Hepatic pseudolesions

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Learning objectives

-To describe imaging characteristics of this relatively common imaging finding.

-To show different kinds of presentation with respect to different causing factors and to interrelationship between different vessels.

-To discuss, how to recognise pseudolesions in different imaging modalities (contrast-enhanced ultrasound, computer tomography, magnetic resonance) and how to avoid confusion of pseudolesion and "real" lesion.
The liver uniquely receives a dual blood supply - approximately 1000-1200 ml/min of blood arrives via the portal vein and approximately 400 ml/min arrives via the hepatic artery. In a non-cirrhotic liver, blood perfusion occurs at pressures of approximately 7 mmHg and 100 mmHg, via the portal vein and hepatic artery, respectively.

There are several communication systems between both vessels, including transsinusoidal, transvasal, and transplexal routes. The most prominent system is the peribiliary plexus (transplexal route) and it is composed of vessels that run around the lobular ducts. This system plays an important role when the portal vein is compromised. Transsinusoidal shunts are governed by an arteriolar inlet sphincter under the influence of different angiogenic factors. These shunts occur in Budd-Chiari syndrome, or may arise for no apparent reason or in response to focal infection or disease that compromise the portal perfusion. Transvasal plexus often occurs in conjunction with peribiliary shunting and via the vasa vasorum of the portal vein. It most commonly occurs in the setting of portal vein occlusion or in cases of invasive hepatocellular carcinoma.

Variable hepatic arteries are relatively common and we find them in approximately 42%. There can exist aberrant and accessory arteries. Aberant artery replaces missing normal artery, accessory arteries coexist with normal hepatic artery.

In the system of *a. hepatica communis* (AHC) can originate variably from a. mesenterica sup. (4%), directly from aorta or branches of a. gastrica sin., a. gastroduodenalis, a. renalis dx. or a. lienalis. AHC can also be duplex, triplex or missing. (12%) - in such case AHC is substituted for one or more variable arteries.

The system of *a. hepatica dextra* (AHD) is variable too, the most common variety is AHD originating from a. mesenteria superior (14 %) or from AHC and in majority of cases, a. cystica descend from AHD. In minority of cases, AHD can be a branch of a. hepatica sinistra (AHS), a. gastroduodenalis, truncus coeliacus, aorta.

*AHS* can be also accessory, eosophageal artery can branch of AHS, but mostly AHS originates from a. hepatica propria or communis, a. gastrica sin., tr. coeliacus or abdominal aorta. Accessory left hepatic arteries occur in approximately 23% and can be multiple.

Variations of hepatic veins manifest as different inflow of segmentary veins, in majority there are drained in v. cava inf. directly, dorsocaudally to porta hepatis. Anatomoses amongst hepatic veins are common, opposite to anastomoses with portal vein system.

*V. portae* (VP) shows just little variability, it may have connections with v. lienalis accessoria, v. phrenica inf., v. pancreaticoduodenalis or v. gastroepiploica dx., rarely VP lies in hepatoduodenal ligament in front of common bile duct and hepatic artery.
Small areas of liver tissue may be supplied by another venous system, the "third inflow", which comprises aberrant veins that enter the liver directly, independently of the portal venous system. Such veins communicate with intrahepatic portal branches to various degrees and lead to focally decreased portal perfusion. However, little overall change in the hepatic arterial perfusion is seen. Because this hemodynamic state is persistent, focal metabolic changes are occasionally observed, typically as sparing in the fatty liver or as accumulations of fat. These communications are not nutritive.
VASCULAR ABNORMALITIES

Due to the interrelationship between different vessels, when individual vessels become compromised, this immediately changes the blood flow in surrounding vessels.

a) portal vein compromise

Decrease in portal flow induce an increase in hepatic arterial flow, whereas a decrease in hepatic arterial flow did not cause an increase in portal flow. In cases of portal vein obstruction and compression, increased hepatic arterial blood flow occurs mainly through the peribiliary plexus. On dynamic computer tomography (CT) or magnetic resonance (MR) of the liver, the decreased portal blood flow leads to areas of parenchymal enhancement during the arterial phase, referred to as transient hepatic attenuation difference (THAD). This area of enhancement, representing increased compensatory arterial flow, is no longer visible during the subsequent portal venous phase. (Figure 1).

Figure 1: Portal vein thrombosis, a,b - arterial phase, c,d,e - portal venous phase of dynamic CT examination. In arterial phase wide branches of hepatic artery are seen, in portal venous phase hypoperfusion of affected parenchyma are visible.
Fig.: Figure 1a : Portal vein thrombosis, a,b - arterial phase, c,d,e - portal venous phase of dynamic CT examination. In arterial phase wide branches of hepatic artery are seen, in portal venous phase hypoperfusion of affected parenchyma are visible. 

