Spontaneous rupture of omental varices : an uncommon cause of hypovolemic shock in cirrhosis

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Abstract

In cirrhotic patients, esophageal and esophagogastric varices are the most common sites of bleeding, often responsible for hypovolemic shock. Hepatocellular carcinoma, blunt abdominal trauma and postprocedural complications are classical causes of hemoperitoneum in hepatic cirrhosis. Rupture of omental varices is another and rarely reported cause of shock in cirrhosis. We report a case of hypovolemic shock caused by ruptured omental varices. Selective review of literature regarding presentation, diagnosis and management of ruptured intraabdominal varices is also part of presentation. (Acta gastroenterol. belg., **2004**, 67, **351-354**).

Key words : hemoperitoneum, omental varices, alcoholic cirrhosis and portal hypertension.

Introduction

Intraabdominal hemorrhage from ruptured varices is an unusual though severe complication of portal hypertension. The development of anastomoses between the portal and systemic circulation occurs at sites where veins, draining into the two systems, are juxtaposed. Such communications exist not only through oesophagus, but can be located in all the digestive tract. Occasionally, they affect extradigestive sites. Spontaneous rupture of omental varices leading to hemoperitoneum is a severe and life- threatening condition with a high mortality rate. Diagnosis and management of ectopic varices are specific and differ from that of esophagogastric varices. In this report, we present a case of hemoperitoneum caused by spontaneous rupture of omental varices and we present a literature review on the other reported cases of ruptured intraperitoneal varices.

Case report

On December 12th 1999, a 49-year-old man with a long history of alcoholic abuse was admitted because of severe hypotension (systolic blood pressure of 85 mm Hg), major confusion and peripheral cyanosis. Rapidly, he developed diffuse abdominal pain without signs of digestive bleeding . He had no history of abdominal surgery or trauma. Initial laboratory tests were as follows : Hb : 5,4 g/dL (normal range, 14-18 g/dL), platelet count : 63 $\times$ 10^9/L (normal range, 150-400 $\times$ 10^9/L), white blood cell count :12.620 $\times$ 10^9/L (normal range, 4-10 $\times$ 10^9/L) and INR : 3,42 (normal range, 1-1,3) and bilirubin : 5,3 mg/dl (normal value, 0-1,2 mg /dL). After hemodynamic stabilization, an oesophagogastroduodenoscopy was performed and showed grade 2 esophageal varices and ulcers in the lower eosophagus. Computed tomography revealed splenic varices and perisplenic ascites (Fig. 1).

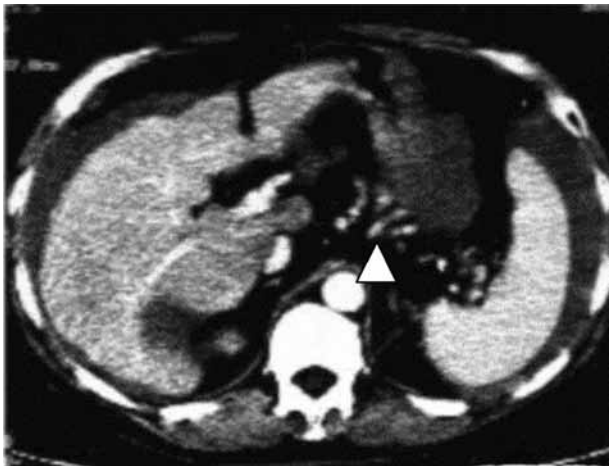

Fig. 1. — Perisplenic ascites and perisplenic dilated varices (white arrow).

Thereafter, hypotension recurred together with increasing abdominal girth. Haemoperitoneum was diagnosed by paracentesis (hematocrit = 22%). During laparotomy, two liters of blood were removed from the peritoneal cavity. The omentum was very fatty with numerous dilated epiploic varices. One of them was bleeding and was ligated allowing to stop the bleeding. The liver was micronodular, consistent with cirrhosis. Other varices were present near the stomach and the oesophagus. There was no ruptured organ.

