Portosystemic collaterals

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Received 12 September 2018
Revised 20 September 2018
Accepted 01 December 2018
Published 11 June 2021

Journal of Current Medical Research and Practice
2021, 6:109–117

This article aims to describe the various portosystemic collateral pathways in patients with portal hypertension on multidetector row computed tomography and their clinical importance, with special emphasis on the uncommon collaterals. This will have important implications both for the clinician and the radiologist. Multidetector row computed tomography with its advanced postprocessing abilities can demonstrate these collaterals to help in therapeutic decision making.

Keywords:
collateral pathways, multidetector row computed tomography, portal hypertension, shunt, varices

J Curr Med Res Pract 6:109–117

2357-0121

Introduction

Portosystemic collateral vessels (PSCVs) are a consequence of portal hypertension that occurs in chronic liver diseases and is responsible for numerous complications, including bleeding esophageal and gastric varices (GVs), and hepatic encephalopathy [1–3]. It was thought that collateral circulation development was due to the passive opening of vascular channels in response to portal hypertension. However, the results of Fernandez *et al.*[4] showed that the formation of portosystemic circulation may be due to angiogenesis driven by vascular endothelial growth factor [4–7].

Portosystemic collateral circulation can be classified into uncommon collateral circulation and common collateral circulation. Common collateral circulation has been studied for a long time, and it includes esophageal and GVs, abdominal and umbilical vein dilation and hemorrhoidal vein dilation [8–11]. However, there are few studies on uncommon collateral circulation, including splenorenal, gastrorenal, retroperitoneal, and cardiac angle venous shunts. Detection of these 'spontaneous' PSCVs provides an important tool in diagnosing portal hypertension and predicting prognosis [8].

A precise description of PSCV is essential to therapeutic decisions, and multidetector computerized tomography portal venography can help in determining the extent and location of PSCVs in patients with portal hypertension [9]. Information about PSCV is especially important before interventional procedures or surgery [10].

The aim of this review was to describe the common and uncommon collateral pathways in patients with portal hypertension at computed tomography examination.

Physiologically, normal portosystemic anastomoses exist (Table 1),[8] and dilatation of these channels in patients with portal hypertension leads to the formation of varices [11].

Description of the portosystemic collateral vessels

In patients with portal hypertension, dilatation of PSCV leads to the formation of varices, which are classified into two groups, the gastroesophageal varices (GOVs) and ectopic varices [8]. This is followed by the spontaneous development of large shunts in the abdomen, which can be anatomically divided into intrahepatic, transhepatic, and extrahepatic shunts (Fig. 1) [12–14].

Table 1 Normal sites of portosystemic anastomoses

Sites	Portal component	Systemic component
Lower esophagus	Left gastric vein	Esophageal veins, which in turn drain into the azygos vein
Rectum	Superior rectal vein, which is a tributary of the inferior mesenteric vein	Middle and inferior rectal veins, which are, respectively, tributaries of the internal iliac and pudendal veins
Anterior abdominal wall	Paraumblical veins, which arise from the left branch of portal vein	Superior and inferior epigastric veins
		The most common path of drainage of paraumbilical veins is through the inferior epigastric veins, which finally reach the external iliac veins
		Paraumbilical veins may also be connected with numerous subcutaneous vessels of the anterior abdominal wall, creating a 'caput medusae' pattern
Retroperitoneum	Tributaries of the splenic and pancreatic veins	Left renal vein
	Tributaries of the splenic and colic veins	The lumbar veins of the posterior abdominal wall or retroperitoneal veins
Bare area of the liver	Portal vein branches	Inferior phrenic (the vein of the diaphragm) and right internal thoracic veins
Patent ductus venosus (rare)	Left branch of portal vein	Inferior vena cava

Varices

Esophageal and paraesophageal varices

The vascular structure of the normal lower esophagus consists of intraepithelial channels, a superficial venous plexus, and deep submucosal and adventitial veins. Moreover, perforating veins exist, which connect the adventitial and deep submucosal veins [15,16].

The majority of the esophageal–cardiac area drains into the portal system via the left gastric vein; any remaining elements drain into the superior vena cava through the azygos system. Submucosal varices of the lower esophagus communicate with those of the cardia. In portal hypertension, the esophageal varices (dilated veins located within the wall of the lower esophagus) are supplied mainly by the anterior branch of the left gastric vein, and can also be supplied by the short and posterior gastric veins [9,17]. Paraesophageal veins are situated outside the wall of the esophagus and supplied by the posterior branch of the left gastric vein [9]. Esophageal varices are present in 55% of patients with liver cirrhosis at initial diagnosis [18]. Overall, 70% of gastrointestinal tract bleeding in cirrhosis is due to the rupture of esophageal varices [19].

