Minimally Invasive Interventions for Nontraumatic Splenic Disorders

A review of nontraumatic clinical applications for splenic arterial interventions and the technical and anatomic considerations unique to these therapeutic options.

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Image-guided, minimally invasive arterial interventions are increasingly performed in the treatment of nontraumatic splenic disorders as an alternative to surgery. This article reviews various nontraumatic clinical applications for splenic arterial interventions, including hypersplenism, sinistral portal hypertension, nontraumatic splenic hemorrhage, and vascular anomalies. Technical and anatomic considerations, as well as potential pitfalls in splenic arterial embolization are summarized.

NONTRAUMATIC INDICATIONS FOR SPLENIC INTERVENTIONS

Hypersplenic Thrombocytopenia

Hypersplenic thrombocytopenia, or hypersplenism, is a growing indication for splenic artery embolization[1] and can be present in a variety of systemic disorders (eg, cirrhosis), with resulting debilitating anemia, thrombocytopenia, and leukopenia. Although hypersplenism has been treated with splenectomy in the past,[2] with advances in transarterial embolization techniques, partial splenic embolization (PSE) has become an effective alternative to splenectomy for improving hematologic derangements in this patient population.[3] For patients with hypersplenic thrombocytopenia secondary to cirrhosis and hepatic tumor burden, PSE may also be performed to facilitate systemic chemotherapy or locoregional treatment such as transarterial chemoembolization (TACE).

Sinistral Portal Hypertension

Sinistral portal hypertension is a rare cause of life-threatening gastrointestinal hemorrhage in which gastric or gastroesophageal varices form secondary to splenic vein obstruction or thrombosis.[4] Originally described by Turril and Mikkelsen,[5] this entity has been challenging to manage until recently, with splenectomy or partial splenectomy as the mainstay of treatment.[2,4,6] However, PSE may be performed in lieu of or as an adjunct to splenectomy/partial splenectomy for patients with this variant of venous hypertension.[1,3] PSE may also be performed in conjunction with transhepatic transvenous variceal obliteration to reduce the likelihood of recurrence of bleeding varices.

Splenic Rupture

Nontraumatic splenic rupture is a rare cause of life-threatening hemorrhage.[7,8] Possible etiologies include primary or metastatic splenic tumors, infection, mechanical rupture secondary to pregnancy or drug treatments, and inflammatory disorders such as pancreatitis, amyloidosis, and vasculitis.[9] As in the case of traumatic splenic injuries, embolization of the spleen can be highly effective in stabilizing these critical, hemodynamically unstable patients.

Vascular Anomalies

Splenic arterial aneurysms and pseudoaneurysms often require treatment due to the high mortality associated

with aneurysm rupture. Although these vascular anomalies may be treated surgically, endovascular methods offer effective treatment with decreased procedural morbidity and mortality. Minimally invasive approaches to treating these vascular anomalies most commonly include permanent embolization or covered stent graft exclusion of the aneurysm or pseudoaneurysm.

VASCULAR ANATOMY OF THE SPLEEN

The splenic artery is the largest of three major branches of the celiac axis (Figure 1). In 8% of patients, the splenic artery arises directly from the abdominal aorta.[10] The main splenic artery typically bifurcates into upper and lower polar divisions, which then divide into four to six intrasplenic segmental branches.[11] However, secondary branches of the splenic artery also provide blood supply to the pancreas and stomach and therefore must be identified to minimize the risk of complications. The dorsal pancreatic and pancreatica magna arteries commonly arise off the proximal and mid-splenic artery and supply the body and tail of the pancreas. Additional possible visceral arteries arising from the splenic artery include the left gastroepiploic, short gastric, and accessory left colic arteries (Figure 2). The left gastroepiploic artery typically arises from the inferior polar splenic artery and supplies the greater curvature of the stomach and portions of the greater omentum. Short gastric arteries arise from the distal splenic artery or polar/segmental branches and supply the gastric cardia and fundus. Rarely, an accessory left colic artery originating from a terminal branch of the splenic artery may supply the colon in the region of the splenic flexure. Appropriate recognition of the aforementioned collaterals is important to minimize the risk of nontarget embolization during splenic artery intervention.

APPROACH TO MANAGEMENT OF NONTRAUMATIC SPLENIC DISORDERS

Technical Considerations

Basic angiographic techniques should be employed for any splenic artery intervention. Once appropriate arterial access is achieved using the Seldinger technique, a base catheter should be advanced to the celiac axis with angiography performed to provide an anatomic road map. Selective catheterization with arteriography of the lobar and segmental branches is subsequently performed to better delineate anatomic structures during the procedure. Because of variations in the tortuosity, caliber, and course of the splenic artery, multiple catheter types and shapes should be readily available. Subselection of polar and segmental splenic branches routinely requires the use of a microcatheter.

