

# Vascular Disorders of the Liver

## *systematic review on cross-sectional imaging*



Seco M, Graça B, Gonçalves B, Belo-Soares P, Curvo-Semedo L, Caseiro-Alves F

### Objectives:

- 1) To perform a systematic review of the vascular disorders of the liver as seen on cross-sectional imaging methods.
- 2) To understand the patho-physiologic mechanisms underlying some vascular lesions.

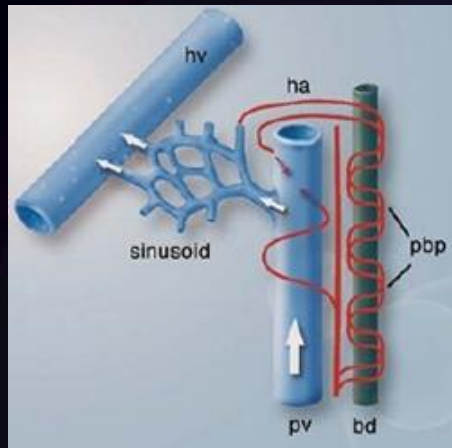
# CONTENTS

- 1) Introduction and liver vascular anatomy
- 2) Vascular pseudolesions and pitfalls
  1. Non-opacified hepatic vessels
  2. Portal venous inflow obstruction and THAD
  3. Intra-hepatic vascular shunts
  4. “Non-portal” venous supply to the liver
  5. Steal phenomena
  6. Post-surgical parasitic arterial flow
  7. Pericaval fat collection
- 3) Hereditary hemorrhagic telangiectasia
- 4) Budd-Chiari syndrome
- 5) Passive hepatic congestion
- 6) Hepatic infarction
- 7) Peliosis hepatis
- 8) Portal vein thrombosis
- 9) Portal hypertension
- 10) Summary
- 11) References

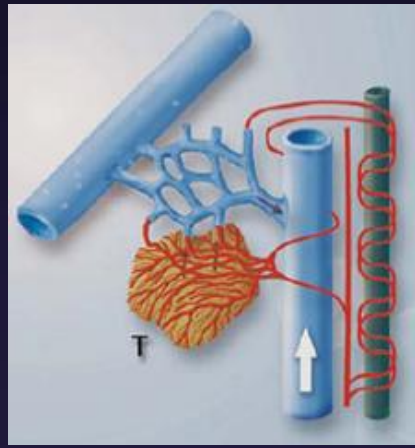
# 1. INTRODUCTION

- The liver, with its dual blood supply, receiving simultaneous arterial (20%) and venous blood (80%), is well suited to cross-sectional imaging, which can provide high intrinsic contrast and temporal resolution.
- The clear-cut separation of the hepatic phases of liver enhancement routinely achieved by state-of-the-art equipment creates additional problems, mostly related to the presence of perfusion abnormalities resulting in areas of abnormal liver enhancement.
- In most cases, they are caused by a selective impairment of the arterial or venous vascular feedings, which may communicate via intra-hepatic anastomoses, at the acinus level, at transplexal, transvasal or even transtumoral routes.
- Connections between the vascular systems in the liver are not restricted to arterio-portal communications but may also occur between the portal and the hepatic or systemic veins.
- In other occasions, the liver may be supplied by accessory hepatic arteries such as the inferior diaphragmatic, capsular or hilar arteries.

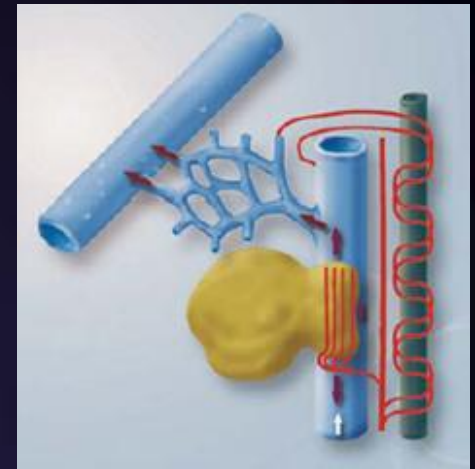
# Drawings of the hepatic microvasculature and arterioportal (AP) communications



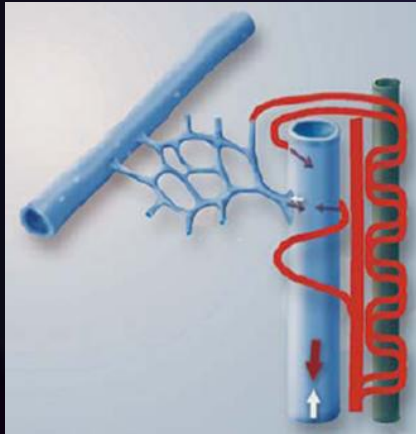
Normal circulation with physiologic AP communications. *ha*, hepatic artery; *hv*, hepatic vein; *pv*, portal vein; *bd*, biliary duct; *pbp*, peribiliary plexus.



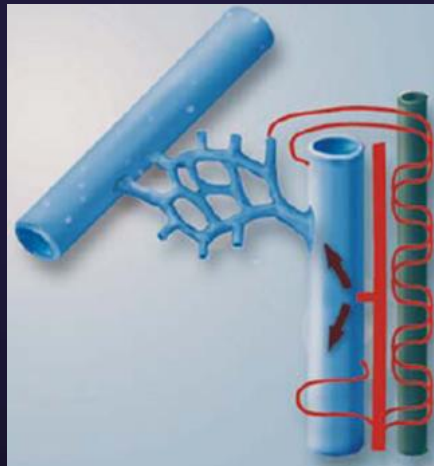
Tumoral AP shunt via a transtumoral route. A direct communication between the feeding arterial vessels of the tumor and the draining portal venules and/or sinusoids is established, resulting in increased arterial flow around the tumor. *T*, tumor.



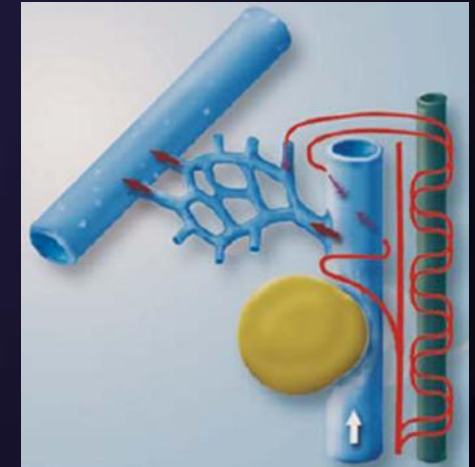
Tumoral AP shunt via a transvasal (vasa vasorum) route.



Cirrhosis with stretching and deformation of hepatic sinusoids causing a compensatory increase of the arterial flow. Note inversion of the direction of the portal vein flow.



Iatrogenic AP shunt caused by a fistulous tract after liver biopsy.



Portal flow reduction due to extrinsic compression resulting in a compensatory increase of the arterial blood flow.



# 1. VASCULAR ANATOMY – *Hepatic artery*



**Normal hepatic arteries.** The common hepatic artery (CHA) arises from the celiac trunk (CT). After giving rise to the gastroduodenal artery (GD), it continues as the proper hepatic artery (PHA), which divides into right hepatic artery (RHA) and left hepatic artery (LHA).

The RHA divides into an anterior and posterior branches and the LHA into branches supplying segment II, III, and IV.



**Aberrant origin of left hepatic artery (LHA),** which arises from the celiac trunk (CT). The common hepatic artery (CHA) has a normal trajectory, giving rise to the right hepatic artery (RHA).



**Complex anatomic variant.** Common hepatic artery (CHA) arises from the celiac trunk (CT), giving only a small branch to segment IV. Right hepatic artery (RHA) arises from superior mesenteric artery (SMA), and the remaining left lobe is supplied by a left hepatic artery (LHA) arising from the left gastric artery (LGA).

Variations in the hepatic arterial anatomy occur in approximately **42% of patients**. The commonest variant is a replaced RHA arising from the superior mesenteric artery (SMA), which is seen in **11% of cases**. A replaced LHA arising from the left gastric artery can also exist. The segment IV artery, which is usually a branch of LHA, originates from the common hepatic artery in **25% of individuals** or from the RHA.

# 1. VASCULAR ANATOMY – *Portal vein*



**Normal portal anatomy.** The main portal vein (PV) divides into right portal vein (RPV) and left portal vein (LPV). The RPV bifurcates into anterior branch (AB) and posterior branch (PB), both of which bifurcate into ascending and descending branches. Each of these four branches supplies a segment of the right lobe. The left portal vein (LPV) divides into three branches, one for each segment of the left lobe.



**Portal vein trifurcation.** Main portal vein divides into right anterior (AB) and posterior (PB) branches and left portal vein (LPV). A right portal vein is not identified.

Abnormal configurations of the portal vein appear in approximately 10% of cases. These variants mainly affect the RPV and may be classified into five types of branching as defined by the origin of the anterior branch.

**Type A** - normal anatomy (92% of cases). **Type B** - represents an early bifurcation or trifurcation.

**Type C** - extraparenchymal branching of the anterior branch from the LPV.

**Type D** - intraparenchymal branching of the anterior branch from the LPV. **Type E** - undivided main portal trunk.

# 1. VASCULAR ANATOMY – *Hepatic veins*



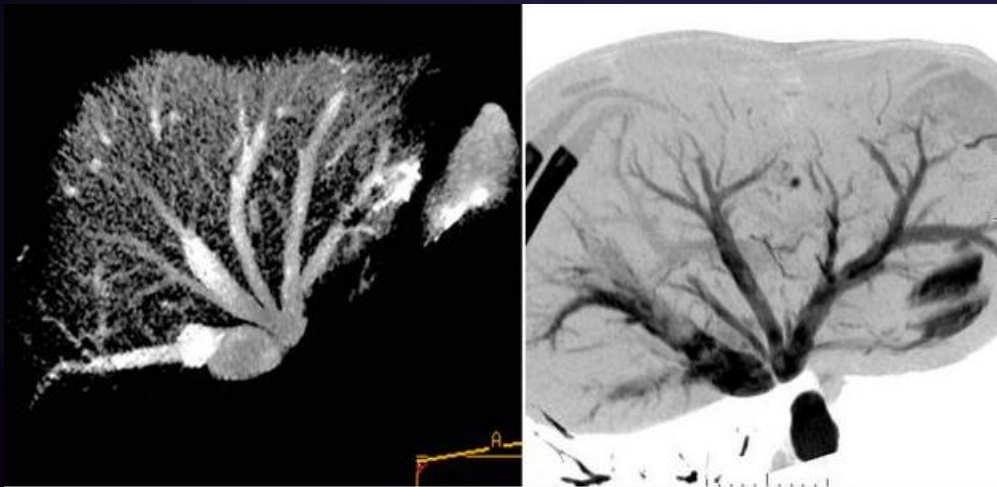
**Normal hepatic veins**, usually consisting of three main hepatic veins: right hepatic vein (RHV), middle hepatic vein (MHV) and left hepatic vein (LHV).

**The right hepatic vein (RHV)** is usually the largest and drains the most part of the right lobe.

**The left hepatic vein (LHV)** drains the II and III segments.

**The middle hepatic vein (MHV)** drains the central portion of the liver (segments IV, V, and VIII).

The site of drainage of the MHV is quite variable, either directly into the inferior vena cava (IVC) or as a common trunk with the LHV.



**Accessory hepatic veins.** They are significant for surgical purposes when their diameter is superior to 5mm.

Relevant variants of the hepatic venous system are short accessory RHVs that drain the posterior segments (VI and VII) independently into the IVC at a variable distance from RHV, happening more often in cases of non-dominant RHV.

The draining veins of segment IV can be multiple in number and very small in calibre, usually draining into the MHV. Left lobe variants are a LHV or segment III vein draining independently into the IVC, or the latter vessel draining into the MHV.

