## GASTRIC VARICES WITHOUT PORTAL HYPERTENSION: ROLE OF LEFT INFERIOR VENA CAVA?

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A variant vascular anatomy was detected during regular analysis of multidetector computed tomography angiography of the abdomen in a 70 year-old female patient, referred to the department of surgery for laparoscopic right colectomy for colon cancer. The anomalous vessel was located left to the aorta, and was consistent with a persistent left inferior vena cava. It was connected by two retroaortic rootlets to the dorsal surface of the regular right inferior vena cava and had four notable tributaries – an anastomosis with the iliolumbar trunk, ovarian vein, renal vein and the inferior phrenic vein. In the upper abdomen, the left inferior vena cava took a tortuous course, passing at first between the spleen and the diaphragm, then curving below the inferior splenic border and terminating in an irregular network in the posterior region of gastric fundus and cardia, close to the splenic hilum, without supradiaphragmatic continuation. Despite this extraordinary termination, there were no signs of portal hypertension or data on previous occurrence of this condition.

Key-words: Stomach, varices - Magnetic resonance (MR), vascular studies - Venae cavae, MR.

Variations of the inferior vena cava (IVC) and its tributaries were once regarded as numerous in form and rare in incidence. These variations, however, have been more frequently reported with the advent of multislice helical CT imaging modalities. A persistent left inferior vena cava is found in 0.2%-0.5% and a double IVC in 0.2-3% of the general population in autopsy series (1-5).

The case of the gastric varices we report hereinafter deserves attention for its atypical appearance, lack of pertinent clinical etiology and the direct anatomical framework of the persistent inferior left vena cava.

## **Case presentation**

The investigation was conducted in accordance with Ethical Principles for Medical Research Involving Human Subjects (6).

A 70-year-old woman was referred to the department of surgery for colon cancer, located in the ascending colon, just proximal to the hepatic flexure. The laparoscopic right colectomy was performed, but the patient developed signs of anastomotic leakage and underwent reoperation with a resulting ileostomy. Final histopathology showed an adenocarcinoma (stage Dukes C) with metastases to local lymph nodes.

The patient had had a previous history of progressive and infiltrating

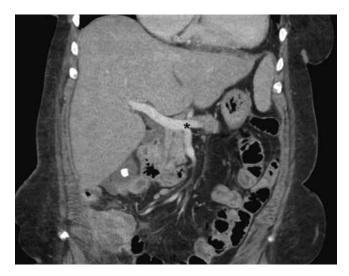


Fig. 1. — Synoptical coronal CT of the abdomen, level of the portal vein confluence (asterisk).

left-sided ovarian adenocarcinoma for which she underwent a bilateral salpingo-oophorectomy, supravaginal hysterectomy and rectosigmoid resection two years earlier, followed by a re-operation for anastomotic leakage, and a sigmoidostomy. No retroperitoneal lymphadenectomy was performed. She had been treated by adjuvant chemotherapy and had undergone introduction of a left JJ (pig-tail) stent, due to the long-standing com-

pression of the left ureter by the pelvic tumor and consequent hydronephrosis.

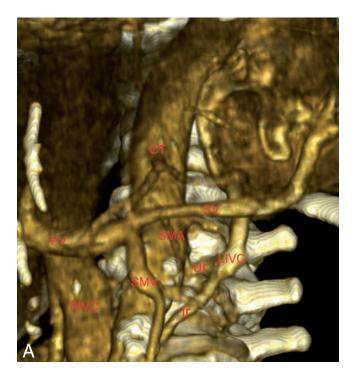
There were no biochemical, radiological or anamnestic findings suggesting clinically significant portal hypertension, such as low platelet count, splenomegaly, portal vein diameter above the upper limit, schistosomiasis, excessive alcohol intake, melena, hematemesis, etc. Therefore, the upper endoscopy was not indicated.