When performing splenic artery interventions, a variety of embolic materials may be used, the choice of which is largely dependent on the familiarity of the operating physician. Permanent occlusion is generally required for most indications such as hypersplenism, sinistral hypertension, and vascular anomalies. Permanent embolic agents include conventional polyvinyl alcohol (PVA), bland microspheres, coils, and liquid embolics such as ethylene-vinyl alcohol (Onyx, Medtronic) and N-butyl cyanoacrylate (or glue). Temporary occlusion with Gelfoam (Pfizer, Inc.) is usually only appropriate for self-limited processes such as traumatic injury and hemorrhage. Metallic coils, covered stents, or a combination of coils and stents may be used to exclude aneurysms and pseudoaneurysms caused by vascular anomalies.

Given the importance of the spleen in immunologic function, some practitioners advocate pneumococcal vaccination to mitigate the risk of pneumococcal sepsis. The lifetime risk of sepsis after splenectomy is approximately 1% to 2%, most commonly secondary to encapsulated organisms such as *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae*.[12] Although this risk is low, postsplenectomy sepsis carries a high rate of mortality approaching 50% to 70%.[13] Some literature suggests that immunization may not be necessary,[13,14] and there is currently no consensus regarding periprocedural vaccination for splenic arterial embolization. However, most practitioners recommend the use of broad-spectrum prophylactic antibiotics prior to the procedure.[15]

Other potential adverse events should be considered when performing splenic artery interventions, particularly embolization. After PSE, postembolization syndrome with fever, nausea, and abdominal pain is common, with an incidence as high as 75%.[16] Rarer major adverse events after embolization include abscess, pleural effusion, ascites, pneumonia, pulmonary embolism, portal vein thrombosis, and liver failure. Minor adverse events include anorexia and ileus. Studies demonstrate a markedly increased risk of major complications associated with splenic embolization volumes > 70%.[17,18] One review exploring risk factors for postembolization portal/splenic vein thrombosis describes a large volume of infarction as the primary risk factor for thrombosis.[19] Cai et al found that the mean splenic infarction volume for patients with portal vein thrombosis after PSE was 71.5%.[20] Efficacy of treatment and overall risk mitigation can be optimized by limiting the end-splenic infarction volume to approximately 60% to 70%.[3,21] Figure 3 demonstrates nonocclusive thrombosis of the portal and splenic veins in a patient shortly after undergoing PSE.

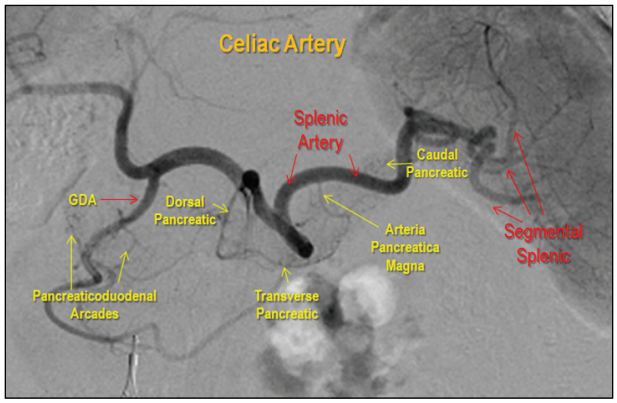

Figure 1. Splenic artery anatomy. Digital subtraction angiogram (DSA) demonstrating conventional celiac arterial anatomy. The dorsal pancreatic and pancreatica magna arteries arise off the tortuous proximal and mid-splenic artery.

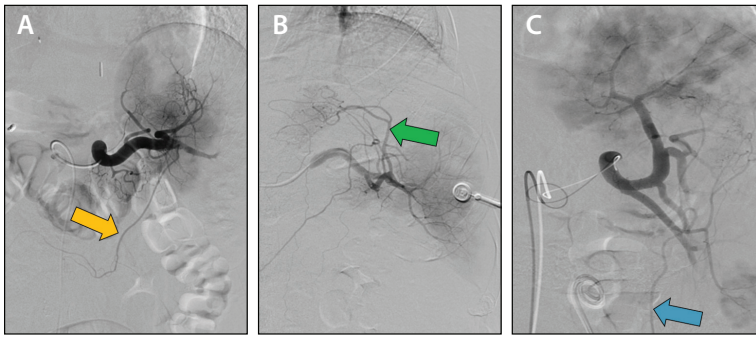

Figure 2. Common collaterals arising from the splenic artery. Left gastroepiploic or gastro-omental artery (yellow arrow) arising from the inferior polar splenic artery (A). Short gastric arteries (green arrow) arising from an intrasplenic segmental splenic artery (B). Accessory left colic artery (blue arrow) arising from an inferior intrasplenic segmental artery (C).

