Splenic Artery Embolization as an Adjunctive Procedure for Portal Hypertension

Mitchell Smith, M.D., M.S.¹ Charles E. Ray, M.D., Ph.D., F.S.I.R.¹

¹ Department of Radiology, University of Colorado, Denver Anschutz Medical Campus, Aurora, Colorado

Semin Intervent Radiol 2012;29:135–139

Address for correspondence and reprint requests Charles E. Ray, M.D., Ph.D., F.S.I.R., University of Colorado Anschutz Medical Campus, Mail Stop L954, 12401 E. 17th Avenue, Room 526, Aurora, CO 80045 (e-mail: Charles.ray@ucdenver.edu).

Abstract

Keywords

- portal hypertension
- spleen
- embolization
- varices

Splenic embolization is a technique that can be used alone or in conjunction with other treatments for the mitigation of portal hypertension and associated physiological effects of portal hypertension. This technique can be used safely when total embolization volume is ~50% and the procedural and periprocedural time periods are covered with antibiotics. In this patient population, partial splenic embolization can decrease the incidence of variceal bleeding, and protection can persist for at least a year. Additionally, liver function tests and serum cell counts can be expected to improve. Although not frequently used as primary therapy for patients with portal hypertension, splenic embolization can often be helpful as an alternative or adjunctive procedure.

Objectives: On completion of this article, the reader will be able to define the role of splenic artery embolization in the treatment of portal hypertension including the indications, technical considerations, outcomes, and complications.

Accreditation: Tufts University School of Medicine is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Credit: Tufts University School of Medicine designates this journal-based CME activity for a maximum of **1 AMA PRA Category 1 Credit™**. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Splenic embolization was first performed in 1973 using autologous clot to treat recurrent gastrointestinal hemorrhage arising from esophageal varices.¹ Initially, patients treated with complete splenic embolization had complications that included abscess, splenic rupture, sepsis, pneumonia, and death.^{2–5} Six years after this initial report, Spigos et al treated 13 patients with partial splenic embolization using antibiotic coverage and postembolization pain control.⁶ Following the adoption of partial embolization and antibiotic coverage, multiple case reports and nonrandomized series reported a reduction in both morbidity and mortality rates compared with complete splenic embolization.^{7–15}

Current indications for splenic embolization include treatment of portal hypertension and control of splenic hemorrhage from trauma or prior to surgical resection. It is also used to improve platelet counts in patients with idiopathic thrombocytopenic purpura, to treat the sequelae of hypersplenism, and to manage splenic steal syndrome (nonocclusive hepatic artery hypoperfusion syndrome [NOHAH]) in liver transplant recipients.^{16–28} NOHAH syndrome is a controversial diagnosis and is discussed separately in this issue of *Seminars of Interventional Radiology*.^{16–22}

Splenic Embolization for Portal Hypertension

In the setting of portal hypertension, splenic embolization can be used to improve liver function; treat or prevent variceal hemorrhage; treat hepatic encephalopathy; and improve red blood cell, leukocyte, and platelet counts.

Liver Function

Since 1986, partial splenic embolization has been noted to improve liver function tests in cirrhotic patients treated with partial splenic embolization.²⁹ In 1996, two papers found 12-month improvements in cholinesterase, cholesterol, total protein, prothrombin time, and albumin levels in 40 patients with portal hypertension treated with partial splenic

embolization.^{30,31} Long-term changes in aspartate aminotransferase and alanine aminotransferase levels in cirrhotic patients are less clear, with some investigators finding transient improvement or no significant changes.^{32–34} The mechanism of these changes is not well elucidated but may be related to increases in hepatic arterial and superior mesenteric vasculature blood flow after embolization.³⁵ Porter et al used a video dilution technique to demonstrate that although splenic blood flow was reduced from 19% to 3% postembolization, there was a concomitant increase in hepatic arterial flow from 3% to 15% and an increase in superior mesenteric blood flow from 6% to 19%.³⁶

