Hepatic Perfusion Abnormalities

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Learning objectives

The purpose of this educational exhibit is to:

1. Describe and explain the pathophysiology of hepatic perfusion abnormalities
2. Demonstrate their imaging appearances at CT and MRI
3. Demonstrate multiple conditions in which these abnormalities may be seen
4. Highlight the importance of hepatic perfusion abnormalities in everyday reporting
Background

THAD (Transient hepatic attenuation differences) and THID (Transient hepatic intensity differences) refer to the imaging appearances of hepatic perfusion abnormalities in CT and MRI respectively. These abnormalities typically appear as hypervascular/hyperattenuating regions in the arterial phase, which revert to normal in the venous and delayed phases. In comparison, relatively few perfusion abnormalities are only seen as hypoattenuating lesions in the venous phase.

Regional variation in the proportions of arterial and venous flow in hepatic parenchyma results in differential enhancement of liver tissue on multiphasic contrast-enhanced cross sectional imaging. This was originally described in 1982 by Itai et al during the development of dynamic CT techniques for the imaging of liver lesions.

Hemodynamics in dynamic liver imaging:

Hepatic arterial phase Fig. 2 on page 11: During the early and late arterial phase, contrast reaching the liver via the hepatic artery is diluted approximately 3:1 within the sinusoids by unopacified portal venous blood, resulting in only minimal hepatic enhancement.

Hepatic venous phase Fig. 3 on page 12: Approximately 10 seconds after the late arterial phase, the iodinated contrast bolus completes transit through the splanchnic organs and opacifies the portal inflow to the liver. Given the duration of the contrast injection in most current protocols, the hepatic arterial inflow also contains contrast during the portal venous phase, resulting in maximal enhancement of liver parenchyma.

Equilibrium phase: Equilibration of contrast between artery, vein and tissue occurs 30 seconds after the hepatic venous phase.

Haemodynamics in perfusion abnormality:

Any pathology which decreases portal inflow, or alters arterial inflow and venous outflow, will result in perfusion abnormality. As there is a large difference between the mean hepatic arterial inflow pressure and mean portal pressure, any communication between
hepatic arterial and portal venous branches at the level of the vessel trunk (or more peripherally) will direct arterial blood into portal vein territory.

Whilst total blood flow to the region remains constant, the arterially derived fraction is larger than in adjacent regions. Consequently, during the arterial phase the region appears markedly enhanced relative to the surrounding parenchyma. During the portal phase the contrast inflow is equivalent.

Aberrant or "non-portal" venous drainage into the liver will also produce perfusion abnormality if there is temporal separation between aberrant contrast inflow and the main portal vein. Non-portal inflow can occur through collateral pathways traversing the liver along embryologic mesenteries in superior or inferior vena caval obstruction, the parabiliary venous system and through pericholecystic veins.

Table 1: Major mechanisms in perfusion abnormality

1. Inflow:
   - Decreased Portal Venous Flow
   - Arterioportal Shunt
   - Increased Arterial Flow
   - Decreased Arterial Flow

2. Outflow:
   - Decreased Hepatic Venous Flow

3. OTHER:
   - Variant Anatomy
   - Miscellaneous including non-portal flow or third inflow

Perfusion abnormality in decreased portal venous flow:

Table 2: Causes of decreased portal venous flow

1) Portal vein obstruction

a) Thrombosis
   i) Multiple etiologies
   ii) Consider septic thrombosis (pylephlebitis) Fig. 7 on page 16 Fig. 8 on page 17 Fig. 9 on page 18

b) Invasion
i) Hepatocellular carcinoma (HCC)

ii) Cholangiocarcinoma Fig. 17 on page 26 Fig. 18 on page 27 Fig. 19 on page 28

c) Compression
   i) Tumor Fig. 10 on page 19 Fig. 11 on page 20 Fig. 12 on page 21
   ii) Infection
   iii) Aneurysm

d) Surgical ligation

2) Biliary obstruction

- Large THID/THAD due to biliary obstruction is uncommon. Fig. 13 on page 22 Fig. 14 on page 23 Fig. 15 on page 24

3) Hepatic parenchymal compression

   a. Ribs
   b. Haematoma
   c. Abscess/collection Fig. 16 on page 25
   d. Tumour

Decreased portal venous blood flow causes perfusion abnormalities by either increased arterial blood flow or by reduced dilution of arterial contrast by unopacified portal venous blood.

