Hepatic Laceration from Wedged Venography Performed before Transjugular Intrahepatic Portosystemic Shunt Placement[1]

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TRANSJUGULAR intrahepatic portosystemic shunt (TIPS) placement is an increasingly used, nonoperative technique for treating variceal bleeding and refractory ascites secondary to portal hypertension (1–3). Since the first clinical TIPS case in 1989, the procedure has undergone significant technical refinement to improve the safety and efficacy of shunt placement (4–7). A major technical challenge of TIPS creation is passage of the transjugular needle from the hepatic vein into the portal vein. Perforation of the liver capsule from an errant needle pass can lead to massive intraperitoneal bleeding. To minimize the number of needle passes required to enter the portal vein, investigators have devised a variety of techniques to visualize the portal vein anatomy including direct transhepatic catheterization of the portal vein (1,2), superior mesenteric artery (SMA) angiography (1), real-time ultrasound (US) guidance (2,8), and refluxing contrast medium into the portal vein with wedged hepatic venography (1,6). While these technical improvements have made TIPS a safe and attractive alternative to conventional surgical shunts, the procedure remains technically challenging and lethal hemorrhagic complications can occur when the liver capsule is perforated during the course of the procedure (9). To our knowledge, there are no reported major complications directly related to the wedged hepatic venogram prior to TIPS. We describe an unusual series of severe liver injuries from wedged hepatic venography during attempts to localize the portal vein.

CASE REPORTS

During July 1992 through October 1994, 85 patients underwent TIPS placement at a single tertiary referral center for bleeding gastroesophageal varices (n = 75) or refractory ascites (n = 10) with use of the method described by LaBerge et al (2). All patients are given general anesthetic and undergo wedged venography to localize the portal vein. Three patients with refractory ascites sustained major bleeding complications from injury to the liver capsule and parenchyma during wedged hepatic venography prior to placement of the shunt.

Patient 1.—A 68-year-old man with alcoholic liver disease was admitted for refractory ascites which was unsuccessfully treated over a 2-month period despite aggressive medical management. Laboratory values following administration of six units of fresh frozen plasma prior to the procedure included a hematocrit of 42% (normal, 40%–52%), platelet count of 83,000/mL (normal, 150,000–400,000/mL), prothrombin time of 14.5 seconds (normal, 10.6–12.2 seconds), partial thromboplastin time of 29.4 seconds (normal, 23–33 seconds), total bilirubin level of 2.2 mg/dL (37.6 μmol/L) (normal, 0.7–1.5 mg/dL [12.0–25.6 μmol/L]), and albumin level of 2.4 g/dL (normal, 3.3–5.0 g/dL). Hepatic venography was performed with injection of 8–10 mL of diluted (50%) nonionic contrast medium (300 mg of iodine per milliliter) and 0.9% saline solution at an approximate rate of 4–5 mL/sec with the catheter wedged in the distal hepatic vein. Venography resulted in subcapsular extravasation of contrast material and partial reflux into the portal vein (Fig 1). The procedure was technically uncomplicated and accomplished without extracapsular needle passes.

Follow-up sonograms obtained on the 1st postoperative day showed a patent shunt and a 3.0 × 4.5-cm subcapsular hematoma in the right hepatic lobe from the wedged hepatic ve-

