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Case Report

Atraumatic splenic rupture precipitated by splenic vein thrombosis

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ABSTRACT

We present the case of a 59-year-old man with atraumatic splenic rupture because of splenic vein thrombosis who was successfully treated with splenic artery embolization.

Case Report

A 59-year-old man with medical history significant for vasculopathy and alcohol abuse presents with elevated lipases and diagnosed with pancreatitis. Because of pain out of proportion on physical examination, the patient was suspected of having mesenteric ischemia. Doppler imaging of the mesenteric vessels was ordered, and subsequently revealed multiple thrombi in the superior mesenteric vein, portal vein, and the splenic vein (Fig. 1). On hospital day 2, the patient experienced a drop in hemoglobin/hematocrit of 11.7 g dL^{-1}/34.2-7.07 g dL^{-1}/20.0 with associated decline of blood pressure to 85/45 mm Hg. The patient was transfused 3 units of packed red blood cells. Abdominal computed tomography (CT) demonstrated a large splenic subcapsular hematoma with a moderate amount of hemoperitoneum and active site of extravasation from the spleen (Fig. 2). In addition, a pseudoaneursym arising from the distal splenic artery was discovered, and the extent of portal and splenic venous thrombosis was appreciated (Figs. 3 and 4). Because of the patient's hemodynamic instability and comorbidities, the patient was an unfit candidate for general anesthesia and general surgery. Interventional radiology was consulted for splenic artery embolization.

Although no active contrast extravasation from the splenic artery was identified, splenic arteriography did discover a 6 to 7-mm pseudoaneurysm near the distal aspect of the splenic artery (Fig. 5). The catheter was positioned proximal to the pseudoaneurysm, and subsequently, a 7-mm Amplatzer vascular plug was deployed to staunch blood flow into the pseudoaneurysm. A 15-minute delayed angiography demonstrated very scant contrast opacification distal to the plug. Vascular flow was maintained proximal to the Amplatzer plug to ensure continued perfusion to the distal pancreatic territory (Fig. 6). On postprocedure day 1, the patient's hemoglobin and/or

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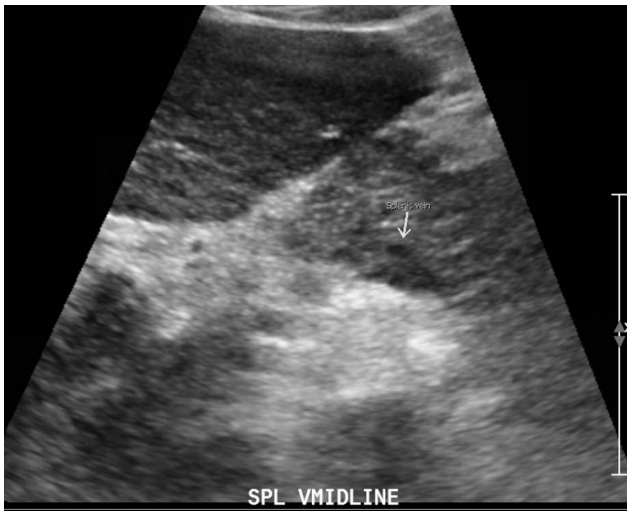

Fig. 1 – Ultrasound of the splenic vein demonstrates acute nonocclusive thrombus (arrow).

hematocrit returned to near baseline of 10.5 g dL^{-1}/35.0 and blood pressure to 110/70 mm Hg. The patient maintained stable hemodynamics 1 week after embolization at which time a splenectomy was performed. The patient tolerated the splenectomy well and was subsequently discharged on postoperative day 14.

Discussion

Atraumatic splenic rupture is a rare phenomenon, with estimates of its incidence between 0.1% and 0.5% [1]. In the medical literature, atraumatic splenic rupture can refer to 2 possible cases, the first being a spontaneous rupture of a normal spleen, more appropriately termed as a spontaneous rupture, and the second being a spontaneous rupture of a diseased spleen, termed a pathologic rupture [2]. A true

