

CASE REPORTS

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Mortality during Transjugular Intrahepatic Portosystemic Shunt Placement

Anna E. Yonker-Sell, M.D.,* Lois A. Connolly, M.D.†

TRANSJUGULAR intrahepatic portosystemic shunting (TIPS) is a new technique to provide a bridge to liver transplantation, prevent gastroesophageal bleeding from portal hypertension, and mobilize refractory ascites in patients with end-stage liver disease. Defined, it is the placement of an expandable metallic stent between the branches of the portal vein and systemic circulation within the liver parenchyma. The role of the anesthesiologist in this procedure is based on the need for conscious sedation and monitoring in a site remote from the operating room. This type of shunt was first attempted in humans in 1983¹ with subsequent modifications that have led to multiple clinical trials supporting its efficacy and safety.²⁻⁷ However, the list of possible complications associated with the procedure, including hepatic encephalopathy, stent occlusion and/or stenosis, bacteremia, portal vein thrombosis, pulmonary emboli, stent migration, hemolysis, acute renal insufficiency, liver capsule rupture, gall bladder laceration, and intraperitoneal hemorrhage,⁸ has increased as its use becomes widespread. Most complications occur after the procedure. An extensive literature review revealed no mortalities reported thus far. We comment on an unfortunate complication occurring during stent placement that ultimately led to the patient's death.

* Resident.

† Assistant Professor.

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Address reprint requests to Dr. Connolly: Department of Anesthesiology, Medical College of Wisconsin, Froedtert Memorial Lutheran Hospital, 9200 West Wisconsin Avenue, Milwaukee, Wisconsin 53226.

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Case Report

A 70-yr-old, 81-kg man with alcoholic cirrhosis was scheduled to undergo an emergent TIPS procedure. Thirty-six hours before admission, while awaiting cardiac catheterization for new onset chest pain, gastroesophageal bleeding developed, requiring a 6-unit packed erythrocyte infusion. He underwent two endoscopies with sclerotherapy but continued to have slow blood loss. While awaiting scheduled TIPS procedure, frank hematemesis and hypotension developed. The patient underwent emergent endoscopy, but the site was not amenable to banding or sclerotherapy. He was scheduled for emergent TIPS procedure. His medical history was significant for the incidental finding of a 5-cm abdominal aortic aneurysm and the recent development of atypical chest pain, with further diagnostic therapy delayed secondary to his gastroesophageal bleeding. His chest pain was relieved with nitroglycerin patch. He had no episodes of heart failure or arrhythmias. Electrocardiography showed normal sinus rhythm without signs of ischemic changes or prior infarction. There was no history of encephalopathy or renal dysfunction. Current medications included sucralfate, nitroglycerin patch, and furosemide. Physical examination was unremarkable with the exception of the presence of abdominal ascites and pedal edema. Laboratory values were as follows: hematocrit 33% after receiving 2 units of packed erythrocytes, platelet count $110,000 \text{ mm}^{-3}$, prothrombin time 15.7 s, partial thromboplastin time 29.9 s, Na 139 mmol/l, K^+ 3.9 mmol/l, Cl 107 mmol/l, HCO_3^- 23 mmol/l, blood urea nitrogen 9 mg/dl, Cr 0.7 mg/dl, glucose 93 mg dl^{-1} , and NH_4^+ 60 $\mu\text{mol/l}$.

Two 18-G peripheral intravenous catheters and a 20-G right radial arterial catheter were placed, and standard ASA monitoring was performed in the angiography suite. Further invasive monitoring was not necessary because of the stability of the patient's chest pain with nitroglycerin patch, normal electrocardiography results, no history of congestive heart failure or arrhythmia, and the relatively hemodynamic stability of the TIPS procedure. Sedation was achieved with $50 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ propofol, 2 mg midazolam in 0.5-mg increments, and 100 μg fentanyl in 25- μg boluses intravenously. Two hours after the start of the procedure, the patient's systolic blood pressure decreased from 110 to 60 mmHg with an increase in heart rate to 110 beats/min. No ST-T changes were noted on electrocardiogram monitoring. This coincided with dilation of the Wallstent device. As the patient lost consciousness, ventilation was supported with 100% O_2 via mask followed immediately by direct laryngoscopy and intubation of the trachea. Hypotension was treated with a total of 200 μg phenylephrine without effect, at which time 100- μg incremental boluses of epinephrine (total 400 μg) were titrated to achieve a systolic blood pressure in the range of 90 mmHg. Massive extravasation of contrast at the junction of the inferior vena cava and right hepatic

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vein was confirmed by fluoroscopy and was determined to have been caused by a tear made during the dilation of the Wallstent device. Blood flow distal to the laceration was intermittently occluded while attempts to bypass the laceration with a balloon stent were performed. The patient's blood pressure was maintained using crystalloid and blood products, including packed erythrocytes, whole blood, and fresh frozen plasma, as well as a dopamine infusion ($10\text{--}15\text{ }\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and $100\text{-}\mu\text{g}$ boluses of epinephrine (total $800\text{ }\mu\text{g}$ over 55 min). Small doses of midazolam and fentanyl were given during this period. A surgical consultation obtained immediately determined that surgical repair was technically impossible because of the site of the laceration. Attempts to repair the laceration with multiple balloon stents failed. Sixty minutes after initiating resuscitative measures, the patient's family opted to withdraw support, and the patient died of irreversible hypovolemic shock.

