An unconventional therapeutic approach for a severe case of septic pylephlebitis involving the portal system using pharmacomechanical thrombolysis and thrombectomy

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ABSTRACT

Pylephlebitis has a high risk of mortality. Even with optimal medical management, the thrombus may propagate and require intervention. We present one such case in which extension of the thrombus from the superior mesenteric vein to the intrahepatic portal veins led to a life-threatening systemic response. Current endovascular techniques tailored with targeted pharmacomechanical thrombolytic therapy were combined with an exploratory laparotomy for septic control and resulted in an accelerated recovery. (J Vasc Surg Cases and Innovative Techniques 2020;6:262-5.)

Keywords: Pylephlebitis; Pharmacomechanical thrombolysis; Thrombectomy

Septic pylephlebitis is an infected thrombus of the portal system developing from an intra-abdominal infection. This infection leads to septic thrombus in the smaller mesenteric veins that propagates into the larger mesenteric veins and portal system, causing occlusive vascular disease. The most common sources of infection are severe pancreatitis, perforated gangrenous appendicitis, and perforated diverticulitis. This rare disease progresses to death in up to 22% of patients.[1] The optimal treatment guidelines for pylephlebitis have been antibiotics and in some cases systemic anticoagulation. More recently, a few case studies have described interventional techniques used to address venous thrombus. The patient's consent was given for case publication.

CASE REPORT

A 69-year-old woman presented to the emergency department with fatigue, vague epigastric pain, and nausea for 2 weeks with rigors and epigastric tenderness. Her leukocyte and platelet counts and liver function test results were normal. A computed tomography (CT) scan of the abdomen showed thickening of the walls of the gastric antrum with fat stranding between the stomach and pancreas (Fig 1, *A*); antibiotic therapy with piperacillin-tazobactam was initiated. Esophagogastroduodenoscopy was performed, and a toothpick was removed from the posterior aspect of the gastric antrum (Fig 1, *B*). The next day, the patient had a temperature of 104°F, with worsening abdominal pain and distention that improved with intravenous hydration. A repeated CT scan was obtained and showed phlebitis with thrombus in the right and left portal veins, with air and thrombus in the superior mesenteric vein (SMV; Fig 1, *C*). She was started on a heparin infusion and continued on piperacillin-tazobactam. The following day, hypotension, tachypnea, thrombocytopenia, and new elevations in liver function values as well as severe upper abdominal tenderness and distention developed. CT of the abdomen showed more extensive pylephlebitis with occlusive thrombus extending into the left intrahepatic portal vein and a small fluid collection adjacent to the SMV (Fig 2). Sepsis and disseminated intravascular coagulopathy had progressed despite full anticoagulation and appropriate antibiotic therapy. After failed medical therapy, the heparin infusion was stopped, platelets were transfused, and the patient was taken emergently to the operating room.

An upper midline laparotomy incision was performed; the lesser sac was opened, and the small posterior antral perforation was closed primarily with sutures and a Graham patch. There was no purulent fluid or hematoma as was previously expected, only a small collection of serous fluid. The SMV was initially approached for endovascular access; however, because of inflammation and friability of surrounding tissues, the proximal middle colic vein was isolated and a venotomy was made. A 14-gauge Jelco Angiocath (Smiths Medical, Minneapolis, Minn) was inserted. Venography through the Angiocath showed that the left portal vein was completely occluded (Fig 3, *A*). A 0.018-inch guidewire was advanced into the portal vein, followed by a 4F Fogarty balloon catheter. The balloon was inflated and pulled back through the SMV where a clot had been seen on CT, but no thrombus was retrieved. A 6F AngioJet (Boston Scientific, Marlborough, Mass) was advanced over a 0.035-inch × 180-cm Glidewire (Terumo Interventional Systems, Somerset, NJ) into the distal left portal vein. The