Gastric varices

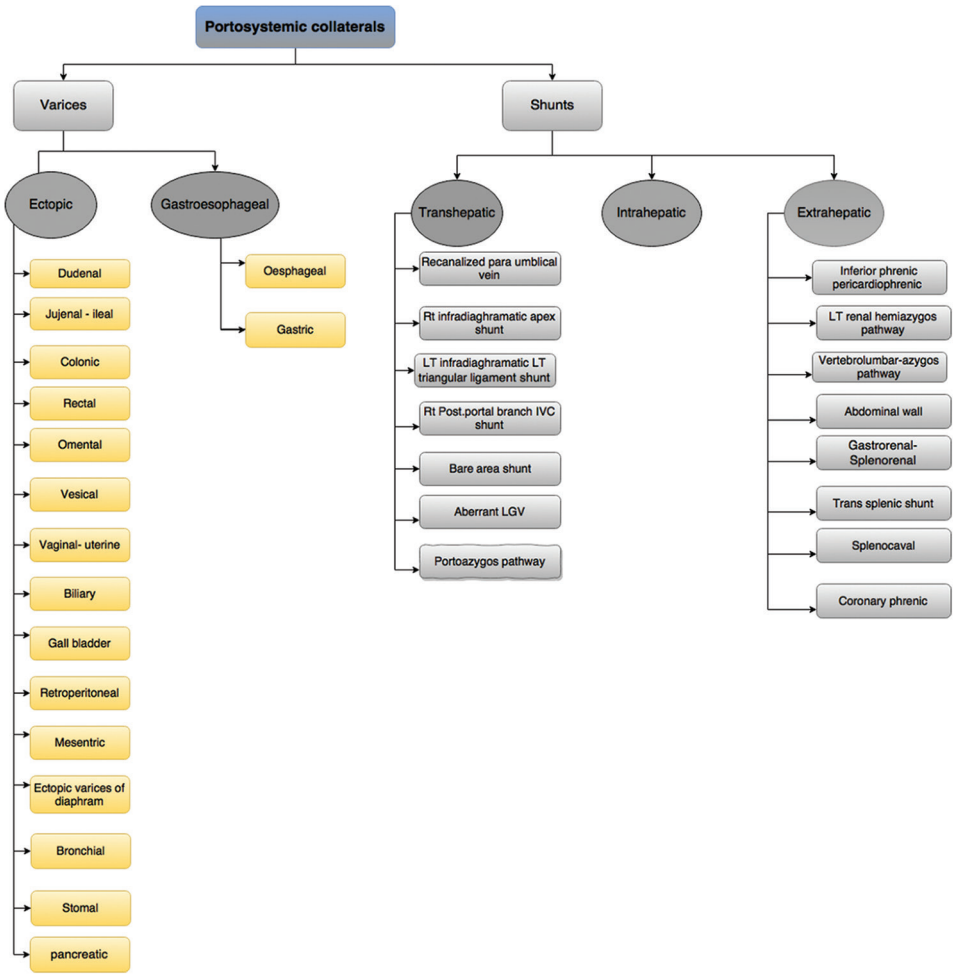
In the wall of the stomach, there are two vein plexuses: perigastric and submucosal. The perigastric plexus is located within the adventitial layer; it penetrates the muscularis mucosae and communicates with the submucosal plexus [20].

GVs are observed in 25% of patients with portal hypertension. They appear at a lower pressure gradient than esophageal varices, but they are responsible for only 10% of cases of gastrointestinal bleeding. The afferents to GVs came from the left gastric vein, short gastric veins and posterior gastric vein [8].

The short gastric veins drain the gastric fundus and the left side of the greater curvature and drain into the splenic vein. In patients with portal hypertension, the reverse flow results in fundal varices [21,22]. The posterior gastric vein is a tributary of the splenic vein and produces primarily fundic varices. In about 81% of cases, it is connected to the superior vena cava through esophageal varices. In about 23% of cases, it is connected to the inferior vena cava (IVC) via the left renal vein (gastrorenal shunt) [21].

