viral infections (mumps, coxsackie B4). About 10% of patients have idiopathic pancreatitis, where no cause is found.[1] Splenic complications in pancreatitis are uncommon, with subcapsular hematoma being extremely rare with an incidence of only 0.4%.[2] Splenic complications of pancreatitis include splenic vein thrombosis, arterial pseudoaneurysm, subcapsular splenic hematoma, and splenic rupture.[3] Most of the time secondary splenic injuries go unnoticed leading to asymptomatic splenic masses. Timely recognition and diagnosis of these masses are difficult due to the absence of specific signs and symptoms.[4] Those who are symptomatic, however, can present with referred abdominal and shoulder pain warranting the use of CT and MRI for further exploration.[5] Early assessment of these hematomas and careful monitoring can decrease the chances of splenic rupture and the need for invasive surgical intervention. The management of these hematomas is ultimately decided by the

patient's condition, taking into consideration the patient's hemodynamic stability status, risk of rupture, and grading of their hematomas.[6] Here we present a unique case of a splenic hematoma in a patient secondary to acute pancreatitis who was treated successfully through conservative management. The work has been done in accordance with CARE guidelines.[7]

2 | CASE REPORT

A 43-year-old male patient was brought to the emergency unit of a tertiary care hospital with left upper quadrant abdominal pain for 1 day. The patient described the pain to be severe in intensity, radiating to the back, and reported no relief with over-the-counter painkillers. It was not associated with nausea, vomiting, and fever. There was no history of hematemesis or melena. No fever or chills and trauma were reported. The patient had a history of multiple admissions previously with left hypochondrial pain and vomiting which were managed conservatively. Records of the patient revealed a history of diabetes mellitus, hypertension, and dyslipidemia.

On evaluation, his vital signs were noted to be stable He was afebrile, oxygen saturations at 98% on room air, respiratory rate was 18 breaths per minute, pulse was 88 beats per minute, blood pressure was 138/75 mmHg, and displayed a Glasgow Coma Scale (GCS) that was 15/15. The findings of the physical examination showed the abdomen was soft, tender, and having sluggish bowel sounds. Initial laboratory investigations revealed increased white blood cell count, and elevated serum lipase, amylase levels, and CRP (Table 1).

A computed tomographic (CT) scan of the abdomen was ordered and displayed pancreatitis with associated fat stranding and mild ascites (Figure 1). The imaging showed no evidence of pancreatic necrosis, cholelithiasis, or ductal dilation.

Based on the CT scan and clinical findings that is abdominal pain and laboratory findings of raised white blood cells, serum amylase and lipase lead to the diagnosis of acute pancreatitis. The patient was admitted to the medical unit and management was initiated for acute pancreatitis he was resuscitated with intravenous (IV) normal saline, ketorolac tromethamine, and omeprazole. On Day 2 of admission, the patient noted mild improvement in his symptoms. However, early on the third day, the patient complained of worsened abdominal pain, now localized more to the left hypochondrium and also had referred pain at the left shoulder. He continued to have discomfort and severe pain despite extensive intravenous analgesics. Repeat investigations revealed hemoglobin dropped from 12.7 g/dL to 7.7 g/dL with no signs of active bleeding from any site. This raised the concern for a concealed hemorrhage, prompting a packed cell transfusion and CT angiogram. The patient was shifted for the CT angiogram to ICU. A total of three packed red blood cells were transfused. An angiogram of the splenic artery and left inferior phrenic artery was done, showing no vascular malformation or active bleeding (Figure 2).

A second abdominal CT scan with contrast was suggested for the review and comparison with previous scan findings and for the identification of the underlying pathology. The CT scan finding showed a splenic hematoma with extracapsular distension and mass effect over the stomach, most likely representing the sequelae of acute pancreatitis (Figure 3).

The patient achieved hemodynamic stability, allowing for the transfer of the patient to a special care unit for close observation. Gradual improvement in the symptoms was observed. A decrease in the splenic hematoma was also observed in the subsequent CT scan (Figure 4). The patient

TABLE 1 Initial laboratory investigations.

Investigations	Results	Normal range
Hemoglobin	12.1 g/dL	(12.3–16.6)
WBC	18,600 cells/L	(4.8–11.3)
Platelet count	434,000 cells/L	(154–433)
Lipase	723 U/L	(6–51)
Amylase	298 U/L	(28–100)
Albumin	4.2 g/dL	(3.5–5.2)
CRP	184.51 mg/L	(0–10)

Abbreviations: CRP, C-reactive proteins; WBC, white blood cell.