Variceal Hemorrhage

Splenic embolization has been used to reduce the incidence of variceal bleeding in patients with portal hypertension. Ohmoto et al compared bleeding rates in 52 patients, half of whom were treated with splenic embolization in addition to variceal ligation. The addition of splenic embolization reduced the rate of rebleeding on follow-up from 39% to 12%.³⁷ Taniai et al compared the recurrence rate for esophageal variceal bleeding in patients treated with either partial splenic embolization and ligation, ligation, or sclerotherapy. Combining partial splenic embolization with ligation reduced the 6-month variceal bleeding rate when compared with ligation alone from 58% to 21%, a result that was significant and persistent for 2 years of follow-up.³⁸ Pålsson et al followed 26 severely ill patients treated with partial splenic embolization for esophageal varices or thrombocytopenia, and they demonstrated a mean survival of 50.5 months with significant increases in hemoglobin, leukocyte, and thrombocyte cell counts.³⁴ Bleeding episode rates secondary to esophageal varices decreased from 4.3% in the pretreatment group to 1.1% after partial splenic embolization. In a review article, Koconis et al reviewed the English-language literature between 1973 and 2005 and found five studies that reported rates of variceal bleeding before and after splenic artery embolization.³⁹ In the 50 combined patients, 2.4 episodes of bleeding per year preembolization was reduced to 0.48 episodes postembolization.³⁹ This effect can also be seen in patient with both esophageal varices and portal venous thrombosis with cavernous transformation; Miyaaki et al demonstrated improvement in both portal vein thrombus burden and hemoglobin that lasted at least 1 year.⁴⁰

Hepatic Encephalopathy

There is limited evidence that splenic artery embolization in conjunction with other treatments may help reduce the incidence of hepatic encephalopathy. Uflacker et al treated two patients with partial splenic embolization who had portosystemic shunts, and they demonstrated improvements in encephalopathy.⁴¹ The addition of partial splenic embolization to transportal obliteration and/or balloon retrograde transvenous obliteration of portosystemic shunts have been shown to have lasting effects on both serum ammonia levels and encephalopathy grade that persist for up to 2 years.⁴²

Blood Cell Counts

Splenic embolization has been repeatedly shown to increase white blood cell, platelet, and red blood cell counts. Increased serum levels of white blood cells is most likely the result of decreased splenic pooling, whereas increased erythrocytes are likely secondary to a combination of a reduction in episodic hemorrhage and longer red cell lifespan.^{34,43,44} Elevation in platelet concentration may be multifactorial, with some improvements mediated through decreased splenic sequestration and some effects resulting from an increase in thrombopoietin levels.

Technical Considerations

Our protocol involves preadministration of broad-spectrum antibiotics prior to the procedure and for up to 2 weeks postprocedure. Pyrexia or systemic infections are contraindications given the increased risk of postprocedural abscess. Common femoral artery access is obtained by palpation or ultrasound guidance with eventual placement of a 5F sheath into the iliac system. A C2 catheter, or rarely a reverse curve catheter, is routinely used to select the celiac axis with an initial angiogram performed to evaluate the splenic arterial branches as well as the location of pancreatic branches. Depending on vascular anatomy, a microcatheter system is used to select the distal main splenic artery or splenic artery branches for embolization, with postembolization angiograms completed from the base catheter. We typically use 500- to 700- μ m particles suspended in contrast medium and embolize ~50% of the spleen volume.

