Pictorial Essay: Multimodality Imaging of the Portal Venous System
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Citation

Abstract
Evaluation of the portal venous system is required in several clinical circumstances. Multiple imaging modalities may be employed for evaluation of the portal venous system. To facilitate a definitive diagnosis the normal anatomy of the portal venous system as well as congenital and acquired abnormalities must be understood. This pictorial essay will review the various imaging appearances of the portal venous system.

NORMAL ANATOMY AND VARIANTS
The portal venous system is composed of three main tributaries, the splenic, superior mesenteric and inferior mesenteric veins as well as several smaller tributaries. The left gastric or coronary vein is the most important of the smaller tributaries and joins the portal vein near its junction with the splenic vein. The normal portal vein divides into left and right lobar veins.

Congenital absence or duplication of the portal vein is rarely seen. (Fig. 1).

FIGURE 1: ANOMALOUS ORIGIN OF THE LEFT PORTAL VEIN

Figure 1
Figure 1a: Power Doppler images demonstrate anomalous origin of the left portal vein (arrow) from the right portal vein.
Spectral waveforms of portal veins are monophasic with hepatopetal flow as opposed to the hepatic veins, which demonstrate cardiac modulation and hepatofugal flow.

First described by Abernethy in 1793, congenital vascular shunts between the main portal vein or its central branches and the inferior vena cava are extremely rare. Two types of congenital porta-caval shunts have been described. Type I shunts include congenital absence of the portal vein with redirection of portal blood directly into the inferior vena cava. Children with this specific anomaly are almost always girls, who often have multiple concurrent congenital anomalies. Type II anomalies consist of a side-to-side primarily extrahepatic connection between the inferior vena cava and an otherwise normal portal vein. Type II anomalies are seen primarily in male patients and are not usually associated with other anomalies. A portosystemic venous shunt is a direct communication between the main portal vein or its proximal branches and the hepatic vein. No known definitive developmental cause has been identified for congenital portacaval shunts. The portal venous system is formed from the vitelline veins, the umbilical veins, and the ductus venosus. Embryologically, the right vitelline vein is instrumental in connecting the extrahepatic portal vein to the intrahepatic portal vein; therefore. Any abnormality in development of the aforementioned structures could lead to a congenital portacaval shunt. Either intrahepatic or extrahepatic. Persistence of the ductus venosus has been offered as a possible etiology for communication between the left portal vein and the inferior vena cava. Hepatic encephalopathy may occur in children with true congenital portacaval shunts due to elevated blood levels of ammonia (Fig. 2) [1].

**FIGURE 2: CONGENITAL PORTACAVAL SHUNT**

This 7-year-old patient had history of elevated blood ammonia levels.

**Figure 3**

Figure 2a: Transverse Gray scale sonographic image demonstrate absence of the left and right portal vein branches with echogenic tracts (arrows) seen in these areas. The portal vein is enlarged.

**Figure 4**

Figure 2b: Sagittal Gray scale sonographic image shows communication of the Portal vein (P) with the Inferior Vena Cava (IVC).
PORTAL VEIN ANEURYSM

There is no clear definition of the portal vein aneurysm and this is likely due to the considerable variation in the size of the portal vein. However, dilation beyond 2 cm may be termed aneurysmal ectasia, especially when a fusiform or a saccular configuration is assumed [1]. Congenital and acquired etiologies have been described. Predominantly asymptomatic, large aneurysms may give rise to symptoms of duodenal compression, common bile duct compression with jaundice and portal hypertension with formation of collaterals (Fig. 3).

FIGURE 3: PORTAL VEIN ANEURYSM

A. B. Gray scale and Color Doppler images demonstrate aneurysmal dilatation of the portal vein (arrow).

PORTAL VEIN OCCLUSION AND PORTAL HYPERTENSION

PORTAL VEIN THROMBOSIS

Portal vein thrombosis often occurs silently but may result in
acute hepatic deterioration, acute gastrointestinal hemorrhage, or the acute onset of ascites. Etiologies include: portal hypertension and stasis; hypercoagulable states; anti-phospholipid antibodies; sepsis; pancreatitis; mass effect and tumor invasion.

Ultrasound diagnostic criteria include venous distension, clot within the lumen and diminished phasicity (Fig. 4).

