International Journal of Gastroenterology and Hepatology
Case Report

A Multidisciplinary Management Approach of a Splenic Artery Pseudoaneurysm Complicating Acute Pancreatitis

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Citation: *Abelardo Broceta Martinez, Carlos M. Pachon Ramos, Matthew D. Razavian, Sahar S. Abdelmoneim, Odalys Frontela. A Multidisciplinary Management Approach of a Splenic Artery Pseudoaneurysm Complicating Acute Pancreatitis. Int Jour Gastro Hepat. 2023;2(1):1-6.*

Received Date: 22 February, 2023; **Accepted Date:** 03 March, 2023; **Published Date:** 10 March, 2023

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ABSTRACT

The early recognition of Splenic Artery Pseudoaneurysm (SAP) as a complication of acute pancreatitis is imperative to implement urgent treatment and prevent any life-threatening events from rupture or hemorrhages which may occur in 58% of cases. We herein present a case of a 37-years-old female who presented to the emergency department with the complaints of acute abdominal pain. The revised Atlanta Criteria to diagnose acute necrotizing pancreatitis was met and a 1.8-centimeter SAP as a complication of acute pancreatitis was noted on imaging. This case report highlights the importance of having a multidisciplinary management approach to promptly recognize SAP in the clinical presentation of acute pancreatitis.

Keywords: Splenic Artery Pseudoaneurysm (SAP); Emergency department; Pancreatitis

INTRODUCTION

The occurrence of Splenic artery pseudoaneurysm (SAP) represents 60% of all visceral aneurysms, making it the most commonly visceral artery affected with pseudoaneurysm.[1] The most common etiologies of SAP include pancreatitis in 52% of cases (acute in 6% and chronic in 46%),[2] abdominal trauma in 29%, post-surgical complications in 3%, and peptic ulcer disease in 2%. The pathophysiology of SAP in pancreatitis is not well understood but it is possible that the release of pancreatic enzymes results in autodigestion of the vessel wall. While SAP can present as an incidental finding on imaging, it also can be catastrophic.[3] Hemodynamic instability can be one of the presentations resulting from massive bleeding into retroperitoneal space, peritoneal cavity, or adjacent organs as pancreatic duct, with the latter presenting as gastrointestinal bleeding.[3] Hence, early recognition of SAP as a complication of acute pancreatitis is imperative to implement a multidisciplinary management approach in aim to prevent any life-threatening events from rupture or hemorrhages which may occur in 58% of cases.[1,3]

To bring to notice such clinical presentation, we report a case of SAP complicating acute pancreatitis. The patient was informed that data concerning the case would be submitted for publication, and he provided an informed consent. We herein present a case of a 37-year-old female who presented to the emergency department with complaints of acute abdominal pain, nausea, vomiting and diarrhea. The revised Atlanta Criteria to diagnose acute necrotizing pancreatitis was met and a 1.8-centimeter SAP as a complication of acute pancreatitis was noted on Imaging. This case report highlights the importance of having a multidisciplinary management approach and having a high index of suspicion regardless of symptomatology to promptly recognize SAP in the clinical presentation of acute pancreatitis.

CASE DESCRIPTION

A 37-years-old female with past medical history significant for preeclampsia (in the context of one vaginal delivery) and esophagitis (as documented on endoscopy) presenting to the Emergency Room (ER) with acute abdominal pain, tenderness, and nausea for the past 15 hours. The patient described the abdominal pain as sharp epigastric and right upper quadrant (RUQ) location, intensity of 7/10, radiating to the back, aggravated by lying down, and improved by sitting. Associated symptoms included episodes of loose stools without blood or mucus and nausea with non-bloody or non-bilious vomiting. The patient denied any similar episodes of pain in the past. The patient denies travel history, sick contact, fever, chills, shortness of breath, chest pain, palpitations, or any urinary symptoms. Her home medications included Labetalol 300 mg two tablets daily and Omeprazole 20mg daily. The patient denied any recreational drug use, Marijuana, or alcohol drinking. Family history was non-contributory. On a physical exam, the patient is alert and oriented to time, person, place, BMI (30.8 kg/m^2), vital signs were blood pressure 118/65 mmHg, heart rate 79 beats per minute, 99 F temperature, respiratory rate 20/min and the patient was saturating 98 % on room air. Chest and heart examinations were unremarkable. Neck examination did not reveal any lymphadenopathy or thyroid gland enlargement. Abdominal examination showed normal bowel sounds on auscultation and was positive for tenderness on light palpation in the epigastrium with no rebound tenderness or guarding. There was no cutaneous manifestations of acute pancreatitis such as Cullen's sign, Grey Turner's sign, Fox's sign. Labs were significant for negative pregnancy tests, Serum Lipase > 4000 (Reference range 0-160 U/L), serum alanine aminotransferase (ALT) 104 U/l (reference range 0-35 U/L), aspartate aminotransferase (AST) levels 246 U/L (reference range 15-46 U/L), alkaline phosphatase (ALKP) 122 (reference range 38-126 U/L), Lactic acid of 2.5, Total bilirubin 1.7 (reference range 0.1-1.2 mg/dL), Direct bilirubin of 0.6 (reference range <0.3 mg/dL), and hepatitis panel were negative. Coagulation profile was within normal values. The remainder of his complete blood count including White blood cell counts, serum electrolytes, serum calcium, serum creatinine, and serum urea, serum cholesterol, and serum triglycerides were all within their reference ranges. COVID rapid test and Urine Drug screen were unremarkable. Serum Ethanol levels were negative. Chest Xray showed normal cardiac silhouette with no focal pulmonary consolidation or pleural effusion. Electrocardiogram (EKG) demonstrated sinus bradycardia (rate 58 beats per minute) with no ST segment, T wave changes. Abdominal Ultrasound was performed and noted Gall Bladder(GB) wall thickening but no intrahepatic biliary duct dilatation. Computed tomography (CT) Abdomen and Pelvis on admission day was subsequently performed and showed an enlarged and edematous appearance of the pancreas. The pancreatic ducts were not dilated with no biliary ductal dilation

