Brief Report

Splenic artery aneurysms encountered in the ED: 10 years’ experience

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Abstract
Objective: Our objective was to report 7 cases of splenic artery aneurysm (SAA) encountered in the emergency department (ED).
Methods: A retrospective survey of our ED database revealed 7 cases of SAA (6 men, 1 woman; mean age, 56 years) of 651347 ED visits over the last decade. Their clinical and imaging features, management, and outcomes were evaluated.
Results: Splenic artery aneurysm in the ED was rare (prevalence, 0.011%). Common presentations included acute abdomen (n = 5) and shock (n = 2). Five cases had liver cirrhosis and portal hypertension. Abdominal radiographs (n = 7) revealed 2 atherosclerotic patients with SAA. Abdominal computed tomography (n = 7) depicted all SAAs (size, 1.5-8 cm; mean, 3.8 cm). Four ruptured SAAs were successfully managed with coils embolization. Among them, 1 patient with ruptured mycotic SAA also received surgery, but the patient died of Klebsiella sepsis 3 months later.
Conclusions: In the ED, ruptured SAA should be included as a rare differential consideration of acute abdomen, especially in middle-aged men with liver cirrhosis and portal hypertension. Although SAA may be an unexpected computed tomographic finding, once diagnosed, endovascular treatment is recommended.

1. Introduction

Splenic artery aneurysm (SAA) is uncommon, although it is the third most common intra-abdominal aneurysm, following abdominal aorta aneurysm and iliac artery aneurysm. In one study, the prevalence of SAA was 0.8% in unselective visceral angiograms and 0.04% to 0.1% at autopsy [1]. Rupture of SAA may lead to catastrophic hemorrhage. Approximately 10% of SAAs are ruptured at the time of diagnosis, with a 75% mortality [2,3]. Modern imaging techniques allow early detection of asymptomatic SAA [1,4,5]. In addition to conventional surgery, alternative therapeutic options include endovascular treatment and laparoscopic repair [1,5-10]. However, SAA encountered in the emergency department (ED) has not been specifically addressed. The objective of this study was to present our
<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age</th>
<th>Sex</th>
<th>Initial presentations</th>
<th>Associated conditions</th>
<th>SAA rupture</th>
<th>Location</th>
<th>Size (cm)</th>
<th>Diagnosis</th>
<th>Management</th>
<th>Outcome</th>
<th>Follow-up</th>
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<tr>
<td>1</td>
<td>77</td>
<td>M</td>
<td>Acute abdomen, RP, fever, shock after 4 h</td>
<td>Gallstone, DM, mycotic aneurysm</td>
<td>+</td>
<td>Middle</td>
<td>2.5</td>
<td>AR, CT, AG</td>
<td>TAE, cholecystectomy, splenectomy, aneurysmectomy</td>
<td>Died of Klebsiella pneumonia after 3 mo</td>
<td>Alive 18 mo</td>
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<tr>
<td>2</td>
<td>55</td>
<td>M</td>
<td>Acute abdomen, RP</td>
<td>HTN (180/100 mm Hg)</td>
<td>+</td>
<td>Middle</td>
<td>5.8</td>
<td>AR, CT, AG</td>
<td>TAE</td>
<td>Alive</td>
<td>4 y</td>
</tr>
<tr>
<td>3</td>
<td>54</td>
<td>M</td>
<td>Acute abdomen, RP, shock after 3 h</td>
<td>LC with PH</td>
<td>+</td>
<td>Distal</td>
<td>1.5</td>
<td>AR, CT, AG</td>
<td>TAE</td>
<td>Alive</td>
<td>6 y</td>
</tr>
<tr>
<td>4</td>
<td>41</td>
<td>M</td>
<td>Acute abdomen, RP</td>
<td>LC with PH, alcoholism, prior pancreatitis</td>
<td>+</td>
<td>Distal</td>
<td>8</td>
<td>AR, US, CT, AG</td>
<td>TAE</td>
<td>Alive</td>
<td>6 y</td>
</tr>
<tr>
<td>5</td>
<td>39</td>
<td>M</td>
<td>Acute abdomen, RP</td>
<td>LC with PH, HTN (190/98 mm Hg)</td>
<td>–</td>
<td>Distal</td>
<td>3.6</td>
<td>AR, US, CT</td>
<td>Conservative</td>
<td>Alive</td>
<td>13 m</td>
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<tr>
<td>6</td>
<td>72</td>
<td>F</td>
<td>Abdominal fullness</td>
<td>LC with PH, HTN (190/98 mm Hg)</td>
<td>–</td>
<td>Proximal</td>
<td>2.2</td>
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<td>Conservative</td>
<td>Alive</td>
<td>9 y</td>
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<tr>
<td>7</td>
<td>54</td>
<td>M</td>
<td>Upper gastrointestinal bleeding</td>
<td>LC with PH, DU</td>
<td>–</td>
<td>Distal</td>
<td>3</td>
<td>AR, CT, endoscopy</td>
<td>Conservative</td>
<td>Died of DU perforation after 1 y</td>
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</table>

AG indicates angiography; AR, abdominal radiographs; ASA, aberrant splenic artery; DM, diabetes mellitus; DU, duodenal ulcer; HTN, essential hypertension; LC, liver cirrhosis; PH, portal hypertension; RP, rebound pain; US, ultrasonography.
experience with 7 cases of SAA encountered in the ED, with an emphasis on patient characteristics, clinical presentations, and diagnostic and therapeutic considerations.