**FIGURE 4: PORTAL VENOUS THROMBOSIS**

**Figure 9**
Figure 4a: CECT shows hypodense thrombus in the right portal vein (arrow) in this patient with a liver metastases (arrowhead) from colon cancer. Multiple metastases are also seen in the liver.

**Figure 10**
Figure 4b: Color Doppler US image from a different patient with pancreatitis shows echogenic thrombus (arrow) in the right portal vein with minimal flow detected. Thrombus was also seen in the left portal vein.

However, portal hypertension alone without thrombosis can
B, C, D. T1 W, T2 W and GRE TOF images show abnormal signal within the portal vein (arrow) suggesting thrombus with collaterals seen on the TOF image (arrowheads).

**Figure 15**

Figure 5e: In another patient post contrast MIP images show complete occlusion of the middle portal vein.

**PORTAL HYPERTENSION PRESINUSOIDAL**

Presinusoidal venous hypertension often occurs in a patient with normal liver function when the portal vein and its branches are compressed before they enter the liver.

**PANCREATIC CANCER AND CHOLANGIOCARCINOMA**

Pancreatic cancer frequently only becomes clinically apparent at an advanced, unresectable stage due to tumor extension and metastases. Computed Tomography and MRI shows extension of splenic or portal venous thrombosis from the pancreatic tumor with or without cavernous transformation, or arterial and portal venous encasement without thrombosis (Fig. 6).

**FIGURE 6: PANCREATIC CANCER INVADES THE PORTAL VENOUS SYSTEM.**
Figure 16
Figure 6a: 3D GRE VIBE axial post contrast image in a patient with adenocarcinoma of the pancreas (arrow) encasing the portal vein (arrowhead).

Figure 17
Figure 6b: Portal vein encasement is best appreciated on the post contrast volumetrically rendered image (arrow).

Cholangiocarcinoma also invades the portal venous system and MRCP imaging demonstrates involvement of the portal venous system and biliary tree (Fig. 7).

FIGURE 7: CHOLANGIOCARCINOMA INVADES THE PORTAL VENOUS SYSTEM.

Figure 18
Figure 7a: 3D gradient echo post contrast VIBE image, 15 min delay, demonstrates a enhancing mass lesion in the region of the porta hepatis with encasement of the portal vein and involvement of the right hepatic duct (arrow).

Figure 19
Figure 7b: Coronal reconstruction from an axial dataset of a portal venous phase image demonstrates the tumor encasing the portal vein (arrow).
Pictorial Essay: Multimodality Imaging of the Portal Venous System

Figure 20
Figure 7c: Post processed Volumetrically rendered image from a portal venous phase clearly demonstrates the cholangiocarcinoma encasing the portal vein (arrow). 3D reconstruction demonstrate invasion of the portal vein (arrow) by cholangiocarcinoma.

PANCREATITIS
Pancreatitis may lead to vascular complications including venous occlusion and formation of pseudoaneurysms. The splenic vein is particularly vulnerable. Recognition of collateral flow via the short gastric veins gastroepiploic veins aids in differentiation. Ultimately cavernous transformation may develop (Fig. 8).

FIGURE 8: CAVERNOUS TRANSFORMATION.

Figure 21
Figure 8a: FSE T2 weighted fat saturated axial image through the liver demonstrates multiple areas of flow void in the region of the porta hepatis and the expected location of the portal vein (arrowheads).

Figure 22
Figure 8b: These areas demonstrate contrast enhancement as seen on the post contrast gradient echo axial image (arrowheads).

Figure 23
Figure 8c: Post contrast CT image in a different patient demonstrates multiple collateral vessels in the region of the portal vein consistent with cavernous transformation (arrowheads).

SEPTIC THROMBOSIS
Inflammatory processes that drain via the portal vein such as appendicitis, diverticulitis, inflammatory or ischemic bowel disease and cholecystitis may lead to presinusoidal portal vein thrombosis. Patients with liver abscesses require a careful evaluation of the portal venous system for a source of infection. Thrombosis of the portal venous system must be excluded if symptoms persist during treatment for sepsis.

MISCELLANEOUS
A wide variety of entities are may be seen in association with portal venous thrombosis, including protein C or S...
deficiency, excessive factor VIII administration, Antithrombin III deficiency and oral contraceptive use. Myeloproliferative conditions such as polycythemia vera and myelofibrosis or paraneoplastic syndromes are also hypercoagulable states. Portal venous thrombosis can also complicate any abdominal surgery, most commonly after splenectomy. After liver transplantation hepatic arterial thrombosis is more common than portal venous thrombosis. This usually arises at the venous anastomosis from stenosis due to endothelial injury (Fig. 9).