We currently administer Pneumovax to patients undergoing splenic embolization, although there is some evidence this may be unnecessary. Skattum et al compared patients who received splenectomy or splenic artery embolization with blood donor controls. In addition to showing an increase in leukocyte and platelet counts, immunoglobulin and memory B cells were not affected in the embolization population, suggesting that immunization may be unnecessary.⁴⁵

Gelfoam has also been used as an agent for splenic embolization. Sangro et al used Gelfoam to reduce splenic blood flow by ~40 to 60%, which significantly increased both white blood cell and platelet counts.⁴⁶ Additionally, some investigators are using the Amplatzer plug for proximal splenic embolization.⁴⁷ Embolization of both the main splenic artery or segmental branches may be used to reach the target embolization volume. There is some evidence from the trauma literature that there may be a slight increase in infarct rate using distal embolization and an increase in infectious complications using a more proximal site.⁴⁸ However, the likelihood of a repeat embolization procedure in portal hypertension patients is not insignificant; for this reason, we do not perform proximal splenic artery embolization with permanent agents that would necessarily preclude repeat embolization procedures. Although in theory we attempt to embolize 50% of the spleen, determining the degree of splenic embolization is both difficult and highly subjective. In our experience, embolizing beyond 50% is easy to do, and for this reason, we perform frequent postembolization angiograms to assess residual flow (—Figs. 1 and 2).

Figure 1 Partial distal splenic embolization. (A) Pre-embolization posteroanterior (PA) projection during arterial phase injection via a base catheter in the proximal splenic artery demonstrates reflux into the common hepatic artery and parenchymal blush throughout the spleen. (B) Postembolization. PA projection during parenchymal phase demonstrates the base catheter advanced into the midsplenic artery. Focal areas of decreased angiographic blush correspond to areas devascularized by Gelfoam slurry, corresponding to embolization of ~40 to 50% of total splenic volume.

Postprocedure, routine groin access precautions as well as immediate access to a patient-controlled analgesic pump are provided. Routine hospital stays are 24 to 48 hours, with the patients following up in clinic 1 week postprocedure. Anti-inflammatory medications (7- to 10-day Medrol dose pack) are administered as well.

Complications

Most of the patients will have postembolization syndrome consisting of fever, nausea, and left upper quadrant pain. These symptoms can usually be controlled with narcotics and antiemetics. Fever is thought to be secondary to release of pyrogens from infarcted spleen and may be treated with steroids.⁴⁹ Additional minor side effects may include anorexia, pleural effusion, ascites, and ileus, all of which can be treated symptomatically.

Koconis et al compiled data looking at the serious complications of splenic embolization procedures.³⁹ These complications include abscess, pleural effusion, ascites, pneumonia,

Figure 2 Proximal main splenic artery embolization. (A) Precoil embolization. Posteroanterior (PA) projection during arterial phase injection demonstrates the base catheter in the proximal splenic artery. Angiographic blush is noted throughout the splenic parenchyma. (B) Postcoil embolization. PA projection during arterial phase injection demonstrates the base catheter in the splenic artery. The coil pack is visualized in the proximal splenic artery with complete devascularization of the splenic parenchyma. The coil pack has been appropriately placed just distal to the dorsal pancreatic artery.

pulmonary embolus, portal vein thrombosis, liver failure, and death. Treatment of pleural effusions and ascites is managed with thoracentesis and paracentesis, respectively, and are typically self-limited. Pneumonia and/or atelectasis usually develop on the left side, which some authors suggest may be mitigated by focusing embolization in the mid and inferior poles of the spleen.^{50,51} Splenic abscess is treated with percutaneous drainage and follow-up computed tomography scan, sinogram, and/or drain upsizing or downsizing as the abscess changes over time. Portal venous thrombosis and pulmonary embolism can be effectively managed with anticoagulation. Depending on the severity of the complication, subspecialty consultation may be helpful in the management of more complex cases; however, few cases require surgical intervention.

