

A Portal Angiographic Sign in a Patient with Esophageal Varices Following Total Gastrectomy and Esophagojejunostomy

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Summary. An increase in arterial inflow at the jejunum was noted in a patient with esophageal varices who had received total gastrectomy and esophagojejunostomy. There was an peculiar angiographic sign in the portal circulation to this situation; namely, the jejunal vein had a divide between the hepatofugal and hepatopetal flow. The divide, termed a "watershed", was obtained at first, and thereafter esophageal varices were identified.

Because a hyperdynamic state in the splanchnic circulation contributes to the formation of esophageal varices, the enhanced jejunal circulation may participate in the development of the varices, and a special angiographic sign—a "watershed"—may be a sign of a splanchnic hyperdynamic state.

Key words—liver disease, splanchnic circulation, portal hypertension, hemodynamics.

INTRODUCTION

In 1977, Inokuchi and his coworkers investigated splanchnic circulation in portal hypertension, and suggested that flow resistance between the left gastric artery and the vein markedly decreased, leading to a remarkable increase in arterial inflow into the portal area. They postulated that the increase in arterial inflow plays a major role in the formation of esophageal varices.¹⁾ This suggestion was confirmed by Aoki and his coworkers, who have proposed a classified guidance for surgical treatment.²⁾ However,

in patients with portal hypertension, it is probable that the increase in arterial inflow takes place not only in the gastric wall but also in other organs connected to the esophagus. Recently, we observed an increase in arterial inflow at the jejunum in a patient with esophageal varices who had had total gastrectomy and esophagojejunostomy, and detected an peculiar angiographic sign in the portal circulation of this situation.

CASE REPORT

A 58-year-old Japanese man was admitted to Niigata University Hospital for the management of recurrent esophageal varices. He had undergone total gastrectomy, splenectomy and esophagojejunostomy (Roux-Y) in May, 1987 because of advanced gastric cancer. He had also suffered from alcoholic liver injury, and esophageal varices associated with liver cirrhosis were noted during surgery.

A liver biopsy showed micronodular cirrhosis. Laboratory findings on admission are shown in Table 1. He was classified as Child's A. Recovery from surgery was uneventful, and esophageal varices improved at once and have progressed gradually since then. Liver dysfunction was not progressive.

The angiographic findings at the second admission are as follows. A superior mesenteric arteriography indicated that the diameter of the arterial branch in the jejunum anastomosed with the esophagus had become enlarged compared with that postoperative at the first admission. Percutaneous transhepatic portography followed by X-ray contrast medium

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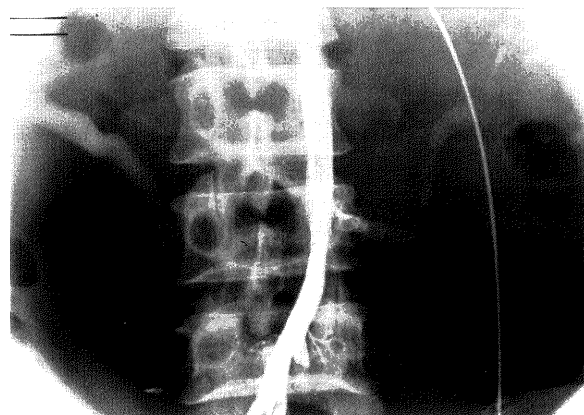
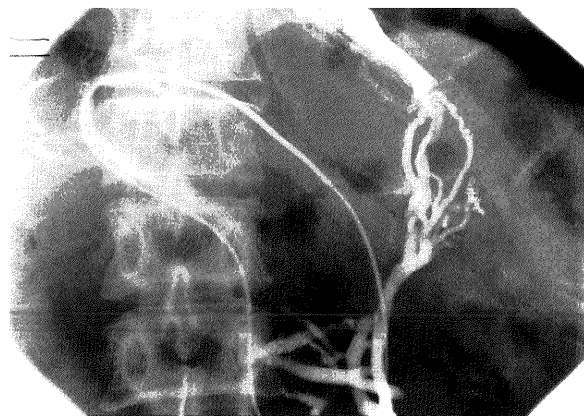
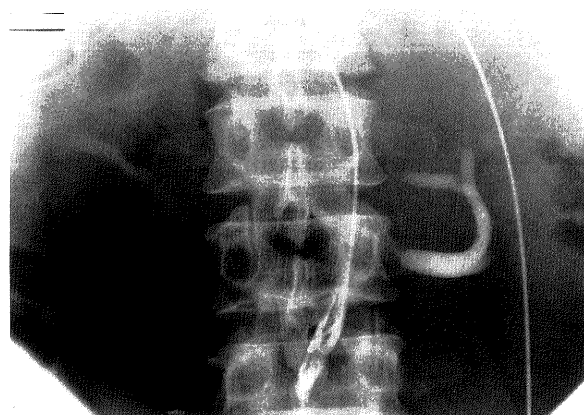
Table 1. Blood chemical scores before (I, May 8, 1987) and after (II, June 22, 1987; III, January 31, 1990) gastrectomy