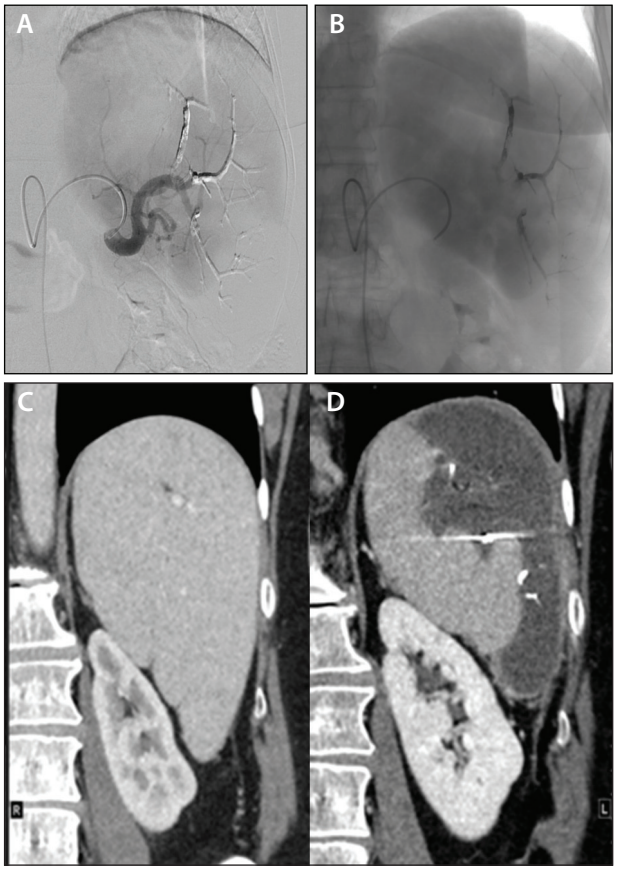

Figure 4. PSE for hypersplenic thrombocytopenia. DSA of the splenic artery after Onyx embolization of three segmental branches; arterial phase (A). Nonsubtracted angiogram of the parenchymal phase demonstrating radiopaque Onyx liquid embolic within the three segmental splenic arterial branches (B). Pre- (C) and post-PSE (D) coronal CT images of the spleen. After PSE, there are new, large geographic areas of tissue infarction within the spleen.

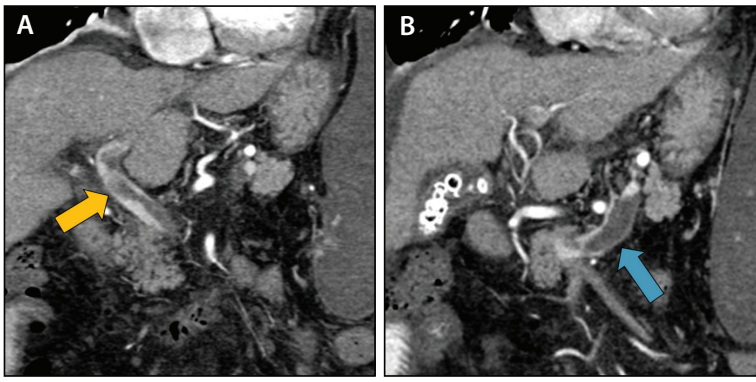

Figure 3. Portal and splenic vein thrombosis after PSE. Coronal contrast-enhanced images demonstrating nonocclusive thrombus identified within the portal vein (yellow arrow) (A) and splenic vein (blue arrow) (B). The patient was successfully treated with anticoagulation, with no further adverse events.

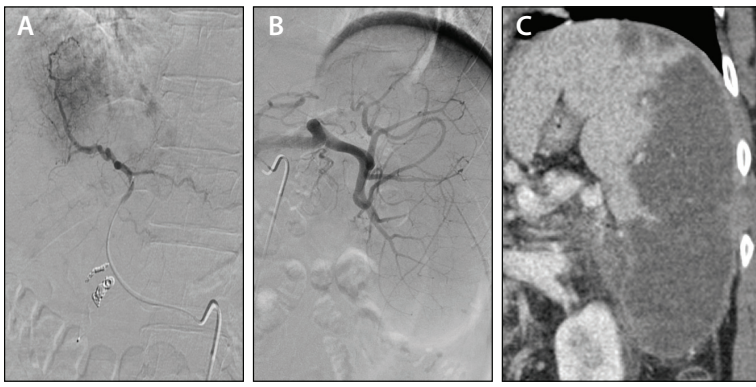

Figure 5. Combined PSE and TACE treatment for hypersplenic thrombocytopenia in a patient with hepatocellular carcinoma. DSA during concomitant TACE procedure (A) and PSE (B). Coronal contrast-enhanced CT image post-PSE demonstrating geographic areas of splenic infarction (C).

