Portosystemic collateral vessels and intrahepatic shunts: Radiological revision

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Learning objectives

Reviewing the pathophysiology of the intrahepatic shunts and the collateral venous systemic.

To exhibit the role of different non-invasive imaging techniques in the diagnostic.

Distinguish the tumor and non-malignant conditions associated with these entities.

Background

Result of the major technological advances in multidetector CT and MRI have been achieved some major improvements in the assessment of hepatic vascularisation.

1. Arterioportal shunts: is the most common pathogenesis with varied (macroscopic fistula, transinusoidal, transvasal, transtumoral and peribiliar), in general secondary malignancies (hemangioma, liver cancer and colangiocarcinoma), interventional procedures or trauma, liver cirrhosis and other less common.

2. Portosystemic venous shunts: considered relatively physiological portal hypertension in patients with congenital while others would be between hepatic portal vein and vein through an aneurysm.

3. Arteriosystemic shunts: are rare and we can see them on the disease Rendu-Osler-Weber and tumors (hepatocellular carcinoma and hemangioma).


The most common are the arteriportal shunts with opacification early portals intrahepatic branches in arterial phase before filling the main door and a transitional liver parenchyma enhancement (THPE) morphology trapezoidal visible in early stages.
PORTOSYSTEMIC VENOUS SHUNTS

Intrahepatic portosystemic (portal to hepatic) venous shunts are uncommon. Pustulated congenital origins of portosystemic shunts include the persistence of an omphalomesenteric venous system with the right horn of the sinus venosus or rupture of a portal vein aneurysm into a hepatic vein (Figures 1 to 6) (Figures 7 to 10). Acquired conditions such as trauma and portal hypertension are also theorized.

Portosystemic shunts are typically identified incidentally as part of the workup for cirrhosis (Figures 11, 12, and 13) or, less commonly, after a patient's condition is diagnosed as hepatic encephalopathy.

Patients with severe portal hypertension can develop extensive portosystemic collaterals draining directly into the inferior vena cava by way of a subcapsular route. Sonography can identify the communication between the portal vein and the hepatic or systemic veins (Figure 14 on page 20).

Park et al have categorized intrahepatic portosystemic shunts into four morphologic types.

The first and most common type consist of a single large tube that connects the right portal vein to the inferior venous cava.

The second type is a localized peripheral shunt in which one or more communications are found in a single hepatic segment.

The third type is a portosystemic shunt through an aneurysm.

The fourth type has multiple communications between peripheral portal and hepatic veins in several segments.

A persistent ductus venosus could be considered as a fifth type of portosystemic shunt.

ARTERIOPORTAL SHUNTS
Arterioportal shunts occur in liver disorders such as cirrhosis, trauma, congenital vascular malformations, and hepatic neoplasms.

Arterioportal shunting associated with cirrhosis (Figures 15, and 16) or hepatocellular carcinoma (Figures 17, and 18) (HCC) is also documented.

The degree of shunting is greater with neoplasm than with cirrhosis and even greater with large tumors. Research shows that the prevalence of arterioportal shunting in HCC is as high as 63% of patients. The hepatic artery supplies HCC almost exclusively, providing the intense early enhancement seen in arterial phase imaging.

The CT or MR imaging findings of arterioportal shunting include early and prolonged enhancement of the portal vein, transient wedge-shaped enhancement peripheral to the tumor (if one is present), and dilated intrahepatic vessels during arterial phase imaging.

Superparamagnetic iron oxide (enhanced MR imaging is clinically useful for differentiating most nontumorous arterioportal shunts from hypervascular tumors, and it is also useful for distinguishing timorous arterioportal shunts from nontumorous arterioportal shunts, especially of T2-weighted gradient-echo images. However, it is necessary to pay attention to the overlaps between the two types of arterioportal shunts.

Intrahepatic nontumorous arterioportal shunts can be idiopathic, resulting from penetrating trauma, or iatrogenic after liver biopsy (Figure 19) on page 25 or percutaneous catheterization of the bile ducts.

Idiopathic intrahepatic nontumoral arterioportal shunts are typically subcapsular or peripheral as seen angiographically or on arterial phase helical CT. They appear as focal increased-attenuation wedge-shaped defects with early portal venous filling (Figure 20 on page 26). Additionally, small idiopathic nontumoral arterioportal shunts have been shown to simulate small hypervascular masses such as HCC, focal nodular hyperplasia, and hemangiomas.

Infrequently, intrahepatic arterioportal shunts may cause life-threatening portal hypertension. Surgical interventions in necessary only when interventional radiologic procedures such as embolization have failed. The shunt or fistula between the hepatic artery and the portal vein is rarely visualized on CT.

Arterioportal shunts are rarely reported in association with hepatic hemangiomas (Figures 21, and 22). The characteristic appearance of hemangiomas on CT or MR imaging, coupled with the identification of a dilated feeding artery and early portal venous filling,
should lead to the correct diagnosis. When subcapsular, small hemangiomas may simulate a small arterioportal shunt without a coexisting lesion. Peripheral wedge-shaped regions of increased perfusion that become isodense or isointense on delayed images can be seen with small hemangiomas an arterioportal shunts.