FIGURE 9: PORTAL VEIN STENOSIS

Figure 24
Figure 9a: 3D gradient echo post contrast projection image demonstrates narrowing of the portal vein (arrow).

Figure 25
Figure 9b: The narrowing can be appreciated on the oblique projection image (arrow).

Figure 26
Figure 9c: In another patient post liver transplant with portal vein stenosis, angiographic image demonstrates the stenosis (arrow), with contrast injected through the transjugular route.
Figure 27
Figure 9d: Post angiographic dilatation. Note the improvement in the stenosis subsequent to angioplasty dilatation (arrow).

**Figure 28**
Figure 10a: Gradient echo 3D post contrast axial VIBE image demonstrates multiple enhancing mass lesions (arrowheads) in a patient with a cirrhotic background, consistent with hepatocellular carcinomas.

**SINUSOIDAL**
Portal hypertension and portal vein thrombosis are most commonly caused by disease at the sinusoidal level of the liver. Cirrhosis causes fibrotic obliteration of the normal sinusoidal architecture and may be due to multiple causes, alcohol abuse, chronic hepatitis, primary biliary cirrhosis and sclerosing cholangitis. Patients with portal hypertension secondary to sinusoidal liver disease demonstrate the features of liver disease usually clinically and imaging shows a small cirrhotic liver, splenomegaly, ascites, and varices or venous collaterals are often seen. Portal hypertension may produce recurrent episodes of life threatening variceal hemorrhage or intractable ascites.

**HEPATOCELLULAR CARCINOMA**
The majority of hepatocellular carcinomas (HCC) are unresectable at the time of presentation. Invasion of the portal vein occurs in up to 70% of cases. Involvement of the main portal vein by tumor makes resection impossible as no surgical technique allows for bypass of this critical structure.

A hepatic mass or neovascularity within the tumoral thrombus allows delineation of the malignant etiology. In addition, the development of periportal arteries feeding the tumor thrombus and running parallel to the thrombosed portal vein may be seen (Fig. 10).

**Figure 29**
Figure 10b: Coronal multiplanar reconstruction of a portal venous phase image demonstrate the tumor extension into the portal vein (arrow) with expansion.

**FIGURE 10: MULTICENTRIC HEPATOCELLULAR CARCINOMA WITH PORTAL VEIN OCCLUSION.**

Abnormalities of liver attenuation both before and during contrast administration are common in HCC. Abnormalities seen on unenhanced scans reflect local changes due to edema, glycogen depletion, and fatty infiltration, whereas abnormal enhancement during the bolus phase of contrast enhancement reflects changes in both arterial and portal flow. These abnormalities, may lead to an overestimation of the size of the tumor and selection of an inappropriate
location for biopsy.

POSTSINUSOIDAL
Post sinusoidal occlusion or obstruction to the flow of blood from the liver occurs in such conditions as Budd-Chiari syndrome, vena caval thrombosis or stenosis, and venoocclusive disease related to therapy during bone marrow transplantation.

BUDD-CHIARI SYNDROME
Hepatic venous occlusion (Budd-Chiari syndrome) is uncommon and typically occurs with ascites, hepatomegaly and abdominal pain. Two thirds of cases have no known cause but other causes include hypercoagulable states.

CT demonstrates a markedly abnormal pattern of hepatic enhancement, with dense early enhancement of the caudate lobe and portions of the left. Portal venous thrombosis must be excluded as this precludes transplantation [4, 5].

PSEUDOTHROMBUS
Laminar flow during the arterial dominant phase of helical dynamic CT scanning is a common flow related artifact producing a pseudothrombus of the portal vein. Repeat imaging resolves this diagnostic problem [3].

COLLATERAL PATHWAYS IN SEGMENTAL OCCLUSION AND PORTOSYSTEMIC SHUNTS.

PERIPANCREATIC VARICES
If cancer involving the he pancreatic head encases the confluence of the portal and superior mesenteric veins, the posterior superior pancreaticoduodenal vein may dilate.