## 2. VASCULAR PSEUDOLESIONS AND PITFALLS

### Vascular Pseudo- Lesions and Pitfalls

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graph LR; A[Vascular Pseudo-Lesions and Pitfalls] --- B[Non-opacified hepatic vessels]; A --- C[Portal venous inflow obstruction/THAD]; A --- D[Intrahepatic vascular shunts]; A --- E["Non-portal" venous supply to the liver]; A --- F[Steal phenomena]; A --- G[Post-surgical parasitic arterial flow]; A --- H[Pericaval fat collection];
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Non-opacified  
hepatic vessels

Portal venous  
inflow  
obstruction/THAD

Intrahepatic  
vascular shunts

"Non-portal"  
venous supply to  
the liver

Steal phenomena

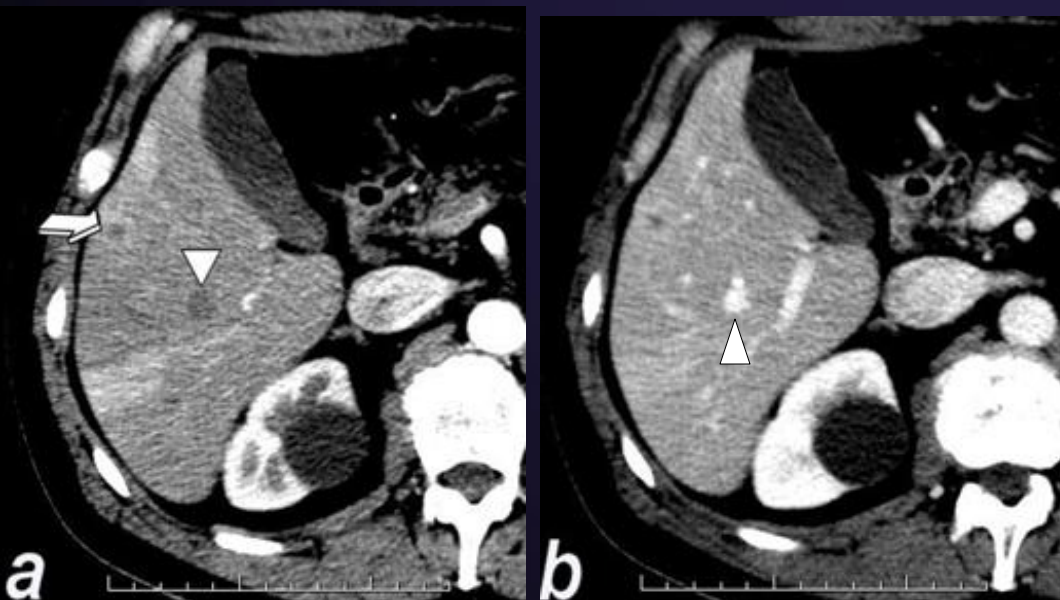
Post-surgical  
parasitic arterial  
flow

Pericaval fat  
collection



## 2.1 NON-OPACIFIED HEPATIC VESSELS

- Due to the high temporal resolution achieved nowadays by dynamic studies performed with multislice CT or hypergradient MRI, the different phases of liver enhancement can be clearly separated.
- Since portal and venous vessel enhancement occurs slightly later than arterial enhancement, at this stage a non-opacified intrahepatic venous vessel may be depicted.
- When seen end-on, this vascular structure may simulate a hypodense focal nodule, mimicking a true focal liver lesion.
- Besides the typical anatomical distribution, the comparison with later phases of enhancement should solve the problem promptly and avoid this common pitfall.

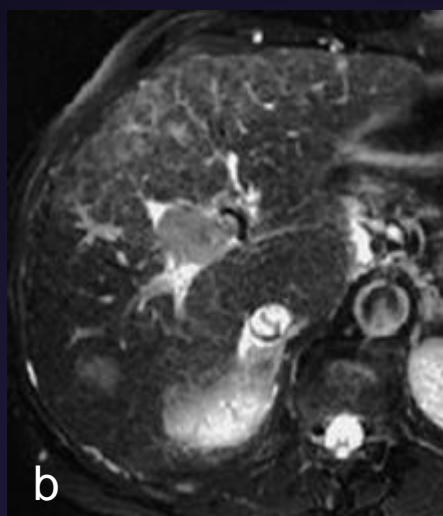
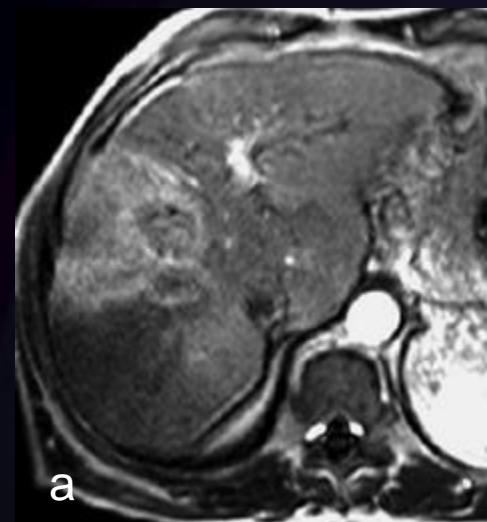


Dynamic MDCT obtained in a patient with rectal cancer during upper abdominal work-up depicting arterial (a) and portal (b) phases of liver enhancement. a) A small centimetre metastasis is depicted showing ring enhancement (arrow) as well as two THAD in segments V and VI related to other intra-hepatic metastasis not shown in the present scans. There is a pseudo-nodular appearance at segment V (arrowhead) that could easily mimic a focal malignant deposit. b) Comparison with the portal phase of liver enhancement clearly shows that the “nodule” corresponds to the unenhanced right hepatic vein seen end-on.

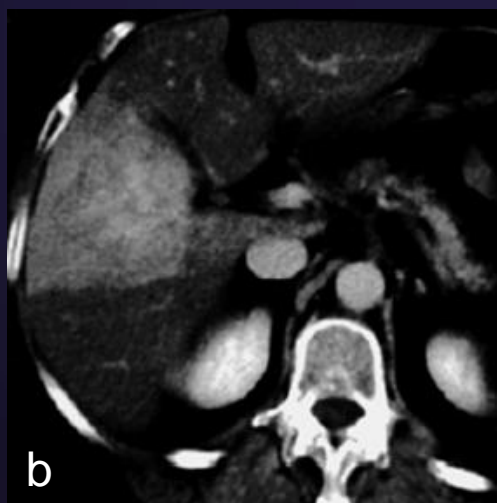
## 2.2 PORTAL VENOUS INFLOW OBSTRUCTION and THAD

- Reduction of the portal blood flow to the liver, either in the portal trunk or in the more peripheral, intra-hepatic portal branches, may give rise, at dynamic studies, to an area of parenchymal staining on the arterial phase due to the increased compensatory arterial flow, showing a rapid return to near isodensity on the subsequent portal venous phase.
- These areas are typically fan-shaped with a broad peripheral base and have been named transient hepatic attenuation differences (THAD).
- Reflecting a local vascular disturbance, THAD are rather non-specific imaging features, since they can be due to other causes, such as arteriovenous communications.
- Areas of hyperattenuation/hyperintensity of liver enhancement can also be depicted on dynamic studies around inflammatory processes such as liver abscesses or acute cholecystitis, artificially increasing the lesion size.

## 2.2 PORTAL VENOUS INFLOW OBSTRUCTION and THAD



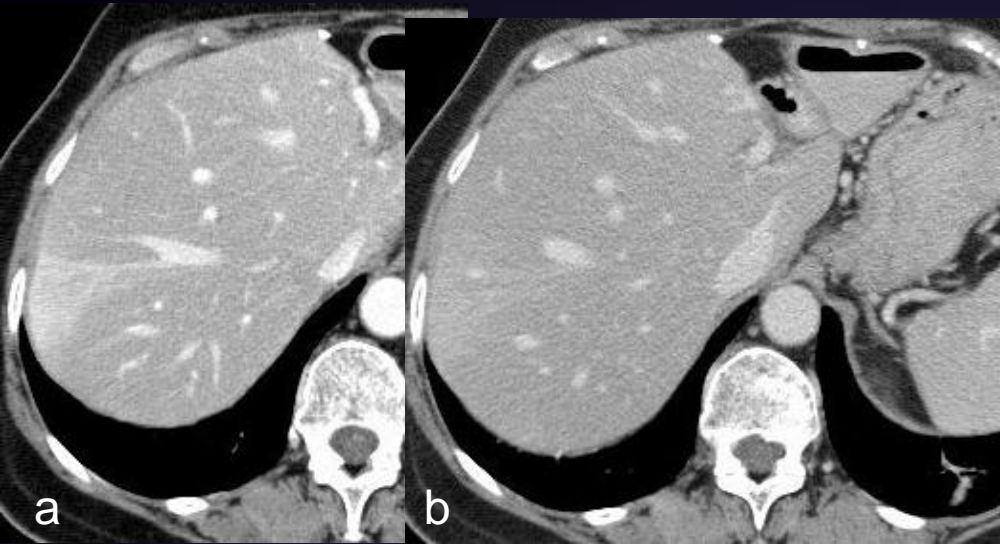
MR imaging of a patient with hepatocellular carcinoma and tumor thrombus within the right portal vein. **a)** The dynamic liver study obtained at the arterial phase of liver enhancement shows a fan-shaped area of hyperintensity with a broad capsular base corresponding to the THAD in the territory affected by the portal flow interruption. **b)** On the T2-weighted fast spin-echo sequence a tumoral portal vein thrombus is depicted causing vessel enlargement.



Dynamic helical CT in a patient with hepatic metastasis from lung cancer located at the central liver. **a)** Due to its central location with probable portal vein involvement there is a peripheral fan-shaped perfusion abnormality artificially increasing the lesion size. **b)** This finding is exacerbated at a later phase of liver enhancement due to the interstitial diffusion of iodinated contrast material within the metastasis.

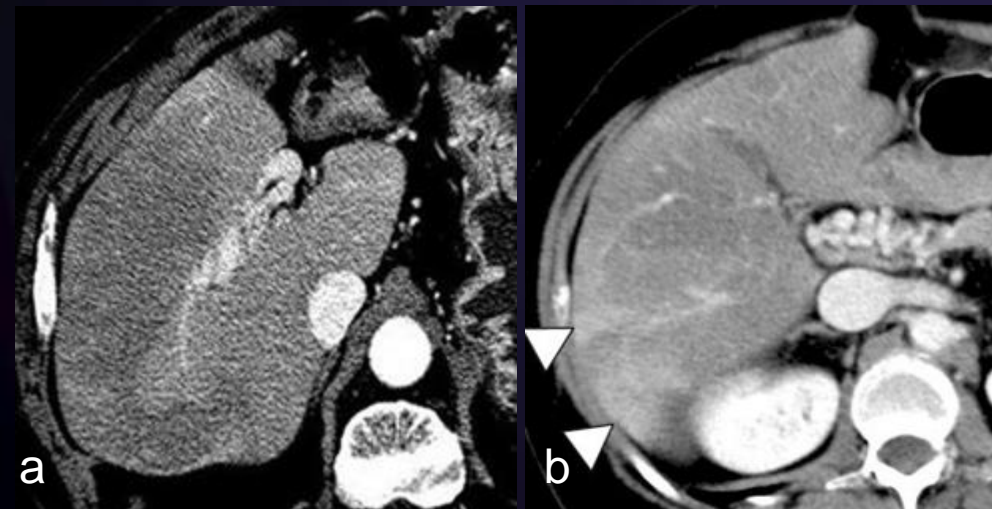
THAD can obscure or artificially increase the size of a focal lesion located within the hyper-attenuating fan-shaped area!

## 2.2 PORTAL VENOUS INFLOW OBSTRUCTION and THAD



Helical CT depicting a peripheral THAD of unknown origin in a patient with previous history of left hepatectomy for metastasis. **a)** A possible vascular mechanism for the THAD seen on segment VII was presumed since small venous collaterals are seen around the stomach (**b**) but without discernible portal vein thrombosis or cavernomatous transformation. **b)** At a later phase of enhancement the THAD returns to near isodensity with the remaining liver. A CT performed six months later did not show any additional abnormalities.

In some instances, THAD's nature is not apparent and a real intra-hepatic vascular mechanism remains to be proved since an associated focal liver lesion may not be seen.