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
Fig.: Figure 1b: Portal vein thrombosis, a,b - arterial phase, c,d,e - portal venous phase of dynamic CT examination. In arterial phase wide branches of hepatic artery are seen, in portal venous phase hypoperfusion of affected parenchyma are visible.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
**Fig.**: Figure 1c: Portal vein thrombosis, a,b - arterial phase, c,d,e - portal venous phase of dynamic CT examination. In arterial phase wide branches of hepatic artery are seen, in portal venous phase hypoperfusion of affected parenchyma are visible.

**References**: Radiology dept., Faculty Hospital Brno - Brno/CZ
Fig.: Figure 1 d: Portal vein thrombosis, a,b - arterial phase, c,d,e - portal venous phase of dynamic CT examination. In arterial phase wide branches of hepatic artery are seen, in portal venous phase hypoperfusion of affected parenchyma are visible. 

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
**Fig.**: Figure 1e: Portal vein thrombosis, a,b - arterial phase, c,d,e - portal venous phase of dynamic CT examination. In arterial phase wide branches of hepatic artery are seen, in portal venous phase hypoperfusion of affected parenchyma are visible.

**References:** Radiology dept., Faculty Hospital Brno - Brno/CZ

b) hepatic artery compromise

Hepatic arteries communicate with each other in the central portion, and blockade of large arteries induces new routes of flow. However, acute obstruction of peripheral arterial flow does not induce recognizable changes in portal blood flow.

c) hepatic vein compromise

When the hepatic vein is acutely obstructed, the portal vein becomes a draining rather than a supplying vein. The result is a compensatory increase in hepatic arterial flow as a result of functional portal flow elimination.

PARENCHYMAL PSEUDOLESIONS
Focal fatty liver (FFL)

FFL is a common metabolic complication of a variety of different insults to the liver. It comprises approximately 10% of adult population. In 30-40% is focal, in 20% multiple. We can see it often in third inflow areas. In ultrasound (US), CT and MR images it has characteristics of fatty tissue and there is no deviation of vessels (Figure 2,3,4).

Figure 2: In ultrasound image (a) FFL presents as hyperechoic areas. In CT (b) as hypodense areas in nativ scan (b).

Fig.: Figure 2a: In ultrasound image (a) FFL presents as hyperechoic areas. In CT as hypodense areas in nativ scan (b).

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
Figure 2b: In ultrasound image (a) FFL presents as hyperechoic areas. In CT as hypodense areas in nativ scan (b).

**References:** Radiology dept., Faculty Hospital Brno - Brno/CZ

Figure 3: CT images of FFL. Equilibrium phase, portal venous phase, arterial phase and nativ scan, respectively. In all of them there is hypodense geographic appearance.
Fig.: Figure 3: CT images of FFL. Equilibrium phase, portal venous phase, arterial phase and nativ scan, respectively. In all of them there is hypodense geographic appearance.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ

Figure 4: MR in FFL case shows increased arterial flow in the right liver lobe to the focal fatty tissue (a), in portal venous phase (b, in T1 weighted image with fat saturation) is the same area hypointense.
Fig.: Figure 4 a: MR in FFL case shows increased arterial flow in the right liver lobe to the focal fatty tissue (a), in portal venous phase (b, T1 weighted image with fat saturation) is the same area hypointense.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
**Fig.**: Figure 4b : MR in FFL case shows increased arterial flow in the right liver lobe to the focal fatty tissue (a), in portal venous phase (b, T1 weighted image with fat saturation) is the same area hypointense.

**References:** Radiology dept., Faculty Hospital Brno - Brno/CZ

**Focal fatty sparing (FFS)**

FFS means focal sparing of fatty infiltration and the most frequently occurs around the gallbladder and regions supplied by an aberrant right gastric vein. They have characteristics of liver tissue. (Figure 5).
Figure 5: In MR examination we can clearly depict the areas of focal fatty liver as well as of focal fatty sparing in dual phase MRI. a) in-phase image with no visible liver lesions, b) out-of-phase image with decreased signal of fatty tissue.

Fig.: Figure 5a: In MR examination we can clearly depict the areas of focal fatty liver as well of focal fatty sparing in dual phase MRI. a - in-phase with no visible liver lesions, b - out-of-phase image with decreased signal of fatty tissue.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
Fig.: Figure 5b: In MR examination we can clearly depict the areas of focal fatty liver as well of focal fatty sparing in dual phase MRI. a - in-phase with no visible liver lesions, b - out-of-phase image with decreased signal of fatty tissue.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ

**Inflammatory pseudotumors (IP)**

IP is an unusual and rare tumor-like condition that is an important differential diagnosis in patients presenting with liver masses. Features are non-specific on US and unenhanced CT. In dynamic imaging on CT or MRI early peripheral enhancement is typically seen, reflecting the inflammatory changes. Than it is followed by homogeneous, complete and persistent enhancement. (Figure 6).

