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CASE REPORT JSLS

Portal Vein Thrombosis After Laparoscopic Splenectomy: An Ongoing Clinical Challenge

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ABSTRACT

Objectives: Portal vein thrombosis (PVT) following open splenectomy is a potentially lethal complication with an incidence of up to 6%. The objective of this report is to describe our management of a recent laparoscopic case, discuss current therapies, and consider antiplatelet therapy for prophylaxis.

Methods: Medical records, laboratory studies, and imaging studies pertaining to a recent case of a laparoscopic splenectomy were examined. Current literature related to this topic was reviewed.

Results: A 16-year-old girl underwent laparoscopic splenectomy for idiopathic thrombocytopenic purpura. Her preoperative platelet count was 96K. She was discharged on postoperative day 1 after an uneventful operation including division of the splenic hilum with an endoscopic linear stapler. On postoperative day 20, she presented with a 5-day history of epigastric pain, nausea, and low-grade fevers without peritoneal signs. Her white blood cell count was 17.3; her platelets were 476K. Computed tomography demonstrated thrombosis of the splenic, superior mesenteric, and portal veins propagating into the liver. Heparinization was begun followed by an unsuccessful attempt at pharmacologic and mechanical thrombolysis by interventional radiology. Over the next 5 days, her pain resolved, she tolerated a full diet, was converted to oral anticoagulation and sent home. Follow-up radiographic studies demonstrated the development of venous collaterals and cavernous transformation of the portal vein.

Discussion: No standard therapy for PVT exists; several approaches have been described. These include systemic anticoagulation, systemic or regional medical thrombolysis, mechanical thrombolysis, and surgical thrombectomy. Unanswered questions exist about the most effective acute therapy, duration of anticoagulation, and the potential efficacy of routine prophylaxis with perioperative antiplatelet agents. PVT following splenectomy occurs with both the open and laparoscopic approach.

Key Words: Laparoscopic splenectomy, Portal vein thrombosis.

INTRODUCTION

Although mesenteric and portal venous thrombosis have been known clinical entities for over 100 years,[1,2] these processes remain perplexing clinical problems with a wide spectrum of severity and many possible modes of therapy. In general, thrombosis of the mesenteric venous system arises from states of inflammation or infection, trauma (including elective abdominal surgery), or neoplasm, with or without an underlying hypercoagulable tendency.[3,4] Presentation has been described as acute, subacute, and chronic. Patients with acute and subacute mesenteric venous thrombosis can present with nonspecific abdominal pain, nausea, anorexia, vomiting, and diarrhea. Physical examination may reveal normal findings or, in the case of venous infarction, fever, guarding, and rebound tenderness.[4]

Portal and mesenteric venous thrombosis after splenectomy has been a well-recognized complication for decades, with a frequency of 7% to 10% documented by duplex ultrasonography.[5,6] Although some authors have noted that splanchnic venous thrombosis (SVT) is more common in patients with myeloproliferative disorders,[7,8] postsplenectomy SVT has also been noted in patients with hemolytic anemia, idiopathic thrombocytopenic purpura, hereditary spherocytosis, and portal hypertension.[9–11] Prompt diagnosis is critical and is typically achieved via color Doppler ultrasonography[12] or computed tomography.[13]

METHODS

We present a case of postsplenectomy SVT, describe our management of this complication, and review the advan-

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tages and disadvantages of various treatment modalities that have been discussed in the literature with emphasis on the consideration of routine antiplatelet agents for SVT prophylaxis.

RESULTS

A 16-year-old girl underwent uneventful laparoscopic splenectomy for idiopathic thrombocytopenic purpura. Ligation and transection of the splenic hilum was performed with an endoscopic GIA stapler. The blood loss was less than 20 mL, the procedure lasted 106 minutes, and she was discharged home on postoperative day 1, tolerating a regular diet. On postoperative day 20, the patient called to report a 5-day history of nausea without emesis, low-grade fevers, and vague, mild abdominal pain. Her pain was periumbilical and epigastric without radiation. Her temperature was 37.6°C, pulse 74, blood pressure 107/50, respiratory rate 20, and the abdominal examination was significant for mild epigastric tenderness without guarding or rebound tenderness. Laboratory studies revealed the following: white blood cell count (WBC), 17 300/mm^3; hematocrit, 36.1%; platelets, 47 6000/mm^3. Blood chemistries and liver function tests were within normal limits. A workup for hypercoagulability states (factor V Leiden, protein C and S deficiency, prothrombin 20210A mutation, antithrombin III deficiency, and lupus anticoagulant) yielded normal results. A computed tomography (CT) scan of the abdomen and pelvis **(Figure 1A)** revealed[1] thrombosis involving the portal vein, extending into the branch veins,[2] thrombus of the splenic and superior mesenteric veins,[3] fat stranding and mild edema of the mesentery, and[4] a significant amount of pelvic free fluid. An abdominal duplex ultrasound examination corroborated the findings of thrombosed splenic and portal veins and demonstrated normal flow through the hepatic artery and veins.

The patient was promptly anticoagulated with a continuous infusion of heparin, maintaining a partial thromboplastin time (PTT) between 60 and 80 seconds. Because of the extent of thrombosis, her pain, and concern for potential venous mesenteric infarction, on hospital day 2 thrombolysis was attempted by interventional radiology. A transjugular intrahepatic portosystemic shunt (TIPS) procedure was attempted, but the portal venous system could not be cannulated (transjugular or transhepatic routes) and the procedure was aborted. The patient's abdominal examination improved slowly while she was maintained *nil per os* (NPO) and on therapeutic heparin. After instituting oral intake and anticoagulation, the patient's abdominal symptoms gradually abated, and she was discharged on hospital day 10 with normal intestinal function.