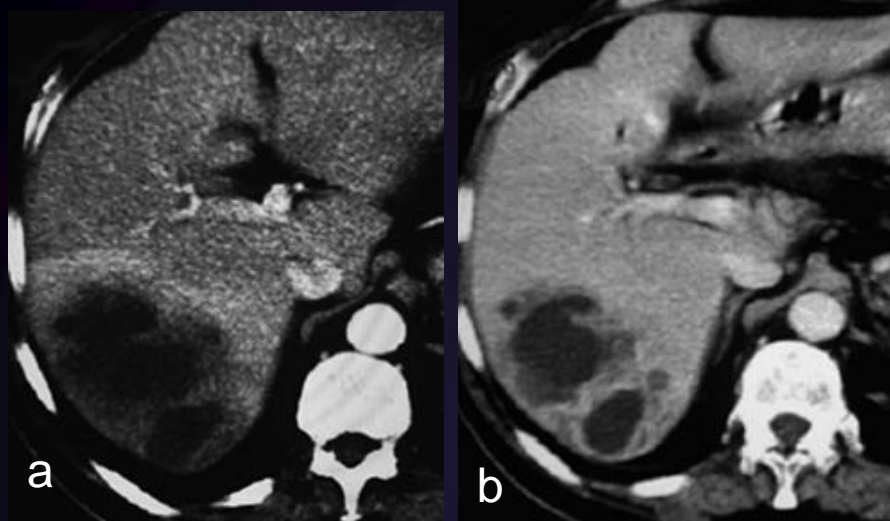


Dynamic multislice helical CT of two cases of portal cavernoma. **a)** The collateral hilar vessels show early staining and there is a better perfusion of the central liver displaying a higher level of enhancement compared to the peripheral parenchyma. **b)** Another patient showing several peripheral THAD at the right liver lobe (arrowheads).

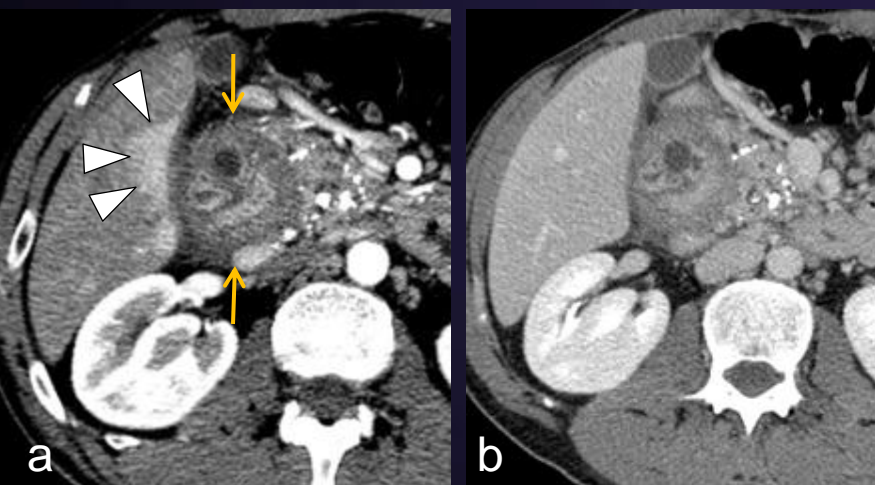
Cavernomatous transformation of the portal vein may cause an impairment of the portal flow to the liver, especially to most peripheral areas, since the hilar collateral vessels are insufficient to adequately supply them!



## 2.2 PORTAL VENOUS INFLOW OBSTRUCTION and THAD



**Dynamic CT of liver abscess.** **a)** There is a peripheral rim of increased attenuation at the late arterial phase of liver enhancement. **b)** A later phase of liver enhancement showing return to isodensity.



**Dynamic CT of groove pancreatitis** (arrows). **a)** Near the inflammatory process, there is an area of increased attenuation at the late arterial phase of liver enhancement (arrowheads) **b)** A later phase of liver enhancement showing return to isodensity.

The local hyperemia related to the inflammation itself increases arterial perfusion and the parenchymal compression exerted by the mass further contributes to locally reduce portal flow.



## 2.3 INTRAHEPATIC VASCULAR SHUNTS

Intra-hepatic shunts can be divided

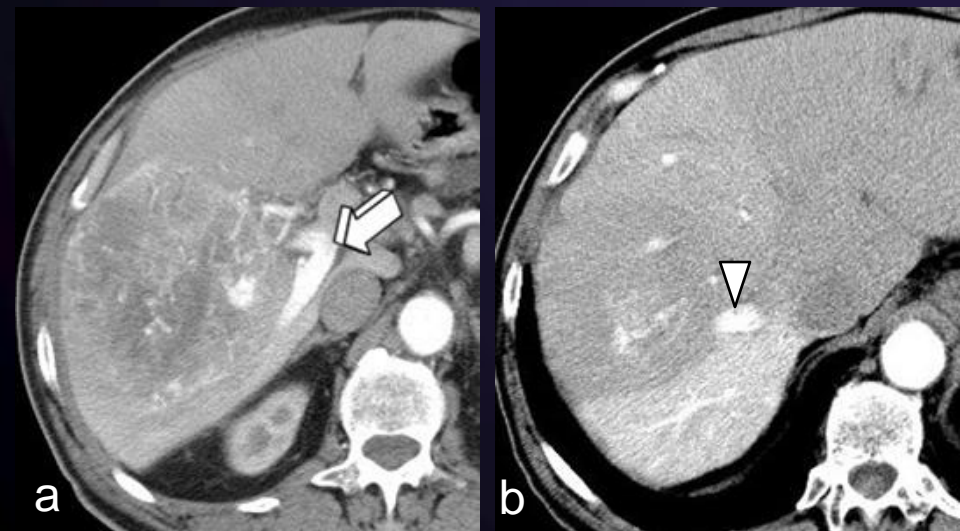
Depending on the underlying cause

Tumorous  
Non-tumorous

According to the vascular  
connection established

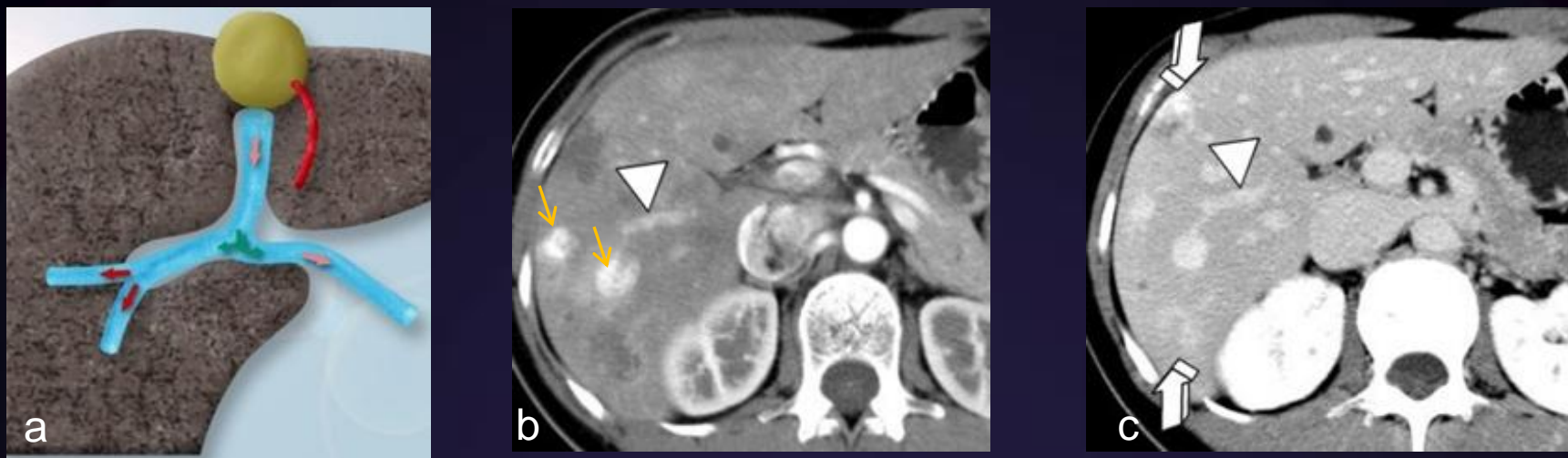
Arterioportal  
Arteriosystemic  
Portosystemic

- Tumoral AP shunting occurs essentially by a transtumoral route (see 4<sup>th</sup> slide).
- These vascular communications are generally below the threshold of detection with cross-sectional imaging, so only the parenchymal perfusion changes are depicted.
- **Imaging findings are manifested** by a peri-tumoral THAD, its size depending on the magnitude of the shunt, associated with early enhancement of the draining veins (portal or systemic). Besides the appearance of a THAD, a localized inversion of the portal flow may occur, demonstrated by early enhancement of portal branches during the arterial phase.



Dynamic helical MDCT study of two hypervascular hepatocellular carcinoma during the arterial phase of liver enhancement. **a)** Early enhancement of the portal vein consistent with **arterioportal shunting** (arrow). **b)** A THAD is seen at the posterior segments of the right liver lobe. An **arteriosystemic fistula** was suspected in this case due to the early enhancement of the right hepatic vein (arrowhead). Arterioportal shunting is generally observed in the context of HCC (transtumoral route, see 4th slide), although it may be iatrogenic, related to previous liver biopsies.

## 2.3 INTRAHEPATIC VASCULAR SHUNTS

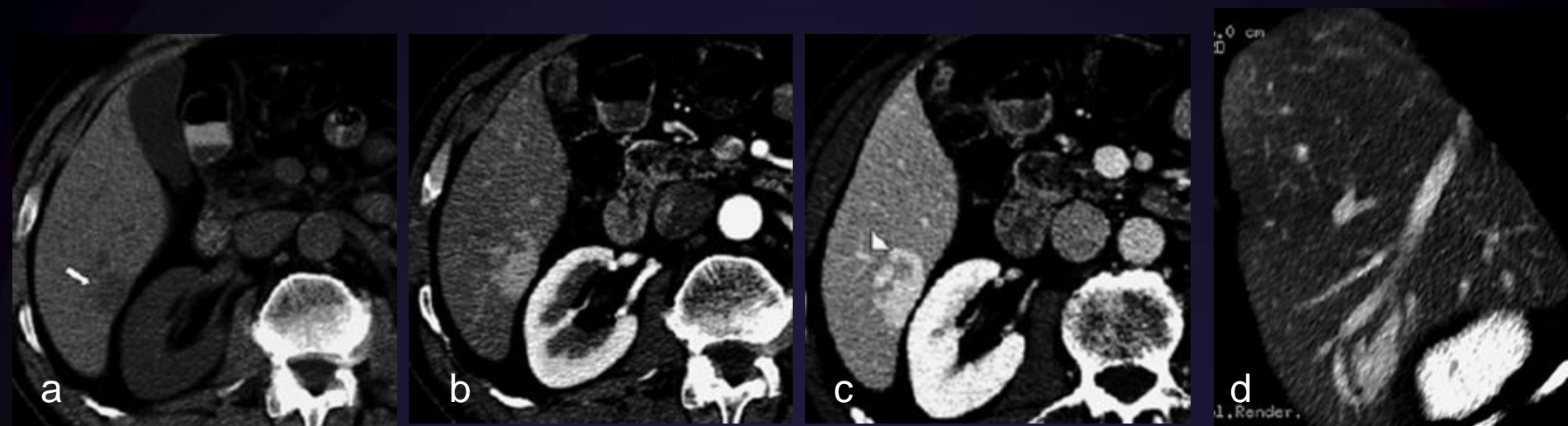


Schematic drawing (a) and dynamic CT (b and c) of a flash-filling hemangioma causing localized inversion of the portal vein flow. The dynamic CT depicts, on the arterial phase of liver enhancement (b), two small hypervascular hemangiomas (orange arrows) showing early and marked enhancement associated with subtle peri-tumoral enhancement representing perfusion abnormalities. There is concomitant enhancement of a small intra-hepatic branch of the portal vein near one of the neoplasms (arrowhead) traducing localized inversion of the portal blood flow. The right liver lobe also shows two additional hemangiomas that display the usual pattern of enhancement (arrows on c).

AP shunts can also occur with other liver tumors, such as the so-called "flash-filling" hemangiomas. In these circumstances the flow in the draining portal vessel may have an inverted direction.

AP shunts may also be seen in liver cirrhosis due to the parenchymal damage which tends to modify the hepatic flow dynamics (see 4<sup>th</sup> slide), and may appear as small arterial-enhancing nodules, which can cause potential confusion with HCC.

## 2.3 INTRAHEPATIC VASCULAR SHUNTS



**Multislice helical CT of intrahepatic portosystemic venous shunts of the internal subtypes.** **a)** On plain CT there is a hypodense nodule on segment VI (arrow). **b) and c)** Arterial and portal phases of dynamic liver study showing progressive enhancement of the “nodule” to the same extent as portal vessels. An abnormal branching structure corresponding to the connecting vein can be seen (arrowhead). **d)** MIP reconstruction showing the direct connection of the intrahepatic “nodule” with the right hepatic vein.

