LEARNING OBJECTIVES

1) To understand the patho-physiologic mechanisms underlying some vascular lesions.

2) To review the CT findings of vascular disorders and perfusion abnormalities.

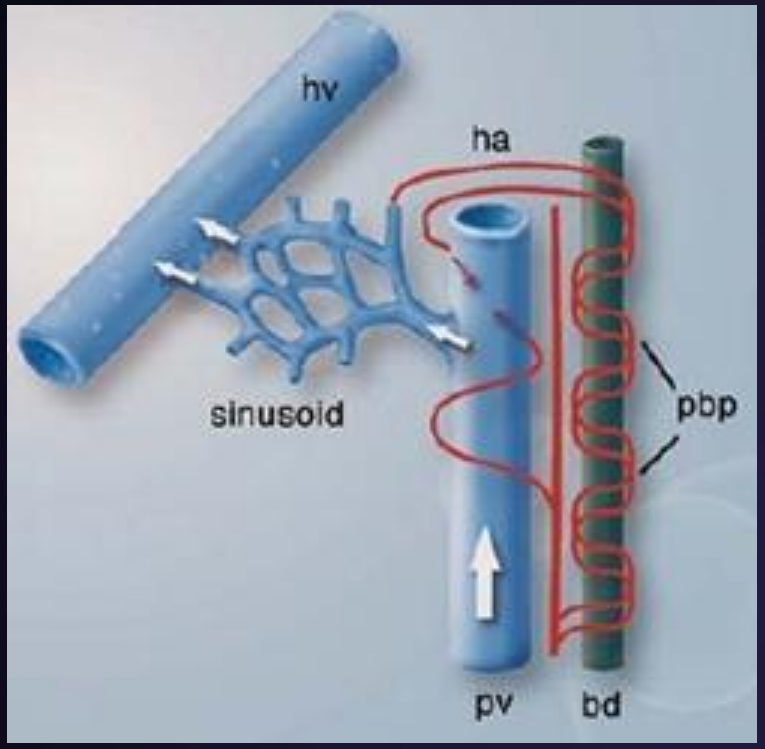
BACKGROUND

- 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.

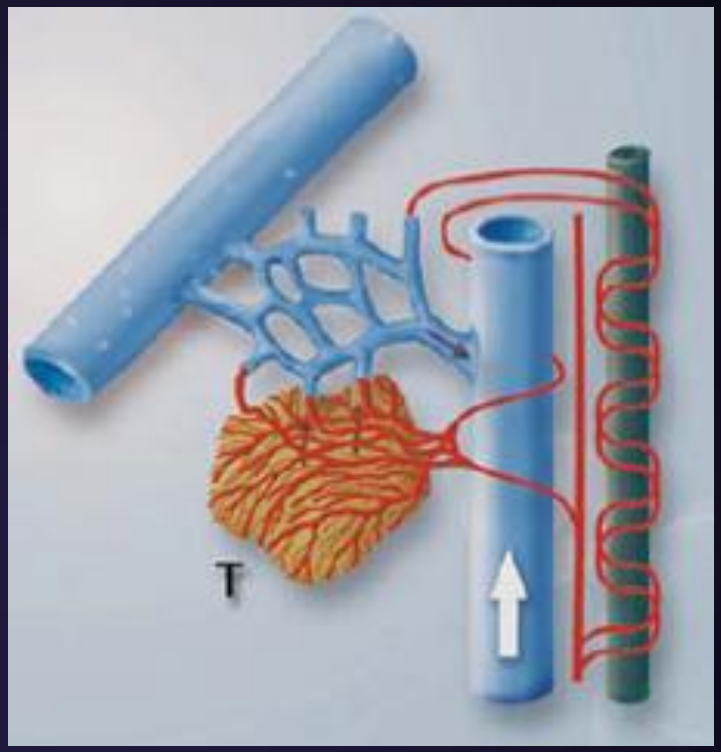
BACKGROUND

- 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.
- On the venous counterpart, an accessory network of systemic veins, the so-called "non-portal venous blood" can drain directly into the liver parenchyma, as it may happen with the parabiliary venous plexus, the cystic veins, the veins of Sappey, or the aberrant drainage of the gastric vein.
- All these feeding vessels may ultimately mimic or conceal focal liver lesions, and thus represent a potential source of interpretation errors.

Drawings of the hepatic microvasculature and arterioportal (AP) communications

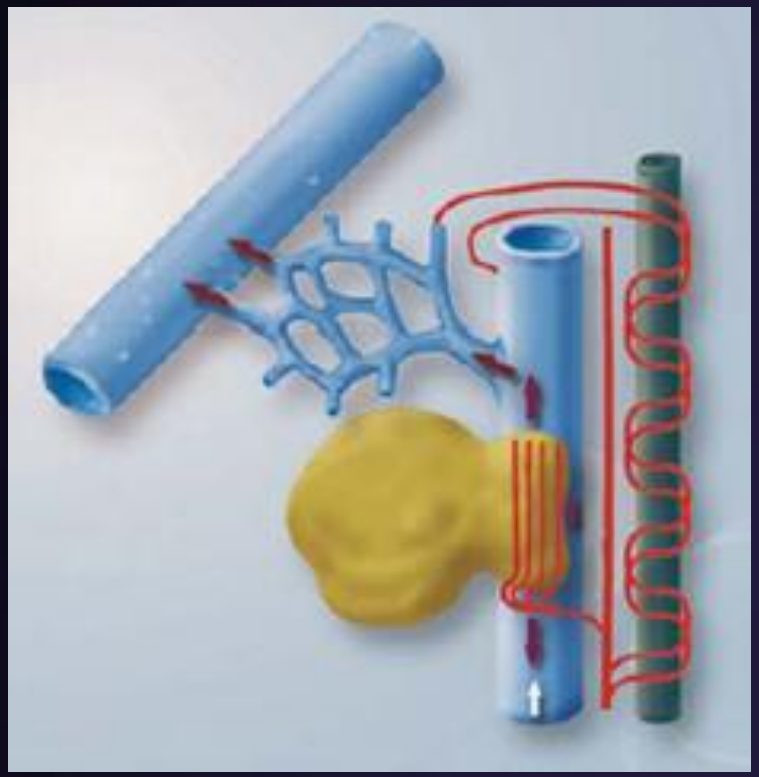


Normal circulation with physiologic AP communications. *ha, hepatic artery; hv, hepatic vein; pv, portal vein; bd, biliary* duct; *pbp, peribiliary plexus.*

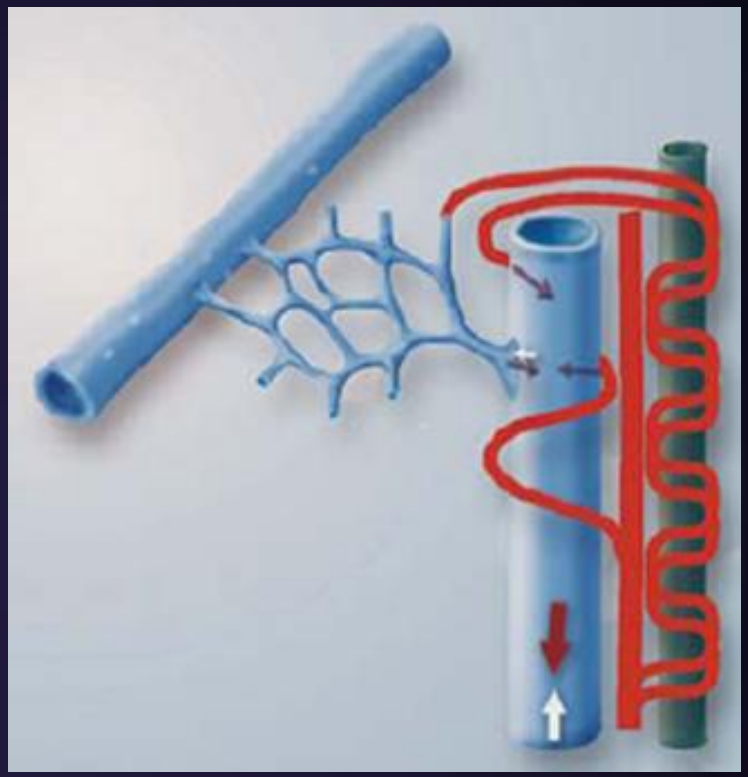


Tumoral AP shunt via a transtumoral route. A direct communication between the feeding arterial vessels of the tumor and the draining portal venules and/or sinusoids is established, resulting in increased arterial flow around the tumor. *T, tumor.*

Drawings of the hepatic microvasculature and arterioportal (AP) communications

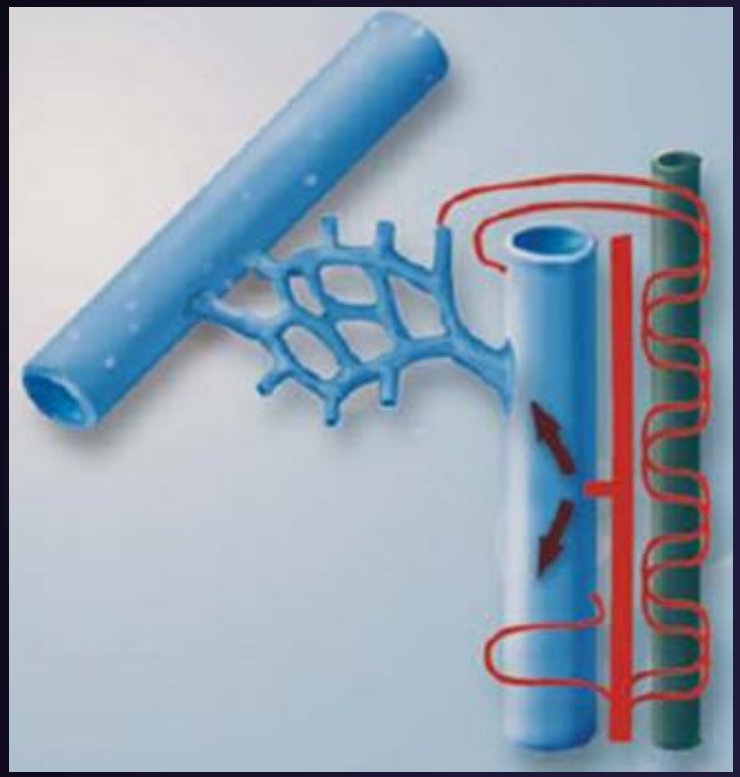


Tumoral AP shunt via a transvasal (vasa vasorum) route.

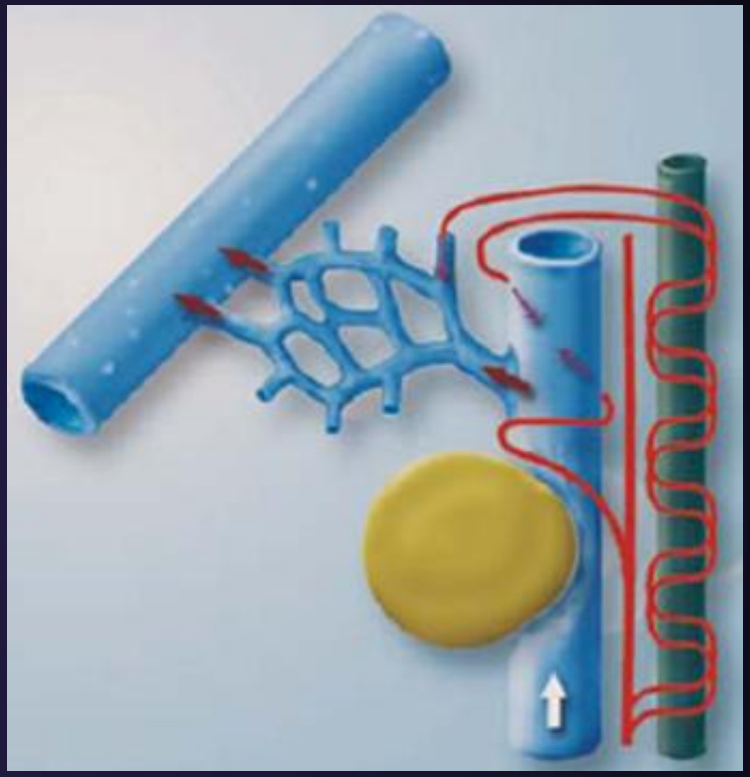


Cirrhosis with stretching and deformation of hepatic sinusoids causing a compensatory increase of the arterial flow. Note inversion of the direction of the portal vein flow.

Drawings of the hepatic microvasculature and arterioportal (AP) communications

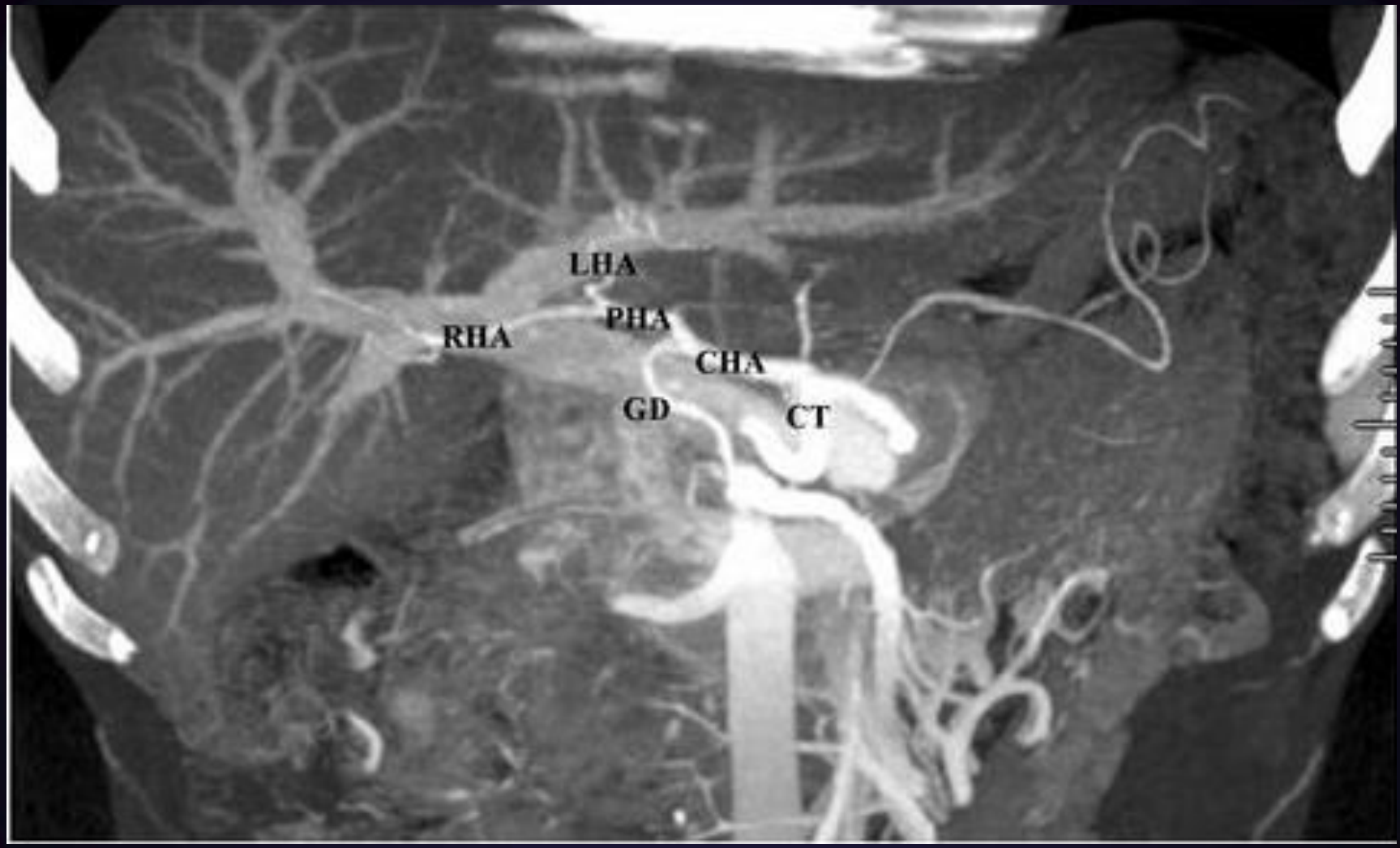


Iatrogenic AP shunt caused by a fistulous tract after liver biopsy.



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

VASCULAR ANATOMY – *hepatic arteries*

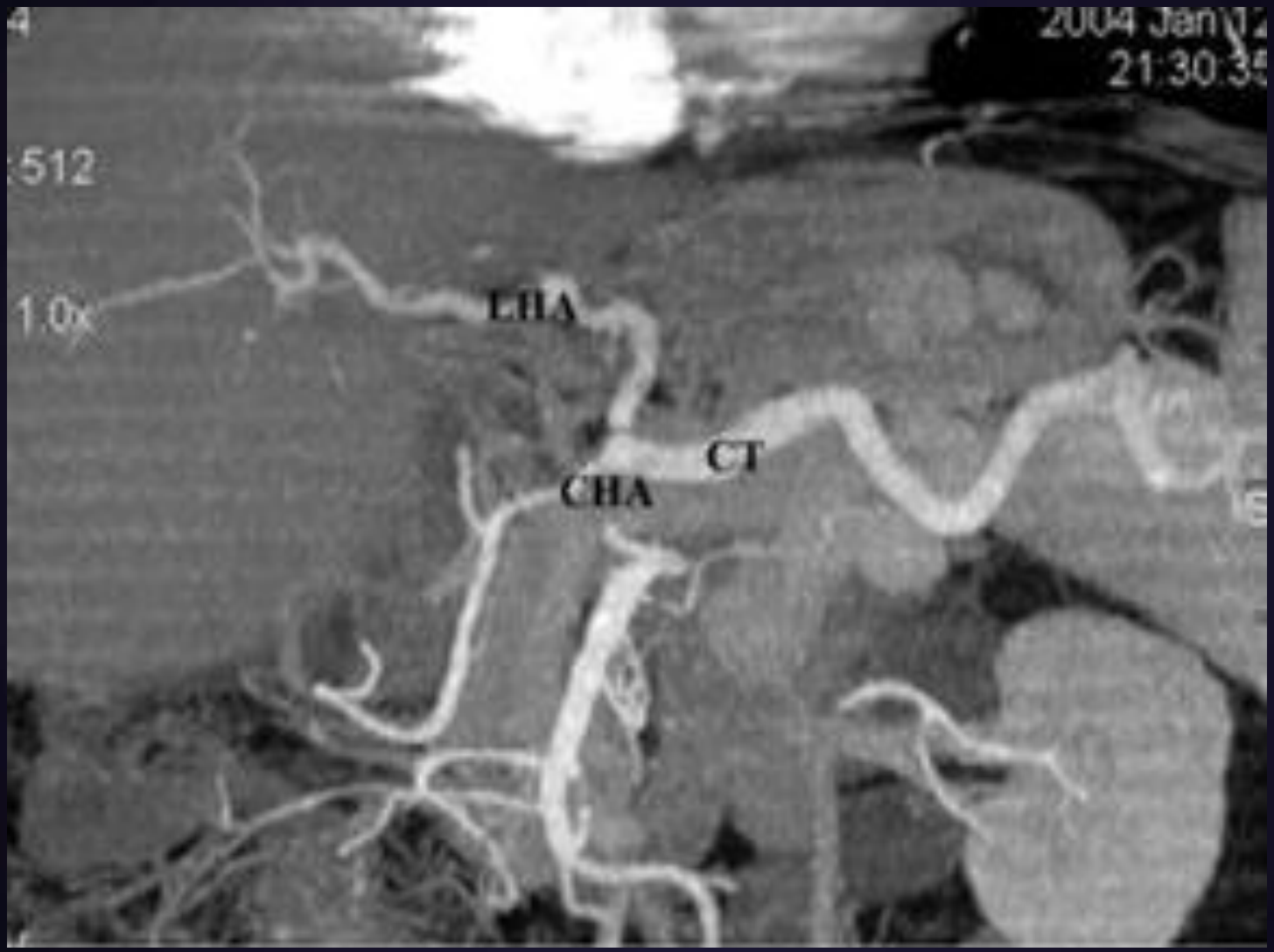


Normal hepatic arteries.

VASCULAR ANATOMY – *hepatic arteries*

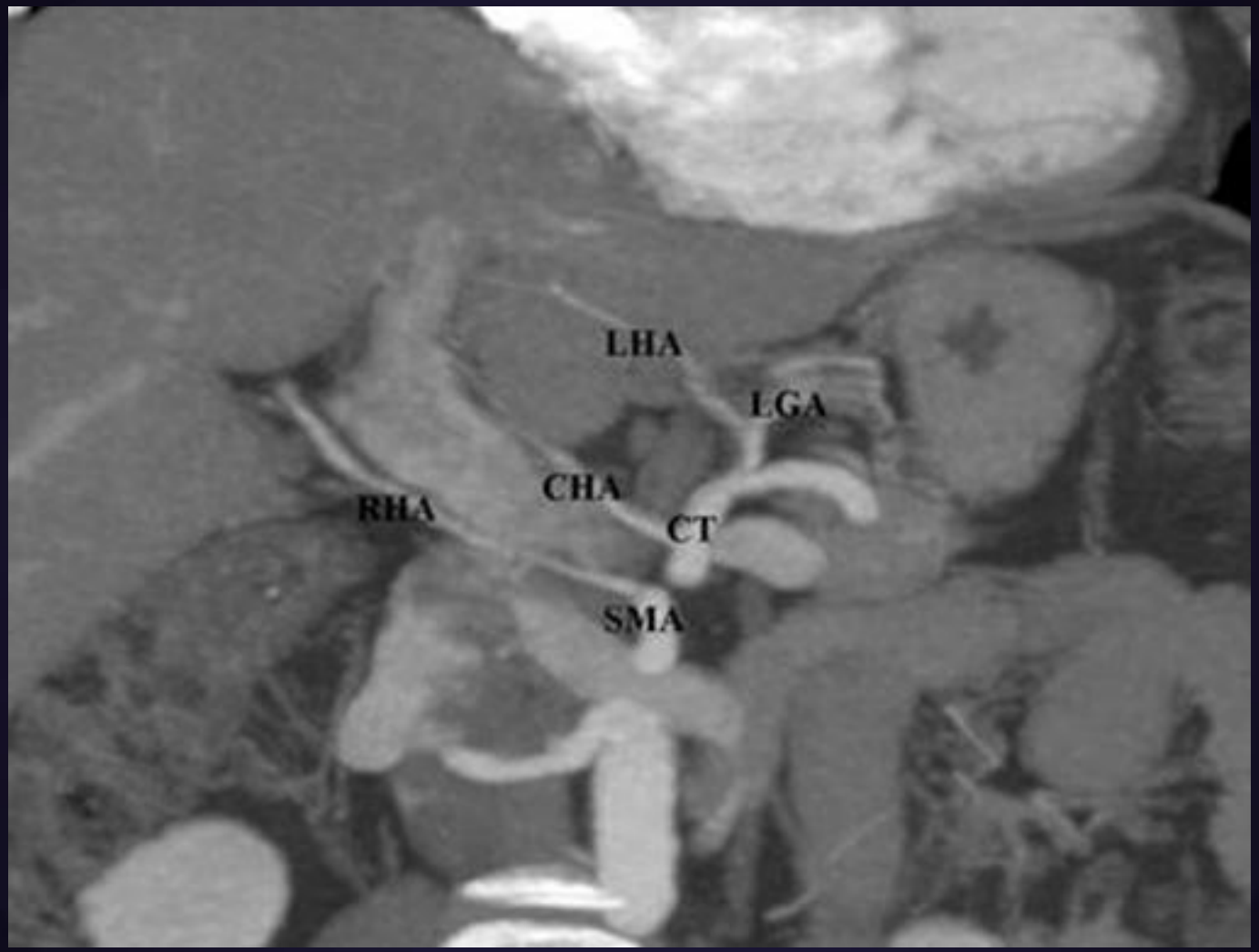
- Variations in the hepatic arterial anatomy occur in approximately 42% of patients.
- The commonest variant is a replaced RHA arising from the superior mesenteric artery (SMA), which is seen in 11% of cases.
- A replaced LHA arising from the left gastric artery can also exist.
- The segment IV artery,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

VASCULAR ANATOMY – *hepatic arteries*



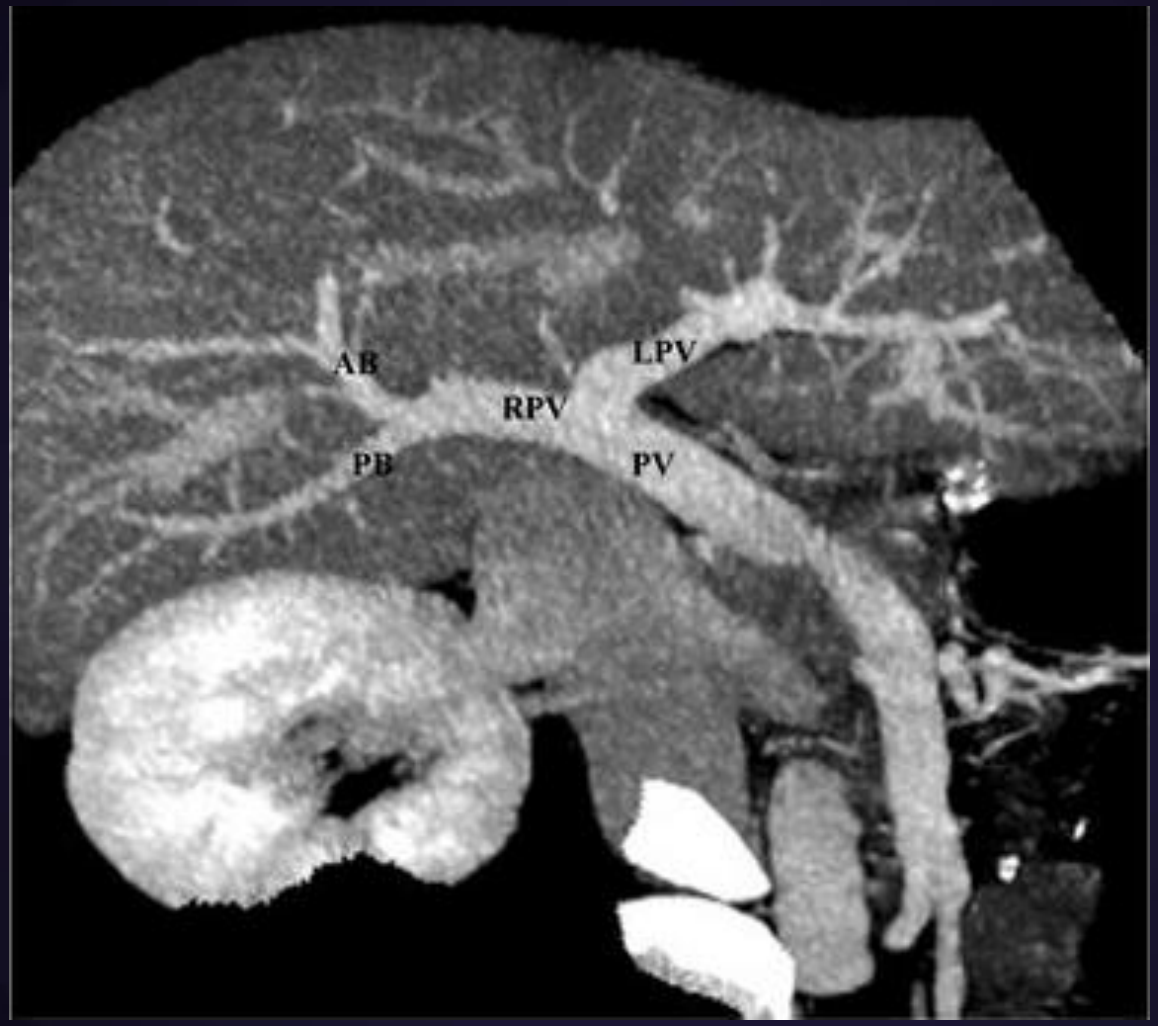
Aberrant origin of left hepatic artery (LHA).