GALLBLADDER AND COMMON BILE DUCT VARICES
Gallbladder and common bile duct varices are also associated with portal vein thrombosis. Dilated epicholedochal venous plexus and veins of sappey should not be confused with wall edema as they drain into the right posterior portal vein (Fig. 11) [6].

In this patient with a history of portal hypertension color Doppler ultrasound demonstrates multiple vascular areas of increased flow within the gallbladder wall (arrows).

ESOPHAGEAL, PARAESOPHAGEAL, AND CORONARY GASTRIC COLLATERAL VESSELS
Traversing the gastrohepatic ligament, a triangular space between the lesser curve of the stomach and the left lobe of the liver, small venous branches along the lesser curve drain into the coronary vein. The coronary vein joins the portal venous system at or near the portal confluence. Vessels larger than 6 mm in the gastrohepatic ligament suggest the presence of varices. On contrast-enhanced study, gastroesophageal varices manifest as high attenuation structures along the lesser curvature of the stomach and are also seen in the esophageal wall (Fig. 12). [3, 5].

FIGURE 12: GASTROESOPHAGEAL VARICES, GASTROHEPATIC AND SPLENIC.

INFERIOR PHRENIC COLLATERAL VEINS PORTOSYSTEMIC SHUNTS
Shunts through the inferior phrenic veins are rare, following their corresponding arteries on the inferior surface of the diaphragm. The right inferior phrenic vein ends at the inferior vena cava whilst the left inferior phrenic vein is often bifurcated. Normally gastroesophageal veins form anastomoses with the left inferior phrenic vein and this may
therefore function as a collateral venous pathway toward the inferior vena cava.

PARAUMBILICAL VEIN AND ANTERIOR ABDOMINAL WALL COLLATERAL VESSELS

The paraumbilical vein arises from the left portal vein and traverses the falciform ligament, eventually draining into the veins of the anterior abdominal wall, producing a “Medusa’s head” appearance. Circular or tubular structures, more than 2 mm in diameter are seen between the medial and lateral segments of the left hepatic lobe at the anterior edge of the falciform ligament (Fig.13).

FIGURE 13: RECANALIZED PARAUMBILICAL VEIN

|image:34|
|image:35|

SPLENORENAL SHUNTS

These collateral vessels originate from the splenic hilum, course medial to the spleen toward the left renal hilum, to anastomose with the left renal vein. Occasionally, splenorenal collateral vessels course behind the spleen and the left kidney producing serpiginous varicosities. Splenorenal collateral pathway may also be surgically created to lower portal venous pressure.

GASTRORENAL SHUNTS

Gastric varices that drain into the esophageal or paraesophageal veins occasionally drain into the left renal vein via a gastrorenal shunt.

RETROCAVAL SHUNTS

These collateral vessels are uncommon and may manifest as nodular or massive lesions in the paraaortic or paracaval spaces. Confusion may occur with other entities such as neurofibromas, lymphangiectasia, or lymphadenopathy.

MESOCAVAIAL SHUNTS WITH INFERIOR MESENTERIC VARICES

These vessels flow into systemic circulations via rectal, pararectal, or hemorrhoidal varices and may present as lower gastrointestinal tract bleeding. If the inferior mesenteric vein is the collateral channel, it occasionally forms mesenteric varices and flows directly into the inferior vena cava [3, 7].

CAVERNOUS TRANSFORMATION OF THE PORTAL VEIN

Cavernous transformation of the portal vein or portal cavernoma is defined as a mass like network of intertwined veins in the heptoduodenal ligament and porta hepatis that acts as an alternative pathway in cases of thrombosis or stenoses of the portal vein or its branches. Development of venous collaterals occurs fairly rapidly and may occur within a 6-week period. The thrombosed portal segment is usually not seen and is replaced by multiple enhancing smaller veins (Fig.8) [3, 7].

ALTERATIONS IN HEPATIC ATTENUATION AND PERFUSION DUE TO PORTAL VENOUS OBSTRUCTION

Contrast laden hepatic arterial blood directly from the aorta reaches the liver before contrast laden portal venous blood, which must first pass through the capillary bed of the intestines and the sinusoids of the spleen. Therefore the density of the liver is affected during all phases of contrast administration. Also in patients with portal vein thrombosis, well defined lobar or segmental hyperperfusion abnormalities with straight boundaries as opposed to round, mass like margins are seen during the arterial phase of spiral dynamic and multiphasic dynamic MR imaging [3, 4].

