Prevalence of Gastroesophageal Varices Supplied by Intrahepatic Portal Branch

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The contribution of the intrahepatic portal branch to the gastroesophageal varices is rare. This anomalous pathway (a large left intrahepatic portal branch) was proved by portographic study and by imaging techniques in a 54-year-old man with cirrhosis. The overall prevalence of this type of collateral in the literature is only 1.9% in 908 patients with portal hypertension. In addition, all are via the left portal venous branch. It may be speculated that the mechanism of this variation is due to dilatation of the rest of the intrahepatic portal system via a small anastomosis due to portal hypertension.

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Introduction

Gastroesophageal varices are an important complication in patients with portal hypertension because of the possible risk of bleeding. It is well known that these variceal channels are usually supplied by the extrahepatic portal branches, such as the left, posterior, and short gastric veins (1-5). However, the contribution of the intrahepatic portal branches to the gastroesophageal varices is seldom described. Recently, we have experienced the case of gastric varices supplied by a large intrahepatic branch. To our knowledge, only 19 cases have been reported in the literature (3, 5-7). Here, we report one case and discuss the prevalence of this anomalous pathway in patients with gastroesophageal varices.

Case Report

A 54-year-old man with cirrhosis (due to alcohol consumption plus HCV infection) was admitted to Kurume University Hospital because of bleeding from esophageal varices. On physical examination, the blood pressure was 116/60 mmHg. The liver and spleen were not palpable, and there were no abdominal masses and dilated veins at the anterior abdominal wall. Laboratory data yielded the following values: white blood cell count, 5,000/mm³; red blood cell count, 324×10⁶/mm³; hemoglobin concentration, 8.2 g/dl; platelet count, 14.3×10⁵/mm³; HBsAg, negative; HCV-Ab, positive; total bilirubin, 0.8 mg/dl; serum aspartate transaminase, 68 U/L; albumin, 3.4 g/dl; prothrombin time, 40%, and ICG-R15, 17%. Real-time ultrasonography showed no ascites, but a large left intrahepatic portal vein running toward the stomach was found (Fig. 1). Endoscopic examination showed esophageal [CW, F2, and red color sign (+)] (8) and nodular tortuous gastric varices. Percutaneous transhepatic portography was carried out to evaluate portal hemodynamics. This study showed that gastric varices seemed to be supplied by a large left intrahepatic portal vein and esophageal varices were supplied by the left, posterior, and short gastric veins (Fig. 2). The former finding was subsequently confirmed by CT scan (Fig. 3). Free portal venous pressure was 22 mmHg. Endoscopic sclerotherapy was then performed using a total dose of 55 ml of 5% ethanolamine oleate for esophageal varices. Follow-up portography showed that a large left intrahepatic portal vein was nearly unchanged, although esophageal varicogram was not visualized. In addition, free portal venous pressure was not modified.

Discussion

In our laboratory, from February 1985 to the end of 1991, percutaneous transhepatic portography has been performed to determine the therapeutic protocol in a total of 65 patients with gastroesophageal varices. In agreement with previous reports (1-5), gastroesophageal varices were found to typically arise from the extrahepatic portal venous branches. In our series, these consisted of the left gastric vein in 60 (92%), posterior transaminase, 91 U/L; serum alanine

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Fig. 1. Ultrasonogram of the left lobe of the liver. A large left intrahepatic portal vein running toward the stomach is visualized.

Fig. 2. Portogram obtained before sclerotherapy. A large intrahepatic portal branch running toward the left subphrenic region is opacified after contrast medium injection in the superior mesenteric vein (upper panel). Esophageal varicogram is opacified from gastric branches after contrast medium injection in the splenic vein (lower panel).

Fig. 3. Enhanced CT scan shows a large left intrahepatic branch (arrow) running toward the upper part of the stomach (arrows).
gastric vein in 29 (45%), and short gastric vein in 27 (42%) (unpublished observation).

In the present case, portography obtained before and after the sclerotherapy indicated that gastric varices was primarily supplied by a large left intrahepatic portal branch, even though esophageal varices was supplied by the extrahepatic portal venous branches. This supplier of gastric varices is interesting because the intrahepatic portal branches do not emerge from the liver in normal anatomy. The mechanism concerning the development of this anomalous collateral vein is not yet clarified. However, it may be speculated that the rest of the intrahepatic portal system via a small anastomosis, as designated the portocoronary anastomosis by Rousselot et al (9, 10), would be dilated due to portal hypertension.

The collateral pathway originating through the intrahepatic portal branches is rarely reported in portographic studies. As listed in Table 1, the overall prevalence of this type of collateral in the literature is only 1.9% among 908 patients with portal hypertension. In addition, all were supplied via the left portal venous branch.

In summary, various feeders of gastroesophageal varices exist in patients with portal hypertension.

References