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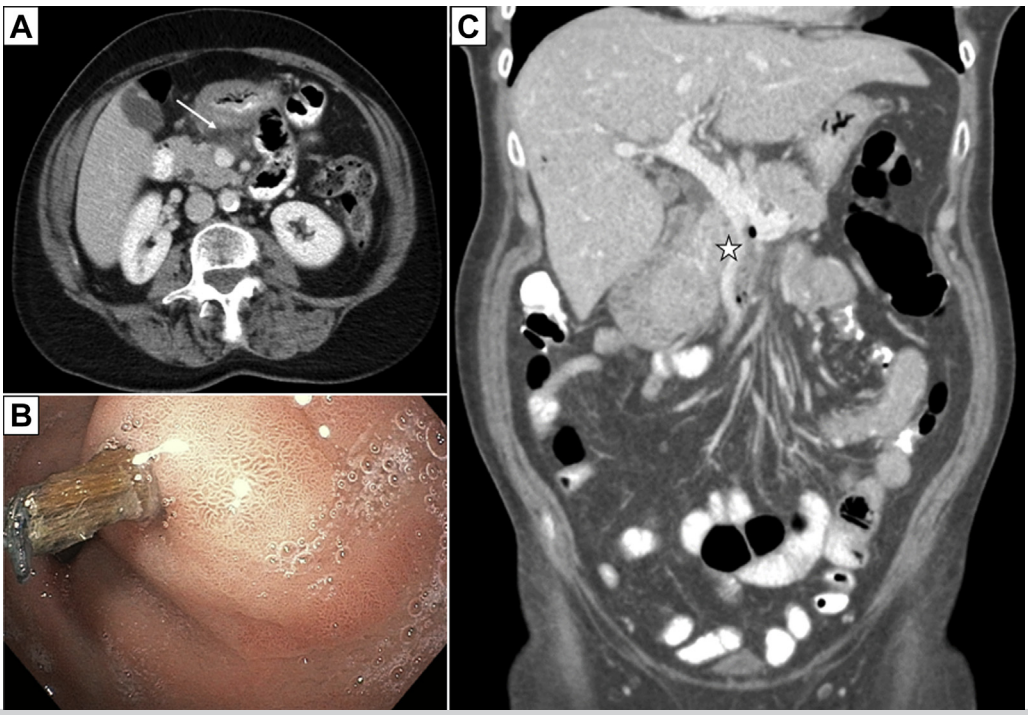

Fig 1. A, Initial computed tomography (CT) scan. Contrast-enhanced axial image shows thickened gastric antrum with surrounding inflammation (*arrow*). **B,** Endoscopic view of toothpick perforating the stomach. **C,** Follow-up CT scan. Contrast-enhanced coronal image shows superior mesenteric vein (SMV) thrombus and intravascular air (*star*).

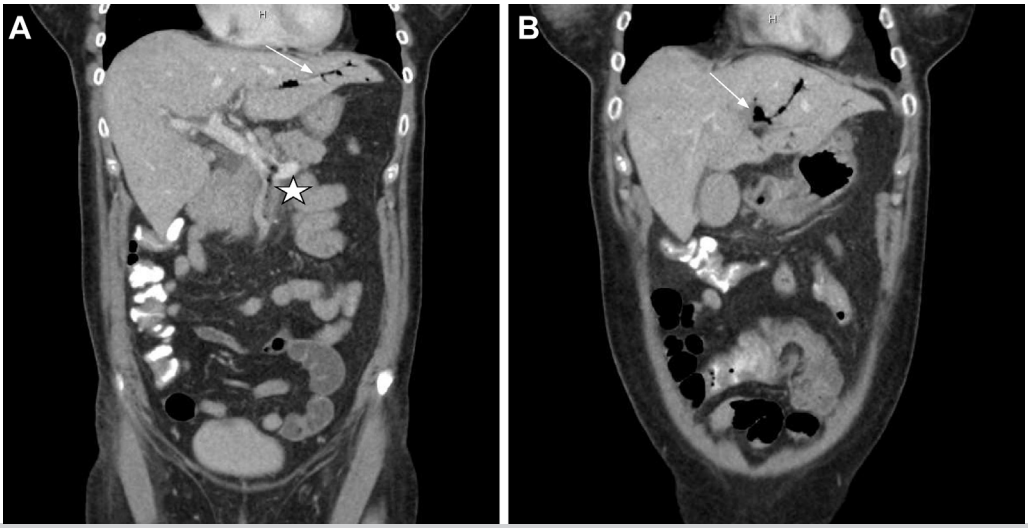

Fig 2. A, Third computed tomography (CT) scan. Contrast-enhanced coronal image shows extension of superior mesenteric vein (SMV) thrombus into portal system (*star*) and portal venous gas (*arrow*). **B,** Additional coronal slice from same scan with obstructed left portal vein and additional portal venous gas.

thrombus was lysed with 3.5 mg of tissue plasminogen activator (tPA) with a dwell time of 15 minutes before pulsed thrombectomy was performed. Venography through the AngioJet showed improvement, but the left portal vein was still distally occluded (Fig 3, *B*). The remaining tPA of AngioJet's standard 10 mg/100 mL normal saline was administered and dwelled 15 minutes, then pulsed thrombectomy was performed. The AngioJet was removed. Completion portal venography through the Berenstein catheter demonstrated patency of the left portal vein (Fig 3, *C*). The catheter was removed and