The pre- and postoperative helical CT was performed with a 64-detector CT scanner (Toshiba, TS\_Aquillion64). Portal-venous phase images were obtained after contrast media administration (320 mg/ml, 80 ml), using a power injector at a rate of 2.5 ml/s. The CT parameters were as follows: pitch was 0.828, tube voltage 120 kV, maximum tube current

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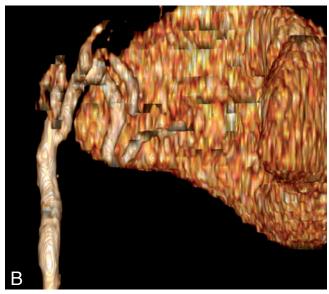


Fig. 2. — A. 3D volume rendering. CT – celiac trunk, LIVC – left inferior vena cava (ur – upper root, lr – lower root), SMA – superior mesenteric artery, SMV – superior mesenteric vein, SV – splenic vein, PV – portal vein, RIVC – right inferior vena cava. B. Detail from Fig. 2 A: arborization of the upper part of LIVC with tortuous gastric varices.

250 mAs, 3 mm reconstructed section thickness, 3 mm reconstruction interval. Analysis was performed by means of 2D multiplanar reconstruction with maximum intensity projection and 3D volume rendering technique using the Osirix algorithm.

In the later clinical follow-up, diagnostic imaging revealed a tumor in the distal ileum, highly suspicious of a local recurrence of her gastroin-

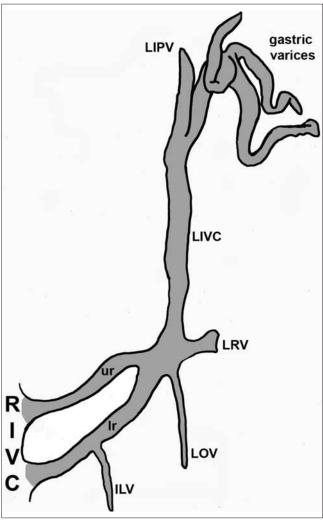


Fig. 3. — Grayscale outline of the LIVC. Captions: ILV – common trunk for iliolumbar / left ascending lumbar vein; LRV – left renal vein, LOV – left ovarian vein; LIPV – left inferior phrenic vein. Other captions as in Figure 2.

testinal cancer, with enlarged paraaortic lymph nodes. During this period the patient was also diagnosed with an aggressive leukemia and died shortly thereafter due to sepsis.

The CT disclosed a 10 mm by 10 mm gallstone in the gallbladder, otherwise the liver and the bile ducts were considered normal. The bipolar diameter of the spleen was 11.7 cm, and the calibers of the splenic vein, superior mesenteric vein and the

Table I. — Morphometry of the persistent LIVC.

	Skeletotopy	Caliber (cm)	Length (cm)
Upper root	L2 / L3	0.884	4.330
Lower root	L3 (lower half)	0.646	5.771
Trunk	L3 –T12 / L1	1.039	10.144

portal vein were 0.872 cm, 0.840 cm and 0.976 cm, respectively (Fig. 1).

Both the pre- and postoperative CTs revealed a vessel located left to the aorta, consistent with a persistent left inferior vena cava (LIVC). Two venous rootlets emerged from the dorsal surface of the regular right IVC, passed to the left retro-aortically, and merged into the trunk of LIVC (Fig. 2).

There were four notable tributaries/branches to the LIVC (Fig. 3 and 4, Table I):

- common trunk for iliolumbar / left ascending lumbar vein;
- stump of the left ovarian vein;
- left renal vein; and
- left inferior phrenic vein

The first branch (common trunk for iliolumbar / left ascending lumbar vein) emerged from the lower root and not from the LIVC trunk itself. In the upper abdomen, the LIVC took a tortuous course, passing at first between the spleen and the diaphragm, then curving below the inferior splenic border and terminating in an irregular network in the posterior region of gastric fundus, close to the splenic hilum (Fig. 2B). In relation to the gastric wall, this network was found to lie extramurally (Fig. 4). According to the Sarin classification, these gastric varices would correspond to group IGV-1. The left ureter coursed lateral and deep to the inferior portion of LIVC. There was no supradiaphragmatic continuation of the LIVC. The right IVC was unremarkable.