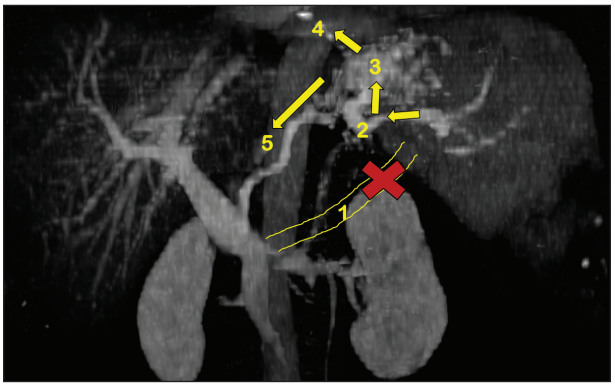

Figure 6. Pathophysiology of sinistral portal hypertension after splenic vein occlusion. In this three-dimensional CT maximum-intensity projection image, splenic vein thrombosis (1) results in hypersplenism (thrombocytopenia) with increased drainage via short gastric veins (2), resulting in gastric varices (3). The dilated varices can either drain uphill into the gastroesophageal veins (4) or antegrade into the coronary vein (5) leading back to the portal venous system.

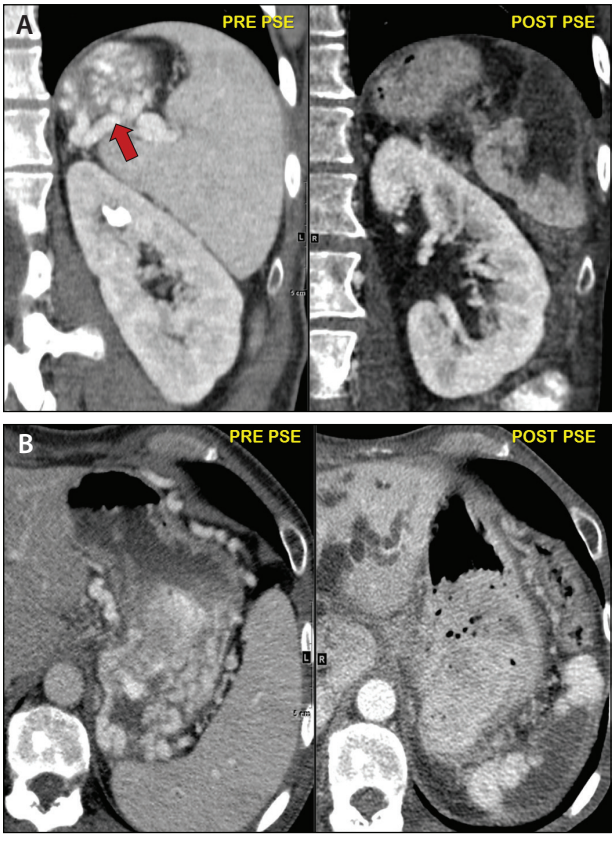

Figure 7. PSE for sinistral hypertension in a patient with pancreatic cancer and bleeding gastric varices. Coronal (A) and axial (B) CT images pre- and post-PSE demonstrating infarction of 60% to 70% of the splenic volume. Note the marked decrease in prominence of the gastric varices.