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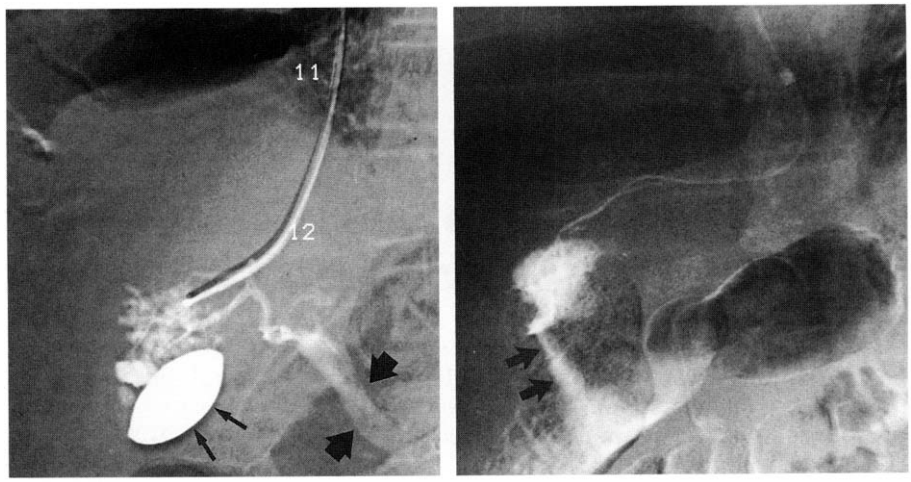
nogram. Over the next 24 hours, the patient developed a decreasing hematocrit and eventual cardiopulmonary decompensation. Emergent laparotomy revealed copious bloody ascites and localized infarction with active bleeding from the liver capsule in the region of the extravasated contrast material. The capsule was oversewn, and the liver bed was packed. The patient subsequently developed multiple organ system failure and died on the 3rd postoperative day despite aggressive medical management including attempts to correct the underlying coagulopathy.

Patient 2.—A 44-year-old man with hepatitis B-induced cirrhosis was admitted for the treatment of refractory ascites. Laboratory values included a hematocrit of 25%, platelet count of 93,000/mL, prothrombin time of 15.4 seconds (after administration of fresh frozen plasma), partial thromboplastin time of 37.8 seconds, total bilirubin level of 1.1 mg/dL (18.8 μmol/L), and albumin level of 2.2 g/dL. The patient was given general anesthesia and a wedged right hepatic venogram was obtained from the internal jugular vein approach as described in the preceding case, except an extra side hole (0.025-inch diameter) was made near the tip of the catheter to reduce the shearing force of the contrast material injection. The venogram demonstrated immediate extravasation of contrast material through the liver capsule and into the peritoneal cavity (Fig 2). The distal hepatic vein was embolized by using two 2-mm-diameter stainless steel embolization coils (Cook, Bloomington, Ind). Transhepatic catheterization of the portal vein was uneventful and without extracapsular passes with use of a Rosch needle (10). The patient remained intubated following shunt placement and was transferred to the intensive care unit with intermittent hypotension and abnormal coagulation indexes.

The hemodynamic instability and coagulopathy worsened over the next 24 hours despite aggressive medical management. An emergent laparotomy revealed massive bloody ascites and a small perforation of the liver capsule along the surface of the posterior right hepatic segment with associated brisk bleeding and infarcted liver parenchyma. The defect was repaired and the liver bed packed; however, the patient died on the 2nd postoperative day from multiple organ system failure.



1. 2.

Figures 1, 2. **(1)** Wedged hepatic venogram obtained with use of a 5-F end-hole catheter with subcapsular extravasation of iodinated contrast medium (small arrows). The main portal vein is seen adjacent to the L-1 vertebral body (large arrows). The subcapsular collection eventually infarcted the parenchyma and perforated the liver capsule, leading to significant intraperitoneal bleeding. **(2)** Wedged hepatic venogram with immediate free extravasation of iodinated contrast medium into the peritoneal cavity (arrows). The distal hepatic vein was embolized with two small coils, but the patient continued to bleed directly from the liver capsule.