GV were classified by Sarin [23], who divided the GV into gastroesophageal (GEV) and isolated gastric varices (IGV). GEV are mainly esophageal varices that extend beyond the esophagogastric junction and were further subdivided into GEV1, which extend for 2–5 cm along the lesser curvature, and GEV2, which extend along the fundus. IGV are not associated with esophageal varices and were divided into IGV1, which are located in the fundus, and IGV2, found anywhere other than the fundus including the body, antrum, or pylorus. GVs are supplied by the left and posterior gastric veins, less common with the short gastric veins, and rarely the gastroepiploic vein in case of surgical exclusion of other main afferent veins [8]. GEV1 are formed by the anterior branch of the left gastric vein, which penetrate the gastric wall at the level of the cardia. GEV2 and IGV1 are usually fed by the short gastric and posterior gastric veins and commonly drain into the esophageal or paraesophageal veins (84%) [8]. In some cases, they drain into the left renal vein by way of gastrorenal shunt, or directly into the IVC through a gastrocaval shunt via the left inferior

Figure 1



Portosystemic collaterals. IVC, inferior vena cava; LGV, left gastric vein.

phrenic and pericardiophrenic vein [8,24,25]. Other smaller venous pathways include ascending lumbar vein, perivertebral venous plexus, intercostal veins, and rarely, the azygos vein [8].

Ectopic varices

Ectopic varices are defined as dilated portosystemic collateral veins that are located in unusual sites other than the gastroesophageal region [26]. Ectopic varices account for 2–5% of gastrointestinal tract variceal bleeding [27–29]. They have a four-fold increased risk of bleeding when compared with esophageal varices and can have a mortality rate as high as 40% [28–32].

Ectopic varices are potentially fatal due to difficulty in controlling the bleeding. Treatment strategies include medical (systemic vasopressin and octreotide) and endoscopic therapy (banding/ligation and injection therapy), decompression using TIPS and partial splenic artery embolisation, antegrade/retrograde obliteration and surgical ligation [27–30,32–35]. Unfortunately, all of them have poor prognosis. Thus, the early diagnosis and therapy of these varices are very important.

Duodenal varices

These are a consequence of intrahepatic portal hypertension and occur in 1–3% of all cases [8]. The most common sites of duodenal varices (DVs) are in the first and second part of the duodenum and less commonly in the distal duodenum [36]. The bleeding from DV is rare due to their smaller diameter, shorter length and deeper location on the outer wall of the duodenum [8]. The afferent vessel includes the superior and inferior pancreaticoduodenal veins, cystic branches of the superior mesenteric veins (SMVs), gastroduodenal vein, and pyloric vein [23,36]. The efferents flow hepatofugally via retroperitoneal shunts (also called veins of Retzius) into the IVC via the following vessels:

(1) Right renal vein (mesenterorenal shunt).
(2) Gonadal vein (mesenterogonadal shunt).
(3) Lumbar veins.
(4) Iliac vein.
(5) Right suprarenal vein.

(6) Right inferior phrenic vein.
(7) Tributary of right renal vein – right inferior adrenal vein.

DVs can also be drained by the subcostal vein and the ascending lumbar vein into the vertebrolumbar–azygos pathway and SVC.

Jejunal and ileal varices

They commonly occur after abdominal surgery [35], and they develop due to collateral circulation through postoperative adhesions between the jejunum or ileum and the abdominal wall. Adhesions bring the parietal surface of the viscera in contact with the abdominal wall, and portal hypertension results in the formation of varices [35,36]. However, they may occur without any prior history of surgical interventions. The afferent vessels include the jejunal and ileal veins (tributaries of the SMV), and the efferents generally drain into the abdominal wall or the veins of Retzius [9,35].

Colonic varices

Colonic varices are found in a segmental distribution, mainly located in the cecum and the rectosigmoid region [8]. Isolated varices of the colon are rare. Recognition of this condition is very important, as colonic varices may be an infrequent cause of massive lower gastrointestinal bleeding [37].

Afferents to colonic varices include the following veins:
(1) Ileocolic vein.
(2) Middle colic vein.
(3) Right colic vein.
(4) Sigmoid colonic vein.

Efferents can drain into veins of Retzius, which include the following vessels:
(1) Right gonadal vein.
(2) Right renal vein.
(3) Systemic lumbar veins.
(4) A part of the veins of the ascending colon drain via the right renal capsular vein into the IVC [38].