VASCULAR ANATOMY – *hepatic arteries*



Complex anatomic variant.

VASCULAR ANATOMY – *Portal vein*

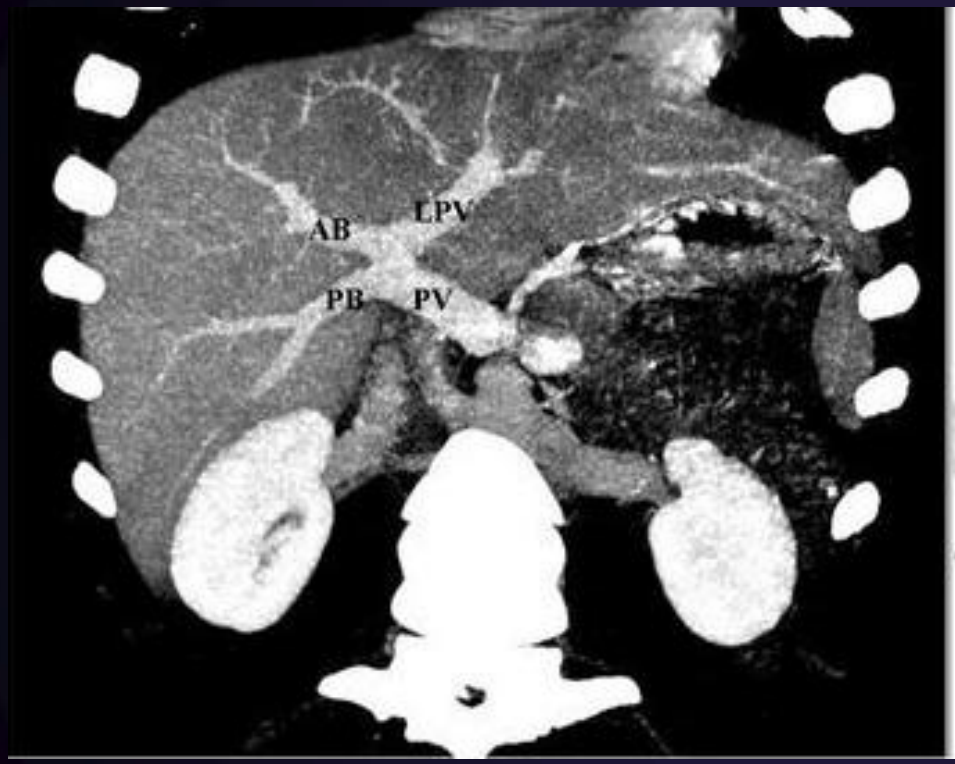


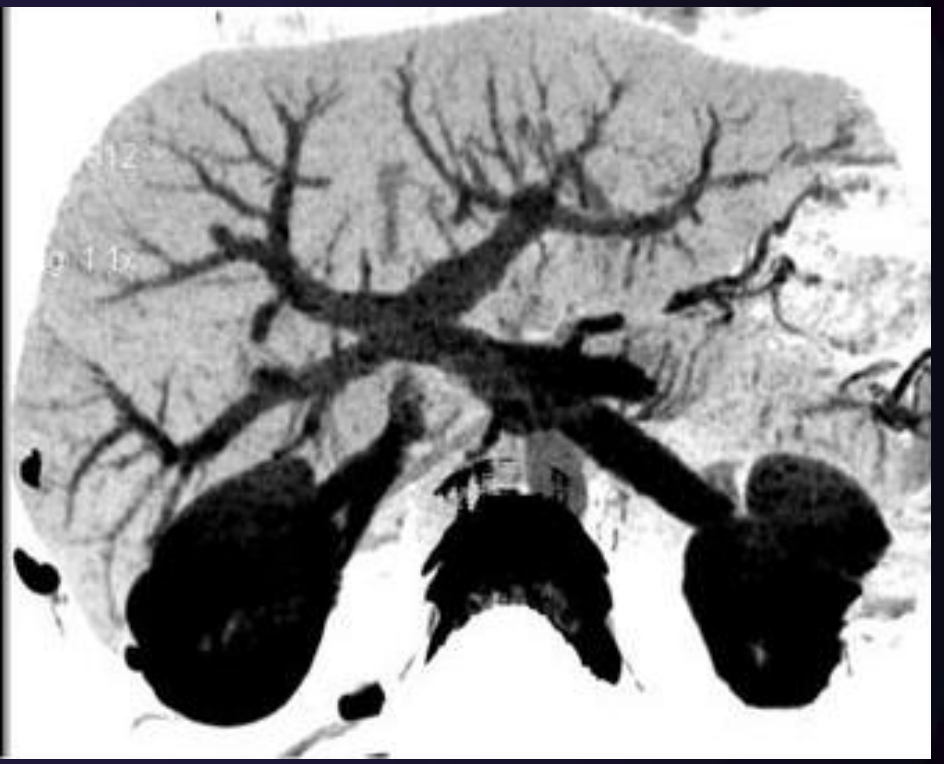
Normal portal anatomy.

VASCULAR ANATOMY – *Portal vein*

- 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.

VASCULAR ANATOMY – *Portal vein*





Portal vein trifurcation.

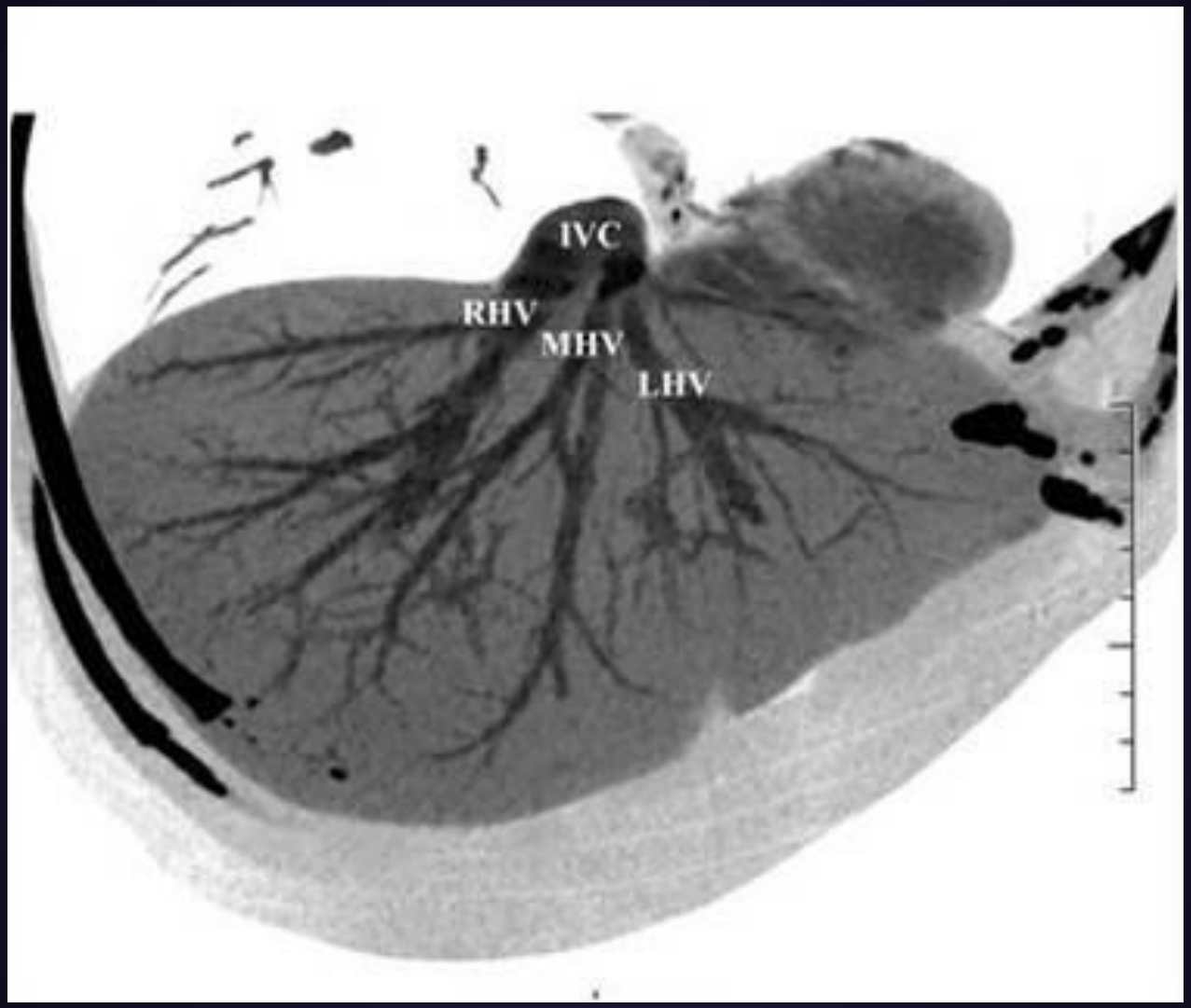
VASCULAR ANATOMY – *Hepatic veins*

- The hepatic venous anatomy is variable, and the most common pattern consists of three main hepatic veins.
- 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.

VASCULAR ANATOMY – *Hepatic veins*

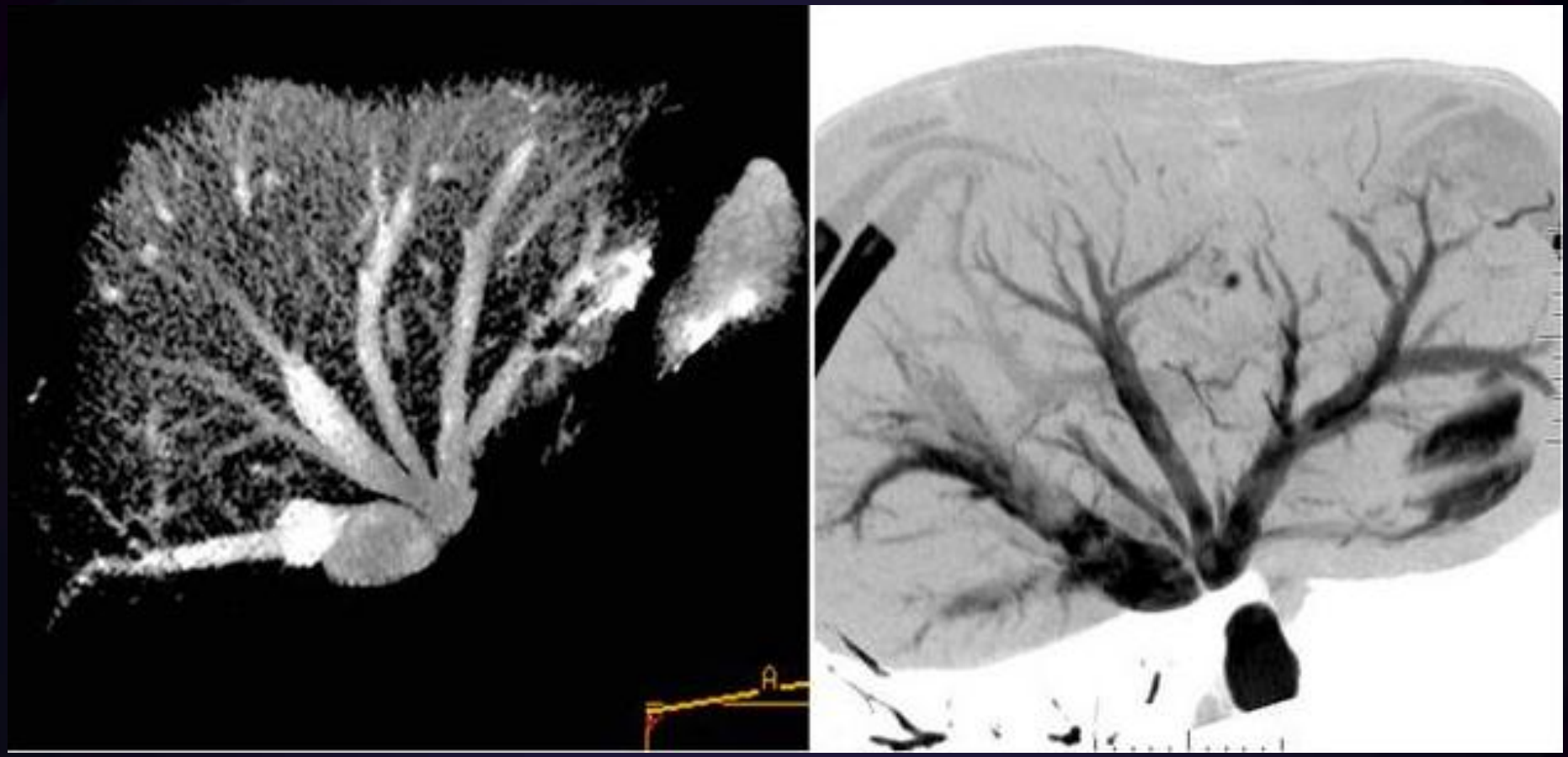
- Relevant variants of the hepatic venous system are short accessory RHVs that drain the posterior segments (VI and VII) independently into the IVC at a variable distance from RHV, happening more usually in cases of non-dominant RHV.
- The draining veins of segment IV can be multiple in number and very 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.
- These accessory veins are important for a surgical standpoint when their diameter is superior to 5mm.

VASCULAR ANATOMY – *Hepatic veins*



Normal hepatic veins.

VASCULAR ANATOMY – *Hepatic veins*



Acessory hepatic veins.

VASCULARES DISORDERS OF THE LIVER

1) Vascular pseudolesions and pitfalls
2) Hereditary hemorrhagic telangiectasia
3) Budd-Chiari syndrome
4) Passive hepatic congestion
5) Hepatic infarction
6) Peliosis hepatis
7) Portal vein thrombosis
8) Portal hypertension

1. VASCULAR PSEUDOLESIONS AND PITFALLS

1.1. Non-opacified hepatic vessels

1.2. Portal venous inflow obstruction/THAD

1.3. Intrahepatic vascular shunts

1.4. “Non-portal" venous supply to the liver

1.5. Steal phenomena

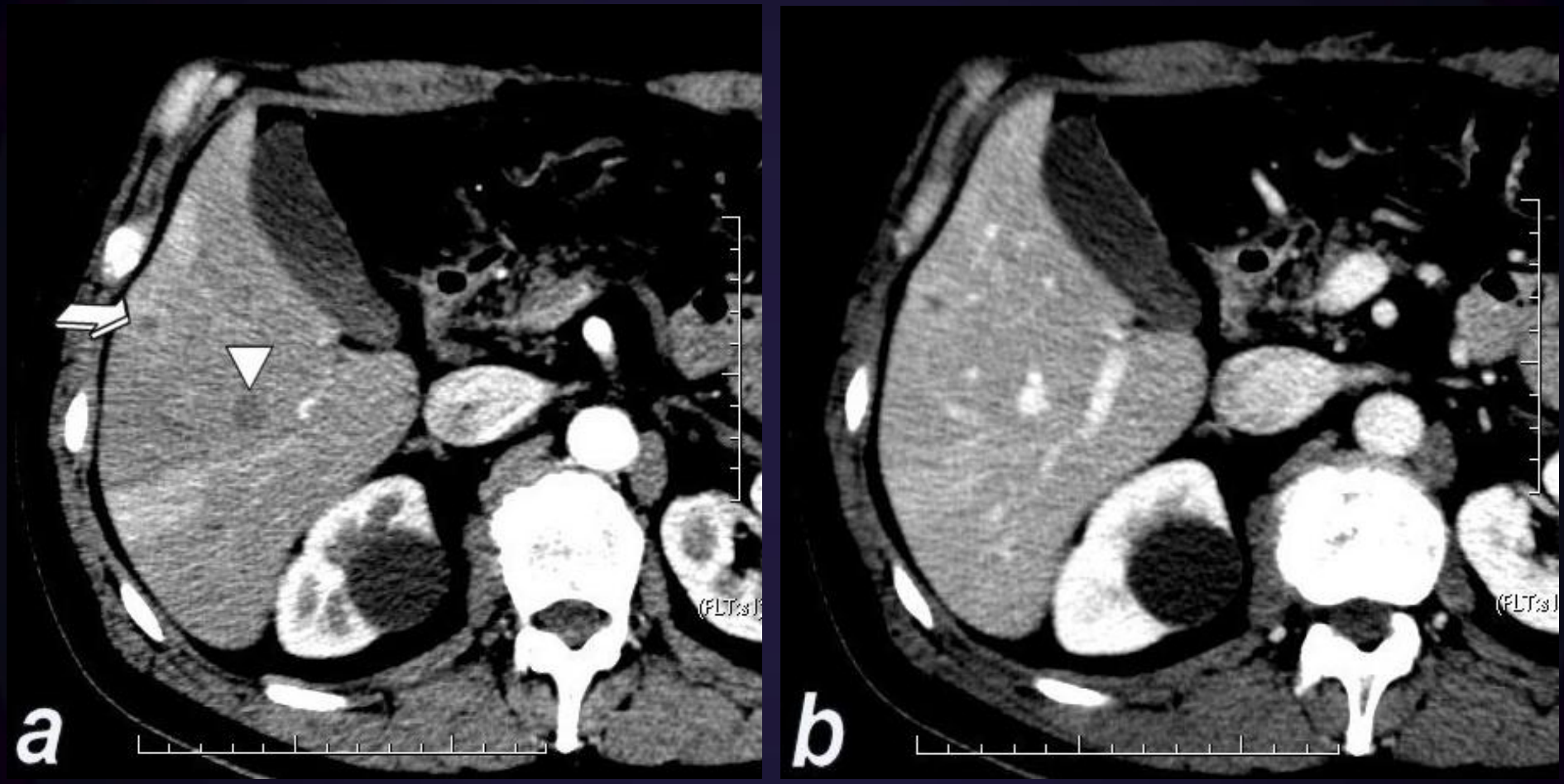
1.6. Post-surgical parasitic arterial flow

1.7. Pericaval fat collection

1.1. NON-OPACIFIED HEPATIC VESSELS

- Due to the high temporal resolution achieved nowadays by dynamic studies performed with multislice CT, 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.

1.1. NON-OPACIFIED HEPATIC VESSELS



Dynamic MDCT obtained in a patient with rectal cancer during upper abdominal work-up depicting arterial (a) and portal (b) phases of liver enhancement.

1.2. 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.

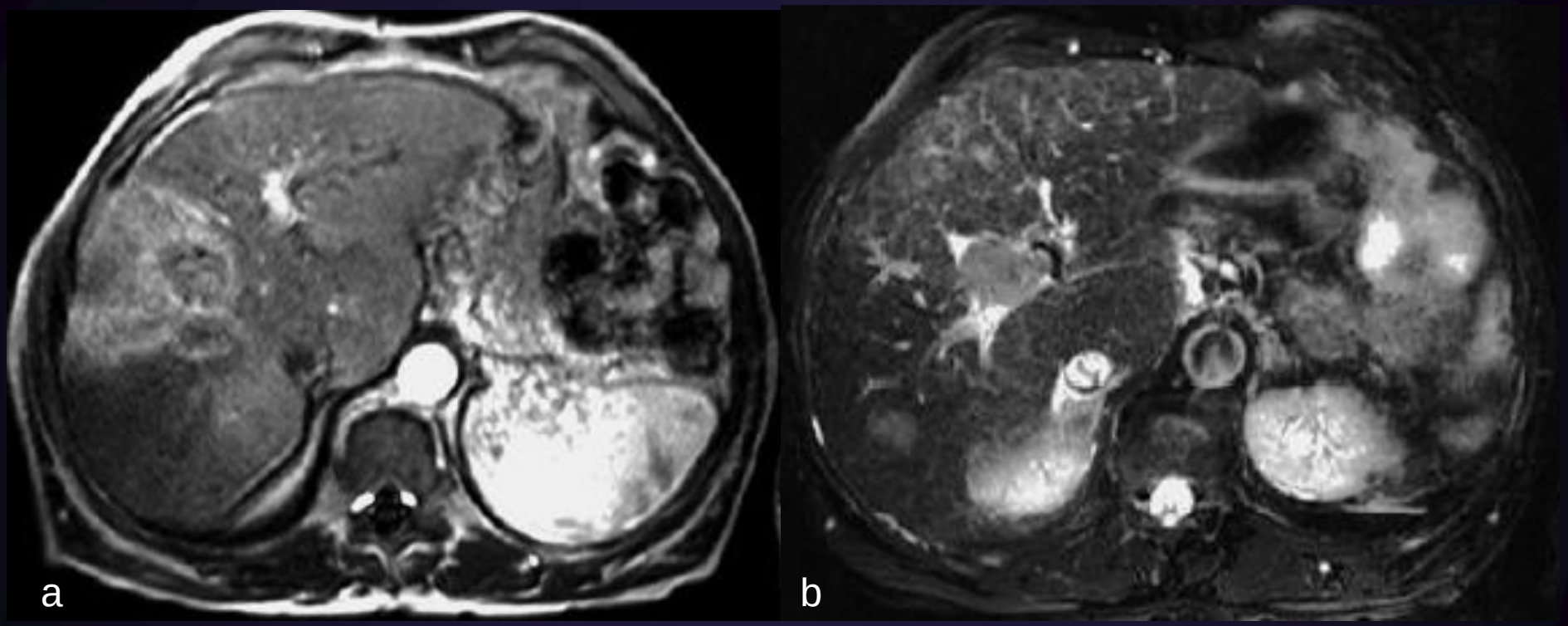
1.2. PORTAL VENOUS INFLOW OBSTRUCTION and THAD

- Areas of hyperattenuation 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.
- 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.

