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Case reports

Hemorrhagic Complications of Large Volume Abdominal Paracentesis

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The incidence of hemorrhagic complications from large volume paracentesis in patients with cirrhosis and portal hypertension is unknown. We have reviewed the cases of 179 outpatients undergoing large volume paracentesis at our institution during a 1-yr period. Of these 179 patients, four developed severe hemorrhagic complications requiring hospital admission and blood transfusion. Three of these patients developed intraperitoneal hemorrhage, one of which was localized to the paracentesis puncture site. One patient experienced an abdominal wall hematoma, localized by ultrasound. The symptoms and signs of hemorrhage became evident anywhere from hours up to 1 wk after completion of the paracentesis procedure. The mechanism of delayed hemorrhage is not known but may relate to the rupture of large intra-abdominal venous collaterals in these patients. The literature does not support a correlation between degree of coagulopathy or thrombocytopenia and risk of bleeding in this setting. To promote early detection of this potentially life-threatening complication, a mechanism should exist for close outpatient follow-up of patients after large volume paracentesis.

INTRODUCTION

Large volume paracentesis (more than 4 L), is an accepted method of managing ascites in patients with cirrhosis and portal hypertension, especially in patients whose ascites is refractory to diuretics and dietary salt restriction (1–3). Diagnostic and therapeutic paracenteses have been reported to be safe procedures, with complication rates of less than 1% (4). Major hemorrhagic complications requiring blood transfusion and/or hospitalization are even less common. However, studies assessing the safety of paracentesis have been confined to patients undergoing diagnostic (less than 60 ml) or small volume therapeutic (less than 2 L) paracentesis. The incidence of hemorrhagic complications after large volume paracentesis in patients with cirrhosis and portal hypertension is unknown. We report four cases of major hemorrhagic complications of large volume paracentesis occurring at our institution over a 1-yr period.

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MATERIALS AND METHODS

One hundred seventy-nine outpatients undergoing large volume paracentesis at the University of Michigan Hospital from 1993–94 were reviewed. The indication for large volume paracentesis was for the control of ascites that had been refractory to diuretic therapy and dietary salt restriction. Vacuum-assisted, greater than 4-L paracenteses were performed in our Medical Procedure Unit with a 14- to 18-gauge angiocath needle, by gastroenterology fellows rotating on a monthly basis. Intravenous albumin infusion was administered to all patients undergoing large volume (more than 4 L) paracentesis. Patients undergoing small volume paracentesis (less than 4 L) did not receive intravenous albumin. Of these 179 patients, four developed severe hemorrhagic complications requiring hospital admission and blood transfusion. These cases are reviewed.

Case 1

A 41-yr-old male with end-stage liver disease secondary to alcohol and hepatitis C virus infection (HCV), and awaiting orthotopic liver transplantation (OLT), had been requiring frequent (twice per week) large volume paracentesis for control of ascites. The patient had previously received 80 paracenteses, of 6–12 L, without complication. The patient underwent a routine large volume paracentesis, via mid-line approach, of 12 L of clear ascitic fluid. Immediately after the procedure, the patient developed light-headedness and abdominal discomfort which spontaneously resolved. The patient was discharged home. Four days later, he experienced a syncopal episode prompting admission to the hospital. On admission, his blood pressure was 80/50 mm Hg, with a pulse rate of 120 bpm, with orthostatic changes. His

blood hemoglobin had fallen to 7.1 mg/dl from a level of 11 mg/dl, 1 wk before paracentesis. His prothrombin time was 14 s and platelet count was 80,000/mm^3. Gastrointestinal hemorrhage was excluded with nasogastric lavage and stool Hemoccult testing. Diagnostic paracentesis revealed grossly bloody ascites with a red blood cell count of 21,000/mm^3. The patient received 3 U of packed red blood cells and was later discharged with a serum hemoglobin of 10 mg/dl. Approximately 1 wk later, the patient was readmitted with abdominal pain and confusion. His blood hemoglobin was 7.0 mg/dl. Gastrointestinal hemorrhage was excluded as an apparent source of bleeding by means of nasogastric lavage and stool examination. Diagnostic paracentesis again revealed grossly bloody ascites with a red blood cell count of 311,000/mm^3. A CAT scan of the abdomen did not reveal an intra-abdominal hematoma. The patient received a blood transfusion and had no further evidence of bleeding. One week later the patient underwent transjugular intrahepatic portosystemic shunt for control of ascites. He received an OLT after an additional 2 months.

Case 2

A 39-yr-old male with end-stage liver disease from alcohol, awaiting OLT, required large volume paracenteses 2–3 times per month for 4 months for control of ascites. The patient developed abdominal pain and distention approximately 1 wk after routine large volume paracentesis. He was admitted to an outside hospital where he underwent diagnostic paracentesis. Although all previous paracenteses had yielded clear ascitic fluid, on this occasion there was grossly bloody ascitic fluid with a red blood cell count of 19,000/mm^3. Further laboratory testing revealed that his serum hemoglobin after receiving 2 U of packed red blood cells was 10 mg/dl, platelet count was 113,000/mm^3, and prothrombin time was 14.5 s. The patient's condition remained stable, without further bleeding, and he was subsequently discharged from the hospital after 4 days. Large volume paracentesis was scheduled for the outpatient management of this patient's ascites. No further episodes of bleeding have been noted.

