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Case report

Flood syndrome managed by partial splenic embolization and percutaneous peritoneal drainage

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ABSTRACT

Flood syndrome is a rare complication of cirrhosis of liver accompanied by ascites and a sudden rupture of umbilical hernia causing drainage of ascitic fluid from abdominal cavity. We report management of a case of Flood syndrome which was caused by rupture of incisional hernia. The clinical picture was similar to well described and widely accepted Flood syndrome. A 70-year-old female with decompensated hepatitis C cirrhosis was transported to the emergency department with a sudden drainage of ascitic fluid after sudden dehiscence of pre-existing incisional hernia and diffuse abdominal tenderness. Initially, she was managed by applying ostomy bag and diuretics to reduce the ascites. On 8th day of admission, a 16 Fr. drain was percutaneously placed in the left lower abdominal quadrant to divert the fluid from the abdominal wall defect. On 13th day, 80% partial splenic embolization (PSE) was attempted to control portal hypertension to reduce the ascites volume. After PSE, the hepatic venous pressure gradient reduced from 28 to 21cm H₂O. The peritoneal drain was removed on 16th day and she was discharged on 22nd day. We conclude that PSE and temporary percutaneous peritoneal drainage are useful option to manage Flood syndrome.

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Figure 1 – Photograph of the abdomen showing an incisional hernia with small ulceration (arrow) of the overlying skin in the lower abdomen. The skin is discolored and irregular. The form of the navel was normal (arrow head).

Introduction

Flood syndrome is a sudden drainage of ascitic fluid from abdominal cavity that accompanies spontaneous rupture of an umbilical hernia [1–3]. It was reported by Frank B Flood [1]. It is a rare and serious complication of long-standing massive ascites in end-stage cirrhotic patients. The optimal management of Flood syndrome has yet to be established [4–10]. We encountered a case of sudden drainage of ascitic fluid associated with spontaneous dehiscence of incisional hernia of abdominal wall. As the clinical picture was similar to well described and widely accepted Flood syndrome, we considered and managed this case as Flood syndrome.

Case report

A 70-year-old female was brought in by ambulance for a sudden drainage of ascitic fluid from abdominal cavity accompanied by a dehiscence of pre-existing abdominal wall incisional hernia associated with diffuse abdominal pain, tenderness, and fever. She gave a history of abdominal distension since 7 months. Past medical history showed that 40 years ago she had transabdominal oophorectomy and received blood transfusion. She reported presence of abdominal hernia after the oophorectomy. She was not followed up for the hernia.

On admission, the patient had a heart rate of 118 per minute, a blood pressure of 122/85 mm Hg, and a respiratory rate of 20 per minute. Her body temperature was 38.8 °C. Her consciousness was lucid. She was jaundiced. A tender incisional hernia with a small ulceration of overlying discolored

skin at the lower abdomen, draining light yellow-colored ascitic fluid was noted (Fig. 1).

Laboratory studies revealed hemoglobin of 10.2 g/dL normal range, (13.5–17.4); total leukocyte count of 3050 / μ L (3500–8000/ μ L); total platelet count of 2.9×10^4 / μ L (12.3 – 33.1×10^4 / μ L); total bilirubin 7.1 mg/dL (0.3–1.3 mg/dL); albumin 2.2 g/dL (3.8–5.0 g/dL); aspartate transaminase 44 U/L (10–32 U/L); alanine transaminase 26 U/L (5–27 U/L); prothrombin time 41.6% (70–130%); international normalized ratio (INR) of 1.8; C-reactive protein 3.51 mg/dL (<0.16 mg/dL); procalcitonin 0.60 ng/mL (<0.3 ng/mL); α -Fetoprotein (AFP) of 98.9 ng/mL (0–20); protein induced by vitamin K absence or antagonist-II (PIVKA-II) of 3081 mAU/mL (<40 mAU/mL). Her serum ammonia was at 35 μ g/dL (normal range, 12–66 μ g/dL). Hepatitis B surface antigen was negative, but hepatitis C virus antibody was positive. The Child-Pugh score was 12 (class C) and model for end-stage liver disease score was 20. These data indicated hepatitis C decompensated cirrhosis with malignancy.