1.2. PORTAL VENOUS INFLOW OBSTRUCTION and THAD

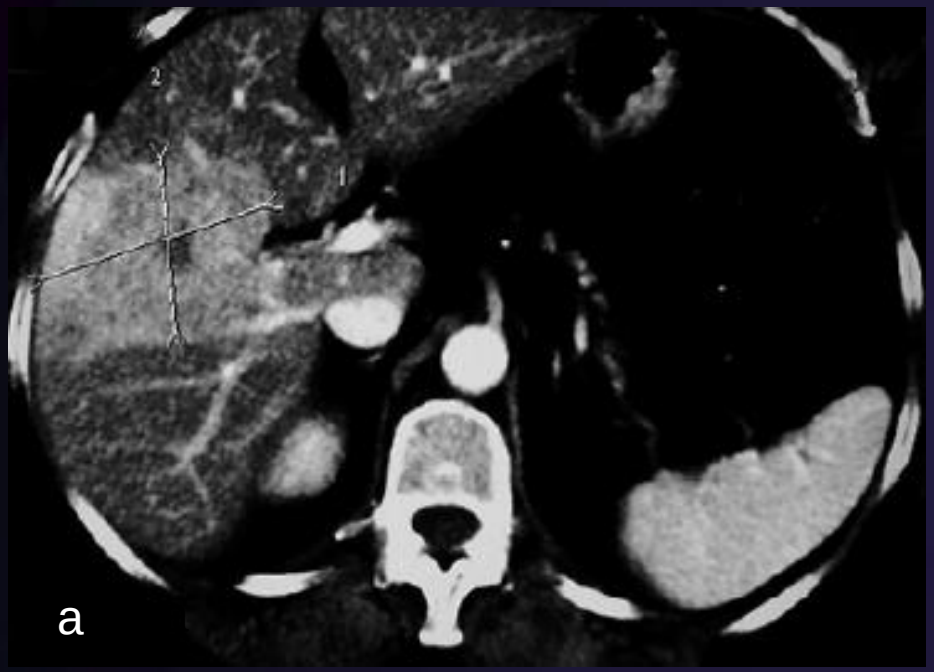
- Cavernomatous transformation of the portal vein may also cause an impairment of the portal flow to the liver, especially to most peripheral areas, since the hilar collateral vessels are insufficient to adequately supply them.
- In some instances, THAD's nature is not apparent and a real intra-hepatic vascular mechanism remains to be proved since an associated focal liver lesion may not be seen.

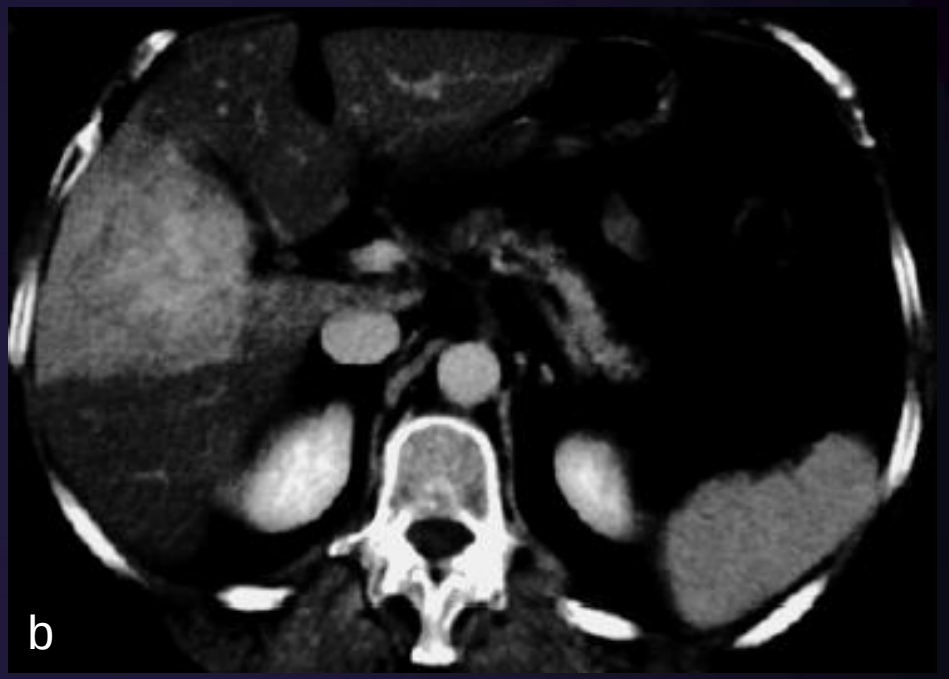
1.2. PORTAL VENOUS INFLOW OBSTRUCTION and THAD



MR imaging of a patient with hepatocellular carcinoma and tumor thrombus within the right portal vein.

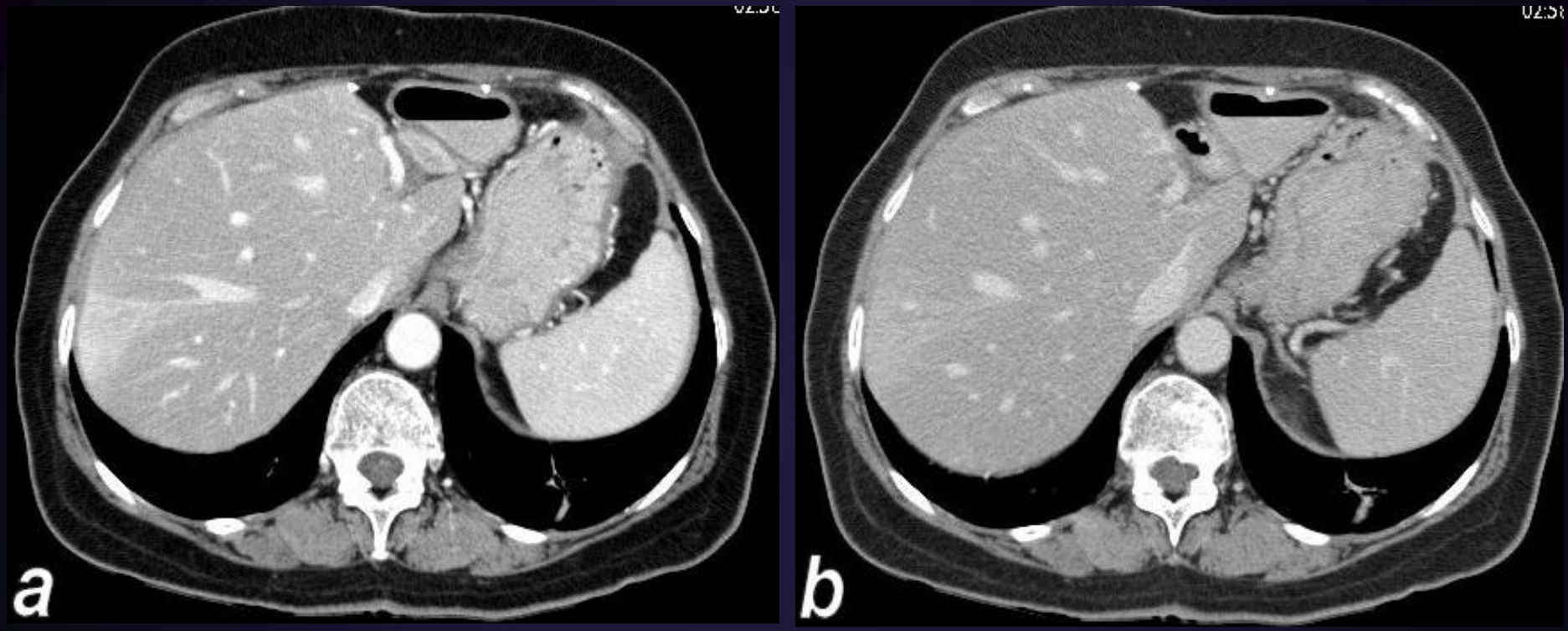
1.2. PORTAL VENOUS INFLOW OBSTRUCTION and THAD





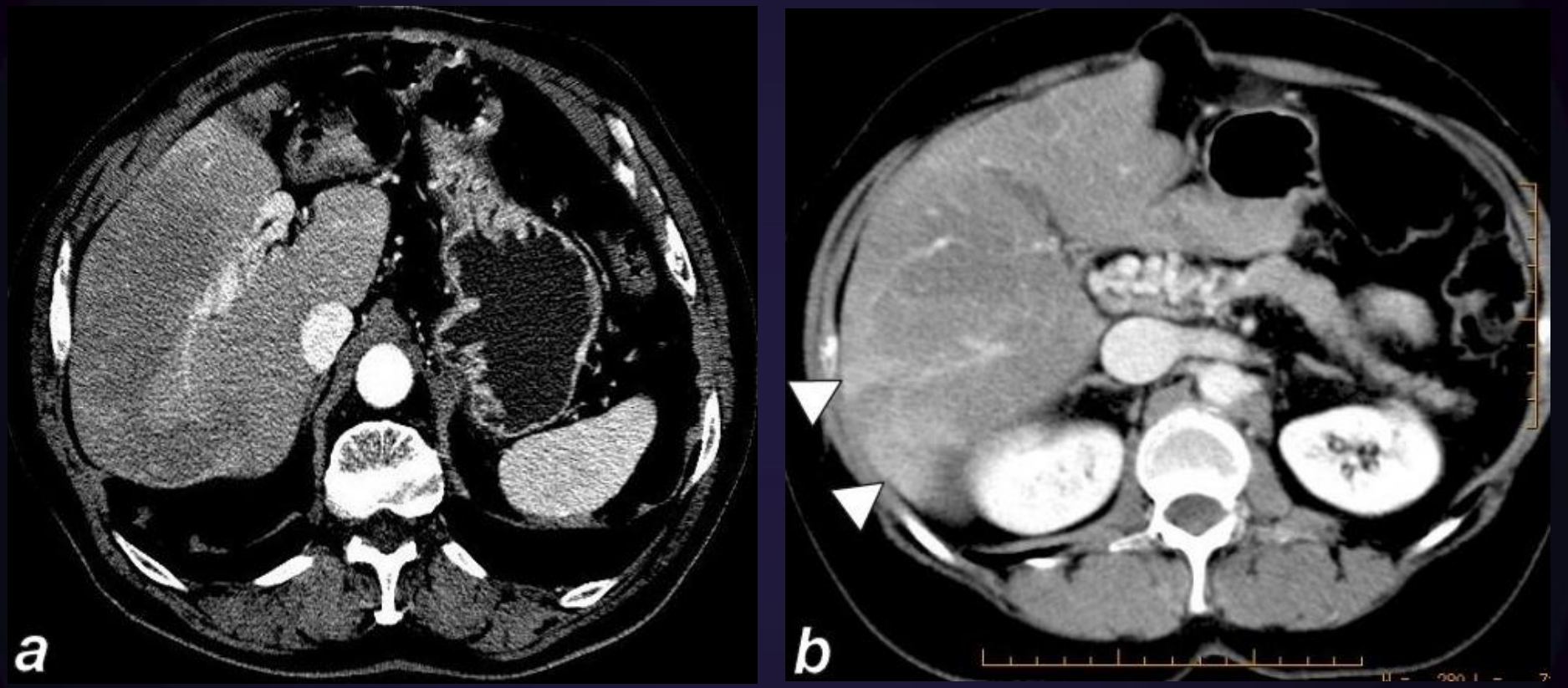
Dynamic helical CT in a patient with hepatic metastasis from lung cancer located at the central liver.

1.2. PORTAL VENOUS INFLOW OBSTRUCTION and THAD



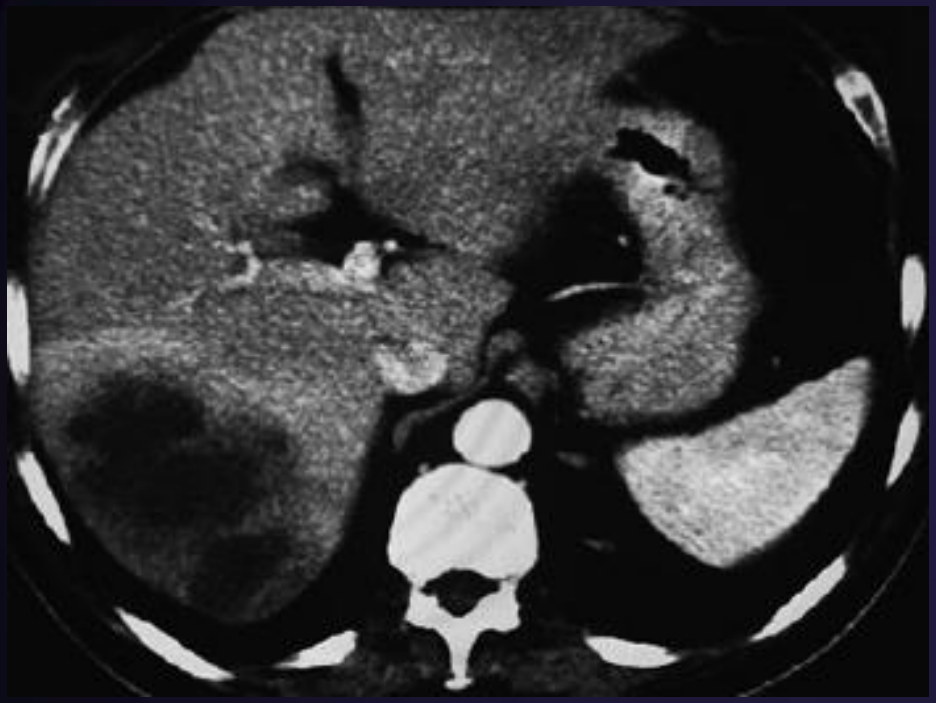
Helical CT depicting a peripheral THAD of unknown origin in a patient with previous history of left hepatectomy for metastasis.

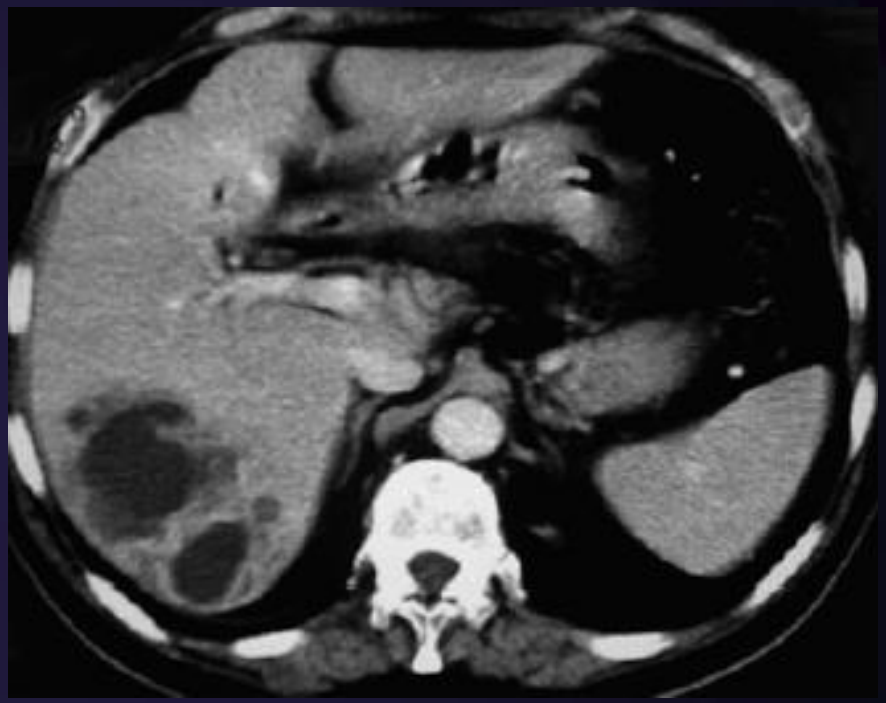
1.2. PORTAL VENOUS INFLOW OBSTRUCTION and THAD



Dynamic multislice helical CT of two cases of portal cavernoma.

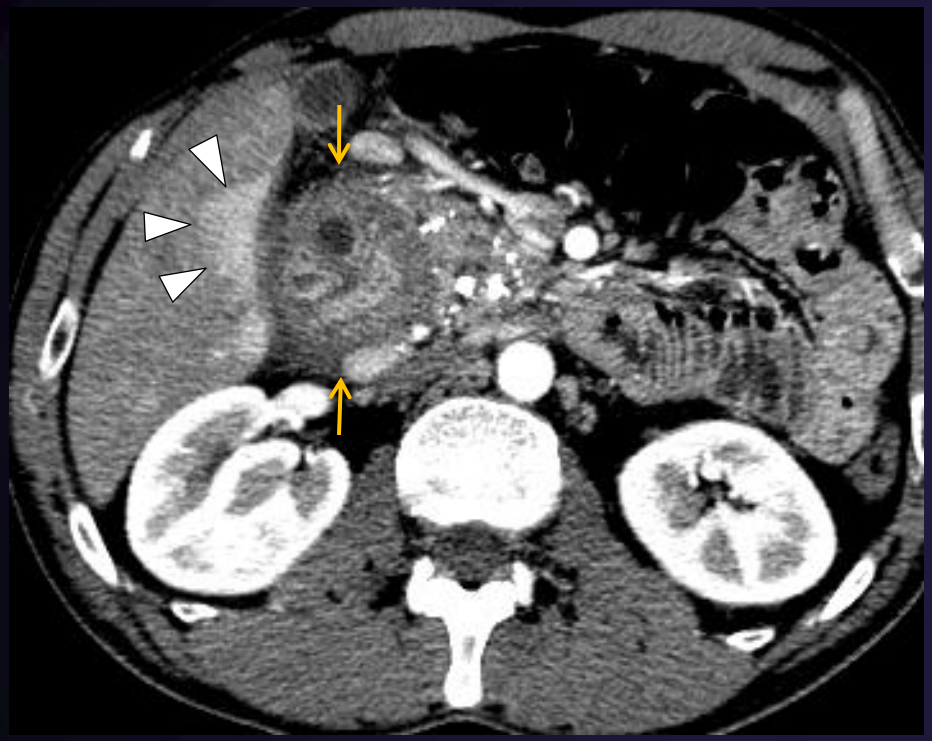
1.2. PORTAL VENOUS INFLOW OBSTRUCTION and THAD

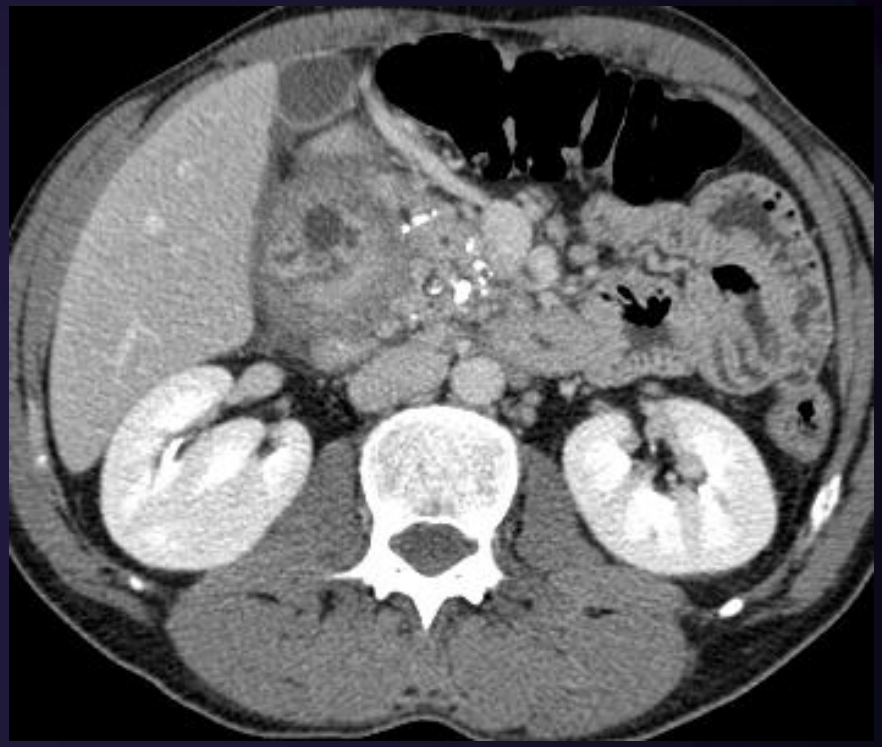




Dynamic CT of liver abscess.

1.2. PORTAL VENOUS INFLOW OBSTRUCTION and THAD





Dynamic CT of groove pancreatitis (arrows).

1.3. INTRAHEPATIC VASCULAR SHUNTS

Intra-hepatic shunts can be divided

Depending on the underlying cause

- a) Tumorous
- b) Non-tumorous

According to the vascular connection established

- a) Arterioportal
- b) Arteriosystemic
- c) Portosystemic

1.3. INTRAHEPATIC VASCULAR SHUNTS

- Arterioportal shunting (AP shunting) is generally observed in the context of hepatocellular carcinoma (HCC), although it may be iatrogenic, related to previous liver biopsy.
- 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.
- 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.

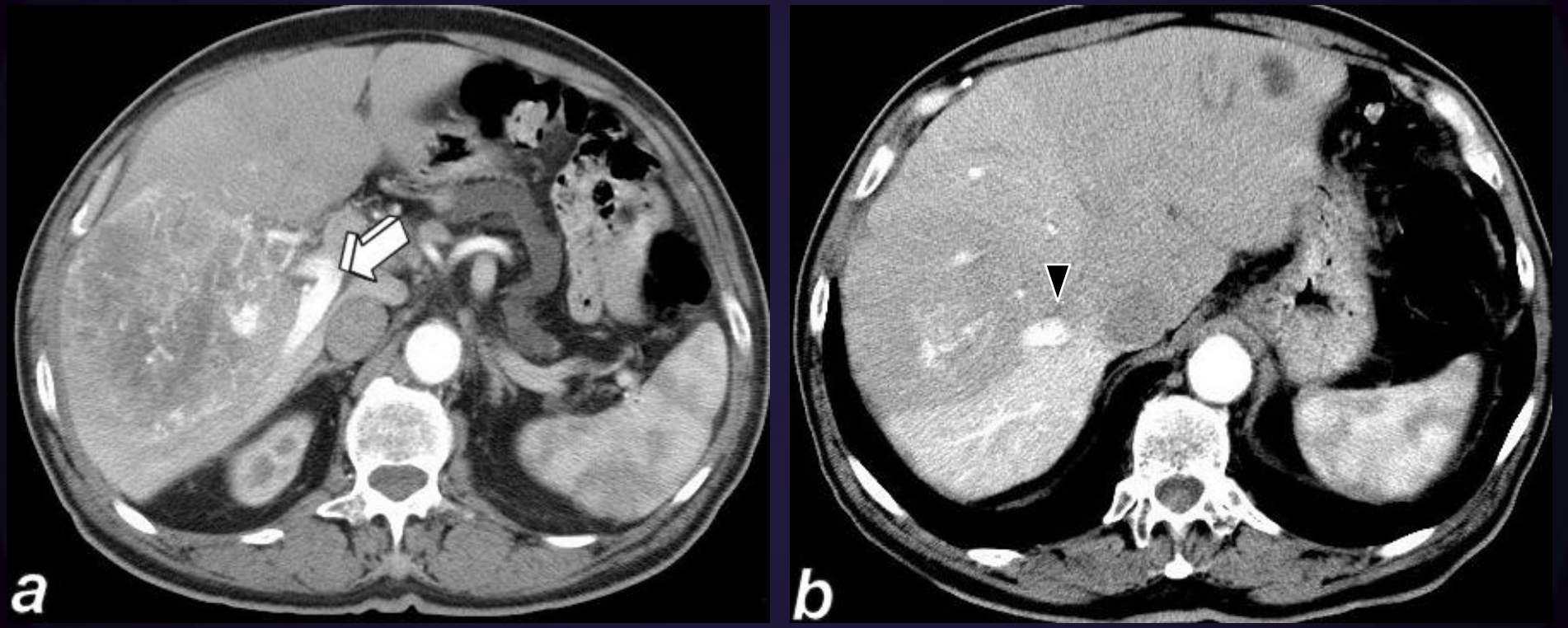
1.3. INTRAHEPATIC VASCULAR SHUNTS

- AP shunts may also be seen in liver cirrhosis due to the parenchymal damage which tends to modify the hepatic flow dynamics, and may appear as small arterial-enhancing nodules, which can cause potential confusion with HCC.
- Some features that can be helpful in differentiation are:
  - HCC have early enhancement with rapid central washout. Pseudolesions usually enhance later and the enhancement persists longer.
  - Coronal enhancement surrounding the lesion is not seen in pseudolesions.
  - A rounded configuration is more likely in HCC.
  - Bulging of the capsule is only seen in HCC.
  - Pseudolesions are isointense to liver on T1 WI and T2 WI.

