Gastroenterologia Japonica
Vol. 18, No. 6

Printed in Japan

—Case Report—

A CASE OF BLEEDING DUODENAL VARICES LOCATED IN THE THIRD PORTION

Mahito NAKAYAMA, M.D., Yoichi KAMURO, M.D. Katsuro SAGARA, M.D.,
Tatsuo SATO, M.D., Koichiro TOGAMI, M.D.* and
Seiki TASHIRO, M.D.*

*Third Department of Internal Medicine and *The First Department of Surgery,*
Kumamoto University School of Medicine, Kumamoto 860, Japan

Summary

We described a patient with isolated duodenal varices of the third portion, preoperatively diagnosed by hypotonic duodenography and endoscopy. These varices seemed to be caused by portal hypertension due to liver cirrhosis. The third to the fourth portion of duodenum should be examined carefully in patients with liver cirrhosis, especially with gastrointestinal bleedings of unknown origin.

Key Words: *gastrointestinal bleeding, duodenal varices.*

Gastrointestinal bleeding of unknown origin often plagues the clinician. Varices of the alimentary tract might cause such bleedings. It is generally recognized that the esophagus is the most common site of varices of the alimentary tract and the fundus of stomach is less frequent. Varices of the rectum, sigmoid colon, cecum and jejunum are seldom reported[1,2]. Alberti reported the first three patients with duodenal varices in 1931[3], and added a fourth in 1933[4]. Since then, there have been several reports of duodenal varices, but almost all of them were located in the bulb[5-7] or the second portion[8,9] of the duodenum. Our patient had varices located in the third portion of the duodenum and at the same time no remarkable varices of the esophagus or stomach were demonstrated. The diagnosis was preoperatively made by hypotonic duodenography and endoscopy.

So far as we know, this in the first case of bleeding from isolated duodenal varices of the third portion. In this paper, we emphasize the importance of hypotonic duodenography and endoscopy in establishing the diagnosis before operation.

Case Report

The patient was a 45-year-old woman. She had no history of alcohol intake. She had two brothers, one of whom had died of liver cirrhosis and the other had died of liver cirrhosis with hepatocellular carcinoma, respectively. They were heavy drinkers. She was well until August, 1979 when she developed fatigue. Liver dysfunction was pointed out by a physician, and received medications frequently. At the beginning of March, 1980, she complained of periorbital and pretibial edema and abdominal dis-

Received June 9, 1983. Accepted August 14, 1983.

Address requests for reprints to: Mahito Nakayama, M.D., Third Department of Internal Medicine, Kumamoto University School of Medicine, 1-1-1 Honjo, Kumamoto City, 860 Japan.

tension. On March 11, she was admitted to Kumamoto University Hospital for further liver evaluation.

Physical examination revealed slight pretibial edema, slight ascites and a firm liver edge palpable 3 cm below the costal margin in the right mid-clavicular line, but no icterus was found. Other findings were normal. Laboratory findings were as follows; peripheral red blood cell count was $326 \times 10^4/mm^3$, hemoglobin 9.4 g/dl, hematocrit 30.2%, white blood cell count $4,500/mm^3$ and platelet count was $10.2 \times 10^4/mm^3$. Prothrombin time and partial thromboplastin time were normal. SGOT was 50 U/L, SGPT 29 U/L, alkaline phosphatase 69 U/L, LDH 178 U/L, total bilirubin 0.5 mg/dl, total serum protein 5.6 g/dl with albumin of 2.7 g/dl and γ-globulin of 1.8 g/dl, total serum cholesterol 128 mg/dl, blood sugar 75 mg/dl, blood urea nitrogen 9 mg/dl, serum creatinine 0.7 mg/dl, venous ammonia 40.1 μg/dl, and serum electrolytes were within normal limits. HBs antigen and antibody were negative. Alfa-fetoprotein was 18.3 ng/ml (N<20 ng/ml). Bromo-sulfaleine (BSP) retention was 22.8% at 45 minutes after the injection. Plasma disappearance rate of indocyanine green was 0.069. Chest and abdominal X-rays were not remarkable. Liver scintigram showed a flying bat pattern, but no space occupying lesions were found. Upper gastrointestinal endoscopy revealed slightly dilated veins in the lower portion of esophagus. On the 27th hospital day, peritoneoscopy was performed. The surface of the liver was nodular to varying degrees, and histologically micronodular cirrhosis was diagnosed.

