CT findings of hepatic vascular disease: what the radiologist needs to know

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Authors: S. Pellegrino¹, D. Giambelluca¹, G. Caruana¹, M. Dimarco¹, M. R. VACCARO NOTTE², D. Picone¹, G. Salvaggio¹, A. Lo Casto¹, R. Lagalla¹; ¹Palermo/IT, ²PALERMO, ITALIA/IT
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Learning objectives

Hepatic vascular diseases are a heterogeneous group of uncommon liver disorders. The goals of this educational exhibit are:

- To identify the usual CT imaging features in the hepatic vascular syndromes.

- To provide brief notes about the clinical and pathophysiologic correlation of the radiologic findings.
Background

Vascular diseases of the liver can result from various conditions involving the hepatic vascular system of the hepatic artery, portal vein or hepatic veins. Diagnostic imaging plays an important role for the correct diagnosis in the majority of such cases. The imaging features of these conditions reflect not only their particular etiology, but also the specific hepatic parenchymal changes depending on the grade, level and duration of the hemodynamic changes associated with the different vascular alteration.

We can distinguish hepatic inflow from hepatic outflow diseases.

The hepatic inflow is constituted of the hepatic artery and portal vein located in the center of each hepatic segment, while the outflow consists of the hepatic veins draining in the inferior vena cava.

The portal vein supplies the 75% of the blood flow to the liver and provides deoxygenated blood that has been drained from the spleen, pancreas and gastrointestinal tract. The proper hepatic artery, a branch of the celiac artery, supplies the 25% of the blood into the liver and provides oxygenated blood in the liver [1].

In the event of a sudden, marked reduction in portal vein blood flow, the arterial blood flow is able to acutely increase, probably through peribiliary vascular plexus, to prevent anoxic tissue necrosis.

The hepatic sinusoid and its adjacent microvasculature are sites of complex anastomosis between the high pressure, low volume hepatic arterial inflow and the low pressure, high volume portal venous input. Resistance to flow from portal venous to hepatic venous vessels through hepatic sinusoids is remarkably low [2].

While the blood supply to liver has a dual origin, all blood leaves the liver through a single venous system: the central veins receive sinusoidal blood and then they unite to form the major hepatic veins. In the sinusoids, the blood is processed by hepatocytes, which can absorb or release nutrients and metabolize toxic chemicals [3].

Deoxygenated blood flow moves out from the lobules through the central veins (located at its center) into the hepatic veins and finally in the vena cava and right atrium.

The study of the functional anatomy of the liver allows the description of a hepatic segmentation based upon the distribution of the portal pedicles and the location of the hepatic veins.

Middle hepatic vein represents the Cantlie line and divides the right from the left liver; right hepatic vein divides right anterior sector and posterior sector. Utilizing the left hepatic
vein, we identify left lateral and left medial sections. The plane of the portal vein (right and left branches) allows us to divide these different portions of liver into lower segments and upper segments. We can represent the hepatic vein system as vertical lines and the portal vein as a horizontal one. We can superimpose on these vessels the eight segments that are described by Couinaud [3].

Third inflow is a normal variant involving an additional venous inflow to the liver, separate from the usual dual blood supply [2]: this system is composed of aberrant veins that directly enter into the liver independently of the portal venous system. Pancreaticoduodenal veins can drain into the posterior part of segment IV and usually deliver a higher amount of insulin in comparison to the portal vein. Therefore, since insulin promotes the synthesis and deposition of fat in the hepatocytes, direct drainage of these veins into the liver can cause focal fatty infiltration [4]. The inferior vein of Sappey drains the anterior abdominal wall vessels. Uncommonly, pseudolesions can consist of focal steatosis. A possible explanation is that aberrant drainage of the inferior vein of Sappey may alter local hepatic metabolism and cause fatty infiltration adjacent to the falciform ligament [4]. On the contrary, gastric and cystic veins contain low concentration of insulin and produce fatty sparing areas, which are typically located in the posterior part of segments II and IV and adjacent to the gallbladder fossa.

Multiphase helical Computed Tomography (CT) allows the evaluation of the liver during both the later hepatic arterial and the portal venous phases after the injection on contrast agent and therefore has become an important modality for the detection and characterization of perfusion abnormalities. The dual blood supply system may cause changes in both the volume and the direction of blood flow when a vascular disease occurs [1].

The increasing use of contrast-enhanced, multiphase helical CT has resulted in more frequent description of the various pathologic conditions involving the hepatic blood vessels. Radiologists with the knowledge of the pathophysiologic processes that are represented on CT images are often in a position to suggest a specific explanation for the observed hepatic abnormality. Correlation of imaging features with the clinical report is often essential for an accurate diagnosis, because some lesion enhancing pattern may mimic mass lesions. Liver biopsy does not usually contribute to a diagnostically useful information and may be contraindicated in some of these conditions.

