





Imaging Approach to Portal Hypertension

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Abstract

Increase in portal venous pressure (termed portal hypertension) is seen in a variety of liver diseases. Imaging tests are useful to detect portal hypertension and identify its cause. Noninvasive tests like abdominal ultrasound and Doppler studies are routinely done in clinical practice for this indication. Cross-sectional studies like computed tomography and magnetic resonance imaging are especially useful to delineate morphological abnormalities in the liver. Invasive tests like assessment of hepatic venous pressure gradient are done less frequently for specific indications. Distinctive imaging findings help differentiate the different causes of portal hypertension like cirrhosis and vascular liver disorders like noncirrhotic portal hypertension, extrahepatic portal venous obstruction, and Budd–Chiari syndrome. Radiological interventions are increasingly used to treat complications of portal hypertension like refractory ascites or refractory bleeding from gastroesophageal varices.

Keywords

- ▶ Portal hypertension
- ▶ Noncirrhotic portal hypertension
- ▶ Budd–Chiari syndrome
- ▶ Liver elastography

Definition

Portal hypertension (PHT) is defined as increase in portal venous pressure due to increase in vascular resistance or increased blood flow or both, resulting in hepatic venous pressure gradient (HVPG) >5 mm Hg.¹ HVPG >10 mm Hg is associated with ascites, portosystemic collaterals, varices, and risk of hepatocellular carcinoma (HCC) and HVPG >12 mm Hg, with increased risk of variceal bleed.¹

Etiology of Portal Hypertension

The etiology of PHT can be classified according to the anatomical location of abnormality into prehepatic, intrahepatic, and posthepatic causes. Intrahepatic causes are further classified into presinusoidal, sinusoidal, and post-sinusoidal causes. Important causes of PHT are summarized in ▶ **Table 1**.¹

Intrahepatic Causes

Cirrhosis

Cirrhosis is advanced stage of liver disease, with histological fibrosis, necrosis, inflammation, and regenerative nodules (RNs) resulting in altered liver morphology.²

Imaging findings in cirrhosis include distinct hepatic and extrahepatic characteristics.³ Hepatic abnormalities include morphological changes and a gamut of focal liver lesions.

Ultrasound and Doppler

Ultrasound (US), which is often the initial imaging modality, shows coarse echotexture, small-sized liver with irregular nodular surface (▶ **Fig. 1A**), blunt edge, and volume redistribution. Typically, caudate (▶ **Fig. 1B**) and lateral liver segments undergo hypertrophy, while the medial segment and right lobe undergo atrophy; however, any combination can occur.² A micronodular liver surface caused by small nodules <3 mm is common with alcoholic liver disease. A macronodular liver

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Table 1 Causes of portal hypertension¹

Prehepatic	Intrahepatic	Posthepatic
	<i>Presinusoidal</i>	
Extrahepatic portal vein thrombosis	Schistosomiasis	Budd–Chiari syndrome
Extraluminal obstruction of the portal vein (neoplasia/lymph nodes/other causes)	Primary biliary cirrhosis	Congenital webs
Splanchnic arteriovenous fistula: congenital	Noncirrhotic portal fibrosis (NCPF)	Right heart failure
Splenic vein thrombosis	Cystic fibrosis	Constrictive pericarditis
Congenital portal vein atresia/stenosis	<i>Sinusoidal</i>	IVC neoplasm
	Cirrhosis	
	Nodular regenerative hyperplasia	
	Infiltrative disorders (lymphoproliferative/myeloproliferative)	
	Congenital hepatic fibrosis	
	<i>Postsinusoidal</i>	
	Veno-occlusive disease	
	Graft vs. host disease (GVHD)	
	Drugs/toxins	

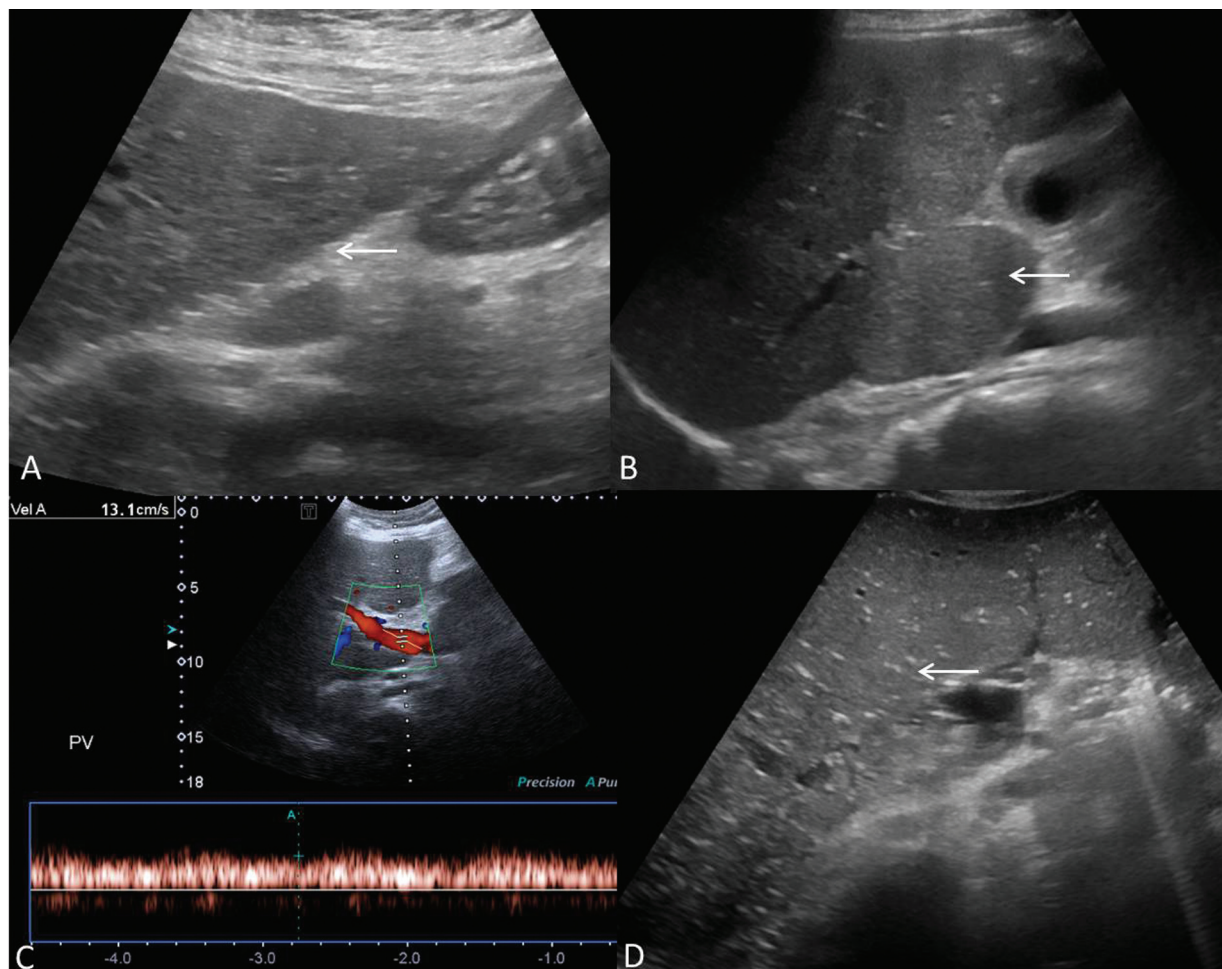


Fig. 1 Ultrasound of the liver in different patients with cirrhosis. (A) High-resolution image showing nodular surface (*arrow*) in the left lobe of liver. (B) Volume redistribution in the form of caudate lobe hypertrophy (*arrow*). (C) Color Doppler showing decreased portal vein velocity of 13 cm/s suggestive of portal hypertension. (D) Multiple echogenic foci in the spleen (*arrow*) representing Gamna–Gandy bodies.

Table 2 Society of Radiologists in Ultrasound (SRU) consensus recommendation for interpretation of liver stiffness values obtained with acoustic radiation force impulse (ARFI) techniques in patients with viral hepatitis and nonalcoholic fatty liver disease (NAFLD)⁸

Liver stiffness measurement	Recommendation
≤5 kPa (1.3 m/s)	High probability of being normal
<9 kPa (1.7 m/s)	In the absence of other known clinical signs, rules out compensated advanced chronic liver disease (CLD). If there are known clinical signs, may need further test for confirmation
9–13 kPa (1.7–2.1 m/s)	Suggestive of compensated advanced CLD but need further test for confirmation
>13 kPa (2.1 m/s)	Rules in compensated advanced CLD
>17 kPa (2.4 m/s)	Suggestive of clinically significant portal hypertension

surface is more frequent with viral hepatitis.⁴ Extrahepatic manifestations on Doppler include features of PHT like enlarged portal vein diameter > 12 mm, decreased portal vein velocity of <16 cm/s (►Fig. 1C; normal velocity is between 20 and 40 cm/s),⁵ hepatofugal flow, enlarged and tortuous hepatic artery,⁴ splenomegaly, and portosystemic collaterals. Portal vein thrombosis can occur in cirrhosis (prevalence: 0.6–15.8%).⁶

Elastography measures the stiffness of tissue and is able to detect different stages of liver fibrosis and can also predict PHT.⁷ US elastography techniques include FibroScan and acoustic radiation force impulse (ARFI) elastography.⁸ FibroScan is a one-dimensional transient elastography technique where shear waves are generated by a body-surface-controlled vibration, whereas ARFI elastography techniques are integrated into conventional US systems and shear waves are generated by the push pulse of a focused US beam. ARFI has few advantages over FibroScan.⁹ There is less operator dependency in ARFI, as there is no need for external compression by the operator. ARFI being integrated to the US system also has significantly lower unsuccessful measurements and is less sensitive to high body mass index and ascites. The liver stiffness measurements (LSM) with US elastography techniques are expressed in meter per second (m/s) or in kilopascal (kPa). Studies comparing fibrosis staging using ARFI and FibroScan have shown that the accuracy in fibrosis staging using ARFI is similar or better than that of FibroScan.^{8,10–12} The update to the Society of Radiologists in Ultrasound (SRU) consensus suggests using a “rule of four” (5, 9, 13, and 17 kPa) for fibrosis staging with ARFI-based techniques in patients with chronic viral hepatitis or nonalcoholic fatty liver disease.⁸ ►Table 2 summarizes these cutoff value recommendations. ARFI is also useful for the follow-up of patients with chronic hepatitis B virus (HBV) infection. Long-term antiviral therapy reduces liver stiffness in chronic hepatitis B patients with significant fibrosis and cirrhosis.¹³