**Intra-hepatic portosystemic venous shunts (IPSVS) can also mimic a hypervascular liver lesion!**

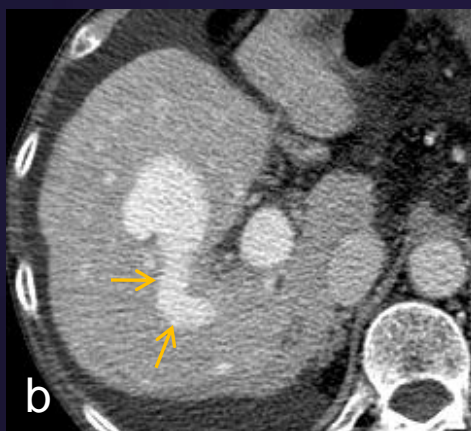
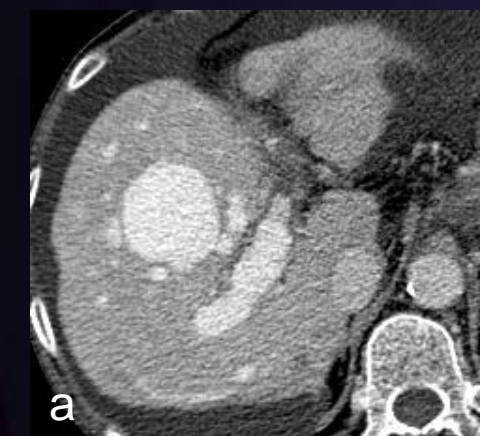
- They can be sub-divided in **internal** and **external** depending whether the portal vein communicates respectively with the hepatic vein or with a systemic vein outside the liver.
- IPSVS are seldom seen outside the setting of liver cirrhosis with portal hypertension, and in those cases a congenital origin has been postulated.
- They can be recognized by the demonstration of a direct connection between a dilated segment of the portal vein and the adjacent draining vein.



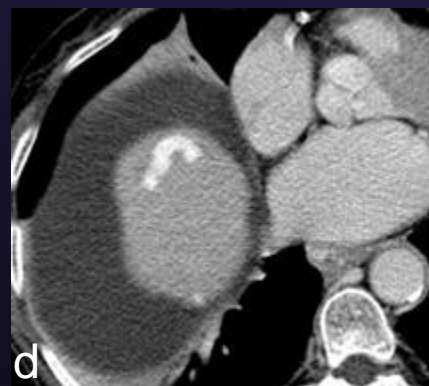
## 2.3 INTRAHEPATIC VASCULAR SHUNTS



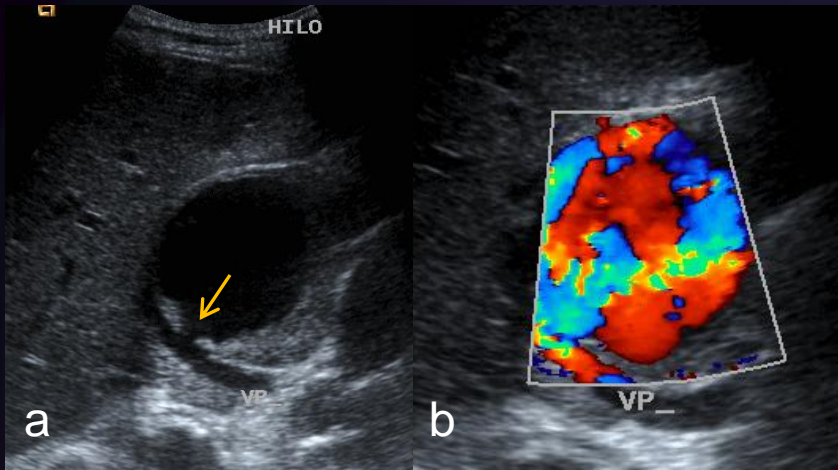
Multislice helical CT of intrahepatic portosystemic venous shunts of the external subtypes. a) Portal venous phase of liver enhancement depicting an abnormal vessel adjacent to the portal vein. b) This venous vessel runs to the periphery of segment V interconnecting with subcapsular systemic veins.



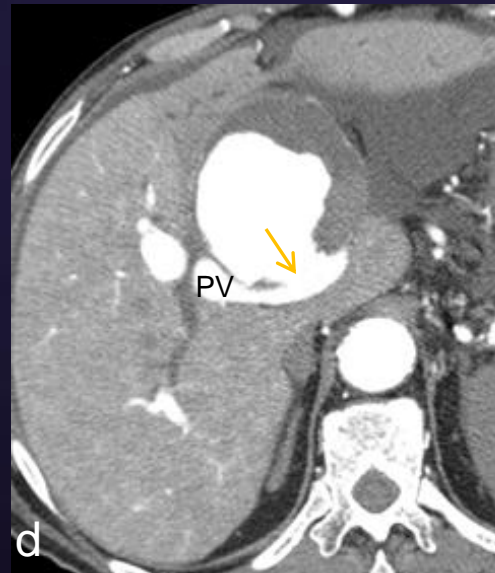
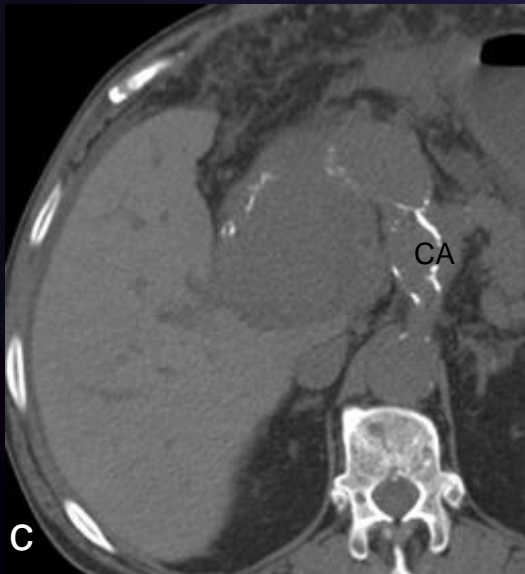
Multislice helical CT of another intrahepatic portosystemic venous shunts of the external subtypes. a) There is a "nodule" with the same enhancement as portal vein. b) A dilated branch (arrows) of the right portal vein constitutes the afferent vessel of this shunt. c) and d) The adjacent draining veins again run to the liver periphery interconnecting with subcapsular systemic veins. This patient had cirrhosis with portal hypertension.



## 2.3 INTRAHEPATIC VASCULAR SHUNTS



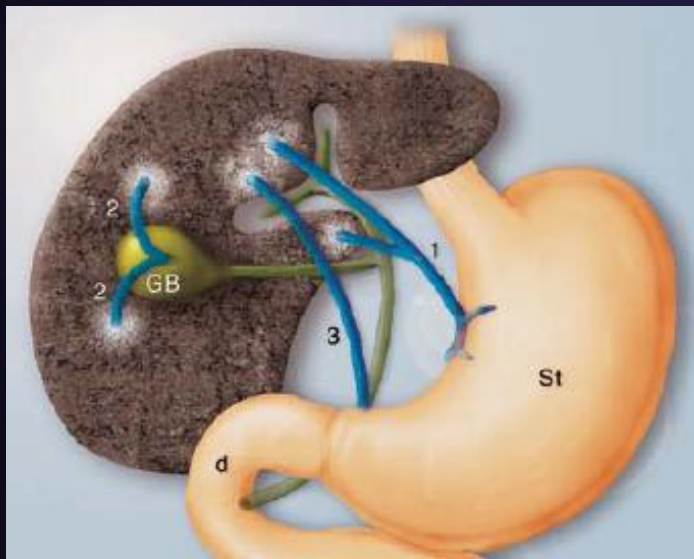
Hepatic artery pseudoaneurysm with a huge intrahepatic arterioportal shunt in a patient with previous pancreatitis. **a)** Ultrasound shows an anechoic formation at the porta hepatis with apparent communication (arrow) with a displaced portal vein (VP). **b)** Color Doppler readily demonstrated detectable flow inside it. **c)** Pre-contrast CT, showing two additional aneurysms of the celiac artery (CA). **d)** and **e)** There is identical simultaneous enhancement of the aorta, hepatic artery pseudoaneurysm (with mural thrombus) and the displaced portal vein, indicating an AP shunt. Like in US the communication is also well depicted (arrow).





## 2.4 “NON-PORTAL” VENOUS SUPPLY TO THE LIVER

- In some instances, veins from digestive organs may not flow into the portal vein trunk but instead directly drain into the liver parenchyma.
- Since parenchymal perfusion to these areas does not depend on portal flow they will show lack of enhancement on CT arterial portography, mimicking hypodense focal lesions.
- On dynamic CT/MRI they show early enhancement due to earlier venous return of less diluted contrast agent when compared with the portal blood.

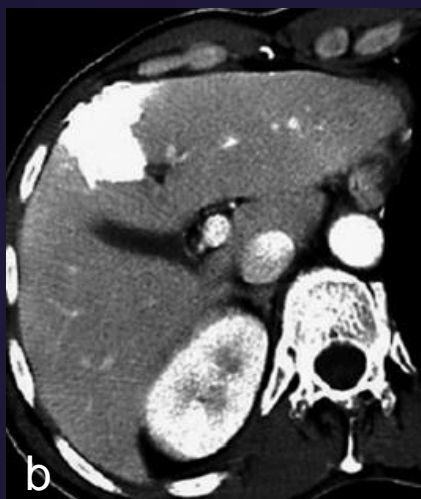
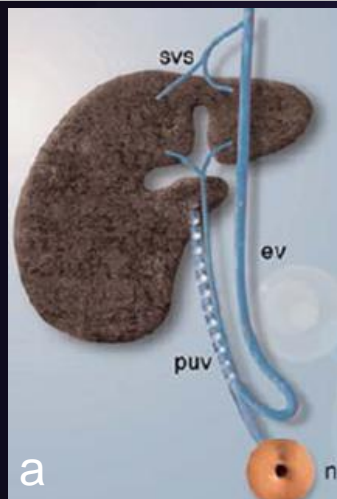


Schematic drawing of non-portal splanchnic perfusion to the liver parenchyma. 1 - aberrant gastric vein drainage to segments I and IV. 2 - cystic veins to segments IV and V. 3 - parabiliary venous system to the posterior aspect of segment IV. GB, gallbladder; ST, stomach; d, duodenum.

Another rare anastomotic network between the portal and systemic circulations can be seen through the veins of the coronary and falciform ligaments interconnecting the diaphragmatic veins to the portal system.

## 2.4 “NON-PORTAL” VENOUS SUPPLY TO THE LIVER

Apart from the vascular variants of the splanchnic circulation other systemic venous shunts can be associated with liver pseudo-lesions, as it is the case of superior vena cava (SVC) obstruction with subsequent development of a thoracic collateral circulation through the intercostal, internal mammary, hemi-azygos and paravertebral veins, ultimately connecting the superior epigastric vein to the portal system via the paraumbilical veins at the round ligament.

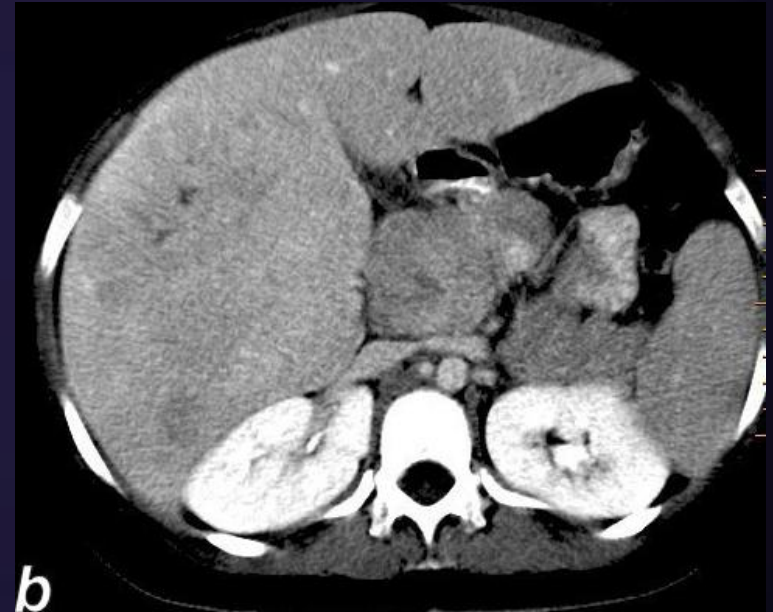


- a)** Schematic representation of the anastomosis of the thoracic vasculature via the epigastric veins interconnecting with the paraumbilical vein at the navel and abutting the superior and inferior veins of Sappey that drain into the liver parenchyma around the falciform ligament. *n*, navel; *puv*, paraumbilical vein; *ev*, epigastric vein; *svs*, superior veins of Sappey. **b)** Patient with superior vena cava syndrome, with intense and early enhancement of segment IV of the left liver lobe, mimicking a hypervascular focal lesion (courtesy of V. Vilgrain). **c)** Another case of superior vena cava syndrome showing subcapsular venous collateral vessels at the right liver lobe leading to a hyperdense pseudo-tumoral focal lesion.

