TRANSIENT HEPATIC PARENCHYMAL ENHANCEMENTS

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**Learning objectives**

Show transient hepatic parenchymal enhancements; review the onset mechanisms and their association with different pathological processes.

**Background**

The liver has a dual blood supply: through the hepatic artery, which accounts for 25% of its inflow and through the portal vein, accounting for the remaining 75%. These have pressures of approximately 100 and 7 mm Hg respectively. The outflow of blood from the liver occurs via the hepatic or suprahepatic veins that drain into the inferior vena cava with an average pressure of about 0 mm Hg. There is also a variable perfusion called the "third flow" or anomalous venous inflow. This is caused by embryological remnants from the "mesenteries" and can reduce or even replace the portal flow, producing artefacts in the hepatic hilum and the falciform ligament area in the images acquired with IV contrast in the portal venous phase.

These features are responsible for transient hepatic enhancements consisting of temporary differences in the capture of intravascular contrast media in hepatic parenchyma areas. They are seen as areas of high attenuation/intensity in the hepatic parenchyma in the arterial phase, which is attenuated or sometimes persists in the portal phase and disappears in later stages. In British and American literature this is known as THAD (transient hepatic attenuation difference) in CT and THID (transient hepatic intensity difference) in MRI.

Traditionally they have been reported with conventional CT, but subsequently their detection increased due to techniques which allow better separation between the arterial and portal phase. These include:

- **CT angiography**, which involves carrying out the CT by injecting the contrast through a catheter located in the hepatic artery or celiac trunk.

- **CT arterial portography**, the catheter is located in the superior mesenteric artery or splenic artery. *(Fig 1)*

- **Multislice CT or MRI Multiphase** with intravenous contrast. The study was carried out in three phases:
• Arterial stage: at 25-35 seconds. Blood with contrast reaches the hepatic artery and non-contrast blood through the portal vein, so that the enhancement of the hepatic parenchyma is slight. In the late arterial phase the main portal has minimal enhancement. This is the ideal phase for detecting hypervascular tumours.

• Portal stage: at 60 - 80 seconds. Blood with contrast arrives to the portal vein and the contrast persist in the arterial system. At this stage the hepatic parenchyma shows maximum enhancement.

• Venous parenchymal stage: Conducted at over 90 secs. It tends to even out the contrast in all the vessels and later with the hepatic parenchyma.

CT arteriography and CT portography allows and a highly selective portal and arterial enhancement but may be limited by anatomic variations (arterial, venous or intrahepatic), preventing a homogeneous uptake of contrast. On the contrary multi-phase CT and MRI are not affected by anatomical variations but have less selective arterial and especially portal enhancements.

**TRANSIENT ENHANCEMENT MECHANISMS**

There is usually a trade-off in the dual hepatic blood supply. The arterial blood flow increases when portal flow decreases, however, this is not always the case vice-versa, when arterial flow decreases portal flow does not increase.

This is because both systems are not independent as there are many communications between them through various routes including [8, 9]:

- **Transsinusoidal**: The hepatic artery contributes to the flow to the sinusoids through small arterioles, which are controlled by angiogenic factors (VEGF). It then passes retrogradely into portal branches due of the high resistance found in the hepatic venules.

- **Peribiliar (through biliary plexus)**: Around perihilar bile ducts is a vascular plexus that communicates through small branches with the hepatic artery and subsequently draining into the hepatic sinusoids and portal vein.

- **Transtumoral**: Usually occurs in hypervascular tumours especially in the hepatomas, where the perinodular arteriolar plexus is responsible for the hyperdense halo in the arterial phase. If the intratumoral shunt is significant the retrograde flow in the portal vein can be easily recognized.
Transvacular: Produced by the vasa vasorum of a branch of the hepatic artery which passes through the wall of a portal branch allowing the passage of blood to the portal system. It is clearly demonstrated in cases of arterialized tumour thrombus in the portal vein.

Images for this section:

Fig. 1: CT images in arterial phase (a) and three images (b, C y d) of CT arterial portography in a patient with hepatoma.
CAUSES OF TRANSIENT ENHANCEMENT

1.- ARTERIOPORTAL SHUNT:

This is produced by organic or functional communication between the hepatic artery and portal vein resulting in increased arterial flow in a localized region of the hepatic parenchyma which has suffered a decrease in portal flow. These communications are opened by the mechanisms described in the previous section. These are especially important when the portal vein is compressed or obstructed. Another mechanism is through macroscopic fistulas caused by iatrogenic causes.