Discussion

The most common cause of portal hypertension in the United States remains alcohol-induced cirrhosis of the liver.⁹ The incidence of initial variceal bleeding in patients with liver disease is between 27% and 48%.¹⁰ Mortality is approximately 50% after each episode of bleeding¹¹ with an initial 6-month mortality risk of 40–60%.^{12,13} Because of this high early mortality, attention has been focused on prompt intervention and prophylaxis. Therapy has included the use of nonselective β blockers, endoscopic sclerotherapy, shunt surgery, and more recently, TIPS. Endoscopic sclerotherapy can control active bleeding in 90% of the patients,^{14,15} and recurrent gastroesophageal bleeding is usually amenable to repeat sclerotherapy. The small percentage of patients who do not respond to sclerotherapy become candidates for the placement of shunts.

Van Buuren *et al.* found that the incidence of long-term complications was comparable between TIPS and surgical shunting, but TIPS was associated with a lower morbidity and mortality.^{16,17} TIPS has the advantage of being performed under local anesthesia with sedation, is nonoperative with procedural trauma limited to a puncture wound, and can be performed safely in patients awaiting liver transplantation.⁵ The presence of a surgical shunt has been associated with a longer duration of surgery during liver transplants, greater procedural difficulty, and greater transfusion requirements compared to patients with TIPS.^{6,8} In addition, the success rate of TIPS procedures appears to be about 90%, suggesting the procedure is technically feasible.⁴

Multiple complications from TIPS have been reported, with the incidence of hepatic encephalopathy and shunt occlusion the most common.⁸ Hepatic encephalopathy has been reported in 10–20% of patients

within 1 month of the procedure.⁸ Shunt occlusion occurs in 5–10% of patients but rarely in the immediate postprocedural period.⁸ Bleeding, including intraperitoneal hemorrhage, biliary hemorrhage, or liver capsule hematomas, was reported by Haag *et al.* in 15 patients; however, all but two resolved spontaneously. Of the two affected, one died from disseminated intravascular coagulopathy, and the other improved after therapy for coagulopathy was instituted.⁴

Currently, mortality has not been associated with the technical aspects of performing a TIPS procedure. The role of the anesthesiologist is to provide the necessary support should inadvertent complications occur, remembering that most angiography suites are not well equipped for the management of an actively bleeding patient in hypovolemic shock. Advocating invasive monitoring in otherwise stable patients is not encouraged, but full pharmacologic and airway support should be available in angiography suites even when a stable course is expected in these otherwise complicated patients. The placement of TIPS appears to be a breakthrough that will be increasingly introduced in most hospitals that require active participation of the anesthesia team.

References

1. Colapinto RF, Stronell RD, Gildiner M, Ritchie AC, Langer B, Taylor BR, Blendis LM: Formation of intrahepatic portosystemic shunts using a balloon dilatation catheter: Preliminary clinical experience. *Am J Radiol* 1983; 140:709–14
2. Rousseau H, Vinel JP, Bilbao JJ, Longo JM, Maquin P, Zozaya JM, Garcia-Villareal L, Coustet B, Railhac N, Railhac JJ: Transjugular intrahepatic portosystemic shunts using the Wallstent prosthesis: A follow-up study. *Cardiovasc Intervent Radiol* 1994; 17:7–11
3. Van Buuren HR, Cheng KH, Pieterman H, Schalm SW, Lameris JS: Transjugular intrahepatic portosystemic shunt: Requiem for the surgical portosystemic shunt? *Scand J Gastroenterol* 1993; 28:48–52
4. Rossle M, Haag K, Ochs A, Sellinger M, Noldge G, Perarnau J-M, Berger E, Blum U, Gabelmann A, Hauenstein K, Langer M, Gerok W: TIPS: To be or not to be? *Gastroenterology* 1994; 107:591–7
5. Freeman RB Jr, FitzMaurice SE, Greenfield AE, Halin N, Haug CE, Rohrer RJ: Is the transjugular intrahepatic portocaval shunt procedure beneficial for liver transplant recipients? *Transplantation* 1994; 58:297–300
6. Sanyal AJ: TIPS: The new kid on the block (letter). *N Engl J Med* 1994; 330:165–71
7. Echenagusia AJ, Camunez F, Simo G, Peiro J, Garay MG, Rodriguez Laiz JM, Banares R: Variceal hemorrhage: Efficacy of transjugular intrahepatic portosystemic shunts created with Strecker stents. *Radiology* 1994; 192:235–40
8. Conn HO: Transjugular intrahepatic portal-systemic shunts: The state of the art. *Hepatology* 1993; 17:148–58

