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A Case Simultaneously Presenting with a Rare Portal Collateral Pathway and Left Gastric Venous Anomaly

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Summary: We had the opportunity to dissect an autopsy case who had developed a rare portal collateral pathway due to increased portal pressure resulting from liver cirrhosis and simultaneous abnormal left gastric venous distribution. The portal collateral pathway consisted of a well-developed communicating branch located between the left renal vein and the left gastric vein. The left gastric vein did not merge into the portal vein, but directly entered the liver after bifurcating near the hepatic hilum. One branch had an anastomosis to the left branch of portal vein in the liver and the other distributed in the hepatic quadrate lobe. We considered this aberrant left gastric vein to be a congenital residue of the embryological left portal vein. The present case is the third Japanese case to have been described minutely in the literature, following the two cases reported by Miyaki *et al.* (1987). Persistence of the umbilical vein and the absence of the celiac trunk were also observed.

It is known, that when the portal vein is occluded, various collateral routes are developed. Representative examples listed in the textbooks of anatomy include the paraunbilical vein, rectal vein and esophageal veins (Gray, 1973; Walls, 1977). However, only a few opportunities exist to dissect and observe such cases during the dissecting practice (Yamada, 1934; Kubota *et al.*, 1957; Sakamoto *et al.*, 1997). Clinically, although there have been reports on the observations of portal collateral routes using imaging diagnostic systems, details of these routes remain unknown (Kokubo *et al.*, 1990; Tajima *et al.*, 1992; Takayasu *et al.*, 1990).

On the other hand, the left gastric vein forms the coronary vein, together with the right gastric vein (Miki, 1973). Embryologically, this is derived from the vitelline veins and is said to be a residue of the left portal vein (Miyaki, 1973, 1978, 1987). Particularly in normal individuals, this is evidenced by the distribution of the left gastric vein directly in the liver, which is an important morphological feature.

We had the opportunity to dissect a cadaver of a patient who had simultaneously a rare developed portal collateral pathway resulting from increased portal pressure caused by liver cirrhosis and an abnormal distribution of the left gastric vein. The rare portal collateral pathway was a well-developed communicating branch located between the left renal vein and left gastric vein. The left gastric vein did not merge into the portal vein, showing direct entry into the liver. The present case is considered of much value clinically and anatomically. The detailed findings of this anomaly are presented in this report.

Findings

This was a case of a 65-old-year female (cadaver No.: 94042) who died from liver cirrhosis and whose history was unknown.

1. Confluence of portal venous roots (Fig. 1)

The cadaver's portal venous trunk was formed after the inferior mesenteric vein entered the superior mesenteric vein, then the splenic vein participated at about 5 mm upward from the point of the confluence. The short gastric veins had two branches: one originated approximately at the dorsal surface of the gastric fundus and the other originated in the vicinity of the cardiac notch. Both veins were directed towards the left and opened to the root of the splenic vein.

2. Gastric veins (Fig. 1, 2)

There was no right gastric vein. Only the left

gastric vein was present between the lesser curvature and the liver. The left gastric vein had an anastomosis to the below mentioned abnormal communicating branch in front of the cardiac region. The left gastric vein had several roots originating from the lesser curvature. The most distal root originated at about distal 1/3 of the lesser curvature. These several roots originating from the lesser curvature became confluent and formed the left gastric vein (circumference: 20 mm). After the left gastric vein ran about 80 mm through the lesser omentum and reached the visceral surface of the liver, it divided into left (circumference: 20 mm) and right (circumference: 14 mm) branches. The left branch directly entered the left lobe from the left side of the round ligament (umbilical vein) and became confluent with the left branch of the portal vein at a site contralateral to the junction of the venous ligament and the left branch of the portal vein. The left branch had a length of 32 mm. The right branch directly entered the quadrate lobe after it traversed the surface of the left branch of the portal vein and contacted the right side of the umbilical vein. Macroscopically, there was no anastomosis present between the right branch of the left gastric vein and other vessels. The right branch had a length of 51 mm.

3. Splenic vein (Fig. 1)

The splenic vein was formed by four roots: three roots originated from the hilum of the spleen and the remaining root originated slightly above the hilum of the spleen. The three roots starting from the splenic hilum became confluent immediately to form a single root, which then ran approximately 110 mm towards the lower right and accepted one root running from outside the hilum of the spleen. After the root further ran 55 mm and became confluent with the mesenteric veins, it formed the portal vein. The root outside the hilum accepted two short gastric veins.