Cross-sectional imaging is better to depict morphological abnormalities. The altered caudate lobe-to-right lobe ratio (>0.65) with the lateral border determined by the main portal vein bifurcation is associated with cirrhosis.¹⁴ The modified caudate-to-right lobe ratio with the right portal vein as the lateral border and an abnormal value >0.9 (►Fig. 2) had higher accuracy of 74 versus 66%.¹⁵ The right posterior hepatic notch sign (►Fig. 3A) indicates a sharp

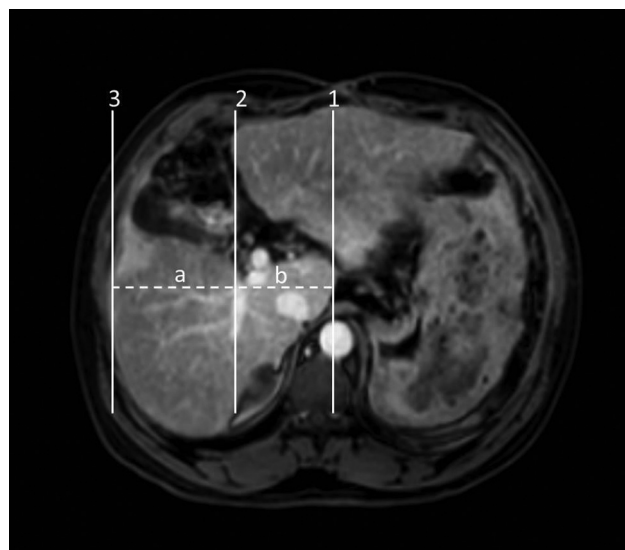


Fig. 2 Axial T1 fat-saturated (FS) postcontrast images through the liver in a patient with cirrhosis demonstrating modified caudate lobe-to-right lobe ratio. This is measured by drawing three parallel lines. Line 1 is through the left margin of the caudate lobe. Line 2 is through the right lateral wall of the right portal vein bifurcation and line 3 is through the lateral margin of the right lobe. A perpendicular line (dashed line) is drawn between the portal vein and the inferior vena cava (IVC). (a) Right lobe measurement and (b) caudate lobe measurement.

indentation along the right posteromedial surface of the liver between the hypertrophied caudate and the atrophic right posterior segment.¹⁶

The expanded gallbladder fossa sign specifies the enlarged pericholecystic space, bounded laterally by the right lobe, medially by the left lateral segment and an associated nonvisualized left medial segment.¹⁷ Widened porta hepatis anterior to the main portal vein at the hilum (►Fig. 3B) is also associated with cirrhosis. Heterogeneous appearance of hepatic parenchyma due to fibrosis is evident on both computed tomography (CT) and magnetic resonance imaging (MRI; seen as areas of high signal intensity on T2-weighted [T2W] MR).⁴

MRI is the best imaging modality to evaluate cirrhosis-associated nodules. RNs are isointense on T1W and T2W sequences. They have predominant vascular supply from the portal vein and hence are indistinguishable on the arterial

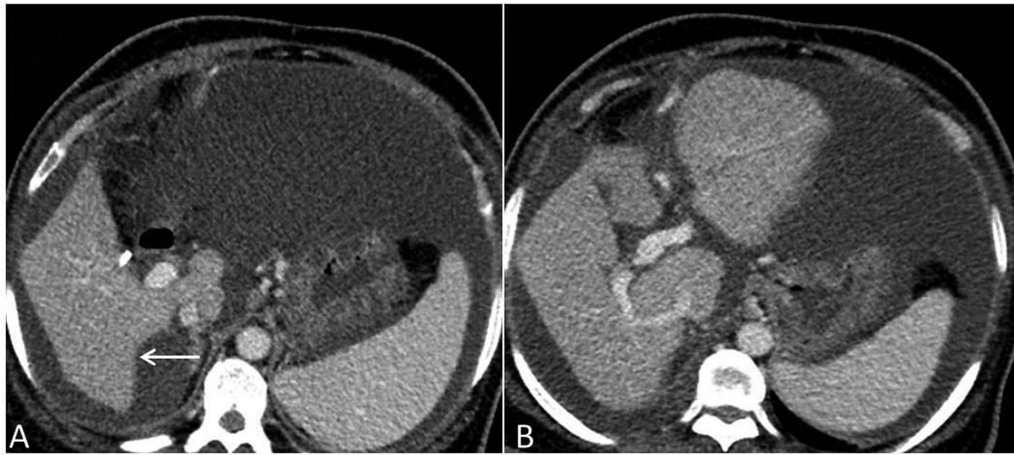


Fig. 3 Axial venous phase images of contrast enhanced CT of 55-year-old male with decompensated chronic liver disease showing (A) right posterior hepatic notch sign and (B) volume redistribution with widened porta hepatis.

phase and isointense on the other phases. These nodules can have T1W hyperintensity or may contain iron, resulting in low signal on both T1W and T2W¹⁸ imaging. Dysplastic nodules (DNs) can be low grade or high grade. Low-grade DNAs are similar to RNs. High-grade DNAs have arterIALIZED flow and hence show early arterial phase enhancement, which becomes isointense on subsequent dynamic postcontrast sequences.¹⁸ These nodules can progress to HCCs with characteristic arterial phase enhancement and washout.

Other extrahepatic findings associated with cirrhosis include ascites, bowel wall thickening, diffuse edema, and gallbladder wall thickening.¹⁹ The foci of hemosiderin in the spleen (Gamna–Gandy bodies) are seen as echogenic foci on US (→Fig. 1D) and T2 hypointense on MRI.

Noncirrhotic portal hypertension (NCPH)

NCPH, a presinusoidal, intrahepatic cause of PHT, accounts for 40% of cases with PHT in India.²⁰ NCPH is characterized by sclerosis and obliteration of medium and small branches of the portal vein with periportal fibrosis resulting in PHT. But features of cirrhosis is typically absent. NCPH is called noncirrhotic portal fibrosis (NCPF) in India, idiopathic PHT (IPH) in Japan, and called hepatportal sclerosis, idiopathic noncirrhotic intrahepatic PHT in the other parts of the world.²¹

The exact etiology is unknown in up to 50% of patients with NCPF. Some known etiologies include umbilical sepsis, portal pyemia, and diarrheal and bacterial infections in infancy. Other causes of NCPF are prothrombotic states, autoimmune diseases, antiretroviral drug such as didanosine, chemotherapy, and radiation.

Clinical Features

NCPF typically presents in the third and fourth decades with no gender predilection. Symptoms include well-tolerated gastrointestinal (GI) bleed, abdominal mass from massive splenomegaly, and anemia from hypersplenism. Jaundice, ascites, and hepatic encephalopathy are uncommon. Liver function test is normal. There are no features of cirrhosis on

Table 3 Stages of noncirrhotic portal fibrosis (NCPF)²²

Stage	Description
Stage I	Nonatrophic liver without subcapsular parenchymal atrophy
Stage II	Nonatrophic liver with subcapsular parenchymal atrophy
Stage III	Atrophic liver with subcapsular parenchymal atrophy
Stage IV	Portal venous occlusive thrombosis

liver biopsy. Portal vein thrombosis is more common in NCPF than in cirrhosis and is associated with poor prognosis.²¹

The stages of NCPF is described in →Table 3.²²

Imaging Findings

Ultrasound and Doppler

Depending on the stage, liver is either normal, enlarged, or small and shrunken. Unlike cirrhosis, the liver surface is smooth in patients with NCPF. Portal venous axis and intrahepatic portal tracts have echogenic wall thickening often >3 mm from periportal fibrosis (→Fig. 4A). Periportal fibrosis can also have a layered appearance with alternative echogenicity and hypoecho-genicity²³ (→Fig. 4B). Massive splenomegaly and normal splenoportal axis are hallmarks of NCPF. Doppler examination can reveal portal vein thrombosis, a known complication of NCPF.

Elastography

Liver elastography helps differentiate cirrhosis from NCPF. In NCPF, liver stiffness is normal or near normal compared with increased stiffness in cirrhosis. On the other hand, splenic stiffness follows an opposite trend in these diseases.²⁴

Cross-Sectional Imaging

Contrast-enhanced CT (CECT) of the abdomen and pelvis allows better evaluation of the structural changes. Diffuse liver atrophy is seen from chronic decrease in the portal supply. But unlike cirrhosis, there is no volume

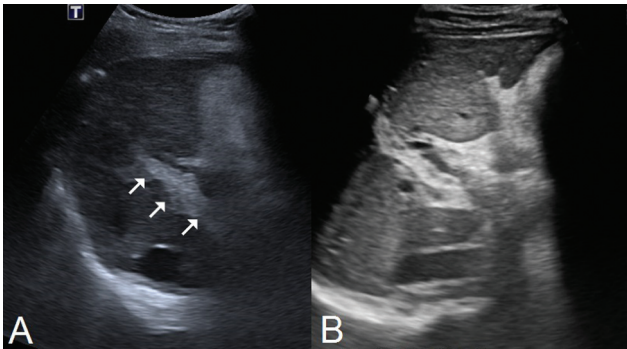


Fig. 4 Ultrasound of the liver of two different patients with non-cirrhotic portal fibrosis showing (A) echogenic wall thickening of the attenuated portal vein (arrow) and (B) layered appearance of periportal fibrosis.

redistribution. In the late stage, liver is shrunken, and its surface becomes nodular. However, segment IV volume is preserved until late in patients with NCPF (**Fig. 5**).^{25,26} Chronic hypoperfusion is also seen as decreased enhancement of the liver periphery in the venous phase and compensatory increased enhancement in the arterial phase and hypertrophied hepatic artery.²⁷ Obliterating portal venous disease can be seen as attenuation, abrupt cutoff, or amputation of small and medium-sized portal vein branches within the liver and this gives a “withered tree” appearance.²³ Features of thrombosis of intra- and extrahepatic portal vein can be seen as filling defects, wall thickening, calcification (**Fig. 5**), or portal cavernoma.²³ CT will also show massive splenomegaly and splenic infarcts. Esophageal (85–90%) and anorectal varices (89%) are common in NCPF.