1.3. INTRAHEPATIC VASCULAR SHUNTS

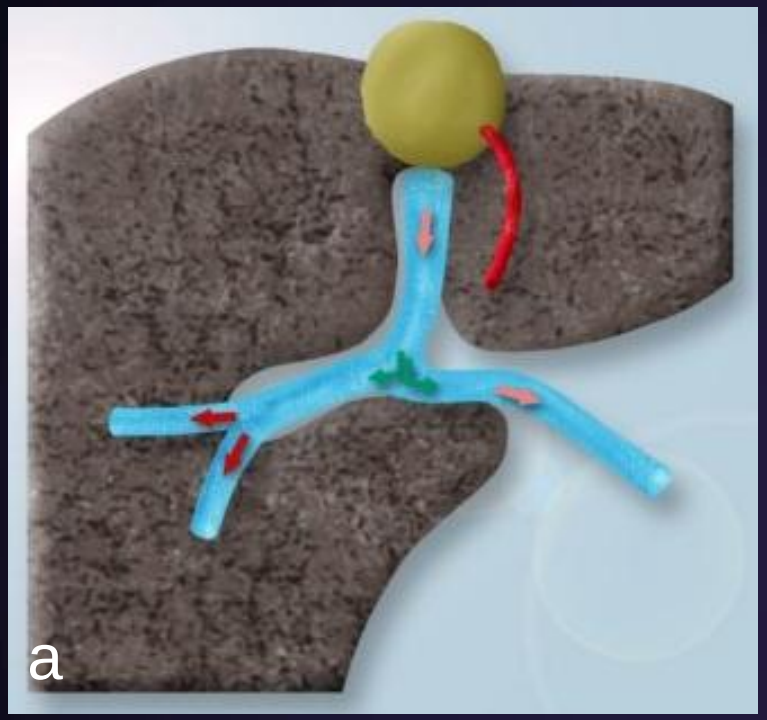
- Intra-hepatic portosystemic venous shunts (IPSVS) can also mimic a hypervascular liver lesion.
- They can be sub-divided in internal and external depending whether the portal vein communicates respectively with the hepatic vein or with a systemic vein outside the liver.
- IPSVS are seldom seen outside 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.

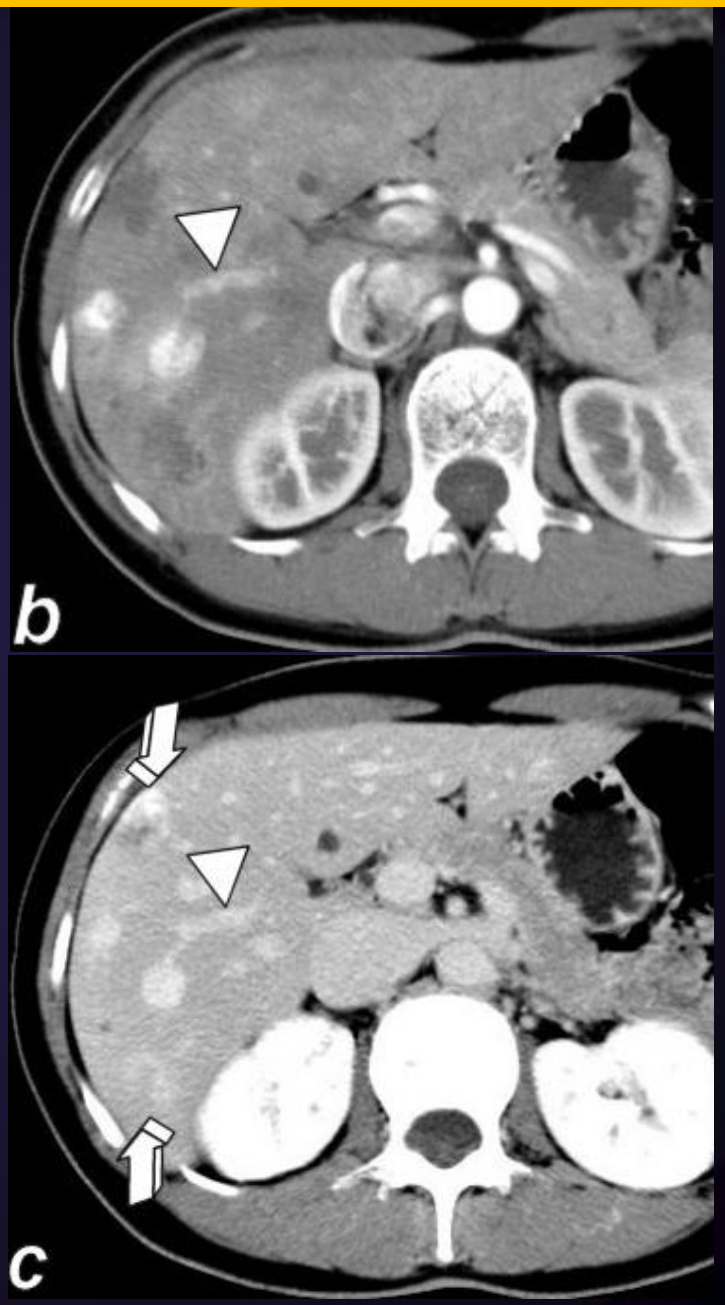
1.3. INTRAHEPATIC VASCULAR SHUNTS



Dynamic helical MDCT study of two hypervascular hepatocellular carcinoma during the arterial phase of liver enhancement.

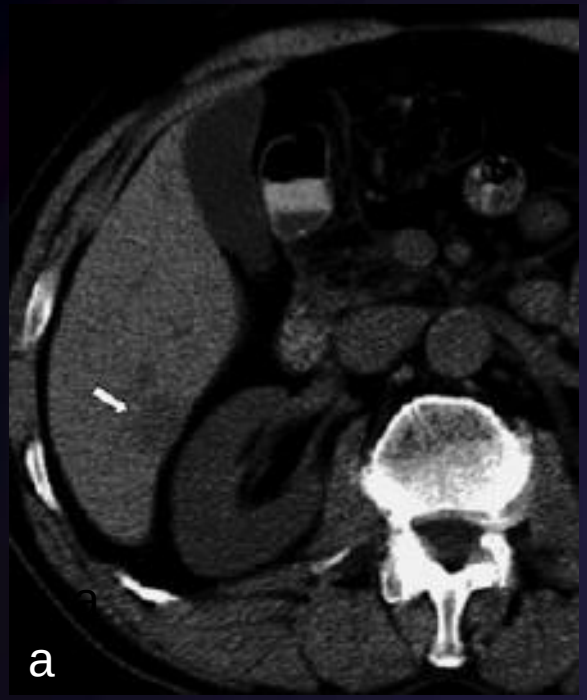
1.3. INTRAHEPATIC VASCULAR SHUNTS

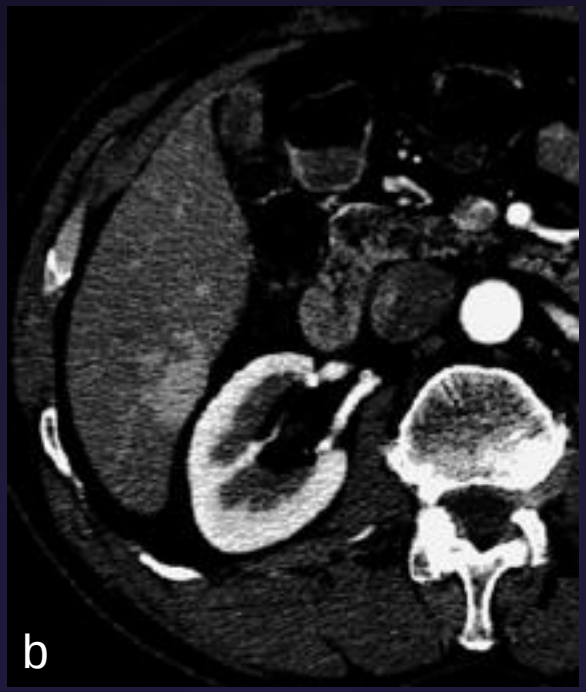


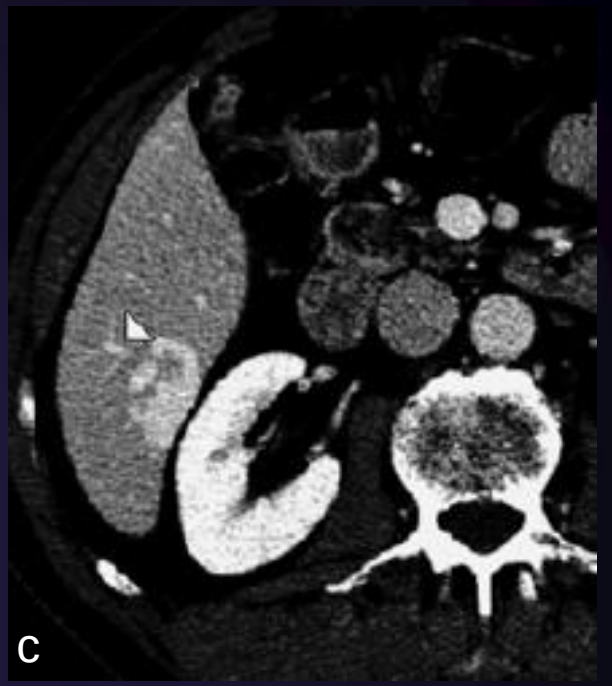


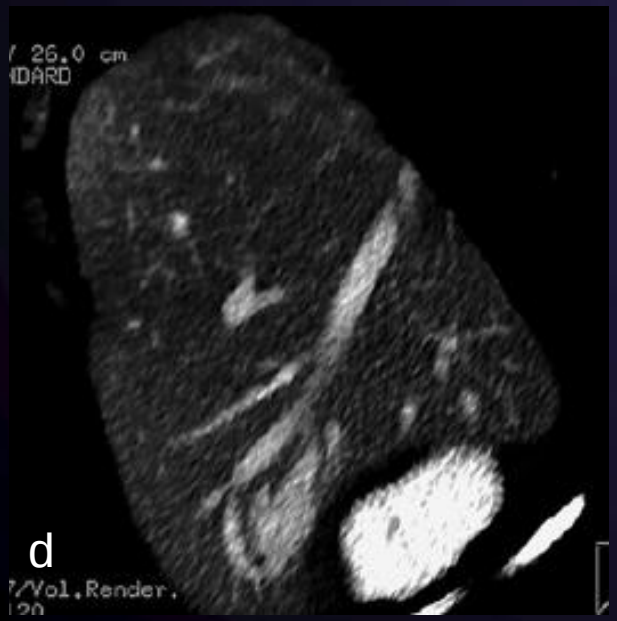
Schematic drawing (a) and dynamic CT (b and c) of a flash-filling hemangioma causing localized inversion of the portal vein flow.

1.3. INTRAHEPATIC VASCULAR SHUNTS



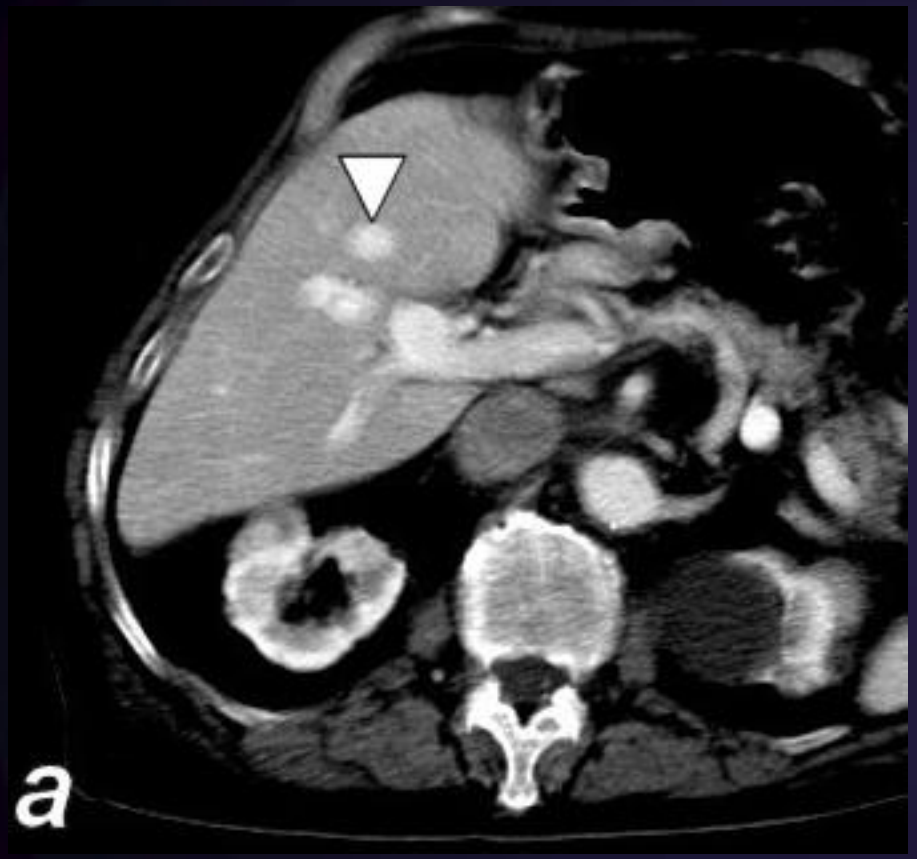


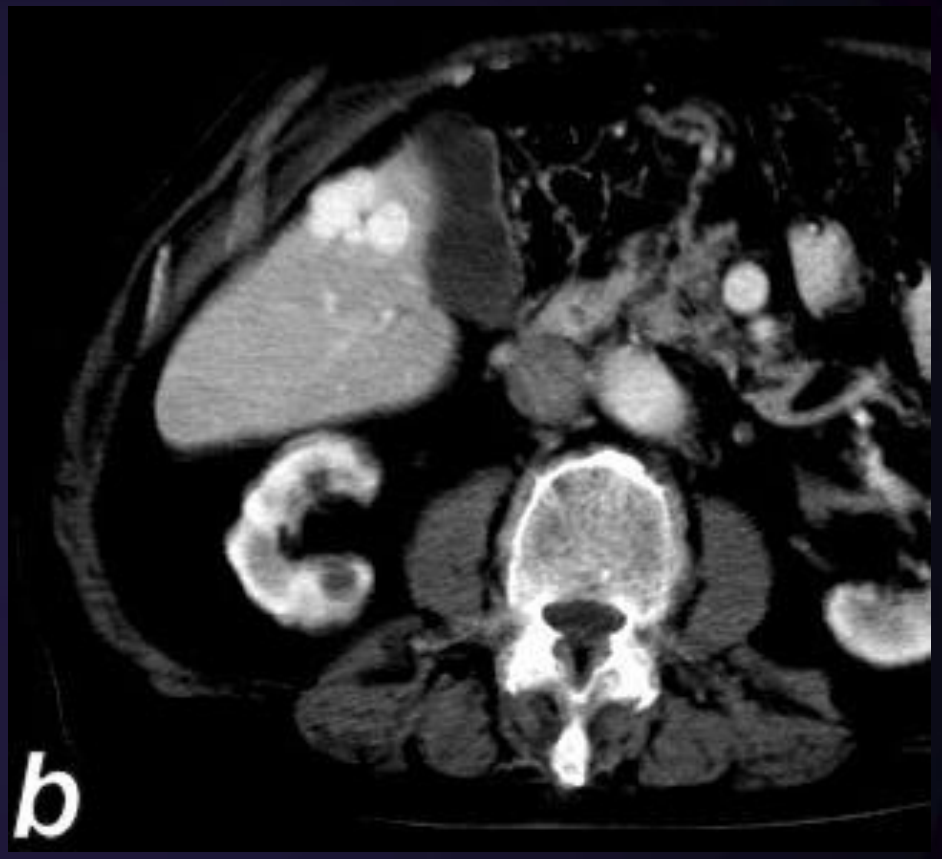




Multislice helical CT of intrahepatic portosystemic venous shunts of the internal subtypes.

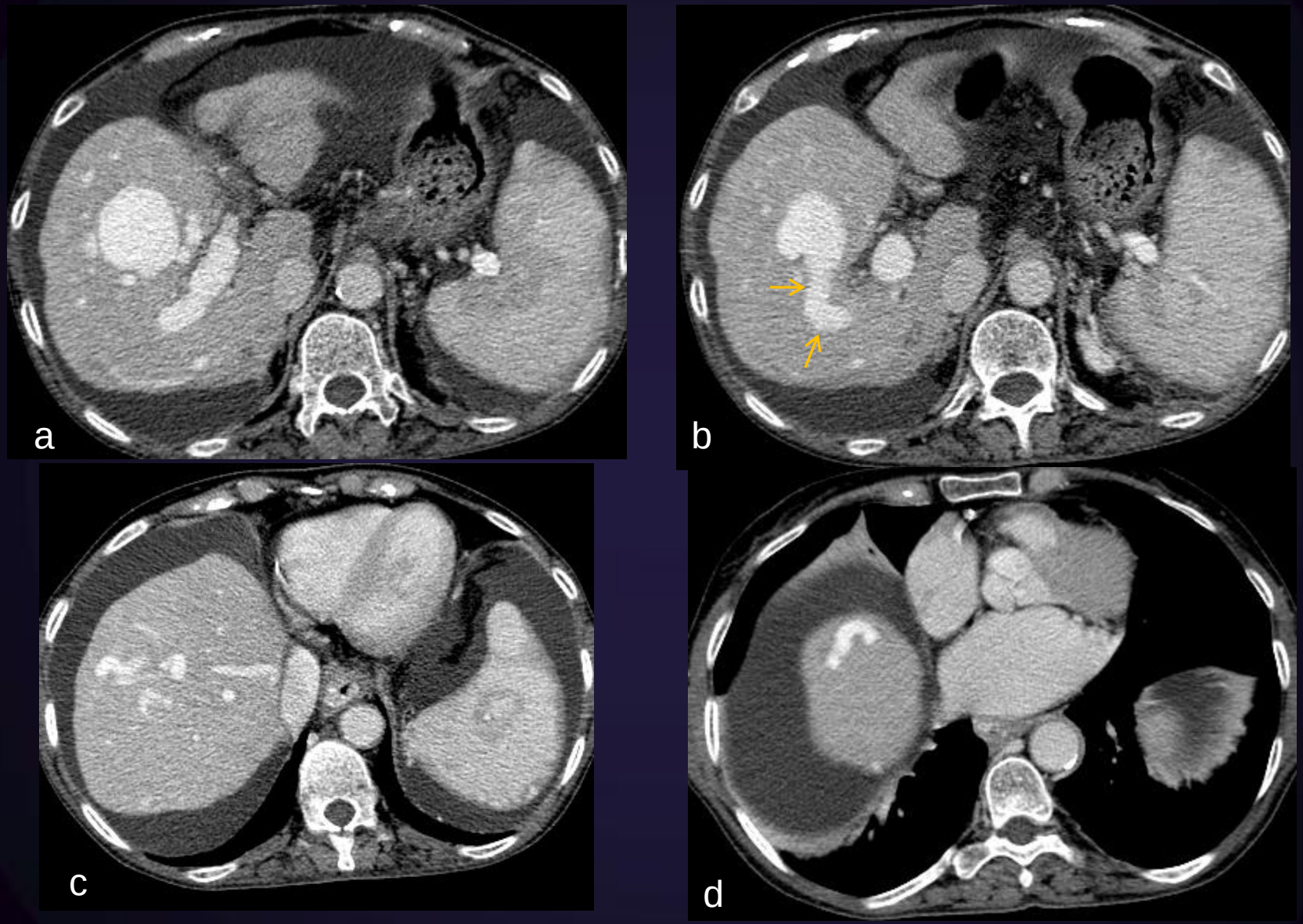
1.3. INTRAHEPATIC VASCULAR SHUNTS





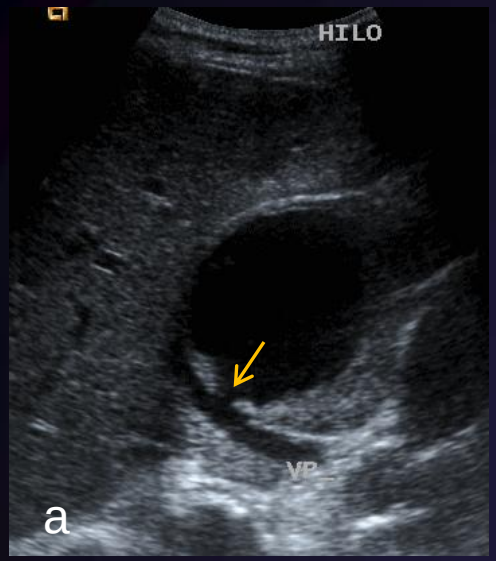
Multislice helical CT of intrahepatic portosystemic venous shunts of the external subtypes.

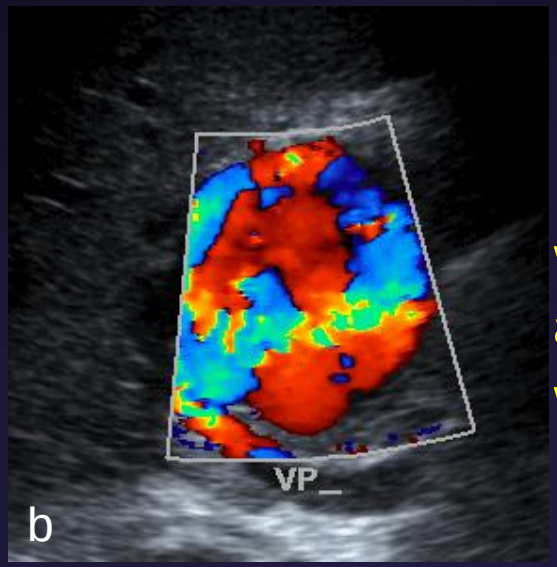
1.3. INTRAHEPATIC VASCULAR SHUNTS



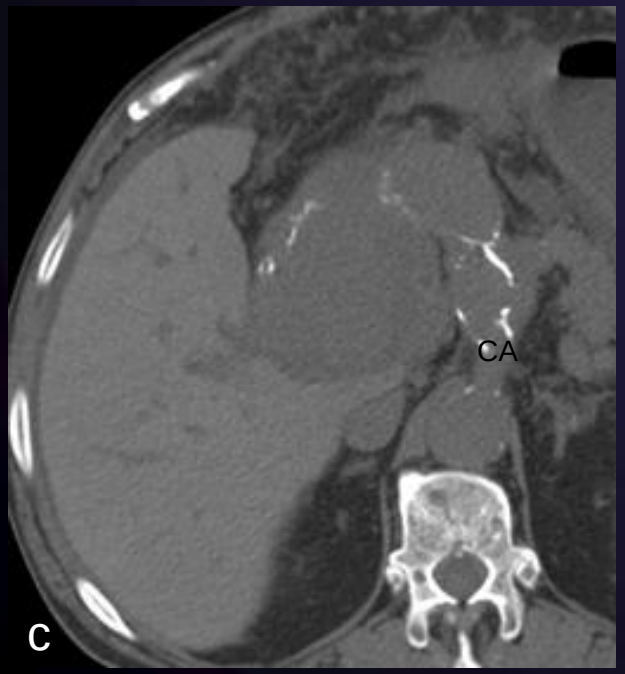
Multislice helical CT of another intrahepatic portosystemic venous shunts of the external subtypes.