On the 36th hospital day, she suddenly experienced an episode of melena. Emergency endoscopy was performed but no bleeding in the esophagus, stomach or duodenal bulb was observed. Melena continued for 12 days, and she received intermittent transfusions of 2,200 ml red blood cells. Thereafter two mild episodes of melena occurred 7 days apart. After improvement of general conditions, on the 71st hospital day she underwent hypotonic duodenography, and clearly lobulated filling defects without ulcerations and umbilications on the surface were demonstrated in the third portion of the duodenum (**Fig. 1**). Upper gastrointestinal endoscopy showed large bluish

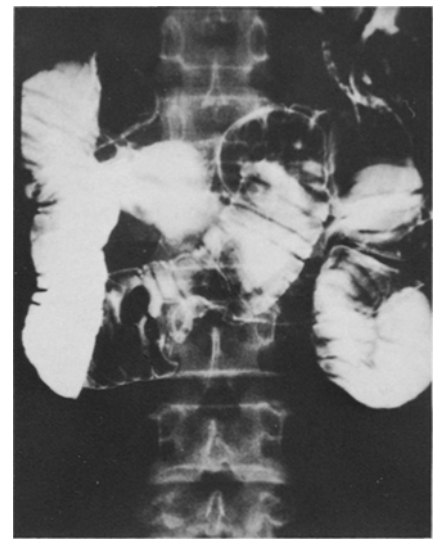

Fig. 1. Hypotonic duodenograph showing lobulated filling defects in the third portion.

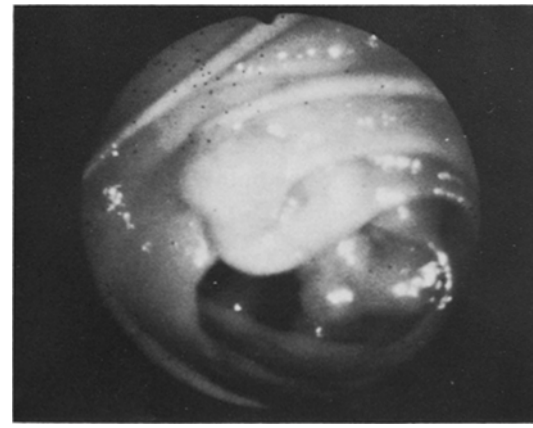

Fig. 2. Endoscopic view of the third portion of duodenum showing bluish varices in the submucosa of the duodenal wall.

varices in the same portion (**Fig. 2**). Bleeding had already ceased. Celiac and superior mesenteric angiography demonstrated remarkably dilated superior mesenteric vein and several varices along it in the venous phase (**Fig. 3**). The site of some varices corresponded to the third portion of duodenum.

On 18, May, she underwent celiotomy. Fine nodular liver cirrhosis, associated with extensive collateral vessels and enlarged spleen were found. Large dilated serpiginous varices were shown on the lateral wall of the third portion of duodenum (**Fig. 4**). The other areas of the duodenum appeared normal. These tortuous varices were dissected together with the spleen and upper gastric and paraesophageal devascularization was carried out. No obstruction of the extrahepatic portal vein and splenic vein was demonstrated. A porto-systemic shunt was not done. The excised specimen showed the varices on the internal surface of duodenum (**Fig. 5**). Microscopic examination revealed an ulceration on the surface of a varix. The postoperative course was uneventful. No varices have recurred during one year follow-up of repeated endoscopy.

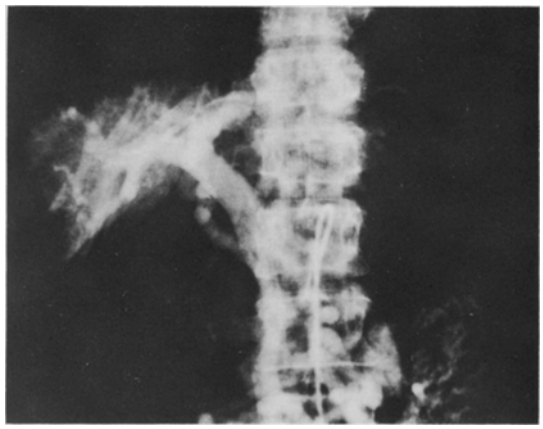

Fig. 3. Superior mesenteric arteriograph (venous phase) demonstrating dilated superior mesenteric vein and several varices along it.