Case 3

A 60-yr-old female with end-stage liver disease due to chronic HCV infection required large volume paracentesis 2–3 times per month for 6 months for control of ascites. Immediately after a routine paracentesis she developed a small abdominal wall hematoma. Approximately 3 wk later, repeat paracentesis of 6 L revealed clear fluid. Shortly thereafter, the patient experienced left lower quadrant abdominal pain at the site of paracentesis, associated with a drop in blood hemoglobin from 10 mg/dl to 7 mg/dl. Other data included a platelet count of 81,000/mm^3 and prothrombin time of 15.2 s. An abdominal sonogram demonstrated a 12 × 5 × 9 cm abdominal wall hematoma. Her serum hemoglobin rose to 9 mg/dl after transfusion of 2 U of packed red blood cells. The patient was discharged from the hospital 3 days later. After an interval of 2 wk, serial large volume paracenteses were again performed for control of ascites. No further episodes of bleeding have occurred.

Case 4

A 55-yr-old male with end-stage liver disease due to alcohol-related liver injury and chronic HCV infection required large volume paracenteses every month for 10 months for control of ascites. Four hours after a routine 10-L paracentesis, the patient experienced severe abdominal pain followed by hypotension and syncope. Gastrointestinal hemorrhage was excluded with nasogastric lavage and frequent stool Hemoccult testing. Diagnostic paracentesis revealed grossly bloody ascites with a red blood cell count of 268,000/mm^3. His blood hemoglobin had fallen to 6.0 mg/dl from a level of 13 mg/dl before paracentesis. His platelet count was 146,000/mm^3 and his prothrombin time was 17 s. The patient was transfused with 4 U of packed red blood cells. CT scan of his abdomen revealed a large hematoma within the peritoneal cavity in the vicinity of the paracentesis site. The patient's blood hemoglobin remained stable without further blood transfusions. He was eventually discharged from the hospital after large volume paracenteses were begun again for control of his ascites. No further episodes of bleeding were noted.

DISCUSSION

We observed four severe hemorrhagic complications from 179 large volume (>4 L) paracenteses performed at our center over a 1-yr period. The symptoms and signs of hemorrhage became evident anywhere from hours up to 1 wk after completion of the paracentesis procedure. Three patients developed intraperitoneal hemorrhage, of which one could be localized to the paracentesis puncture site. One patient experienced an abdominal wall hematoma documented by ultrasound. Bleeding as a direct result of needle trauma (abdominal wall hematoma, intraperitoneal hematoma at the puncture site) tended to occur early post-procedure. The mechanism for delayed hemorrhage into the peritoneal cavity is unknown.

Studies reporting the apparent safety of paracentesis have not addressed the incidence of hemorrhagic complications of large volume paracentesis (4). Hemorrhage from abdominal paracentesis occurs either during the procedure or begins some time afterward. Bleeding during the procedure has been attributed to a variety of factors, including puncture of superficial epigastric vessels with resultant abdominal wall hematoma, as well as puncture of intra-abdominal venous collaterals, including paraumbilical veins (5, 6). Intra-abdominal hemorrhage occurring post-procedure is a rare complication. Mallory and Schaefer (7) report four cases of severe intraperitoneal hemorrhage following diagnostic paracentesis. Onset of hemorrhage, as detected by clinical symptoms, varied from 6 to 48 h after the procedure and could not be directly attributed to coagulation or clotting

defects. Liebowitz (8) reports two cases of intra-abdominal hemorrhage occurring post-paracentesis. One case involved puncture of venous collateral vessels, and one involved trauma to the serosa overlying the small intestine. The literature does not suggest that the mechanisms for immediate or delayed hemorrhage related to paracentesis are necessarily the same.

In our cases, it appeared that bleeding usually began after the ascitic tap had been completed. This was suggested by the presence of clear fluid throughout the paracentesis. This phenomenon raises interesting questions as to possible alternative mechanisms of hemorrhage. Liebowitz (9) has reported six cases of esophageal varix rupture occurring from 9 to 72 h after large volume paracentesis (volume not specified). A postulated mechanism was rapid decompression of the splanchnic circulation due to release of intra-abdominal pressure from drainage of the ascitic fluid. This could result in a marked increase in portal pressure and portosystemic blood flow through esophagogastric collaterals with secondary esophageal varix rupture and subsequent hemorrhage. Although this mechanism would be difficult to substantiate, it is not without precedent. An analogous system is known to exist in patients with chronic urinary retention, undergoing rapid decompression of the urinary bladder via catheterization. Hematuria occurs in approximately 16% of patients undergoing rapid bladder evacuation, most occurring within the first 24 h (10).

Three of our four patients developed intraperitoneal hemorrhage from several hours to 1 wk after large volume paracentesis, without evidence of gastrointestinal hemorrhage. Multiple reports in the literature demonstrate the existence of large intra-abdominal venous collaterals, including recanalized umbilical vein, in patients with portal hypertension and ascites (11–13). It is not unreasonable to speculate that rapid decompression of splanchnic circulation could occur after large volume paracentesis and result in transient increased pressure in these venous collaterals with secondary rupture and hemorrhage. Regardless of the exact mechanism, serious hemorrhage after large volume paracentesis does occur. Perhaps more important, intra-abdominal hemorrhage can occur up to 1 wk after large volume paracentesis. To avoid this complication, one should consider lower volume and slower drainage of ascitic fluid in patients with chronic refractory ascites. The literature does not support a correlation between degree of coagulopathy and risk of bleeding in this setting, but in patients with markedly elevated prothrombin time and severe thrombocytopenia, correction of these parameters may be prudent. In addition, a mechanism should exist for close outpatient follow-up of patients after the procedure. For many patients, frequent large volume paracentesis has become the only method available for control of massive ascites. Until other nonsurgical methods for the control of diuretic refractory ascites are developed and approved, it is important to recognize the existence and timing of this serious complication of large volume paracentesis.

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