Intraperitoneal haemorrhage from anterior abdominal wall varices

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Summary: Patients with oesophageal varices frequently present with gastrointestinal haemorrhage but bleeding from varices at other sites is rare. We present a patient with hepatitis C-induced cirrhosis and partial portal vein occlusion who developed spontaneous haemorrhage from anterior abdominal wall varices into the rectus abdominus muscle and peritoneal cavity.

Introduction

Portal hypertension is most often seen in patients with chronic liver disease but may also occur in those with portal vein occlusion. Thrombosis of the portal vein is recognized in both cirrhotic patients, those with previous abdominal surgery, sepsis, neoplasia, myeloproliferative disorders, protein C or protein S deficiency. Oesophageal varices develop when the portal pressure is maintained above 12 mmHg. Patients with oesophageal varices often present with severe haematemesis. The mortality associated with the first haemorrhage is approximately 50% but varies with the severity of any underlying liver disease. For this reason the prognosis is better in patients with prehepatic portal hypertension.

Extraoesophageal varices are recognized in intrahepatic portal hypertension but they are rare and seldom bleed. In contrast in prehepatic portal hypertension they are both more common and bleed more frequently. This may in part be due to extraoesophageal varices being of a greater size, as rupture is more likely when the wall tension is high. We describe a patient with both cirrhosis and partial portal vein occlusion in whom bleeding occurred from large anterior abdominal wall varices.

Case report

A 48 year old Egyptian male professional presented with collapse after experiencing severe central abdominal pain of sudden onset. Over the preceding 3 months he had noticed abdominal and ankle swelling. Four years earlier chronic active hepatitis had been diagnosed in Egypt and treated with prednisolone and azathioprine.

Examination revealed a well nourished, jaundiced man with stigmata of chronic liver disease who was anaemic and shocked with a pulse of 100 mm and blood pressure 60/20 mmHg. The abdomen was distended, diffusely tender and there was splenomegaly. Rectal examination was normal.

Investigation revealed a haemoglobin of 4.5 g/dl, platelets 129 x 10^9/l, prothrombin time 39 seconds (control <16.5 seconds). Serological tests were positive for anti-hepatitis B core antigen and hepatitis C (p22). Autoantibody tests were negative as were the schistosomal serology and hepatitis B surface antigen.

Abdominal ultrasonography demonstrated ascites, a small irregular liver, splenomegaly and massively dilated umbilical and anterior abdominal wall veins. An ultrasound-guided diagnostic paracentesis was performed at a site distant from the vessels. This revealed uniformly blood-stained fluid. Contrast enhanced abdominal computed tomographic (CT) scan confirmed the ultrasound findings, demonstrated oesophageal varices and showed the left rectus abdominus muscle to be distended (Figure 1). Calcification was noted in the region of the portal vein. At angiography the superior mesenteric vein was narrowed near the head of the pancreas where calcification was again seen. The portal vein filled poorly and a large varix drained into the umbilical vein which was grossly dilated. The appearances suggested haemorrhage...
into the left rectus abdominus muscle with subsequent bleeding into the peritoneal cavity as a consequence of portal hypertension in a patient with cirrhosis and partial portal vein occlusion.

Ten days after admission the patient suffered a further episode of shock with abdominal distension. A repeat CT scan showed an increase in size of the left rectus abdominus muscle. After resuscitation a laparotomy was performed. The peritoneal cavity contained 5–6 litres of altered blood, the spleen was massively enlarged and the liver shrunken; there was no evidence of splenic trauma. Numerous varices were seen in both the intraperitoneal and extraperitoneal spaces associated with the ligamentum teres. The spontaneous haemorrhage appeared to have occurred from one of these but the exact site could not be localized. The abdominal paracentesis had been performed at a point 7–8 cm caudal to any varices. The anterior abdominal wall varices were under run and a wedge liver biopsy performed. This showed cirrhosis with areas of inflammation and piecemeal necrosis, compatible with the consequences of hepatitis C infection.

The patient made a good initial postoperative recovery but on day 4 developed hypotension and hepatic failure. Attempts at resuscitation were unsuccessful. Postmortem examination revealed ruptured oesophageal varices with blood in the stomach and small bowel. There was no evidence of recent intra-abdominal haemorrhage and the anterior abdominal wall varices remained collapsed. The portal vein was partially occluded with calcified thrombus.