In CT and MRI they are displayed as [10]:

- Early filling of peripheral portal branches without enhancement of the main portal vein during the arterial phase.

- Early filling of peripheral portal branches with enhancement of the main portal without filling the superior mesenteric or splenic vein.

- THAD: A wedge-shaped peripheral parenchymal area with well-defined edges which is enhanced temporarily during the arterial phase. This is due to the passage of contrast from a high pressure system such as the hepatic artery to a low pressure one such as portal branches, which makes the enhancement in this area of the parenchymal greater than the normal adjacent parenchyma, where the contrast is diluted by mixing with non-contrast portal flow. Vessels with normal characteristics can be seen crossing through it.

Causes of arterioportal shunt:

• Tumours: In hypervascular tumours it has been reported that the tumour produces a primary increase in arterial flow resulting in a hyperperfusion of the adjacent parenchymal without a demonstrable portal hypoperfusion, this is called "the siphoning effect". Small tumours may act directly on the main branches of the right or left hepatic artery causing hyperperfusion of the lobe containing the tumour. It is much rarer that the contralateral segment in the same lobe containing the tumour transiently presents less enhancement, what is known as "steal phenomenon" [3, 4, 5].
• **Hepatocellular carcinoma (Fig 2, 3 and 4):** The presence of THAD associated to small hepatomas is relatively uncommon, unlike the oversized hepatocellular carcinomas where transient perfusion abnormalities are common [11]. Hepatomas can affect the portal flow and less frequently venous flow, consequently transvascular, transsinusoidal and transtumoral shunts are opened. When the portal vein is affected the peribiliar route may be involved.

• **Hemangioma (Fig 5, 6 and 7):** A high percentage (25%) of hemangiomas are accompanied by arteriportal shunt. Hemangiomas are usually small (<3 cm) and with rapid and intense enhancement [11].

• **Cholagiocarcinoma:** The presence of THAD is an important indirect sign of vascular invasion [8].

• **Metastasis:** Cause SAP due to obstruction or compression of the portal vein directly [8]. (Fig 8)

• **Cirrhosis:** The incidence of shunts in cirrhosis in the absence of tumours lesions is very high (>13%). It is believed that they appear due to the obstruction of small suprahepatic branches which retrogradely cause, through the sinusoids, filling of the small portal branches, which connect with arterial branches. In later stages the porta becomes a drainage system rather than a system of blood inflow [1, 8, 10]. As a result there is a secondary increase of arterial flow which produces a functional SAP. (Fig 9)

• **Trauma, biopsy, radiofrequency and localized treatments:** Often after interventional radiology procedures. They can cause an organic or functional communication in the event that procedure damage the portal vein and causes it thrombosis [1, 8]. These usually spontaneously close very quickly. (Fig 10)

• **Malformations, aneurysms.**

• **Idiopathic.** (Fig 11)

### 2.-**PORTAL THROMBOSIS:**

The perfusion disorders are caused by obstruction or decreased flow of the main portal vein or in the lobar or segmental regions, either by thrombosis, compression, ligature or iatrogenic embolization or tumour invasion.

Usually seen on CT as an area of high attenuation of segmental morphology during the arterial phase which is attenuated in the portal phase and disappears in the equilibrium
phase. When associated with a focal hepatic lesion, depending on its location, the area of perfusion alteration may be wedge-shaped or fan-shaped. If the lesion is within the arterial phenomenon and induces SAP or portal thrombosis, perfusion alteration will have a wedge-shape. However if it is at the apex of the arterial phenomenon and causes portal compression or infiltration, the THAD will be fan-shaped [5]. This is frequently associated with malignancy.

Causes of portal vein thrombosis:

- Liver Tumors. (Fig 12 and 13)
- Pancreatic or biliary tumors. (Fig 14, 15 and 16)
- Infection. (Fig 17, 18 and 19)
- Ligature or embolization. (Fig 20)
- Venous compression.
- Hypercoagulable states.
- Idiopathic.

3.-HEPATIC VEIN OCCLUSION:

It results from the obstruction of the hepatic veins. Their occlusion produces an increase in sinusoidal pressure with gradient inversion between the sinusoids and portal vein, which makes the portal vein become a draining vein producing a functional SAP [8].