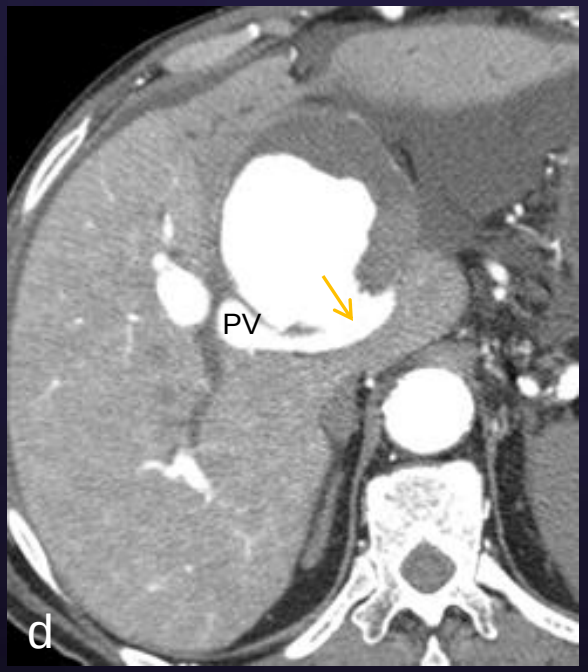
1.3. INTRAHEPATIC VASCULAR SHUNTS

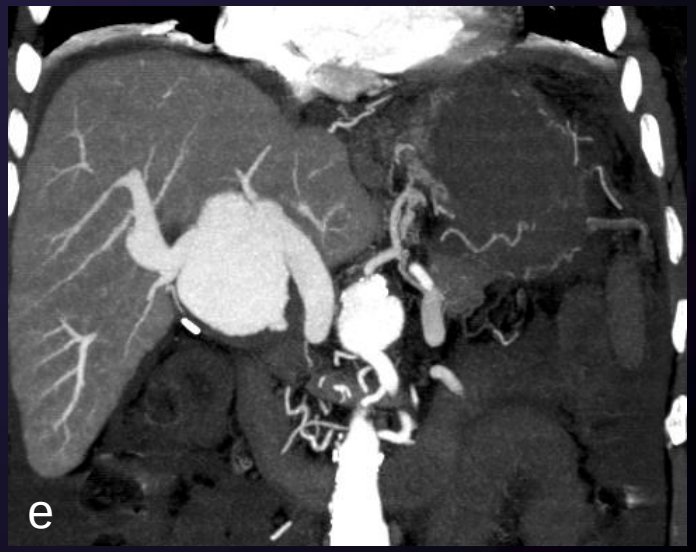




Hepatic artery pseudoaneurysm with a huge intrahepatic arterioportal shunt in a patient with previous pancreatitis.







1.4. “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 they show early enhancement due to earlier venous return of less diluted contrast agent when compared with the portal blood.
- 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.

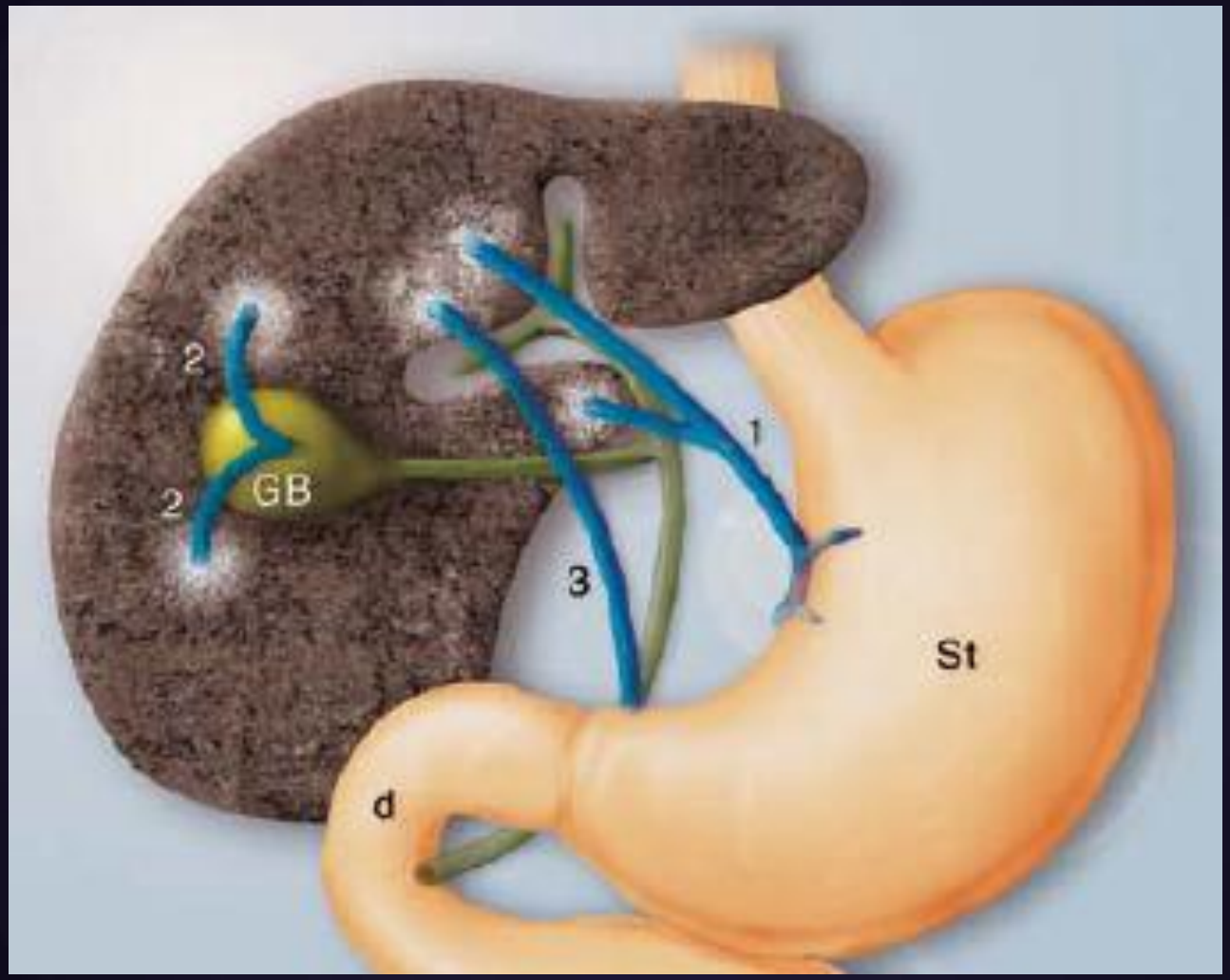
1.4. “NON-PORTAL” VENOUS SUPPLY TO THE LIVER

- 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.

1.4. “NON-PORTAL” VENOUS SUPPLY TO THE LIVER

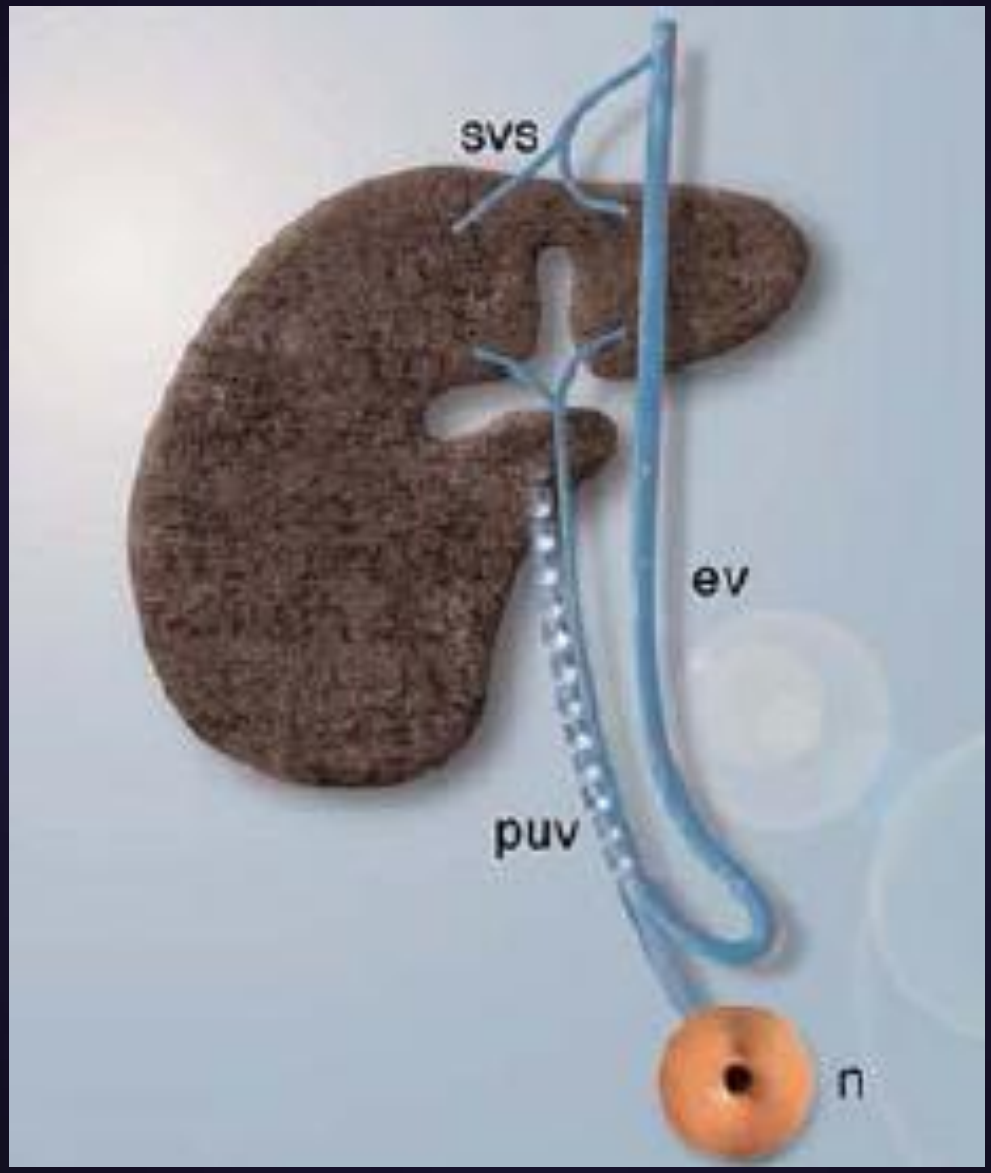
- In cases of SVC obstruction the recruitment of the collateral circulation can cause a dense focal parenchymal stain in the early phases of enhancement around the round ligament, the left portal vein or even at subcapsular areas, corresponding to an early arrival of minimally diluted contrast agent to those areas.
- The parenchymal staining in these cases can be so intense that it can mimic a true hypervascular neoplasm.

1.4. “NON-PORTAL” VENOUS SUPPLY TO THE LIVER

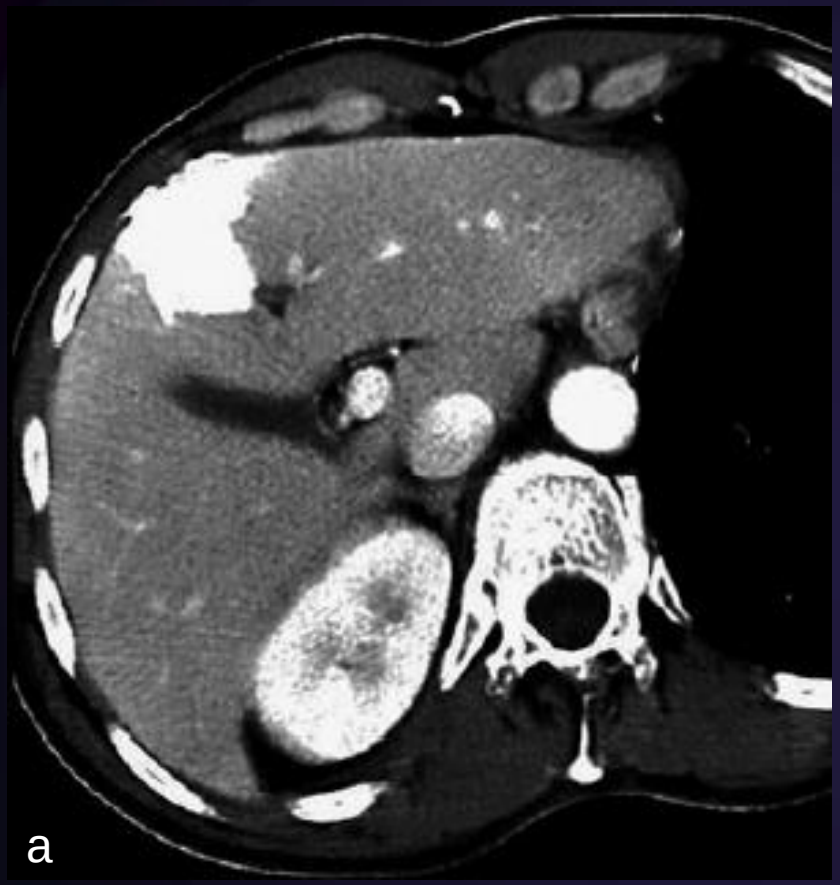


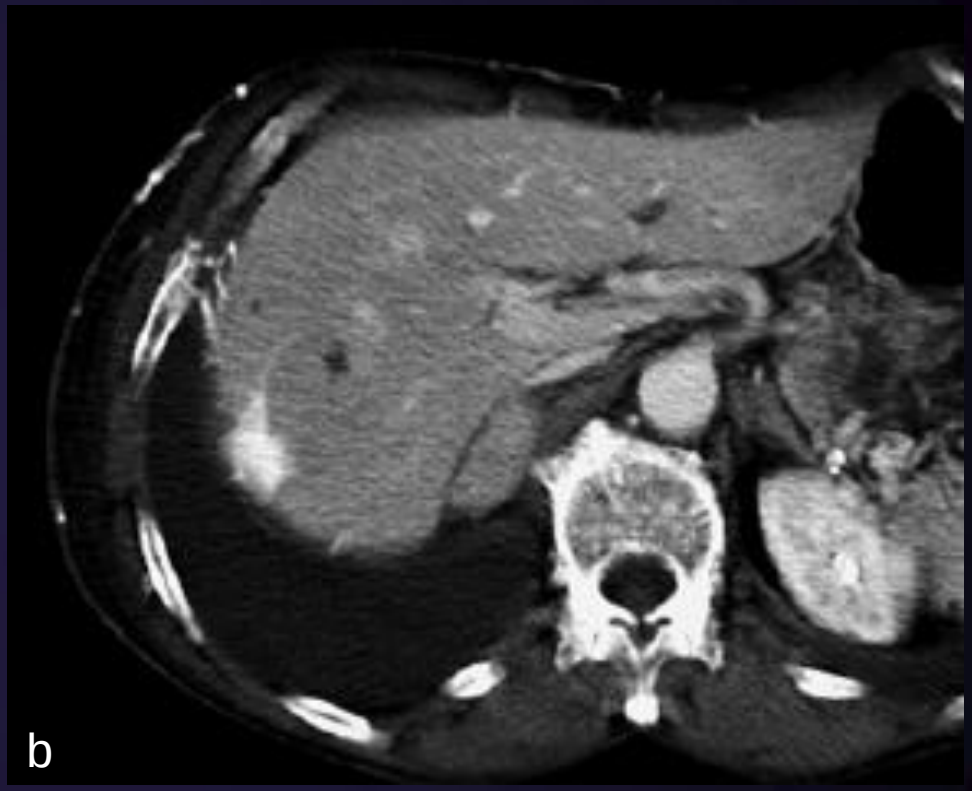
Schematic drawing of non-portal splanchnic perfusion to the liver parenchyma.

1.4. “NON-PORTAL” VENOUS SUPPLY TO THE LIVER

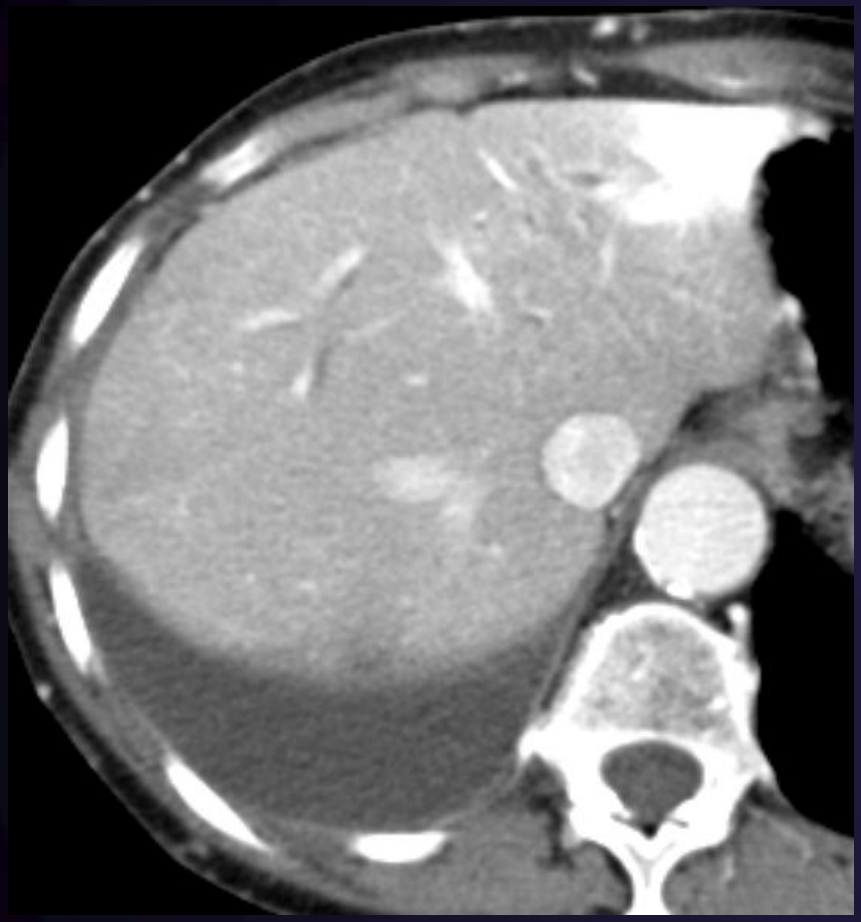


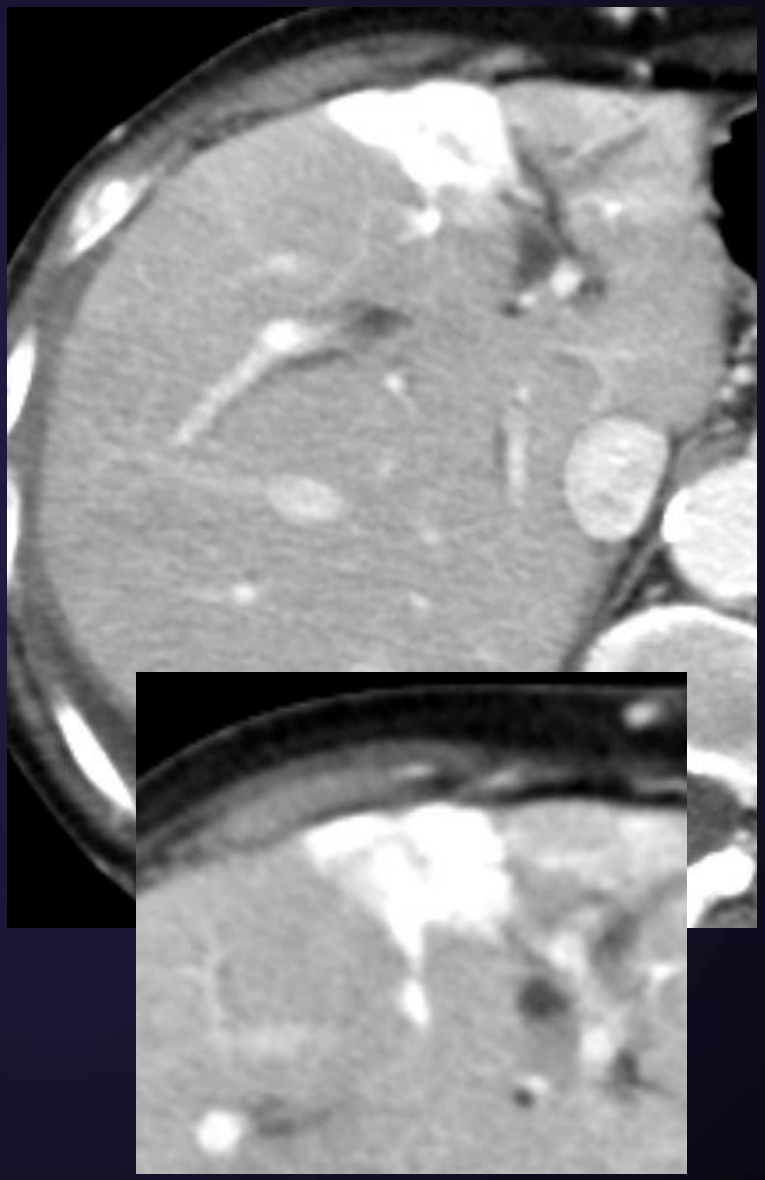
1.4. “NON-PORTAL” VENOUS SUPPLY TO THE LIVER





1.4. “NON-PORTAL” VENOUS SUPPLY TO THE LIVER

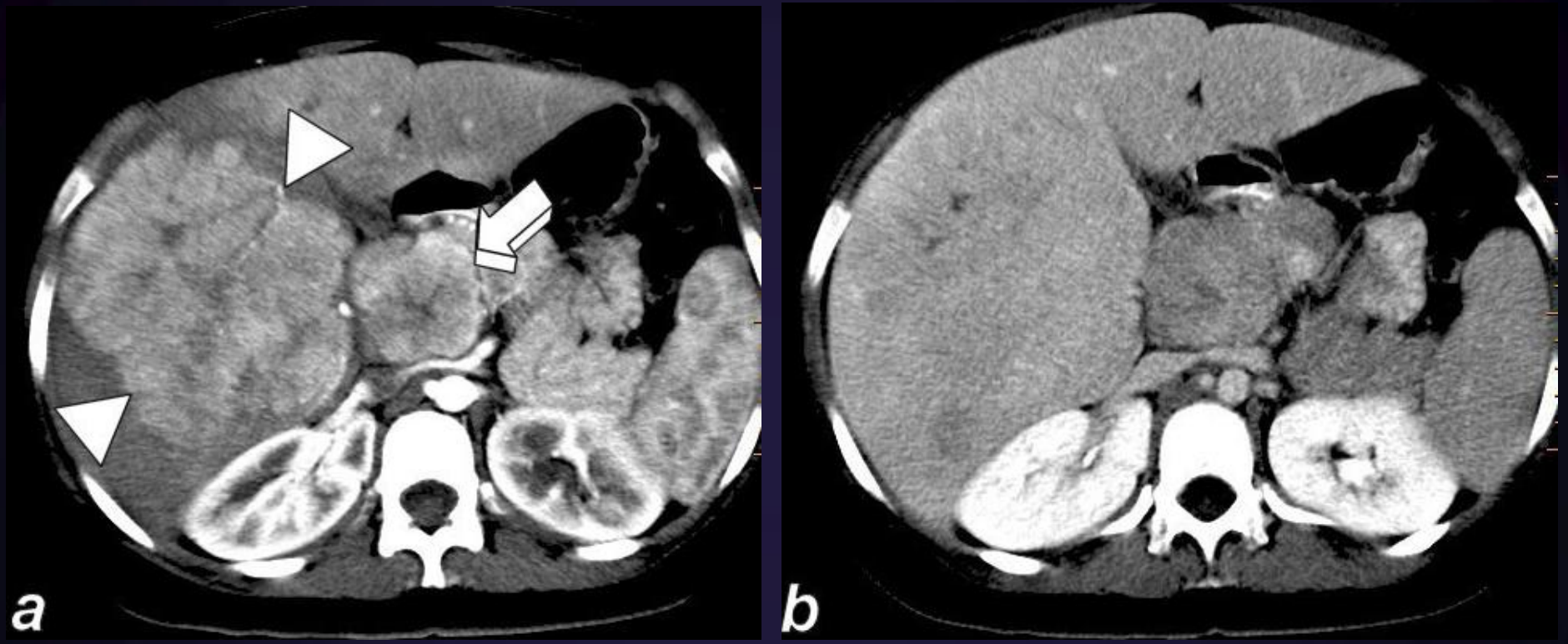




1.5. STEAL PHENOMENA

- 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 phenomenon is transitory and on the portal venous phase the affected parenchyma returns to isodensity.

1.5. STEAL PHENOMENA



Dynamic helical CT of an hypervascular liver metastasis from non-functioning malignant neuroendocrine pancreatic tumor.