	I	II	III
White blood cells (/ μ l)	3100	5000	4000
Red blood cells ($\times 10^4$ / μ l)	409	426	363
Platelets ($\times 10^4$ / μ l)	5.7	10.5	23.7
Total protein (g/dl)	6.4	8.4	6.4
Albumin (g/dl)	3.2	4.1	3.3
Cholinesterase (IU/l)	3702	5454	3169
Glutamate oxaloacetate transaminase (IU/l)	65	35	60
Glutamate pyruvate transaminase (IU/l)	46	27	48
Total bilirubin (mg/dl)	1.3	1.4	1.0
Direct bilirubin (mg/dl)	0.8	0.8	0.6
Hepaplastin test (%)	68	44	60
Indocyanine green disappearance rate (/min)	0.062	0.049	0.081
Indocyanine green retention rate (R_{15}) (%)	38.8	46.3	27.5

infusion into the portal or superior mesenteric vein showed the hepatopetal direction. No hepatofugal flow toward the esophageal varices was detected (Fig. 1). In unsequential selective infusion into the jejunal vein that had been connected to the esophagus, the greater part of the X-ray contrast medium flowed in the hepatofugal direction (Fig. 2). There was a divide between the hepatofugal and hepatopetal flow in the jejunum when Lipiodol was infused into the jejunal vein (Figs. 3 and 4). The venous pressure in the divide was estimated to be 23–24 mmHg, whereas in the proximal and distal portions of the divide it was 21–24 mmHg.

DISCUSSION

In treating a case of esophageal varices associated with jejunal hyperdynamic circulation following total gastrectomy, we discovered an peculiar angiographic

**Fig. 1.** Superior mesenteric venogram: only a hepatopetal flow is seen.**Fig. 2.** Selective portal venogram to the anastomosed jejunum: only a hepatofugal flow to the esophagus is seen.**Fig. 3.** Stasis of Lipiodol is seen in the splanchnic portal area. Lipiodol was infused slowly into the jejunal vein approximately 2 to 6 cm distal to the confluence of the superior mesenteric vein.

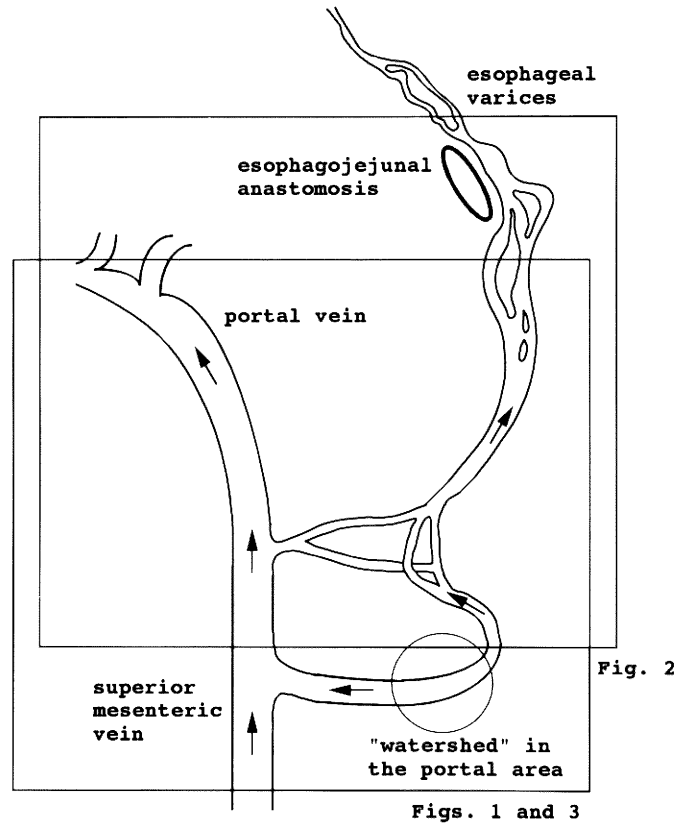


Fig. 4. An image composed of Figs. 1, 2 and 3. *Arrows* indicate the direction of the blood flow.

sign in the portal circulation which may be specific to this situation.

The mechanism forming the angiographic sign is not easy to explain, but, as was pointed out by several investigators, when pressure in the portal circulation is high, blood in the splanchnic vascular bed returns to the heart through not only the portal vein but also the collateral pathways, and there is a divide between the portal and collateral circulation.¹⁻³⁾ The angiographic sign may hold true for the divide. Conversely, this sign suggests the existence of portal hypertension and collateral circulation.

One of the collateral pathways is the esophageal pathway, and because an increase in pressure in the venous structure produces varices,^{4,5)} an increase in esophageal inflow contributes to the development of esophageal varices.⁶⁾ In this study, jejunal circulation was judged to be in a hyperdynamic state, and venous inflow to the esophagus was enhanced. It is likely that the angiographic sign also implies an increase in esophageal inflow.

It has been indicated that an increase in arterial

inflow in the gastric wall contributes to the formation of esophageal varices.^{1,2)} However, in this case, the influence of gastric circulation was excluded because the stomach had been removed. In connection with this, as mentioned above, esophageal varices can be induced when the esophagus is connected to a hyperdynamic collateral circulation. Thus, a hyperdynamic state in the splanchnic circulation may result in varices formation with or without the stomach intact.

There was a divide between the hepatofugal and the hepatopetal flow in the splanchnic angiographic findings. In a previous report, we called such a divide a "watershed", and emphasized that this watershed may be a sign specific to esophageal varices complicated with portal hypertension.³⁾ In this case, the same angiographic sign appeared after total gastrectomy, and subsequently, esophageal varices were identified. It can therefore be expected that the "watershed" sign has a diagnostic significance common to esophageal varices with portal hypertension.

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