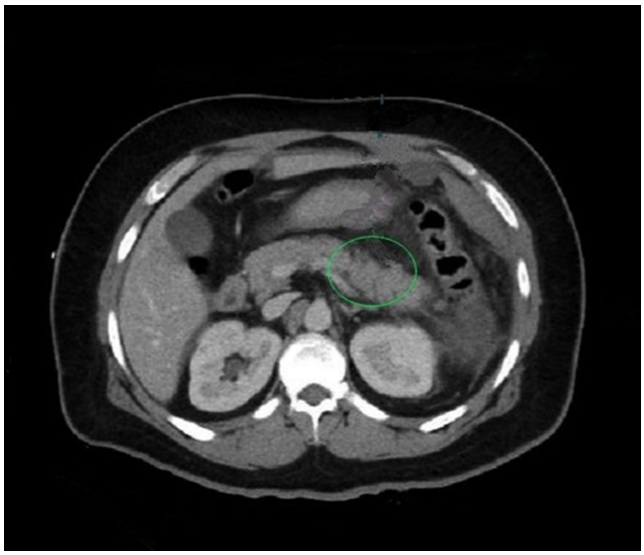

FIGURE. 1 Computed tomographic (CT) scan of the abdomen showing peripancreatic inflammatory changes in the distal pancreas.

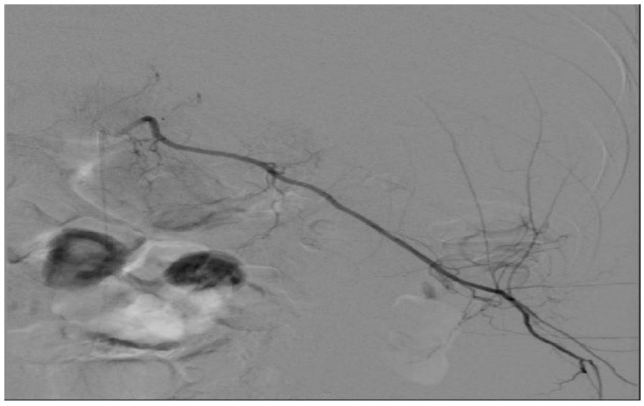

FIGURE. 2 Computed tomographic (CT) angiogram displaying a splenic artery without any vascular malformation nor active bleeding.

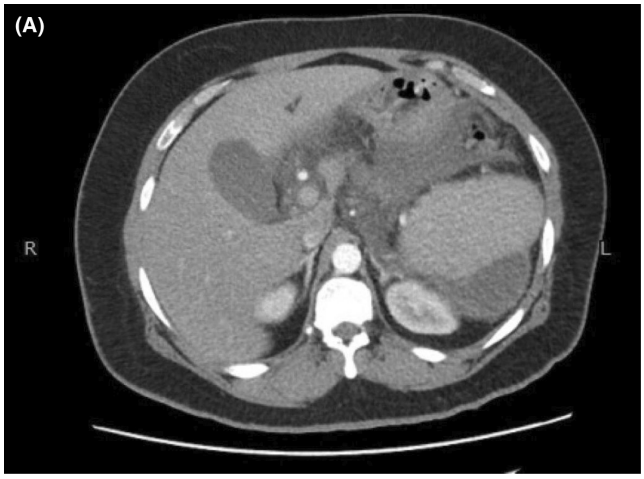

FIGURE. 3 Computed tomographic scan of the abdomen representing splenic hematoma with extracapsular distension and mass effect over the stomach (A).

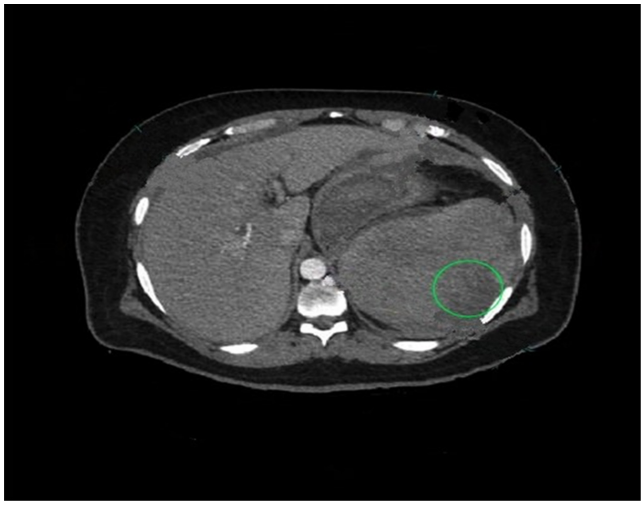

FIGURE. 4 Follow-up computed tomographic scan of the abdomen representing regression of splenic hematoma represented by a green circle.

was ultimately discharged on oral medicines that included syrup antacid 10 mL oral before meals, capsule dexlansoprazole 60 mg oral before breakfast and dinner, tablet ondansetron HCL 8 mg oral before breakfast and dinner, tablet tramadol + paracetamol 50 mg oral two times a day.