Congenital arterioportal shunts are a rare cause of portal hypertension. They can be associated with hereditary hemorrhagic telangiectasia, Ehlers-Danlos syndrome, and biliary atresia, although in most case reports they are not associated with any other disease.

The helical TC findings of arterioportal shunts are as follows: early enhancement of the peripheral portal vein branches during the hepatic arterial phase and before the main portal vein is enhanced, enhancement of the peripheral portal vein branches and main portal vein without enhancement of the superior mesenteric and splenic veins, an appearance that has been considered diagnostic on hepatic angiograms; and transient, peripheral, wedge-shaped hepatic parenchymal enhancement during the hepatic arterial phase (THPE).

**ARTERIOSYSTEMIC SHUNTS**

Arteriosystemic shunts involving the liver are rare and typically associated with benign and malignant neoplasms. The first description of arteriosystemic venous shunting with a cavernous hemangioma was seen in the setting of HCC and arterioportal shunting.

The actual incidence of arteriosystemic shunting in the setting of HCC is unknown, but the incidence of hepatic venous invasion by tumor is thought to be approximately 15%. Therefore, when arteriosystemic shunting to the hepatic veins is observed, invasion of the hepatic veins by HCC should be suspected.

Hereditary hemorrhagic telangiectasia or Osler-Weber-Rendu disease is an autosomal dominant disorder that may involve virtually every organ and is reported to involve the liver in 8-31% of patients. The hepatic manifestations of this disorder include vascular malformations, cirrhosis, and fibrosis.

Arterioportal or arteriosystemic venous shunts *(Figure 23)* on page 29 are also described as manifestations of this congenital disorder. However, identification of a specific abnormal communication in the presence of multiple hepatic vascular malformations can be challenging. The appearance of an early enhancing portal or
hepatic vein during the arterial phase of the CT or MR imaging examination is the clue to the diagnosis (Figure 24 on page 30).

PORTOSYSTEMIC COLLATERAL VESSELS (Figure 25) on page 31

Gastroesophageal varices

The most common and most clinically important portosystemic shunt is through gastroesophageal varices (Figures 26, and 27) The blood flow to gastroesophageal varices is predominantly from the left gastric vein (or coronary vein). This vein originates at the portal venous confluence, traverses the gastric fundus, and drains into the veins of the lower esophageal plexus.

Shunting also occurs from the splenic vein through the short gastric veins and into the esophageal plexus. These varices flow into the deep, intrinsic, longitudinal veins of the lower esophagus, which dilate and are responsible for the bleeding encountered in cases of gastroesophageal varices (Figure 28) on page 34. These veins can increase in size sixfold and carry up to a half liter of blood per minute.

With splenic vein thrombosis, collateral flow can course via a gastroepiploic varix through the stomach wall. The result can be variceal bleeding from isolated gastric varices.

Recanalized paraumbilical vein

The umbilical vein, which runs in the falciform ligament, atrophies after birth and becomes the ligamentum teres. Coursing through this ligament are additional smaller paraumbilical veins. These veins may hypertrophy in the presence of elevated portal venous pressure and are then known as recanalized paraumbilical vein (Figure 29). on page 35

Occasionally, a paraumbilical varix leads to enlargement of the superficial abdominal wall veins, causing the so-called caput medusa. However, caput medusa is a rare consequence of paraumbilical varix. The paraumbilical vein originates from the left portal vein, courses along the falciform ligament, and usually extends toward the umbilicus.

In utero, the umbilical vein penetrates the anterior abdominal wall at the umbilicus on its way from the placenta. With recanalization of the paraumbilical veins, which share this
course, the varix may appear as an umbilical hernia at physical examination. If it is just deep to the skin, it may be mistaken for herniated intestine at physical examination.

**Splenorenal and retroperitoneal varices**

Common thinking about splenorenal varices is they originate from the splenic hilum and travel directly to the renal vein. This situation is actually the exception. Most varices in the left flank are convoluted, travelling a great distance before communicating with the systemic circulation and not always communicating directly with the renal vein *(Figure 30 on page 36)*.

Varices originating from the splenic hiliar region may course cephalically toward the diaphragm and pass along the diaphragmatic surface posterior or lateral to the spleen, eventually communicating with the renal vein.

Occasionally it is possible to show communication with retroperitoneal space *(Figure 31)* on page 37.