In the arterial phase a transient wedged-shaped alteration of the hepatic enhancement can be seen similar to that described in the portal vein thrombosis. The only difference is that in the case of portal vein thrombosis the apex of the perfusion abnormality points to the hepatic hilum while in the hepatic venous occlusion the apex points to the inferior vena cava [1, 8]. A reticular or mosaic pattern that tends to disappear in the equilibrium phase usually appears in the portal phase. In Budd-Chiari syndrome these alterations normally respect the caudate lobe because it has its own draining veins [1].

Causes of hepatic venous occlusion:

- Right heart failure. (Fig 21 and 22)
• Soft thrombus or tumour thrombus. (Fig 23)

• Tumour or hematoma compression.

• Pericardial diseases.

• Budd-Chiari syndrome. (Fig 24)

• Thrombosis of the inferior vena cava.

**4.-COLLATERAL FLOW (FLOW THIRD):**

Usually produced by the paraumbilical venous system and by the parabiliary venous system. The first consists of persistent paraumbilical veins within and adjacent to the falciform ligament, which most likely produce portosystemic shunts in cases of portal hypertension. This system connects with the internal thoracic vein in the epigastrium and with the inferior epigastric vein, forming the inferior vein of Sappey that collects the drainage of the anterior abdominal wall. The most common manifestation is a perfusion defect in the falciform ligament (segment IV b) in the portal phase or in CT portography [9]. (Fig 25 and 26)

The venous drainage of the diaphragm and chest wall occurs through the superior vein of Sappey, which manifests itself conspicuously in the case of obstruction of the superior vena cava [9]. When the superior vena cava is obstructed multiple collaterals vessels dependent of the right-side of the heart are developed. There are four main routes: Azygos-hemiazygos, vertebral venous plexus, lateral thoracic vein and thoracoabdominal superficial vein and internal mammary. The first two communicate with the inferior vena cava and the last two with superficial epigastric vein and paraumbilical veins, which in turn communicate with the portal system. [17] These routes cause abnormalities in hepatic enhancement and are displayed as areas of high attenuation in arterial phase located in: (Fig 27, 28, 29 and 30)

1.-Anterior part of segment IV.

2.-Subdiaphragmatic portion of the liver.

3.-Posterior portion of the right hepatic lobe.

4.-Lateral segment of the left hepatic lobe.

The parabiliary system collects the drainage of the gastric antrum through the right gastric vein and part of the pancreaticoduodenal arcade. These pass through the hepatoduodenal ligament towards the hepatic hilum. Also part of the fundus and the gallbladder body drain
through cystic veins communicating directly with portal branches. This system, like the paraumbilical, causes perfusion defects in portal phase and CT portography in the dorsal part of segment IV and adjacent to the gallbladder [9]. (Fig 31)

Anomalous venous drainage has been established as a cause of focal deposits of fat or areas of preserved parenchyma in the previously mentioned localizations which are displayed in CT in portal phase. This is due to hormonal or nutritional changes influencing hepatic metabolism, secondary to alterations in hepatic flow [9].

5.-OTHER CAUSES OF HEPATIC PERFUSION ALTERATION:

- **Infection:** Local inflammation can cause hyperemia of the hepatic artery and regional portal or venous flow obstruction in cases of cholecystitis, cholangitis and abscesses. The inflamed parenchymal shows a wedge-shaped segmental enhancement in arterial phase or an area of lesser enhancement in CT portography. In the case of cholecystitis it may be secondary to an inflamed hepatic artery that produces hyperemia and to a dilated aberrant cystic vein which causes increased blood flow [1, 8]. In the case of an abscess it may be arterial hyperemia or due to the existence of multiple inflammatory cells that produce stenosis of portal venules, suprahepatic veins or both, resulting in a decrease in portal and/or venous flow thereby increasing arterial flow. (Fig 32)

- **Extrinsic compression:** Compression of the hepatic parenchyma can produce temporary or permanent portal flow compression which appears as a perfusion defect in portal phase. If compression is more permanent minor SAP can be produced due to decreased portal perfusion. This may be due to compression of the ribs, peritoneal implants or hepatic subcapsular collections. The perfusion abnormalities are reversible when compression ceases [1, 8, 9].