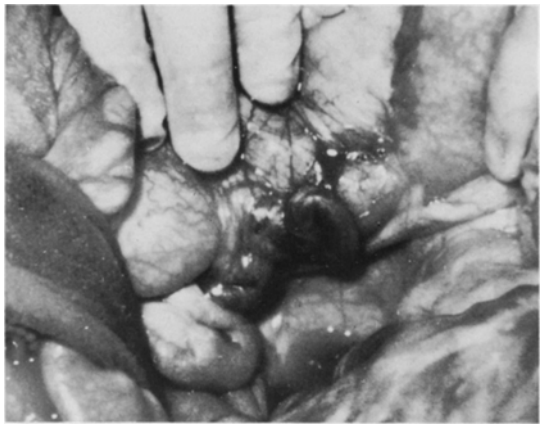

Fig. 4. Large dilated serpiginous varices are seen on the lateral wall of the third portion of duodenum.

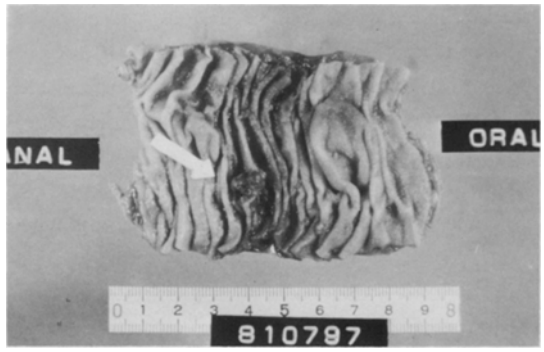

Fig. 5. Excised specimen showing the varices on the internal surface of duodenum. Ulceration can be seen on the surface of a varix (arrow).

Discussion

Gastrointestinal varices are one of the most significant signs in the diagnosis of portal hypertension, but bleeding from unknown sites often presents problems. The esophagus and gastric fundus are common sites of alimentary tract varices[1]. Varices of other areas of the alimentary tract such as the rectum, sigmoid colon, caecum and jejunum have often been reported[1,2]. There have been several reports on duodenal varices, but almost all were located in the bulb or the second portion of the duodenum.

In this patient, no remarkable varices of the

esophagus or stomach were demonstrated by routine upper G.I. series. After the episodes of melena, hypotonic duodenography was carried out and clearly lobulated filling defects were observed in the third portion of the duodenum. Formation and rupture of varices isolated in the third portion are very rare in patients with liver cirrhosis.

In infancy, varices of the alimentary tract might arise from obstruction of the extrahepatic portal vein due to omphalitis, congenital atresia and so on. In adult, varices can often be caused by the obstruction of the extrahepatic portal vein or splenic vein due to neoplasm, pancreatitis, pancreatic pseudocyst and thrombosis. However celiac and superior mesenteric angiography did not demonstrate such lesions in this case. The duodenal varices of this patient might have been caused by portal hypertension resulting from liver cirrhosis. There have been several reports on the surgical treatment of duodenal varices. Partial resection of the duodenum (the third to the fourth portion), splenectomy and upper gastric and paraesophageal devascularization were performed in this case. The postoperative course was uneventful and there has been no more bleeding or varices formation during a follow-up period of over one year. Her liver function has not deteriorated. It would appear from this case that surgical treatment of duodenal varices is effective for the prevention of subsequent varices formation and bleeding.

Although bleeding from duodenal varices isolated in the third portion is a rare complication of liver cirrhosis, the possibility should be considered and these patients should be examined carefully, especially with episodes of gastrointestinal bleeding of unknown origin. Hypotonic duodenography and gastrointestinal endoscopy can contribute to accurate diagnosis of such patients.

References

1) Ruzicka FF Jr: Portography in "Alimentary Tract Roentgenology" Vol 2, by Margulis AR, Burhenne HJ, CV Mosby Co, St Lois, 1973, p 1359
2) Hamlyn AN, et al: Portal hypertension with varices in unusual sites. Lancet 2: 1531, 1974
3) Alberti W: Über den röntgenologischen Nachweis von Varizen im Bulbus duodeni. Fortsschr Roentgenstr 43: 60, 1931
4) Alberti W: Über den röntgenologischen Nachweis von Varizen im Bulbus duodeni. ibid 47: 467, 1933
5) Richter RM, et al: Duodenal Varices. Arch Surg 95: 269, 1967
6) Bateson EM: Duodenal and antral varices. Br J Radiol 42: 744, 1969
7) Rappazzo JA, et al: Duodenal varices: endoscopic diagnosis of an unusual source of upper gastrointestinal hemorrhage. Gastroenterol Endoscopy 27: 227, 1981
8) Shearburn EW, et al: Duodenal varices treated by portacaval shunt. Arch Surg 93: 425, 1966
9) Richardson JD, et al: Duodenal varices. Am Surg 42: 201, 1976