Gastric varices are less common (25%) but often present with GI bleed (**Fig. 6**).^{21,23}

Hemodynamic Studies

In late stages of NCPF, it is hard to differentiate NCPF from cirrhosis based only on imaging findings. In this setting, hemodynamic studies are useful. HVPG, intrahepatic pressure, and hepatic venous pressure are normal or near normal in NCPF.²³

Magnetic Resonance Imaging

Periportal fibrosis is seen as hyperintense periportal cuffing on T2W images.⁷ Chronic hemodynamic disturbances can lead to focal nodular hyperplasia like lesions in the liver. Dynamic contrast-enhanced MRI is useful in characterizing these lesions.²⁶

Extrahepatic Portal Vein Obstruction

EHPVO refers to primary chronic thrombosis of extrahepatic portal vein with or without involvement of intrahepatic portal vein branches, splenic or superior mesenteric vein in the absence of liver disease or neoplasm, and resultant cavernous transformation of portal vein.²³

Etiopathogenesis

EHPVO is a common cause of PHT among children in developing countries, accounting for ~54% of cases.²⁸ The etiology is unknown in most cases. In children, it may be linked to neonatal umbilical sepsis or sequelae of the umbilical vein cannulation, whereas in adults, various prothrombotic states may predispose to this condition.²³

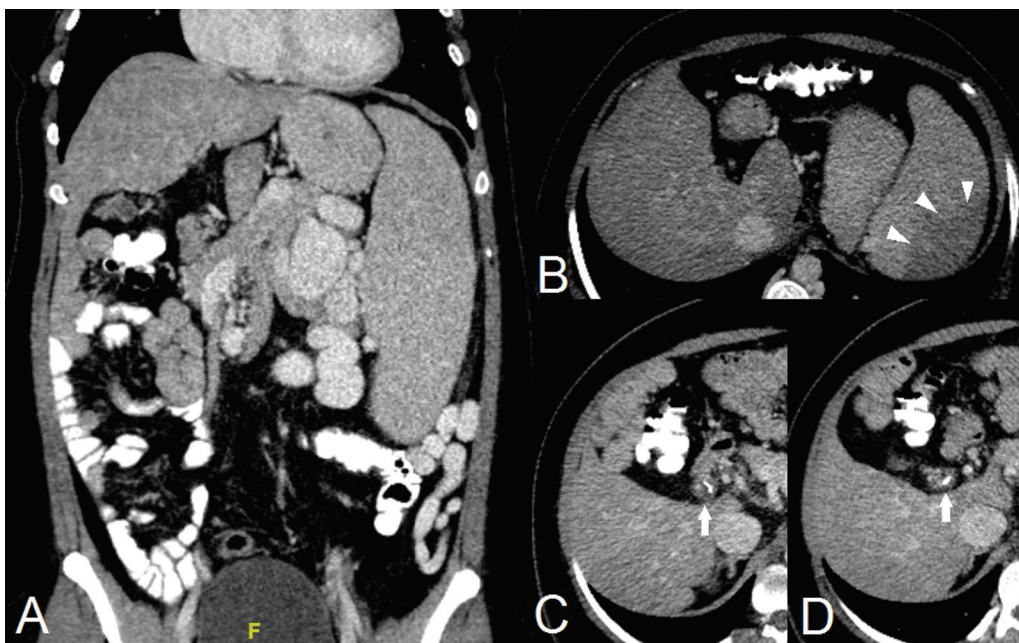


Fig. 5 Axial and coronal reconstructions of venous phase images of contrast-enhanced computed tomography (CT) of a 23-year-old female patient with noncirrhotic portal hypertension (NCPH) and gastrointestinal (GI) bleed showing (A,B) a small liver with smooth margins and massive splenomegaly. Note the infarcts in the upper pole of spleen (arrowheads in B). But there is no ascites. (C,D) Hypodense filling defects and calcifications in the portal vein due to portal vein thrombosis.

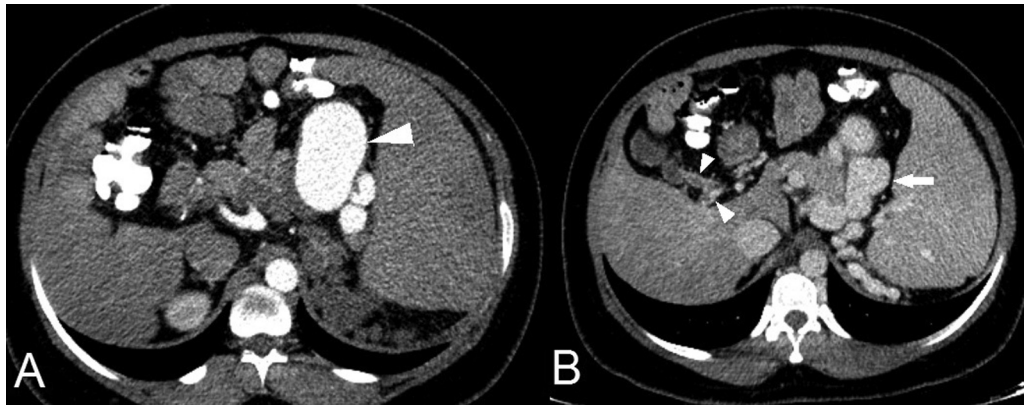


Fig. 6 Contrast-enhanced computed tomography (CECT) of the same patient in ►Fig. 2. (A) The arterial phase shows a splenic artery aneurysm (arrowhead) from long-standing massive splenomegaly. (B) Portal vein thrombosis (arrowheads) and multiple dilated perigastric collaterals (arrow). For the gastrointestinal (GI) bleed, the patient underwent embolization of splenic artery aneurysm and endoscopic glue injection of gastric varices.

Portal vein thrombosis results in the formation of portoportal collaterals as alternate pathways to shunt blood to the liver. These portoportal collaterals are pericholedochal (surrounding bile duct) plexus of Petren, epicholedochal (in the wall of the bile duct) plexus of Saint, and pericholecystic venous plexuses, which become dilated and tortuous. Splenomegaly and portosystemic collaterals develop as portal pressures increase further.

The etiopathogenesis of PHT is chiefly a result of increased vascular resistance. As the disease progresses, there is increased blood flow to the portal venous system resulting in worsening of elevated portal pressures and complications of PHT.²

Abnormalities in the extrahepatic biliary tract occur in patients with EHPVO due to either extrinsic compression of the bile duct by collaterals or biliary stricture due to prolonged ischemia and is termed portal cavernoma cholangiopathy (PCC). The prevalence of clinically significant PCC ranges from 5 to 50% in patients with EHPVO, although morphological changes in the biliary tract can be seen in

70 to 100% of cases.²⁹ It may be irreversible if there is fibrotic stricture or masslike cavernoma with predominant fibrotic soft tissue compressing the duct, whereas shunt surgery may decrease varices resulting in reversibility.³⁰

Imaging Findings

Ultrasound and Color Doppler

Ultrasonography (USG) is the initial modality to evaluate EHPVO. The portal vein is replaced by multiple tortuous venous channels at the porta and peripancreatic regions. The pericholecystic vessels also frequently enlarge and become tortuous. The liver surface is smooth and there is no volume redistribution; however, these can set in later in the disease course due to long-standing perfusion alterations. As PHT develops, there is moderate to gross splenomegaly and portosystemic collaterals (►Fig. 7). Doppler demonstrates absent flow in the main portal vein and decreased monophasic flow toward the liver in the portoportal collaterals. The periportal collaterals can sometimes appear as masslike

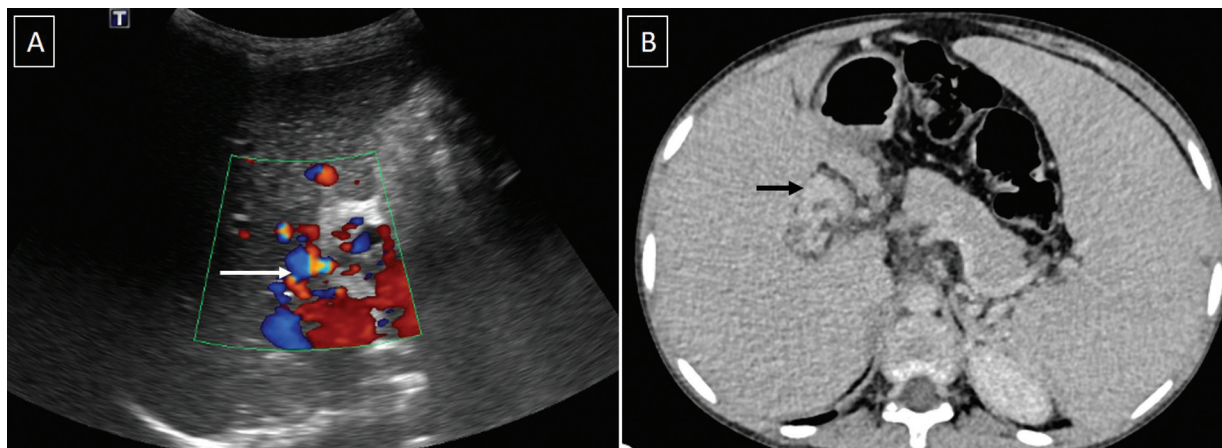


Fig. 7 Color Doppler image (A) in a patient with extrahepatic portal vein obstruction (EHPVO) shows nonvisualized main portal vein replaced by multiple tortuous collaterals at the porta. Contrast-enhanced computed tomography (CECT) axial section (B) of the same patient shows portal cavernoma (black arrow) splenomegaly and relatively smooth liver surface.