or obstructing calculi visualized in the biliary tree. The gall bladder was distended (4.5cm) without radio-opaque stones, wall thickening or pericholecystic fluid, or reactive duodenitis. These findings are suggestive of acute interstitial edematous pancreatitis. The patient had no systemic inflammatory response syndrome (SIRS) criteria. BISAP Score (entailing: BUN, Impaired mental status, SIRS, Age> 60 years, and presence of pleural effusion) calculated from the labs and data on the first 24 hours of admission was score 0 (indicating less than 1% of mortality), and CT severity index (Balthazar score) was C (indicating mild interstitial pancreatitis without pancreatic or extra pancreatic necrosis). Given the patient clinical presentation of abdominal pain, and lipase levels elevation more than three times the upper limit of normal, the revised Atlanta Criteria to diagnose mild acute interstitial edematous pancreatitis (IEP) was met. The blood tests and imaging studies ruled out gallstones, alcohol, or hyperlipidemia as possible causes of acute pancreatitis. The patient was admitted to the Intensive Care Unit for further management. The patient was made NPO, and IV fluids (2-3 liters Lactate Ringer IV) were started in a peripheral line. Pain control was managed with morphine IV. On the 5th day of hospital admission, the patient continued to have severe RUQ abdominal pain so a repeat CT abdomen with and without contrast agent was performed and reportedly showed (Figure 1 A and B) the following findings: Gallbladder (GB) is less prominent compared to admission CT; Common Bile Duct (CBD) is not dilated; GB sludge is identified with no ductal dilation or calculi; and mild GB wall hyperemia which is likely reactive to adjacent pancreatitis. Additionally, evolving changes of acute pancreatitis with low degree of enhancement mostly around the tail of the pancreas and few other punctate foci of low-non enhancement. Complication of acute pancreatitis are identified including 1.8 centimeter splenic artery pseudoaneurysm (SAP), abdominopelvic ascites and interval worsening of reactive gastritis, enteritis and colitis and diffuse mesenteric edema, as well as trace bilateral pleural effusions. There no evidence of abscess formation, portal venous thrombosis. Evidence of hepatomegaly with hepatic steatosis was also noted. Given the repeat CT findings, patient clinical presentation of persistence of abdominal pain, the revised Atlanta Criteria to diagnose acute necrotizing pancreatitis was met and complicated with SAP. The patient was kept on medical treatment for acute pancreatitis and her symptoms gradually resolved on the 7th day of hospital admission, her ALT and AST normalized, and Lipase trended downward and normalized (4000> 2601 > 2340> 660> 138). A multidisciplinary team approach was coordinated between gastroenterologist, general surgeons, and interventional radiologist (IR). Based on the evaluation of IR, the decision was reached that there was no indication for percutaneous transcatheter embolization intervention of SAP at this time given the small size and clinical stability of the patient. General surgeon team also decided that the best approach is future follow-up in 6 weeks post hospital discharge to evaluate the need for Cholecystectomy after the acute nature of the presentation is resolved. The patient clinically improved, and all her symptoms resolved, and she was counseled on the importance of outpatient follow-up, and she was discharged home.