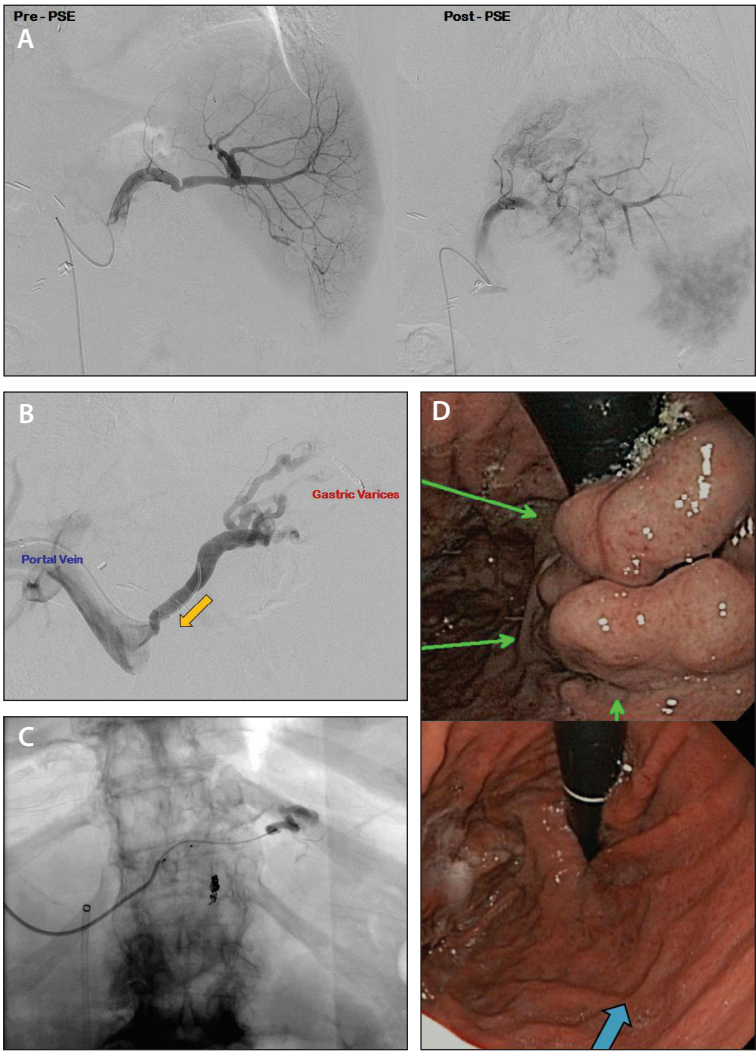

Figure 8. Combined PSE and transhepatic transvenous obliteration of varices in a patient with a pancreatic tail mass resulting in splenic vein thrombosis, hypersplenic thrombocytopenia, and bleeding gastric varices. PSE was performed to infarct approximately 60% to 70% of the splenic volume using 300- to 500-µm microspheres (A). This was done to treat the thrombocytopenia and decrease inflow into the dilated gastric varices. Transhepatic transvenous obliteration of gastric varices was then performed. A transhepatic approach was used to directly access the coronary vein and gastric varices. Venography demonstrates antegrade drainage of the gastric varices into the coronary vein and portal vein (yellow arrow) (B). The coronary vein was occluded using a vascular occluder plug (C). After passing a 2.4-F microcatheter alongside the plug to access the varices, sclerotherapy was performed with 3% sodium tetradecyl foam until the entire variceal network was sclerosed. The arrows demonstrate resolution of the varices after sclerotherapy (D).

Hypersplenism

Hypersplenism is a common indication for PSE. Hypersplenic thrombocytopenia, leukopenia, and anemia accompany a range of underlying pathologies, including cirrhosis with portal hypertension, thalassemia, idiopathic thrombocytopenia, and hereditary spherocytosis. A low blood count is often seen in the setting of chemotherapy, despite widespread use of marrow-proliferating agents and may be another indication for embolization in the setting of splenomegaly. Hypersplenism is thought to cause derangement of peripheral blood counts due to increased consumption and sequestration of the corpuscular elements of blood within the enlarged spleen. PSE is an alternative to surgical splenectomy and is thought to work both by decreasing splenic sequestration and destruction, as well as increasing thrombopoietin levels; the preservation of splenic tissue also helps maintain part of the spleen's immunologic function.

In a study of outcomes after PSE, white blood cell and platelet counts increased and peaked at approximately 3 days and 2 weeks, respectively, after embolization.[18] Most publications and practitioners recommend targeting between 50% to 70% embolization. A study of PSE for the treatment of hypersplenic thrombocytopenia in cirrhosis demonstrated relapse of clinical symptoms in patients who underwent < 50% embolization of the spleen.[22] Complications were significantly increased in those who underwent > 70% embolization. In a study by Zhu et al, 50% of patients treated with > 70% embolization of splenic volume had major complications ranging from bacterial peritonitis to death.[18] In order to further minimize procedural risk, some practitioners perform staged PSE, targeting approximately 30% to 40% embolization over two separate sessions.[23] Figure 4 illustrates PSE for the treatment of hypersplenic thrombocytopenia; in this case, embolization was successful and the patient's thrombocytopenia resolved.

PSE plays a large role in the treatment of hematologic derangement related to cirrhosis and portal hypertension. In addition to effectively correcting blood counts, PSE appears to benefit cirrhotic patients by decreasing portal venous pressure and in turn reducing variceal bleeding, improving hepatic function, and reducing hepatic encephalopathy.[17] In cirrhotic patients with hypersplenism and hepatocellular carcinoma, oncologic treatment options may be limited by bleeding risk and hepatic dysfunction. Effective PSE may therefore facilitate the delivery of both systemic chemotherapy and locoregional treatment. For example, patients with liver-dominant tumors and severe thrombocytopenia who are unable to safely undergo systemic therapy due to hypersplenic thrombocytopenia may qualify for systemic treatments after PSE. In addition to "distal" end-organ embolization with PSE, Bhatia et al described the use of "proximal" splenic embolization as a potential alternative treatment for chemotherapy-induced thrombocytopenia.[24] In this retrospective report, the authors concluded that proximal splenic embolization is not only safe and effective in treating patients with chemotherapy-induced thrombocytopenia, but given its lower incidence of postembolization syndrome and favorable side effect profile compared with distal PSE, this procedure may be performed on an outpatient basis.

