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Large extrahepatic portosystemic shunt without portal hypertension

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Abstract

Extrahepatic portosystemic shunt usually occurs secondary to severe portal hypertension, and it is rare to encounter it in patients without portal hypertension. We report herein a large extrahepatic portosystemic shunt between the left gastric vein and left renal vein without portal hypertension in which color Doppler sonography was useful not only for detection but evaluation of the effect of embolization.

Key words: Color Doppler—Portosystemic shunt—Ultrasound—Embolization.

Extrahepatic portosystemic shunt usually occurs in patients with severe portal hypertension, and it is frequently associated with portosystemic encephalopathy [1]. The literature includes only a few cases not associated with portal hypertension [2, 3], and these reports dealt with shunts between the superior mesenteric vein and the inferior vena cava. We encountered recently a case of a large portosystemic shunt between the left gastric vein and left renal vein without portal hypertension in which embolization using stainless steel coils markedly improved the patient's encephalopathy. To our knowledge, this paper is only the second report of such an unusual shunt. We wish to stress the usefulness of color Doppler sonography not only for detection of this unusual shunt but for evaluation of the effect of treatment.

Case Report

A 62-year-old man suffering from chronic alcoholism was admitted to our hospital because of confusion. His medical history included a partial gastrectomy for a recurrent duodenal ulcer 30 years ago and treatment for alcohol abuse 8 years ago. During surgical intervention for his duodenal ulcer no other abnormality was found including in the abdominal vessels. He complained of morning confusion and abnormal behavior for the last 9 years, and he has been on medication for chronic alcoholism in a mental hospital. His medical doctor pointed out that he had a high serum ammonia level—up to 348 μg/dl (normal $<$ 70 μg/dl)—and suspected hepatic encephalopathy and requested ultrasonography for evaluation of the liver. Before this time the serum ammonia had not been measured, nor had ultrasonography been done. The serum chemistry was normal except for severe anemia (red blood cells $212 \times 10^4/\mu$l, hemoglobin 6.5 g/dl, hematocrit 19.4%) and a high serum ammonia level (189 μg/dl; normal $<$ 94 μg/dl). Esophagogastric endoscopy showed no abnormality. Sonography revealed an abnormal vessel (1.5 cm in diameter) behind the pancreas running backward. The liver was normal on sonography, and there were no other abnormalities in the abdomen. On conventional sonography, it was difficult to evaluate the relation between this vessel and the neighboring vascular system. Color Doppler sonography clearly demonstrated that this vessel originated from the confluence of the superior mesenteric vein and the splenic vein and communicated with the left renal vein. It contained a backward constant flow (Fig. 1A).

The flow velocity and flow volume of the portal system were as follows: main portal vein (0.29 m/s, 0.52 L/min), splenic vein (0.19 m/s, 0.30 L/min), superior mesenteric vein (0.30 m/s, 0.55 L/min), and the abnormal vessel (0.05 m/s, 0.34 L/min). Thus the shunt rate (shunt volume/total portal volume) was 39.5%. There were no other abnormal vessels in the abdomen. Arterial portography via the superior mesenteric artery showed the markedly dilated left gastric vein communicating directly with the left renal vein (Fig. 1B). There were no other shunts or collateral circulations in the abdomen.

Embolization therapy using 10 stainless steel coils was attempted. The catheter was introduced via the ileocecal vein to the superior mesenteric vein through a small incision. Ten stainless coils were successfully delivered into the left gastric vein. Portal pressure measured during this procedure was normal (main portal vein 9 mm Hg, left renal vein 8 mm Hg).

Color Doppler sonography after embolization showed complete obstruction of the left gastric vein. The flow velocity and flow volume of the portal system were as follows: main portal vein (0.22 m/s, 0.88 L/min), splenic vein (0.11 m/s, 0.08 L/min), and superior mesenteric vein (0.20 m/s, 0.75 L/min). On careful observation, multiple fine collaterals appeared around the pancreas (Fig. 2A). Superior mesenteric portography performed 1 week after embolization showed disappearance of the shunt flow (Fig. 2B). The postembolization course was uneventful, and the serum ammonia level rapidly normalized. The patient is now symptom-free and is receiving no special medical therapy.