Figure 6: Inflammatory pseudotumor surrounding the small fish bone located in the left liver lobe. In arterial phase (a) strong peripheral enhancement is visible. In portal venous (b) and late phase (c), the lesion is enhancing completely, excepting small hyperdensity in the centre, representing the corpus alienum.
Fig.: Figure 6a: Inflammatory pseudotumor surrounding the small fish bone located in left liver lobe. In arterial phase (a) strong peripheral enhancement is visible. In portal venous (b) and late (c) phase, the lesion is enhancing completely, excepting small hyperdensity in the centre, representing the corpus alienum.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
**Fig.**: Figure 6b: Inflammatory pseudotumor surrounding the small fish bone located in left liver lobe. In arterial phase (a) strong peripheral enhancement is visible. In portal venous (b) and late (c) phase, the lesion is enhancing completely, excepting small hyperdensity in the centre, representing the corpus alienum.

**References**: Radiology dept., Faculty Hospital Brno - Brno/CZ
**Fig.**: Figure 6c: Inflammatory pseudotumor surrounding the small fish bone located in left liver lobe. In arterial phase (a) strong peripheral enhancement is visible. In portal venous (b) and late (c) phase, the lesion is enhancing completely, excepting small hyperdensity in the centre, representing the corpus alienum.

**References**: Radiology dept., Faculty Hospital Brno - Brno/CZ

**Parenchymal compression**

Diaphragmatic compression of liver parenchyma due to contraction of diaphragmatic muscle bundles may create hypodense pseudonodular areas, especially in segment VII and VIII, typically in patients deep inspiration. Pseudolesions due to rib compression are most commonly seen in the subcapsular region of the liver. Due to compression, portal perfusion decreases (Figure 7).

Figure 7: On post-contrast CT in arterial phase the round hypodense lesion can be seen in the right liver lobe.
Fig.: Figure 7: On post-contrast CT in arterial phase the round hypodense lesion can be seen in the right liver lobe.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ

VASCULAR PSEUDOLESIONS (VP)

VP may be functional (THAD), or organic (vascular malformations). At all events, they are associated with many different causes, mostly intrahepatic shunts and we can divide them to arteriportal, arteriosystemic and portosystemic, depending on the vascular connection.

Arteriportal shunts are the most common form of intrahepatic shunts, and are commonly associated with HCC (rarely with hyperdynamic haemangiomas) or with iatrogenic causes, such a liver biopsy. Usually, they are seen as early enhancement of portal vein branches during the arterial phases of CT or MR dynamic studies (Figure 8).
Figure 8: Hyperdynamic haemangioma in MR examination. In arterial phase (a,b) hyperintense area is visible near the nodule. In the portal venous phase (b) the area appears hypointense due to wash-out of contrast medium from this area and enhancement of the surrounding liver.

Fig.: Figure 8a: Hyperdynamic haemangioma in MR examination. In arterial phase (a,b) hyperintense area is visible near the nodule. In the portal venous phase (b) the area appears hypointense due to wash-out of contrast medium from this area and enhancement of the surrounding liver.

**References:** Radiology dept., Faculty Hospital Brno - Brno/CZ
Fig.: Figure 8b: Hyperdynamic haemangioma in MR examination. In arterial phase (a,b) hyperintense area is visible near the nodule. In the portal venous phase (b) the area appears hypointense due to wash-out of contrast medium from the area and enhancement of the surrounding liver.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
Fig.: Figure 8c: Hyperdynamic haemangioma in MR examination. In arterial phase (a,b) hyperintense area is visible near the nodule. In the portal venous phase (b) the area appears hypointense due to wash-out of contrast medium from this area and enhancement of the surrounding liver.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ

Arteriovenous malformations (AVMs) are congenital abnormalities. The blood vessels shunt through direct arteriovenous connections, without neoplastic tissue between these anomalous vessels. On unenhanced CT AVMs generally appear as hypoattenuating areas within the liver. In the arterial and early portal venous phases after contrast media administration, these lesions enhance intensely and homogeneously, similar to contrast observed in the surrounding vascular structures. In dynamic MR imaging, the pattern is similar, however MR is useful tool - signal hypointensity on T2 weighted images can distinguish AVMs from haemangiomas (Figure 9, 10, 11).
Figure 9: US examination of AVM. In B-mode US examination, small hyperechoic lesion near the hepatic vein is seen (a,b), in contrast-enhanced US multiple, irregular and tortuous arterial vessels are visible (c,d).