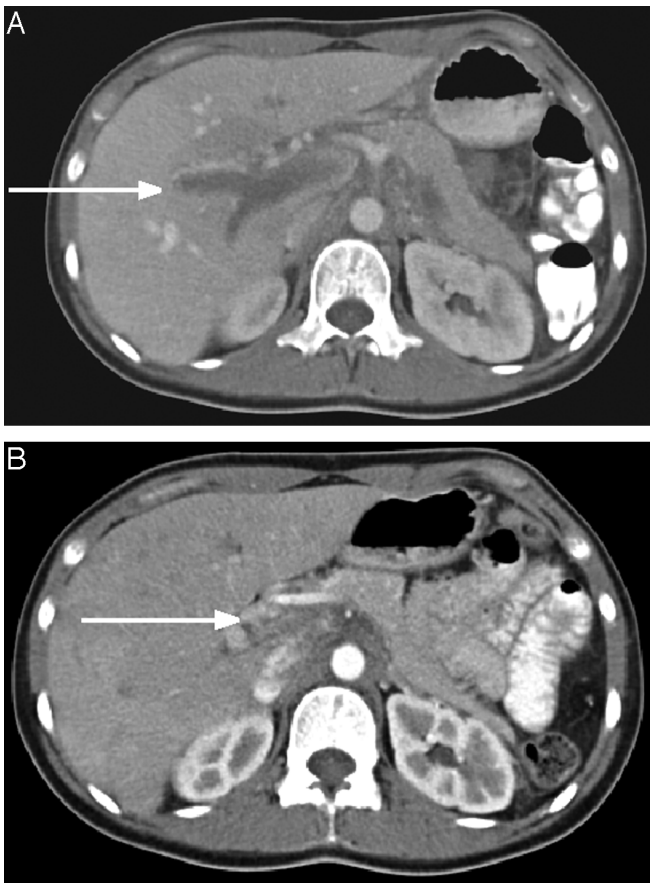

Figure 1. (A) Computed tomography (CT) scan on readmission following laparoscopic splenectomy. Arrow indicates dilated thrombosed portal venous system. (B) Follow-up CT scan 2 months after readmission, demonstrating cavernous transformation of the porta hepatis (arrow).

A follow-up computed tomographic scan 2 months postsplenectomy showed persistent thrombus in the mesenteric venous system with early collateralization and cavernous transformation of the porta hepatis **(Figure 1B).** After a 6-month period, anticoagulation was terminated. At that time, a surveillance ultrasound examination demonstrated flow within the portal vein with extensive collateralization around the porta hepatis. To date, the patient remains clinically stable and asymptomatic.

DISCUSSION

The treatment of patients with postsplenectomy portal vein or splenic vein thrombosis, or both, includes a wide

spectrum between anticoagulation only and emergency laparotomy. Surgical intervention is infrequently required but should not be avoided in cases of peritonitis or when infarcted bowel is suspected. In contrast to arterial intestinal infarction, venous infarction causes an unclear demarcation between viable and nonviable bowel, and "second-look" laparotomy has been proposed to improve survival.[14] Surgical thrombectomy is warranted only in select cases of isolated thrombosis of the superior mesenteric vein, and portosystemic shunt surgery is both infrequently feasible and associated with prohibitively high morbidity.

Other less invasive modes of therapy have been used when, as is often the case, no evidence is present of bowel infarction. In cases of acute mesenteric thrombosis, most authors agree that prompt intravenous anticoagulation is warranted.[15–17] Early anticoagulation promotes recanalization of the portal vein and reduces the risk of splanchnic venous infarction without increasing the risk of variceal bleeding.[18,19] The probability of recanalization is related to the extent of thrombosis.[19] The most appropriate duration of this therapy is unknown, but some authors have demonstrated success with oral anticoagulation for 3 months,[20] and a general guideline of 6 months to 1 year has also been suggested.[4]

Systemic medical thrombolysis with urokinase or recombinant tissue plasminogen activator (tPA) has been used, but carries a higher risk of bleeding complications. Urokinase, a previously effective thrombolytic made from human sources, has been taken off the market, pending a recombinant formulation. In recent years, catheter-based, regional medical thrombolysis has successfully treated SVT. Local thrombolysis can be achieved via a variety of techniques. Pharmacologic thrombolytics can be administered from the arterial circulation (superior mesenteric artery infusion)[21,22] or venous circulation (by the transjugular or transhepatic route–the latter may carry a higher risk of bleeding complications). In addition, devices have been developed to mechanically disrupt and evacuate thrombus, and several case reports describe various combinations of the aforementioned techniques in the treatment of mesenteric thrombosis.[23–25] Although these success stories exist, some authors question the effectiveness of catheter-based techniques, citing underlying pylephlebitis, extension of thrombus to venular levels, rethrombosis, and inadequate outflow as potential obstacles.[26] Currently, no clear indications for interventional radiology techniques exist, and therapy should be based on each patient's clinical circumstances and the abilities of individual institutions.

The potential role of antiplatelet agents in the prevention of SVT has been discussed in the literature. Although quantitative changes in platelet levels have been shown not to correlate with the development of postsplenectomy SVT,[27] qualitative changes in platelet function have been suggested to play a role in thrombotic tendencies.[28] One author[8] has instituted treatment with aspirin and low-dose heparin with good results, but to date, no current standard of care exists regarding the routine use of antiplatelet agents in postsplenectomy patients.

CONCLUSION

In summary, PVT following splenectomy occurs with significant frequency and should be ruled out in patients who present with vague abdominal symptoms. The course of this complication, while potentially devastating, is usually benign and responds well to anticoagulation alone. The indications and overall efficacy of catheter-based thrombolysis remains to be determined, as does the routine use of postoperative antiplatelet agents or anticoagulation.

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