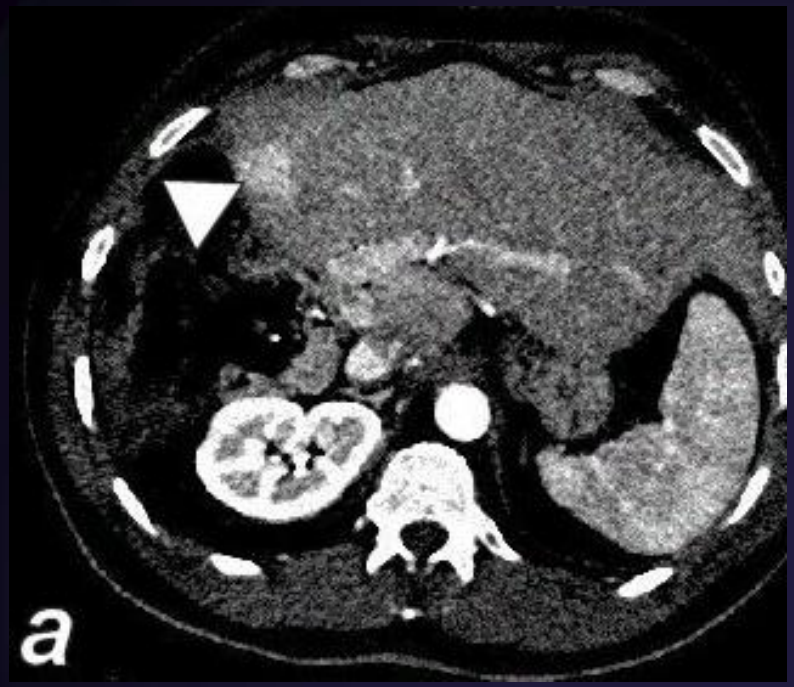
1.6. POST-SURGICAL PARASITIC ARTERIAL FLOW

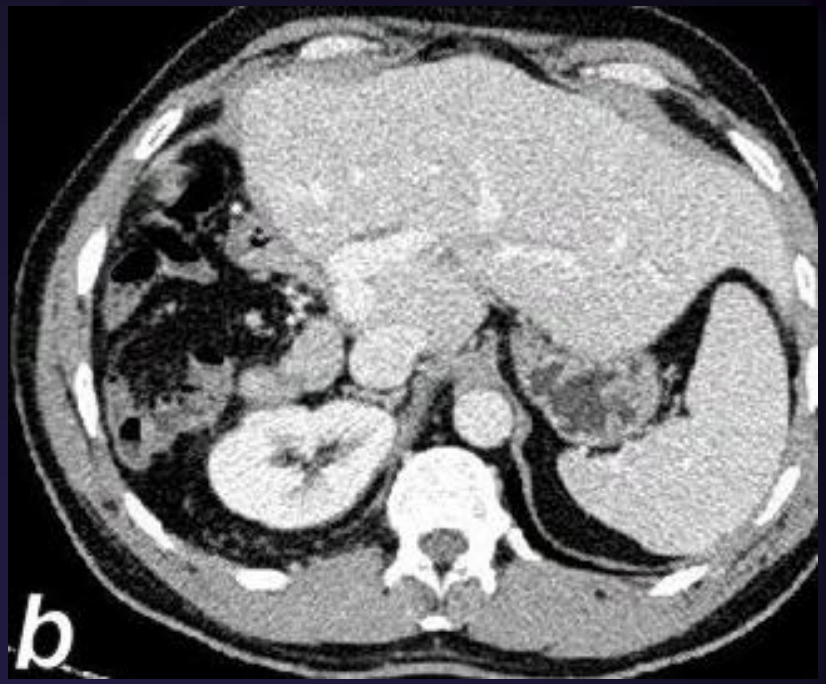
- In patients submitted to previous liver surgery, contrast-enhanced cross-sectional imaging can show early-enhancing non-neoplastic areas. They can be ill-defined, irregular, serpiginous, nodular or wedge-shaped, and are mostly seen on the resection margin along the liver edge.
- When these areas display 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.

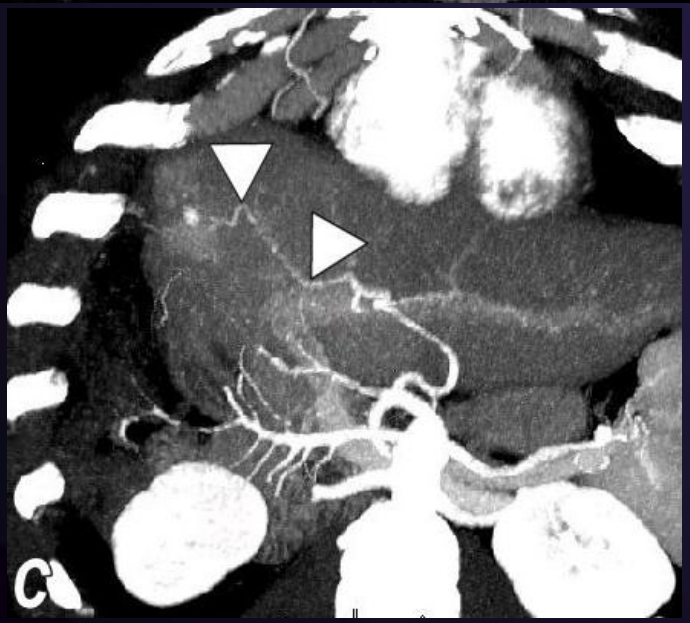
1.6. POST-SURGICAL PARASITIC ARTERIAL FLOW

- These pseudo-lesions possibly result from the aggressive surgical procedure with hepatic artery ligation to control bleeding.
- Alternative pathways develop due to a localized increased arterial flow through accessory hepatic arteries, interlobar collaterals, right inferior diaphragmatic artery or even thin branches of the gastroduodenal or pancreatic arteries.

1.6. POST-SURGICAL PARASITIC ARTERIAL FLOW





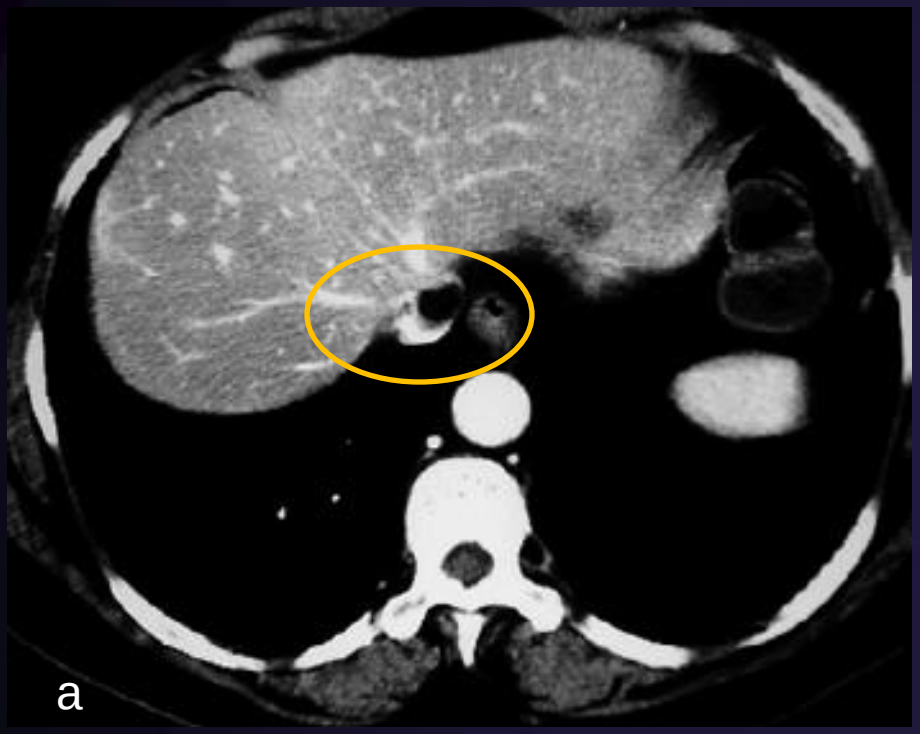


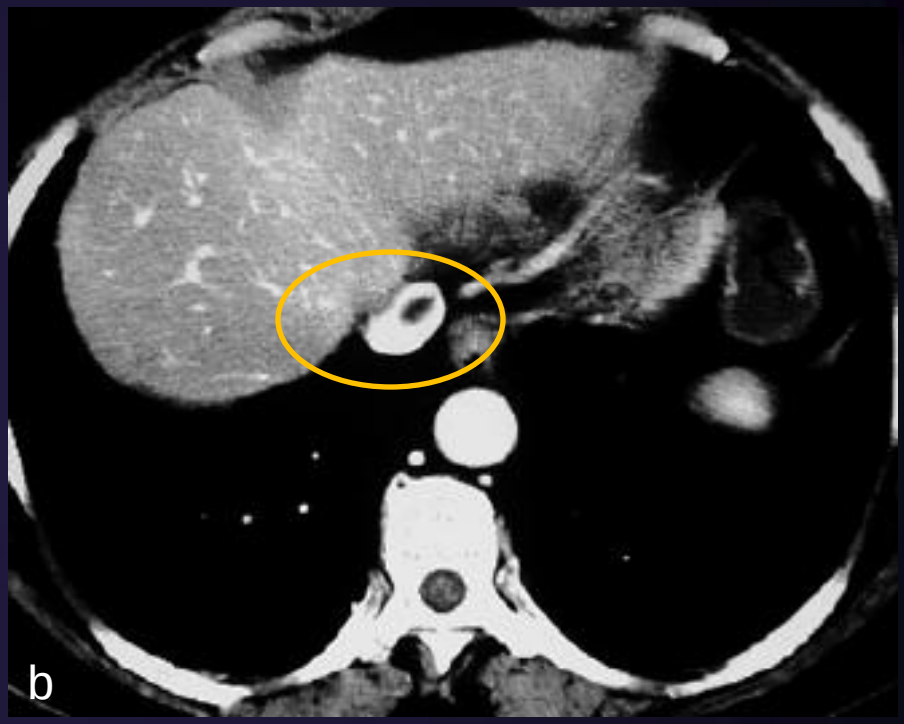
Cross-sectional studies in a patient with previous history of right hepatectomy.

1.7. 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 band the vessel itself.

1.7. PERICAVAL FAT COLLECTION

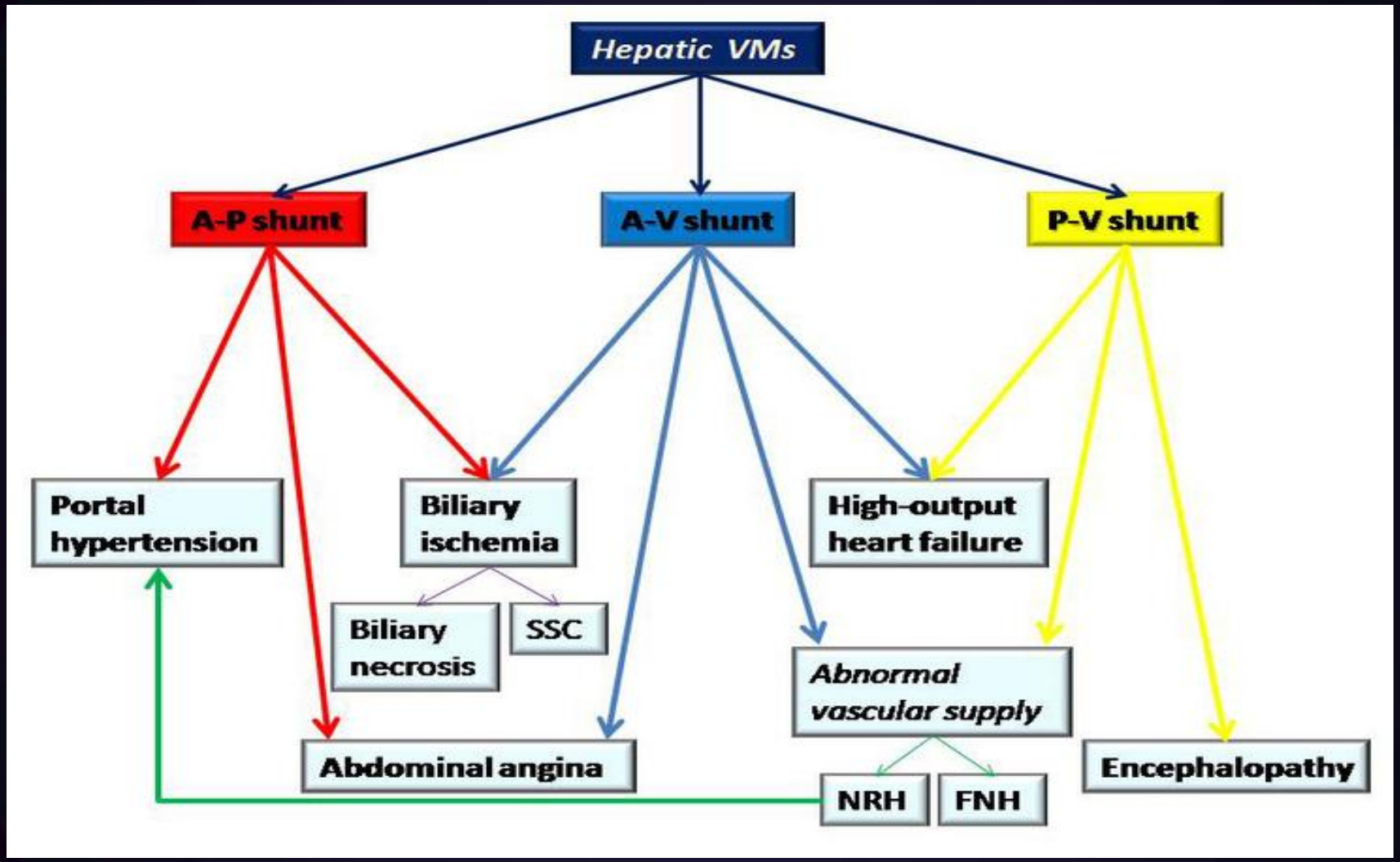




2. HEREDITARY HEMORRHAGIC TELANGIECTASIA

- Hereditary hemorrhagic telangiectasia, 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.

2. HEREDITARY HEMORRHAGIC TELANGIECTASIA



Schematic representation of the pathophysiology of different presentations of liver involvement in HHT: the clinical findings are mainly due to vascular shunts.

2. 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)***
  2. Hepatic artery to hepatic veins (***A-V shunts)***
  3. Portal vein to hepatic veins (***P-V shunts)***

2. HEREDITARY HEMORRHAGIC TELANGIECTASIA

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.

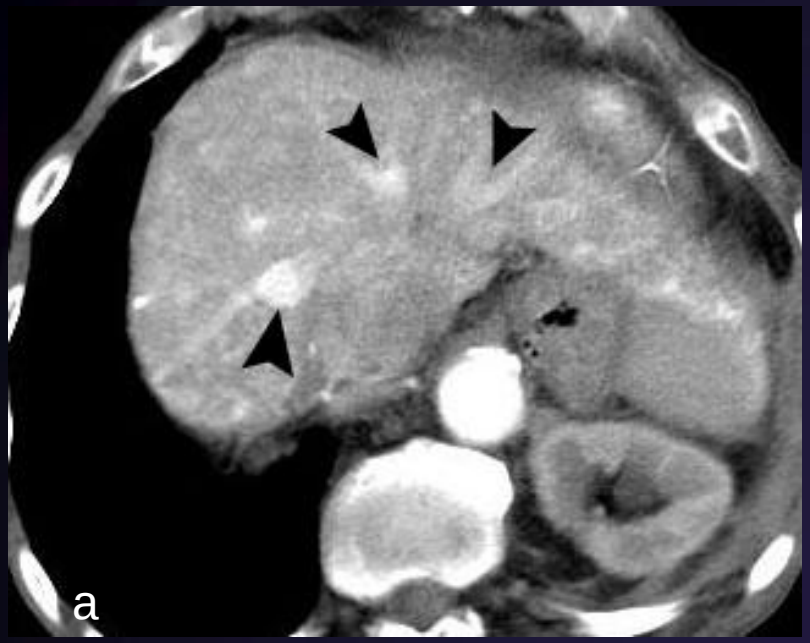
2. HEREDITARY HEMORRHAGIC TELANGIECTASIA

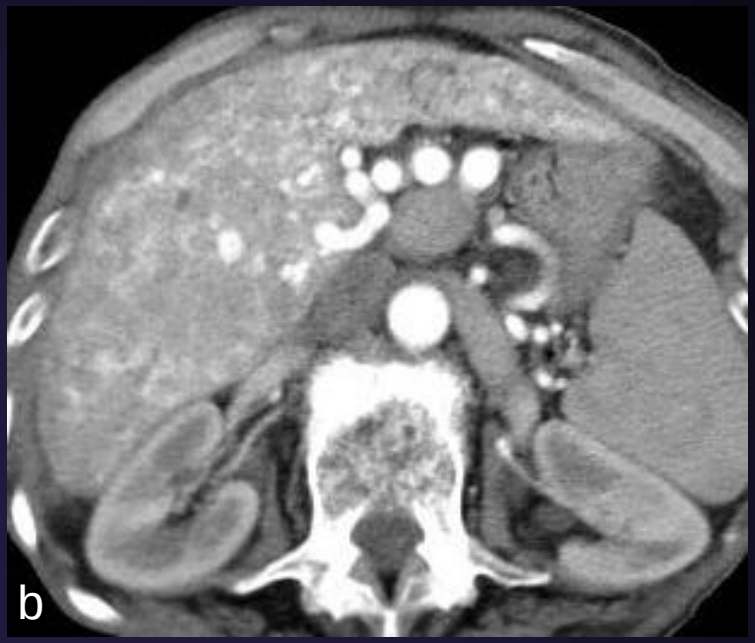
2. Hepatic artery to hepatic veins (***A-V shunts)***
   - Early enhancement of one or more hepatic veins during the early arterial phase.
   - Enlargement of the early filled hepatic vein(s).
   - Heterogeneous enhancement (mosaic pattern) during the arterial phase; Hepato-splenomegaly.
   - Ascites;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

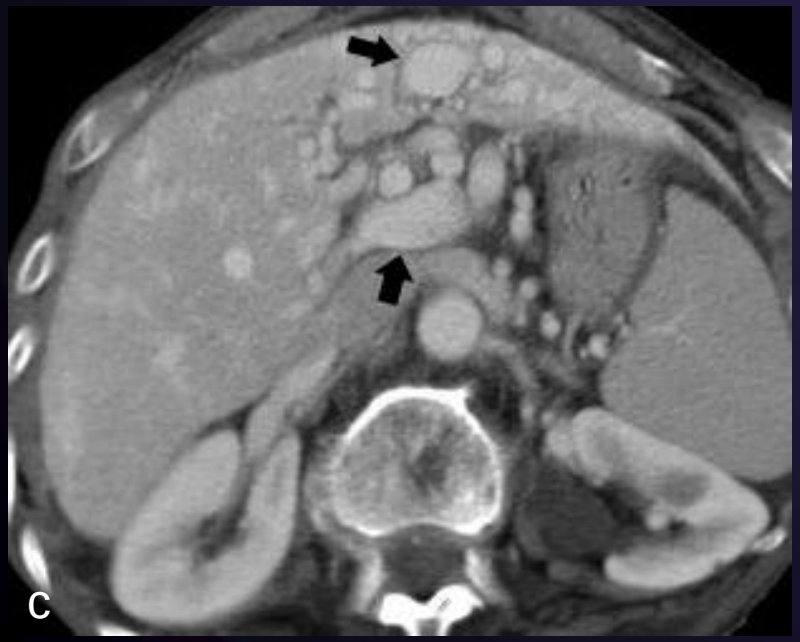
2. HEREDITARY HEMORRHAGIC TELANGIECTASIA

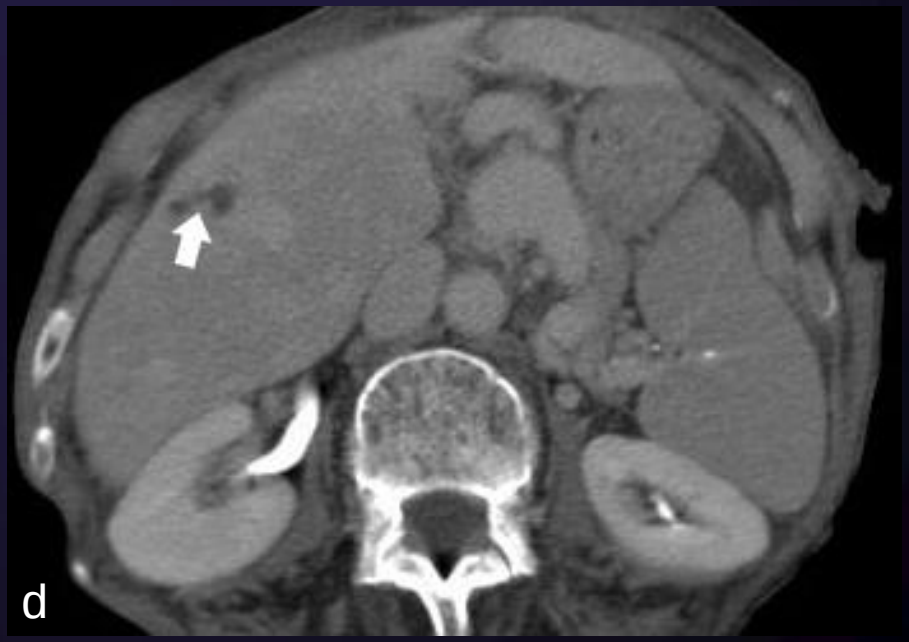
3. Portal vein to hepatic veins (***P-V shunts)***
   - Dilated portal branches communicating with large-calibre hepatic or systemic veins on portal venous phase.

2. HEREDITARY HEMORRHAGIC TELANGIECTASIA









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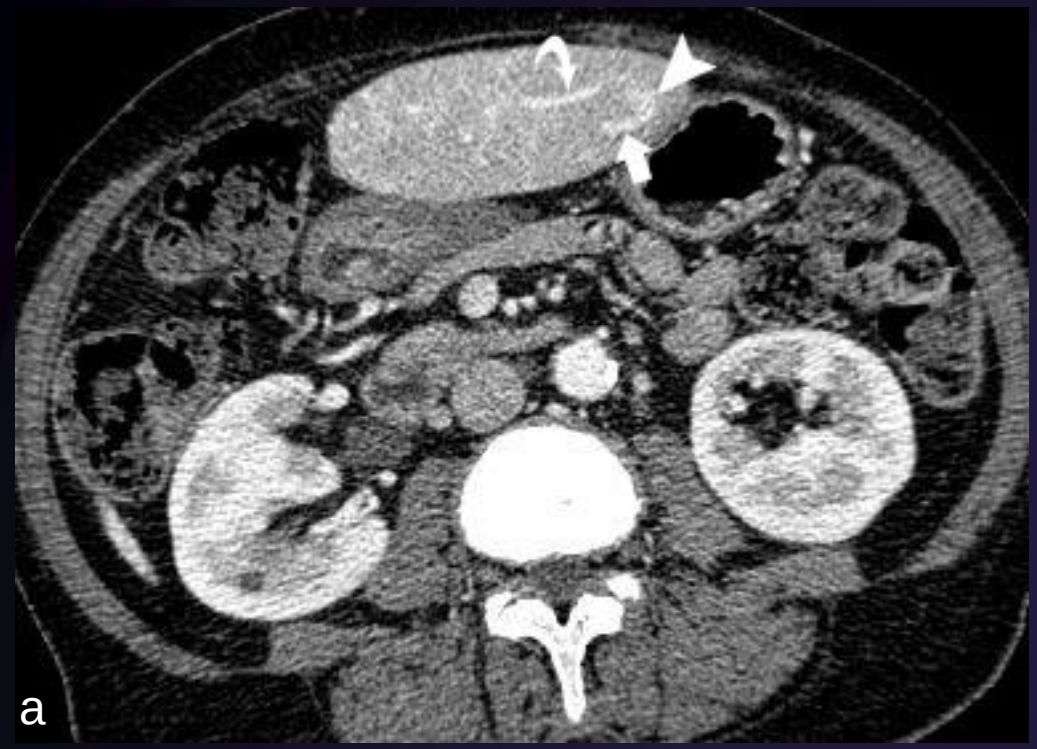