In cases of SVC obstruction the recruitment of the collateral circulation can cause a dense focal parenchymal stain in the early phases of enhancement around the round ligament, the left portal vein or even at subcapsular areas, corresponding to an early arrival of minimally diluted contrast agent to those areas.

## 2.5 STEAL PHENOMENA

- On the arterial phase, the parenchyma around a hypervascular neoplasm can be significantly hypodense compared to the remainder of the normal liver since the arterial blood flow is strongly diverted to feed the hypervascular liver lesion.
- This phenomenon may be seen around benign or malignant liver tumors, being essentially dependent on the amount of arterial vascularization of the hepatic neoplasm.
- The steal phenomena is transitory and on the portal venous phase the affected parenchyma returns to isodensity.



Dynamic helical CT of an hypervascular liver metastasis from non-functioning malignant neuroendocrine pancreatic tumor. a) The primary tumor (arrow) and the liver metastasis (arrowheads) enhance similarly during the arterial phase of liver enhancement. The parenchyma around the metastasis is less dense than the normal left liver lobe due to steal phenomena. b) This finding is transitory and not depicted on the later phases of the dynamic study.



## 2.6 POST-SURGICAL PARASITIC ARTERIAL FLOW

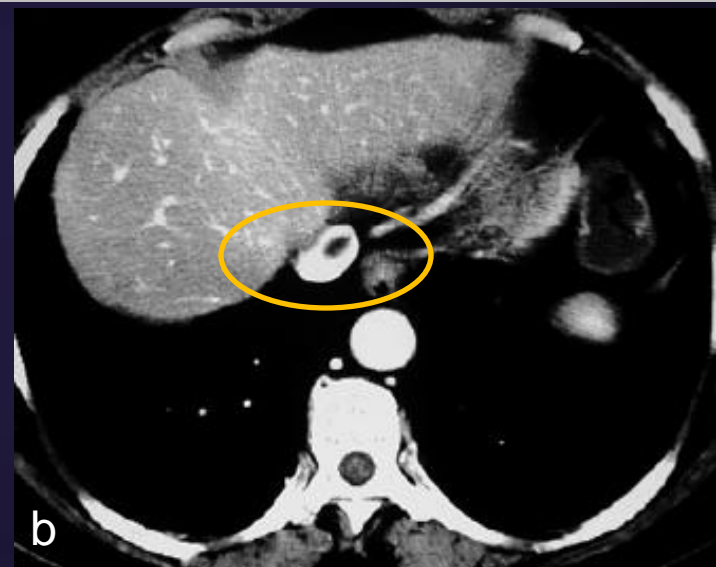
- In patients submitted to previous liver surgery, contrast-enhanced cross-sectional imaging can show early-enhancing non-neoplastic areas. They can be ill-defined, irregular, serpiginous, nodular or wedge-shaped, and are mostly seen on the resection margin along the liver edge.
- When these areas display a nodular appearance, they can be easily mistaken by a recurrent or "de novo" hypervascular neoplasm, namely HCC. A clue to their diagnosis resides on lack of detection on the plain scans and absence of washout through the later phases of liver enhancement.
- These pseudo-lesions possibly result from the aggressive surgical procedure with hepatic artery ligation to control bleeding, so that alternative pathways develop (accessory hepatic arteries, interlobar collaterals, right inferior diaphragmatic artery or even thin branches of the gastroduodenal or pancreatic arteries).



**Cross-sectional studies in a patient with previous history of right hepatectomy.** a) Dynamic helical CT showing a round hypervascular lesion adjacent to the resection margin in early arterial phase (arrowhead). b) This finding is not seen in a later phase of enhancement due to the interstitial diffusion of iodinated contrast agent from the portal supply. c) 3D MIP reconstruction depicts the arterial supply to the lesion from a branch of the right diaphragmatic artery (arrowheads).

## 2.7 PERICAVAL FAT COLLECTION

- This anatomic variant can be a source of error, mimicking intracaval fat-containing lesions or even lipomatous liver masses.
- The typical location is around the inferior vena cava (IVC) and due to volume averaging effects an intracaval thrombus may even be erroneously diagnosed.
- In general, observation of the adjacent contiguous slices is enough to solve any diagnostic dilemma, but in doubtful cases a second series of scans obtained at end-expiration can be performed in order to modify the anatomical relationship between the pericaval fat and the vessel itself.

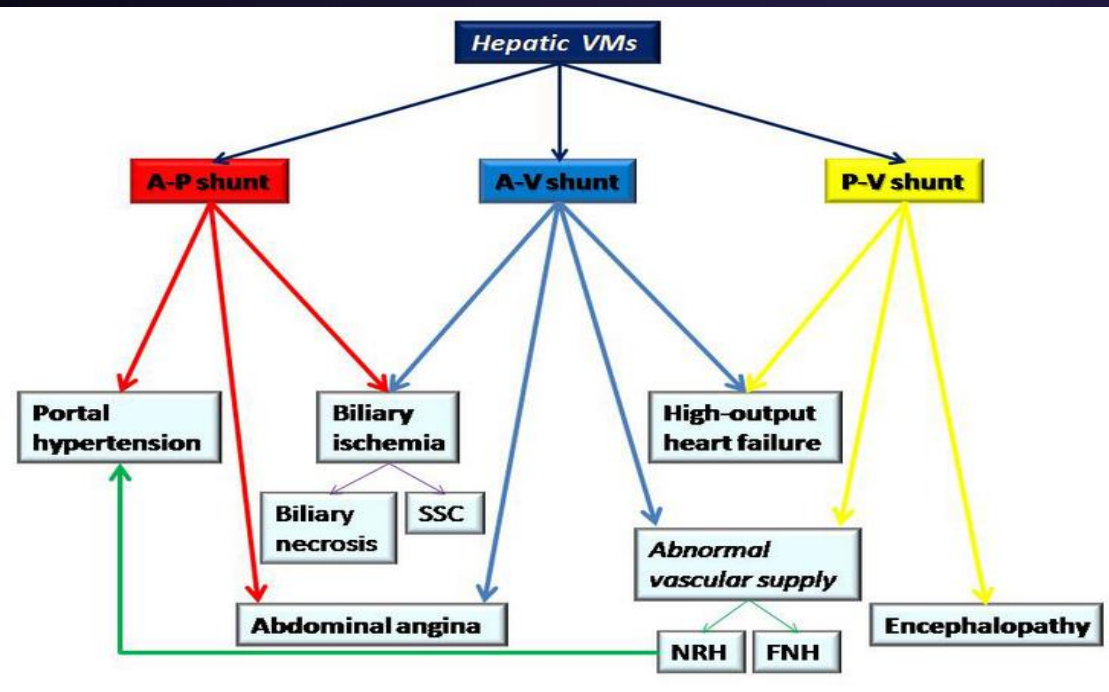


**Pericaval fat collection.** Helical dynamic CT obtained in a non-cirrhotic patient in end-inspiration (a) and during the portal phase of liver enhancement (b). There is a small fat collection around the IVC simulating an intracaval thrombus.



### 3. HEREDITARY HEMORRHAGIC TELANGIECTASIA

- Hereditary hemorrhagic telangiectasia (HHT), also known as Osler-Rendu-Weber disease, is a vascular disease with an autosomal dominant transmission, occurring with an estimated frequency of 10-20 per 100,000 individuals.
- Among visceral involvement, liver involvement has been considered uncommon, ranging from 8% to 31% in retrospective studies.
- Intra-hepatic telangiectases appear as rounded, strongly-enhancing lesions smaller than 10 mm, with a predominant peripheral distribution.
- Large confluent vascular masses are larger than 10 mm in diameter and consist of vascular pools showing early filling during the arterial phase and persistent enhancement.



Schematic representation of the pathophysiology of different presentations of liver involvement in HHT: the clinical findings are mainly due to vascular shunts. VMs- vascular malformations; SSC- secondary sclerosing cholangitis; NRH- nodular regenerative hyperplasia; FNH- focal nodular hyperplasia; A-P – arterio-portal; A-V – arterio – hepatic veins; P-V – portal-hepatic veins.

### 3. HEREDITARY HEMORRHAGIC TELANGIECTASIA

- Imaging findings are believed to be a consequence of the predominant pattern of hepatic shunting in each patient:

#### 1. Hepatic artery to portal vein shunt (**A-P shunts**)

- Early and prolonged enhancement of the portal vein during the early arterial phase.
- Sub-capsular, peripheral THADs on arterial phase; Dilatation of the hepatic artery and its branches.
- Dilatation of the portal vein > 13 mm, often with collateral vessels.
- Splenomegaly > 13 cm; Biliary strictures, dilatations and cysts.

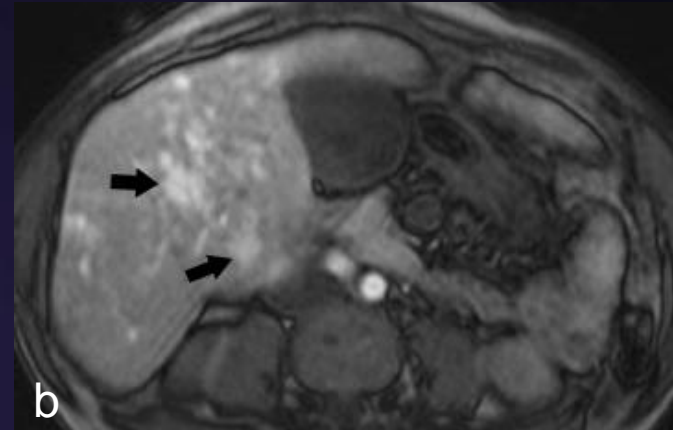
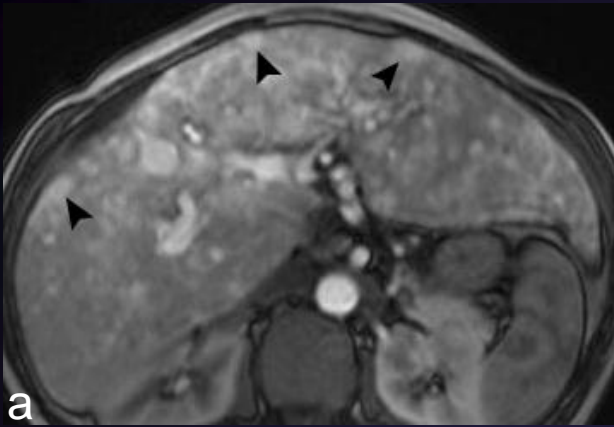
#### 2. Hepatic artery to hepatic veins (**A-V shunts**)

- Early enhancement of one or more hepatic veins during the early arterial phase.
- Enlargement of the early filled hepatic vein(s).
- Heterogeneous enhancement (mosaic pattern) during the arterial phase; Hepato-splenomegaly.
- Ascites; Biliary strictures, dilatations and cysts.