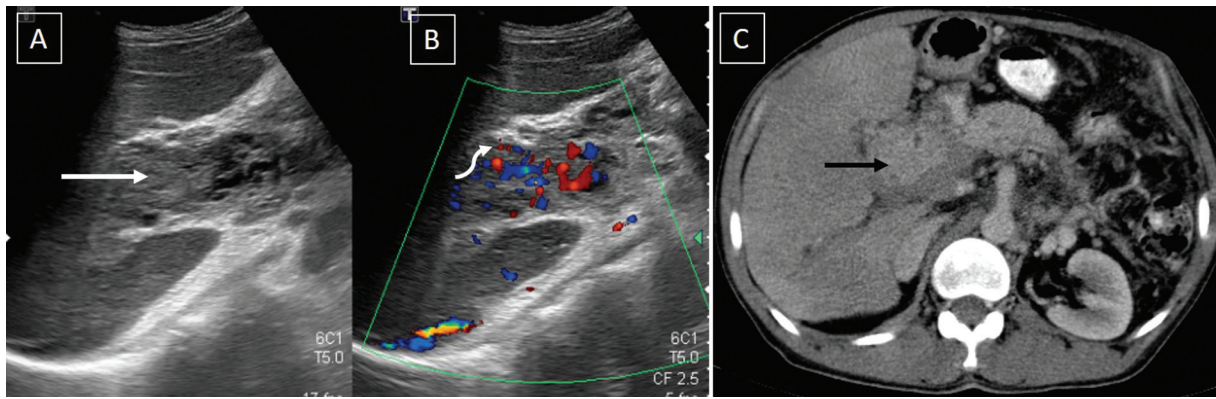


Fig. 8 (A) Ultrasonography (USG) in a patient with extrahepatic portal vein obstruction (EHPVO) shows masslike soft-tissue thickening at the porta due to extensive fibrosis. (B) Color Doppler shows few vascular channels within the masslike soft-tissue thickening. (C) Contrast-enhanced computed tomography (CECT) axial section shows enhancement of small vascular channels in the masslike cavernoma at the porta.

soft-tissue thickening with few vascular channels due to extensive fibrosis. Color Doppler detects color flow in vascular channels within this masslike thickening (→**Fig. 8**).

Intrahepatic biliary radicle dilatation in PCC can also be detected on USG.

Computed Tomography

CECT helps assess the extent of vascular involvement, clearly demonstrates the portosystemic circulation, and excludes other etiologies of PHT.

The liver morphology is largely preserved till later stages when perfusion abnormalities can result in volume redistribution and surface irregularity.³¹ Vasodilatation and increased splenic circulation also result in splanchnic aneurysms.

CECT is valuable in preshunt evaluation to assess the extent of portal vein thrombosis and patency of the splenic vein, superior mesenteric vein, renal vein, and inferior vena cava (IVC) along with any anatomic variations or large collaterals if present. In postoperative patients, CECT also helps assess the shunt patency and obliteration of the collaterals.

Magnetic Resonance Cholangiopancreatography

Magnetic resonance cholangiopancreatography (MRCP) is the imaging of choice to evaluate biliary changes in EHPVO. The findings include extrinsic indentation resulting in a wavy contour of the biliary duct, focal stricture with upstream dilatation, and sharp angulation of the bile duct (→**Fig. 9**). Cholelithiasis, choledocholithiasis, and hepatolithiasis can also occur. The stricture can be long segment (>2 cm long) or short segment (≤2 cm).³²

Based on morphology and etiopathogenesis, biliary abnormalities in EHPVO are classified into three types: varicoid type (due to pericholedochal collaterals causing extrinsic compression), fibrotic type (stricture due to ischemia, inflammation with typical delayed enhancement), and mixed type (undulation and strictures with associated dilatation).³³ A radiological classification based on MRCP by Llop et al suggests the likelihood of developing symptoms in PCC. Minimal irregularities are classified as grade 1 PCC, stenosis without dilatation as grade 2 PCC, and stenosis with dilatation (dilatation defined as intrahepatic duct measuring ≥4mm and extrahepatic duct caliber measuring ≥7mm)

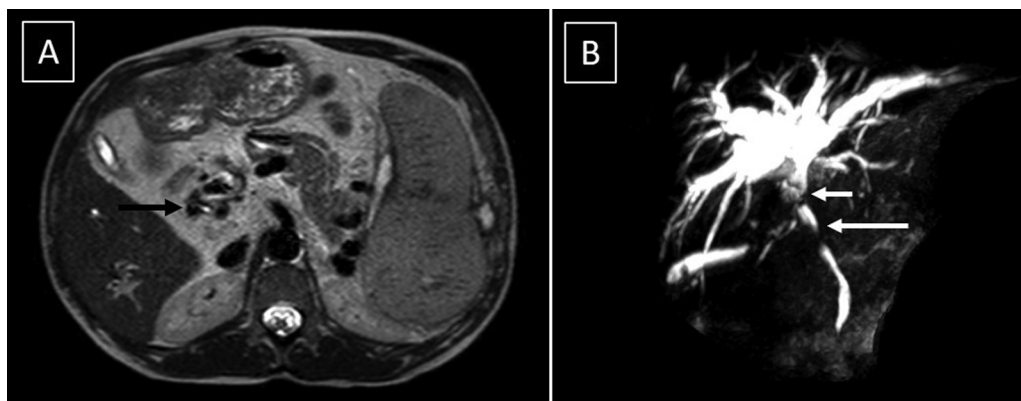


Fig. 9 T2-weighted (T2W) axial image (A) in a patient with extrahepatic portal vein obstruction (EHPVO) and portal cavernoma cholangiopathy shows multiple tortuous vascular flow voids at the porta surrounding the biliary duct causing narrowing of lumen (black arrow). (B) Magnetic resonance cholangiopancreatography (MRCP) image shows short segments of narrowing in the extrahepatic bile duct and resultant bilobar symmetric intrahepatic bile duct (IHBRD, intrahepatic biliary radical dilatation).

as grade 3, with grade 3 PCC associated with higher risk of symptomatic disease.³⁴

Budd–Chiari syndrome

Budd–Chiari syndrome (BCS) refers to disorders resulting from partial or complete hepatic venous outflow obstruction in the hepatic veins or IVC up to the cavoatrial junction. It occurs more commonly in young adults and in women^{35,36} and is an important posthepatic cause of PHT.

Etiopathogenesis

BCS is classified based on etiology into primary and secondary types. Primary BCS occurs due to primary endoluminal venous problems such as webs, thrombosis, and stenosis. Inherited hypercoagulable states such as protein C, protein S deficiency, antithrombin 3 deficiency, and acquired prothrombotic states like myeloproliferative disorders may predispose to BCS.^{37,38} Secondary BCS results from extrinsic compression of veins by abscesses and cysts, and infiltration by tumors like HCC, renal cell carcinoma, and others.^{38,39}

The hepatic venous outflow obstruction leads to increased hepatic sinusoidal pressure and decreased portal perfusion. There is hepatic congestion, which can progress to ischemic injury, hepatocyte necrosis, fibrosis, and ultimately cirrhosis.^{35,39}

Imaging

Imaging findings reflect the duration of BCS with varying changes in acute, subacute, and chronic stages.

Acute BCS

US and Doppler will show hepatomegaly, ascites, and enlarged caudate lobe, which may compress the IVC. An enlarged caudate vein (≥ 3 mm) may suggest BCS.³⁶ Distended hepatic veins with hypoechoic thrombus may be seen, which evolves over time to echogenic thrombus with reduction in caliber of the vein. On Doppler, there may be loss of phasic variation of the waveform, absent flow, or reversed flow.^{38,39} In the presence of a web, there will be increase in flow

velocity and color aliasing at the site of narrowing.³⁹ Portal vein may show hepatofugal flow.³⁸

Computed Tomography and Magnetic Resonance Imaging

The acute changes of increased sinusoidal pressure and hepatic congestion due to outflow obstruction cause hepatomegaly with patchy reduced enhancement in the peripheral portions of the liver on the early phase of CECT and MRI with relatively increased enhancement in the caudate lobe, which is enlarged³⁷ (► **Fig. 10**). Reversal of enhancement with increased enhancement in the peripheral portions of the liver and relative decreased enhancement in the caudate lobe on the venous phase is termed the “flip flop” phenomenon.

Subacute Budd–Chiari Syndrome

In the subacute stage, imaging will show volume distribution in the liver with multiple collaterals. These changes are also seen in chronic BCS from which it may be difficult to differentiate on imaging.

Chronic Budd–Chiari Syndrome

In chronic BCS, morphologic changes in the liver are those of chronic liver disease with volume redistribution (► **Fig. 11**). The parenchymal edema seen in acute BCS is now replaced by fibrosis with delayed enhancement on CECT or MRI.³⁵ Marked caudate lobe hypertrophy and multiple RNs are a feature of chronic BCS. RNs (0.5–4 cm in size) represent a hyperplastic response in parts of the liver with decreased portal perfusion, hyperarterialization, and preserved venous outflow. On CT, these nodules are hyperenhancing in the arterial phase with enhancement often persisting into the venous phase with no “washout.”³⁶ On MRI, these nodules are hyperintense on T1W MRI and iso- to mildly hypointense on T2W MRI.

The collateral pathways that open up in chronic BCS include comma-shaped intrahepatic venovenous collaterals, intrahepatic subcapsular collaterals, left renal hemiazygos pathway inferior phrenic- pericardiophrenic collaterals, and

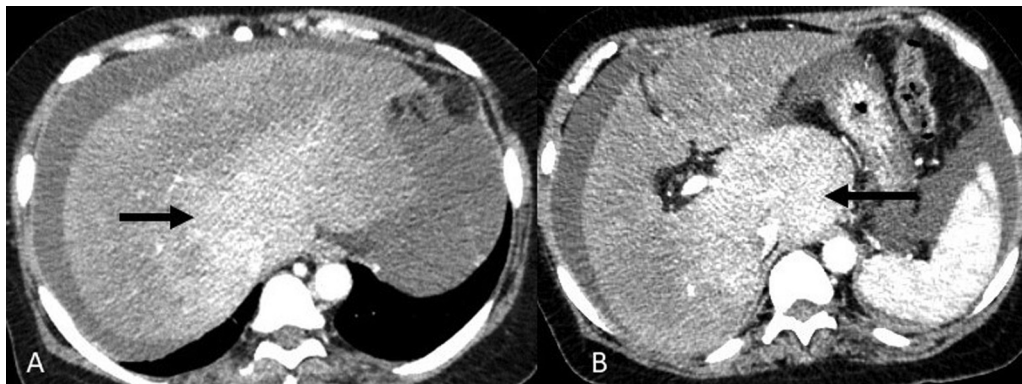


Fig. 10 Contrast-enhanced computed tomography (CECT) axial images of a patient with acute Budd–Chiari syndrome shows hypoenhancement of the peripheral portions of the liver with a relatively increased enhancement in the caudate lobe (arrow), which is enlarged. There is ascites and nonvisualized hepatic veins.