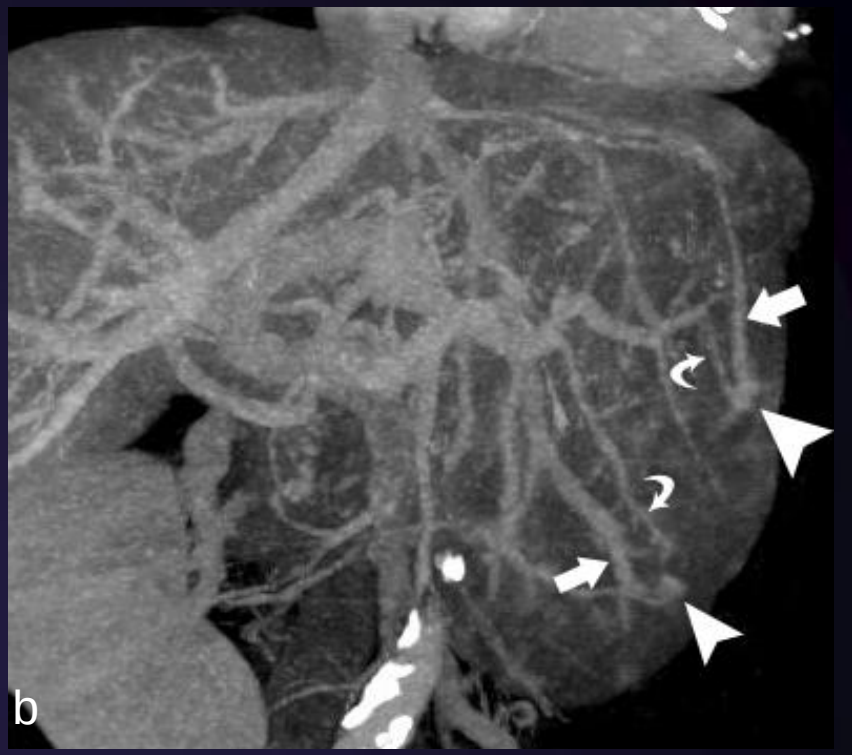

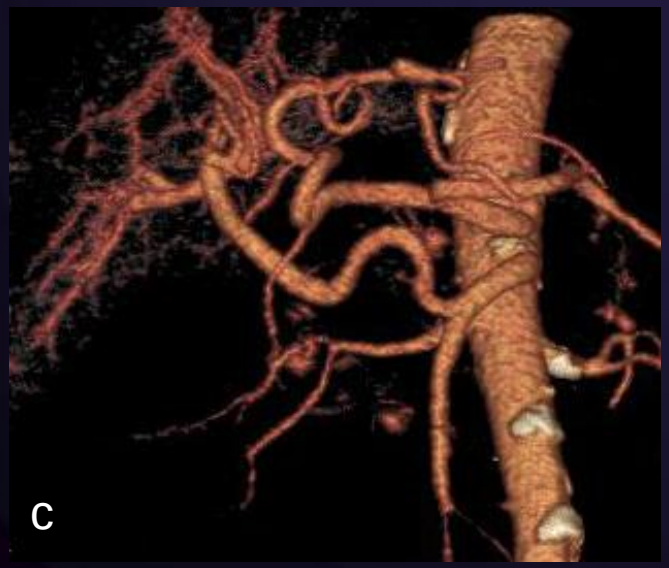

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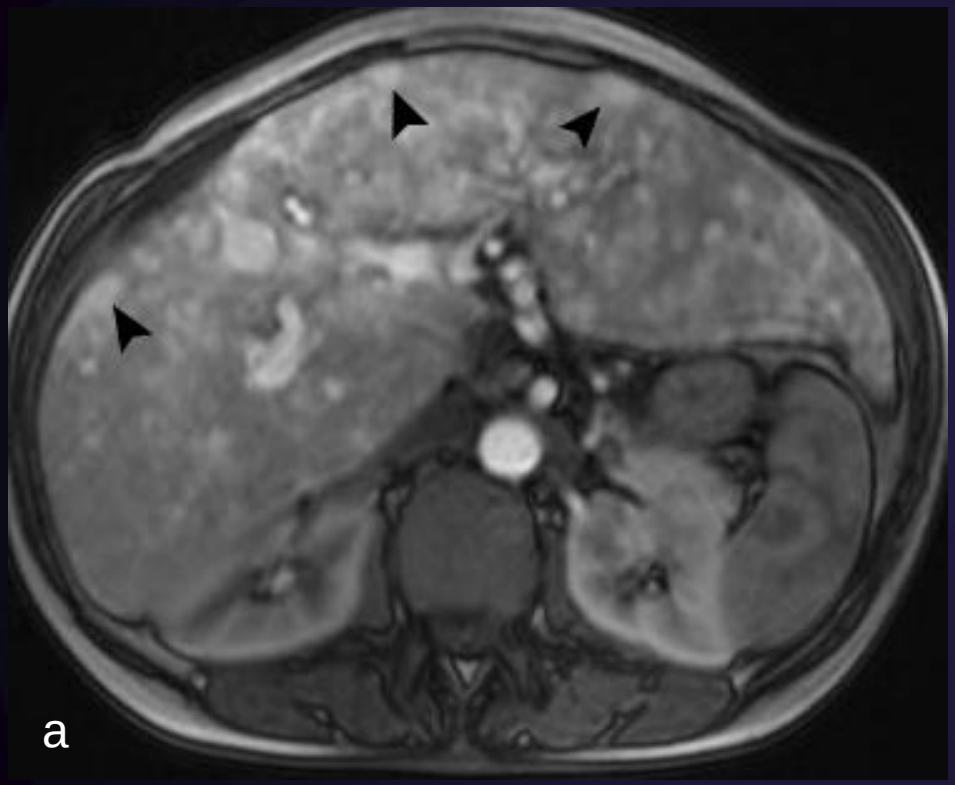

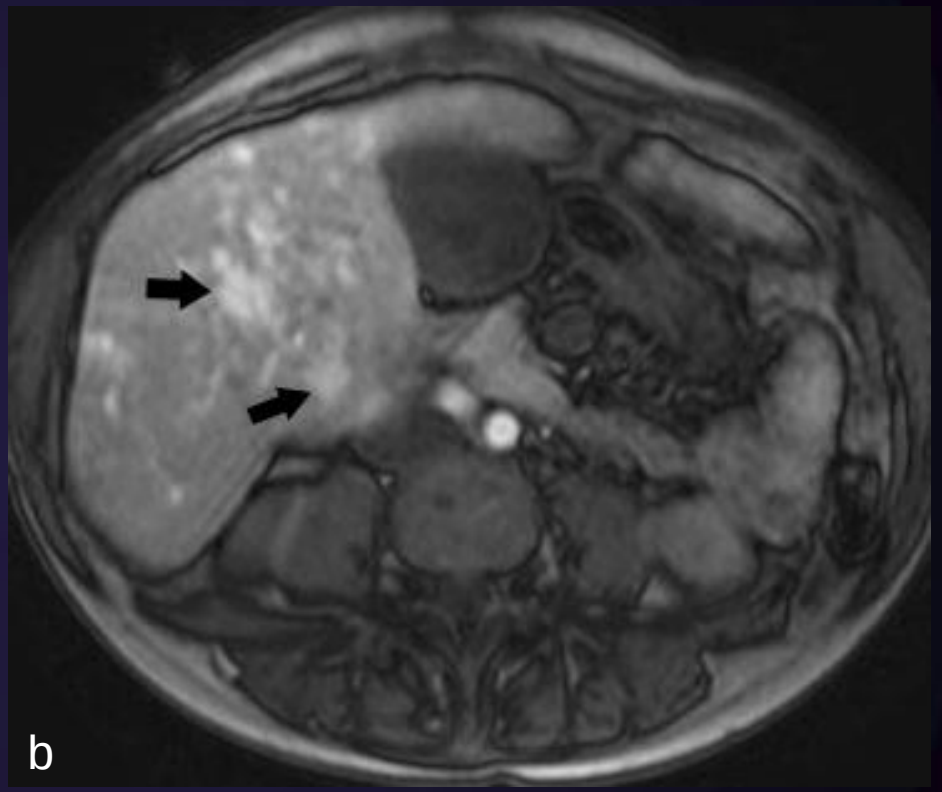

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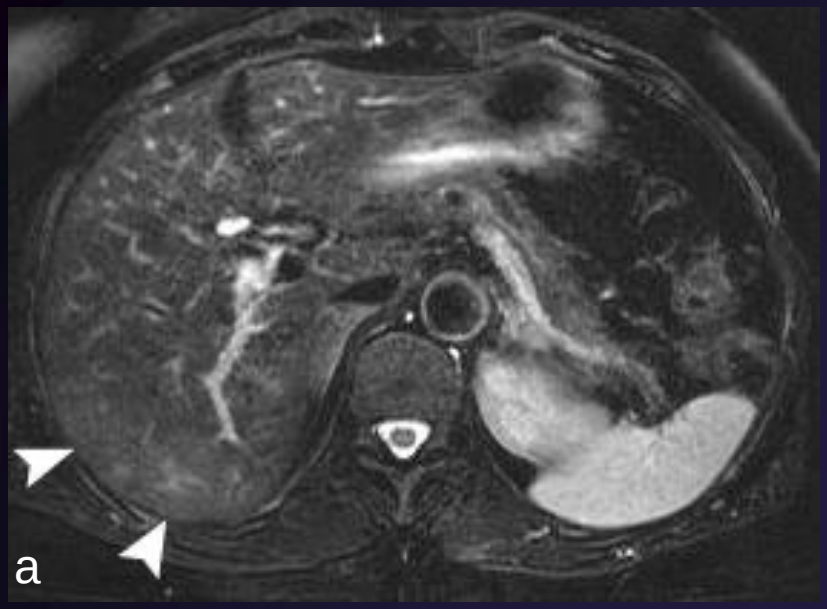
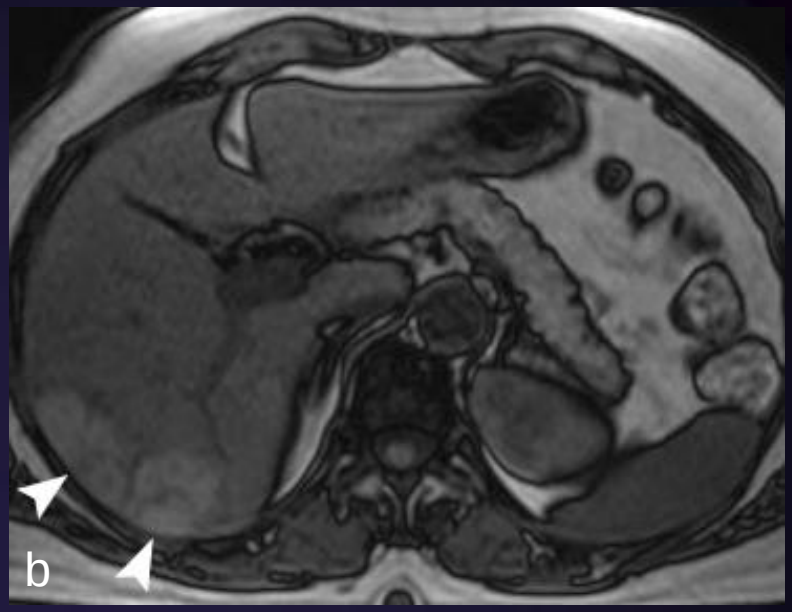
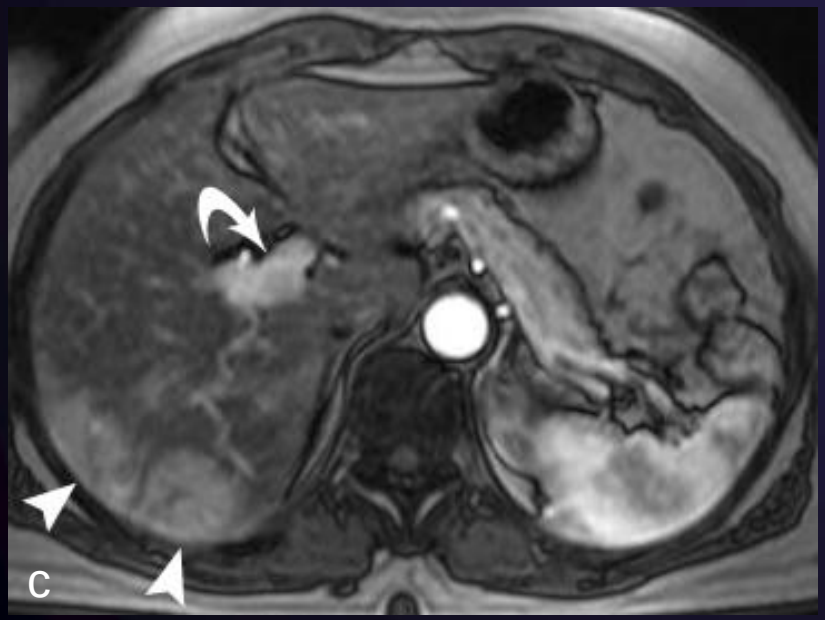
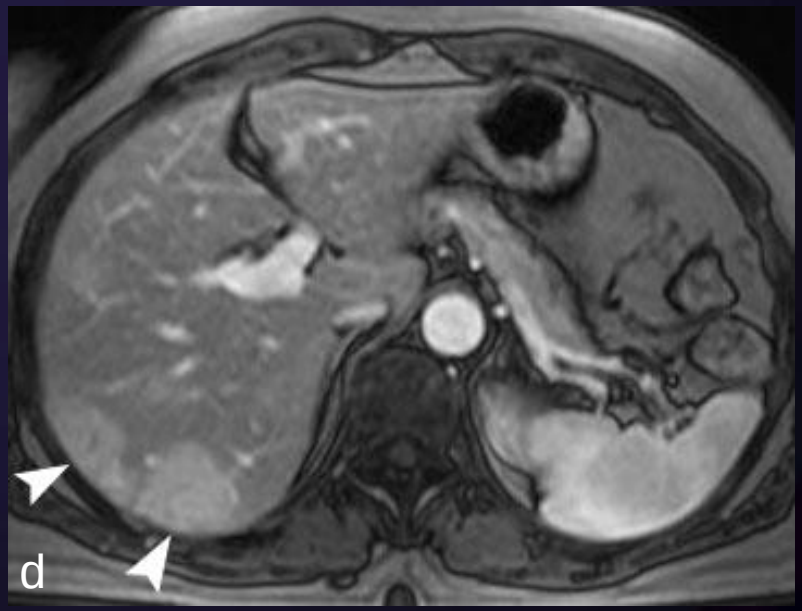








3. BUDD-CHIARI SYNDROME

- 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).
- On the acute phase, besides enlargement of the liver, the post-sinusoidal obstruction causes a severe reduction of the portal venous flow with a compensatory increase of the arterial flow.
- Functional intra-hepatic arterio-portal shunts are recruited,which may ultimately lead to a complete reversal of flow within the portal vein. Dynamic studies will show an isolated and vigorous enhancement of the portal vein at the arterial phase.

3. BUDD-CHIARI SYNDROME

- On the later phases of enhancement a mottled parenchymal appearance is the result of the efferent vessel obstruction, causing stasis and distal accumulation of the intravascular contrast material.
- Thrombi may be identified in the ICV or hepatic veins.
- Ascitis is usually present .

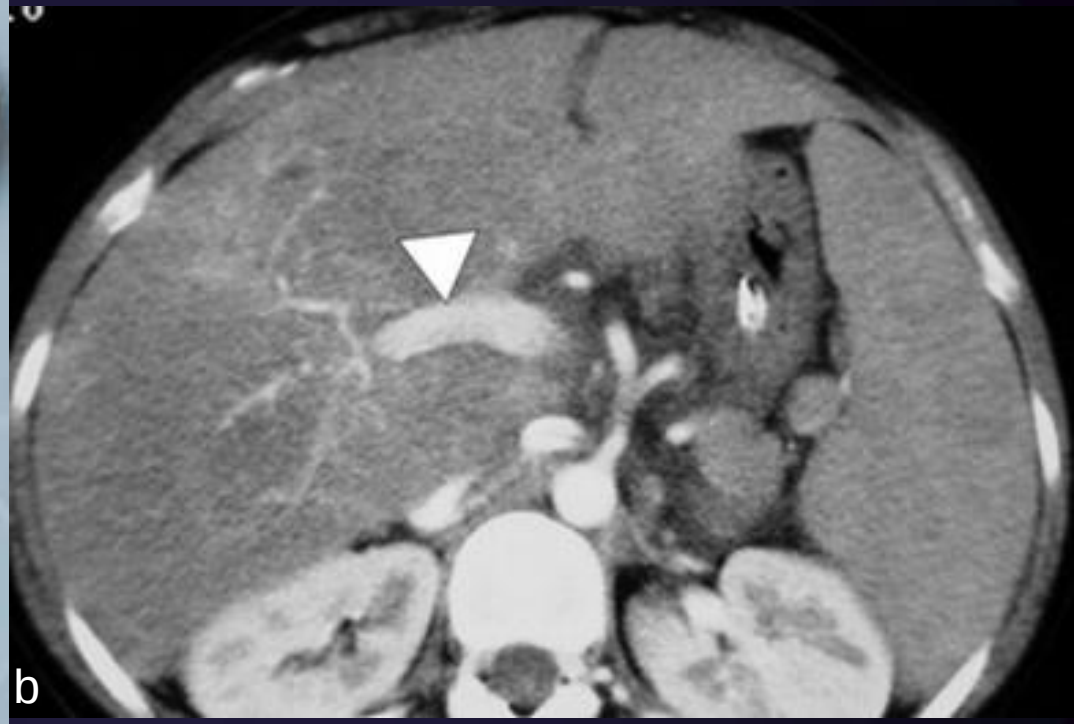
3. BUDD-CHIARI SYNDROME

- 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, 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 (often enlarged) due to its separate, autonomous venous drainage.

3. BUDD-CHIARI SYNDROME

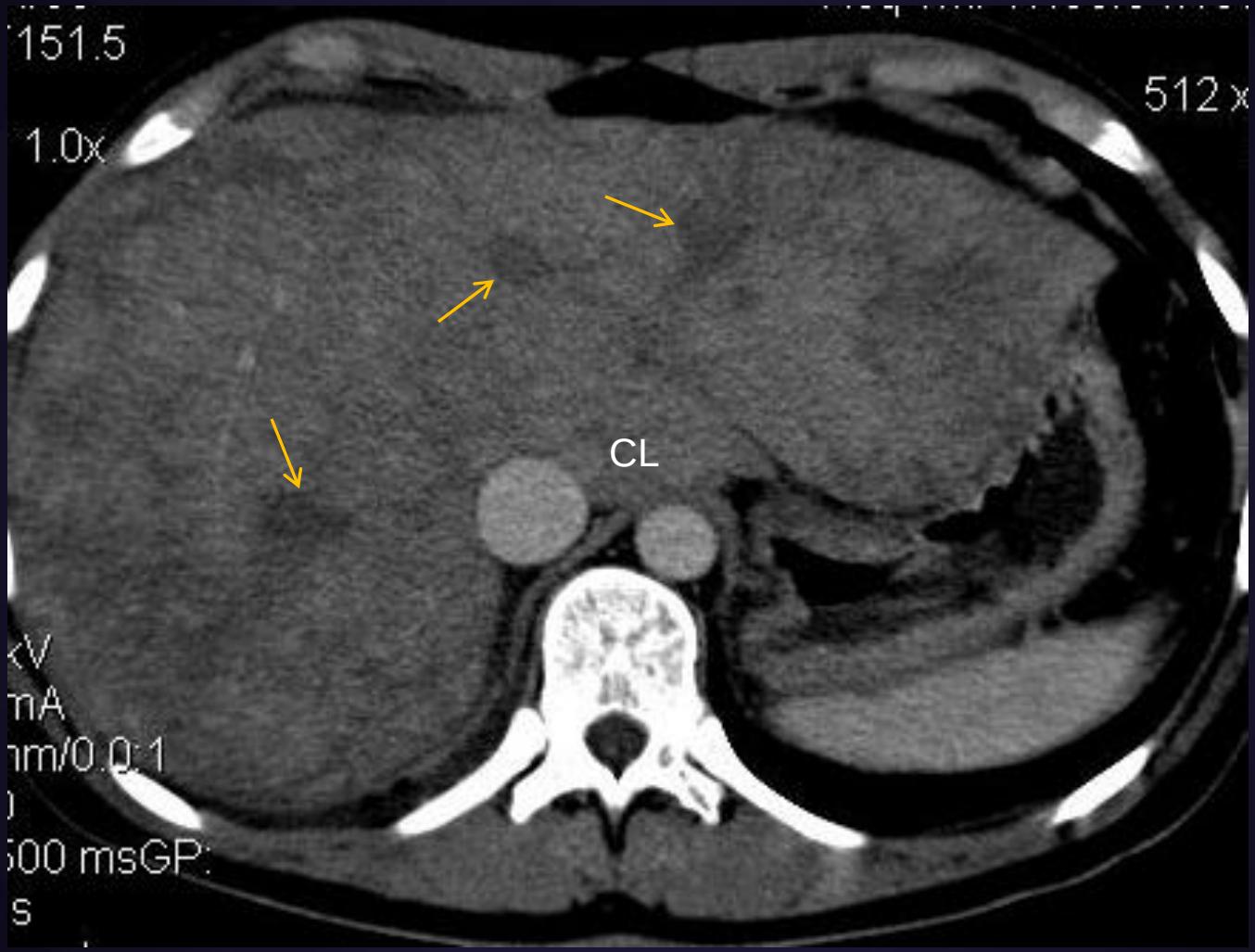
- Some patients develop large benign regenerative nodules, which are usually multiple and less than 4 cm in diameter.
- The regenerative nodules in Budd-Chiari syndrome differ from regenerative nodules secondary to cirrhosis in these respects:
  - More often hyperintense on T1 WI
  - Usually enhance on arterial-phase images
  - Larger lesions (> 1cm) often have central scars

3. BUDD-CHIARI SYNDROME



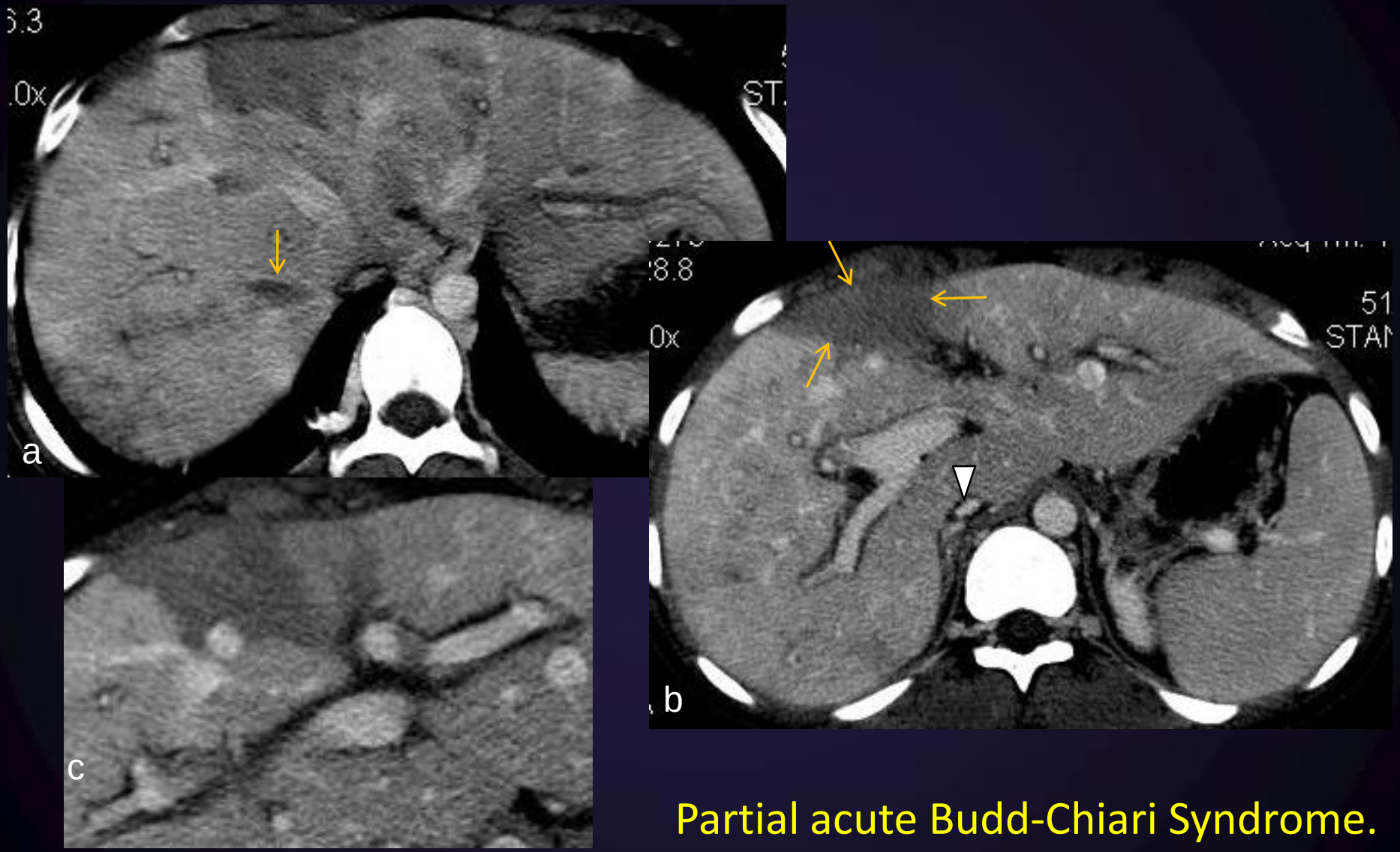
Abnormal liver hemodynamics in the acute phase of Budd-Chiari syndrome.