Fig.: Figure 9a: US examination of AVM. In B-mode US examination small hyperechoic lesion near the hepatic vein is seen (a,b), in contrast-enhanced US multiple, irregular and tortuous arterial vessels are visible (c,d).

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
Fig.: Figure 9b: US examination of AVM. In B-mode US examination small hyperechoic lesion near the hepatic vein is seen (a,b), in contrast-enhanced US multiple, irregular and tortuous arterial vessels are visible (c,d).

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
Fig.: Figure 9c: US examination of AVM. In B-mode US examination small hyperechoic lesion near the hepatic vein is seen (a,b), in contrast-enhanced US multiple, irregular and tortuous arterial vessels are visible (c,d).

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
Fig.: Figure 9d: US examination of AVM. In B-mode US examination small hyperechoic lesion near the hepatic vein is seen (a,b), in contrast-enhanced US multiple, irregular and tortuous arterial vessels are visible (c,d).

References: Radiology dept., Faculty Hospital Brno - Brno/CZ

Figure 10: CT in late arterial phase, the same patient as in fig. 9.

Several polygonal areas of different enhancement in the liver are recognizable.
**Fig.**: Figure 10a: CT in late arterial phase, the same patient as in fig. 9. Several polygonal areas of different enhancement in the liver are recognizable.

**References:** Radiology dept., Faculty Hospital Brno - Brno/CZ
Fig.: Figure 10b,c: CT in late arterial phase, the same patient as in fig. 9. Several polygonal areas of different enhancement in the liver are recognizable.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ

Figure 11: MR examination of the liver. The small T2 hyperintense lesion is demarcated (left), in T1 weighted image (right) the lesion is hyperintense with hypointense centre (a). After contrast media administration, in arterial phase (b,c) multiple tortuous vessels, as well as transient hepatic intensity differences are visible. In portal venous phase (d) the intensity of liver parenchyma is homogeneous and thick vascular structures can be seen.
Fig.: Figure 11a: MR examination of the liver. The small T2 (left) hyperintense lesion is demarcated, in T1 weighted image (right) the lesion is hyperintense with hypointense centre (a). After contrast media administration, in arterial phase (b,c) multiple tortuous vessels, as well as transient hepatic intensity differences are visible. In portal venous phase (d) the intensity of liver parenchyma is homogeneous and thick vascular structures are seen.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ

Fig.: Figure 11b: MR examination of the liver. The small T2 (left) hyperintense lesion is demarcated, in T1 weighted image (right) the lesion is hyperintense with hypointense centre (a). After contrast media administration, in arterial phase (b,c) multiple tortuous vessels, as well as transient hepatic intensity differences are visible. In portal venous phase (d) the intensity of liver parenchyma is homogeneous and thick vascular structures are seen.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
Fig.: Figure 11c: MR examination of the liver. The small T2 (left) hyperintense lesion is demarcated, in T1 weighted image (right) the lesion is hyperintense with hypointense centre (a). After contrast media administration, in arterial phase (b,c) multiple tortuous vessels, as well as transient hepatic intensity differences are visible. In portal venous phase (d) the intensity of liver parenchyma is homogeneous and thick vascular structures are seen.

References: Radiology dept., Faculty Hospital Brno - Brno/CZ
**Fig.**: Figure 11d: MR examination of the liver. The small T2 (left) hyperintense lesion is demarcated, in T1 weighted image (right) the lesion is hyperintense with hypointense centre (a). After contrast media administration, in arterial phase (b,c) multiple tortuous vessels, as well as transient hepatic intensity differences are visible. In portal venous phase (d) the intensity of liver parenchyma is homogeneous and thick vascular structures are seen.

**References:** Radiology dept., Faculty Hospital Brno - Brno/CZ
Conclusion

Recognition of pseudolesions of the liver at different imaging modalities is important because of their close resemblance to primary liver cancers or metastases. Knowledge of the haemodynamics in both hepatic parenchyma and liver tumor is very important for proper characterization of liver tumors and to avoid erroneous identification of pseudolesions. Radiologists need to understand the underlying mechanism of these pseudolesions to better recognize the wide range of their appearances at different imaging methods.

In summary, radiologists should be aware of these pseudolesions of the liver to obviate unnecessary examination or biopsy. Understanding the underlying mechanisms of pseudolesions may help radiologists recognize the wide variety of their appearances.
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References


5. Yoshimitsu K., Honda H., Kuroiwa T. et al.:Unusual hemodynamics and pseudolesions of the non cirhotic liver at CT. Radiographics;2001,21,81-96