Abdominal ultrasonography and contrast-enhanced CT showed presence of incisional hernia, dehiscence, ascites, splenomegaly and 3 cm sized hepatocellular carcinoma at segment 3 and segment 6 of the liver (Fig. 2a and b). 3D-CT demonstrated large splenorenal shunt. The spleen volume was 812 mL, the liver volume was 570 mL, and spleen/liver volume ratio [11] was 1.43 (Fig. 3a). Endoscopy confirmed absence of gastroesophageal varices.

Blood and ascitic fluid cultures on 1st day were positive for streptococcus dysgalactiae which was sensitive to benzylpenicillin potassium and ampicillin sodium. A diagnosis of Flood syndrome due to incisional hernia dehiscence was made. This was complicated by bacteremia and bacterial peritonitis. She was treated with intravenous benzylpenicillin potassium for the first 7 days. The patient developed hyperkalemia, IV antibiotic was switched to ampicillin sodium for the next 14 days. Initially, an ostomy bag was placed over the hernia to collect ascitic fluid leakage. She was treated with furosemide of 80 mg, spironolactone of 100 mg, and tolvaptan of 7.5 mg a day. Despite all these efforts 2 to 3 liters of ascitic fluid was drained each day.

Repeat blood culture on 3rd day was negative for bacteria. On 8th day, a 16 Fr. drain was placed in the left lower abdominal quadrant to divert the fluid from the abdominal wall defect (Fig. 4). The retention rate of indocyanine green at 15 minutes (ICG15) on 9th day was 77% (<10%). On 13th day, hepatic venous cannulation via the right arm was performed and hepatic venous pressure gradient (HVPG) was evaluated (Fig. 5). HVPG was 28.0 cm H₂O. Venous phase of superior mesenteric arteriography revealed large splenorenal shunt (Fig. 6). PSE using gelatin sponge and microcoils was performed to control portal hypertension to reduce the ascites volume (Fig. 7a and b). The HVPG reduced to 21.0 cm H₂O immediately after PSE. 3D-CT after PSE on 16th day revealed that the viable spleen volume decreased to 114 mL and corrected spleen/liver volume ratio was 0.22 (Fig. 3b).

Ascitic fluid cultures on 9th and 16th days were negative for bacteria. The patient slowly recovered, and the peritoneal drain was removed on 16th day. She was discharged on 22nd day.

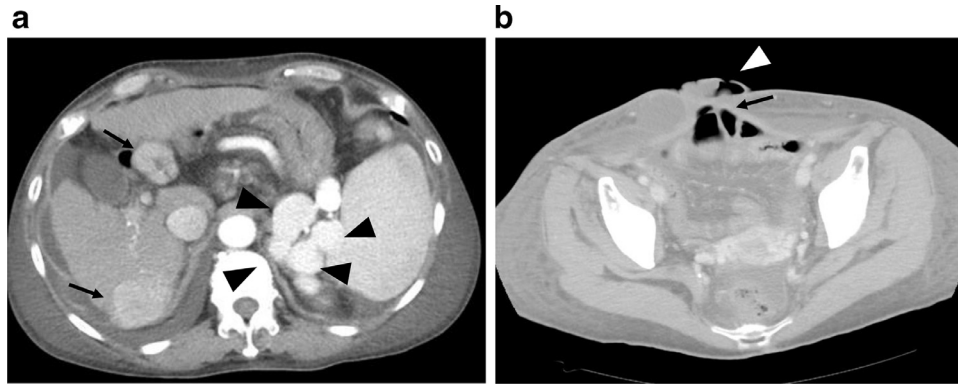


Figure 2 – (a) Contrast-enhanced upper abdominal CT showing hepatocellular carcinoma (arrow), splenomegaly, and large splenorenal shunt (arrow head). (b) Contrast-enhanced lower abdominal CT showing incisional hernia (arrow) and dehiscence of abdominal wall (arrow head).