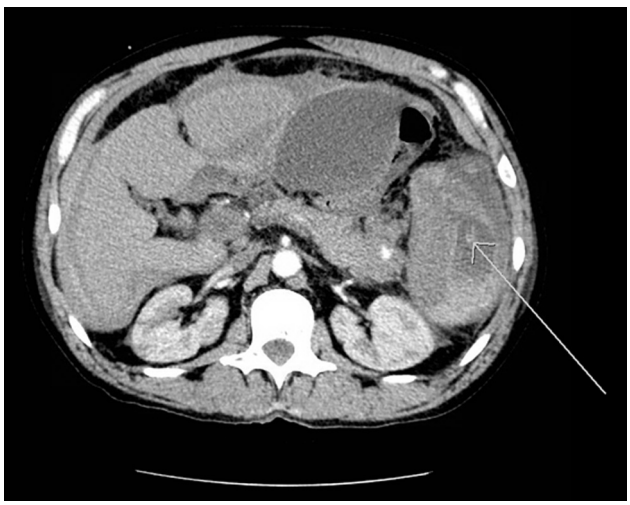

Fig. 2 – Contrast-enhanced computed tomography abdomen demonstrates a small area of contrast extravasation (arrow) in an enlarged, heterogeneous spleen with multiple areas of parenchymal devascularization. Hemoperitoneum was also evident.

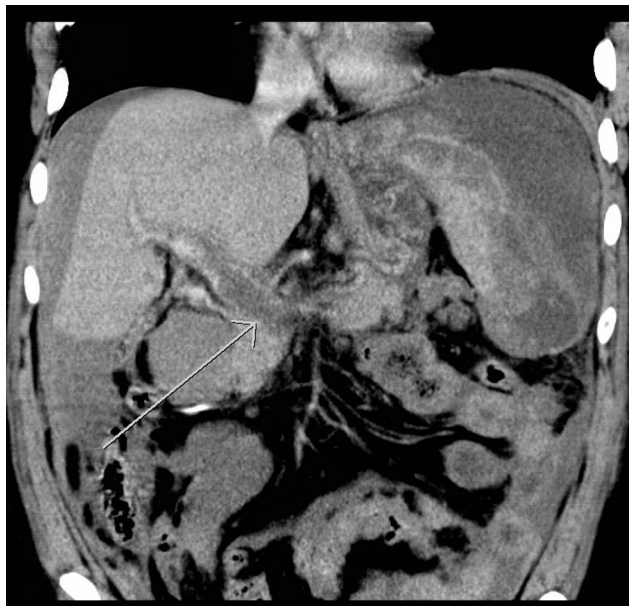

Fig. 3 – Contrast-enhanced axial computed tomography abdomen pelvis shows thrombus formation at the portal confluence with extension into the splenic vein (arrow).

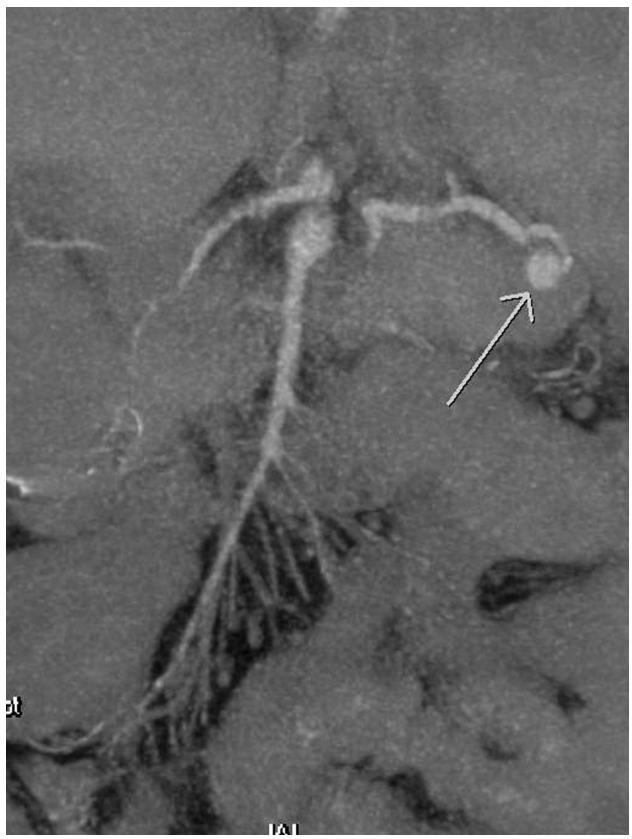

Fig. 4 – Coronal maximum intensity projection shows pseudoaneursym at the distal splenic artery (arrow).