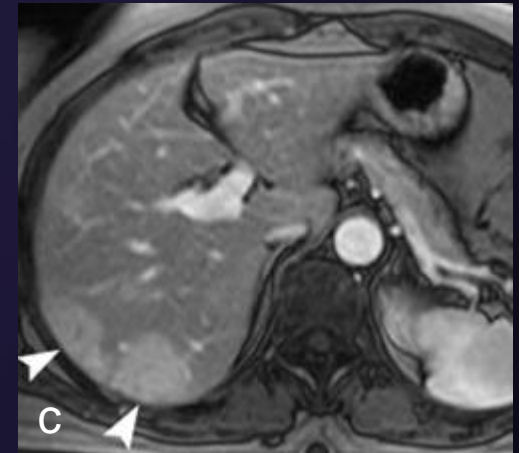
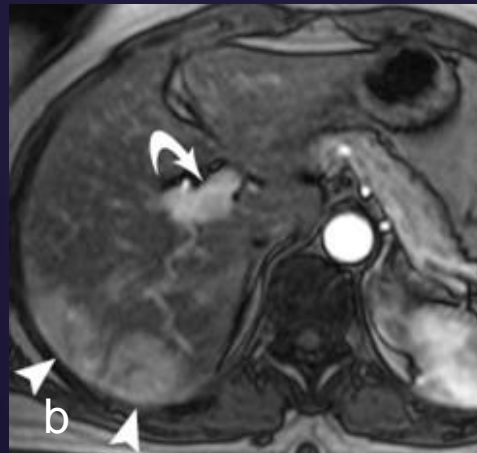
#### 3. Portal vein to hepatic veins (**P-V shunts**)

- Dilated portal branches communicating with large-calibre hepatic or systemic veins on portal venous phase.

### 3. HEREDITARY HEMORRHAGIC TELANGIECTASIA



**MRI. a)** Multiple small-sized rounded vascular lesions corresponding to intra-hepatic telangiectases are seen at the periphery of the liver (black arrowheads). **b)** Some early-filling lesions which correspond to large confluent vascular masses are depicted in the right lobe of the liver (black arrows).

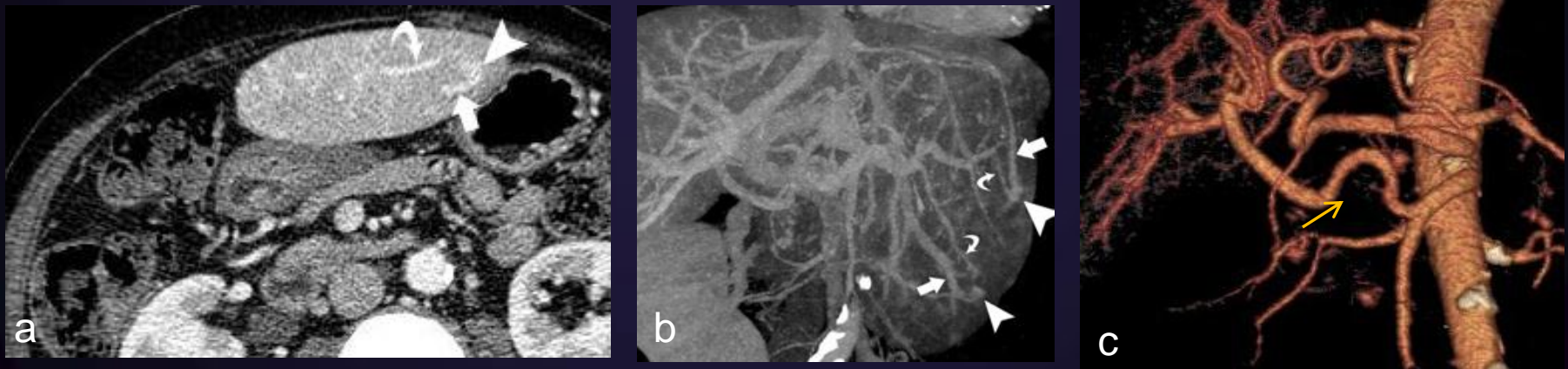


**MRI. a)** T2-w FS images depict several rounded mildly hyperintense lesions which consist of large confluent vascular masses (arrowheads). **b) and c)** On the dynamic, contrast-enhanced images they experience early filling and remain permanently enhanced throughout the entire study (arrowheads). On the arterial phase, simultaneous opacification of the portal vein, consistent with the presence of AP shunting, is apparent (b, curved arrow).

### 3. HEREDITARY HEMORRHAGIC TELANGIECTASIA



**CT. a)** In the early arterial phase there is opacification of the enlarged hepatic veins (arrowheads) while the SVC is still unenhanced, demonstrating the presence of an **AV shunt**. **b)** The hepatic artery is markedly tortuous and shows an enlarged calibre, while the liver enhancement is diffusely heterogeneous due to the presence of several small-sized rounded vascular lesions corresponding to **intra-hepatic telangiectases**. **c)** The portal vein (both the main trunk and some intra-hepatic branches, particularly in the left liver lobe) is markedly dilated and enhances maximally during the portal venous phase (arrows). **d)** Focal cystic dilatation of the intra-hepatic biliary tree of the right liver lobe is seen (arrow).



**CT. a) and b)** In the portal venous phase some vascular malformations (arrowheads) connecting the portal venous tree (curved arrows) with branches of hepatic veins (arrows) are detected and correspond to the presence of **P-V shunts**. **c)** This patient also shows tortuosity and dilatation of the hepatic artery. Note that the right hepatic artery (arrow) arises from the superior mesenteric artery.



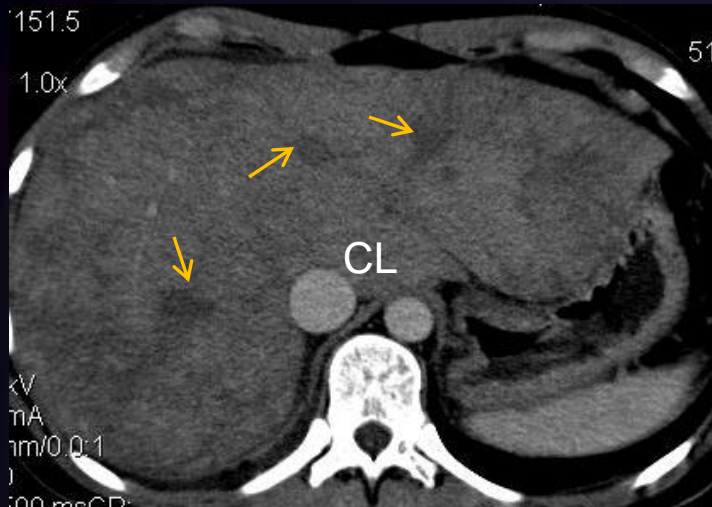
## 4. BUDD-CHIARI SYNDROME

Rare disorder that results from obstruction to hepatic venous outflow, at the level of either the large hepatic veins or the inferior vena cava (IVC).

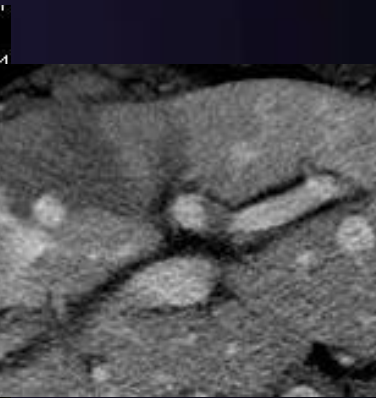
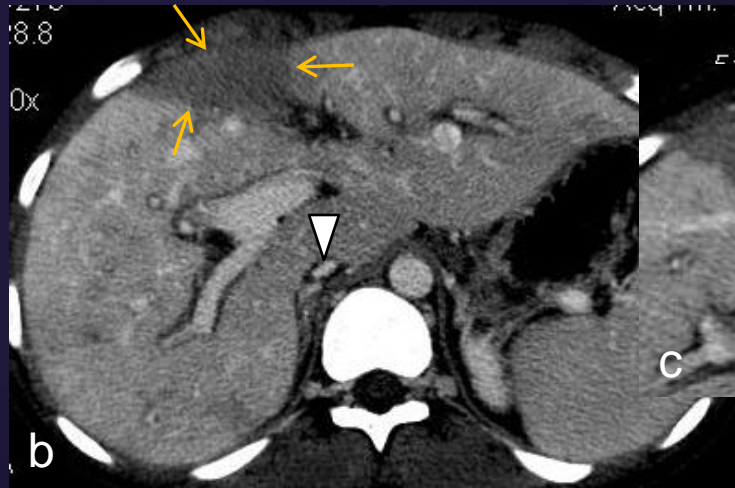


**Abnormal liver hemodynamics in the acute phase of Budd-Chiari syndrome.** a) Due to the hepatic venous flow obstruction there is a rising pressure in the portal system resulting in reduced intra-hepatic portal venous flow and compensatory increase of the arterial flow. Since there is a normal pressure gradient between the arterial and portal systems, **functional AP shunts are recruited**, which may ultimately lead to total inversion of the blood flow direction within the main portal vein, which acts as a draining vein. b) In the arterial phase of liver enhancement iodinated blood conveyed by the hepatic artery perfuses the central areas of the liver. Due to complete flow reversal within the portal vein early and isolated enhancement is observed (arrowhead).

## 4. BUDD-CHIARI SYNDROME

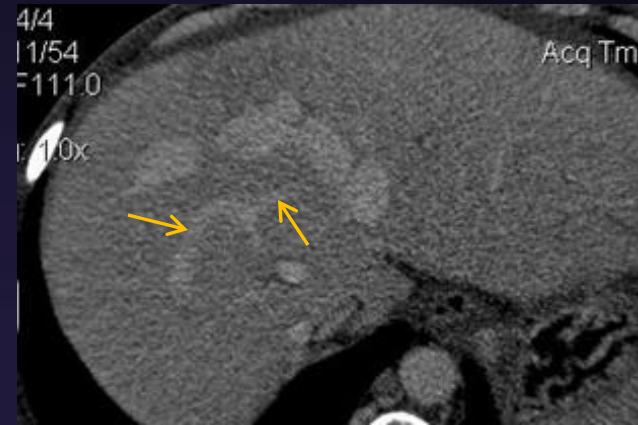
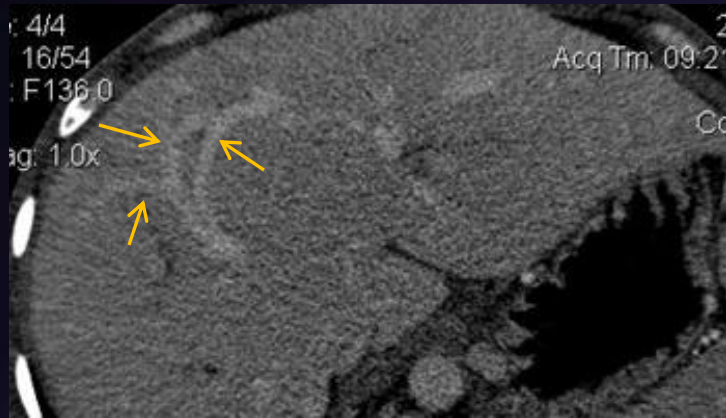


**Acute Budd-Chiari Syndrome.** On the later phases of enhancement a **mottled parenchymal appearance** is the result of the efferent vessel obstruction, causing pooling and distal accumulation of the intravascular contrast material. There is lack of hepatic vein enhancement (arrows) and the caudate lobe (CL) is enlarged.

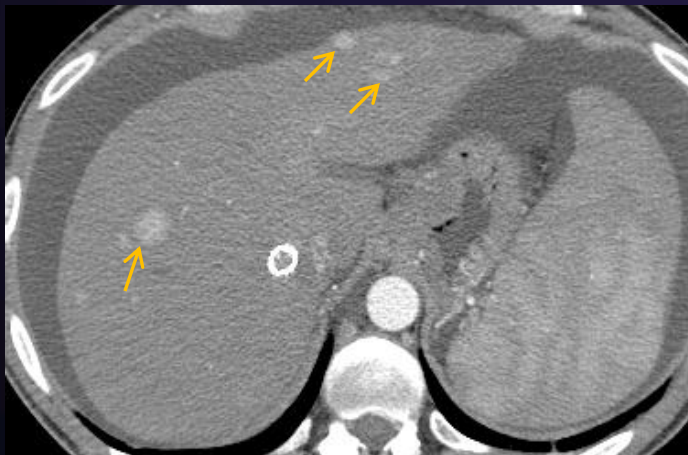


**Partial acute Budd-Chiari Syndrome.** **a)** There is occlusion of the right hepatic vein (arrow). **b)** Parenchymal swelling has compressed the vena cava to a slit (arrowheads). There is also a nonenhancing peripheral wedge-shaped region (segment IV), consistent with hepatic infarction. **Hepatic infarctions are occasionally present in Budd-Chiari syndrome.** **c)** Note also intrahepatic periportal lucency, a nonspecific finding, possibly due to lymphedema.