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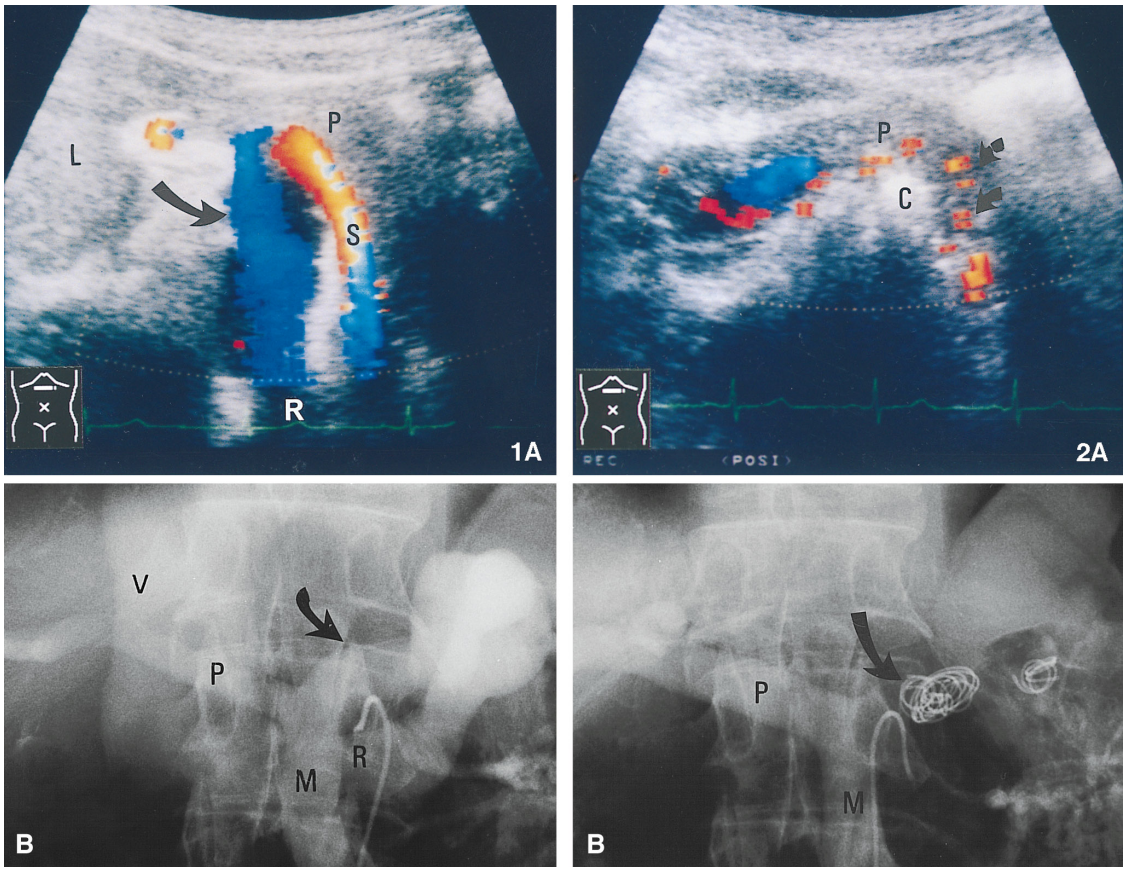

Fig. 1. A Color flow imaging on admission. Color flow imaging in the transverse scanning plane passing through the level of the pancreas visualizes a large vessel running backward from the confluence of the superior mesenteric vein and the splenic vein. *P*, pancreas; *L*, liver; *S*, splenic vein; *R*, left renal vein; *arrow*, abnormal vessel. **B** Superior mesenteric portography demonstrates the markedly dilated left gastric vein communicating with the left renal vein. Inferior vena cava was also seen at this phase. *P*, main portal vein; *M*, superior mesenteric vein; *V*, inferior vena cava; *R*, left renal vein; *arrow*, dilated left gastric vein.

Fig. 2. A Color flow imaging after embolization confirms the disappearance of the abnormal vessel shown in Figure 1. Note the multiple fine collaterals around the pancreas. *P*, pancreas; *C*, steel coil; *arrow*, fine collaterals. **B** Superior mesenteric portography after embolization demonstrates the disappearance of the portosystemic shunt. *P*, main portal vein; *M*, superior mesenteric vein; *arrow*, steel coil.

Discussion

Large extrahepatic portosystemic shunt provokes so-called portosystemic encephalopathy due to unaltered portal blood passing directly into the systemic vein [4]. There is little doubt that in the presented case the neuropsychological disturbance resulted from the portosystemic shunt between the left gastric vein and left renal vein, because after embolization the serum ammonia level rapidly normalized and the patient's complaints disappeared. In this case, although a congenital anomaly can be considered, the cause of the portosystemic shunt remains unknown.

This case has two major unusual points: (1) a large extrahepatic portosystemic shunt without portal hypertension, and (2) an isolated shunt between the left gastric vein and left renal vein. The literature includes only one case of such an unusual shunt, reported by Matsumuro et al. in 1988 [5]. They reported a 60-year-old woman with a complaint of recurrent episodes of neurologic disturbance of 10 years' duration. There were no signs of portal hypertension, and hepatic function was normal. Laboratory data showed a high concentration of serum ammonia (86 μg/dl; normal $<$ 75 μg/dl), and arterial portography disclosed a large portosystemic shunt between the left gastric vein and left renal vein. Our case closely resembles their case in terms of the clinical history and portographic findings.

A definitive diagnosis of extrahepatic portosystemic shunt has been made usually by demonstrating an outpouring of contrast medium from the portal system into the venous system on portography. The final diagnosis can now also be determined by direct demonstration of the communication between the portal

system and the venous system on color Doppler sonography.

Color Doppler sonography is used extensively to evaluate vessels in the abdomen. It is most useful for determining flow direction and flow pattern [6]. However, the most important advantage of the technique is its noninvasiveness and repeated utilization. Although approximate, color Doppler sonography yields much useful information about portal hemodynamics. In fact, in our case color Doppler sonography permitted us to measure the shunted blood flow and to evaluate the effect of embolization: Almost 40% of the portal flow passed through the portosystemic shunt, and after embolization the shunt flow had ceased, and the blood flow in the main portal vein increased.

There is no standard treatment for portosystemic shunts. However, the satisfactory result obtained following embolization in our case suggests that it may be the treatment of choice for an extrahepatic shunt. Thus an aggressive surgical intervention need not be attempted.

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