## Discussion

The embryogenesis of the IVC is a composite process, with some stages still uncertain or under debate. It is based on sequential formation, regression, anastomosing and fusion of multiple paired axial venous channels: postcardinal, subcardinal, supracardinal, azygos and subcentral. The double IVC derives from the persistence of both the right and left supracardinal veins. Although the left IVC is considered to be strictly subrenal (3-5, 7, 8), there are extremely rare reports of its suprarenal hemiazygos continuation (9). In the case reported by Lao et al. (10), the left vena cava crossed the midline to the right and emptied into the azygos vein.

Embryologic changes affecting the veins are considered to also induce variations in lymphatic drainage (1).

With regard to the two IVC confluence and/or anastomoses, the LIVC crosses the aorta anteriorly (5, 7, 8), or posteriorly (4), at the level of the left renal vein. A highly positioned preaortic crossing may lead to intermittent celiac trunk compression syndrome (3). Our case had two retroaortic roots connecting the right and left IVC at the levels of second and third lumbar vertebra, respectively. In addition, the number of the vertebrae was normal, and no major vascular anomalies were found except the LIVC in question. This is in accordance with the fact that the developmental anomalies of the

cardinal vein systems are relatively isolated (5, 7-9). On the other hand, the duplication of the vena cava may also be associated with a retrocaval ureter, which descends between the venae cavae and the aorta (4). In our case, no congenital anomalies of the urinary system were observed. Further, taking into account the caudocranial direction of the blood flow for the ovarian vein and the iliolumbar vein, it would be unlikely that the initial gynecological operation has elicited the development of a collateral venous channel.

The ratio between the two IVC calibers has not been often addressed in the literature, only with regard to the determination of duplication type (2) or the regression of the right IVC (5). Our case shows the persistent LIVC to have a caliber approximately one-third of its counterpart.

The most enigmatic part of our LIVC case was its cranial portion. Unlike the reports in which the persistent LIVC continues the hemiazygos course and opens into the coronary sinus (5), in our case the persistent vessel did not traverse the diaphragm. Instead, it dispersed in a network in the region of gastric fundus, resembling the MDCT picture of gastric varices (11). It has been noted that, unlike the cardiac veins and varices which are continuous with esophageal varices, fundic varices develop independently and can be very large in caliber (12). This is a consequence of the hepatopetal direction of blood flow towards the stomach and inferior diaphragmatic

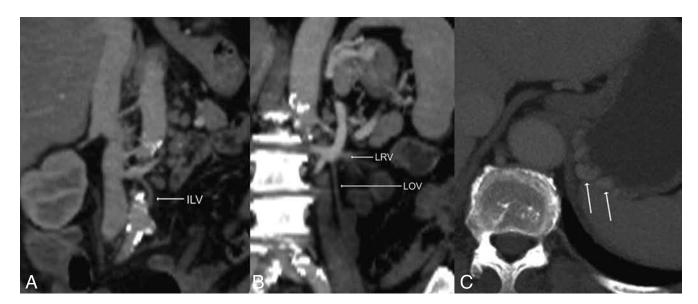


Fig. 4. — 2D multiplanar reconstruction. A) and B) coronal views; C) axial view: arrows – fundic termination of LIVC. Caption as in Figure 3.

veins, which plays a buffer role in portal hypertension (13). However, in our case there was no data or any clinical findings in favor of portal hypertension. We also considered the possibility of congenital extrahepatic portocaval shunt – the Abernethy syndrome (14), or similar malformations reported in animals (15), but, as already mentioned, there were no other abnormalities of the circulatory system.

The only plausible explanation we can offer could be an enlarged venous communication between the persistent LIVC, the left gastric/ gastroomental veins and the left inferior phrenic vein. A similar pattern, an anastomosis between an abnormally communicating enlarged vein between the left gastric vein and the esophageal tributary of the left inferior phrenic vein, which opened into the left renal vein, has been found to occur in 40% of apparently healthy specimens (16). There have been reports on portosystemic shunts involving the left inferior phrenic vein and draining into the renal vein (17), but they were intrahepatic and probably acquired through iatrogenic or traumatic episodes, or as a consequence of portal hypertension.

In the light of the fact that the number of the patients undergoing multiple complex surgical procedures is increasing, the preoperative awareness for the new forms of vascular variations and anomalies, such as the case here presented, becomes mandatory.

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