Rectal varices

Afferents to rectal varices: Afferent to rectal varices is the superior rectal vein [tributary of inferior mesenteric vein (IMV)] [8]. The blood from the superior rectal vein goes to the extrinsic rectal venous plexus, which lies outside the rectum below the level of peritoneal reflection. From the extrinsic rectal venous plexus, the blood flows by perforators into the intrinsic rectal venous plexus (IRVP), which consists of two groups of veins – the superior and inferior groups. The superior group of veins form the zone of internal hemorrhoids in the submucosa and pass up for a distance of about 10 cm above the pectinate line to form the superior rectal vein. The rectal varices are formed from this superior group of upper submucosal veins of IRVP. The inferior group of IRVP lying in the anal subcutaneous tissue passes down to form the inferior rectal vein and contributes to the formation of external hemorrhoids [39].

Efferents from rectal varices: Efferents from the rectal varices are the middle and inferior rectal veins (tributaries of the internal iliac) [1].

Omental collateral vessels

Afferents to omental varices: Afferents to the omental varices are the superior or IMVs.

Efferents from omental varices are as follows:
(1) The retroperitoneal or pelvic veins.
(2) Omental veins may also drain into GEV.

Rupture of the omental varices has a fatal prognosis [23].

Vesical varices

Vesical varices are rare in patients with portal hypertension because the bladder wall is an unusual collateral route for the venous splanchnic blood. Generally, reported cases of vesical varices have a history of abdominal surgery [39–41].

Vaginal and uterine varices

Venous drainage of the vagina and uterus

The uterus has an extensive venous plexus; these vessels are unlikely to develop varices. The vagina also has a venous plexus, which drains into the internal iliac vein via the bilateral vaginal veins. The plexuses are in communication with each other and with the vesical and hemorrhoidal plexuses.

Afferent to vaginal and uterine varices

The afferent to the vaginal and uterine varices is the superior portion of the hemorrhoidal plexus [42,43].

Efferents from vaginal and uterine varices

The efferents from the vaginal and uterine varices are the venous plexuses of the uterus and vagina, as well as the internal iliac vein and uterine veins.

Biliary varices

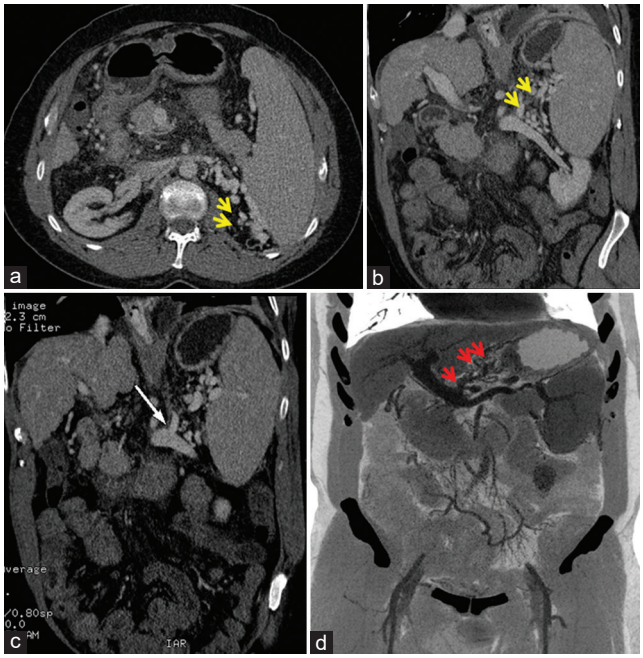
Two preformed venous systems near the extrahepatic bile ducts contribute to the formation of biliary varice:

the paracholedochal (PACD) veins of Petren, and the epicholedochal (ECD) venous plexus of Saint. The PACD plexus runs parallel to the common bile duct (CBD), and the ECD plexus forms a reticular mesh on the surface of the CBD [44,45]. Early in portal hypertension, engorgement of the PACD veins of Petren and the ECD venous plexus of Saint occur. Dilatation of the PACD collaterals may cause extrinsic compression and protrusion into the thin CBD, whereas dilatation of the ECD collaterals may make the normally smooth intraluminal surface of the CBD irregular [46,47]. In portal hypertension, dilatation of the PACD veins will occur first, and ECD varices have not been described without the accompanying PACD varices [48]. Endoscopic ultrasound scan is the investigation of choice in tracing the origin, caliber, entry, and course of intracholedochal varices throughout the CBD [49].