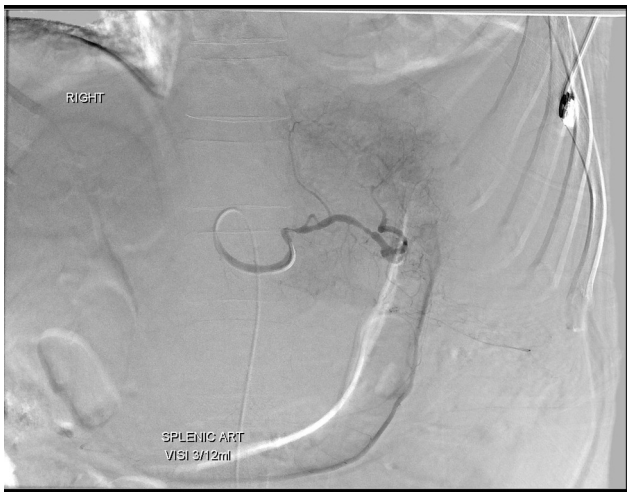

Fig. 5 – A 6 to 7-mm pseudoaneurysm is seen at the distal splenic artery. No indication of active extravasation in the splenic parenchyma. Several subtle areas of nodular luminal irregularity could represent distal third and fourth order splenic artery pseudoaneurysms.

spontaneous rupture is thought to be much less common [3]. Atraumatic splenic rupture of a pathologic spleen is more extensively documented, and the rupture of the spleen is thought to manifest because of underlying diseases [4].

Findings from a systematic review of atraumatic splenic rupture indicate that a vast majority of atraumatic splenic rupture patients have 1 etiologic factor that precipitates atraumatic splenic rupture. An abridged table from Renzulli et al

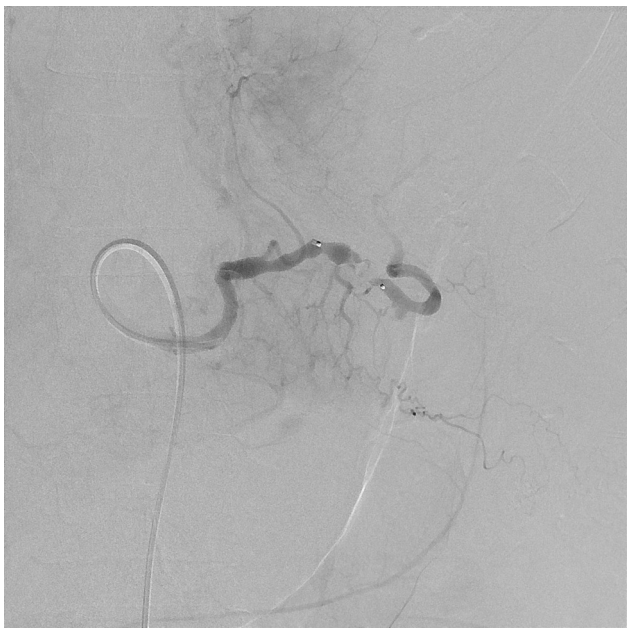

Fig. 6 – The distal construct of the Amplatzer plug shows very minimal contrast opacification with significantly reduced flow seen in the splenic arterial bed distally. There is preserved perfusion of the distal pancreatic bed territory.

Table 1 – Common etiologies for atraumatic splenic rupture.

Etiologies for atraumatic splenic rupture	Number of factors (%)*
Malignant hematologic disorders	152 (16.4)
Acute myelogenous leukemia	21 (2.3)
Non-Hodgkin's lymphoma	55 (5.9)
Viral infectious disorders	137 (14.8)
Infectious mononucleosis	102 (11.0)
Cytomegalovirus infection	13 (1.4)
Local inflammatory and neoplastic disorders	101 (10.9)
Chronic pancreatitis	65 (7.0)
Acute pancreatitis	20 (2.2)
Primary neoplastic disorders	75 (8.1)
Angiosarcoma	31 (3.3)
Peliosis	18 (1.9)
Normal spleen	59 (6.4)
Normal spleen without triggering factor	41 (4.4)
Normal spleen with triggering factor	18 (1.9)

* A total of 926 etiologic factors were present in the 845 patients. A total of 711 patients had only one etiologic factor. Percentages are approximated and are calculated based on a total of 926 factors.

listing common etiologies is provided in Table 1. The systematic review noted that approximately 7% of patients had no etiologic factor for atraumatic splenic rupture [5]. Splenomegaly was seen in more than half (55%) of atraumatic splenic rupture patients, whereas a normal sized spleen was observed in approximately 15% of patients [5]. This could lend credence to the possibility of splenomegaly being a common feature of atraumatic splenic rupture. Approximately 84% of atraumatic splenic rupture patients received treatment in the form of total splenectomy. Although transcatheter arterial embolization can be used in unstable patients, less than 1% of patients received this form of treatment. The study also found both an age greater than 40 years and splenomegaly to be significant factors for predicting atraumatic splenic rupture-related mortality [5].