4. Abnormal communicating branch (Fig. 1, 3)

Abnormal communicating branch (hereafter referred to as "communicating branch") communicated between the left renal vein and the left gastric vein. The venous communication from the kidney to the stomach was as follows. The left renal vein was composed of the superior, middle and inferior roots. First, the superior and middle roots departed from the renal hilum and became confluent. The inferior root participated in the confluent trunk and formed the left renal vein (circumference: 32 mm). The left ovarian vein was exhausted to the inferior root. The left renal vein traversed in front of the abdominal aorta at the level of L2 and L3, then entered the inferior vena cava. When the left renal vein traversed in front of the abdominal aorta 17 mm right from the point of confluence of the inferior branch, the communicating branch (circumference: 34 mm) originated from the left renal vein. The origin of the communicating branch accepted minor veins from the left adrenal gland. The communicating branch ascended 25 mm on the anterior medial side of the left adrenal gland and accepted the left adrenal vein. After further ascending 40 mm, the communicating branch formed a large varix towards the lateral (left) and was abruptly directed towards the medial (right). Here, as the communicating branch traversed the diaphragmatic muscular side of the left inferior phrenic artery, it was pressed by the artery. After the communicating branch ran 30 mm in the upper lateral (right) direction from the swollen site, it was directed left and upward again and was greatly swollen. After the communicating branch ascended 17 mm on the diaphragm, it bifurcated into two branches. One branch had a circumference of 14 mm and the other 25 mm. The narrow branch passed from the dorsal side of the fundus of the stomach to the cardiac notch and was distributed to the ventral surface of the fundus. After the thick branch passed from the cardiac notch and reached ventral side of the cardia, it was anastomosed to the left gastric vein.

5. Round ligament of the liver and paraumbilical vein (Fig. 1)

(1) Round ligament of the liver (umbilical vein)

The round ligament of the liver originating from the umbilicus ascended about 60 mm passing through the falciform ligament of the liver and gradually increased in size to reach along the right side of the well-developed paraumbilical vein. When the round ligament reached a site 120 mm upward from the umbilicus, it obliquely traversed the peritoneal side of the paraumbilical vein, while abruptly increasing in size (circumference: 13 mm). It then developed a sigmoid bend 140 mm upward of the umbilicus and became confluent (circumference: 28 mm) with the paraumbilical vein. It extended to the visceral surface of the liver immediately, and after running approximately 50 mm along the round ligament fissure of the liver, it was anastomosed to the left branch of the portal vein. It had a circumference of 32 mm inside the fissure. A clot existed in the upper round ligament of the liver 20 mm upward from the umbilicus, suggesting persistence of the umbilical vein. The venous ligament of the liver was closed and had the appearance of a fibrous string.

(2) Paraumbilical vein

A well-developed paraumbilical vein existed between the left inferior epigastric vein and the round ligament of the liver. The left inferior epigastric vein penetrated through the posterior lamella of the rectus sheath approximately at the level of the umbilicus and ascended after forming the paraumbilical vein. After reaching a site approximately 40 mm upward from the umbilicus, the paraumbilical vein abruptly developed a sigmoid bend and contacted the left side of the round ligament of the liver. As a result, the paraumbilical vein had a circumference of 14 mm. The paraumbilical vein then ascended approximately 60 mm along the left side of the round ligament of the liver (or umbilical vein) and became confluent with the round ligament (or umbilical vein).

6. Celiac trunk (Fig. 2)

No celiac trunk was observed in this cadaver. The splenic artery and the left gastric artery arose directly from the abdominal aorta. The common hepatic artery originated from the superior mesenteric artery. Details of these arteries will be described separately elsewhere.

Discussion

The major findings in the present case included the presence of well-developed communicating branch between the left renal vein and the left gastric vein, and the course and distribution of the left gastric vein. Persistence of the umbilical vein due to increased portal pressure has been described previously by Kubota *et al.* (1957), whose findings were similar to those of the present investigation.