Gallbladder varices

Gallbladder varices are dilated tortuous vascular structures around or within the gallbladder wall. They present in about 12% of patients with portal hypertension but are more frequent in those with extrahepatic portal hypertension (30%) [49]. They link the cystic vein branch of the portal system to the systemic anterior abdominal wall collaterals. They may also be associated with portal cavernous transformations such as a bypass around a focal portal vein thrombosis. Gallbladder varices may be due to back-pressure within the portal venous system [49,50].

Figure 2



Multislice computed tomography angiography axial and coronal (a–c) and coronal maximum intensity projection show dilated tortuous splenorenal collaterals (yellow arrows) that drain into the inferior vena cava through the left renal vein (a and b) causing splenorenal shunt (white arrow). Dilated left gastric vein with dilated perigastric (c) collaterals are also seen (red arrows) (d).

Retroperitoneal shunts

Retroperitoneal shunts develop from the mesenteric or colic veins and drain into the renal veins or directly into the IVC. Collaterals may develop in the prepancreatic, perisplenic, perianal, paravertebral, and retrocaval areas. There is an increase in the risk of hepatic encephalopathy, as these collaterals have a large diameter. However, gastrointestinal bleeding is not a common complication [8].

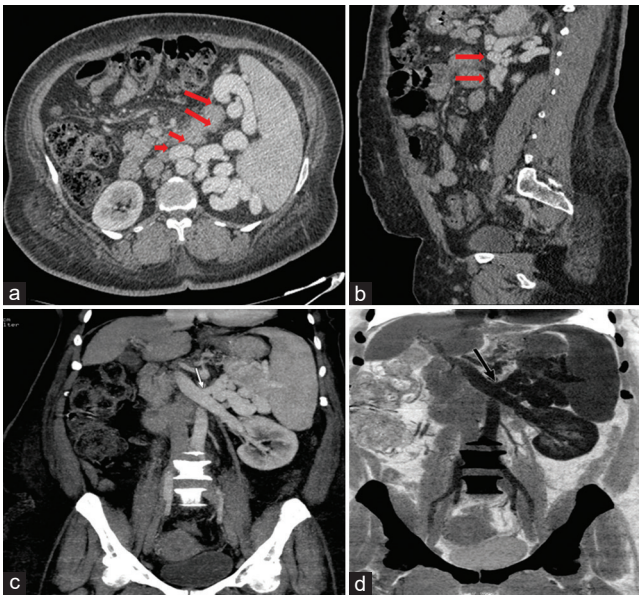
Mesenteric collaterals

Afferent vessels are branches of the SMV and IMV. Efferent vessels are the retroperitoneal or pelvic veins (also called the veins of Retzius) that drain into the IVC. Various pathways of veins of Retzius are defined according to the receiving vein (mesentericogonadal/renal/caval or iliac) [51,52].

Bronchial varices

Bronchial varices develop through collateral channels that normally exist between the tracheal and esophageal venous systems [53]. There are three case reports in bronchial varices secondary to portal hypertension [53–55]. Two of these were in patients with alcoholic liver cirrhosis and esophageal varices [54,55], whereas the third was secondary to

Figure 3



Multislice computed tomography angiography axial, sagittal, and coronal (a–c) and coronal MIP (d) show dilated tortuous splenorenal collaterals (red arrows) that drain into the inferior vena cava through the left renal vein causing a splenorenal shunt (black and white arrows). Shrunken liver, splenomegaly, no ascites.

extrahepatic portal vein stenosis [53]. All the cases presented with hemoptysis.

Anastomotic and stomal varices

In case of chronic portal vein thrombosis, collaterals usually develop through the hepaticoduodenal ligament, resulting in the formation of a portal cavernoma. In case of previous hepatobiliary surgery, formation of the classical portal cavernoma can be prevented by the surgical dissection of preformed primitive vascular structures in the hepatoduodenal ligament [8]. In this case, collateral channels can develop at unusual locations as portoportal varices in patients with enteroenteric anastomosis[56] and dilated communicating channels between jejunal veins and intrahepatic portal vein branches in patients with hepaticojejunostomy [57].

Stomal varices occur in case of surgically created bowel stomas due to communication between the high-pressure portal venous network of the mesentery and the low-pressure network of systemic veins in the abdominal wall.