The most common reported cause of atraumatic splenic rupture is malignant hematologic disorders, followed by infectious and inflammatory disorders [3,5]. Although unclear, potential pathophysiologic mechanisms for atraumatic splenic rupture have been hypothesized, such as distention of the spleen because of infiltration of splenic parenchyma and hemorrhage of the splenic capsule due to infarct [6]. Infiltration of splenic parenchyma is thought to be a possible mechanism for malignant hematologic disorders [6,7].

As presented in this case, pancreatitis is also thought to be a common cause of atraumatic splenic rupture [3,5] because of the sequela of splenic vein thrombosis [7]. Rupture of the spleen from splenic venous thrombosis revolves around gradual accumulation of pressure and congestion within the splenic parenchyma due to venous outflow obstruction. Increased intracapsular pressure will drop arterial perfusion pressure thereby causing the spleen to undergo necrotic dehiscence.

Aside from pancreatitis, there have been reported cases of splenic vein thromboses due to cardiac surgery, septic emboli from infective endocarditis, and atherosclerosis from chronic renal failure [8–11]. There also has been a reported incidence of toxic megacolon from underlying ulcerative colitis, which can cause splenic venous thrombosis and lead to splenic rupture

[12]. Thrombophilia in patients afflicted with paroxysmal nocturnal hemoglobinuria has been documented to have caused splenic rupture through splenic venous thrombosis [13]. Heparin-induced thrombocytopenia has also been implicated in development of splenic venous thrombosis that ultimately lead to splenic rupture [14,15]. Furthermore, acquired amyloidosis due to injectable drugs and protein S deficiency were also observed as causes of atraumatic splenic rupture [16,17].

It has been reported that a diagnosis of a ruptured spleen is frequently not considered in the absence of trauma, even in the presence of pain in the upper left quadrant and hemodynamic instability, which can lead to devastating outcomes [18]. In these instances, the use of diagnostic tools may greatly increase an accurate diagnosis of atraumatic splenic rupture. The systematic review found that for a diagnosis of free-intraperitoneal fluid, ultrasonography was used in 24.6% of patients and CT was used in 23% of patients [5]. Diagnosis of atraumatic splenic rupture was made by ultrasonography in 18.6% of patients and by CT in 32.4% of patients [5]. Use of CT is the preeminent technique for diagnosis [19], since CT can provide for a conclusive diagnosis of atraumatic splenic rupture [20]. The use of ultrasound is also supported, with the literature purporting a sensitivity of 72%-78% and a specificity of 91%-100% for the diagnosis of atraumatic splenic rupture [6,20,21].

The 2 most common forms of atraumatic splenic rupture treatment are total splenectomy and conservative treatment [5]. Management via conservative and nonoperative means have low rates of success, and will ultimately later necessitate a splenectomy due to rebleeding [6]. The atraumatic splenic rupture-related mortality rate tends to increase significantly if patients have to undergo secondary surgery after conservative treatment [5]. The definitive treatment of atraumatic splenic rupture is splenectomy. From the systematic review by Renzulli et al [5], there was a 7.4% atraumatic splenic rupture-related mortality rate in patients receiving a total splenectomy. Nonetheless, there are lifelong risks associated with the total splenectomy procedure, such as increased risk for sepsis and infections, and an increased risk for thromboembolic complications, arteriosclerosis, and coronary heart disease [22]. When considering therapeutic options, assessment of hemodynamic stability, whether the patient is fit for general anesthesia, the amount of blood products used, degree of hemoperitoneum, and magnitude of splenic injury are paramount in guiding treatment [5]. In the setting of atraumatic splenic rupture precipitated by splenic vein thrombosis, there has been documented utilization of conservative management and eventual regression of a chronic-appearing subcapsular splenic hematoma in an otherwise hemodynamically stable patient [19].

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