Evaluation of these serious complications suggests that both operator experience and embolization volume may be related to poor outcome. In 11 patients where the data were available, 73% had a serious complication when the embolization volume was 70%.³⁹ Of note, the four deaths reported

since 1990 were in patients who had portal hypertension secondary to cirrhosis. A rough estimate of mortality rate in this population from splenic embolization is 1% (compared with a reported 2.8% mortality from transjugular intrahepatic portosystemic shunt).^{39,52}

Conclusion

Splenic embolization is a technique that can be used alone or in conjunction with other treatments for the mitigation of portal hypertension and associated physiological effects of portal hypertension. This technique can be used safely when total embolization volume is ~50% and the procedural and periprocedural time periods are covered with antibiotics. In this patient population, partial splenic embolization can decrease the incidence of variceal bleeding, and protection can persist for at least a year. Additionally, liver function tests and serum cell counts can be expected to improve. Although not frequently used as primary therapy for patients with portal hypertension, splenic embolization can often be helpful as an alternative or adjunctive procedure.

References

- Maddison FE. Embolic therapy of hypersplenism. *Invest Radiol* 1973;8:280–281
- Goldstein HM, Wallace S, Anderson JH, Bree RL, Gianturco C. Transcatheter occlusion of abdominal tumors. *Radiology* 1976;120(3):539–545
- Castaneda-Zuniga WR, Hammerschmidt DE, Sanchez R, Amplatz K. Nonsurgical splenectomy. *AJR Am J Roentgenol* 1977;129(5):805–811
- Wholey MH, Chamorro HA, Rao G, Chapman W. Splenic infarction and spontaneous rupture of the spleen after therapeutic embolization. *Cardiovasc Radiol* 1978;1(4):249–253
- Witte CL, Ovitt TW, Van Wyck DB, Witte MH, O'Mara RE, Woolfenden JM. Ischemic therapy in thrombocytopenia from hypersplenism. *Arch Surg* 1976;111(10):1115–1121
- Spigos DG, Jonasson O, Mozes M, Capek V. Partial splenic embolization in the treatment of hypersplenism. *AJR Am J Roentgenol* 1979;132(5):777–782
- Dent D, Alsabrook G, Erickson BA, et al. Blunt splenic injuries: high nonoperative management rate can be achieved with selective embolization. *J Trauma* 2004;56(5):1063–1067
- Delcour C, Spiegl G, Brion JP, De Vaere S, Struyven J. Complications in splenic embolization. *Ann Radiol (Paris)* 1982;25(7):453–454
- Vazquez Estévez J, Lassaletta L, Perez-Higueras A, Utrilla J, Diez-Pardo JA. Partial splenic embolization in hypersplenism. *Acta Paediatr Scand* 1988;77(4):593–596
- Uflacker R, d'Albuquerque LAC, de Oliveira e Silva A, de Freitas JM, Gama-Rodrigues JJ. Embolization to reverse severe recurrent hepatic encephalopathy. *Arq Gastroenterol* 1988;25(special issue):21–25
- Kumpe DA, Rumack CM, Pretorius DH, Stoecker TJ, Stellin GP, DH. Partial splenic embolization in children with hypersplenism. *Radiology* 1985;155(2):357–362
- Vazquez Estévez J, Lassaletta L, Perez-Higueras A, Utrilla J, Diez-Pardo JA. Partial splenic embolization in hypersplenism. *Acta Paediatr Scand* 1988;77(4):593–596
- Delcour C, Spiegl G, Brion JP, De Vaere S, Struyven J. Complications in splenic embolization. *Ann Radiol (Paris)* 1982;25(7):453–454
- Back LM, Bagwell CE, Greenbaum BH, Marchildon MB. Hazards of splenic embolization. *Clin Pediatr (Phila)* 1987;26(6):292–295
- Reynolds M, Donaldson JS, Vogelzang RL. Giant iatrogenic splenic pseudocyst. *J Pediatr Surg* 1989;24(7):700–701; discussion 701–702