1. Abnormal communicating branch

Yamada (1934) described a case of a 66-year-old man who had a large communicating branch which originated from the renal vein, reached the gastric cardia in the same manner as observed in the present case, accepted the left gastric vein, and ultimately entered the portal vein (trunk). Yamada (1934) only described the patient's history of gastritis and did not describe the abnormality of the liver itself.

The present case was considered to be a well-developed portal collateral pathway. The evidence was clearly demonstrated, based on a comparison with normal observations (Fig. 4). This normal case was of a non-cirrhotic 64-year-old female (cadaver No.: 94051) who died from acute cardiac failure. The findings in this case suggested that poorly-developed veins may have developed into portal collateral routes, instead of the above-discussed abnormal communicating branch. It is of note that the left inferior phrenic vein entered the left adrenal vein, which in turn entered the left renal vein. After a part of the roots of the left inferior phrenic vein became confluent in the vicinity of the left side of the hiatus esophageus, it descended and became confluent with some venous roots originating around the upper pole of the left kidney and the center of the diaphragm, which formed the left phrenic vein and was exhausted to the left adrenal vein. The left adrenal vein descended along the upper medial surface of the left adrenal gland and became confluent with the left renal vein. A branch of the inferior phrenic artery ran along each root of the left phrenic vein. The arterial branch traversed the diaphragmatic serous side of the vein to compress it.

As apparently seen from this normal case, the left inferior phrenic vein may enter the left renal vein via the left adrenal vein. According to Yamamoto *et al.* (1973), when the portal pressure is increased, a portal blood pathway passing from the left gastric vein into the azygos vein via the esophageal veins on the lesser curvature side and another pathway of the short gastric venous system originating from the upper pole of the spleen into the esophageal veins on the greater curvature side become prominent around the esophagus and the cardiac region. According to the predictions of Miyoshi *et al.* (1983), when the portal pressure is increased, esophageal varices appear prominently. Location of the phrenic artery on the phrenic vein when the vein crosses the artery means that the vein is compressed by the artery and greatly swollen. These findings suggest that the communicating branch between the left renal vein and the gastric cardiac veins might have formed a portal collateral pathway as a result of dilatation of the left phrenic vein due to increased portal pressure caused by liver cirrhosis. Recently, Sakamoto *et al.* (1997) reported similar anomaly in Japanese male, and emphasized the importance of the connection between the left gastric vein and the left inferior phrenic vein near the gastro-esophageal transitional region.

2. Left gastric vein

The finding of particular note in the present case was the direct entry of the left gastric vein into the liver (Fig. 1, 2). Anatomically, there have been four similar Japanese cases reported in the literature. Unfortunately, however, two of the four cases had an unknown configuration (Ryu, 1929 and Mori, 1941) and detailed information was obtained only in the remaining two cases (Miyaki *et al.*, 1987). According to Miyaki *et al.* (1987), the incidence of

Table 1. Number and frequency (%) of exhaustion site of the left gastric vein in Japanese

Author \ Site	V. portae	V. lienalis	Junction*	Liver	Obscure	Total
Aso[1])	32 (54.2)	21 (35.6)	6 (10.2)	0	0	59
Mori[11])	67 (47.9)	60 (42.9)	9 (6.4)	1 (0.7)	3 (2.1)	140
Rokutanda[12])	38 (47.5)	34 (42.5)	7 (8.8)	0	1 (1.3)	80
Ryu[13])	33 (66.0)	10 (20.0)	5 (10.0)	1 (2.0)	1 (2.0)	50
Total	170 (51.7)	125 (38.4)	27 (8.2)	2 (0.6)	5 (1.5)	329

Junction*: the left gastric vein empties into the junction of the lienal vein with the portal vein. (Adult: Mori[11]) and Ryu[13]), Fetus: Aso[1]) and Rokutanda[12])

the cases was 0.8% (2 out of 245 cadavers), which almost coincides with the present authors' finding of the location of the opening of the left gastric vein based on other investigators' papers (Table 1). Both of the two cases reported by Miyaki *et al.* (1987) showed direct entry of the left gastric vein into the liver via the omentum minus and an anastomosis to the branches bifurcating from the left branch of the portal vein. While the left gastric vein and normal right gastric vein were present in Case 1, the right gastric vein was absent in Case 2. In the present case, a vein corresponding to the right gastric vein was lacking and most of the veins in the lesser curvature were collected in the left gastric vein. The left gastric vein divided into right and left branches of the same size immediately before entering the liver. The left branch became confluent with the left branch of the portal vein after its entry immediately into the left hepatic lobe, whereas, the right branch was directly distributed to the hepatic quadrate lobe. There was no anastomosis between the right branch and other vessels macroscopically. These findings may indicate that the course of the left gastric vein observed in the present case was congenital, and became prominent due to increased portal pressure caused by liver cirrhosis.