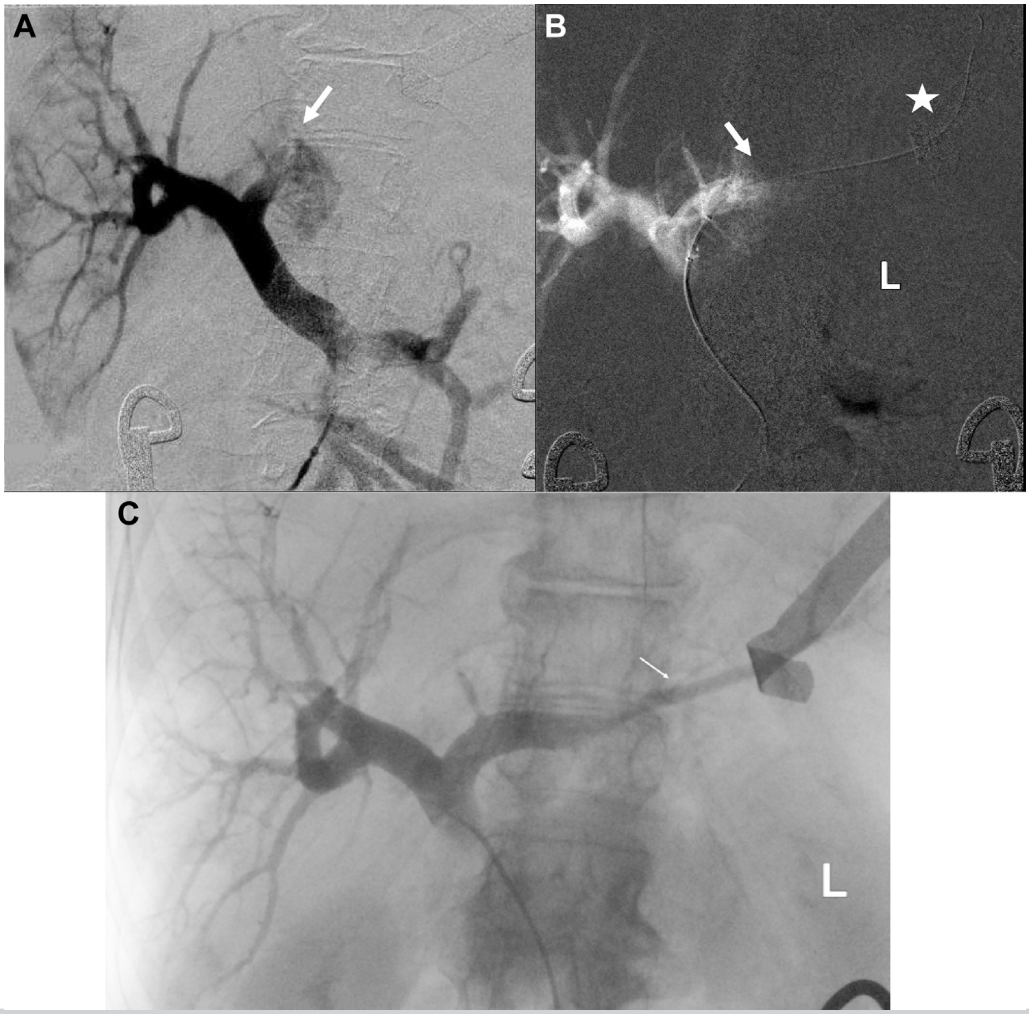

Fig 3. A, Initial venogram showing occlusion of left portal vein (*arrow*). **B,** Venogram through lysis catheter after 3.5 mg of tissue plasminogen activator (tPA) showing improvement but continued occlusion of left portal vein (*arrow*); lysis catheter is present distally in left portal system (*star*). **C,** Completion venogram showing patency of left portal vein (*arrow*).

the vein closed. A 10-mm Jackson-Pratt drain was left in the lesser sac; no active bleeding was identified despite the tPA, and the abdomen was closed.

Heparin was restarted postoperatively, and antibiotics were continued; the patient showed rapid clinical improvement. A repeated CT scan at 1 week showed continued patency of the left portal vein. Preoperative blood cultures eventually grew *Streptococcus intermedius*, *Streptococcus anginosus*, *Streptococcus mitis*, and *Streptococcus oralis*, identifying the causative organisms, with subsequent cultures showing no growth. Ultimately, she was discharged on a 2-week course of intravenous antibiotics and apixaban. Apixaban at a dose of 5 mg twice a day for 3 months was chosen for its ease of use and efficacy in patients with portal venous thrombus. At 12 months of follow-up, ultrasound examination showed complete patency of the portal and mesenteric veins without signs of thrombus (Fig 4). She is doing well with no signs of infection or sequelae of portal vein thrombosis.