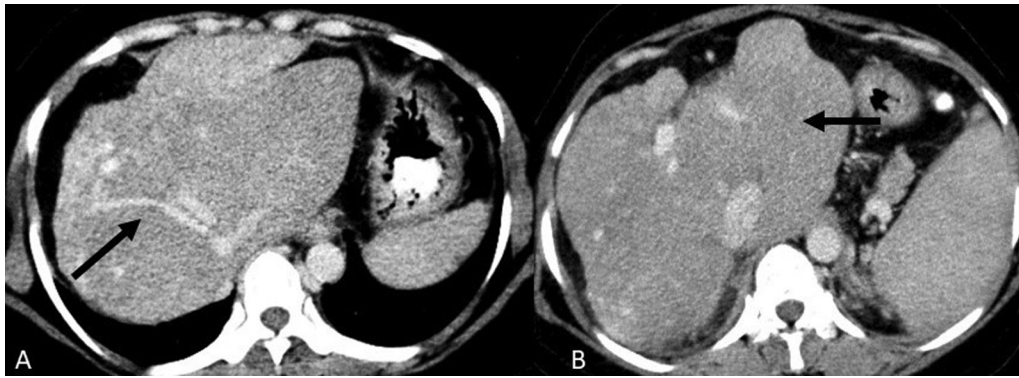


Fig. 11 Contrast-enhanced computed tomography (CECT) axial images of a patient with chronic Budd–Chiari syndrome with a markedly enlarged caudate lobe (*arrow*), irregular liver margins, and the presence of intrahepatic collaterals (*arrow*).

collaterals of the abdominal wall.³⁸ Enlarged right hepatic artery may be seen.

Collateral Pathways in Portal Hypertension and Related Imaging

The increased portal pressure in PHT redirects blood through alternate pathways into low-pressure systemic veins, leading to formation of an extensive network of portosystemic collateral vessels.^{40,41} These collaterals include dilated end organ veins called varices and dilated channels that connect the portal and systemic vascular beds called shunts. The common varices and shunts in PHT are listed in **Table 4**.^{41,42}

The collaterals can also be classified as tributary collaterals, developed collaterals, or bridging collaterals.⁴³

Tributary collaterals are normal tributaries of the portal venous system in which flow reversal occurs and transforms into large shunts. They include the left gastric vein, short gastric vein, and superior and inferior mesenteric veins.

Developed collaterals are reopened congenital connections that should have been closed if there was no PHT. They include recanalized paraumbilical vein, splenorenal, gastrorenal, and splenoretroperitoneal collaterals.

Bridging collaterals occurs in EHPVO or splenic vein thrombosis to restore the hepatopetal flow. In EHPVO, bridging collaterals are the peribiliary venous collaterals forming a cavernoma (**Fig. 12 and 13**). In splenic vein occlusion, the bridging collaterals are the short gastric vein to the left gastric vein or the gastroepiploic vein to the superior mesenteric vein, both of which finally drain into the portal vein.

Table 4 Summary of common portosystemic collaterals^{41,42}

Collaterals	Afferent	Efferent
Varices		
Esophageal/paraesophageal	Left gastric vein	Azygos–hemiazygos veins
Gastric/perigastric	Left gastric vein/short gastric and posterior gastric veins	Esophageal/paraesophageal veins
Duodenal	Superior and inferior pancreaticoduodenal veins, cystic branches of the superior mesenteric veins, gastroduodenal vein, and pyloric vein	Veins of Retzius into the inferior vena cava (IVC)
Jejunioileal	Jejunal and ileal veins	Abdominal wall veins/veins of Retzius
Colonic	Ileocolic, right, middle colic, or sigmoid colic vein	Right gonadal vein, right renal vein, and systemic lumbar veins
Pancreatic	Ventral and dorsal pancreatic veins, and pancreaticoduodenal veins	IVC
Uterovaginal	Superior hemorrhoidal plexus	Uterine and hypogastric veins to the IVC
Vesical	Mesenteric veins	Internal and external iliac veins
Pericholecystic	Cystic vein or a branch of the right portal vein	Hepatic vein, intrahepatic portal vein, or anterior abdominal wall collaterals
Bronchial	Tracheobronchial plexus of veins	Pulmonary veins, bronchial veins, and esophageal/paraesophageal varices
Mesenteric	Superior and inferior mesenteric veins	Retroperitoneal or pelvic veins (veins of Retzius)

(Continued)

Table 4 (Continued)

Collaterals	Afferent	Efferent
Omental	Superior or inferior mesenteric veins	Retroperitoneal or pelvic veins or gastroesophageal veins
Retroperitoneal	Colic or mesenteric branches	Retrogastric varices or inferior phrenic veins to the left renal vein or directly into the IVC
Rectal/perirectal	Superior rectal veins	Middle and inferior rectal veins, tributaries of the internal iliac and pudendal veins
Shunts		
Intrahepatic shunts	Portal vein branches	Hepatic veins or directly to the intrahepatic IVC
Transhepatic shunts		
Recanalized paraumbilical vein	Left portal vein	Anterior abdominal wall veins and iliofemoral veins
Right infradiaphragmatic/apex type shunt	Left portal vein	Internal thoracic vein and intercostal vein
Left infradiaphragmatic	Portal branch of the left lateral segment of the liver	Intercostal vein or left pericardiophrenic vein to left
Left triangular ligament shunt		Inferior phrenic vein or left triangular ligament
Right posterior portal branch IVC shunt	Right posterior portal vein	IVC or the right adrenal vein
Bare area shunt	Right posterior portal vein	Intercostal vein or the right inferior phrenic vein
Aberrant left gastric vein shunt	Left portal vein	Hepatogastric ligament
Extrahepatic shunts		
Gastrorenal	Gastric varices or posterior or short gastric veins	Left renal vein
Splenorenal	Splenic vein	Left renal vein
Gastrocaval	Gastric varices or posterior gastric vein	Left inferior phrenic and pericardiophrenic vein to the IVC
Splencaval	Splenic vein or perisplenic collaterals	Hypogastric vein into the IVC
Trans-splenic	Splenic vein	Intercostal veins
Mesenterico-gonadal/renal/caval	Mesenteric veins	Right gonadal vein
Splenic/coronary pulmonary vein	Left gastric vein or splenic vein	Inferior pulmonary vein, pericardiophrenic vein, or intercostal vein
Spleno-azygous/phrenic	Splenic vein	Left inferior phrenic vein, posterior abdominal wall veins, or hemiazygos vein
Pancreaticoduodenal hemiazygos	Ventral and dorsal pancreatic veins, and pancreaticoduodenal veins	Posterior abdominal wall veins or hemiazygos vein
Left renal hemiazygous	Left renal vein	Hemiazygous vein
Vertebrolumar azygous	Ascending lumbar vein	Azygous vein
Anterior abdominal wall	Superficial epigastric and inferior epigastric vein	Superior epigastric and lateral thoracic veins
Inferior phrenic pericardiophrenic	Inferior phrenic vein	Pericardiophrenic vein

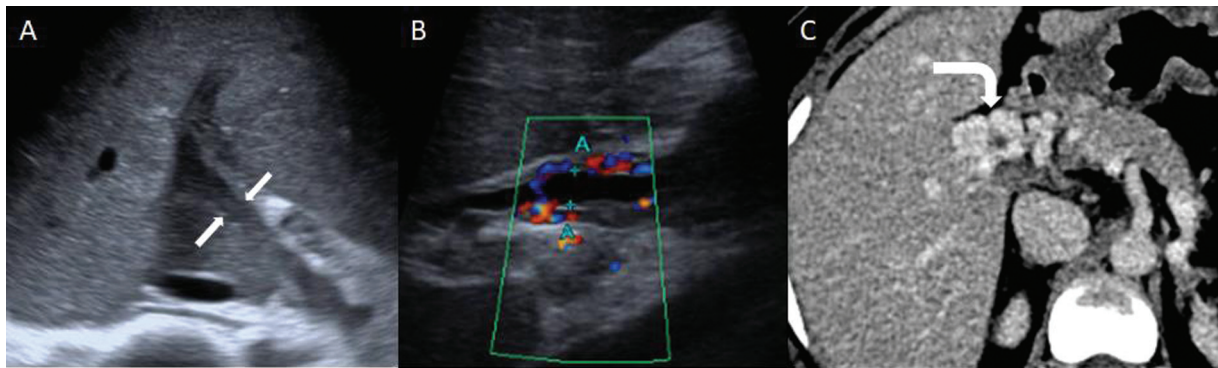


Fig. 12 Bridging collaterals in extrahepatic portal vein obstruction (EHPVO). (A) Ultrasonography (USG) image shows echogenic thrombus completely occluding the portal vein (*between arrows*). (B) The color Doppler image shows small developing peribiliary vessels around CBD. (C) Computed tomography (CT) done after a year shows large caliber peribiliary venous collaterals (*curved arrow*) forming cavernoma.