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9. Greig JD, Garden OJ, Carter DC: Prophylactic treatment of patients with esophageal varices: Is it ever indicated? *World J Surg* 1994; 18:176-84
10. Kleber G, Sauerbruch T, Ansari H, Paumgartner G: Prediction of variceal hemorrhage in cirrhosis: A prospective follow-up study. *Gastroenterology* 1991; 100:1332-7
11. Grace ND: The side-to-side portocaval shunt revisited. *N Engl J Med* 1994; 330:208-9
12. Korula J, Balart LA, Radvan G, Zweiban BE, Larson AW, Kao HW, Yamada S: A prospective, randomized controlled trial of chronic esophageal variceal sclerotherapy. *Hepatology* 1985; 5:584-9
13. Westaby D, MacDougall BRD, Williams R: Improved survival following injection sclerotherapy for esophageal varices: Final analysis of a controlled trial. *Hepatology* 1985; 5:827-30
14. Burroughs AK, Hamilton G, Phillips A, Mezzanotte G, McIntyre N, Hobbs KEF: A comparison of sclerotherapy with staple transection of the esophagus for the emergency control of bleeding from esophageal varices. *N Engl J Med* 1989; 321:857-62
15. Westaby D, Hayes PC, Gimson AES, Polson RJ, Williams R: Controlled clinical trial of injection sclerotherapy for active variceal bleeding. *Hepatology* 1989; 9:274-7
16. Rikkers LF, Burnett DA, Volentine GD, Buchi KN, Cormier RA: Shunt surgery *versus* endoscopic sclerotherapy for long-term treatment of variceal bleeding. *Ann Surg* 1987; 206:261-71
17. Henderson JM, Kutner MH, Millikan WJ Jr, Galambos JT, Riepe SP, Brooks WS, Bryan C, Warren WD: Endoscopic variceal sclerosis compared with distal splenorenal shunt to prevent recurrent variceal bleeding in cirrhosis. *Ann Int Med* 1990; 112:262-9

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Anaphylactoid Reaction to Protamine Confirmed by Plasma Tryptase in a Diabetic Patient during Open Heart Surgery

Makoto Takenoshita, M.D.,* Masahiro Sugiyama, M.D.,† Yasutsugu Okuno, M.D.,† Yoshimi Inagaki, M.D.,‡
Ikuto Yoshiya, M.D.,§ Yasuhisa Shimazaki, M.D.¶

ANAPHYLACTOID reactions to protamine during open heart surgery in diabetic patients who use insulin preoperatively have been reported.¹ We describe a similar case in which the anaphylactoid reaction to protamine was confirmed by increased plasma tryptase concentration and a positive skin test.

Case Report

A 53-yr-old, 171-cm, 52-kg man was scheduled for a patch closure of an atrial septal defect. No transfusion was expected. Preoperative

hemodynamics included; blood pressure 110/70 mmHg, heart rate 64 beats/min, cardiac index $2.4 \text{ l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$, Qp/Qs 2.8, and ejection fraction 47%. Medical history included diabetes mellitus treated with diet control and oral hypoglycemics until 6 months earlier, when NPH insulin had been added. He claimed no allergy to food or drugs.

Anesthesia was induced with diazepam and fentanyl and maintained with 33% N₂O, fentanyl, and pancuronium bromide before cardiopulmonary bypass. Duration of cardiopulmonary bypass was 55 min, and no blood was transfused. About 15 min after cardiopulmonary bypass, an injection of protamine (protamine sulfate, 10 mg/ml, Shimizu) was started *via* the peripheral vein. Within about 10 min, when 12.5 ml protamine had been injected, the systolic blood pressure decreased from approximately 100 to 40 mmHg. The heart rate increased from 105 to 135 beats/min. Initially, hypovolemia was assumed to be the cause of the hypotension and the remaining fluid in the oxygenator and plasma protein fraction (PPF) was infused rapidly. Within 10 min, systolic blood pressure recovered gradually but remained at 70-80 mmHg, and dopamine ($3 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) was given. Edema on the face, ears, lips, and conjunctiva were noticed. The end-tidal carbon dioxide, sampled from the distal end of the endotracheal tube, decreased from 30-35 to 15-20 mmHg. Peak inspiratory pressure increased, and decreased deflation of the lungs was observed. Within 10 min after administering protamine, SpO₂ gradually decreased from 100% to 84% and recovered to 100% about 40 min after protamine. Arterial blood gas analyses (table 1) demonstrated hypoxia, hypercarbia, and hemoconcentration. Although

* Assistant Professor, Department of Anesthesiology.

† Resident, Department of Anesthesiology.

‡ Instructor, Department of Anesthesiology.

§ Professor and Chairman, Department of Anesthesiology.

¶ Associate Professor, First Department of Surgery.

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