PORTAL VENOUS GAS

Portomesenteric vein gas is most commonly caused by mesenteric ischemia but may have a variety of other causes, and is idiopathic in approximately 15% of cases. CT imaging shows tubular areas of decreased attenuation in the liver, predominantly in the left lobe. Intrahepatic portal vein gas must be differentiated from air pneumobilia, which also has a left lobe predilection. However collections of portal vein gas are smaller and more numerous and are located in the periphery (Fig. 14) [8].

FIGURE 14: PORTAL VENOUS GAS

|image:36|
|image:37|
|image:38|

B, C. CT demonstrates air in the biliary system from gallstone ileus not to be mistaken for air in the portal venous system.

INTRA HEPATIC PORTOSYSTEMIC VENOUS SHUNTS

Intrahepatic spontaneous shunts may be congenital or acquired. They are categorized according to their intrahepatic course as (1) inferior vena caval shunts between
the posterior branch of the right portal vein and the IVC, (2) paraumbilical shunts between the left portal vein and the paraumbilical vein or (3) miscellaneous shunts (Fig.15).

**FIGURE 15: HEPATIC VENOUS/VENOUS AND HEPATIC/PORTAL COMMUNICATION**

![Image 39](image:39)

![Image 40](image:40)

In this patient with congestive heart failure, passive hepatic venous congestion is seen with spontaneous communication between the right and middle hepatic veins (arrows). Gray scale (A) and power Doppler (B) images are shown.

**ARTERIOPORTAL SHUNTS**

Hepatic tumors, liver cirrhosis, trauma and interventional procedures may all give rises to the formation of shunts (Fig.16).

**FIGURE 16: IATROGENIC ARTERIOVENOUS FISTULA**

![Image 41](image:41)

![Image 42](image:42)

![Image 43](image:43)

Selective Celiac axis injection shows early enhancement of the portal vein branches, indicating the presence of an arterioportal shunt. This patient had a history of multiple liver biopsies.

Portal flow is reversed or reduced. Doppler findings include a dilated hepatic artery more than 10 mms, hepatic artery and portal venous shunts with pulsatile portal flow and in hepatic artery and hepatic vein shunts waveform changes may be seen in severe cases. Dynamic CT imaging shows early enhancement of the affected portal vein, transient lobar or segmental hyperperfusion anomalies and dilated intrahepatic vessels with surrounding irregularly enhanced vessels. Transient wedge shaped enhancement may also be seen peripherally to a tumor.

**TRANSJUGULAR INTRAHEPATIC PORTOSYSTEMIC SHUNTS (TIPS)**

TIPS is the method of choice for decompression of the hypertensive portal system. Portal decompression is achieved through a percutaneously established shunt with expandable metallic stent placed between the hepatic and portal veins within the liver.

A baseline study should be obtained 24 hours post TIPS looking for portal hypertension. Functional shunts show portosystemic gradients less than 15 mm hg with no bleeding or ascites. Thrombosis of the TIPS is usually the result of a bile leak (Fig.17) [3].

**FIGURE 17: TIPS THROMBOSIS**

![Image 44](image:44)

![Image 45](image:45)

![Image 46](image:46)

**CONCLUSION**

Evaluation of the portal venous system is required in several clinical circumstances. Multiple imaging modalities are now available for evaluation of the portal venous system. A variety of techniques including arterial and transhepatic portography, splenoportography, computed tomography, and ultrasound are currently available for evaluation of the portal venous system. Selection of a modality is multifactorial, and considerations such as cost, availability, accuracy, invasiveness of the procedure and local expertise with the procedure must be included in any decision making process. Some of these methods are limited by flow dynamics, which may be altered by portal hypertension. A wide variety of portal venous abnormalities may be detected at cross-sectional imaging. In order to make a definitive diagnosis the normal anatomy of the portal venous system and the various imaging appearances of acquired abnormalities must be understood. Ultrasound is noninvasive and relatively inexpensive.

Helical volumetric CT and fast gradient echo MR techniques makes it possible for imaging to be completed during the vascular phase of a contrast enhanced study within a single breath hold clearly depicting the anatomic characteristics of vascular structures. However CT involves the use of contrast agents and ionizing radiation whereas MRI allows blood flow to be imaged noninvasively in a multiplanar format irrespective of direction of flow and without contrast agents or ionizing radiation.

**References**

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