Ectopic varices of the diaphragm

Cardiophrenic varices, particularly on the right side, are usually located at a cardiophrenic angle, and their rupture is rare.

Afferent to diaphragmatic varices

Cardiophrenic varices are collaterals from the paraumbilical vein.

Efferent from diaphragmatic varices

The efferent from the diaphragmatic varices is the internal mammary vein.

Shunts

Intrahepatic collaterals

The causes of intrahepatic collaterals are as follows:

(1) Congenital due to persistent embryonic venous anastomoses.
(2) Acquired due to cirrhosis, traumatic episodes, or rupture of a portal venous aneurysm into a hepatic vein.

Park *et al.*[58] classified them into the following types: (i) single tubular shunt connecting the right portal vein to the IVC (most common type), (ii) localized peripheral shunt in which one or more communications are found in a single hepatic segment, (iii) portosystemic shunt through a portal vein 'aneurysm', and (iv) multiple communications between peripheral portal and hepatic veins in several segments.

Transhepatic

Transhepatic PSCV involve intrahepatic branches of the portal vein that communicate with a systemic vein outside the liver, including the IVC, coronary vein, vertebral plexus, and hemiazygos vein [11]. Sappy described vessels that play an important role in the origin of transhepatic portosystemic shunt [11]. The different locations are as follows:

(1) Upper part of falciform ligament (superior veins of Sappey).
(2) Lower part of falciform ligament (inferior veins of Sappey).
(3) Ligamentum teres in the central part of falciform ligament (the recanalized umbilical vein).
(4) Left triangular ligament (left inferior phrenic vein and intercostal vein).
(5) Right triangular ligament (right inferior phrenic vein).
(6) Gastrohepatic omentum (cystic veins and branches of left gastric veins).
(7) Bare area of liver (diaphragmatic veins).
(8) Ligamentum venosum (patent ductus venosus, if present).

Recanalized paraumbilical veins

Afferent to umbilical varices: In patients with portal hypertension, multiple paraumbilical vessels can arise from the left portal vein. They follow the tract of the falciform ligament and then accompany the teres ligament to the umbilicus.

Efferents from umbilical varices: The efferents of umbilical varices are the superior and inferior epigastric veins. The inferior epigastric veins drain into the external iliac veins. Paraumbilical vessels may also anastomose with internal thoracic veins and drain into the superior vena cava. In rare cases, paraumbilical veins may also be connected with numerous subcutaneous vessels of the anterior abdominal wall, creating a 'caput medusae' pattern or may drain into the femoral vein [59].

Right infradiaphragmatic shunt/apex-type shunt

In this shunt, the collateral vein arising from a peripheral branch of left portal vein drains into the internal thoracic vein and the intercostal vein and reaches the right heart via the brachiocephalic vein [11,60]. This vein is also called the superior vein of Sappey [11].

Left infradiaphragmatic shunt/left triangular ligament shunt

The collateral vein arising from the peripheral portal branch of the left lateral segment communicates with the left inferior phrenic vein at the left triangular ligament, and it drains into the IVC or the left renal vein through the intercostal vein or the left pericardiophrenic veins [11,60].

Right posterior portal branch-inferior vena cava shunt

Collateral vessel arising from the right posterior portal vein runs across the posterior surface of the liver, into the IVC directly or through the adrenal vein [9,11–60].

Bare area shunt

The peripheral branch of the right posterior portal vein runs across the surface of the liver and drains into the intercostal vein or the right inferior phrenic vein [11].

Aberrant left gastric vein draining into the left portal vein

The left gastric vein drains most of the esophageal–cardiac area and a part of the fundic area before joining the portal vein. Aberrant veins may drain directly into the main left portal vein. In the presence of portal hypertension, the left gastric vein acts as a hepatofugal collateral carrying the blood from the portal vein to the systemic circulation; however, in splenic vein obstruction, it acts as a hepatopetal collateral. It has two branches [17]:

(1) An anterior branch that penetrates the cardia (2–3 cm below the gastroesophageal junction) and divides into submucosal branches that house cardiac varices that are contiguous to the submucosal varices of the lower part of the esophagus.
(2) A posterior branch that feeds paraesophageal varices: In 78% of cases, the left gastric vein connects to the superior vena cava via the esophageal and paraesophageal varices. In 12% of cases, the IVC is attained via the left inferior phrenic vein, the adrenal vein, then the renal vein (gastrorenal shunt). Exceptionally, the left gastric vein may anastomose directly to the IVC [21,61].