PSE may also be performed in conjunction with TACE. In addition to improved platelet counts and decreased bleeding risks, some studies have shown reduced variceal bleeding risk in patients undergoing both treatments.[25] Figure 5 shows a patient with hypersplenic thrombocytopenia and hepatocellular carcinoma who was safely treated with concomitant PSE and TACE.

Sinistral Portal Hypertension

Sinistral (or left-sided) portal hypertension is recognized as a possible cause of upper gastrointestinal variceal hemorrhage.[26] Pancreatic pathologies such as neoplasm, pseudocysts, and postinflammatory sequelae of chronic pancreatitis can cause obstruction or thrombosis of the adjacent splenic vein. Other nonpancreatic pathologies that rarely cause underlying splenic vein thrombosis/obstruction include splenic vein injury/laceration, spontaneous splenic vein thrombosis, and other nonpancreatic neoplasms.[4] Obstruction or thrombosis of the splenic vein causes increased pressure within the portal venous system and redistribution of flow through the short gastric and posterior gastric or gastroepiploic veins to the coronary veins, which results in the formation of gastric and gastroesophageal varices.[26] This phenomenon is illustrated in Figure 6. Because gastric varices most commonly derive from right-sided portal hypertension, this etiology should first be excluded prior to considering a diagnosis of sinistral hypertension. In patients with upper gastrointestinal varices, splenomegaly, and normal liver function, the possibility of sinistral hypertension should be considered.

Traditionally, this condition has been managed surgically with splenectomy,[6] which decompresses the underlying gastric varices by decreasing the arterial inflow into the left-sided portal system. In patients who are poor surgical candidates or in cases in which a more conservative approach may be warranted, endovascular therapies including PSE offer a safer alternative therapeutic approach. Total splenic artery embolization may also be performed in the preoperative setting to minimize the risks of blood loss.

Available endovascular techniques for the management of gastric varices secondary to sinistral hyperten-

sion include PSE, direct transvenous obliteration, and PSE combined with transvenous obliteration. PSE directly lowers splenic artery inflow, thereby decreasing outflow to the gastric veins. In the setting of symptomatic sinistral hypertension and hypersplenic thrombocytopenia, PSE may be the preferred option given that this single therapy may result in treatment of both conditions. With transvenous obliteration, the gastric varices are directly catheterized via systemic or transhepatic collaterals followed by subsequent embolization/sclerotherapy of the varices.[27] Although this method may initially control variceal bleeding, new varices may develop over time due to continued inflow from splenic drainage.

Recent studies suggest that combined PSE and transhepatic transvenous obliteration may most effectively treat gastric varices related to sinistral hypertension. In patients undergoing combined therapy, rebleeding rates were 6.7% at 1 year compared to 36.7% in patients who underwent transhepatic transvenous obliteration alone.[28] Figures 7 and 8 show enlarged, bleeding gastric varices due to pancreatic cancers compressing the splenic veins, resulting in thrombosis. Both patients were successfully treated with PSE alone (Figure 7) or combined PSE and transhepatic transvenous obliteration of the varices (Figure 8).

Splenic Rupture

The most common pathologic etiologies of nontraumatic splenic rupture include neoplasm (30%), infection (27%), and inflammatory causes (20%).[29] Neoplastic causes include malignant and nonmalignant hematologic disorders, primary splenic tumors, and splenic metastases. In patients with hematologic malignancies, male gender, severe splenomegaly, and previous cytoreductive chemotherapy are all associated with increased occurrence of splenic rupture, with splenomegaly considered the greatest risk factor.[7] Infectious causes include bacterial, viral, protozoal, and fungal organisms. Malaria is frequently cited as the most common cause of splenic rupture in endemic regions.[7] Inflammatory causes include pancreatitis, amyloidosis, and vasculitis. Less common causes of pathologic rupture may be related to drug/treatment (eg, anticoagulation, thrombolytics, granulocyte macrophage colony-stimulating factor) and mechanical causes, such as congestive splenomegaly and pregnancy. Spontaneous splenic rupture has also been reported in normal spleens.

The pathology of spontaneous splenic rupture is not completely understood. Some postulate that increased intrasplenic tension stretches and increases the susceptibility of the splenic capsule. Infiltrative malignant processes may also directly disturb, invade, and weaken the capsule. Nontraumatic splenic rupture is a rare and difficult diagnosis, but rapid diagnosis and treatment are essential due to high mortality rates approaching 10% to 15%.[8] Patients may present with acute left upper quadrant abdominal pain, hypotension, tachycardia, and anemia. Rapid diagnosis is aided by abdominal ultrasound and diagnostic paracentesis confirming intraperitoneal blood products. In hemodynamically stable patients, CT may also be useful for diagnosis. Treatment options for these causes include catheter-directed embolization, surgery, and conservative management. The literature supports aggressive therapy via urgent surgical or endovascular therapy. Depending on specific angiographic findings, partial or total splenic artery embolization may be performed in these cases (Figure 9).

