Collateral Pathways in Portal Hypertension

Malay Sharma*, Chittapuram S. Rameshbabu**

*Jaswant Rai Speciality Hospital, Saket, Meerut 250 001 and **Dept. of Anatomy, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh, India

Presence of portosystemic collateral veins (PSCV) is common in portal hypertension due to cirrhosis. Physiologically, normal portosystemic anastomoses exist which exhibit hepatofugal flow. With the development of portal hypertension, transmission of backpressure leads to increased flow in these patent normal portosystemic anastomoses. In extrahepatic portal vein obstruction collateral circulation develops in a hepatopetal direction and portoportal pathways are frequently found. The objective of this review is to illustrate the various PSCV and portoportal collateral vein pathways pertinent to portal hypertension in liver cirrhosis and EHPVO. (J Clin Exp Hepatol 2012;2:338–352)

Almost any vein in the abdomen may serve as a potential collateral channel to the systemic circulation. Presence of abnormal collateral vessels appears to be one of the most sensitive (70–83%) and specific sonographic signs for the diagnosis of portal hypertension.[1] When blood flow through a vessel or a vascular bed is obstructed due to occlusion, as in EHPVO, or distortion, as in liver cirrhosis, collateral pathways open up as blood bypasses the occlusion or obstruction, always flowing down a pressure gradient from a high pressure to a low-pressure vessel or bed. The formation of portosystemic pathways occurs due to reopening of collapsed embryonic channels or reversal of the flow within existing adult veins.[2,3] The number of collateral channels depends on the severity of portal hypertension *i.e.* the differential gradient driving the flow between the portal and the systemic circulations and the duration of portal hypertension. The more severe and more prolonged the portal hypertension, the higher are the number of portosystemic pathways. However, this traditional hypothesis has been challenged and it has been suggested that the formation of portosystemic collateral circulation may be due in part to angiogenesis driven by vascular endothelial growth factor.[4,5]

Almost 50 years have passed since the first detailed description of portosystemic collateral veins (PSCV) in portal hypertension appeared.[6] Precise mapping of PSCV is essential to therapeutic decisions and multidetector computerized tomography (MDCT) is sometimes used for cartography of PSCV.[7] EUS is increasingly being used for evaluation of PSCV and additionally provides an option for performing therapeutic interventions.[8] In addition to PSCV, PPCV pathways are frequently found in extrahepatic portal vein obstruction (EHPVO). The objective of this review is to illustrate the various PSCV and PPCV pathways pertinent to portal hypertension in liver cirrhosis and EHPVO.

NORMAL PORTOSYSTEMIC ANASTOMOSES

The left gastric vein (LGV) anastomoses with the esophageal veins, which in turn drain into the azygos vein (AV). The superior rectal vein (SRV) anastomoses with the middle and inferior rectal veins (IRV), which are, respectively, tributaries of the internal iliac and the pudendal veins. The paraumbilical vein (PUV) anastomoses with subcutaneous veins in the anterior abdominal wall. In the retroperitoneal region, tributaries of the splenic and pancreatic veins anastomose with the left renal vein. Short veins also connect the splenic and colic veins to the lumbar veins of the posterior abdominal wall. The veins of the bare area of the liver also communicate with those of the diaphragm, as well as the right internal thoracic vein.[9]

THE DIRECTION OF COLLATERAL FLOW

The direction of collateral flow is always to bypass the occluded portion of the vessel. When vascular obstruction

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Address for correspondence: Dr. Malay Sharma, Gastroenterologist, Jaswant Rai Speciality Hospital, Saket, Meerut 250 001, Uttar Pradesh, India.
Tel.: +91 9837031148 (mobile)
E-mail: sharmamalay@hotmail.com
Abbreviations: PSCV: portosystemic collateral veins; EHPVO: extrahepatic portal vein obstruction; PPCV: portoportal collateral vein; PV: portal vein; SV: splenic vein; SMV: superior mesenteric vein; AV: azygos vein; SRV: superior rectal vein; IRV: inferior rectal veins; GEV: gastroepiploeic vein; LGEV: left gastroepiploic vein; RGEV: right gastroepiploic; GV: gastric varices; DV: duodenal varices; LGV: left gastric vein; RGV: right gastric vein; PSPDV: posterior superior pancreatico duodenal vein; AIPDV: anterior inferior pancreatico duodenal vein; PIPDV: posterior inferior pancreatico duodenal vein; IPDV: inferior pancreatico duodenal vein; MCV: middle colic vein; ASPDV: anterior superior pancreatico duodenal vein; IVC: inferior vena cava; SVC: superior vena cava; SMV: superior mesenteric vein; IMV: inferior mesenteric vein; ERVP: extrinsic rectal venous plexus; IRVP: intrinsic rectal venous plexus; PHB: portal hypertensive biliopathy; PACD: paracholedochal; ECD: epicholedochal; CBD: common bile duct; GT: gastrocolic trunk; FJT: first jejunal trunk; LPV: left portal vein; CT: computed tomography; US: ultrasonography; PVT: portal vein thrombosis; PUV: paraumbilical vein; BCS: Budd–Chiari syndrome
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is intrahepatic, collateral vessels drain away from the liver (hepatofugal collateral circulation). When the obstruction is extrahepatic, the collateral circulation usually develops toward the portal vein beyond the site of obstruction and thus drains toward the liver (hepatopetal collateral circulation). However in cirrhosis hepatopetal pathways can be present and in EHPVO hepatofugal pathways can be found.