In this educational exhibit, we describe the most common imaging findings of vascular diseases in inflow vessels (portal vein and hepatic artery) and in outflow vessels (hepatic veins) and provide clinical and pathophysiological correlation of the radiological features.
Findings and procedure details

Hepatic infarction

Hepatic infarction is defined as an area of coagulation necrosis from hepatocyte cell death caused by local ischemia, which in turn results from the obstruction of circulation to the affected area [1]. It is an extremely rare situation because of the liver supply. Hepatic infarction can occur when there is both hepatic arterial and portal vein flow compromise; in fact, in most cases, it results from either insult to the hepatic artery or portal vein thrombosis superimposed on hepatic arterial occlusion [1].

It may occur as a complication of hepatic artery stenosis or thrombosis after liver transplantation, or it may be secondary to hypercoagulability, vasculitis, or infection in sepsis and shock (Fig. 1).

Typically infarction presents on CT as an ill-defined wedge-shaped area of hypoattenuation, which is mostly peripheral without mass-effect on adjacent structures in post-contrast images (Fig. 2, Fig. 3).

Portal vein thrombosis

Decreased portal vein inflow and increased arterial inflow are two conditions that can occur in portal vein thrombosis.

Portal vein thrombosis is characterized by a thrombus developed in the main portal vein, and/or its right or left branches, or by the permanent obliteration that results from a prior thrombosis. It may be seen with many clinical conditions including cirrhosis, abdominal neoplasms, intrabdominal inflammatory processes such as Crohn disease, diverticulitis and appendicitis, hypercoagulable states, and trauma [5]. Recently, portal vein thrombosis was found to be associated with metabolic syndrome, especially with central abdominal obesity.

Thrombus is usually seen as a hypodense filling defect in the lumen of the portal vein, with partial or complete occlusion on contrast-enhanced CT (Fig. 4). Unenhanced scans have been shown to be of minimal benefit in the identification of thrombus, except in the case of early thrombosis, because it appears hyperdense (Fig. 5).

Occlusion of a branch of the portal vein by a thrombus can manifest as transient hepatic attenuation differences in the late arterial phase, showing increased enhancement of the lobe or segment previously supplied by the vein due to hepatic arterial compensatory flow (Fig. 6).

Cavernous transformation of the portal vein is a sequela of chronic portal vein thrombosis and/or occlusion, which leads to the development of numerous periportal collaterals,
which appear as a mass of veins in the porta hepatis on contrast-enhanced CT (Fig. 7, Fig. 8) [6].

**Hereditary Hemorrhagic Telangiectasia (Osler-Weber-Rendu Syndrome)**

It's a rare hereditary autosomal dominant vascular disorder. Hepatic involvement occurs in up to 30% of cases and consists of vascular, parenchymal, and biliary lesions with characteristic telangiectasias and vascular shunts.

On arterial phase, liver shows characteristically mosaic heterogeneous perfusion enhancement. These abnormalities are best seen during arterial phases, almost always disappearing in the hepatic phase as the hepatic parenchyma becomes homogeneous (Fig. 9, Fig. 10). The cause of this mosaic appearance is multiple arteriovenous shunts showing different attenuation and telangiectasias [7].

Telangiectasias are hypervascular rounded nodules with predominant peripheral location (varying from a few millimeters to 1 cm in size) in the arterial and late arterial phases, often becoming isointense in the portal venous and delayed phases (Fig. 11) [7].

Sometimes integrated confluent vascular masses appear as larger vascular pools (25%) and these masses are characterized by early and persistent enhancement during the arterial phase (Fig. 12).

Opacification of the hepatic veins during the arterial phase was considered as an indirect sign of the presence of hepatic-arteriosystemic venous shunt (Fig. 13); arterial dilatation (a common hepatic artery greater than 7 mm in diameter) can be seen and it is a consequence of an increased volume flow in the hepatic artery and veins caused by intrahepatic fistulas (Fig. 9) [8].

Early and prolonged enhancement of the portal vein during the arterial phase was considered an indirect sign of the presence of arteriportal shunt.

**Budd Chiari syndrome**

Budd-Chiari syndrome results from the hepatic venous outflow obstruction at any level, from the small hepatic veins to the junction of the inferior vena cava and the right atrium (Fig. 14, Fig. 15). Thrombosis is the most common cause of hepatic vein obstruction, most commonly results from a hypercoagulable states.

The imaging findings of Budd-Chiari syndrome are variable and depend on the stage of the disease.

