is probably resulted by the complex circulatory dysfunction of both cirrhosis and infection. But, vasoconstrictors act in the liver also, reducing the hepatic blood flow and aggravate portal hypertension and liver failure (J Hepatology 1985; 1:325-337). These effects account for the poor outcome of patients with SBP. The mortality of an episode of SBP is estimated about 30%, comparable with that of variceal hemorrhage (Gastroenterology 1999; 117:495-499). It is known also that large-volume paracenteses (that means removal of large quantity of albumin), results in increased portal pressure (Gastroenterology 1997; 113:579-586). These observations support the usefulness of plasma expansion by albumin (in combination with antibiotics), in the good outcome of patients with SBP and renal impairment (N Engl J Med 1999; 341; 403-409). Over the last 15 years dramatic improvement in survival of SBP has occurred. This is attributed to earlier diagnosis, to use of third-generation cephalosporins, and to synchronous supportive care (Hepatology 1998; 27:264-272). But, 30 to 40 percent of patients develop renal failure, which is the major cause of death (Hepatology 1994; 20:1495-1501). The use of intravenous albumin in the management of cirrhotic ascitis is not accepted by everyone. Some investigators support that large-volume paracenteses are safe if accompanied by intravenous administration of albumin at a dose 6-8g per liter of ascitic fluid removed, since it prevents the deterioration of renal function (Gastroenterology 1988; 94:493-502). Others support that, since up to a third of patients have never used diuretics, the renal impairment caused by paracenteses is of little clinical importance, the benefits in terms of survival is unclear, and the albumin has high cost, then the paracenteses with the administration of albumin is generally unnecessary or at least unnecessary in many patients (Arch Intern Med 1995; 155:373-379). In the study of Sort et al (N Engl J Med 1999; 341:401-409) there was clearly benefit from the administration of albumin plus an antibiotic in patients with cirrhosis and SBP. Advanced cirrhosis and sepsis are separate diseases, but they have similar mechanisms in causing hemodynamic disturbances. Probably these patients represent a subgroup, in whom the plasma expansion prevents the worsening of renal function and the development of hepatorenal syndrome (N Engl J Med 1999; 341:443-444). Of course, care must be taken in early identification of predisposing factors of SBP. Predisposing factors at first episode are the ascitic fluid low levels of total proteins, serum bilirubin and albumin concentrations, prothrombin time, and gastrointestinal hemorrhage (Gastroenterology 1993; 104:1133-1138). High risk subgroup are these cirrhotics with ascitic levels of total proteins <1g/dL, bilirubin concentration >3.3mg/dL and platelet count $<98000/\mu$L (Gastroenterology 1999; 117:414-419). So, the close follow-up, the early identification and treatment of SBP has significant role in outcome of these patients, because after the developing of this complication these patients are candidates for liver transplantation, since their probability of survival the next year without antibiotic prophylaxis is $<40\%$ (Hepatology 1988; 8:27-31).

S. KALATHENOS

Retrograde transvenous obliteration of gastric varices

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Balloon-occluded retrograde transvenous obliteration is an effective new method of angiographic sclerotherapy for treating gastric varices. This minimally invasive interventional radiologic procedure, which was introduced by Kanagawa et al (Endosc Digest 1991; 8:1067-1071), leads to the occlusion of gastric varices through a gastrorenal shunt. Kawanaka et al (Am J Gastroenterol 1995; 90:508-510) were the first to report a successful reversion of hepatic encephalopathy using this angiographic technique.

In this study from Japan, the clinical efficacy, techniques and complications associated with balloon-occluded retrograde transvenous obliteration of gastric varices are presented and evaluated. Between December 1994 and November 1997, balloon-occluded retrograde transvenous obliteration of gastric varices was performed on 20 patients who had demonstrated gastric varices in danger of rupture, according to gastroscopic findings and, gastrorenal shunts, as shown by contrast medium-enhanced spiral computed tomography in the portal phase. Eleven patients had esophageal varices, sixteen had viral hepatitis, one primary biliary cirrhosis, three had alcoholic hepatitis, four complicating hepatocellular carcinoma, one had undergone resection of esophageal cancer and three patients had hepatic encephalopathy.