2. Materials and methods

From March 1995 to March 2006, a total of 7 cases of SAA were found after a retrospective investigation of the ED database. The medical records of the patients were reviewed for clinical manifestations, known prior diseases, pertinent laboratory data, and outcomes. All patients had plain abdominal radiographs and abdominal computed tomography (CT). Two of them also received sonographic evaluation.

Emergent transarterial embolization (TAE) was performed in 4 patients with ruptured SAAs diagnosed by CT. Transarterial embolization was done using coils with or without gelfoam cubes. Technical success was defined as complete exclusion of the aneurysm on controlled arteriogram after the procedure. One patient underwent surgery immediately after TAE. A follow-up CT was performed 5 days, 3 months, and 9 months after TAE.

3. Results

The clinical and imaging findings of the patients are summarized in Table 1.

3.1. Clinical features

There were 6 men and 1 woman in our study, ranging in age from 39 to 77 years (mean, 56 years). Of the 7 patients, 5 presented with an acute abdomen with sudden-onset diffuse abdominal or epigastric tenderness with rebound pain; patient 1 also had fever and subsequently developed shock 4 hours after arrival in the ED, and patient 3 was hemodynamically stable at admission but suddenly developed shock 3 hours later. One patient presented with abdominal fullness and another with gastrointestinal bleeding. Medical histories revealed liver cirrhosis with portal hypertension in 5 patients, essential hypertension in 2, diabetes mellitus and gallstones in 1, duodenal ulcer in 1, and alcoholism with a prior history of pancreatitis in 1. Laboratory studies revealed elevated white blood cell counts in 4 patients, mildly elevated levels of serum aspartate and alanine aminotransferase in 5 patients, and mildly decreased hemoglobin levels in 5 patients.

3.2. Imaging features

Abdominal radiographs (n = 7) showed splenomegaly with a calcified-rim nodule in the splenic hilum in patient 7 and a calcified-rim nodule in the left L1 through L2 paraspinal region in patient 6, suggesting possible calcified SAAs (Fig. 1). Two patients underwent transabdominal transarterial embolization with coils with or without gelfoam cubes.
sonography, which revealed SAA in patient 4 (Fig. 2) but missed the diagnosis in patient 5. All 7 patients underwent abdominal CT, which consistently revealed SAA. The lesion was located in the distal part of the splenic artery in 4 patients, the middle part in 2, and the proximal part in 1. The aneurysm sizes ranged from 1.5 to 8 cm, with a mean of 3.8 cm. Two SAAs had calcified rims. Four SAAs were ruptured focally with irregular arterial outpouching from the aneurysmal sac, contrast-medium leakage, perisplenic hematoma, and regional fluid collection and/or ascites. The sizes varied (1.5, 2.5, 5.8, and 8 cm; mean, 4.45 cm). In patient 1, who had diabetes, perianeursymal gas with adjacent inflammatory strands was demonstrated, compatible with mycotic aneurysm (Fig. 3). In patient 3, the SAA was small and was initially overlooked; the images were reviewed, and SAA was identified retrospectively (Fig. 4). Liver cirrhosis and portal hypertension (engorged portal vein, presence of varices and/or splenomegaly) were demonstrated in 5 patients. Gallstones were demonstrated in 1 patient.