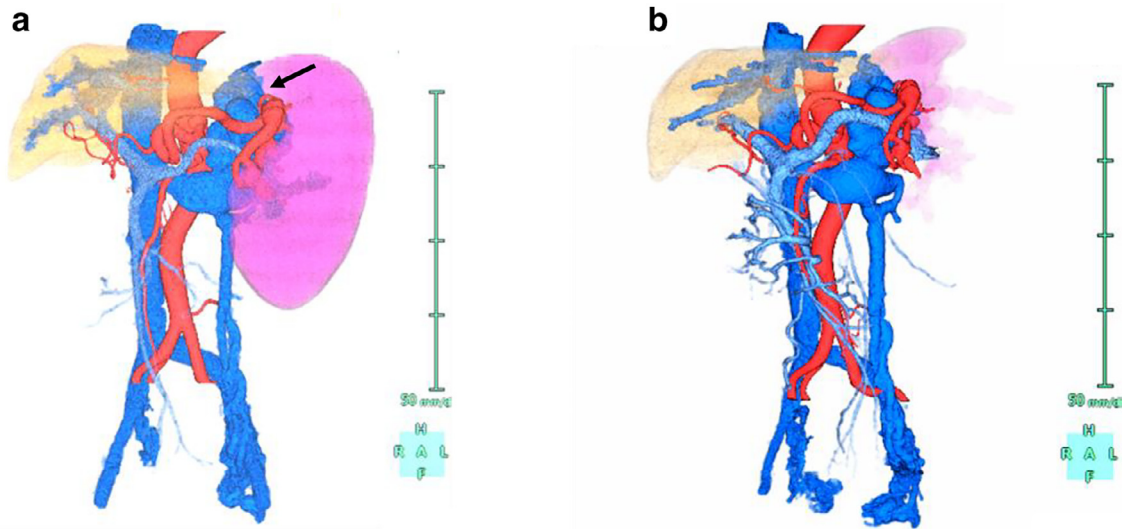


Figure 3 – (a) 3D-CT before PSE showing large splenorenal shunt (arrow). The spleen/liver volume ratio was 1.43. (b) 3D-CT after PSE revealed that the corrected spleen/liver volume ratio was 0.22.

Discussion

Flood syndrome in patients with ascites associated with end-stage cirrhosis is a serious complication and may be fatal [1–3]. Optimal management of Flood syndrome has not been established [4–10]. We have reported usefulness of PSE in the management of portal hypertension [12–13]. Use of PSE in the management of Flood syndrome has not been reported. This report describes a case of Flood syndrome and was managed by PSE and temporary percutaneous peritoneal drainage.

Original Flood syndrome was described as sudden drainage of ascitic fluid from abdominal cavity that accompanied spontaneous rupture of an umbilical hernia. It was reported in 1961 by Flood FB [1]. He reported 5 cases who had died on 3, 9, 13, 27, and 27 days after spontaneous rupture of umbilical hernia. He reported complications such as renal insufficiency in 5, peritonitis in 3, hepatic failure in 3, hypoproteinemia in 2,

hyponatremia in 4, and hypotension in 3. Although the clinical picture was similar as that with umbilical hernia rupture, our case was associated with spontaneous dehiscence of incisional hernia of abdominal wall.

About 20% of patients with chronic liver cirrhosis develop umbilical hernias [14–15]. Portal hypertension is one of the most common cause of ascites in liver cirrhosis. Flood FB [1] reported 5% (5/101) of patients with cirrhosis and ascites were admitted to his hospital because of umbilical hernia rupture in a period of two and a half years.