- **Hemodynamic alterations:** The Rendu-Osler-Weber syndrome is characterized by multiple telangiectasias, dilated vessels and arteriovenous communications. Hepatic shunts are very frequent and may occur between branches of the hepatic artery and suprahepatic vessels and/or portal vessels. CT shows arterial and venous dilatation, simultaneous arterial and venous enhancement... In the case of SAP heterogeneous enhancement of the parenchyma is observed with multiple wedge-shaped hyperdense areas in arterial phase [1]. (Fig 33)

- **Focal eosinophilic necrosis:** This is a focal hepatic lesion caused by tissue damage due to eosinophils. This is almost always accompanied by peripheral eosinophilia. It is particularly problematic in patients with an underlying neoplastic disease. It is usually displayed during the portal phase as a focal lesion with a blurred edge, a non-spherical shape and subtle hypoattenuation without ring enhancement [19].
• Idiopathic. (Fig 34)

OTHER LESIONS THAT MAY MIMIC PERFUSION ABNORMALITIES:

• Acute alcoholic hepatitis. (Fig 35)

• Steatosis. (Fig 36)

• Hepatic infarction. (Fig 37)

Images for this section:
**Fig. 2:** Hematoma in segment VI of the right hepatic lobe. In this MRI dynamic study there are common findings as capsular retraction, arterial enhancement, wash-out in portal phase and pseudocapsule.