3.3. Treatment outcome and follow-up

All 4 ruptured SAAs were managed with emergent TAE. In patients 1 and 2, the ruptured SAAs were located in the middle portion of the splenic artery and were embolized with the sandwich method by applying coils 1 to 2 cm upstream and downstream of the aneurysm; gelfoam cubes were also added in patient 2 for increased packing of the SAA (Fig. 5). In patients 3 and 4, the SAAs were located in the distal portion of the splenic artery and were successfully occluded with coil packing at the distal supplying artery; gelfoam cubes were also applied in patient 3. Immediate follow-up splenic arteriograms revealed technical success in all 4 patients. Because patient 1 had a mycotic aneurysm, surgical treatment with a cholecstectomy, splenectomy, and aneurysmectomy was performed immediately after TAE. However, the patient died 3 months later of Klebsiella pneumonia and sepsis. After TAE, patients 2, 3, and 4 showed mildly elevated levels of serum amylase (186-214 U/L; mean, 200 U/L; reference range, 27-137 U/L) and serum lipase (196-257 U/L; mean, 227 U/L; reference range, <190 U/L), which normalized after 1 to 2 weeks. They were alive and well at 18 months, 4 years, and 6 years after the procedure, respectively. Of our 7 patients, 3 received conservation treatment. Patient 5 had a 3.6-cm SAA in the distal portion of the splenic artery; he refused surgery and remained alive and well at the 13-month follow-up. In patients 6 and 7, the SAAs had calcified walls and were probably atherosclerotic. Abdominal angiographic evaluation of patient 6 showed an atherosclerotic SAA in the proximal part of an aberrant splenic artery originating from the splenomesenteric trunk (Fig. 1), and this aberrant SAA remained stable at the 9-year follow-up. Patient 7 died of duodenal ulcer perforation and multiple-organ failure 1 year after treatment.

4. Discussion

Splenic artery aneurysms are more common in women than men, with a 4:1 female-to-male ratio, and commonly affect multiparous women during pregnancy [2-4,11]. Approximately 80% to 95% of SAAs are asymptomatic and are incidentally found during evaluation of unrelated symptoms [1,3,6-8,11]. However, SAAs may rupture, resulting in severe abdominal pain or even hypovolemic shock. In these circumstances, the patient may be brought to the ED for management. Nevertheless, as shown by our experience, an SAA is indeed rare in the ED, with a prevalence of approximately 0.011%. Among 100 cases of
SAA reported by Trastek et al [3], 80 were women, but only 3 had acute rupture. In contrast, 6 of 7 ED-encountered SAAs in our series affected middle-aged men who commonly presented with acute abdomen, and there was a surprisingly high rupture rate of 59% (4/7 cases). Noteworthily, as in 2 of our cases, there is a so-called double rupture phenomenon, which may initially occur as bleeding confined to the lesser sac, with the patient in a transient hemodynamically stable status and decrease clinical alertness. This is followed by an unpredictable onset of shock due to subsequent intraperitoneal SAA rupture, which can be fatal [1,5].

The pathogenesis of SAA is not well understood. Atherosclerosis, essential hypertension, trauma, septic embolism, pancreatitis, liver disease, and portal hypertension have been reported as risk factors for SAA [4-8,11,12]. Essential hypertension seems to play a role in atherosclerotic weakening of the splenic artery wall and formation of an aneurysm. Atherosclerotic changes are observed in up to 99% of the SAAs examined histologically but are most likely secondary to medial degeneration [3]. In our series, essential hypertension associated with an atherosclerotic SAA could only be identified in 1 case. In pregnant women, etiologic factors including aorta compression by the uterus with portal...

Fig. 5 Patient 2. A, Abdominal CT reveals a ruptured SAA (arrows) at the middle part of the splenic artery. B, Digital subtraction angiogram confirms the ruptured SAA (arrows). C, Follow-up angiogram after coil embolization shows complete obliteration of the SAA (arrows). D, Follow-up CT angiogram 9 months after embolization shows persistent obliteration of the SAA (arrows) and collateral reconstitution of the distal part of the splenic artery (open arrows).
Splenic artery aneurysms encountered in the ED

Spleenic artery aneurysms (SAAs) represent a rare entity that poses a diagnostic and management challenge in the emergency department (ED). The splenic artery, originating from the splenomesenteric trunk [15], is the smallest visceral artery. SAAs are defined as an abnormal outpouching of blood vessels more than 20% the diameter of the normal vessel and can be divided into true aneurysms and pseudoaneurysms [1].

Establishing the correct diagnosis of CT findings in the splenic artery is important for patients with cirrhosis with an acute abdomen and detailed scrutiny of underlying diseases. As in patient 1, CT was capable of revealing a clinically occult mycotic SAA. However, SAAs may be small and may be overlooked if radiologists and ED physicians do not keep this condition in mind, as occurred in patient 4. On the other hand, in the ED, SAAs rupture with intra-abdominal hemorrhage may be an incidental or unexpected finding on CT, as a part of the workup of abdominal pain. When CT reveals intra-abdominal hemorrhage, the bleeding site or origin of active extravasation of the contrast material should always be available for ED patients [4].

Various imaging modalities including plain radiographs, sonography, CT, and angiography have been used to identify SAAs [1,4,5,12-14]. Radiographically, as in our study, only 2 SAAs were identified as calcified-rim nodule in the left upper abdomen [13]. On sonograms, SAAs typically manifest as a left hypochondrial pulsatile cystic lesion if it is not obliterated by overlying stomach gas [14]. Magnetic resonance angiography plays a role in the diagnosis of SAA in liver transplantation but may not always be available for ED patients [4].

