Gastric perforation after endoscopic ligation for gastric varices

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Gastric variceal bleeding is a serious complication of liver cirrhosis. Although it occurs less frequently than esophageal variceal bleeding, the associated mortality rate is higher. Endoscopic ligation has been used to treat gastric variceal hemorrhage with preliminary results that suggest that it is efficacious in arresting active bleeding. Because of limited experience, however, there are few reports of complications of endoscopic gastric variceal ligation. This is a report of a patient with gastric variceal bleeding in whom a gastric perforation developed soon after band ligation. The patient recovered completely after emergency laparotomy with simple closure of the perforation.

CASE REPORT

A 62-year-old man with a 20-year history of chronic hepatitis C infection and known cirrhosis presented with hematemesis. Gastroscopy disclosed tortuous and nodular-form varices with active bleeding in the fundus of the stomach. The varices were ligated with 10 bands by using an XQ-30 endoscope (Olympus Optical Co. Ltd., Tokyo, Japan), an overtube, and endoscopic pneumatic ligating devices (Sumitomo Bakelite Co., Ltd., Tokyo, Japan). The pneumatic device consists of a clear 3-layer cylinder (outer cylinder, siding cylinder, and inner cylinder), air tube, and an “0” ring plate. With this device, the “0” ring is

Figure 1. Photomicrograph of resection specimen. Arrowheads indicate edge of transmural perforation of gastric wall. Ulcerative mucosa indicated by arrow. Residual tortuous varices (V) in submucosa (SM) and inflammatory cell infiltration in submucosa, muscularis propria (MP), and serosa (S) are present (H&E, orig. mag. ×5).

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induced ulcerations was achieved. Gastroscopy 1 month later revealed complete eradication of the gastric varices. The patient experienced another episode of hematemesis 15 months later. Gastroscopy disclosed recurrent gastric varices with bleeding. Hemostasis was achieved by endoscopic ligation with 6 bands. Gastroscopy 5 months thereafter disclosed residual varices over the fundus and anterior wall of the proximal body of the stomach. The fundal varices were ligated with 2 bands and another band was placed at the anterior wall of the proximal body. The procedure was completed without incident. However, the patient developed severe epigastric pain 12 hours later that rapidly evolved into generalized abdominal pain.

On presentation to the emergency room the patient had an extremely toxic appearance. He denied use of immunosuppressive or nonsteroidal anti-inflammatory drugs. Examination revealed a distended abdomen with absent bowel sounds. On palpation of the abdomen, muscle guarding and diffuse tenderness with rebound pain were elicited. Laboratory data included the following: white cell 11,100/mm³ (normal: 4800-10,800 mm³), hematocrit 39.3% (37%-47%), platelet 118,000/mm³ (130,000-400,000/mm³), prothrombin time 14 seconds (11.5 sec.), albumin 3.2 g/dL (3.7-5.3 g/dL), blood urea nitrogen 38 mg/dL (7-20 mg/dL), creatinine 1.2 mg/dL (0.7-1.5 mg/dL), alanine aminotransferase 74 IU/L (0-40 IU/L), aspartate aminotransferase 98 IU/L (5-45 IU/L), amylase 512 IU/L (10-100 IU/L), alkaline phosphatase 61 IU/L (10-40 IU/L), glucose 123 mg/dL (65-115 mg/dL), sodium 143 mmol/L (135-147 mmol/L), potassium 3.9 mmol/L (3.4-4.7 mmol/L), and total bilirubin 0.7 mg/dL (0.2-1.6 mg/dL). A chest radiograph showed bilateral subphrenic free air.

The patient underwent emergency laparotomy at which a round perforation about 1 cm in diameter was found on the anterior wall of the proximal body of the stomach. A circular resection of the perforated area was performed with simple closure of the wound. Histopathologic evaluation of the resection specimen demonstrated transmural necrosis of the gastric wall and peritonitis (Fig. 1). The patient was treated by intravenous administration of cefazolin, 1 g every 6 hours; metronidazole, 500 mg every 8 hours; netilmicin, 150 mg every 12 hours; and nothing by mouth for 5 days. Oral intake was then resumed and the patient was discharged 10 days later. Gastroscopy 1 month later revealed complete eradication of the gastric varices and healing of the suture site.

**DISCUSSION**

The optimal treatment for gastric variceal bleeding has not been defined. Endoscopic variceal sclerotherapy with absolute alcohol or ethanalamine olate is unsatisfactory because the rate of recurrent bleeding is high and the bleeding is frequently uncontrollable. Endoscopic injection of N-butyl-2-cyanoacrylate is more effective than a conventional sclerosant. However, this carries the risk of damage to the endoscope and serious complications may occasionally occur. The procedure described by Hassab, which consists of a splenectomy together with devascularization of the stomach and distal esophagus, has an associated mortality rate of up to 38.4%. The transjugular intrahepatic portosystemic shunt may increase the severity of encephalopathy and is less efficacious in the treatment of gastric variceal bleeding. Retrograde balloon occlusion was hailed as an effective treatment for fundal variceal bleeding, but this procedure has an associated high risk for the subsequent development of esophageal varices.

Endoscopic ligation is an optimal method for treating esophageal variceal bleeding. However, experience with this technique in the treatment of gastric variceal bleeding is limited. Preliminary studies have shown that the endoscopic ligation of gastric varices is safe and effective. However, 2 cases of gastric perforation occurring 3 days after ligation of cardial and fundal varices with a detachable snare have been reported. Although the volume of tissue ligated by a pneumatic ligator is more uniform and smaller than that ligated with a detachable ligator, nevertheless this case of gastric perforation after an endoscopic band ligation was encountered.

Esophageal perforation may sometimes occur when balloon tamponade is superimposed on a postligation esophageal ulcer or as a result of blunt trauma caused by overtube insertion for the purpose of variceal ligation. However, endoscopic ligation never causes esophageal perforation because only the mucosa and submucosa are ligated, not the muscular layer. Immunosuppression has been considered a detrimental factor for esophageal perforation after endoscopic variceal ligation, but our patient did not use any immunosuppressive agent before or after the ligation procedure. The gastric perforation occurred soon after an endoscopic ligation procedure. Thus, transmural trapping of the gastric wall might account for the perforation, although the exact mechanism of the “transmural” ligation is not known. However, if repeated endoscopic gastric ligation increases the potential for gastric perforation, it would be worthwhile to investigate whether gastric variceal ligation should be reserved for acute hemostasis and not used repeatedly to achieve eradication.

Gastric perforation is a rare complication of endoscopic ligation of gastric varices. Because preliminary results indicate that endoscopic gastric variceal ligation effectively controls bleeding and leads to rapid eradication of gastric varices, this procedure may become more popular. The rate and range of complications associated with it are not established and more such complications may become evident in the course of time. This lack of

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data should be taken into consideration when the procedure is to be used.

REFERENCES