Follow-up at 4 week was done, subsequent CT imaging were suggested to the patient which showed progressive regression of hematoma and the patient remained symptom-free.

3 | DISCUSSION

Acute and chronic pancreatitis can have deleterious effects on the spleen, as the organ lies near the pancreas. Moreover, diseases of the pancreas can progress to affect the spleen, leading to complications such as pseudocysts, splenic hematomas, splenic ruptures, and hemorrhagic spleens. Subcapsular hematomas, of note, are commonly seen in patients with chronic pancreatitis.[8]

The spleen is more commonly affected by diseases involving the distal part of the pancreas, as anatomically these structures abut one another.[6] The distal part of the pancreas has the possibility of coming into contact with the hilum of the spleen (enclosed by splenic vessels) in times of swelling. Involvement of the spleen in chronic pancreatitis has been well documented and is being increasingly reported in acute pancreatitis.[9] Pancreatic pseudocysts by way of tryptic erosion can cause splenic vein thrombosis, splenic arterial hemorrhage, and splenic infarction. Pancreatic enzymes that dissect into the spleen may directly erode the splenic parenchyma, small intrasplenic or hilar vessels resulting in intrasplenic hemorrhage The blood may be contained within the pseudocyst or it may dissect beneath the splenic capsule to form a subcapsular haematoma.[10]

Patients with subcapsular splenic hematomas may present with diffuse left upper quadrant pain, possible abdominal guarding, and referred pain in the left shoulder. Elevation of the left diaphragm, tachycardia, hypotension, and decreased hemoglobin can raise suspicion of splenic complications.[6] Imaging and vitals analysis plays an important role in aiding the diagnosis of asymptomatic splenic hematomas and their detection. The CT scan and CT angiogram findings displayed no open splenic rupture as the hematoma was confined to the splenic capsule. The patient remained asymptomatic, but the dropping of hemoglobin and CT scan suggested splenic complications. Previous studies have reported that splenic hematomas and ruptures are mainly present in patients with histories of chronic pancreatitis,[11] whilst our patient had no prior history of pancreatitis or any pancreatitis-specific symptoms.

The management of splenic hematomas following acute or chronic pancreatitis remains controversial.

Conservative approach or surgical interventions depend on the patient's hemodynamic status, size and grading of hematoma, persistent symptoms, and risk of rupture. Some studies have suggested that the splenic hematomas caused by acute pancreatitis can be managed through nonsurgical means and that, if necessary, their surgical indication is based on relevant clinical findings.[12] Conservative treatment includes observation and/or embolization (requiring strict follow-up with ongoing ultrasound or CT scans to observe the reduction in the size of the hematoma). Surgical management includes laparotomy, splenectomy, and percutaneous drainage.[13]

Given the fact that our patient was hemodynamically stable, had no active bleeding, and had no vascular malformations seen on the angiogram, a conservative approach was followed. A similar subcapsular splenic hematoma following pancreatitis was treated conservatively and significant resolution of the hematoma in the abdominal CT was reported and have proposed conservative treatment in hemodynamically stable patients showing clinical improvement.[14] It was also suggested that splenic hematomas larger than 5 cm caused by pancreatitis should be managed surgically through percutaneous drainage or laparotomy as early as possible to reduce pressure and avoid future splenic ruptures.[15]

4 | CONCLUSION

Splenic hematoma secondary to acute pancreatitis is a rare phenomenon that can be treated conservatively when diagnosed early. Conservative measures prove to be effective for patients who are hemodynamically stable and who do not require emergent surgical intervention. Early diagnosis of this splenic complication with acute pancreatitis needs to be further explored in the literature as it can be life-threatening to patients.

AUTHOR CONTRIBUTIONS

Zulfeqar Ali: Conceptualization; data curation; investigation; supervision; validation; writing – original draft. **M.Masood Karim:** Conceptualization; investigation; methodology; resources; writing – original draft. **Maria Javeed:** Conceptualization; data curation; investigation. **Qaisar Ali Khan:** Methodology; project administration; writing – original draft; writing – review and editing. **Christopher Farkouh:** Writing – original draft; writing – review and editing. **David Zepeda:** Writing – original draft. **Parsa Abdi:** Writing – original draft. **Michelle Anthony:** Writing – original draft. **Mathew Farkouh:** Writing – review and editing. **Arooba Khan:** Writing – review and editing. **Bikona Ghosh:** Writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The author declares no competing conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

PATIENT CONSENT

Written informed consent was obtained from the patient to publish this case report and any accompanying images and can be available upon editor's request.

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