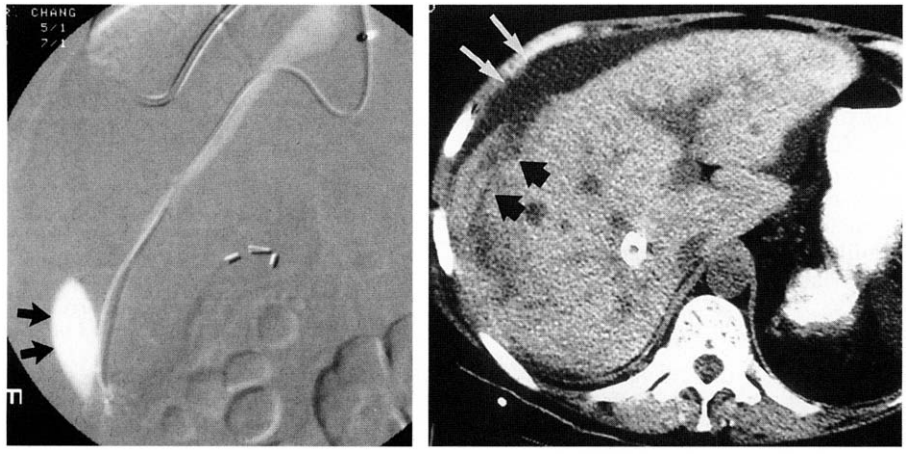
Patient 3.—A 29-year-old woman with systemic lupus erythematosus and noncirrhotic portal hypertension was admitted for treatment of massive refractory ascites and recurrent pleural effusions. Laboratory values included a hematocrit of 27%, platelet count of 153,000/mL, prothrombin time of 11.3 seconds, partial thromboplastin time of 25.4 seconds, total bilirubin level of 0.4 mg/dL (6.8 μmol/L), and albumin level of 2.0 g/dL. Wedged hepatic venography was performed with use of 100% gaseous carbon dioxide instead of iodinated contrast medium to reduce the risk of capsular injury. Immediately following the manual injection of CO_2 (noncompressed total volume of 60 mL at room temperature injected at 40–60 mL/sec), a large subcapsular gas collection was noted along the right lateral margin of the liver (Fig 3a). The TIPS was placed in routine manner without immediate complications.

Over the next 24 hours, there was a significant decrease in hematocrit and marked elevation of liver function indexes though the patient underwent aggressive measures to correct the coagulopathy. An abdominal computed tomographic (CT) scan revealed a moderate-sized hepatic subcapsular hematoma with laceration of the liver parenchyma (Fig 3b). A diagnostic paracentesis revealed grossly bloody ascites, and celiac angiography was performed to locate a potential bleeding source. No active hemorrhage was identified and the TIPS was widely patent. The patient developed multiple organ system failure and died 13 days following TIPS placement.

DISCUSSION

TIPS placement is rapidly evolving into a safe and effective technique for treating gastroesophageal varices and refractory ascites secondary to portal hypertension (11). TIPS creation offers the advantage of direct decompression of the portal system without the inherent risk associated with laparotomy in patients with advanced liver disease.

Percutaneous portosystemic shunting continues to undergo refinement as the number of centers performing the procedure increases. Consequently, as investigators gain experience



a. b.

Figure 3. **(a)** Digital subtraction wedged hepatic venogram obtained with use of CO_2 gas. A large subcapsular collection of free gas is present (arrows). **(b)** Follow-up abdominal CT scan shows a subcapsular hematoma (white arrows) and a frank parenchymal laceration (black arrows).

with the technique, reports of technical complications have emerged (9). The most difficult and potentially hazardous step in TIPS placement is the transhepatic needle pass from the hepatic vein into the portal vein tributaries. Reports describe a variety of complications related to attempted needle passage into the portal vein including extracapsular puncture (6,9), biliary duct puncture (6,9), hepatic artery puncture (12,13), gallbladder puncture (6,9), right kidney puncture (9), and extrahepatic portal vein puncture (14).

Several techniques have been proposed to localize the portal vein before TIPS placement to reduce the complications of needle-related injuries and to decrease the time required to place the shunt. These include wedged hepatic venography with iodinated contrast media (1,6) or CO_2 (15), direct transhepatic catheterization of the portal vein (1,2), SMA angiography (1), transmesenteric portal vein catheterization through a surgical cutdown (16), US guidance of the transjugular needle (17), US-guided placement of a metallic marker into liver (18), US-guided placement of wire into the portal vein (19), US-guided catheterization of the umbilical vein (20), magnetic resonance angiographic guidance (21), and ex vivo evaluations of human livers (22,23).

While there is no generally accepted method of portal vein visualization, many of the larger clinical series use either wedged hepatic venography or catheterization of the SMA for guidance (6,14). Wedged hepatic venography is the simplest and quickest method of identifying the portal vein, since no additional catheterization, preparation, or equipment is needed. Other techniques to localize the portal circulation add considerable time and expense, especially in the setting of the critically ill patient who is actively bleeding and requires urgent portal vein decompression (16–21). We have found that these alternative techniques are most useful in the early phase of experience with the procedure when the investigator is learning to guide the transhepatic needle. The main disadvantage of wedged hepatic venography is the lack of consistency in refluxing contrast material through the hepatic sinusoids into the portal vein; in only 40% of our cases was the venogram sufficiently diagnostic.