In our experience, CT is an excellent tool for diagnosing SAA. Besides assessing the location and size of the SAA, it also reveals ruptured aneurysms, intra-abdominal hemorrhage, and associated underlying diseases. As in patient 1, CT was capable of revealing a clinically occult mycotic SAA. However, SAAs may be small and may be overlooked if radiologists and ED physicians do not keep this condition in mind, as occurred in patient 4. On the other hand, in the ED, SAA rupture with intra-abdominal hemorrhage may be an incidental or unexpected finding on CT, as a part of the workup of abdominal pain. When CT reveals intra-abdominal hemorrhage, the bleeding site or origin of active extravasation of the contrast material should cautiously be identified. The differential diagnoses of intra-abdominal hemorrhage include traumatic solid visceral or mesenteric injuries, intra-abdominal tumor bleeding (most commonly hepatocellular carcinoma rupture in our country), abdominal aortic aneurysm rupture, and, uncommonly, visceral aneurysm rupture. Although rare, awareness of the possibility of SAA rupture in patients with cirrhosis with an acute abdomen and detailed scrutiny of CT findings in the splenic artery are important for establishing the correct diagnosis.

Consistent with prior reports [1-12], in 4 of 7 cases encountered in the ED, the SAA originated from the distal splenic artery, which is the most frequently affected site. Only 1 SAA was found in the proximal part of an aberrant splenic artery, originating from the splenomesenteric trunk [15]. An SAA rarely exceeds 3 cm in diameter, although giant SAAs up to 30 cm have been documented [1-12,16]. The risk factors for SAA rupture are difficult to assess, and various factors including the presence of calcification, patient’s age, and aneurysmal size have been evaluated, but no definitive relation to aneurysmal rupture can be established [1,4,5,7,8]. Lee et al [12] reported that ruptured SAAs in patients with portal hypertension were larger than in those without portal hypertension (5.5 vs 5.1 cm). In our series, the average size of the ruptured SAAs was 4.45 cm. Although a ruptured SAA with a diameter of less than 2.5 cm is rare [3,6], the smallest ruptured SAA in this series was only 1.5 cm.

Regardless of how SAA is discovered, surgical or endovascular treatment is advocated in patients with SAA-related symptoms, patients with expanding SAA, women who are pregnant or expecting to be pregnant, in liver transplant candidates, and patients with SAA measuring 2.5 cm or greater [1-7]. Classically, SAA can be surgically repaired or directly ligated with or without splenectomy or a partial pancreatectomy [6,7,12]. Elective repair of SAA is safe, with a 0% to 1.3% mortality. However, emergent repair of ruptured SAA is associated with mortality of up to 40% [1,3,5,7,12]. Laparoscopic SAA repair has also been described as a minimally invasive method that may be preferable for selected patients in whom open surgery entails prohibitively high risks [9,10]. However, the clinical feasibility of laparoscopic surgery for a ruptured SAA in the ED setting has not been validated.

The application of TAE for SAA as a treatment alternative has been substantiated by low morbidity rates ranging from 14% to 25% and high success rates ranging from 75% to 100%. Some studies have even advocated TAE as the treatment of choice for all visceral aneurysms [1,8,17,18]. Some TAE-related complications of SAA have been described [1,5-8], including splenic infarction, inadvertent embolism to other visceral arteries, abscess formation, arterial disruption, and access site hematoma. Saltzberg et al [7] reported that major complications developed in 4 of 11 patients with distal SAAs treated with TAE, but those TAEs were performed with coil plus adjunctive n-butyl-2-cyanoacrylate (NBCA) adhesive. Because NBCA is a liquid adhesive, embolization using NBCA will lead to permanent occlusion of the distal-end arteries, and thus, severe splenic infarcts and pancreatitis are probable. In our study, the TAEs in all 4 patients with ruptured SAAs (coil embolization in 2, coil and minimal use of gelfoam cubes in 2) were technically successful. Except for patient 1 who had a mycotic SAA and in whom subsequent surgery was needed to remove a septic focus and for complete treatment, the other 3 patients had good recoveries with only minimal transient elevation of pancreatic enzymes and no recurrence in long-term follow-up. In our experience, TAE is an effective method for treating most of the SAAs, even if the patient is hemodynamically unstable. However, as in patient 1, who had a mycotic SAA, urgent referral to a vascular surgeon after TAE is mandatory. In addition, in SAA not amenable by TAE due to complex...
anatomy or technical failure even after repeated TAE, referral for surgical repair is warranted [1,7].

In summary, this case series highlights that SAA should be included as a rare differential consideration of acute abdomen, especially in middle-aged men with liver cirrhosis and portal hypertension. Although SAA rupture may be an unexpected CT finding as part of the workup of abdominal pain in the ED, once diagnosed, timely TAE is usually effective. If SAA is not amendable by endovascular treatment, surgical repair is necessitated.

References