- Saad WE. Management of nonocclusive hepatic artery complications after liver transplantation. *Tech Vasc Interv Radiol* 2007;10(3):221–232
- Rasmussen A, Hjortrup A, Kirkegaard P. Intraoperative measurement of graft blood flow—a necessity in liver transplantation. *Transpl Int* 1997;10(1):74–77
- Saad WEA. Liver transplant-related vascular disease. In: Dake M, Geshwind J-F, eds. *Abrams Angiography: Interventional Radiology*. 2012. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins
- Nüssler NC, Settmacher U, Haase R, Stange B, Heise M, Neuhaus P. Diagnosis and treatment of arterial steal syndromes in liver transplant recipients. *Liver Transpl* 2003;9(6):596–602
- Lima CX, Mandil A, Ulhoa AC, Lima AS. Splenic artery steal syndrome after liver transplantation: an alternative technique of embolization. *Transpl Proc* 2009;41:1990–1993
- Geissler I, Lamesch P, Witzigmann H, Jost U, Hauss J, Fangmann J. Splenohepatic arterial steal syndrome in liver transplantation: clinical features and management. *Transpl Int* 2002;15(2–3):139–141
- Uflacker R, Selby JB, Chavin K, Rogers J, Baliga P. Transcatheter splenic artery occlusion for treatment of splenic artery steal syndrome after orthotopic liver transplantation. *Cardiovasc Intervent Radiol* 2002;25(4):300–306
- Fujitani RM, Johs SM, Cobb SR, Mehlinger CM, White RA, Klein SR. Preoperative splenic artery occlusion as an adjunct for high risk splenectomy. *Am Surg* 1988;54(10):602–608
- Hiatt JR, Gomes AS, Machleder HI. Massive splenomegaly. Superior results with a combined endovascular and operative approach. *Arch Surg* 1990;125(10):1363–1367
- Kimura F, Itoh H, Ambiru S, et al. Long-term results of initial and repeated partial splenic embolization for the treatment of chronic idiopathic thrombocytopenic purpura. *AJR Am J Roentgenol* 2002;179(5):1323–1326
- Miyazaki M, Itoh H, Kaiho T, et al. Partial splenic embolization for the treatment of chronic idiopathic thrombocytopenic purpura. *AJR Am J Roentgenol* 1994;163(1):123–126
- Kimura F, Ito H, Shimizu H, et al. Partial splenic embolization for the treatment of hereditary spherocytosis. *AJR Am J Roentgenol* 2003;181(4):1021–1024
- Thanopoulos BD, Frimas CA. Partial splenic embolization in the management of hypersplenism secondary to Gaucher disease. *J Pediatr* 1982;101(5):740–743
- Hirai K, Kawazoe Y, Yamashita K, et al. Transcatheter partial splenic arterial embolization in patients with hypersplenism: a clinical evaluation as supporting therapy for hepatocellular carcinoma and liver cirrhosis. *Hepatogastroenterology* 1986;33(3):105–108
- Murata K, Shiraki K, Takase K, Nakano T, Tameda Y. Long term follow-up for patients with liver cirrhosis after partial splenic embolization. *Hepatogastroenterology* 1996;43(11):1212–1217
- Sakata K, Hirai K, Tanikawa K. A long-term investigation of transcatheter splenic arterial embolization for hypersplenism. *Hepatogastroenterology* 1996;43(7):309–318
- Hidaka H, Kokubu S, Saigenji K, Isobe Y, Maeda T. Restoration of thrombopoietin production after partial splenic embolization leads to resolution of thrombocytopenia in liver cirrhosis. *Hepatology* 2002;23(4):265–273
- Tajiri T, Onda M, Yoshida H, Mamada Y, Taniai N, Kumazaki T. Long-term hematological and biochemical effects of partial splenic embolization in hepatic cirrhosis. *Hepatogastroenterology* 2002;49:1445–1448
- Pålsson B, Hallén M, Forsberg AM, Alwmark A. Partial splenic embolization: long-term outcome. *Langenbecks Arch Surg* 2003;387(11–12):421–426
- Nishida O, Moriyasu F, Nakamura T, et al. Interrelationship between splenic and superior mesenteric venous circulation