There have been clinical reports describing an abnormal course and distribution of the left gastric vein, particularly anastomosis of the left gastric vein to the left branch of the portal vein (Kokubo *et al.*, 1990; Tajima *et al.*, 1992; Takayasu *et al.*, 1990). However, these report have only given angiographic findings and lacked detailed morphological descriptions.

Embryologically, the portal vein is derived from symmetrical vitelline veins. The original vitelline veins appear symmetrically, but portions of these veins are lost or they become anastomosed to each other, thereby forming a single portal vein. On the other hand, it has been reported that before the appearance of the vitelline veins, the subintestinal vein appears along the intestinal tract. Although the subintestinal vein disappears at some stage, it remains in part as the coronary vein (Miki, 1973). Partial appearance and disappearance of the bilateral vitelline veins and the morphology of the bilateral portal veins can be understood readily from the hepatic portal venous distribution in fowls. In humans, it has been reported that the direct entry of the coronary vein into the liver represents a residue of the proximal left vitelline vein, which corresponds to the left portal vein (Miyaki, 1973, 1978, 1987). According to Miyaki (1994) the residue of the left portal vein of mammals has been described in the literature in only five human cases including his two cases and one case of a Japanese bear.

As presented above, although the formation of collateral routes due to increased portal pressure should be taken into account, the macroscopic observation that part of the left gastric vein is distributed directly in the liver without forming an anastomosis to the portal system, and the lack of the right gastric vein suggest that the present case had a congenital residue of the left portal vein. Anatomically, this was the third Japanese case of this type, following the two cases reported by Miyaki *et al.* (1987).

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Abbreviations in Figures

Organs; Ap: appendix fibrosa, Di: diaphragm, Du: duodenum, Es: esophagus, Gb: gallbladder, Lg: round ligament, Lk: left kidny, Ll: left lobe of the liver, Ls: left suprarenal gland, Pa: pancreas, Rk: right kidney, Rl: right lobe of the liver, Sp: spleen, St: stomach.

Arteries; Ctr: coelic trunk, Pha: proper hepatic artery, Lpa: left inferior phrenic artery, Sma: superior mesenteri artery.

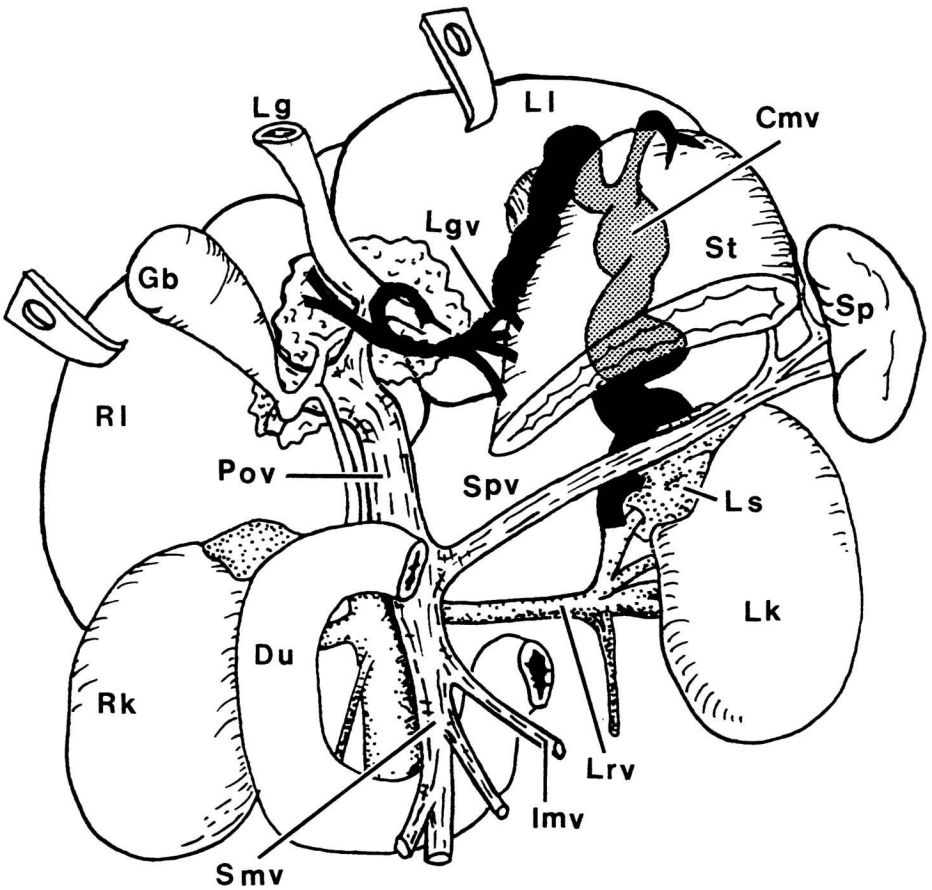
Veins; Cmv: communicating branch between the left renal and left gastric veins, Imv: inferior mesenteric vein, Lgv: left gastric vein, Lpv: left inferior phrenic vein, Lrv: left renal vein, Pov: portal vein, Smv: superior mesenteric vein, Spv: splenic vein.