In acute Budd-Chiari syndrome, the morphologic of the liver usually is normal, and occlusion of the hepatic veins with ascites are the typical finding (Fig. 16).
exhibits patchy, decreased peripheral enhancement caused by portal and sinusoidal stasis and stronger enhancement of the central portion of the liver parenchyma [9].

In subacute or chronic Budd-Chiari syndrome, the morphologic changes in the liver are the result of the type of venous involvement, and portosystemic and intrahepatic collateral vessels are often found [9]. Contrast-enhanced CT is useful for depicting regions of hypoperfused liver parenchyma and hepatic veins thrombosis (Fig. 17).

Chronic Budd-Chiari syndrome is also characterized by the development of multiple regenerative nodules, which can be viewed as a response to a focal loss of portal perfusion and hyperarterialization in areas with preserved hepatic venous outflow [9].

**Passive hepatic congestion**

Passive congestion occurs with the stasis of blood flow within liver parenchyma as a result of impaired hepatic venous drainage secondary to cardiac disease [1]. Elevated central venous pressure is directly transmitted from the right atrium to the hepatic veins [10]. On contrast-enhanced CT scans of patients with passive hepatic congestion, an inhomogeneous, mottled, reticulated-mosaic pattern of parenchymal may be observed in arterial phase in association with early enhancement of hepatic veins due to contrast reflux from right atrium into inferior vena cava (Fig. 18, Fig. 19). In portal phase liver parenchyma become omogenous [1].

**Hepatic sinusoidal obstruction syndrome**

Hepatic sinusoidal obstruction syndrome (SOS) is a vascular disorder that has been described at first as a complication of oral contraceptive therapy; now it is associated with several other conditions such as pregnancy, granulomatous disease, neoplasm, rheumatoid arthritis, HIV infection, Hodgkin's disease, inflammatory disorders.

It is caused by toxic injury to sinusoidal endothelial cells. Histologically, it is the result of sinusoidal congestion and centrolobular hemorrhagic necrosis due to non-thrombotic occlusion of the central hepatic veins, whereas large hepatic veins remain patent [12].

This disease has long been recognized as a consequence of poisoning with pyrrolizidine alkaloids-containing plants, consumed either as contaminated flour or as traditional or herbal remedies. Currently, the most common cause for the disease is toxicity from various chemotherapeutic agents or regimens, particularly, but not exclusively, when used for myeloablation prior to hematopoietic stem cell transplantation [11].

Today, SOS has been associated with more than 20 drugs including conventional doses of some immunosuppressive and chemotherapeutic agents. [11].

On contrast-enhanced CT, hepatic veins congestion causes a mottled pattern of contrast enhancement in the hepatic arterial and early portal venous phases with
decreased enhancement in the liver periphery. The areas of decreased enhancement are due to decreased portal flow, hepatic congestion and ischemia. On delayed images enhancement of the liver becomes more homogeneous (Fig. 20).

On MRI, the presence of reticular hypointensity on hepatobiliary phase images obtained using gadoxetic acid-enhanced MR imaging is highly specific for the diagnosis of SOS (Fig. 21) [12].
Fig. 1: Axial Arterial CT images of a 47-year-old woman, with septic shock and mesenteric ischemia, show thrombosis of celiac tripod (arrows) in two different levels (a, b).

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Fig. 2: Axial CT images on arterial phase, obtained from different levels in the same patient of Fig 1, depict well-defined wedge-shaped poorly enhancing regions in both hepatic lobes, which are predominantly peripheral and extended to the capsular surface (arrows). Portal venous gas is also associated (arrowheads).

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**Fig. 3:** Axial Portal CT images of the same patient in Fig 1-2 demonstrate that lesions remain hypoattenuating, representing regions of necrotic tissue.

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**Fig. 4:** Axial Portal CT image of a 78 year-old-man, with Chron disease and abdominal pain, shows thrombosis of main portal vein (arrow). Ischemia of upper pole of spleen is also observed (arrowhead). These findings are consistent for acute portal vein thrombosis.
Fig. 5: Axial non-enhanced CT scan of a 68 year-old-man, with nephrotic syndrome and abdominal pain, shows thrombosis in portal confluence (arrows). Thrombus is hyperattenuating, as in case of acute thrombosis.
Fig. 6: CT image on arterial phase of a 34-year-old woman, in hormonal therapy, with partial right branch portal thrombosis (arrowhead), determining homolateral lobar transient hepatic intensity difference (arrows).

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Fig. 7: Axial CT image on portal phase of a 82-year-old woman, with a story of HCV-related cirrhosis, shows partial thrombosis in portal vein (arrow) and multiple collateral vessels that develop in the porta hepatitis (arrowhead). These findings are consistent with portal cavernomatosis.