Portoazygos shunt

It is a communication between the main portal and the azygos veins. Only a solitary case report is available. In this report, the shunt presents between the posterior aspect of the main portal vein and the azygos vein along the right aspect of the thoracolumbar vertebrae [62].

Extrahepatic collaterals

Inferior phrenic–pericardiophrenic collaterals

The right inferior phrenic vein drains directly into the IVC, whereas the left inferior phrenic vein has two branches: one ending in the left renal vein and the other ending in the IVC. In about 75% of cases, the ostia of the left hepatic vein and left inferior phrenic veins are close to each other, or the former terminates at the latter [63]. The inferior phrenic–pericardiophrenic collaterals can be formed via both intrahepatic and extrahepatic collaterals in Budd–Chiari syndrome, wherein the intrahepatic collaterals come via the subcapsular collaterals, and the extrahepatic collaterals come via the left renal vein. The pericardiophrenic vein has diaphragmatic branches that anastomose with the inferior phrenic vein and in case the blockage extends above the joining of inferior phrenic veins in IVC forming inferior phrenic–pericardiophrenic collaterals [64,65].

Left renal–hemiazygos pathway

Multiple vessels join the left renal vein (the inferior phrenic, capsular, adrenal, and gonadal veins); hence, it is more complex than the right. The left renal vein also communicates with the retroperitoneal vein by the lumbar, ascending lumbar, and hemiazygos veins in two-third of cases [63].

Vertebrolumbar–azygos pathway

In patients with Budd–Chiari syndrome, venous flow within the IVC reverses to the common iliac vein and then continues through the ascending lumbar vein, anastomosing with the azygos system. Flow may reverse through the left renal vein to the gonadal, ureteric, or capsular vein (perirenal collaterals), onward to the iliac vein, and then to the vertebral venous system. The flow may also be directed through the intravertebral, paraspinal, and extravertebral plexuses (Batson's plexus). The ascending lumbar veins parallel the spine as they ascend through the retroperitoneum [63].

Abdominal wall

The inferior epigastric, circumflex iliac, and superficial epigastric veins form the superficial collateral system of the abdominal wall. The inferior epigastric vein anastomoses with the superior epigastric vein and with the internal mammary vein to reach the subclavian vein. Flow may follow the common femoral vein through the superficial circumflex iliac vein to the superficial epigastric vein anastomosing with the lateral thoracic vein [66,67].

Gastrorenal and splenorenal shunts

Gastrorenal shunts form between the gastric and perigastric varices and the left renal vein, generally through the left inferior phrenic vein. Rupture of GVs expose the patient to a risk of digestive hemorrhage (Figs. 2 and 3).

Direct splenorenal shunts are described between the inferior pole of the spleen and the external side of the kidney. They constitute a direct communication between the splenic vein and the left renal vein. They are not associated with gastrointestinal bleeding; thus, they are considered to be less life threatening. However, enlarged shunts are significantly associated with hepatic encephalopathy [68].

Indirect splenorenal shunts: It is indirect communication between the splenic vein and the left renal vein through the short and posterior gastric veins. The flow coming from the gastric and perigastric varices is drained into the renal vein through the left inferior phrenic then adrenal veins (splenogastrophrenoadrenalorenal shunt). Sometimes, the shunt bridges directly to the adrenal vein, bypassing the gastric area (splenoadrenalorenal shunt). These shunts create a risk of digestive hemorrhage by rupture of GVs [69].

Trans-splenic shunt

Trans-splenic shunts are extremely rare, with only two published case reports. One of these was in an adult patient with compensated cirrhosis and may be associated with intrasplenic collaterals [70].

Splenocaval/phrenic/azygos shunt

The splenic vein or the perisplenic collaterals communicate with the hypogastric vein and ultimately drain into the IVC (splenocaval shunt). The splenic vein can also communicate with the left inferior phrenic vein, hemiazygos vein or the posterior abdominal wall veins [59].

Coronary/splenic-inferior pulmonary/inferior phrenic/intercostal veins

The left gastric vein or splenic vein communicates with the inferior pulmonary vein, pericardiophrenic vein or with the intercostal vein [59].

Financial support and sponsorship

Nil.

Conflicts of interest

None declared.

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