Massive Hemorrhage during Percutaneous Transhepatic Portal Vein Thrombolysis

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Interventional radiologists are fortunate that continuous development and improvement of new devices and technologies allow expansion of minimally invasive procedures. With new device implementation, there is a responsibility of the operator to fully understand the device design and utilization for procedure effectiveness and efficiency, and most importantly for patient safety.

Portal vein thrombosis (PVT) is associated with portal hypertension, and definitively portends differences in patient prognosis and management. In the setting of PVT, portal vein thrombolysis is an effective technique for treating thrombosis, improving prognosis, and expanding options for management, but comes with associated risks, the most concerning of which is periprocedural bleeding.

Case Report

A 51-year-old man with a history of ulcerative colitis, primary sclerosing cholangitis, and cirrhosis was found to have extensive intra- and extrahepatic partially occlusive PVT noted on a routine surveillance computed tomography (CT) scan (►Fig. 1A). His previous surveillance CT scan 8 months before was without any evidence of PVT (►Fig. 1B), and the acuity of thrombus development over the previous 8 months was unknown. His PVT was clinically silent and no significant liver insult was present by lab analysis. He had “grade A” cirrhosis as per the Child-Pugh classification, and his underlying liver function was excellent. However, with his history of primary sclerosing cholangitis, it was presumed that he would eventually undergo liver transplantation. With concern that PVT would preclude or complicate the eventual liver transplantation, PVT thrombolysis was requested.

The patient presented as an outpatient to the interventional radiology (IR) division for the elective procedure. A percutaneous transhepatic approach was chosen for planned mechanical and pharmacological thrombolysis. A single puncture was performed into a branch of the right portal vein under ultra-

sound guidance. The AngioJet device (Possis Medical, Inc., Minneapolis, MN) was initially utilized for mechanical pulse spray of thrombus with 6 mg tissue plasminogen activator (tPA), followed by further mechanical lysis with aspiration. With significant thrombus burden remaining, a coaxial system was placed through a 6F sheath into the branch of the right portal vein, and an 18-cm infusion length EKOS catheter (EKOS Corporation, Bothell, WA) was placed through the PVT into the superior mesenteric vein (SMV) for ultrasound-assisted thrombolytic infusion. To facilitate intrahepatic thrombolysis, a shorter length sheath was chosen such that the working length of the infusion catheter would be external to the sheath and within the intrahepatic right portal vein (►Fig. 2). tPA infusion was started at 1 mg/h, with concomitant heparin at 400 units/h through the sheath. The patient was admitted to the intensive care unit (ICU) for thrombolysis monitoring.

Overnight, the patient developed severe abdominal pain and hypotension. Hemoglobin and hematocrit had fallen from 14.9 g/dL and 44.5% preprocedure to 8.5 g/dL and 25.7%, respectively. Fibrinogen decreased from 244 to 107 mg/dL. The patients' clinical condition continued to worsen with increasingly tense abdominal distension, worsening renal function, respiratory failure requiring intubation, and hypotension unresponsive to large volume fluid and blood product resuscitation. The infusion was stopped, and the patient was brought emergently to the IR suite, where it was discovered that the sheath had dislodged from the liver (►Fig. 3). The sheath was upsized to a 7F × 30 cm transhepatic sheath, which was advanced into the portal vein in an attempt to tamponade any further bleeding (►Fig. 4). Immediately postprocedure, a CT scan was performed demonstrating massive hemoperitoneum (►Fig. 5). In grave condition, but with even worsened prognosis in the setting of abdominal compartment syndrome, the patient was taken emergently to the operating room for hematoma evacuation. The patient did not recover, and in multiorgan-system failure expired the same day.

Figure 1 (A) Axial contrast-enhanced CT scan in portal venous phase from October 2011 demonstrates cirrhotic liver morphology with patency of the right portal vein (arrow). (B) Subsequent CT scan from June 2012 demonstrates near-completely occlusive thrombus of the right portal vein. Adjacent images showed extensive thrombus also involving the main portal vein. CT, computed tomography.

Discussion

Nonneoplastic PVT is encountered in 0.6 to 26% of individuals with liver cirrhosis.^{1,2} The prevalence of PVT increases with the severity of cirrhosis, being less than 1% in patients with compensated cirrhosis but 8 to 25% in candidates for liver transplantation.³⁻⁵ Acute PVT can lead to intestinal congestion and ischemia, presenting as abdominal pain or distention; diarrhea; rectal bleeding; fever; lactic acidosis; and sepsis. Without relief of the obstruction, intestinal perforation, peritonitis, shock, and death from multiorgan failure can occur. In contrast, chronic PVT can be nearly clinically silent, except for the presence of varices, cutaneous collaterals, or ascites.⁶

Figure 2 Spot fluoroscopic radiograph (A) and zoomed in (B) images demonstrate final positioning of percutaneous intrahepatic sheath and EKOS infusion catheter (EKOS Corporation, Bothell, WA). The most proximal radio-opaque marker shown is that of the infusion catheter (arrowhead), with the distal tip of the sheath positioned even further laterally, in the subcapsular region (arrow).

Figure 3 Spot fluoroscopic radiograph demonstrating retraction of the initially placed shorter sheath, in an extracapsular location (arrow).

Figure 4 Spot fluoroscopic radiograph demonstrating upsizing and extension of sheath, now completely transhepatic and terminating in the portal vein (arrow).