Umbilical hernia complications such as strangulation, incarceration, and rupture in patients with cirrhosis carry high mortality rate. Mortality rate of 60%–80% after supportive care and 6%–20% after surgical management has been reported [4–6]. Managements of these patients have been controversial. Previously, umbilical hernia repair, transjugular intrahepatic portosystemic shunt (TIPS), peritoneovenous shunt, and sur-

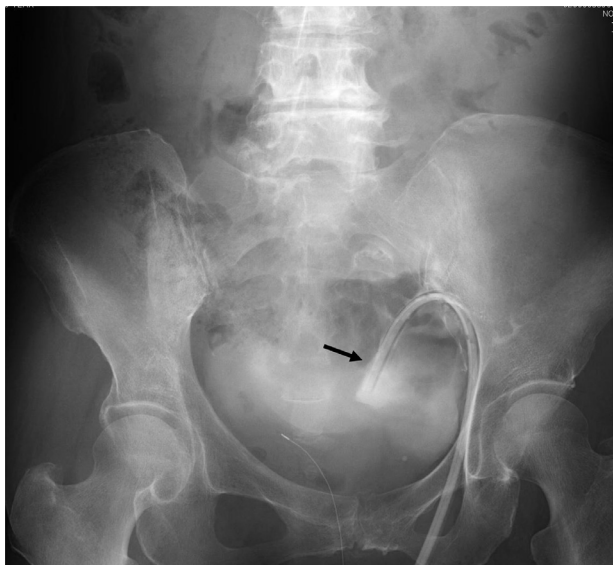


Figure 4 – Peritoneal drainage tube was percutaneously inserted into the left lower quadrant (arrow).



Figure 6 – Venous phase of superior mesenteric arteriography revealed large splenorenal shunt (arrow).

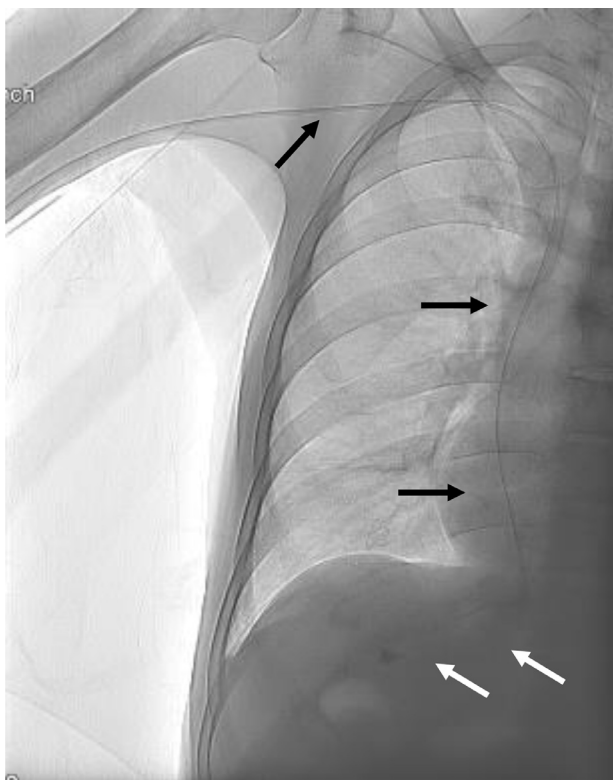


Figure 5 – Hepatic venous cannulation was performed via the right arm (arrow).

gical portosystemic shunt have been reported as treatments for Flood syndrome [7].

Surgical portosystemic shunts are no longer performed nowadays because of high mortality (10%), risk of encephalopathy (50%), and degradation of hepatocellular function [8]. Liver transplantation is the ideal treatment because it

treats both ascites and its cause. However, our case was elderly and complicated with hepatocellular carcinoma. O'Connor M [9] reported good results with simultaneous umbilical herniorrhaphy and peritoneovenous shunt in patient with leaking ascites. In our case, peritoneovenous shunt was contraindicated because of bacteremia and bacterial peritonitis.