Splenic Artery Vascular Anomalies

Splenic artery aneurysms and pseudoaneurysms account for approximately 60% of all visceral aneurysms,[30] with an overall incidence of up to 10.4% in the general population. These aneurysms are four times more common in women, and risk factors include pregnancy, trauma, portal hypertension, and atherosclerosis. Splenic artery aneurysms have a 2% to 10% overall risk of hemorrhage, and risk factors for rupture include size > 2 cm, pregnancy, interval enlargement, portal hypertension, and history of liver transplantation. Typically, endovascular treatment is warranted when these aneurysms are symptomatic or measure > 2 cm due to the high mortality rate after rupture (range, 25%–70%).[31] Symptoms of aneurysms include epigastric or left upper quadrant abdominal pain, nausea, anorexia, or palpation of a pulsatile abdominal mass; however, > 90% of splenic artery aneurysms are asymptomatic.[32]

Pseudoaneurysms of the splenic artery require intervention due the high risk of massive hemorrhage, regardless of size. Over 50% of patients with splenic artery pseudoaneurysms present with hemodynamic instability, necessitating emergent intervention. The causes of splenic artery pseudoaneurysms most commonly include pancreatitis, recent surgery, and trauma. An adjacent pancreatic pseudocyst is also thought to be a risk factor for splenic artery pseudoaneurysm formation.[33]

Compared to surgery, endovascular management of splenic artery aneurysms and pseudoaneurysms offers decreased morbidity and mortality. A variety of techniques are available for the treatment of these vascular anomalies, including exclusion of the aneurysm via embolization of the artery both distal and proximal to the aneurysm (Figure 10), direct embolization of the aneurysm sac, exclusion by covered stent placement, or a combination of these techniques (Figure 11). Although covered stent graft placement across the neck of an aneurysm seems ideal in that splenic blood flow is

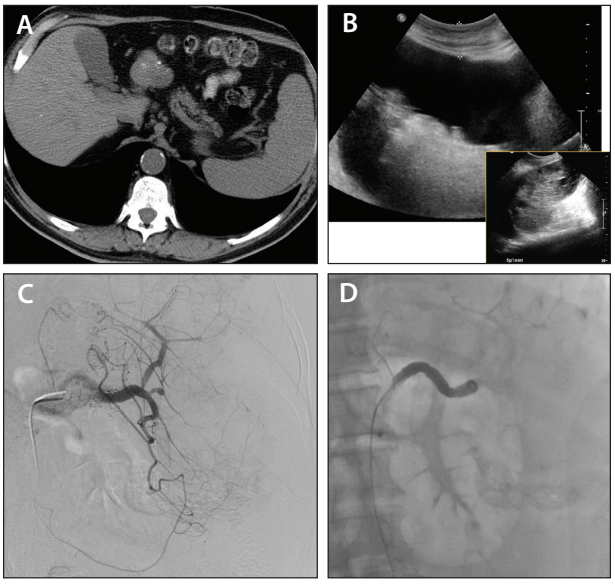

Figure 9. Endovascular management of nontraumatic splenic rupture (spontaneous). A patient with metastatic angiosarcoma to the spleen with acute abdominal distention and left upper quadrant pain. The CT (not shown) and ultrasound demonstrated an enlarged, heterogeneous spleen with new large abdominal ascites. A diagnostic paracentesis revealed serosanguinous fluid. Given the patient's acute blood loss requiring transfusions, the patient was referred to the interventional radiology department for splenic artery embolization. Axial nonenhanced CT (A) performed prior to the bleed demonstrating no significant perisplenic fluid. Ultrasound (B) demonstrating new perisplenic fluid with heterogeneous enlargement of the spleen. A diagnostic paracentesis was performed revealing serosanguinous fluid. Splenic artery angiography (C) was performed with subsequent embolization of the spleen utilizing PVA particles distally and proximal splenic artery coils resulting in complete resolution of the life-threatening splenic hemorrhage (D).