Varices

The most common varices seen in PHT are esophageal/paraesophageal varices (50%), followed by gastric/perigastric varices (10–35%).⁴² Varices elsewhere (ectopic varices) are less common.⁴² They include duodenal, jejunoileal, colonic, pancreatic, mesenteric, vesical, pericholecystic, retroperitoneal, and rectal/perirectal varices. Esophageal varices are the most common to bleed in cirrhotic patients.⁴⁴ The risk of bleeding from the gastric varices is lower than that from the esophageal varices; however, the

severity of bleeding is significantly higher because of their large size.⁴⁴ The ectopic varices, although uncommon, have a fourfold increased risk of bleeding when compared with the esophageal varices.⁴² Ectopic variceal bleeds are difficult to control by any means, and larger bleeds are potentially fatal.⁴²

Shunts

Shunts can be divided into intrahepatic, transhepatic, and extrahepatic shunts.⁴² Intrahepatic shunts can be single or multiple shunts connecting the portal vein branch to the

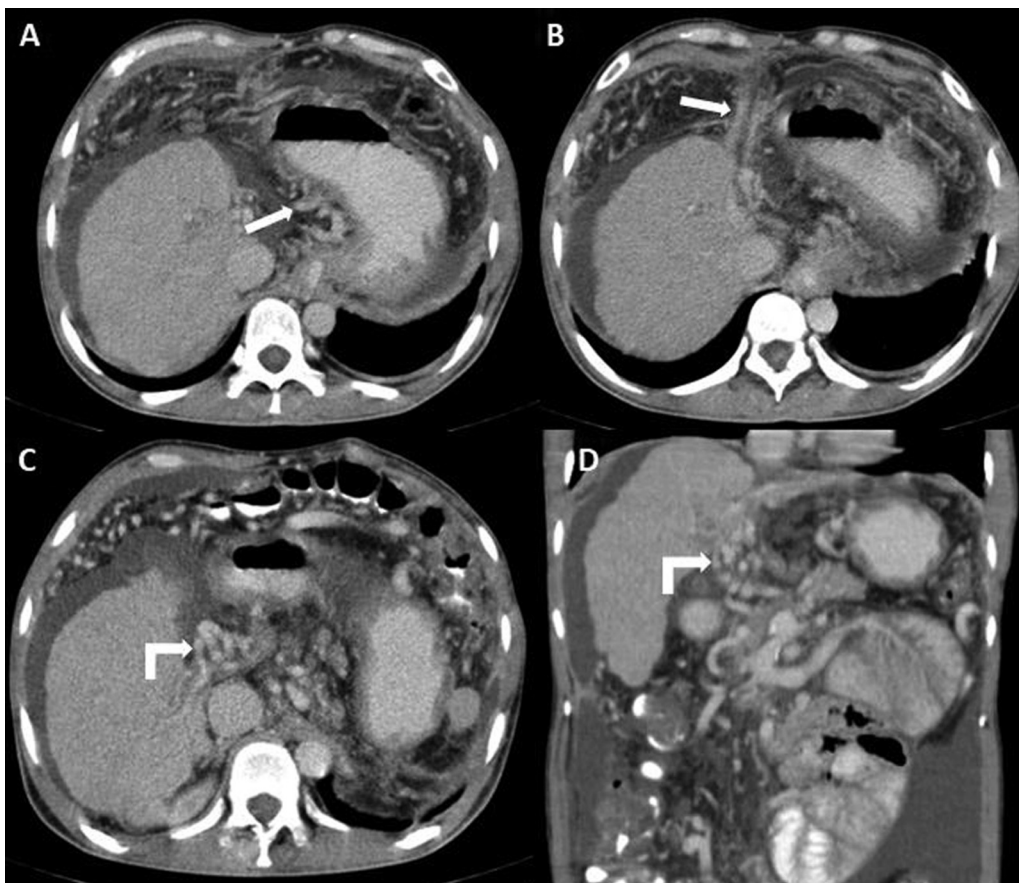


Fig. 13 Types of collaterals in portal hypertension. (A) Computed tomography (CT) image shows a dilated left gastric vein (*arrow*), an example of tributary collateral. (B) CT image shows a recanalized paraumbilical vein (*arrow*), an example of developed collateral. (C, D) CT images show nonvisualization of the portal vein with multiple peribiliary collaterals (*bend arrows*) replacing it, which is an example of bridging collateral.

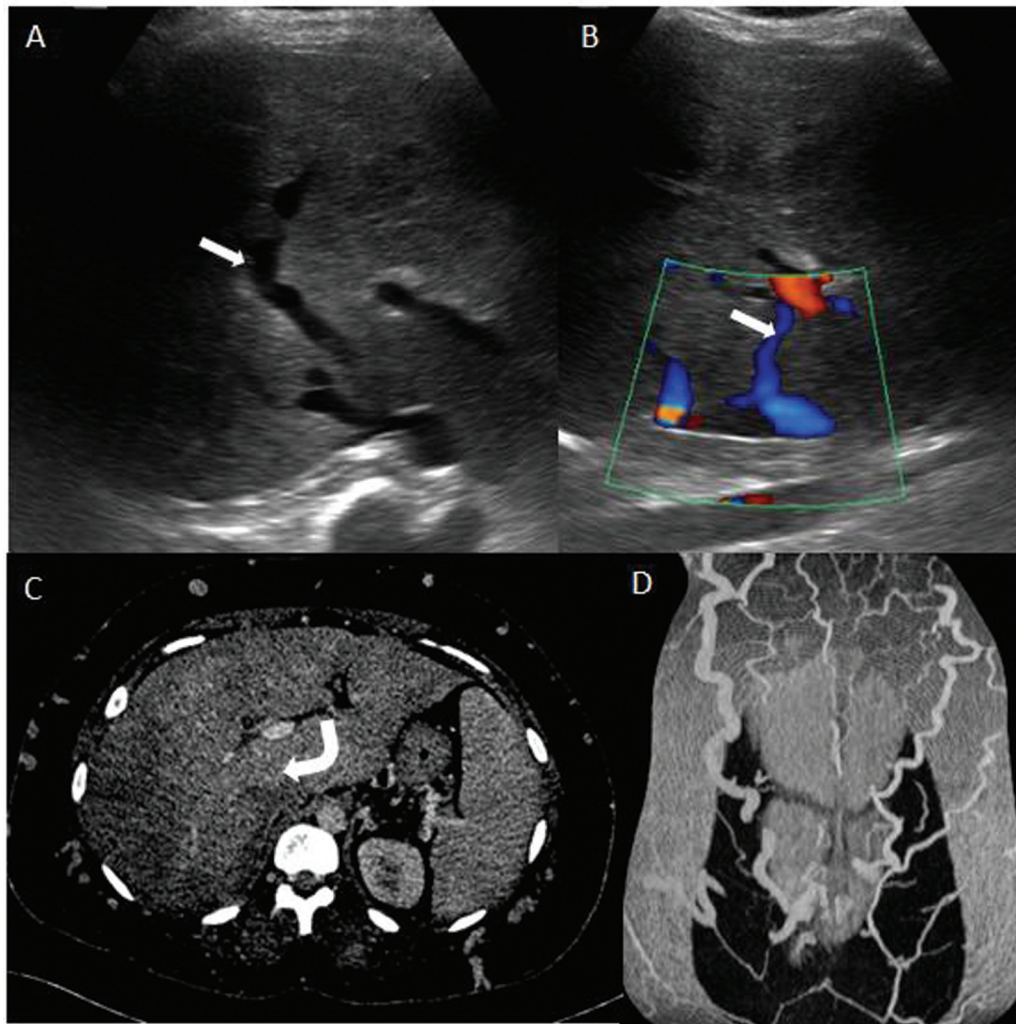


Fig. 14 Collaterals in Budd–Chiari syndrome. (A) US and (B) color Doppler images show intrahepatic tortuous collaterals (*straight arrows*). (C) Computed tomography (CT) axial image shows the nonopacification of intrahepatic inferior vena cava (IVC; *curved arrow*) with inhomogeneous attenuation of liver parenchyma. (D) The maximum-intensity projection CT image shows extensive superficial anterior abdominal wall shunts typically seen in Budd–Chiari syndrome.

intrahepatic IVC or hepatic veins. The transhepatic shunts connect the intrahepatic branches of the portal vein with a systemic vein outside the liver. The commonest transhepatic shunt is the recanalized paraumbilical vein. The extrahepatic shunts are between the extrahepatic portal vein tributaries with a systemic vein outside the liver. Among these shunts, the intrahepatic shunts are almost exclusively seen in BCS and very rare in other causes of PHT.⁴⁵ The extrahepatic shunts in BCS are also characteristic and include left renal hemiazygous, vertebral lumbar azygous, superficial anterior abdominal wall, and inferior phrenic pericardiophrenic shunts⁴⁴ (→ **Fig. 14**).

Diagnosis of portal hypertension

Diagnosis of PHT is by demonstrating its direct or associated signs clinically or with imaging.⁴⁶ Direct signs include dilated splenoportal axis (main portal vein diameter >13 mm and splenic vein diameter >10 mm), reduced or reversed portal vein flow and presence of collaterals.⁴⁷ The important associated findings include splenomegaly (length >12 cm) and ascites.^{48,49}

US and Doppler has the advantage of being a noninvasive, cost-effective, real-time test. It helps identify patency and direction of flow in relevant vessels (portal vein, splenoportal axis, hepatic veins, and IVC) and frequently identifies collaterals. The normal portal vein flow on Doppler is always hepatopetal with a fairly uniform flow and slight undulation/phasicity due to respiration and cardiac activity. The normal mean flow velocity is 12 to 18 cm/s (or peak velocity from 20 to 40 cm/s). As PHT starts to develop, the flow in the portal vein decreases and loses normal undulation and becomes monophasic. With more severe PHT, there will be reverse/hepatofugal flow. However, if intra- and transhepatic collaterals beyond the level of portal vein are present, the forward flow in portal vein is maintained even with severe PHT.⁵⁰ To increase the objectivity and reproducibility of US, optimization of technique is needed.⁴⁷ The portal vein diameter should be measured at the point it crosses anterior to the IVC. For Doppler evaluation, the most important technical requirement is the need to keep the angle of insonation less than 60 degrees. The respiration

pattern can greatly influence the flow in the portal system. Scanning should be done in gentle respiration. Performing scan in arrested or deep inspiration can increase the portal vein caliber and halt or even reverse the flow, and hence should be avoided.