**Other** *(Figure 32)* on page 38

**Images for this section:**
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Fig. 2: Figure 2. A incidental portosystemic venous shunt with an aneurysm vein. Sonography aspect
**Fig. 3:** Figure 3. A incidental portosystemic venous shunt with an aneurysm vein. Color Doppler sonography aspect
**Fig. 4**: Figure 4. A incidental portosystemic venous shunt with an aneurysm vein. Enhanced CT, images show the communication into hepatic vein and portal vein.
Fig. 5: Figure 5. A incidental portosystemic venous shunt with an aneurysm vein. Enhanced CT, images show the communication into hepatic vein and portal vein.
Fig. 6: Figure 6. A incidental portosystemic venous shunt with an aneurysm vein. Enhanced CT images show the communication into hepatic vein and portal vein.
**Fig. 7:** Figure 7. A incidental portosystemic venous shunt with an aneurysm vein. Non enhanced CT axial image
**Fig. 8:** Figure 8. A incidental portosystemic venous shunt with an aneurysm vein. Non enhanced CT sagittal image
**Fig. 9**: Figure 9. Ultrasound shows a large hypoechoic peripheral lesion
Fig. 10: Figure 10. Color doppler ultrasound demonstrated a big aneurysm in a portosystemic venous shunt.
**Fig. 11:** Figure 11. Enhance CT images shows at left hepatic lobe a communication into left hepatic vein and left portal vein. Portosystemic venous intrahepatic shunt
Fig. 12: Figure 12. Enhance CT images shows at left hepatic lobe a communication into left hepatic vein and left portal vein. Portosystemic venous intrahepatic shunt
Fig. 13: Figure 13. Enhance CT images shows at left hepatic lobe a communication into left hepatic vein and left portal vein. Portosystemic venous intrahepatic shunt
Fig. 14: Figure 14. 58-year-old man with portosystemic shunt undergoing evaluation for cirrhosis. Sonogram shows direct communication between right portal vein and hepatic vein.
**Fig. 15:** Figure 15. Early enhancement of the peripheral portal vein branches during the hepatic arterial phase and before the main portal vein is enhanced in a patient with hepatic cirrhosis and portal thrombosis vein.
**Fig. 16:** Figure 16. Early enhancement of the peripheral portal vein branches during the hepatic arterial phase and before the main portal vein is enhanced in a patient with hepatic cirrhosis and portal thrombosis vein.
**Fig. 17:** Figure 17. Early enhancement of the peripheral portal vein branches during the hepatic arterial phase and before the main portal vein is enhanced in a patient with hepatocellular carcinoma and portal thrombosis vein.
Fig. 18: Figure 18. Early enhancement of the peripheral portal vein branches during the hepatic arterial phase and before the main portal vein is enhanced in a patient with hepatocellular carcinoma and portal thrombosis vein
Fig. 19: Figure 19. Arterioporal shunt nontumorous in a patient with liver biopsy antecedent ten days ago.
**Fig. 20:** Figure 20. Contrast-enhanced arterial phase CT scan shows wedge-shaped subcapsular region of increased attenuation (straight arrow). Note early portal venous filling (curved arrow), representing small idiopathic nontumoral arterioportal shunt confirmed angiographically.
Fig. 21: Figure 21. Axial CT shows an arterioportal shunt macroscopic in a patient with a hemangioma liver into IV segment
Fig. 22: Figure 22. Coronal CT shows an arterioportal shunt macroscopic in a patient with a hemangioma liver into IV segment
Fig. 23: Figure 23. Arteriosystemic shunt. MIP reconstructions
Fig. 24: Figure 24. Arterioportal, arteriosystemic shunt, and subcapsular venous lagos in a patient with hereditary hemorrhagic telangiectasia or Osler-Weber-Rendu
Fig. 25: Figure 25. Drawing illustrates the collateral vessels in portal hypertension. AWV = abdominal wall vein, GEV = gastroesophageal vein, IMV = inferior mesenteric vein, IVC = inferior vena cava, LGV = left gastric vein, LPV = left portal vein, LRV = left renal vein, MV = mesenteric vein, PDV = pancreaticoduodenal vein, PEV = paraesophageal vein, PV = portal vein, RPPV = retroperitoneal-paravertebral vein, SMV = superior mesenteric vein, SRV = splenorenal vein, SV = splenic vein, UV = umbilical vein.

Fig. 26: Figure 26. Esophageal varices. Axial CT image agittal reconstructions CT image
Fig. 27: Figure 27. Prominent esophageal varices. Sagittal reconstructions CT image
Fig. 28: Figure 28. Paraesophageal varices in a patient who had an hepatocellular carcinoma. Axial CT image
**Fig. 29:** Figure 29. Recanalized paraumbilical vein. Axial CT image, sagittal, and coronal MIP reconstructions CT images
Fig. 30: Figure 30. Splenorenal ang gastrorenal shunt. Axial CT scan shows dilated vessels (arrows) around the retrogastric and splenic hila
Fig. 31: Figure 31. Retroperitoneal varices in a patient with portal hypertension. Coronal MIP reconstructions CT image demonstrates a tortuous, dilated retroperitoneal shunt (arrows) that communicates with the inferior vena cava (I) through the left renal vein (R).
Fig. 32: Figure 32. Inferior hemorrhoidal varices. A lobulated mass protruding into the rectal lumen
Conclusion

The technical possibilities of new equipment multidetector CT (64 or more crowns) has allowed a significant step forward in the analysis of hepatic vascularization and certainly are a real alternative to conventional angiographic studies.

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Images for this section:
Fig. 1: Figure 1. Uxía, and Iria are my daughters
References