**Fig. 3:** CT in arterial and portal phase. Big hepatoma in the RHL with early filling of portal branches (right portal vein is filling before main porta).
**Fig. 4:** Angiography of the same patient in Fig 3. Shows intravenous contrast in hepatic artery with early enhancement of main porta in arterial phase.
**Fig. 5:** (a, b, c, d) This dynamic MRI shows a hemangioma with transient perfusion disorder of triangular morphology in arterial phase which is normal in portal phase. In portal phase the nodule shows the typical enhancement of hemangiomas. (e) T2 image shows a typical hemangioma heavily hyperintense in T2.
**Fig. 6:** Dynamic CT of small hemangioma in segment II with perfusion disorder in arterial phase. It's thought that shunts are opened by transinusoidal mechanism.
Fig. 7: Small hemangioma in segment VI with associated fistula
Fig. 8: MRI in a patient with pancreatic neuroendocrine tumor with hypervascular metastasis in segment VIII of the right hepatic lobe.
**Fig. 9:** Patient with hepatitis C and ampullary tumor. MRI shows multiples THID (a: arterial phase; b: portal phase) without associated lesions and without signal alteration in T2. (9c). Functional fistulas are the most probably etiology.
Fig. 10: CT in a patient with a small hepatocarcinoma treated with chemoembolization, TACE, (10a and 10b) who has THAD, MRI in other patient with hepatocarcinoma treated with radiofrequency (10c)
**Fig. 11:** (11a) CT axial image and (11b) MRI study with reconstructions. We can see an A-V fistula with early filling of hepatic vein through a communication with hepatic artery. There is a right branch of hepatic artery which is slightly dilated and early filling of the right hepatic vein in arterial phase with a THID associated.
**Fig. 12:** Patient with colon cancer and hepatic metastasis who presents multiples areas of high attenuation and triangular morphology associated. This areas desappear in portal phase and are due to segmental thrombosis of the portal vein; Thrombosis is seen in portal phase.
Fig. 13: (a) CT in a patient with pancreatic cancer, hepatic perfusion disorders and small focal lesions. (b) CT three months later shows growth of the metastasis in the area of the THAD. It is important to recognize perfusion disorders because it can appear before the lesion is visible.
Fig. 14: CT in a patient with mass in pancreatic tail, shows multiples areas of THAD due to thrombosis of periferic portal branches. Also shows thrombosis of splenic vein (due to compression of the mass) with splenic perfusion abnormality. These findings disappear in portal phase.
**Fig. 15**: (15a) MRI in a patient with cholangiocarcinoma which affects left bile duct with lobar perfusion alteration. (15b). Steatosis in the left lobe in other patient with hiliar cholangiocarcinoma.
**Fig. 16:** Klatskin tumor with perfusion disorder in LHL and dilatation of the biliary duct. Dynamic CT and MRI images. In hilar cholangiocarcinomas are frequent the THAD with lobar distribution due to compression or affectation of the portal branches, although as occur in this case, there is not thrombosis of the left portal vein.
Fig. 17: In cases of portal thrombosis with posterior recanalization and cavernomatosis, hilar hepatic parenchyma is well vascularized, however periferic hepatic parenchyma receives less portal flow so it can show a THAD [1]. In this case it shows a patient with pylephlebitis (17a) and posterior development of portal cavernomatosis (17b).
Fig. 18: CT in a patient with segmental thrombosis of the portal vein of infectious etiology. In these cases perfusion disorders usually are more prominent. In addition this patient presents thrombosis of the splenic vein with splenic infarct.
Fig. 19: Acute infectious trombosis of the left portal vein with important alteration of hepatic perfusion, it is abnormal even in portal phase.
Fig. 20: CT in a patient with embolization of the right portal vein by interventional radiology procedure. This technique leads to hypertrophy the LHL and subsequently be able to resect the RHL. The liver shows typical THAD indicating that the procedure has been effective.
**Fig. 21:** CT in a patient with right heart failure and retrograde filling of the inferior vena cava, suprahepatic veins and hepatic perfusion disorder in arterial phase.
**Fig. 22:** Patient with thrombus in the right atrium and secondary stasis. CT shows a mosaic pattern which usually disappear in later phases.
Fig. 23: Patient with thrombosis of segmental suprahepatic branch and perfusion alteration associated.
Fig. 24: CT in a patient with hepatic vein thrombosis and hepatic perfusion disorder associated in portal phase
**Fig. 25:** Perfusion defect in portal phase in the area of falciform ligament which disappears in later phase.
Fig. 26: MRI in a patient with pancreatic cancer and vascular invasión. (26a) T1 images in-phase and out-of-phase show a focal steatosis area near of falciform ligament. (26b) T1 images after intravenous contrast administration show perfusion disorder in the same location.
Fig. 27: Perfusion disorder in LHL and abundant collateral circulation; indirect signs of thrombosis of the superior vena cava.
Fig. 28: CT and multiplanar reconstructions in other patient with thrombosis of the superior vena cava show typical perfusion alterations and collateral circulation, in this case through anterior abdominal wall veins (Burow and Baumgarten) to umbilical and Sappey veins. Intravenous contrast was injected from the left arm.
Fig. 29: Same patient that in Fig 28 with thrombosis of the superior vena cava, this time the contrast was injected from the right arm. There is retrograde flow into epigastric veins and then to the femoral and iliac veins (volume reconstruction). There is not THAD (axial images).
Fig. 30: CT axial images and reconstructions in other patient with obstruction of the superior vena cava show perfusion disorder in subdiaphragmatic region and in the LHL in CT with contrast injected through left arm (30a). Prominent collateral circulation in thoracic wall which drain to inferior vena cava through phrenic veins in CT images with contrast injected from the right side. In this case there is not perfusion disorder (30b).
**Fig. 31**: Contrast MRI and sequences in-phase and out-of-phase show perfusion disorder in perivascular region associated to a steatosis focal area. This finding is seen in 15% of the cases (or an area free of fat infiltration).
Fig. 32: Patient with acute cholecystitis and perfusion disorder in segment VI and VII of the right hepatic lobe.
**Fig. 33:** Abdomen CT in a patient with Rendu-Osler-Weber syndrome shows multiple dilated vessels and heterogeneous enhancement of hepatic parenchyma.
Fig. 34: CT and MRI in a patient who presents multiple transient hepatic enhancement without associated lesion. The only finding was a cyst in pancreatic tail.
Fig. 35: In alcoholic hepatitis we can see hepatomegaly and heterogeneous parenchyma due to diffuse steatosis and edema as well as focal perfusion abnormalities.
Fig. 36: Steatosis decreases the attenuation of the hepatic parenchyma in CT without contrast and vessels appear of low attenuation. Is more difficult see steatosis in a CT with contrast, however if it’s focal, as in this case, the affected area is similar to a perfusion abnormality, nevertheless the attenuation difference is not transient, it is seen in portal and later phases.
**Fig. 37:** When arterial flow is affected there is not compensatory increase of portal flow as we have seen previously. Besides if there is affectation of portal flow we can see hepatic infarction as in this patient.
Conclusion

This poster reviews the mechanisms that produce transient alterations of hepatic perfusion. Cases are presented associated with focal lesions (malignant and benign) and vascular changes, indicating their peculiarities. It is important to understand these phenomenom in order to avoid misdiagnosis or an overestimation of size. Knowledge about them can facilitate the detection and characterization of lesions.

Personal Information

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