## 4. BUDD-CHIARI SYNDROME



On the chronic phase of Budd-Chiari disease the venous obstruction is well established, giving rise to the appearance of typical **comma-shaped branching vascular structures** (arrows), corresponding to an intra-hepatic network of venous collaterals trying to bypass the obstruction. These abnormal vessels tend to be peripherally located and most prominent around the caudate lobe (which is often enlarged due to its separate, autonomous venous drainage).



Some patients with **chronic Budd-Chiari Syndrome** develop large **benign regenerative nodules**, which are usually multiple and less than 4 cm in diameter. Post-contrast arterial phase CT (and MR) images demonstrate **hypervascular lesions** (arrows) that enhance homogeneously. Note the presence of a TIPS.



## 5. PASSIVE HEPATIC CONGESTION

- Complication of congestive heart failure or constrictive pericarditis.
- Elevated central venous pressure leads to decreased hepatic blood flow, elevated hepatic venous pressure and arterial hypoxia, leading to sinusoidal congestion, dilatation and perisinusoidal edema.
- The prominence of the hepatic veins and IVC helps to distinguish passive hepatic congestion from Budd-Chiari syndrome, that has poorly visualized or thrombosed intra-hepatic caval or hepatic veins.
- Ancillary findings: cardiomegaly, pleural effusions, ascites and intrahepatic perivascular lucency.

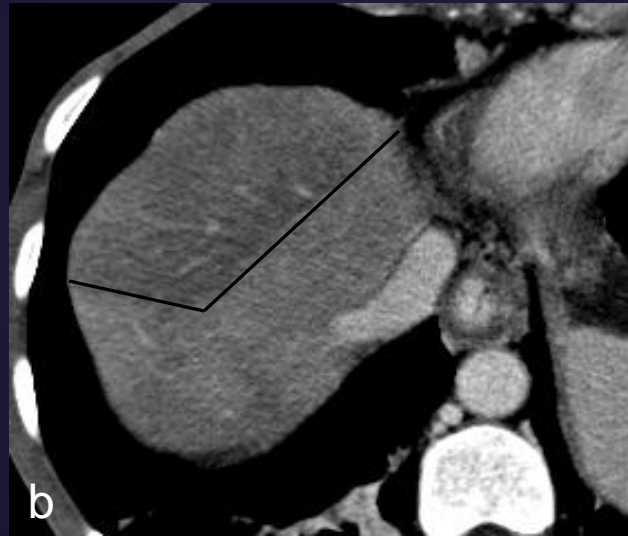
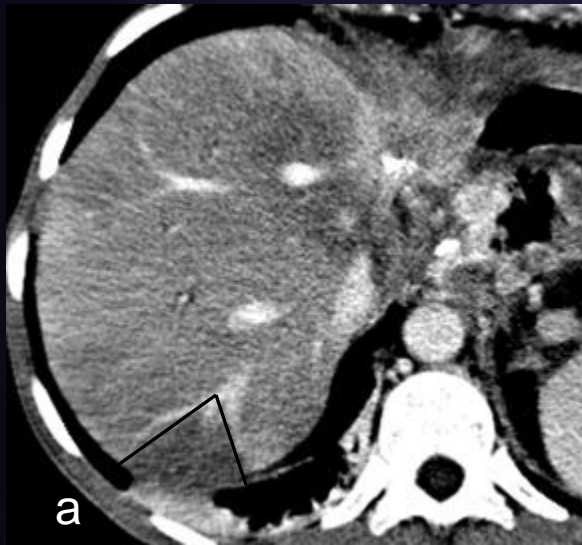


**Major imaging features of passive hepatic congestion resulting from severe right-sided heart failure. a)** Intense reflux of the iodinated contrast agent within the hepatic veins is seen shortly after the injection in a forearm vein, with severe delay in aortic enhancement. **b)** Mottled appearance of the liver parenchyma in the latter phase of liver enhancement due to stagnation of iodinated contrast material within hepatic sinusoids. There is a hypodense area on the spleen corresponding to a local infarction (arrowhead).



## 6. HEPATIC INFARCTION

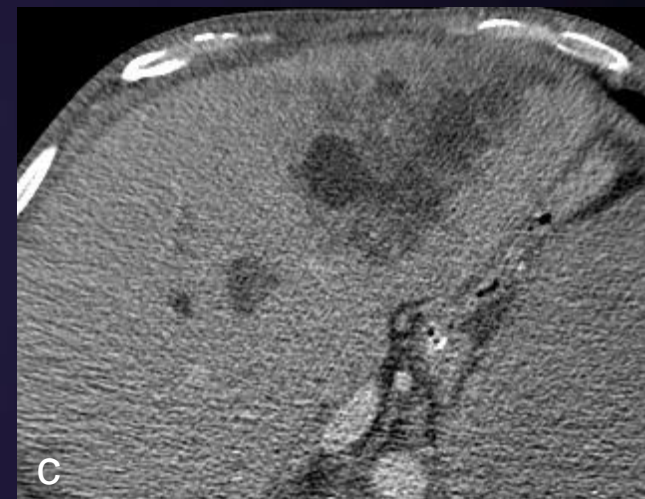
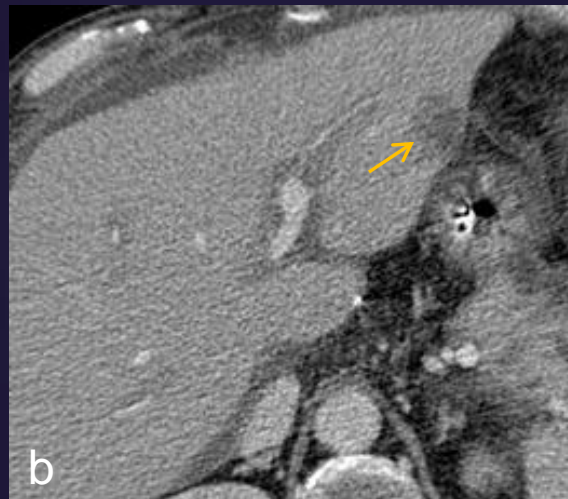
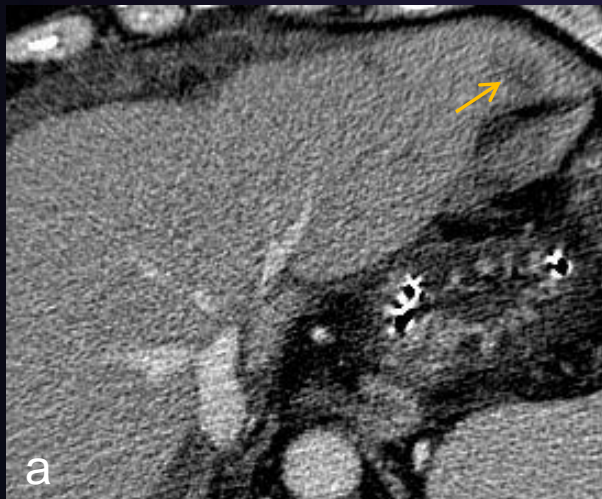
- Because the liver has a dual blood supply, hepatic infarction is uncommon. However, recent increase in the use of liver transplantation and laparoscopic cholecystectomy have led to an increase in the incidence of liver infarction.
- Three major configurations have been described:
  - 1) Wedge-shaped, usually peripheral
  - 2) Rounded, either peripheral or central
  - 3) Irregularly shaped lesions paralleling bile ducts



**Hepatic infarctions.** a) and b) Contrast enhanced CT shows peripheral wedge-shaped regions of low attenuation in a patient with hepatic artery occlusion by tumor (not shown). c) Contrast enhanced CT of a postoperative patient (central liver wedge resection) showing heterogeneous low attenuation of the right lobe due to right hepatic artery occlusion.

## 6. HEPATIC INFARCTION

- Necrosis may result in central gas collections.
- Chronic changes include atrophy of the involved segment and formation of cystic bile collections secondary to bile duct necrosis.
- On MRI, acute infarcts are hypointense on T1 and hyperintense on T2 images owing to edema, with lack of enhancement after Gd injection.



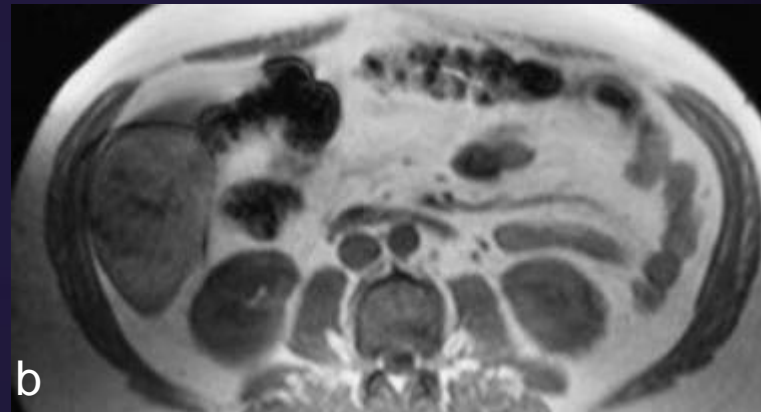
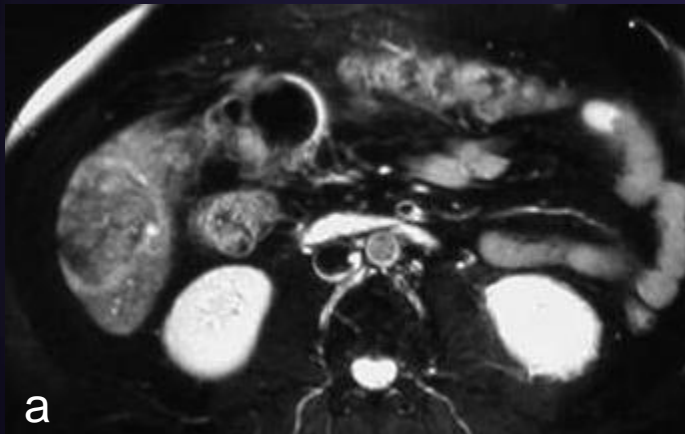
**Hepatic infarctions, atypical configurations.** a) and b) Peripheral, somewhat rounded regions of low attenuation (arrows) in a liver transplant, consistent with **rounded infarctions**. c) Another posttransplant hepatic infarction appearing as an irregularly shaped lesion paralleling bile ducts, caused by hepatic artery occlusion. **These atypical configurations of hepatic infarctions tend to occur in liver transplant patients.** In b) note also periportal low attenuation due to lymphedema, a normal finding in the first 6 months after transplantation.

## 7. PELIOSIS HEPATIS

- Peliosis hepatis is an uncommon benign disorder of the liver, characterized by oval and irregularly shaped blood-filled spaces, which are lined either by hepatocytes or endothelial cells.
- These cavities communicate with the sinusoids, many of which are dilated.
- A number of theories have been postulated to explain the etiology, such as outflow obstruction of the blood flow at the sinusoidal level, hepatocellular necrosis, or direct lesions of the sinusoidal barrier.
- The imaging appearance of peliosis hepatis is very variable and depends on lesion size, the extent of communication with the sinusoids, the presence of hepatic steatosis and the presence of complications such as thrombosis or hemorrhage within a lesion.

## 7. PELIOSIS HEPATIS

- On precontrast CT images the lesions are usually iso- to hypodense. However, in some cases hyperdense spots are found in these lesion, representing focal hemorrhage.
- Following contrast injection the lesions are hypoattenuating. During a later venous stage the liver mostly becomes homogeneous again.
- At MRI the multiple hepatic foci in peliosis hepatis show a high signal on T2-weighted images and a variable signal on T1- and proton density-weighted images, presumably reflecting various stages of subacute hemorrhage.



**MR.** In this patient with a **focal form of peliosis hepatis**, the lesion is heterogeneous with predominant low-signal intensity on T2 w FS (a) and T1-w (b) images, due to areas of old hemorrhage with deposition of hemosiderin.