DISCUSSION

Historically, treatment of acute suppurative thrombosis of the portal system including the major proximal tributaries was limited to antibiotic therapy with or without systemic anticoagulation.[1] The treatment modalities for pylephlebitis are based mainly on case reports as the incidence is low. Antibiotic therapy alone produces unpredictable results as the infected thrombus becomes stabilized and difficult to penetrate; liver abscesses develop and rapidly lead to life-threatening propagation in a large portion of patients.[1] There is a theoretical benefit to adding systemic anticoagulation, but it is difficult to attribute patient outcomes solely on the basis of this as results are mixed.

Catheter-directed thrombolysis and thrombectomy have gained traction as this approach produces quicker recanalization rates with decreased risk of systemic

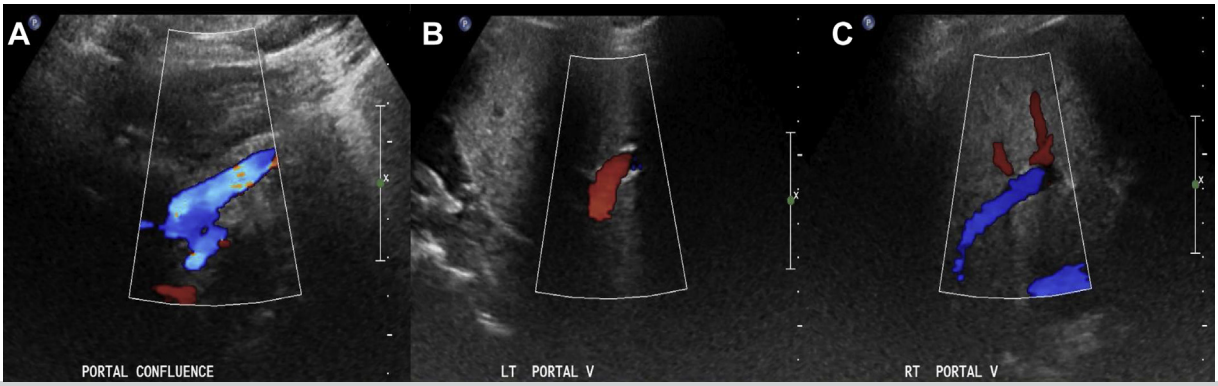

Fig 4. A, The 1-year post-treatment ultrasound image showing patency of superior mesenteric vein (SMV). **B,** Ultrasound image showing patency of left portal vein. **C,** Ultrasound image showing patency of right portal vein.

hemorrhage due to targeted thrombolysis. However, treating portal thrombus presents a unique challenge because of the difficulty of accessing the portal system. When adapted for the use of pylephlebitis, these techniques are modified for the treatment of infected thrombus by infusing thrombolytics and even antibiotic therapy at the site of thrombus.[2] One case study used the surgically recanalized umbilical vein for thrombolytic access because of the inability to use the splanchnic veins for fear of overwhelming intra-abdominal contamination from perforated appendicitis.[3] Treatment of portal vein thrombosis in cirrhosis with infusion of thrombolytics into the superior mesenteric artery has also been established as a treatment option to recanalize the portal system.[4]

Catheter-directed thrombolysis has been described as a method of treating thrombosis of the SMV and portal vein. Recombinant tPA was instilled through a multiside hole catheter inserted into the jejunal vein, and abdominal closure was delayed for 36 hours.[5] This case was effective, but ongoing thrombolysis carries the risk of uncontrolled hemorrhage and delays abdominal closure.

Treatment of pylephlebitis with AngioJet has been described through a transhepatic approach. This approach, however, has the risk of severe hepatic hemorrhage as seen in one of eight patients of the study group, with nearly complete resolution of clot burden in only three of eight patients.[6]

We present a case in which during treatment of the intra-abdominal source of the pylephlebitis, thrombolysis was performed with an AngioJet catheter, which was then removed before closing the abdomen. Had this intervention not been successful in establishing patency of the portal system, we would have considered either placement of an infusion catheter intraoperatively for ongoing thrombolysis with delayed abdominal closure or transhepatic thrombolysis.

CONCLUSIONS

Pylephlebitis continues to represent a highly mortal complication of severe abdominal infections. Medical therapy with antibiotics and optimal anticoagulation alone was unsuccessful in the presented case and many others. The patient required surgical exploration for clinical deterioration and primary source control; concomitant thrombolysis and thrombectomy were used for control of the propagating infectious portal thrombus, which was likely the source of septic shock. We contend that this simultaneous combination of techniques provided a successful, durable, and efficient clinical outcome in this case.

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