ORDER OF APPEARANCE OF COLLATERAL

The vascular structure of the bowel wall is complex. There are intraepithelial channels, a superficial venous plexus, and deep submucosal and adventitial veins. In addition, perforating veins connect the adventitial and deep submucosal veins. Backpressure transmitted through the tributaries of the portal vein results in the engorgement of the collaterals outside the gut wall in a para-esophageal, para-gastric para-rectal or paracholedochal location.[10–12] In turn, this is followed by dilatation of veins on the surface of the visceral wall in a peri-esophageal, peri-gastric or perirectal location. Presence of perforating veins allows the transmission of this backpressure to the 'backwaters' of the gut wall and results in the formation of varices in a submucosal or subepithelial location. These submucous veins are, thus, the first sites of 'bloodlogging' and become varicose before those upon the outer surface of esophagus in portal hypertension (Figure 1).[13]

In patients with oesophageal varices dilated deep intrinsic veins displace the superficial venous plexus, assume a subepithelial position and are easily seen on endoscopy as the red color sign on varices *i.e.* telangiectasiae, cherry red spots, hemocystic spots and red wale markings or as the varices themselves. PSCV present in the peri-esophageal, peri-gastric and perirectal areas are easily seen by EUS.[14–18] Veins on the mucosal aspect can cause gastrointestinal luminal bleeding and those outside the wall may cause extraluminal *i.e.* pleural or peritoneal bleeding.[19,20]

CLASSIFICATION OF COLLATERAL PATHWAYS

The simplest classification of PSCV puts esophagogastric varices into one group and all other varices as

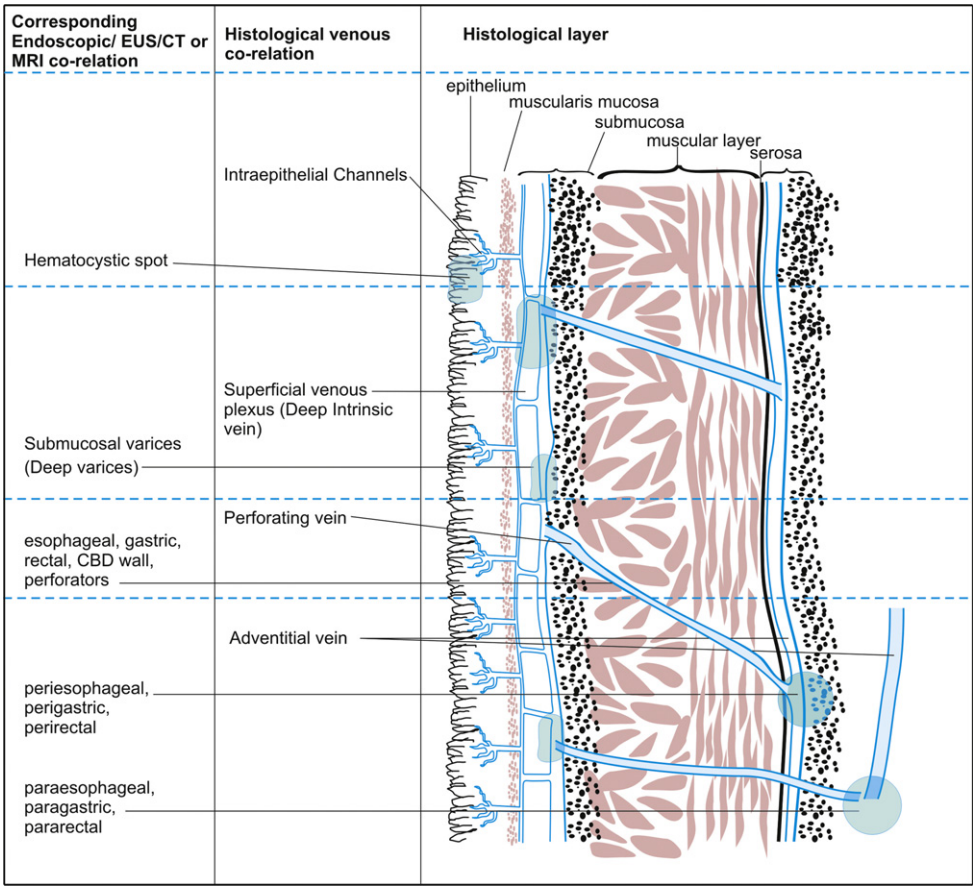

Figure 1 Venous anatomy in portal hypertension. Backpressure results in formation of varices on or outside the wall of the gut. The varices outside the wall are called para-in location and varices adjacent to the muscular layer are called peri-in location. From these locations the blood travels through the muscular layer via perforators to the deep intrinsic veins, which lie in submucosa. The hematocystic spots represent focal weakness on the variceal wall.