At the microscopic level Fig. 4 on page 13, diminished portal inflow can lead to lead to arterio-portal (AP) shunting via the peribiliary plexus which surrounds the bile ductules (transplexal shunting), the vasa vasorum of the portal venules (transvasal shunting), and at the level of the sinusoid itself (trans-sinusoidal shunting). Of these, transplexal shunting is thought to be the dominant route in the setting of portal vein obstruction, occlusion, or increased sinusoidal pressure. A fourth type of shunting (transtumoral shunting) is seen in parenchymal neoplasms.

The cause of perfusion abnormalities in biliary obstruction is thought to be secondary to obstruction of peribiliary plexus.
Perfusion abnormality in AP shunting:

In addition to microscopic shunting in reduced portal venous flow, AP shunting can occur due to arteriovenous (AV) fistulas (traumatic or iatrogenic) and malformations. Fig. 22 on page 31 Fig. 23 on page 32

Table 3: Causes of AP shunting

1. Cirrhosis Fig. 20 on page 29 Fig. 21 on page 30
2. Iatrogenic
   a. Biopsy-related AV fistula
3. Tumor
   a. Most commonly HCC
4. Trauma

In the cirrhotic liver, fibrotic structural distortion of the microvascular anatomy results in occlusion of hepatic venules and retrograde filling of small portal branches. These AP shunts are theorized to occur via the transsinusoidal or transplexal route. Peripheral AP shunts in cirrhosis are a common cause of false-positive diagnosis of hepatocellular carcinoma (HCC) in contrast-enhanced CT or MRI scans of the liver. The characteristic wedge-shaped appearance of a hypervascular lesion on the arterial phase that becomes isodense/isointense during the portal venous and delayed phases should permit accurate distinction from HCC. These shunts can also occur in non-cirrhotic liver. Fig. 24 on page 33 Fig. 25 on page 34

In tumours, in addition to the transplexal route, transtumoral shunts also may play a role in perfusional abnormalities.

Perfusional abnormality in increased arterial flow:

Hypervascular tumours like HCC may produce perfusion abnormalities by "siphoning" or increasing arterial flow in the absence of a decrease in portal flow, or by stealing arterial flow from adjacent liver parenchyma. (It can also occur by stealing arterial flow from adjacent liver parenchyma.)
With this "siphon effect", the adjacent parenchyma may appear hypervascular in arterial phase, whilst with steal effect the adjacent parenchyma may have reduced attenuation. AP shunting and/or siphon effect are commonly seen with small flash-filling hemangiomas.

Local parenchymal inflammation due to cholangitis or abscess may produce regional arterial hyperaemia, in addition to decreased portal flow secondary to elevated sinusoidal pressure.

Table 4: Causes of increased arterial flow:

1. **Tumor**
   a. Essentially all liver tumors are fed by the hepatic artery Fig. 30 on page 39 Fig. 31 on page 40
   b. Small haemangiomas Fig. 26 on page 35 Fig. 27 on page 36

2. **Inflammation**
   a. Cholecystitis Fig. 28 on page 37 Fig. 29 on page 38
   b. Abscess

**Perfusion abnormality in decreased hepatic arterial flow:**

Hepatic perfusion abnormalities can result from collateral vascular supply (eg. internal mammary artery) or from transplexal shunting.