Explanation of Figures

Plate I

Fig. 1. Schematic drawing of an unusual portal collateral route and aberrant left gastric vein. A rare collateral vein of the portal vein connects the left gastric vein with the left renal vein via parietal veins. The left gastric vein divided into left and right branches to enter the liver. The left branch of the left gastric vein anastomosed to the left branch of the portal vein at a site contralateral to the junction of the venous ligament and the left branch of the portal vein, whereas the right branch distributed to a part of the quadrate lobe on the right side of the hepatic round ligament (or umbilical vein).

Fig. 2. Direct entry of the left gastric vein into the liver. The left gastric vein had several roots (arrows) originating from the lesser curvature of the stomach. After the left gastric vein ran through the lesser omentum and reached the visceral surface of the liver, it divided into left and right branches to enter the liver directly.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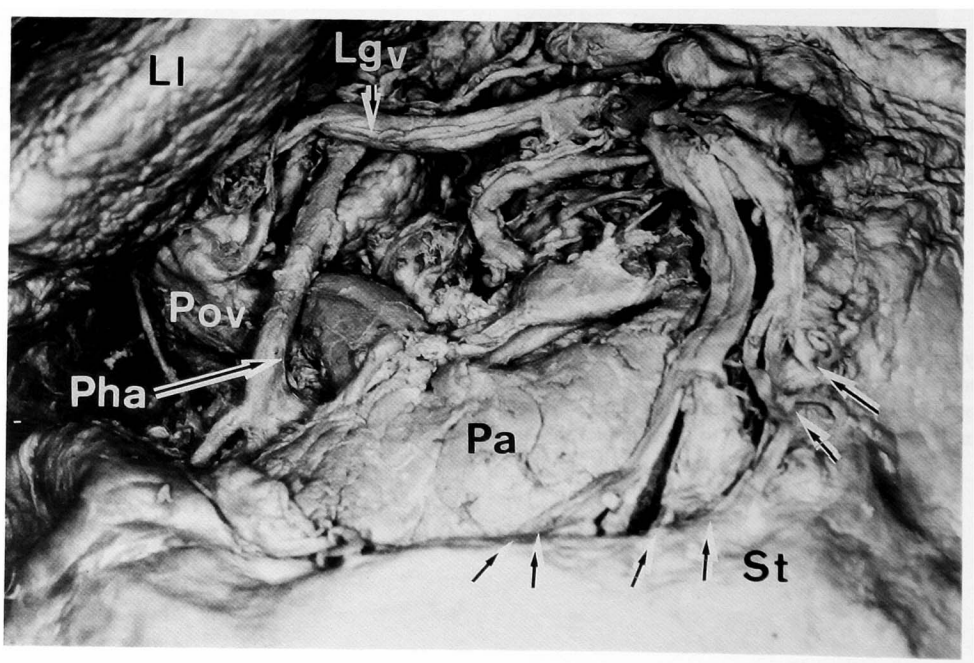
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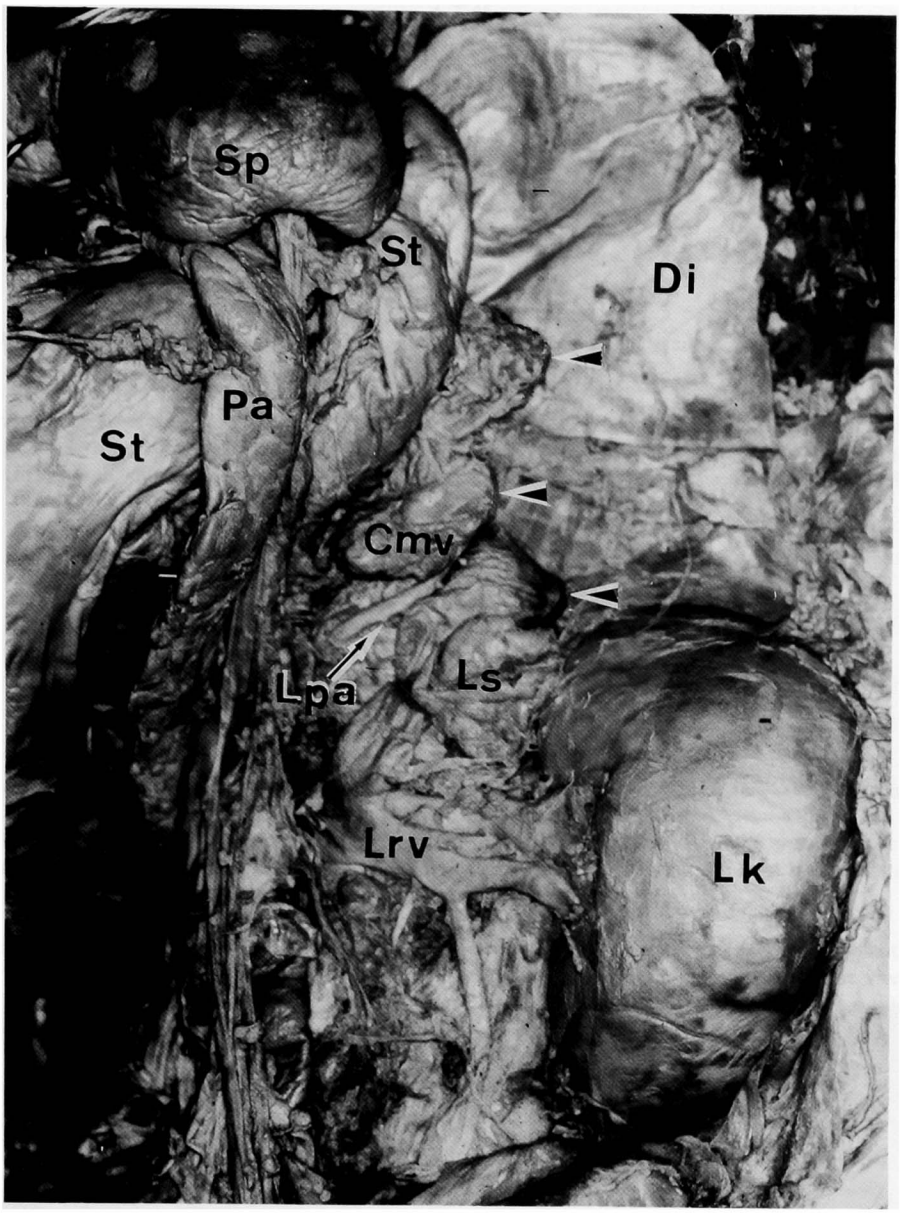

Plate II

Fig. 3. Greatly swollen communicating branch (arrow head: varicosity) between the left gastric and left renal veins. It is thought to be secondary communication and enlargement of the left suprarenal and left inferior diaphragmatic veins due to the increase of the portal pressure of liver cirrhosis. The vein is traversed by the left inferior phrenic artery. Stomach, pancreas and spleen were turned over on the right side to show the left half of the internal surface of the abdominal posterior wall.