Potentially significant liver injury from wedged venography was described in the 1970s when venography was routinely used for evaluation of liver disease. Bookstein et al reported three cases of subcapsular extravasation of contrast medium with no clinical sequelae with injection of 3 mL over 2 seconds (60% methylglucamine diatrizoate) in a series of 40 patients (24). Casteneda-Zuniga et al used a dog model to study the effects of extravasated contrast medium in the liver and injected 6 mL at a rate of 2 mL/sec (60% methylglucamine diatrizoate) (25). Histologic analysis of these livers demonstrated extensive hemorrhagic infarction and severe generalized congestion of the hepatic lobules. These findings prompted the conclusion that "the risk of parenchymal damage is real; and even when the amount of contrast injected is low, this additional parenchymal damage may be a significant burden to a severely diseased liver; it is probably not clinically and pathologically important to the normally functioning liver" (25). The main reason for the hepatic injuries described in this report is due to overvigorous injection during wedged venography.

Because the viscosity of the iodinated contrast mixture creates a tremendous shearing force when injected through a small end-hole catheter, we switched to the use of CO_2 as described by Rees et al (15). CO_2 is easy to use, inexpensive, and has no nephrotoxic effects in this setting. The anatomy of the portal vein and its bifurcation is often more completely visualized with CO_2 than with iodinated contrast material. Rees et al reported extravasation of CO_2 gas into the liver parenchyma that evolved into small subcapsular hematomas on follow-up US examinations in several patients. These were all clinically insignificant with no major complications reported. Our injury was due to overinjection of the gas with the catheter wedged very distally in the hepatic vein approximately 2 cm from the hepatic capsule.

Patients considered for TIPS placement are at risk for a hemorrhagic catastrophe because of poor coagulation parameters even with aggressive transfusions of plasma and other blood products. It is our conjecture that ascites increases the risk of significant bleeding complications from both errant needle passes and venographically-induced capsular injuries though there are no clinical studies, to our knowledge, that have specifically evaluated this parameter. The presence of massive ascites may impair localized tamponade of a small capsular tear resulting in continued bleeding

into the peritoneal cavity. If there is clinical evidence of bleeding from a lacerated liver capsule, angiography is virtually useless in detecting slow, oozing bleeding from the capsule. Embolization of the distal hepatic vein adjacent to the site of contrast material extravasation is also minimally effective if the source of bleeding is from the liver parenchyma through the torn capsule into the ascites-filled abdomen. Embolotherapy or immediate exploratory laparotomy with repair of the liver capsule may be the only therapeutic maneuvers possible in these patients who continue to bleed. From our experience, these patients inevitably will die from continued bleeding and associated complications.

Our experience demonstrates that severe liver injury and intraperitoneal bleeding can occur with either liquid or gaseous contrast medium during wedged venography prior to TIPS and should serve as a warning that significant massive and lethal hepatic injury can occur if injection is too forceful, especially in patients with massive ascites. We have subsequently used CO_2 in 18 additional patients with exquisite delineation of the portal anatomy in 16. CO_2 gas is now used exclusively during wedged venography but with significantly less volume and force (noncompressed volume of 30–50 mL injected at 15 mL/sec) and we have had no further traumatic hepatic injuries. CO_2, when injected at lower rates, is probably superior to and safer than iodinated contrast media for wedged venography. The development of an automated CO_2 injection pump will allow more uniform and consistent boluses than manual injection. We recommend that the catheter should be wedged in a relatively central portion of the liver, avoiding the periphery near the capsule, and care must be taken to purge any remaining blood or saline solution in the catheter with 1–2 mL of CO_2 gas prior to the actual angiographic injection. In instances when we cannot reflux the gas into the portal vein, we typically proceed with the transhepatic needle pass. If there is difficulty puncturing portal branches, a small volume of iodinated contrast media is directly injected through the needle. This will often result in satisfactory reflux into the portal system. Care must be taken not to overinject, as the extravasated contrast material may obscure the underlying anatomic landmarks. We hope continued refinement of techniques will further increase the safety profile of the TIPS procedure in these high-risk and often critically ill patients.

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