- manifested by transient splenic arterial occlusion using a balloon catheter. *Hepatology* 1987;7(3):442–446
- 36 Porter BA, Frey CF, Link DP, Lantz BM, Pimstone NR. Splenic embolization monitored by the video dilution technique. *AJR Am J Roentgenol* 1983;141(5):1063–1065
 - 37 Ohmoto K, Yamamoto S. Prevention of variceal recurrence, bleeding, and death in cirrhosis patients with hypersplenism, especially those with severe thrombocytopenia. *Hepatogastroenterology* 2003;50(54):1766–1769
 - 38 Taniai N, Onda M, Tajiri T, Toba M, Yoshida H. Endoscopic variceal ligation (EVL) combined with partial splenic embolization (PSE). *Hepatogastroenterology* 1999;46(29):2849–2853
 - 39 Koconis KG, Singh H, Soares G. Partial splenic embolization in the treatment of patients with portal hypertension: a review of the English Language literature. *J Vasc Interv Radiol* 2007;18(4):463–481
 - 40 Miyaaki H, Ichikawa T, Nakao K, et al. Portal hypertensive gastropathy with portal thrombosis successfully treated with partial splenic embolization. *Clin J Gastroenterol* 2009;2(3):218–221
 - 41 Uflacker R, d'Albuquerque LAC, de Oliveira e Silva A, de Freitas JM, Gama-Rodrigues JJ. Embolization to reverse severe recurrent hepatic encephalopathy. *Arq Gastroenterol* 1988;25(special issue):21–25
 - 42 Yoshida H, Mamada Y, Taniai N, et al. Long-term results of partial splenic artery embolization as supplemental treatment for portal-systemic encephalopathy. *Am J Gastroenterol* 2005;100(1):43–47
 - 43 Rios R, Sangro B, Herrero I, Quiroga J, Prieto J. The role of thrombopoietin in the thrombocytopenia of patients with liver cirrhosis. *Am J Gastroenterol* 2005;100(6):1311–1316
 - 44 Hidaka H, Kokubu S, Saigenji K, Isobe Y, Maeda T. Restoration of thrombopoietin production after partial splenic embolization leads to resolution of thrombocytopenia in liver cirrhosis. *Hepatol Res* 2002;23(4):265–273
 - 45 Skattum J, Titze TL, Dormagen JB, et al. Preserved splenic function after angioembolisation of high grade injury. *Injury* 2012;43(1):62–66
 - 46 Sangro B, Bilbao I, Herrero I, et al. Partial splenic embolization for the treatment of hypersplenism in cirrhosis. *Hepatology* 1993;18(2):309–314
 - 47 Zhu X, Tam MD, Pierce G, et al. Utility of the Amplatzer Vascular Plug in splenic artery embolization: a comparison study with conventional coil technique. *Cardiovasc Intervent Radiol* 2011;34(3):522–531
 - 48 Schnüriger B, Inaba K, Konstantinidis A, Lustenberger T, Chan LS, Demetriades D. Outcomes of proximal versus distal splenic artery embolization after trauma: a systematic review and meta-analysis. *J Trauma* 2011;70(1):252–260
 - 49 Yoshida H, Mamada Y, Taniai N, Tajiri T. Partial splenic embolization. *Hepatol Res* 2008;38(3):225–233
 - 50 Israel DM, Hassall E, Culham JA, Phillips RR. Partial splenic embolization in children with hypersplenism. *J Pediatr* 1994;124(1):95–100
 - 51 Zhu X, Yang RJ, Wang TW, Chen H, Zhang HZ. The effect of transcatheter partial splenic embolization in patients with hepatocellular carcinoma concomitant hypersplenism. *Radiol Prat* 2002;17:214–216
 - 52 Papatheodoridis GV, Goulis J, Leandro G, Patch D, Burroughs AK. Transjugular intrahepatic portosystemic shunt compared with endoscopic treatment for prevention of variceal rebleeding: a meta-analysis. *Hepatology* 1999;30(3):612–622