The procedure that was followed for the connection

of the gastric varices to the left adrenal vein was carefully planned with contrast - medium enhanced computed tomography. A 6-F balloon catheter was inserted from either the right femoral or the right jugular vein and was wedged into the left adrenal vein. Then, left adrenal venography was performed with the balloon inflated. The degree of progression of the gastric varices and of collateral veins was classified into five grades, according to the results of adrenal venography during balloon occlusion. Grade 1 refers to well-opacified gastric varices without evidence of collateral veins, grade 2-4 refers to progressive increase in number and size of collateral veins and gradual decrease in the opacification of gastric varices due to increasing blood flow velocity and grade 5 reveals most progression, as the occlusion of the left adrenal vein is infeasible, because of an existing very large gastrorenal shunt with rapid blood flow. The sclerosing agent (consisting of a mixture of 10% ethanolamine oleate and the same dose of a nonionic contrast medium) was injected into the gastric varices during balloon occlusion, with a maximum dose of 30 ml, used at one time. Intravenous administration of 4,000 units of human haptoglobin was performed to prevent hemolysis and subsequent renal failure, which may be induced by ethanolamine oleate. Collateral veins that had developed were treated with embolization, which was essential for the occlusion of gastric varices with a grade greater than grade 2. The injected mixture remained in the varices for 1-3 hours during balloon oclusion and was (as much of the injected amount as possible) withdrawn under fluoroscopic observation. The procedure was terminated when test injection of the contrast - medium showed the formation of clots in the varices. Follow-up consisted of fiberoptic endoscopy, computed tomography and endoscopic ultrasound examination (or combination of the above). These procedures were performed at 2 weeks, 1 month, 3 months and every 3 months after the initial balloon-occlusion (follow-up period 3-36 months; mean, 16.6 months).

Technical success was achieved in all patients. The clinical symptoms of hepatic encephalopathy in the three patients improved remarkably. Follow-up endoscopy three months after the procedure revealed the disappearance of gastric varices in fifteen patients and reduced variceal size in five. During the follow-up period, 19 patients had no recurrence of gastric varices and three had aggravation of the esophageal varices, but were successfully controlled with endoscopic injection sclerotherapy.

In conclusion, balloon-occluded retrograde transvenous obliteration is a feasible alternative to a transjugular intrahepatic portosystemic shant for patients with large gastrorenal shunts or hepatic encephalopathy (or both).

COMMENTS

In portal hypertension, the consequence of increased hepatic vascular resistance is collateral vessels with portosystemic shunting. Large portosystemic collaterals drain large amounts of blood from the portal venous system, thus reducing blood flow or even giving rise to hepatofugal flow. According to Watanabe et al, gastric varices are seen in 57% of the patients with varices due to portal hypertension and 39% of patients with gastric varices have gastrorenal collaterals (Gastroenterology 1988; 95:434-440).

Gastric varices have a lower bleeding risk factor than esophageal varices (Hepatology 1992; 16:1343-1349). Bleeding of esophageal varices can be prevented by endoscopic injection sclerotherapy, but this is not always effective for gastric varices, especially for fundal varices. Furthermore, the more often esophageal varices are controlled with endoscopic injection sclerotherapy, the more often gastric varices develop because of portal pressure exerted on the perigastric vein (J Gastroenterol Hepatol 1996; 11:51-58). Once gastric variceal bleeding occurs, it is associated with high mortality during the acute episode and a high rate of recurrence in the patients who survive (Endoscopy 1987; 10:7-12). Immediate control of bleeding is, therefore, required.

The methods that are used to control gastric varices are vasoactive agents infusion, surgical intervention, endoscopic injection sclerotherapy, percutaneous transhepatic obliteration and transjugular intrahepatic portosystemic shunt.

