Case Report

Splenic artery pseudoaneurysm presenting with massive rectal bleeding

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\textbf{ABSTRACT}

Splenic artery pseudoaneurysm is relatively rare and its rupture is usually fatal. We report a case of a 48-year-old male with 2 prior episodes of alcoholic pancreatitis that presented with massive per rectal bleeding from rupture of a splenic artery pseudoaneurysm into the transverse colon. Gold standard of diagnosis is CT angiography of the abdomen. We present the first case in the literature where a diagnosis has been made with noncontrast CT of the abdomen and described the radiologic features that facilitated this diagnosis.

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\textbf{Introduction}

Splenic artery pseudoaneurysm is relatively rare and its rupture can be fatal; the diagnosis is usually established at post-mortem. We describe the first case in the literature of preoperative diagnosis on noncontrast CT of abdomen of a splenic artery pseudoaneurysm rupturing into the transverse colon. The features are here in described.

\textbf{Case report}

A 48-year-old gentleman presented to the Emergency Room with a 6-hour history of massive bleeding per rectum. He denied other symptoms such as coffee ground vomitus, nausea, or change in bowel habit. He had prior episodes of minor, bright red rectal bleeding in the preceding 2 months with increasing frequency in the 3 weeks before presentation.
There was a long history of alcohol abuse and tobacco dependence as well as 2 previous hospitalizations at another institution for acute alcoholic pancreatitis, and 6 and 2 months prior to the current presentation. On both occasions, he was treated conservatively and discharged free of known complications. However, epigastric pain had been persistent since his last episode of pancreatitis and he had increase in severity the day prior to presentation. He denied any recent alcohol binges and stated his last consumption was more than 6 weeks ago.

Physical examination revealed an East Indian male in mild painful distress with obvious cachexia and pallor in stage 2 hemorrhagic shock. His abdomen was soft with mild tenderness in the epigastrium without any obvious masses. On rectal examination, there was bright red blood without hemorrhoids, masses, or perianal disease. Laboratory investigations revealed normocytic anemia of Hb 6.8 mg/dL consistent with acute blood loss, acute kidney injury as evidenced by (creatinine 1.4 mg/dL, blood urea nitrogen of 30 mg/dL) and normal coagulation studies. Liver function tests as well as serum amylase and lipase were all within normal limits.

Intravenous fluid resuscitation with 0.9% saline was initiated and compatible blood was transfused within 1 hour. Due to the patient’s kidney injury, an emergent computed tomography (CT) scan of the abdomen and pelvis, without intravenous contrast was performed. This suggested the possibility of a splenic artery pseudoaneurysm near the tail of the pancreas with fistulization into the splenic flexure of the colon and actively leaking (Figs. 1 and 2).

Emergency laparotomy was performed and the findings confirmed the CT findings. At surgery, the spleen was resected along with the distal pancreas, the splenic flexure of the colon, and the attached aneurysmal sac. There was no intraperitoneal bleeding or evidence of mesenteric hematoma. The portion of the sac densely adherent to the stomach was left in situ. A primary anastomosis of the colon was also performed.

The postoperative course was uneventful, the patient discharged after 5 days and followed as an outpatient with a vaccination protocol following splenectomy.

Discussion

Albeit rare, rupture of a splenic artery aneurysm (SAA) is an important cause of gastrointestinal bleeding due to its high mortality and uncommon occurrence. [1] Following the aorta and the common iliac arteries, aneurysms of the splenic artery are reported as the third most common intra-abdominal aneurysm and the most common visceral arterial aneurysm. [2] Many authors agree that its prevalence is estimated at less than 1% in the general population, 7%-20% in patients with liver cirrhosis and portal hypertension, and 8%-13% in patients following liver transplantation [3,4].

Common risk factors for true aneurysmal dilatation of the splenic artery include hypertension, diabetes mellitus, hyperlipidemia, cirrhosis, portal hypertension, female sex, and pregnancy. In a retrospective analysis involving 233 patients with visceral arterial aneurysms over 10 years, Pitton et al. concluded that degenerative atherosclerosis was the main cause of SAA [5]. Hormonal changes and increased splenic artery outflow during pregnancy have not only increased the incidence of SAA, but has shown to increase the size of a pre-existing lesion as well as the risk of rupture [3].

As for pseudoaneurysms of the splenic artery, the most common risk factors include acute or chronic pancreatitis, pancreatic pseudocyst, and abdominal trauma [3]. The local release of pancreatic enzymes in conditions such as acute pancreatitis leads to the breakdown of the elastin fibers and degeneration of the vessel wall predisposing to false aneurysmal dilatation of the splenic artery. Although less common than true aneurysms, pseudoaneurysms of the splenic artery are more likely to rupture and produce complications, given its histopathology [6].