Table 5: causes of decreased arterial flow:

1. Iatrogenic
   a. Surgical ligation
   b. Thrombosis
2. Atherosclerosis
3. Embolic phenomena
4. Vasculitis Fig. 32 on page 41 Fig. 33 on page 42 Fig. 34 on page 43 Fig. 35 on page 44

**Perfusion abnormality in decreased hepatic venous flow:**

Table 6: Causes of decreased hepatic venous flow:

- Hepatic vein thrombosis Fig. 36 on page 45 Fig. 37 on page 46
Hepatic vein branch thrombosis results in AP shunting and retrograde portal venous flow. In Budd-Chiari syndrome, venous drainage from the caudate and precaval segments is maintained. This produces a characteristic diffuse perfusion abnormality distinct from the peripheral or lobar types.

More commonly, retrograde transmission of elevated right heart pressure to the sinusoids produces the characteristic reticulated and mottled appearance of passive congestion.

**Perfusion abnormalities secondary to third inflow:**

The liver is a unique organ in terms of its dual blood supply. Small areas of liver tissue, however, are known to be supplied by another venous system. This system may be composed of aberrant veins or parts of normal veins that directly enter the liver independently of the portal venous system. Such veins communicate with intrahepatic portal branches to various degrees, focally reducing portal perfusion but causing little change in the hepatic arterial perfusion. This results in the appearance of "pseudolesions". Cholecystic veins, parabiliary venous system and paraumbilical system constitute the third inflow system

**a. Cholecystic Vein through the Liver Bed:**

These include small branches that directly enter the liver through the liver bed (segments IV and V) or those that run through the Calot triangle and join the parabiliary veins at the porta hepatis. They dilute the portal perfusion causing perfusion abnormalities. Fig. 5 on page 14

**b. Parabiliary Venous System:**

This venous network is within the hepatoduodenal ligament and collects venous blood from the head of the pancreas, distal part of the stomach, and the bile duct system. These veins usually join the portal venous system but occasionally enter the liver directly around the porta hepatis. The cholecystic vein (via the Calot triangle) and the pancreaticoduodenal vein (conveying the blood from the region of the head of the pancreas) join the parabiliary system to form the superior and infero-lateral portion of the network respectively. The right gastric or pyloric vein draining the distal part of the stomach forms the medial portion of the network. The aberrant drainage of these vessels
into the liver causes perfusion abnormality, typically at the dorsal aspect of segment IV. The abnormality is dependent on the direction and amount of blood flow through these anastomoses. In addition, the presence of hypervascular inflammation, a tumour, or surgical intervention in this region may change the flow through these anastomoses. Fig. 5 on page 14

c. Epigastric-Paraumbilical Venous System: Fig. 6 on page 15

This system consists of small veins around the falciform ligament that drain the venous blood from the anterior part of the abdominal wall directly into the liver. This flow dilutes the portal perfusion at these sites, causing perfusion abnormalities.

The system consists of superior and inferior vein of Sappey and the vein of Burow. The superior vein of Sappey drains the upper portion of the falciform ligament and medial part of the diaphragm. It communicates with branches of the superior epigastric or internal mammary veins. The inferior vein of Sappey drains the lower portion of the falciform ligament. It communicates with branches of inferior epigastric veins around the navel. Both Sappey veins enter peripheral portal branches of the left hepatic lobe.

The vein of Burow also communicates with branches of inferior epigastric veins around the navel. However, it does not enter the liver directly but terminates in the middle portion of the collapsed umbilical vein. There are small communicating intercalary veins between it and the inferior vein of Sappey.

This venous system is an important route for venous return to the heart in the setting of superior or inferior vena cava obstruction. When the vena cavae are obstructed, major collateral routes are recruited to bypass the obstruction. This can result in perfusion abnormality as undiluted venous blood from the arm and leg traverses the liver parenchyma.