CT and MRI are usually considered the second-line modality in diagnosing PHTN when US is inadequate due to patient factors causing poor acoustic window. CT interpretation is not based on flow dynamics, but based on morphological changes in vessels and organs and the presence of collaterals, which are always better depicted than in US.⁵¹ MRI also provides morphologic information comparable to the CT scan. Phase contrast MRI technique can offer quantitative information about the portal venous flow.⁵² Other than for the cases with inadequate US, CT and MRI are important in preoperative planning of portosystemic shunt surgeries to identify technically feasible shunts and complicating factors.⁵³

Interventional radiology (IR) in portal hypertension

IR plays a major role in diagnosis and management of PHT.⁵⁴ HVPG and transjugular liver biopsy (TJLB) are invasive diagnostic tests. The HVPG represents the portal pressure indirectly, to optimize the medication to control PHT. TJLB is performed through a transvenous access, avoiding the liver

capsule. It is safer in patients with ascites or deranged bleeding parameters.

Transjugular intrahepatic portosystemic shunts (TIPS) are performed in patients of PHT who do not respond to endoscopic management of variceal bleeding. TIPS is also performed in patients with refractory ascites secondary to PHT. It reduces portal pressure. Adding US to the fluoroscopic guidance during the creation of TIPS increases the success rate and reduces the complication and radiation dose.^{55,56} A covered stent placed in the parenchymal tract increases long-term patency. A complication of TIPS is hepatic encephalopathy.

Balloon-occluded retrograde obliteration (BRTO) of gastric varices is done to treat bleeding gastric varices.⁵⁷ The access is through the left renal vein into the variceal outflow from the stomach wall. The varices are obliterated by temporarily keeping a balloon inflated and retrogradely injecting sclerosants like sodium tetradecyl sulfate. BRTO increases the portal pressure, in contrast to TIPS (→Fig. 15). Bleeding ectopic varices like the paraumbilical collateral veins, when suitable, may be accessed percutaneously under image guidance for sclerotherapy.

An acutely thrombosed portal vein may be accessed percutaneously or via a transjugular access for pharmacological thrombolysis and mechanical thrombectomy. Chronically

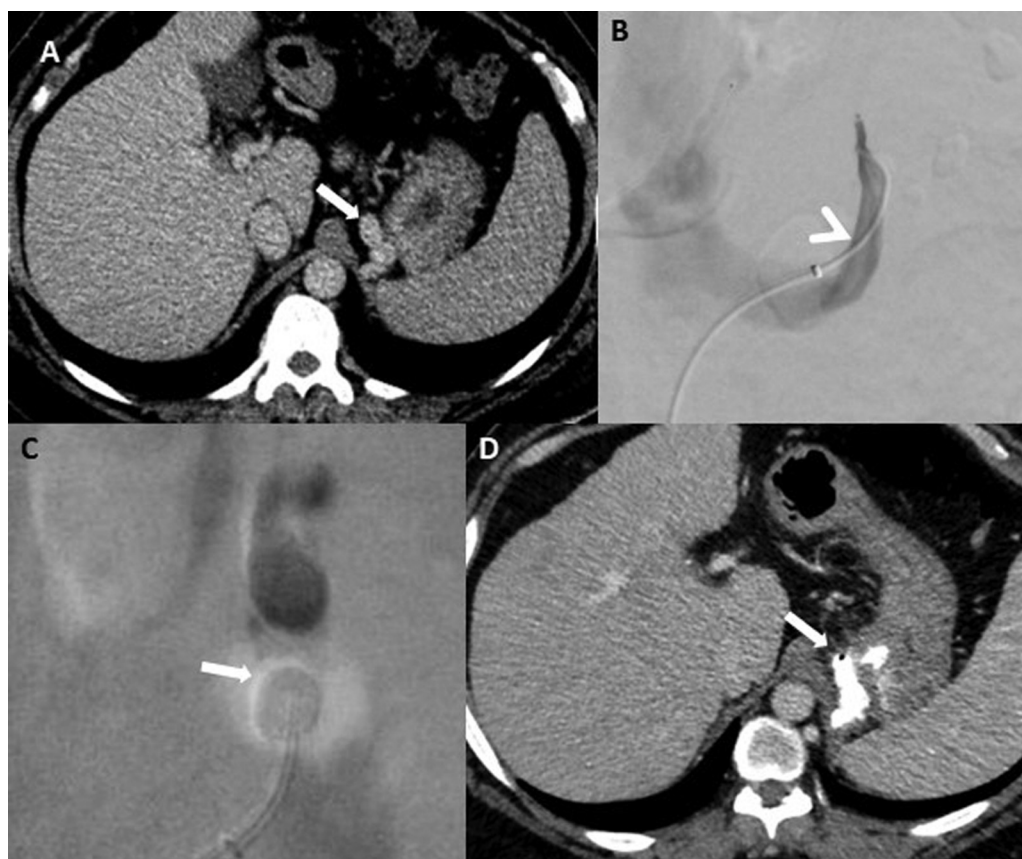


Fig. 15 Balloon retrograde obliteration (BRTO) done for a patient with bleeding gastric varices. (A) The preprocedure computed tomography (CT) image shows a large collateral (arrow) in the cardia of the stomach. (B) Digital subtraction venogram image shows the transfemoral venous approach with the tip of the catheter in the efferent vein (arrowhead) joining the left renal vein. (C) Venogram image shows an inflated balloon in the efferent vein (arrow) with contrast stasis in the collateral. Lipiodol mixed with 3% sodium tetradecyl sulfate foam was then used to embolize the varices (not shown). (D) The postprocedure CT image shows dense contrast within the collateral (arrow), which represents lipiodol-mixed sclerosant within the collateral.

occluded portal veins can be treated by TIPS to reduce portal pressure. Left-sided PHT (LSPH) or sinistral hypertension is a potentially life-threatening cause of GI hemorrhage.⁵⁸ It usually occurs as a result of isolated obliteration of the splenic vein. Partial splenic embolization is a technique to reduce the flow into the spleen and indirectly reduce the splenic venous outflow pressure in the collateral veins. Usually, the subdiaphragmatic portion of the spleen is spared to minimize the postprocedural symptoms.

IR is an option when surgically created shunts require revisions, which may be achieved by balloon angioplasty or stenting.⁵⁴

Recanalization of occluded IVC or hepatic veins in patients with BCS facilitates physiological restoration.⁵⁹ Recanalization of the hepatic vein could be attempted through the transjugular or percutaneous accesses. Catheter-assisted techniques increase the chances of recanalization.⁶⁰

Stenting should be considered when balloon angioplasty is not successful in retaining the patency or reducing pressure gradient. If the hepatic veins are not repairable, TIPS is considered to treat significantly symptomatic patients. The tract in such patients is created between the IVC and the portal vein, known as direct intrahepatic portosystemic shunt (DIPS). Multiple IR procedures may be required over a period of time in patients of BCS.⁶¹

A percutaneous peritoneovenous shunt is a palliative option in a selective subgroup of patients with refractory ascites who are not suitable for other specific treatment options.⁶²

Imaging thus has a diagnostic role to identify the etiology and the severity of PHT. It also has a varied therapeutic role to control bleeding and other secondary manifestations of this condition.

Conflict of Interest

None declared.