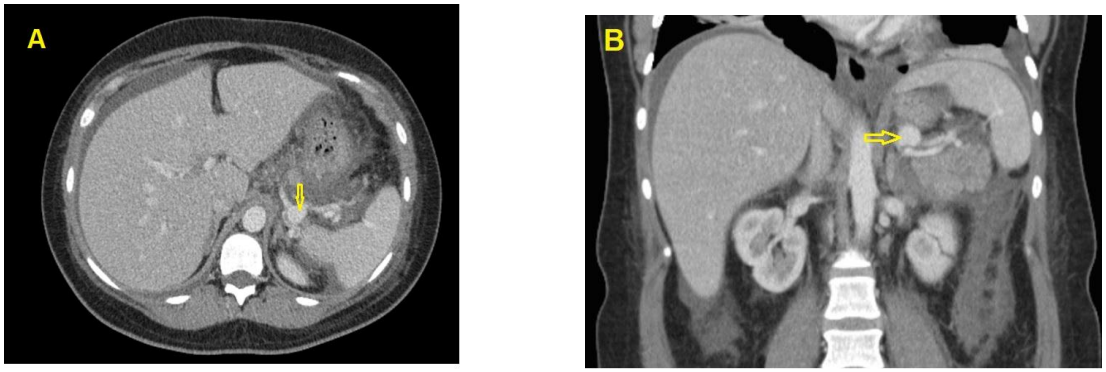

Figure 1: Contrast enhanced Abdominal CT (A) transverse view, (B) coronal view both showing splenic arterial pseudoaneurysm (SAP) , arrow.

DISCUSSION

Splenic artery pseudoaneurysm (SAP) is described as a focal dilation of the artery diameter that is more than fifty percent of the normal diameter and is reported to occur in about sixty percent of patients diagnosed with visceral artery aneurysm.[1] In the literature, the most common etiology of SAP is pancreatitis occurring in 52% of cases (acute in 6% and chronic in 46%), abdominal trauma in 29%, post-surgical complications in 3%, and peptic ulcer disease in 2%.[1,3]

Our case report adds to a handful list of cases reporting on SAP as a complication of acute pancreatitis.[2,4] While the pathophysiology of SAP in pancreatitis is not completely understood, it was shown that a possible mechanism is through autodigestion of the artery elastic walls by the pancreatic enzymes resulting in pseudoaneurysm formation.[4] It was reported that such formation of pseudoaneurysm may take from one to four months post an episode of pancreatitis.[3] In a case series of 157 patients with SAP, abdominal pain was the most common symptom of presentation (50% of cases) followed by abdominal hemorrhage (30% of cases) and nausea, back pain, or chest pain in 20%. In our case, abdominal pain was the main presenting symptom. Tessier and colleagues also reported on a case series of ten patients with SAP (mean age of 51.2 years,[1] range, 35-78 years) in whom 80% were symptomatic and average diameter was 1.7 cm. In their series, the authors reported that chronic pancreatitis was the majority of causes followed by trauma. The authors also recommended repairing SAP regardless of symptoms or size. To our knowledge, our patient did not experience any similar episodes of abdominal pain in the past, and she denied alcohol intake hence, the clinical presentation was concurrent with acute pancreatitis and SAP was 1.8 cm in diameter. Hence this case report adds to the smaller percentage of SAP complicating acute pancreatitis. On the contrary, others documented the association of SAP with recurrent pancreatitis.[5,6]

Awareness and prompt recognition of SAP risk factors, presenting symptoms, diagnostic imaging, and management options is crucial. SAP has a wide variety of presentations from an incidental finding on non-invasive imaging to a life threatening acute hemodynamic instability because of acute rupture.[7] The diagnostic imaging modality of choice to detect SAP is abdominal CT which can distinguish SAP from pseudocyst while enhanced contrast multi-sliced CT or Magnetic resonance imaging (MRI) can be of added value by detecting SAP luminal enhancements and localizing the pseudoaneurysm in relationship to the associated vessels.[8]

However, challenges in the diagnosis can arise from missing smaller pseudoaneurysms on CT imaging in cases where peripancreatic fluid collection or pseudocyst coexists.[8] Hence, the most definitive diagnosis is achieved with abdominal angiography which in turn shows a hyperdense area within a peripancreatic collection. In our case, the diagnosis of SAP was not clear with the initial abdominal CT scan due to the presence of concomitant peripancreatic fluid collection. However, given the persistence of the patient's symptoms on the 5th hospitalization day, a repeat enhanced abdominal CT detected the presence of a 1.8 cm SAP.

Asymptomatic SAP has been also reported in 2.5% of cases.[9] While the natural history of asymptomatic SAP is unclear, it was reported to be more prone for rupture than a true splenic artery aneurysm.[9] However, in the current literatures, there no evidence of correlation between the size of SAP rupture. In our case, the symptoms resolved on the 7th day of hospitalization and the multidisciplinary care team including the IR team concluded that the patient is a poor candidate for embolization, hence the recommendation was to continue with an outpatient surgical follow-up.

To date, there are no specific guidelines for the management of SAP in acute pancreatitis. The current standard of therapy is to control SAP complications such as bleeding by either non-invasive intervention (including endovascular trans arterial catheter embolization or stent placement) or by surgical intervention (either direct ligation of bleeding vessels or by splenectomy with or without distal pancreatectomy including the SAP) in cases of failed embolization.[10] Hence tailoring a multidisciplinary team approach including early consultation with surgeons in managing SAP is warranted for sound management plan.

CONCLUSION

Despite the rare occurrence of Splenic Artery Pseudoaneurysm (SAP), evaluation by a multidisciplinary team approach of gastroenterologist, general surgeons, and interventional radiologist is warranted. Our case report adds to the literatures reporting on the association between acute pancreatitis and SAP.

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