Bleeding gastric varices can be injected with the tissue-adhesive cyanoacrylate, as described in few uncontrolled studies (Endoscopy 1986; 18:25-26). This technique was not popularized mainly because of the expertise it requires, particularly in avoiding damage to the endoscope and because of potentially severe complications. More recently, the injection of thrombin was performed with success in 11 patients with bleeding gastric varices, but these preliminary results should be confirmed with further and extended research (Gut 1994; 35:1287-1289).

It is difficult to control bleeding gastric varices, especially fundal varices, by endoscopic injection sclerotherapy (Gastrointest Endosc 1986; 32:264-268).

The high blood flow volume through gastrorenal or splenorenal shunts results in a rapid loss of the sclerosant into the systemic circulation during sclerotherpay. Furthermore, gastric varices are too large to ligate endoscopically.

Surgical portosystemic shunt is a method to decrease the high portal pressure and subsequently decompress the esophageal and gastric varices. In particular, the Hassab operation, which includes davascularization of the upper half of the stomach and esophagus and splenectomy, can eliminate gastric varices (Surgery 1967; 61:169-176). However, patients with gastric varices usually have cirrhosis of the liver and are in a compromised condition. As a result, the mortality rate is 42-56% for elective surgery and higher for emergency procedures (Am J Surg 1986; 152:290-293).

Percutaneous transhepatic obliteration is an effective, non-surgical procedure for treating esophageal and gastric varices, but is more invasive than balloon-occluded retrograde transvenous obliteration and difficult to perform repeatedly. In addition, intrahepatic hemorrhage may occur hwn hemorrhagic diathesis is present.

Transjugular intrahepatic portosystemic shunt (TIPS), first reported by Rosch et al in 1969 (Radiology 1969; 92:1112-1114), involves percutaneous decompression of portal hypertension. Complications, such as stent dislocations, hemoperitoneum and hepatic encephalopathy, sometimes occur and the 30-day mortality rate is 21% (JAMA 1995; 273:1824-1830).

Conversely, balloon accluded retrograde transvenous obliteration is less invasive than surgery, percutaneous transhepatic obliteration or TIPS. Balloon occluded retrograde transvenous obliteration causes the gastric varices to coagulate due to stoppage of the voluminous blood flow in the varices. The mechanism of treating gastric varices seems to be similar to that induced by endoscopic injection sclerotherapy. Collateral veins, such as the inferior phrenic, hemiazygos, or pericardial veins, should be occluded, for a grade of collateral development greater than 2.

Aggravation of esophageal varices is the most important complication of this method. Obliteration of the gastrorenal shunt causes portal venous pressure to increase and new collateral veins to the esophageal varices to develop. In such cases, endoscopic injection sclerotherapy is effective to preclude rupture of esophageal varices.

One of the patients in this study developed cardiogenic shock, immediately after injection of 10ml of the sclerosing agent. Cardiogenic shock is the first time to be reported as a complication of intravenous administration of the sclerosant.

Other complications reported are pulmonary edema, hemothorax and disseminated intravascular coagulation. Migration of the embolic coil at the inferior phrenic vein was another complication caused by a reversal of the direction of the blood flow. Partial obstruction of the renal vein, followed by gross hematuria, was caused once, due to dislocation of the balloon part of the catheter. Hemolysis and, consequently, renal tubular disturbance and renal insufficiency, due to ethanolamine oleate, were prevented, as haptoglobin was administered (one unit of haptoglobin binds 1mg of hemoglobin).

Follow-up results showed successful treatment of gastric varices. These midterm results also showed that the effect persisted for more than a year without aggravation. Additionally, the clinical symptoms of the three patients with encephalopathy improved remarkably. Even though the hemodynamics of balloon-occluded retrograde transvenous obliteration requires future investigations, this method offers good control of gastric varices in patients with gastrorenal shunts and has minimal complications.

E. LIAPI

Epidemics of diarrhea caused by a clindamycin-resistant strain of clostridium difficile in four hospitals

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Clostridium difficile is recognized as a major nosocomial pathogen throughout the world. Epidemics of C. difficile-associated diarrhea are often linked to a single strain capable of causing disease. Large outbreaks of diarrhea caused by a newly recognized strain of C.