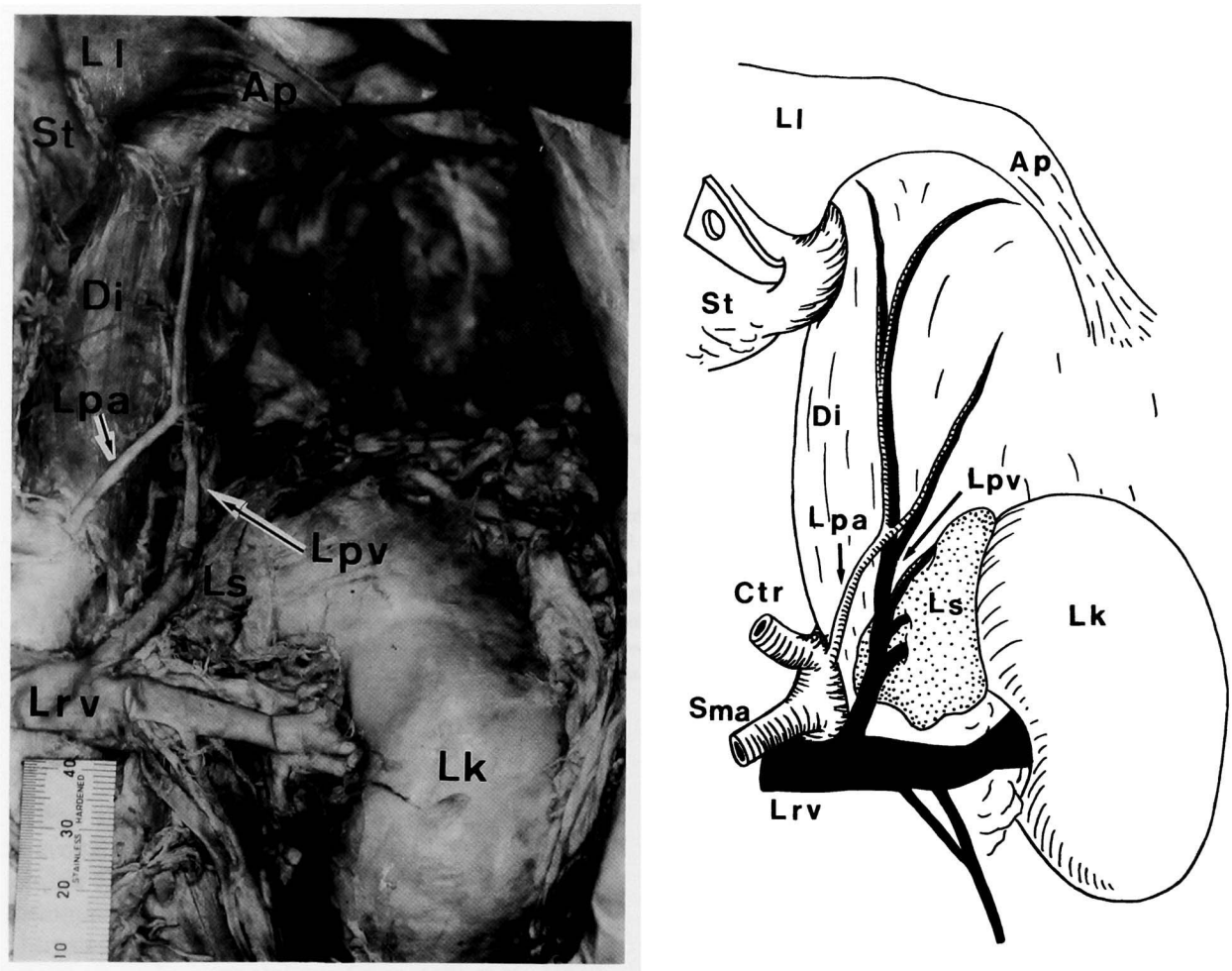

Plate III

Fig. 4. Phrenico-adrenal venous connection found in another normal case. The left adrenal vein descended along the upper medial surface of the left adrenal gland and became confluent with the left renal vein. A branch of the inferior phrenic artery traversed the diaphragmatic serous side of the vein, which suggests that the vein is compressed by the artery and greatly swollen in case of the formation of collateral routes due to increased portal pressure.