References

- Bloom S, Kemp W, Lubel J. Portal hypertension: pathophysiology, diagnosis and management. *Intern Med J* 2015;45(01):16–26
- Martí-Bonmatí L, Delgado F. MR imaging in liver cirrhosis: classical and new approaches. *Insights Imaging* 2010;1(04):233–244
- Sangster GP, Prevgliano CH, Nader M, Chwoschtschinsky E, Heldmann MG. MDCT imaging findings of liver cirrhosis: spectrum of hepatic and extrahepatic abdominal complications. *HPB Surg* 2013;2013:129396
- Gupta AA, Kim DC, Krinsky GA, Lee VSCT. CT and MRI of cirrhosis and its mimics. *AJR Am J Roentgenol* 2004;183(06):1595–1601
- Iranpour P, Lall C, Houshyar R, et al. Altered Doppler flow patterns in cirrhosis patients: an overview. *Ultrasonography* 2016;35(01):3–12
- Kinjo N, Kawanaka H, Akahoshi T, et al. Portal vein thrombosis in liver cirrhosis. *World J Hepatol* 2014;6(02):64–71
- Gherlan GS. Liver ultrasound elastography: more than staging the disease. *World J Hepatol* 2015;7(12):1595–1600
- Barr RG, Ferraioli G, Palmeri ML, et al. Elastography assessment of liver fibrosis: society of radiologists in ultrasound consensus conference statement. *Radiology* 2015;276(03):845–861
- Xu XY, Wang WS, Zhang QM, et al. Performance of common imaging techniques vs serum biomarkers in assessing fibrosis in patients with chronic hepatitis B: a systematic review and meta-analysis. *World J Clin Cases* 2019;7(15):2022–2037
- Dietrich CF, Bamber J, Berzigotti A, et al. EFSUMB guidelines and recommendations on the clinical use of liver ultrasound elastography, update 2017 (long version). *Ultraschall Med* 2017;38(04):e16–e47
- European Association for the Study of the Liver. Electronic address: easloffice@easloffice.eu; Clinical Practice Guideline Panel; Chair; EASL Governing Board representative; Panel members. EASL Clinical Practice Guidelines on non-invasive tests for evaluation of liver disease severity and prognosis - 2021 update. *J Hepatol* 2021;75(03):659–689
- Ferraioli G, Wong VW, Castera L, et al. Liver ultrasound elastography: an update to the world federation for ultrasound in medicine and biology guidelines and recommendations. *Ultrasound Med Biol* 2018;44(12):2419–2440
- Su TH, Liao CH, Liu CH, et al. Acoustic radiation force impulse US imaging: liver stiffness in patients with chronic hepatitis b with and without antiviral therapy. *Radiology* 2018;288(01):293–299
- Harbin WP, Robert NJ, Ferrucci JT Jr. Diagnosis of cirrhosis based on regional changes in hepatic morphology: a radiological and pathological analysis. *Radiology* 1980;135(02):273–283
- Awaya H, Mitchell DG, Kamishima T, Holland G, Ito K, Matsumoto T. Cirrhosis: modified caudate-right lobe ratio. *Radiology* 2002;224(03):769–774
- Ito K, Mitchell DG, Kim MJ, Awaya H, Koike S, Matsunaga N. Right posterior hepatic notch sign: a simple diagnostic MR finding of cirrhosis. *J Magn Reson Imaging* 2003;18(05):561–566
- Ito K, Mitchell DG, Gabata T, Hussain SM. Expanded gallbladder fossa: simple MR imaging sign of cirrhosis. *Radiology* 1999;211(03):723–726
- Watanabe A, Ramalho M, AlObaidy M, Kim HJ, Velloni FG, Semelka RC. Magnetic resonance imaging of the cirrhotic liver: an update. *World J Hepatol* 2015;7(03):468–487
- Aubé C, Bazeries P, Lebigot J, Cartier V, Boursier J. Liver fibrosis, cirrhosis, and cirrhosis-related nodules: imaging diagnosis and surveillance. *Diagn Interv Imaging* 2017;98(06):455–468NCPF
- Toros AB, Gokcay S, Cetin G, Ar MC, Karagoz Y, Kesici B. Portal hypertension and myeloproliferative neoplasms: a relationship revealed. *ISRN Hematol* 2013;2013:673781
- Sarin SK, Kumar A, Chawla YK, et al; Members of the APASL Working Party on Portal Hypertension. Noncirrhotic portal fibrosis/idiopathic portal hypertension: APASL recommendations for diagnosis and treatment. *Hepatol Int* 2007;1(03):398–413
- Nakanuma Y, Tsuneyama K, Ohbu M, Katayanagi K. Pathology and pathogenesis of idiopathic portal hypertension with an emphasis on the liver. *Pathol Res Pract* 2001;197(02):65–76
- Arora A, Sarin SK. Multimodality imaging of obliterative portal venopathy: what every radiologist should know. *Br J Radiol* 2015;88(1046):20140653
- Furuichi Y, Moriyasu F, Taira J, et al. Noninvasive diagnostic method for idiopathic portal hypertension based on measurements of liver and spleen stiffness by ARFI elastography. *J Gastroenterol* 2013;48(09):1061–1068
- Glatard AS, Hillaire S, d'Assignies G, et al. Obliterative portal venopathy: findings at CT imaging. *Radiology* 2012;263(03):741–750
- Krishnan P, Fiel MI, Rosenkrantz AB, et al. Hepatoportal sclerosis: CT and MRI appearance with histopathologic correlation. *AJR Am J Roentgenol* 2012;198(02):370–376
- Waguri N, Suda T, Kamura T, Aoyagi Y. Heterogeneous hepatic enhancement on CT angiography in idiopathic portal hypertension. *Liver* 2002;22(03):276–280
- Sarin SK, Khanna R. Non-cirrhotic portal hypertension. *Clin Liver Dis* 2014;18(02):451–476
- Duseja A. Portal cavernoma cholangiopathy: clinical characteristics. *J Clin Exp Hepatol* 2014;4(Suppl (Suppl 1)):S34–S36

- 30 Dhiman RK, Saraswat VA, Valla DC, et al. Portal cavernoma cholangiopathy: consensus statement of a working party of the Indian national association for study of the liver. *J Clin Exp Hepatol* 2014;4(Suppl (Suppl 1)):S2–S14
- 31 Gupta P, Kalra N, Gulati A, et al. Changes in liver morphology in patients with extrahepatic portal venous obstruction: a retrospective magnetic resonance imaging study. *Br J Radiol* 2019;92(1098):20180890
- 32 Pargewar SS, Desai SN, Rajesh S, Singh VP, Arora A, Mukund A. Imaging and radiological interventions in extra-hepatic portal vein obstruction. *World J Radiol* 2016;8(06):556–570
- 33 Shin SM, Kim S, Lee JW, et al. Biliary abnormalities associated with portal biliopathy: evaluation on MR cholangiography. *AJR Am J Roentgenol* 2007;188(04):W341–7
- 34 Llop E, de Juan C, Seijo S, et al. Portal cholangiopathy: radiological classification and natural history. *Gut* 2011;60(06):853–860
- 35 Cura M, Haskal Z, Lopera J. Diagnostic and interventional radiology for Budd-Chiari syndrome. *Radiographics* 2009;29(03):669–681
- 36 Brancatelli G, Vilgrain V, Federle MP, et al. Budd-Chiari syndrome: spectrum of imaging findings. *AJR Am J Roentgenol* 2007;188(02):W168–76
- 37 Ferral H, Behrens G, Lopera J. Budd-Chiari syndrome. *AJR Am J Roentgenol* 2012;199(04):737–745
- 38 Das CJ, Soneja M, Tayal S, et al. Role of radiological imaging and interventions in management of Budd-Chiari syndrome. *Clin Radiol* 2018;73(07):610–624
- 39 Bansal V, Gupta P, Sinha S, et al. Budd-Chiari syndrome: imaging review. *Br J Radiol* 2018;91(1092):20180441
- 40 Philips CA, Arora A, Shetty R, Kasana V. A comprehensive review of portosystemic collaterals in cirrhosis: historical aspects, anatomy, and classifications. *Int J Hepatol* 2016;2016:6170243
- 41 Sharma M, Rameshbabu CS. Collateral pathways in portal hypertension. *J Clin Exp Hepatol* 2012;2(04):338–352
- 42 Arora A, Rajesh S, Meenakshi YS, Sureka B, Bansal K, Sarin SK. Spectrum of hepatofugal collateral pathways in portal hypertension: an illustrated radiological review. *Insights Imaging* 2015;6(05):559–572
- 43 McIndoe AH. Vascular lesions of portal cirrhosis. *Arch Pathol (Chic)* 1928;5:23–40
- 44 Bandali MF, Mirakhor A, Lee EW, et al. Portal hypertension: Imaging of portosystemic collateral pathways and associated image-guided therapy. *World J Gastroenterol* 2017;23(10):1735–1746
- 45 Cho O-K, Koo JH, Kim YS, Rhim HC, Koh BH, Seo HS. Collateral pathways in Budd-Chiari syndrome: CT and venographic correlation. *AJR Am J Roentgenol* 1996;167(05):1163–1167
- 46 Berzigotti A, Seijo S, Reverter E, Bosch J. Assessing portal hypertension in liver diseases. *Expert Rev Gastroenterol Hepatol* 2013;7(02):141–155
- 47 Zwiebel WJ. Sonographic diagnosis of hepatic vascular disorders. *Semin Ultrasound CT MR* 1995;16(01):34–48
- 48 Berzigotti A, Ashkenazi E, Reverter E, Abraldes JG, Bosch J. Non-invasive diagnostic and prognostic evaluation of liver cirrhosis and portal hypertension. *Dis Markers* 2011;31(03):129–138
- 49 Indiran V, Vinod Singh N, Ramachandra Prasad T, Maduraimuthu P. Does coronal oblique length of spleen on CT reflect splenic index? *Abdom Radiol (NY)* 2017;42(05):1444–1448
- 50 Wachsberg RH, Bahramipour P, Sofocleous CT, Barone A. Hepatofugal flow in the portal venous system: pathophysiology, imaging findings, and diagnostic pitfalls. *Radiographics* 2002;22(01):123–140
- 51 Kang HK, Jeong YY, Choi JH, et al. Three-dimensional multi-detector row CT portal venography in the evaluation of portosystemic collateral vessels in liver cirrhosis. *Radiographics* 2002;22(05):1053–1061
- 52 Leyendecker JR, Rivera E Jr, Washburn WK, Johnson SP, Diffin DC, Eason JD. MR angiography of the portal venous system: techniques, interpretation, and clinical applications. *Radiographics* 1997;17(06):1425–1443
- 53 Fehrenbach U, Gül-Klein S, de Sousa Mendes M, et al. Portosystemic shunt surgery in the era of TIPS: imaging-based planning of the surgical approach. *Abdom Radiol (NY)* 2020;45(09):2726–2735
- 54 Kirby JM, Cho KJ, Midia M. Image-guided intervention in management of complications of portal hypertension: more than TIPS for success. *Radiographics* 2013;33(05):1473–1496
- 55 Keshava SN, Kota GK, Mammen T, et al. Direct intrahepatic cavoportal shunts in Budd-Chiari syndrome: role of simultaneous fluoroscopy and trans-abdominal ultrasonography. *Indian J Gastroenterol* 2006;25(05):248–250
- 56 Keshava SN, Moses V, Sharma A, et al. Technical and medium-term clinical outcomes of transjugular intrahepatic portosystemic shunt with fluoroscopy and additional trans-abdominal ultrasound guidance. *Indian J Radiol Imaging* 2021;31(04):858–866
- 57 Choi YH, Yoon CJ, Park JH, Chung JW, Kwon JW, Choi GM. Balloon-occluded retrograde transvenous obliteration for gastric variceal bleeding: its feasibility compared with transjugular intrahepatic portosystemic shunt. *Korean J Radiol* 2003;4(02):109–116
- 58 Pereira P, Peixoto A. Left-sided portal hypertension: a clinical challenge. *GE Port J Gastroenterol* 2015;22(06):231–233
- 59 Sharma A, Keshava SN, Eapen A, Elias E, Eapen CE. An update on the management of Budd-Chiari syndrome. *Dig Dis Sci* 2021;66(06):1780–1790
- 60 Keshava SN, Moses V, Surendrababu NR. Cannula-assisted and transabdominal ultrasound-guided hepatic venous recanalization in Budd Chiari syndrome: a novel technique to avoid percutaneous transabdominal access. *Cardiovasc Intervent Radiol* 2009;32(06):1257–1259
- 61 Ahmed M, Keshava SN, Moses V, et al. Transjugular intrahepatic portosystemic shunt through the strut of a previously placed stent: technical feasibility and long-term follow-up results. *Cardiovasc Intervent Radiol* 2018;41(11):1794–1798
- 62 Won JY, Choi SY, Ko HK, et al. Percutaneous peritoneovenous shunt for treatment of refractory ascites. *J Vasc Interv Radiol* 2008;19(12):1717–1722