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**Fig. 8:** Axial CT images on portal phase of a 68 year-old-woman, with chronic HCV cirrhosis and portal cavernomatosis. The upper level image (a) shows collateral vessels in hepatic hilum (arrows); the lower level image (b) shows dilatated epicholedochal and paracholedochal veins around gallbladder (arrowheads).

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**Fig. 9:** Axial CT images, obtained from different levels on arterial phase of a 55-years-old woman with a clinical history of hematemesis and abdominal pain, show hepatic perfusion abnormalities, identified as an inhomogeneous attenuating pattern within the liver parenchyma and multiple telangiectasias, associated with dilatation of the Hepatic Arteries (arrows). Fig.a shows dilatation of Hepatic artery in falciform ligament; Fig.b shows dilatation of Hepatic Proper Artery; Fig.c shows dilatation of Hepatic Common Artery. These findings are suggestive for Rendu-Osler-Weber Syndrome.

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**Fig. 10:** Axial CT images on portal phase, in the same patient of Fig 9. Parenchymal abnormalities are best seen during arterial phases, almost always disappearing in the hepatic phase as the hepatic parenchyma becomes homogeneous.

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Fig. 11: Axial CT image on arterial phase shows multiple Telangiectases (arrows): hypervascular rounded nodules are hyperattenuating in the early and late arterial phases and isoattenuating in the hepatic phase.

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**Fig. 12:** Axial CT image on arterial phase shows Large Confluent Vascular Masses (arrows). They are defined as large areas of multiple telangiectases that coalesce or large shunts that are directly visible.

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**Fig. 13:** Axial CT image on portal phase shows dilatation of Hepatic Veins (arrows).

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**Fig. 14:** Coronal CT image on portal phase of a 53 year-old man, with a story of HCC, shows neoplastic thrombosis in inferior vena cava (arrows) and diffuse disomogenous enhanced areas in the peripheral hepatic parenchyma (arrowheads). HCC is visible in the upper part of the liver and is irregularly surrounded by capsule.

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Fig. 15: Axial (a) and coronal CT image (b) on portal phase, obtained in a 75 year-old woman, with lung cancer and metastasis, show thrombosis of inferior vena cava (arrows) and an area of patchy enhancement (arrowheads) in the subdiaphragmatic part of liver.

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Fig. 16: Axial CT image on portal phase of a 24 year-old woman, with a diagnosis of inflammatory bowel disease and with high D dimer level, shows hepatic veins thrombosis (arrows) and a subcapsular triangular ipodense area (arrowhead). The diagnosis was Budd-Chiari syndrome.

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**Fig. 17:** Axial CT images on arterial (a) and portal phase (b), obtained in a 76 year-old woman with chronic Budd Chiari syndrome. Portal veins are not opacified; liver is bigger, with a mottled pattern of contrast enhancement in early portal venous phase (b) and decreased enhancement of its periphery, due to venous congestion.

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**Fig. 18:** Coronal CT image on early arterial phase of a 83 year-old woman shows cardiomegaly and early enhancement of dilated inferior vena cava and hepatic veins (arrows) due to contrast reflux from right atrium into inferior vena cava.
**Fig. 19:** Axial CT image of passive hepatic congestion due to congestive heart failure in a 75-year-old woman. (a) Axial contrast-enhanced CT image obtained on the early arterial phase shows retrograde enhancement of dilated hepatic veins (arrows). (b) Axial contrast-enhanced CT image obtained on the portal venous phase at the same level shows delayed antegrade enhancement of the hepatic veins because of impaired venous drainage due to elevated central venous pressure, with heterogeneous enhancement of the hepatic parenchyma.

**Fig. 20:** Axial CT images on arterial Phase, in two different levels, of a 36 year-old woman in contraceptives therapy, show reticular enhancement pattern, similar to patchy pattern enhancement in Budd-Chiari syndrome. In this case, SOS is responsible of hepatic congestion.
**Fig. 21:** MRI on hepatospecific phase at different levels of a 47-year-old man, with right colectomy for adenocarcinoma, after eight cycles of chemotherapy with Oxaliplatin (four months in range time), demonstrate diffuse hypointense reticulations with densely coalescent areas (arrows). This is a characteristic feature of Sinusoidal Obstructive Syndrome.
Conclusion

A broad spectrum of variants and pathologic conditions can involve the liver vasculature. Imaging is essential for the evaluation of the normal hepatic vascular anatomy, as well as for the determination of the patency of the vessels and the diagnosis of abnormalities. Familiarity with the pathogenesis and imaging features of these vascular entities can aid the radiologic diagnoses and guide appropriate patient management.
Personal information

Silvia Pellegrino, silviapellegrino82@libero.it
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