While PVT was once considered an absolute contraindication to orthotopic liver transplantation, with advancement of medical care, surgical techniques, and radiologic interventions, it is now possible to perform transplantation in patients with PVT.⁶ Despite innovations, however, PVT can lead to thrombosis-related cavernous transformation and involvement of the splenomesenteric confluence, which continues to preclude attempts at transplantation. In addition, PVT has been shown to cause increased technical difficulties and increased postoperative morbidity and mortality rates.⁷ To decrease these transplant-related risks and the risk of preoperative upstream thrombus extension or cavernous transformation, pretransplant endovascular thrombolysis has gained favor in some centers in the management of PVT.

Both transjugular intrahepatic and direct transhepatic interventional approaches have been described for direct thrombolysis of PVT^{8,9}; transcatheter techniques include mechanical thrombectomy, pharmacologic lysis, and combination techniques. While routinely performed many years ago, transplenic access of the portal vein is now uncommonly performed.

Transjugular hepatic vein to portal vein access (transvenous intrahepatic portosystemic shunt [TIPS]) has advantages in portal vein thrombolysis including the ability to access the portal vein with larger devices, theoretically less bleeding risk than percutaneous direct approaches, and the natural creation of portal vein outflow to facilitate against rethrombosis. TIPS access to the portal vein has the disadvantage of providing poor access to peripheral intrahepatic portal vein branches and, if left in place following the lysis procedure, may lead to hepatic encephalopathy. Percutaneous transhepatic direct portal vein access allows for access along the long axis of the portal vein, providing for improved catheter control, as well as more easily providing access to peripheral portal vein branches. Disadvantages of the direct transhepatic approach include restriction of device size

Figure 5 (A and B) Postsheath repositioning noncontrast CT scan of the abdomen demonstrates large subcapsular hematoma and large volume hemoperitoneum. A portion of the replaced transhepatic sheath (arrow) and contrast related to sheath repositioning and portography (arrowhead) is shown in right portal vein branches. CT, computed tomography.

limited by access size and increased bleeding risk due to the transcapsular approach.¹⁰

The major concern of the transhepatic route is postprocedural bleeding, including large volume intraperitoneal hemorrhage. Bleeding following percutaneous direct portal vein access in the absence of transhepatic tract closure has been reported to exceed 30%.¹¹ Postprocedural bleeding rate is increased when patients are anticoagulated or receiving pharmaceutical thrombolytic therapy. In patients with cirrhosis and portal hypertension, bleeding risk is often further increased secondary to gastroesophageal varices and portal hypertensive gastropathy.

Upon review, there were a number of errors made during this procedure and clinical case. First, despite being handled

by a relatively experienced operator, the EKOS catheter was a device with which the operators were relatively unfamiliar. This operative team had never before used the device clinically, and despite an in-service presentation (including hands-on using the device in a dry lab setup), the device had never been visualized fluoroscopically. As such, the proximal radio-opaque marker on the catheter was misinterpreted on the initial postprocedural fluoroscopic radiographs as being that of the distal end of the sheath. The distal end of the sheath was in fact far more lateral in position, and barely positioned in the liver parenchyma at all (►Fig. 2A). In retrospect, it was determined that bleeding occurred via the tract into the subcapsular space; the subcapsular hematoma further displaced the sheath until it was in a completely extraparenchymal position. This sheath retraction led to further bleeding due to the loss of tract tamponade effect. With new and improved devices continuously becoming available, which make interventionalists better at what they do, it is critical that all new technologies be fully understood for effective and safe implementation. The authors have now made it a standard practice to fluoroscopically visualize new technologies, in addition to holding it in their hands, to simulate the expected appearance of a device during actual clinical practice.

Second, despite the asymptomatic and overall wellness of the patient on presentation, this patient had a serious disease, one which portends a tenuous postprocedural course at best. When initially introducing new technologies into one's clinical practice and learning about subtleties of use, a more conservative approach would be to first gain experience using the device in a lower risk patient population. The obligatory inexperience inherently associated with implementing new technology is generally better tolerated by healthier patients.

Finally, communication issues arose during this case that delayed necessary action. The case was primarily performed by an attending operator, without much trainee involvement, and the attending operator of the case directly signed out the case to the on-call attending interventionalist. The on-call IR fellow, who primarily responds to IR-related issues overnight, was not directly made aware of the patient. In this case, initial communication from the ICU to the IR fellow on-call was met with some confusion, as the fellow was not aware that the patient was, in fact, undergoing thrombolysis and is very much a patient of the IR service. That initial miscommunication further led to subsequent delay as the fellow tried to manage the case independently before contacting the on-call attending. Here, a lack of standardization, and straying from the more regular divisional practice of directly involving the first-call fellow in the sign-out conversation, resulted in miscommunication and delays in necessary subsequent man-

agement steps. While it can be difficult in even a moderately busy division with multiple attending operators, fellows, and other trainees to rigorously establish this daily communication structure, it is imperative for every single patient.

In summary, the authors opine that the following three issues were all contributory to the poor outcome in this patient:

1. New technology should not be used for the first time in difficult or high-risk patients—one should gain experience in lower risk patients before using it in more challenging cases/patients.
2. In addition to familiarizing oneself with new devices by holding it in one's hands, fluoroscopically visualize new technology to most simulate the appearance of a device in a clinical case.
3. Apply rigorous standards of communication and when communication strays from standardization have measures in place to get standardization back on track—sign out must always include the primary on-call contact person.

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