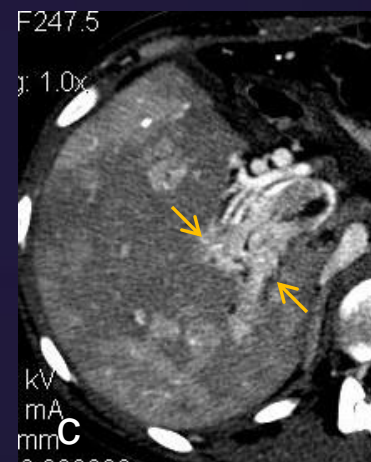
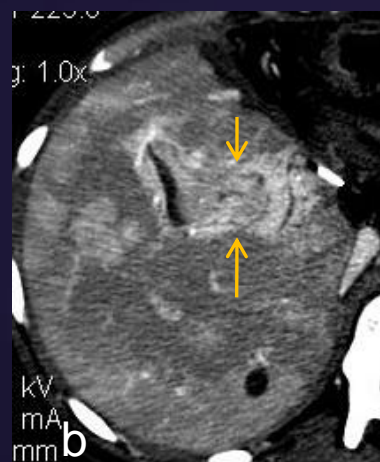
Angiography can be of use since it can differentiate peliosis hepatis from a vascular malformation or tumour. Angiography shows multiple round collections of contrast material, best seen in the late arterial phase.



## 8. PORTAL VEIN THROMBOSIS



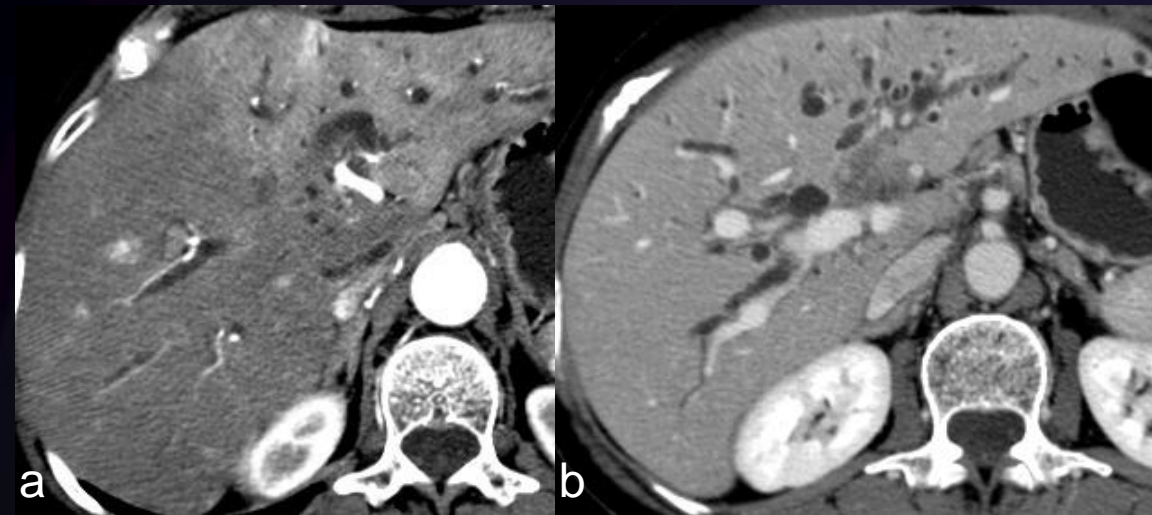
**Bland portal vein thrombus.** Post-contrast CT images demonstrate an intraluminal filling defect. **Non-tumorous portal venous thrombus should not enhance in the portal venous phase.** There is marked enhancement of the wall of the portal vein (arrows), likely due to flow through dilated vasa vasorum.



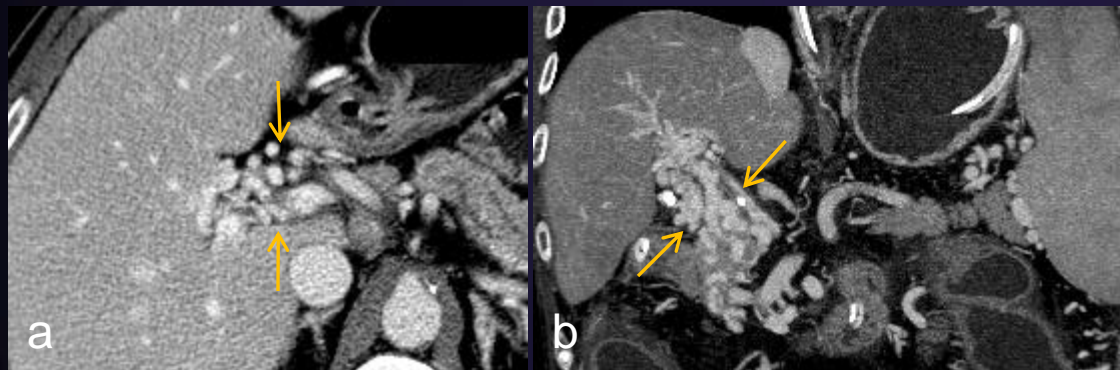
**Tumoral thrombus in a patient with recurrent HCC after left hemihepatectomy.** **a)** Pre-contrast CT shows marked enlargement of portal vein. **b) and c)** In the late arterial phase there is diffuse enhancement within the obstructed portal vein (arrows) indicating the presence of tumor thrombus. There is also peripheral areas of increased attenuation, which disappear in the portal venous phase (**d**), representing THAD.

Two flow-related phenomena that can be seen during dynamic contrast-enhanced imaging are **THAD** and **diminished enhancement during the portal venous phase**.

## 8. PORTAL VEIN THROMBOSIS



Left portal vein thrombosis in a patient with cholangiocarcinoma. **a)** Note the THAD resulting in increased attenuation of the left hepatic lobe in the arterial phase. **b)** In portal phase there is bile duct dilatation due to the tumor (not shown) but the liver parenchyma is homogeneous.

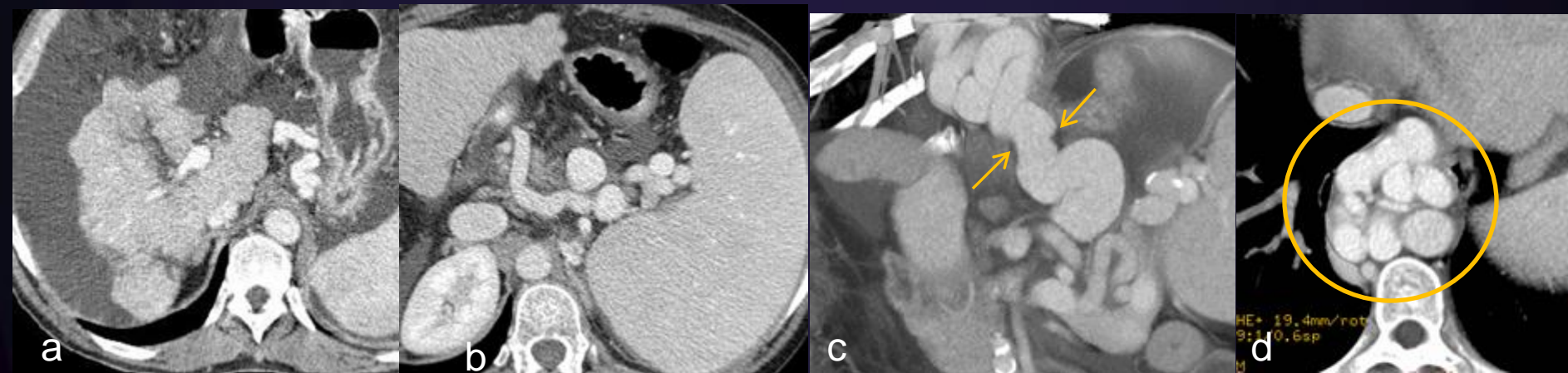


Chronic cavernous transformation of the portal vein. **a) and b)** Contrast-enhanced CT shows a tangle of collateral vessels (arrows) that have formed in the porta hepatis after thrombosis of the main portal vein. The splenomegaly in **b)** is due to secondary portal hypertension.

- Decreased hepatic lobar attenuation on precontrast images is postulated to be due to hepatic glycogen depletion and increased hepatocyte fat content.
- Portosystemic collateral veins may develop as a consequence of portal venous hypertension.

## 9. PORTAL HYPERTENSION

- Results from increased portal venous resistance, the cause of which may be prehepatic, intrahepatic or posthepatic.
- Rarely may be due to a hyperkinetic condition caused by an arterial-portal fistula (traumatic, congenital, neoplastic).
- The pressure increase induces the formation of portosystemic collaterals which are usually dilated pre-existing vessels, but active angiogenesis is also seen.



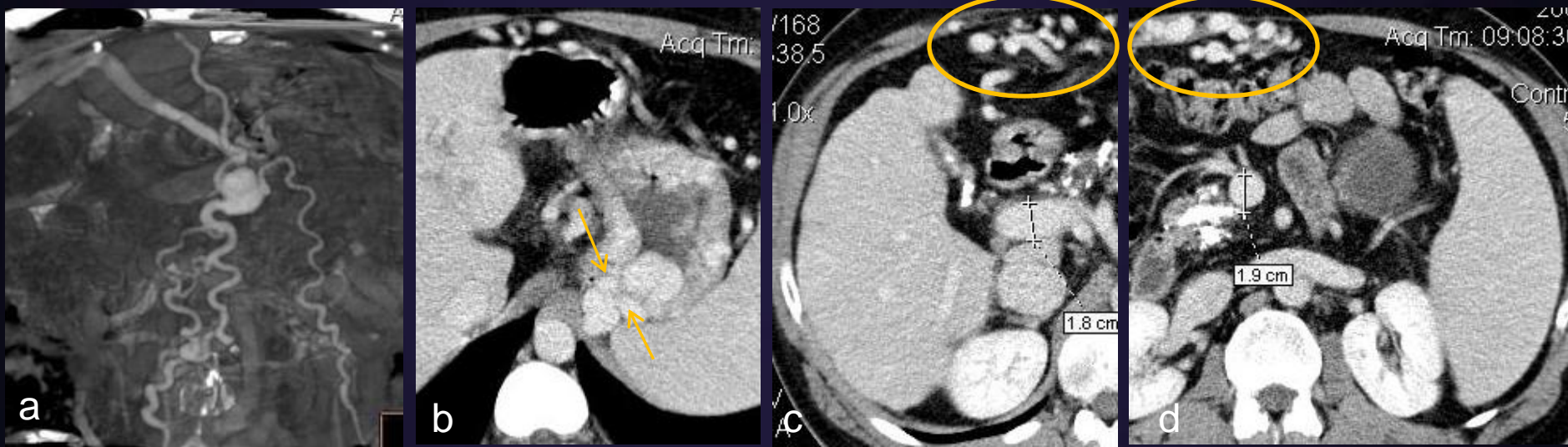
**Contrast-enhanced CT of common collateral pathways in portal hypertension.** **a)** Dilatation of a vascular structure, adjacent to the lesser curvature of the stomach, corresponding to a right gastric varix. **b)** Tangle of vessels are seen at the splenic hilum, corresponding to short gastric varices. **c) and d)** Marked enlargement of left gastric vein (arrows) leading to tortuous vascular structures adjacent to the lower esophagus, corresponding to paraesophageal varices (circle).



## 9. PORTAL HYPERTENSION

Common collateral pathways include:

- the left gastric, posterior gastric and short gastric veins to the esophageal and paraesophageal veins;
- gastrosplenic, gastrosplenic and splenorenal shunts to the left renal vein
- several pathways including the omental, paraumbilical, hemorrhoidal and retroperitoneal (Retzius) veins.



**Contrast-enhanced CT in portal hypertension.** a) Patent paraumbilical vein connecting with subcutaneous vessels of the anterior abdominal wall, creating the caput medusae sign. b) Stomach fundus varix (arrow). c) and d) An increased diameter of the portal vein (> 13mm) and the superior mesenteric and splenic veins (>10mm) with development of paraumbilical collaterals (> 3mm) are highly specific although only moderately sensitive signs of portal hypertension. In this case the diameter of portal vein is 1.8 cm and that of the superior mesenteric vein is 1.9 cm. There are also marked paraumbilical collaterals (circle) and splenomegaly.



## 10. SUMMARY

- Vascular disorders of the liver may not only mimic but also conceal focal liver lesions.
- Awareness of the imaging spectrum and knowledge of the underlying mechanisms of vascular disorders of the liver can avoid a substantial amount of interpretative pitfalls, increasing the diagnostic accuracy in this group of conditions.

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