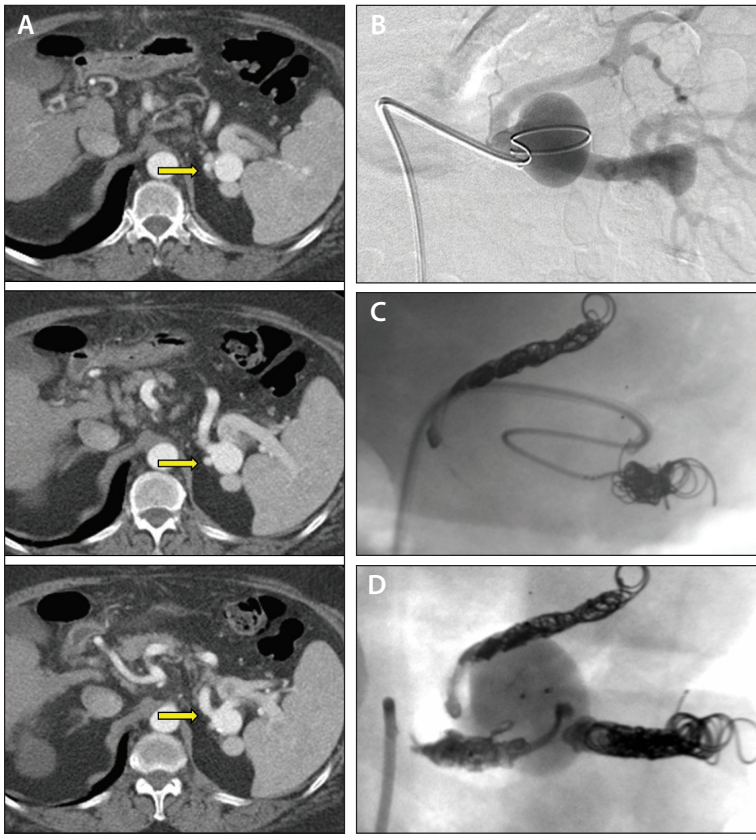

Figure 10. A splenic artery aneurysm. Contrast-enhanced axial CT serial images demonstrating a 2.6-cm splenic artery aneurysm (A). A splenic artery angiogram (B) demonstrates a fusiform aneurysm near the splenic hilum with inflow via the main splenic artery and dual outflows to the superior and inferior polar branches. Embolization of the "backdoor" polar branches distal to the aneurysm sac utilizing detachable coils (C) followed by embolization of the aneurysm sac with Onyx 34 liquid embolic and embolization of the main splenic artery inflow utilizing a vascular plug and Onyx 34 (D).

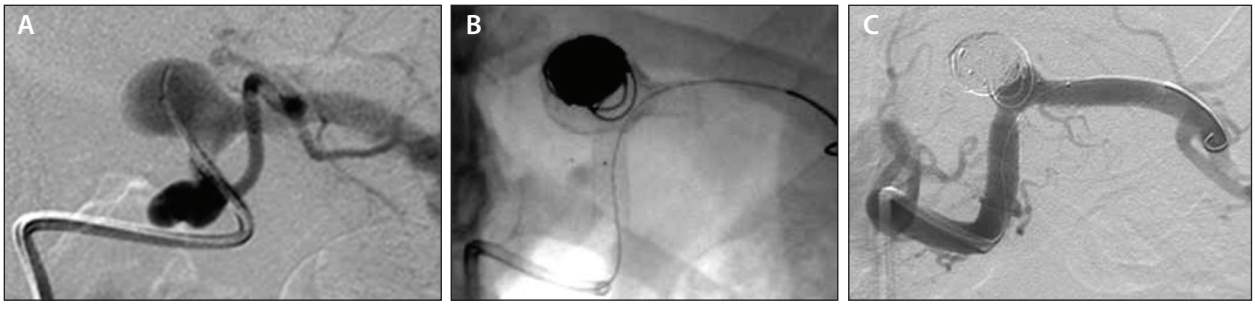

Figure 11. Endovascular treatment of an asymptomatic, partially calcified splenic artery pseudoaneurysm. An expandable bare-metal stent was deployed across the origin of the pseudoaneurysm. A microcatheter was then advanced through the interstices of the metal stent, and the pseudoaneurysm was embolized utilizing metallic coils until flow was no longer detected within the sac. DSA (A) demonstrating the presence of a saccular pseudoaneurysm arising from the main splenic artery. Bare-metal stent placement across the neck of the pseudoaneurysm with subsequent coil embolization through the interstices of the stent (B). Final DSA demonstrating complete exclusion of the treated pseudoaneurysm, with preservation of the main splenic artery (C).

preserved, this can often be technically challenging due to the tortuosity of the splenic artery and large sheath diameter often necessary for stent graft deployment.

CONCLUSION

Although splenic artery interventions have historically and most commonly been performed in the setting of trauma, there is an increasing role for transarterial interventions in the treatment of nontraumatic splenic disorders. Transcatheter splenic interventions can be safely utilized for the treatment of hypersplenism, variceal bleeding related to sinistral hypertension, nontraumatic splenic rupture, and aneurysms and pseudoaneurysms involving the splenic artery. Splenic interventions may also be safely performed in patients with cancer to help facilitate systemic and locoregional therapies that could not otherwise be delivered due to chemotherapy-induced thrombocytopenia.[24] Image-guided, minimally invasive splenic intervention is a viable and safe alternative to surgical intervention in the treatment of many nontraumatic splenic disorders. ■

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