SAA poses a diagnostic dilemma in the Emergency Department setting and needs to be differentiated from tumors, notably Gastrointestinal Stromal Tumors which can present similarly [7].
While up to 80% of SAA are asymptomatic and discovered incidentally, frequent complaints by patients with symptomatic SAA are nonspecific and include symptoms such as epigastric or left upper quadrant pain, nausea, vomiting, and anorexia. As with aneurysms of any artery, the natural progression of SAA is enlargement of the aneurysm over time with the potential for rupture especially when greater than 2 cm. Due to its anatomic relations, aneurysms of the splenic artery may rupture into the abdominal cavity resulting in a hemoperitoneum and may present as an acute abdomen, with left shoulder tip pain (Kehr’s sign) and hypovolemic shock. There are reported cases of erosion and rupture into adjacent organs such as the stomach (presenting with symptoms of an upper gastrointestinal bleed), the duodenum (melena and hematemesis), as well as erosion into the splenic vein (resulting in an arteriovenous fistula with mesenteric steal and ischemic small bowel) [7]. As with the case outlined above, there have been cases where an SAA has eroded into the colon and resulted in hematochezia with hypovolemic shock [1,8]; less than 200 such cases are described in the literature [9].

The gold standard for the diagnosis of SAA is CT angiography of the abdomen/abdominal aorta. However, additional imaging modalities such as USS with color Doppler and MRA of the abdomen have been reported with varying sensitivities and specificities [2]. CT angiography is favored in the diagnosis of SAA because of its accuracy and cost benefits as well as the advantage of providing the clinician with useful anatomic relations for consideration of different treatment modalities. Despite the many positives, the use of CT angiography has its downsides, in particular, the administration of intravenous contrast material in patients with kidney disease as well as those with allergies to the dye. As outlined in our case, the patient suffered a prerenal acute kidney injury secondary to hypovolemic shock, which contraindicated the use of IV contrast material due to the risk of potentiating the patient’s renal failure. As such, our patient underwent CT imaging of the abdomen without the use of contrast material to better determine the cause of his gastrointestinal hemorrhage.

This is the first case presented, to our knowledge, which made the diagnosis of splenic artery pseudoaneurysm using CT imaging, where intravenous contrast was not administered. Radiologically, there are no direct pathognomonic features that would pointedly suggest a splenic artery pseudoaneurysm on a noncontrast CT.

This lesion had the appearance, on CT, of a mixed density lesion with its epicenter in the pancreatic tail associated with peripancreatic fat stranding (Fig. 1). The lesion indented, and was indistinguishable from, the greater curvature of the stomach and the splenic flexure of the colon. The intrinsic hemorrhagic component within the mass was evident by the appearance of hyperdensities on the CT. These features supported a splenic artery pseudoaneurysm as an increasingly plausible differential diagnosis.

At this time, the other possible differentials to consider could be narrowed down to a solid pseudopapillary pancreatic neoplasm, pancreatic hematoma, or acute pancreatitis. Neoplasm was considered less likely due to the fact that there was evidence of erosion into the colonic wall, which would less likely be seen in a well-encapsulated solid tumor. There was also nothing in the patient’s history, such as trauma, to suggest that a pancreatic hematoma may be a likely diagnosis.

There was discontinuity in the colonic wall, with focal dehiscence and absence of medial wall at the splenic flexure along with a focal hyperdensity (blood/clot) protruding into the lumen (Fig. 2). Diffuse intraluminal hyperdense layering was noted in the colon and the rectum, suggestive of blood (Figs. 3 and 4).

All these features combined, along with the clinical history of per rectal bleeding and the fact that there was CT evidence of blood within the entire colon, pointed to a working diagnosis of splenic artery pseudoaneurysm, eroding into the colon.
There was also evidence to suggest chronic pancreatitis, namely pseudocyst in the pancreatic head and calcifications within the pancreatic tail.

Treatment of SAA can be through open surgery or endovascular approaches where embolization can be done. In a review of literature, [9] during open surgery the most durable procedure was splenectomy with or without distal pancreatectomy, which resulted in no reported failure. Resection of adjacent structures is done based on invasion. Of note, in our case we were able to preserve the stomach in comparison to the case described by Maharaj et al. [1]. This was possible because of CT diagnosis of SAA and intraoperative confirmation of the same where there was no invasion into the stomach and no need to remove the sac since it was not a tumor.

Timely imaging in conjunction with the history and clinical findings facilitated early surgical intervention, a good clinical outcome, and avoidance of adherent stomach.

**Supplementary material**

Supplementary material associated with this article can be found, in the online version, at doi: 10.1016/j.radcr.2019.03.038.

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