Discussion

The patient’s presentation was due to spontaneous rupture of abdominal wall varices.

The success of the operation, confirmed at postmortem, will have increased the pressure and wall tension inside the oesophageal varices. The association between variceal wall tension and risk of haemorrhage is recognized and thus in retrospect ligation of the anterior abdominal vessels may have precipitated oesophageal variceal haemorrhage, which led to the patient’s death.

An alternative treatment would have been to decompress the portal circulation either surgically or by a transjugular intrahepatic portosystemic stent-shunt (TIPSS). Surgical shunts are either selective, nonselective or partial. A selective shunt such as the distal splenorenal anastomosis maintains the pressure in the mesenteric and portal system whilst decompressing the oesophagogastric bed. This is successful therapy for oesophageal variceal bleeding but would not have been appropriate in this case. Nonselective shunts direct the portal circulation into the inferior vena cava via end-to-side, side-to-side or prosthetic interposition anastomoses. Although effective in preventing bleeding, because the anastomoses are wide and allow large volume flow, they are associated with a high incidence of chronic hepatic encephalopathy. Our patient was a professional man who would have been incapacitated by this complication. He had developed severe encephalopathy after both bleeding episodes and we believed the likelihood of encephalopathy was too great to perform a nonselective shunt.

Partial shunts are designed to avoid chronic encephalopathy. They have smaller anastomotic windows, do not fully reduce the portal-systemic gradient and may in part preserve portal flow. The anastomosis may be made directly between the portal vein and the inferior vena cava or via a prosthetic insert of fixed diameter. The experience with partial shunts is limited. Neither the adequacy of pressure reduction nor the duration of patency are yet well established. Thrombotic occlusion of nonselective shunts is a well-recognized problem and the same may be true of partial shunts. Because of these uncertainties we rejected this surgical option.

A TIPSS is introduced by radiologically guiding a wire and subsequently a stent between a branch of the hepatic vein, through the parenchyma of the liver to a radicle of the portal vein. Presumably because the decompression is not complete, encephalopathy has not been a major complication in the limited number of reports. The technique does not need surgical intervention and repeat balloon dilatation of stents can be performed if the portal pressure is inadequately reduced. TIPSS
may become the treatment of choice for portal hypertension when the portal vein is patent. Portal vein occlusion does not preclude the technique but does not increase the difficulty of deployment. As the imaging in our patient suggested portal vein calcification and partial portal vein occlusion we were deterred from attempting the procedure.

The development of varices at points of portal-systemic anastomosis is well recognized. Whilst small intestinal, colonic, and rectal varices are recognized to bleed there are few reports of extra-intestinal lesions bleeding. Haemorrhage from round ligament, adhesion related and mesenteric varices has been reported and there are two previous reports of rupture of periumbilical varices. The presence of partial portal vein occlusion in our patient is of interest as Lebrec has suggested that whilst only 1–3% of cirrhotic patients bleed from ectopic varices the incidence may be ten times this in patients with extrahepatic portal hypertension.

The commonest cause of spontaneous intraperitoneal haemorrhage remains ectopic pregnancy which may be either fallopian or intra-abdominal. Pregnancy can rarely be associated with hepatic rupture and intra-abdominal haemorrhage which may also occur during labour. In non-pregnant women pelvic tumours or rupture of the corpus luteum may cause intra-abdominal haemorrhage. This is more easily recognized if the patient is receiving peritoneal dialysis.

Non-gynaecological intraperitoneal bleeding may occur from primary or secondary hepatic tumours or from other intra-abdominal tumours. Pancreatic pseudocysts may bleed into the peritoneal cavity and haemoperitoneum may also be seen as a complication of spontaneous visceral perforations or arterial haemorrhage.

Portal hypertension seldom results in extraoesophageal variceal haemorrhage but when bleeding is not into the gastrointestinal tract the diagnosis is difficult to make. Hepatocellular carcinoma is a common complication of cirrhosis and may present with intraperitoneal haemorrhage. Therefore bleeding into the abdominal cavity must be considered in all cirrhotic patients with unexplained hypotension and a decreased circulating volume. Prior to the advent of ultrasound and CT imaging the diagnosis was often only made at laparotomy. In this setting the prognosis is poor in patients with liver disease and a 30% mortality rate has been reported. Ultrasound-guided paracentesis offers an easy and rapid method of diagnosis and allows planning of therapy.

References


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