During the postoperative period, the patient developed transient hepatic failure and needed ventilatory assistance during 72 hours. The patient left the hospital 12 days later and was treated with isoproterenol 40 mg twice a day.

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Table 1. — **Main characteristics of patients with "hemoperitoneum due to intra-abdominal varices" reported in the literature**

N, (ref), age/sex	Cause of port hypertension	Site of bleeding	Therapy	Outcome
1. (4) 74/M	AC	Anterior to the right kidney	None	Died
2. (5) 52/M	C	Pouch of Douglas	None	Died
3. (6) NA/M	AC	Veins of Retzius at cecum	Ligation and porto-caval shunt	Died
4. (7) 76/M	C	Subdiaphragmatic veins of Sappey	None	Died
5. (8) 38/M	AC	Root of mesentary	Ligation	Died
6. (9) 38/M	C	Periumbilical	Ligation	Survived
7. (10) 28/M	AC	Veins of Retzius at hepatic flexure	Ligation and portocaval shunt	Died
8. (11) 64/M	AC	Veins of colo-parietal ligament	Laparotomy	Survived
9. (12) 60/M	AC	Region of spleen	Intraarterial vasopressin	Died
10. (13) 39/M	AC	Veins of Retzius at right colic gutter	Ligation	Died
11. (14) 49/M	AC	Retroperitoneal varix in sub-hepatic space	Ligation	Survived
12. (15) 58/M	AC	Gastrohepatic ligament	Ligation	Died
13. (15) 50/M	AC	Veins of Retzius at ascending colon	Ligation	Survived
14. (15) 25/M	AC	Pancreatic veins	Ligation	Died
15. (16) 73/M	AC	Veins in gastrohepatic omentum and perisplenic space	Mesocaval shunt	Died
16. (17) 58/F	AC	Veins in round ligament	Ligation	Died
17. (18) 49/M	AC	Varices in superior mesenteric venous system	None	Died
18. (19) 50/M	C	Periumbilical varices	Ligation	Died
19. (20) 67/F	PNC	Omental varices	Ligation	Died
20. (21) 21/M	NRH	Umbilical vein	Ligation and portocaval shunt	Survived
21. (22) 21/M	NRH	Veins in surface of liver, spleen and mesentery	None	Died
22. (23) 45/M	AC	Retroperitoneal varix near right colon	Ligation	Survived
23. (24) 29/M	EPVO	Ectopic vessel along the splenorenal ligament	Ligation	Survived
24. (25) 58/M	AC	Retroperitoneal varix	Ligation	Survived
25. (26) 68/M	AC	Gastrosplenic varices	Ligation	Died
26. (27) 41/M	AC	Varices in the gallbladder	None	Died

Abbreviations : N, number ; (Ref), reference ; M, male ; F, female ; AC, alcoholic cirrhosis ; NRH, nodular regenerative hyperplasia ; EPVO, extra-hepatic portal vein obstruction. NA, not available ; PNC : postnecrosis cirrhosis.

During the next 24 months, the patient was followed-up as an outpatient, he had stopped drinking and liver tests had improved.

Discussion

In patients with ascites, haemoperitoneum can develop spontaneously (from hepatocellular or ovarian carcinoma or from ruptured intraperitoneal varix in the setting of portal hypertension) or can follow abdominal trauma and diagnostic or therapeutic procedures (1). Ectopic varices are an unusual cause of gastrointestinal haemorrage in cirrhotic patients, but account for up to 5% of all variceal bleeding (2). Localisation include stoma (26%), duodenum (17%), jejunum or ileum (17%), colon (14%), rectum (8%) and the peritoneum (9%) (3).

In the literature, 26 other reported cases of spontaneous bleeding from intraabdominal varices were identified (4-27) and are summarized in Table 1. Including our patient, there were 25 men and 2 women with a mean age of 49.2 years. Intrahepatic portal hypertension was present in 26 of the 27 patients. Only one of these patients had undergone previous abdominal surgery (28). A few reports (29-30) have suggested that previous abdominal operations or inflammatory abdominal diseases may be predisposing factors for the development of mesenteric varices by creating new portosystemic communications in peritoneal adhesions. Wall tension is thought to be the major determinant of hemorrhage from varices (31). Tension in the varix wall is proportional to transmural pressure across the vessel wall and the radius of the vessel. Therefore, the major determinants of rupture of ectopic varices are likely to be vessel size and portal pressure.