Elective herniorrhaphy is recommended after the patient is stabilized using optimal medical management [14]. TIPS is the most widely used method. However, TIPS has wide variety of complications and risks such as hemorrhage, encephalopathy, TIPS dysfunction, liver failure, and incarceration of hernia. Its morbidity risk can be as high as 20% [10]. Telem DA [6] reported 4 cases of preoperative TIPS in conjunction with semi-elective herniorrhaphy. They reported 1 of 4 had worsened encephalopathy and 1 had liver decompensation needing liver transplantation.

Because large splenorenal shunt existed in our patient, TIPS was less favorable. We applied PSE instead of TIPS. We have reported that PSE can reduce the splenic venous blood flow volume and portal venous pressure [12]. In liver cirrhosis, spleen/liver volume ratio increases [11]. In this case, PSE could reduce HVPG and spleen/liver volume ratio, which was effective in the management of ascites.

While control of bleeding from esophagogastric varices became easy by endoscopic treatment such as sclerotherapy or variceal ligation, splenomegaly is often left without being treated. We believe that the management of splenomegaly is important in the treatment of portal hypertension. Surgical techniques such as Sugiura procedure [16] and Hassab's operation [17] are some of the accepted surgical treatments for splenomegaly. Here, we want to propose a new concept of "Splanchnic Caput Medusae" in which enlarged spleen is her face and portal collateral pathways are her snake hairs. This concept is completely different from an old concept of Caput Medusae, in which abdomen is her face and perinavel dilated subcutaneous veins are her snake hairs [18]. We have to treat

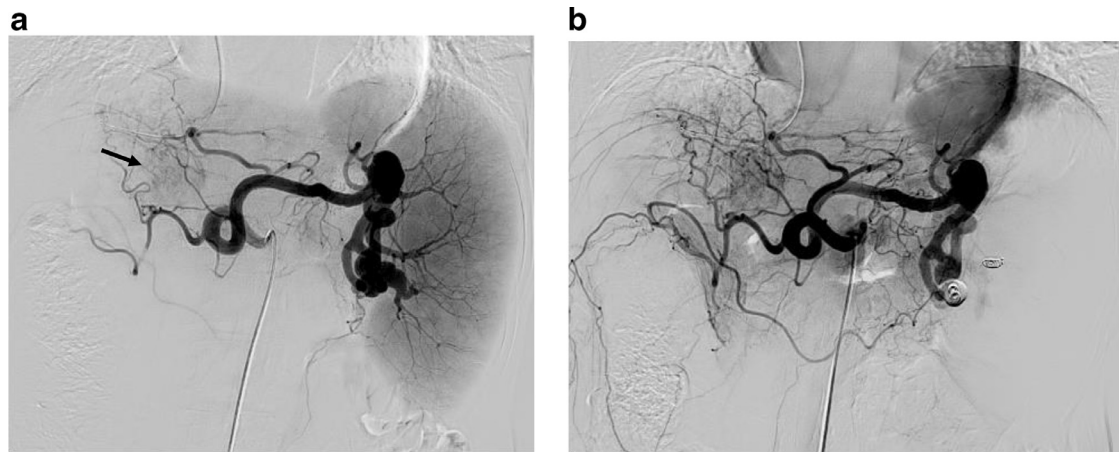


Figure 7 – (a) Celiac arteriography before PSE showing splenomegaly and hepatocellular carcinoma (arrow) in the left hepatic lobe. (b) Celiac arteriography after PSE showing 80% embolization of spleen.

splenomegaly and portal collaterals in a good balance for portal hypertension. In the new concept of “Splanchnic Caput Medusae”, PSE is considered as the treatment of Medusae’s face.

We conclude that PSE and temporary percutaneous peritoneal drainage are useful option to manage Flood syndrome.

Patient consent statement

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Declaration of Competing Interest

The authors declare no conflicts of interest associated with this manuscript.

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