Table:7 Third inflow/ Other causes of perfusion abnormalities

**Variant anatomy**
- Aberrant arterial inflow
- Collateral vessels
- Vena caval obstruction

**Accessory veins**
- Capsular veins
- Accessory cystic vein
- Aberrant right gastric vein Fig. 38 on page 47
Images for this section:

Fig. 1: Non-contrast liver image

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**Fig. 2: Arterial Phase**

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Fig. 3: Venous Phase

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Fig. 4: Hepatic microcirculation

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Fig. 5: Non-portal and parabiliary system

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**Fig. 6:** Paraumbilical venous system

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Fig. 7: Wedge shaped abnormality in arterial phase

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Fig. 8: Isoattenuation in venous phase

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Fig. 9: Thrombus in the portal vein.

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**Fig. 10:** Arterial phase showing perfusion abnormality in metastasis

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Fig. 11: venous phase showing isointensity. Metastatic nodule is now clearly evident

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Fig. 12: Clear demarcation of nodule in hepatic-biliary phase

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**Fig. 13:** Perfusion abnormality in cholangitis- Artrial phase

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Fig. 14: Perfusion abnormality in cholangitis- Venous phase

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Fig. 15: Perfusion abnormality in cholangitis- Delayed phase

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**Fig. 16**: Perfusion abnormality in perihepatic collection

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Fig. 17: Compression and infiltration of portal vein by cholangiocarcinoma resulting in perfusion abnormality-Arterial phase

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**Fig. 18:** Compression and infiltration of portal vein by cholangiocarcinoma resulting in perfusion abnormality-venous phase

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Fig. 19: Compression and infiltration of portal vein by cholangiocarcinoma resulting in perfusion abnormality-Thrombus in the portal vein

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Fig. 20: AP Shunt perfusion abnormalities in cirrhosis-Arterial phase

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**Fig. 21:** AP Shunt perfusion abnormalities in cirrhosis-Venous phase

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Fig. 22: Perfusion abnormality in Arterio-portal fistula- Arterial phase

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Fig. 23: Perfusion abnormality in Arterio-portal fistula- venous phase

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Fig. 24: AP Shunt perfusion abnormalities in non-cirrhosis-Arterial phase

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**Fig. 25:** AP Shunt perfusion abnormalities in non-cirrhosis-Venous phase

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**Fig. 26:** Perfusion abnormality in flash filling haemangioma-Arterial phase

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Fig. 27: Perfusion abnormality in flash filling haemangioma - venous phase

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**Fig. 28:** Perfusion abnormality in emphysematous cholecystitis- Arterial phase

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Fig. 29: Perfusion abnormality in emphysematous cholecystitis - venous phase

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**Fig. 30:** Perfusion abnormality in HCC due siphoning-Arterial phase

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**Fig. 31:** Perfusion abnormality in HCC due siphoning-Venous phase

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**Fig. 32:** Perfusion abnormalities in vasculitis secondary to collaterals-arterial phase

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**Fig. 33:** Perfusion abnormalities in vasculitis secondary to collaterals-venous phase

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**Fig. 34:** Perfusion abnormalities in vasculitis secondary to collaterals-3D

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Fig. 35: Perfusion abnormalities in vasculitis secondary to collaterals-3D

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Fig. 36: Hepatic vein thrombosis- Arterial phase

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Fig. 37: Venous phase

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**Fig. 38:** Perfusion abnormality in aberrant gastric vein

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Fig. 39: Perfusion abnormality in CCF - Arterial phase

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**Fig. 40:** Perfusion abnormality in CCF-Venous phase

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Findings and procedure details

The hepatic perfusion abnormalities are seen in MR and CT dynamic imaging of the liver. Liver images are acquired in arterial/late arterial phase, venous phase and equilibrium phase.

These abnormalities typically appear as hypervascular/hyperattenuating regions in the arterial phase, which revert to normal in the venous and delayed phases. A few perfusion abnormalities are only seen as hypoattenuating lesions in the venous phase.

Please see the background section for images.
Conclusion

Hepatic perfusion abnormalities are a reasonably common incidental finding that can mimic other pathologies. Whilst the pathophysiology is complex, recognition of the appearances described will avoid false positive diagnosis of hypervascular tumours. Overestimation of the size of liver tumours may also be avoided.
Personal information

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References