Diagnosis of rupture of intraperitoneal varices must be evoked when sudden abdominal pain, abdominal distension, tachycardia and arterial hypotension occur. A characteristic clinical feature is the rapid enlargement of abdominal girth, in contrast to the relatively slow accumulation of nonhaemorrhagic ascites. Diagnosis is confirmed by a heavyly blood-stained ascitic fluid tap (the haematocrit is typically above 5%) (14).

Angiography was performed in 5 patients (12,13,14, 16,21). The bleeding site was localized (in the spleen) in only one case (12). The diagnosis is further suggested by computed tomography scan showingof retroperitoneal varices and intra- abdominal haemorrhage. Only laparotomy can confirm the rupture of the abdominal varix and allows direct control of the bleeding by varix ligation.

Rupture of intraperitoneal varices has a poor prognosis : 18 patients died (mortality rate 67 %). Causes of death were uncontrolled bleeding and/or hepatic failure (14). In 11 of the patients, death occurred despite direct ligation of bleeding varices. There was no survivor among the 6 patients treated without surgery. Only 9 patients survived and 8 of them had been treated by direct ligation of bleeding varices.

Management of bleeding intra-abdominal varices is difficult since there have been no randomised trials related to the infrequency of this condition. Therefore, management depends on local expertise and the cause of portal hypertension. A systematic approach for any patient presenting with bleeding from possible ectopic varices has been proposed (32).

The transhepatic approach is used to catheterize the portal vein and to identify the ectopic varices and their drainage into the systemic venous system. Management of this situation is made more difficult by the relative contraindication to transhepatic obliteration of the varices caused by the accompanying ascites. The transjugular, intrahepatic approach to the portal system is another possibility for angiographic embolization. Angiographic embolization of varices should be proposed when the portal vein is patent with the goal to occlude the feeding vein to the ectopic varices. When embolization fails to control bleeding, options include either creating a transjugular intrahepatic portosystemic stent shunt (TIPS) or proceeding with surgery. The choice of TIPS or surgery depends on the underlying liver function. Surgical ligation followed by shunt should be attempted in patients of Child- Pugh A and in patients with an extrahepatic portal vein thrombosis. TIPS placement at the time of variceal obliteration may be considered in Child-Pugh B or C but it should probably be defered unless the initial embolization therapy fails to arrest haemorrhage.

Emergency surgery may be performed in these patients with the aim of ligating the bleeding varix, but this carries a high mortality. It appears that in patients with intrahepatic portal hypertension, TIPS offers a highly effective means of controlling haemorrhage in the short term. Data about the efficacy of TIPS implantation in ectopic varices are relatively rare. TIPS can be a uniquely effective method for the treatment of peristomal or rectal varices regardless of Child's class (33). Emergency surgery (ligation of the varix) should be performed in patients with extrahepatic portal vein thrombosis.

There are no data available regarding the use of beta adrenergic-blockers or nitrates in the long-term management of patients with ectopic varices. Due to the well-known efficacy of beta-blocker therapy for secondary prophylaxis of bleeding from oesophagogastric varices, these agents should also be recommended in the prevention intra-abdominal variceal bleeding (34).

In summary, clinicians caring for patients with cirrhosis and portal hypertension should be aware that intra-abdominal bleeding is a potential complication of portal hypertension, needing a multidisciplinary approach between hepatologists, endoscopists, interventional radiologists and surgeons. This rare entity has a high short-term mortality and needs rapid diagnosis by abdominal paracentesis. Embolization and surgical ligation of bleeding varices are the only treatments improving survival. Surgical shunt or TIPS couldbe considered for selected patients for whom embolization fails to control bleeding.

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