3. BUDD-CHIARI SYNDROME



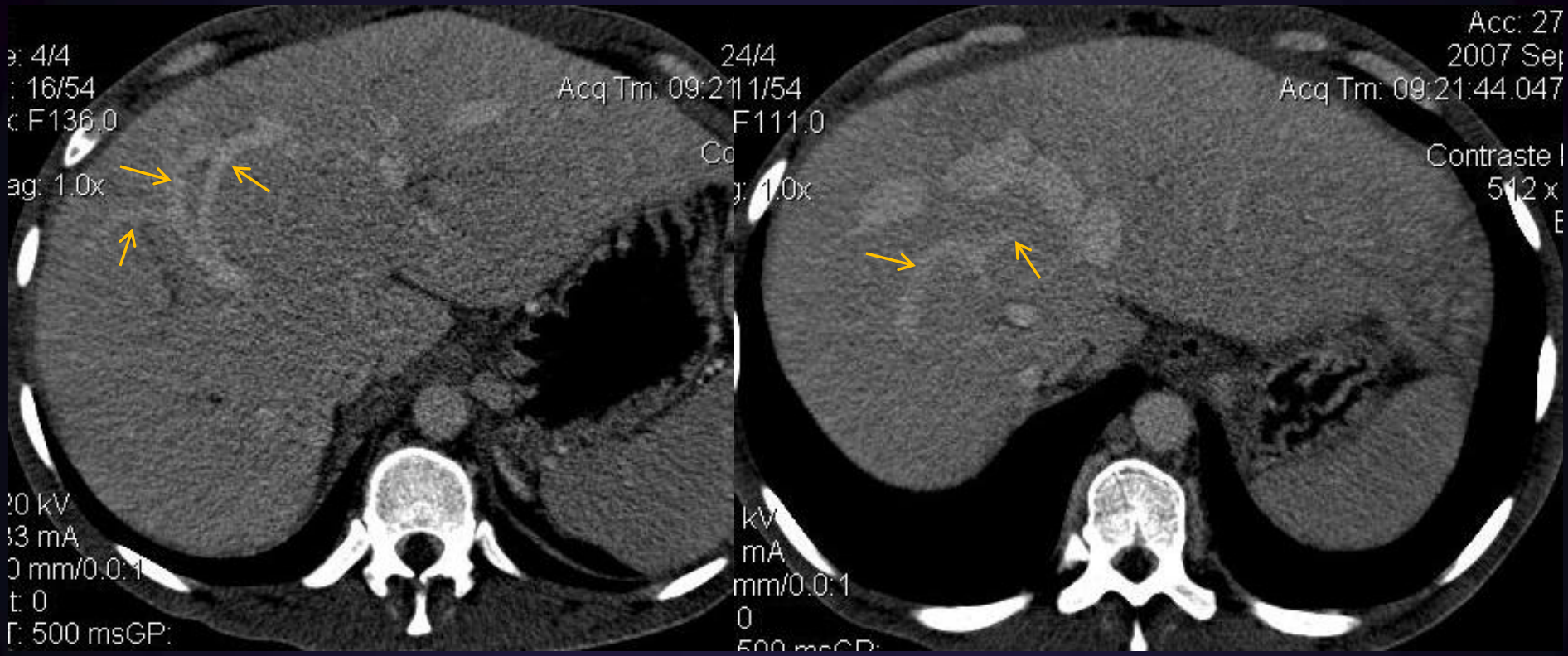
Acute Budd-Chiari Syndrome.

3. BUDD-CHIARI SYNDROME



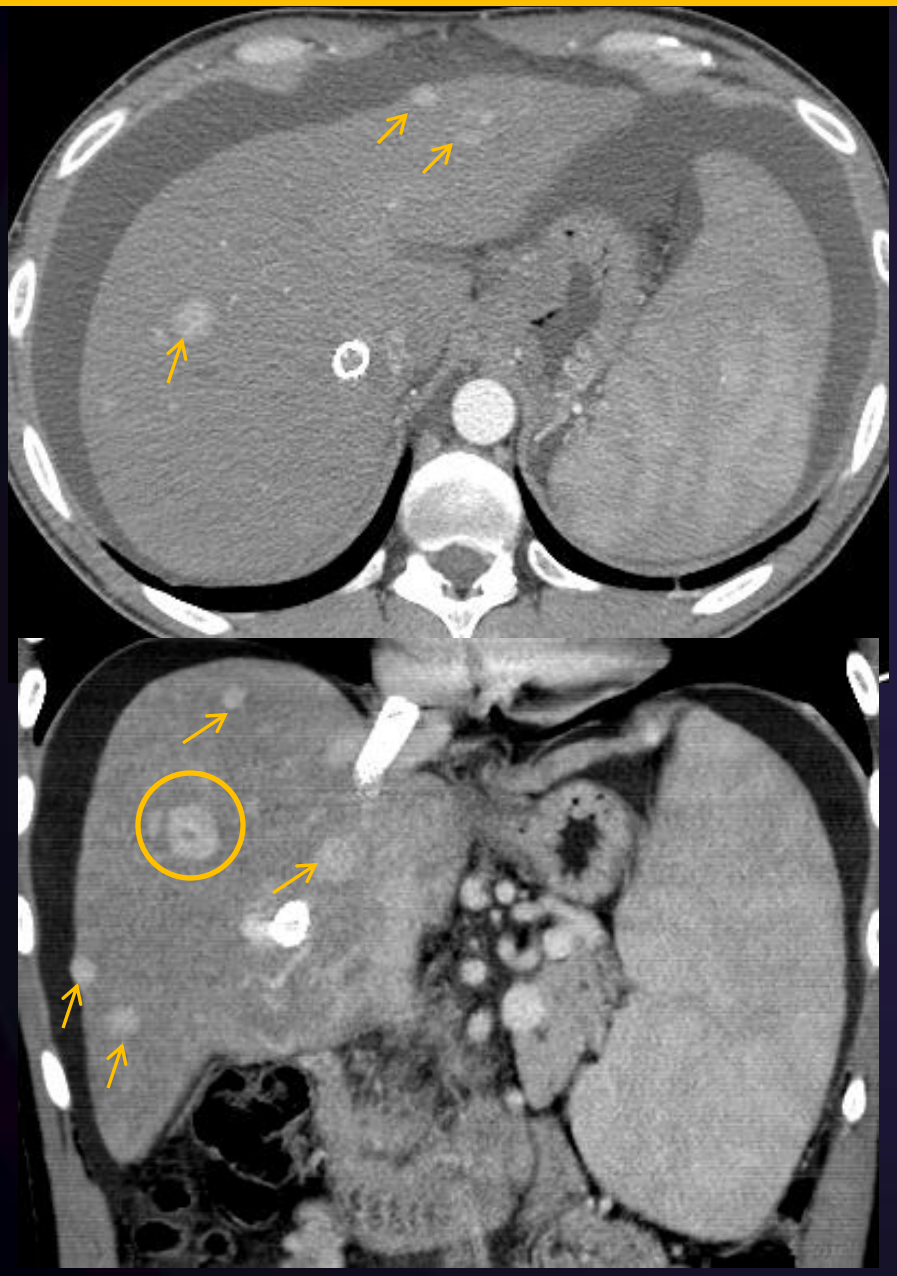
Partial acute Budd-Chiari Syndrome.

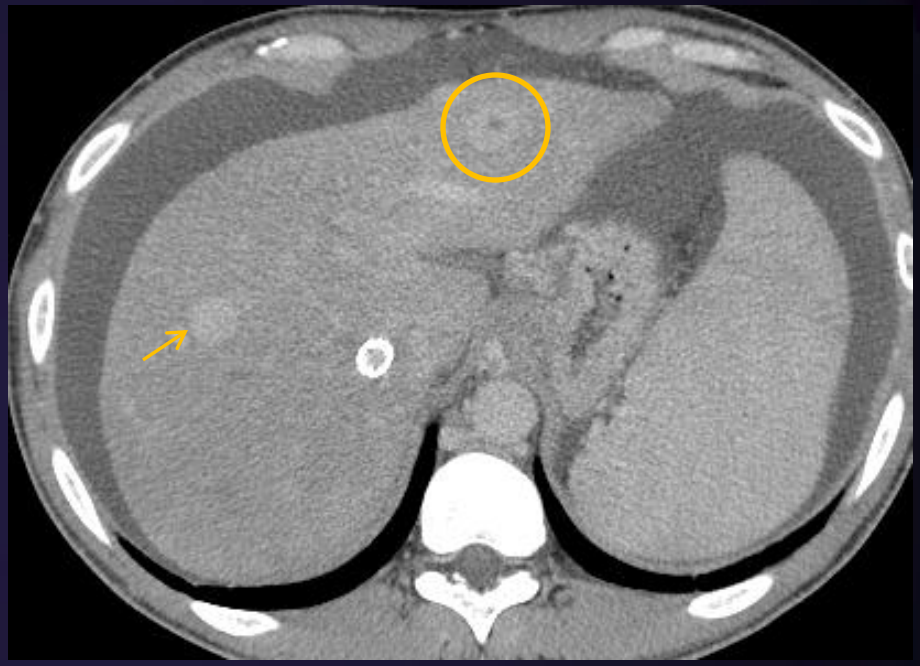
3. BUDD-CHIARI SYNDROME



Chronic phase of Budd-Chiari disease.

3. BUDD-CHIARI SYNDROME





Chronic phase of Budd-Chiari disease.

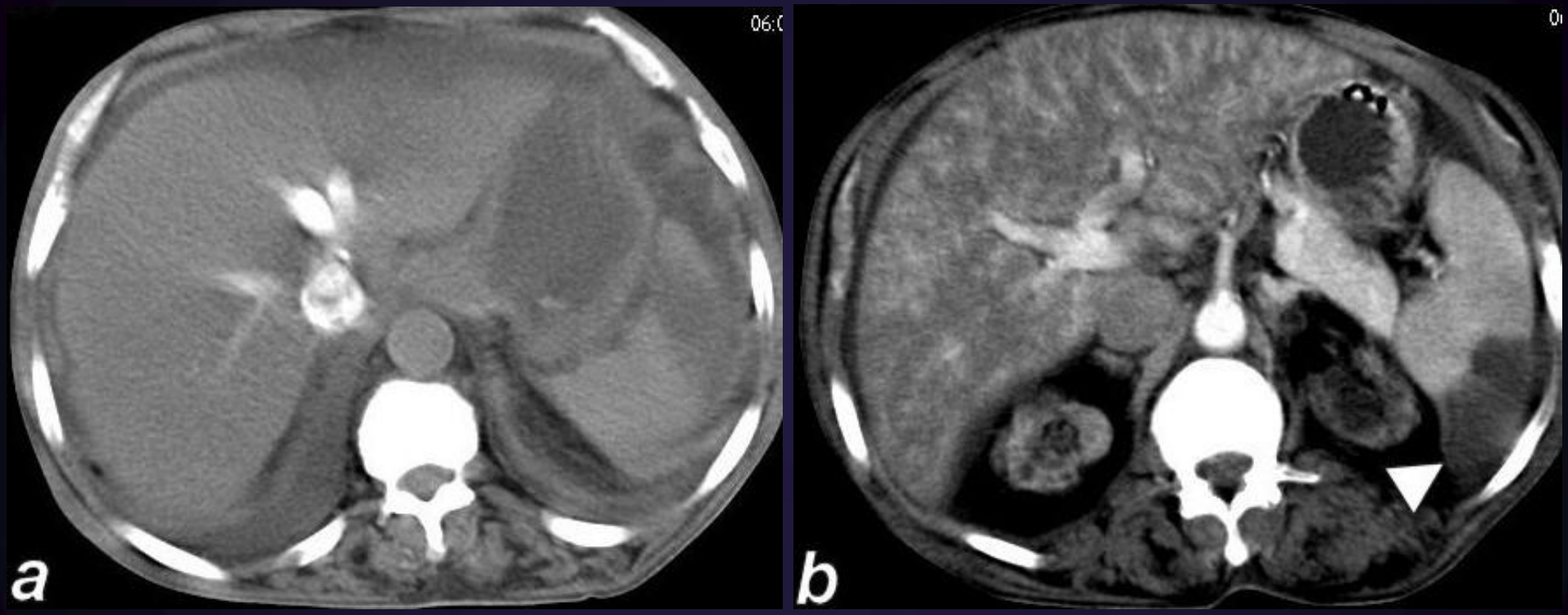
4. PASSIVE HEPATIC CONGESTION

- Complication of congestive heart failure or constrictive pericarditis.
- Elevated central venous pressure leads to decreased hepatic blood flow, elevated hepatic venous pressure and arterial hypoxemia, leading to sinusoidal congestion, dilatation and perisinusoidal edema.
- Major imaging features are a diffusely mottled hepatic parenchymal enhancement pattern and reflux of contrast-enhanced blood from the right atrium into the IVC and hepatic veins.

4. PASSIVE HEPATIC CONGESTION

- The proeminence of the hepatic veins and IVC helps distinguish passive hepatic congestion from Budd-Chiari syndrome, that has poorly visualized or thrombosed intra-hepatic cava or hepatic veins.
- Ancillary findings: cardiomegaly, pleural effusions, ascites and intrahepatic perivascular lucency.

4. PASSIVE HEPATIC CONGESTION

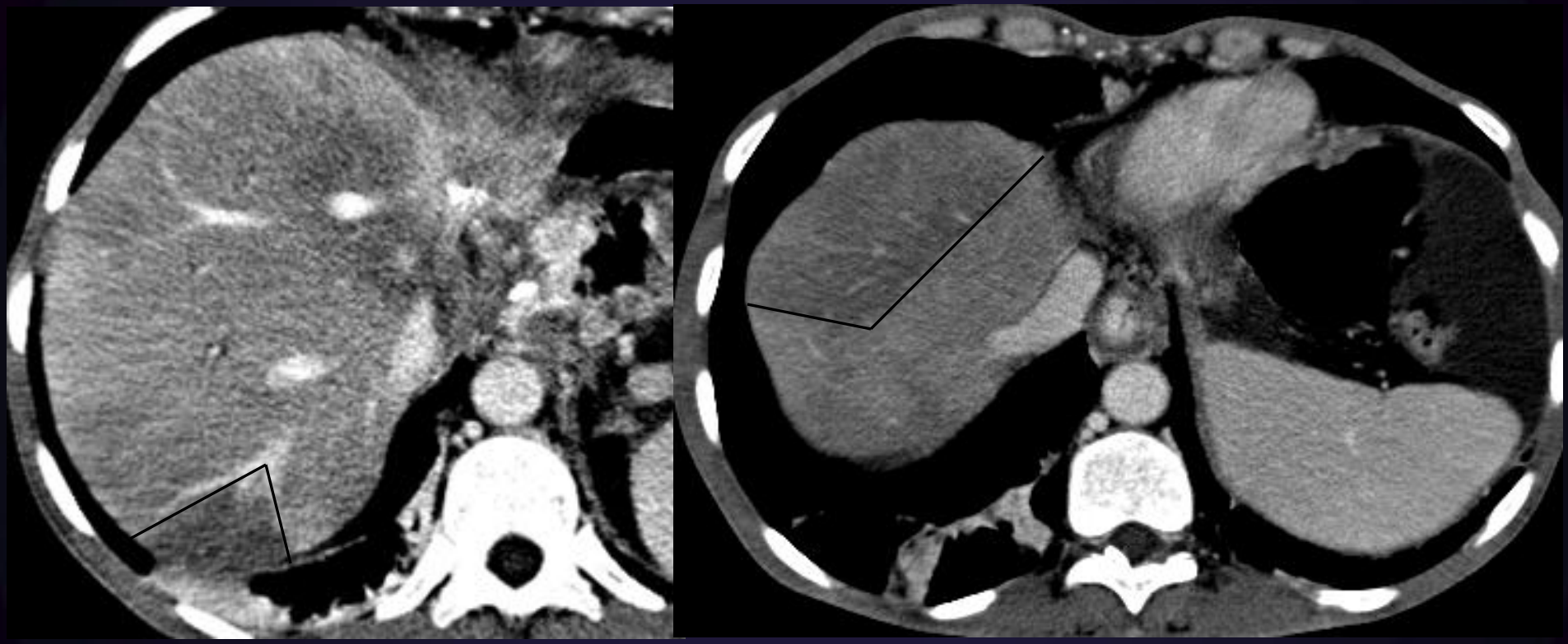


Major imaging features of passive hepatic congestion resulting from severe right-sided heart failure.

5. HEPATIC INFARCTION

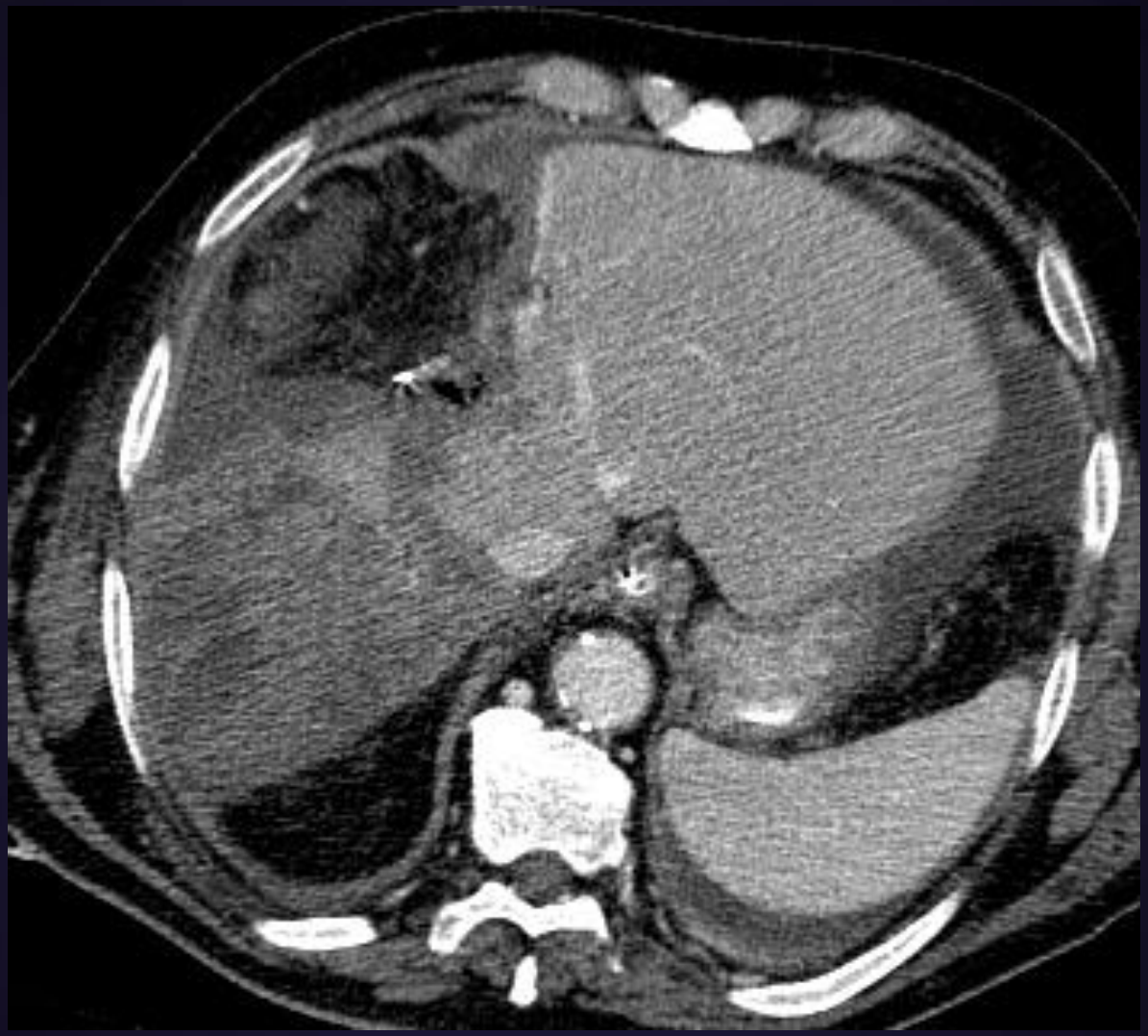
- Because the liver has a dual blood supply, hepatic infarction is uncommon. However, recent increases in the use of liver transplantation and laparoscopic cholecystectomy have led to an increase in the incidence of liver infarction.
- Three major configurations have been described:
  - Wedge-shaped,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

5. HEPATIC INFARCTION



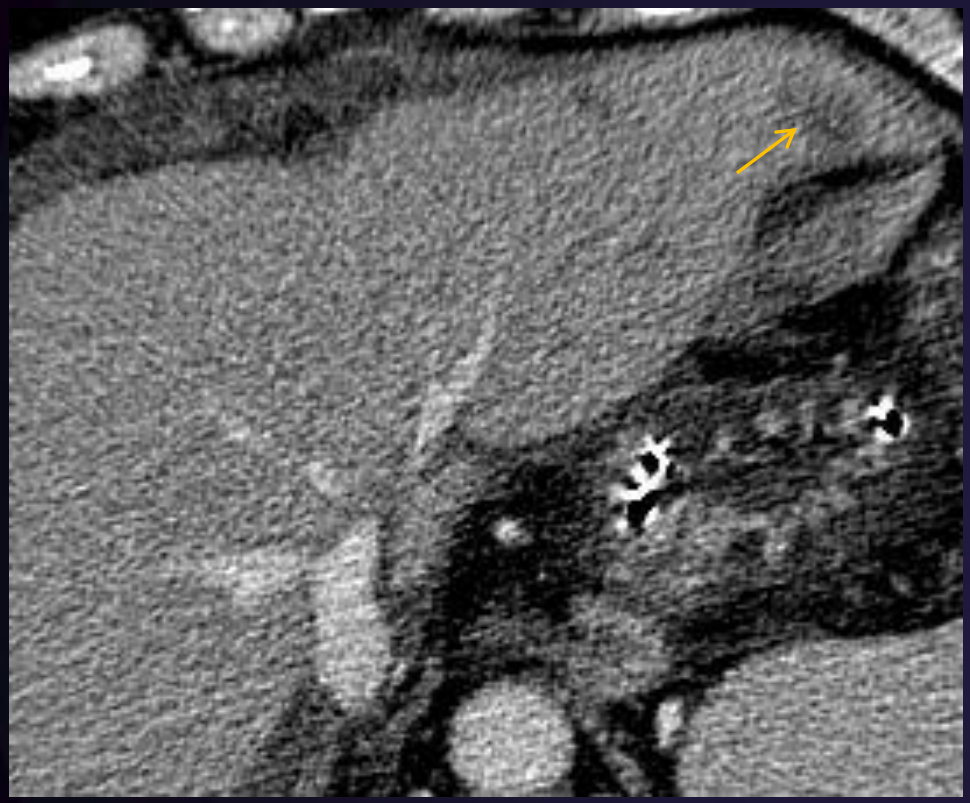
Hepatic infarctions.

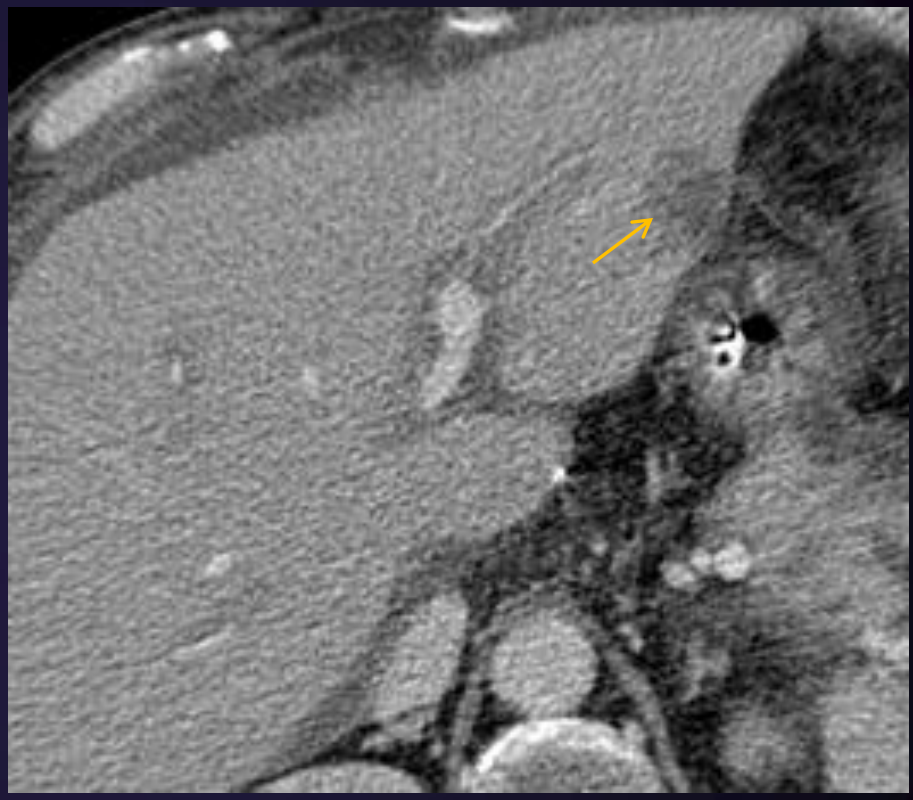
5. HEPATIC INFARCTION



Hepatic infarction.

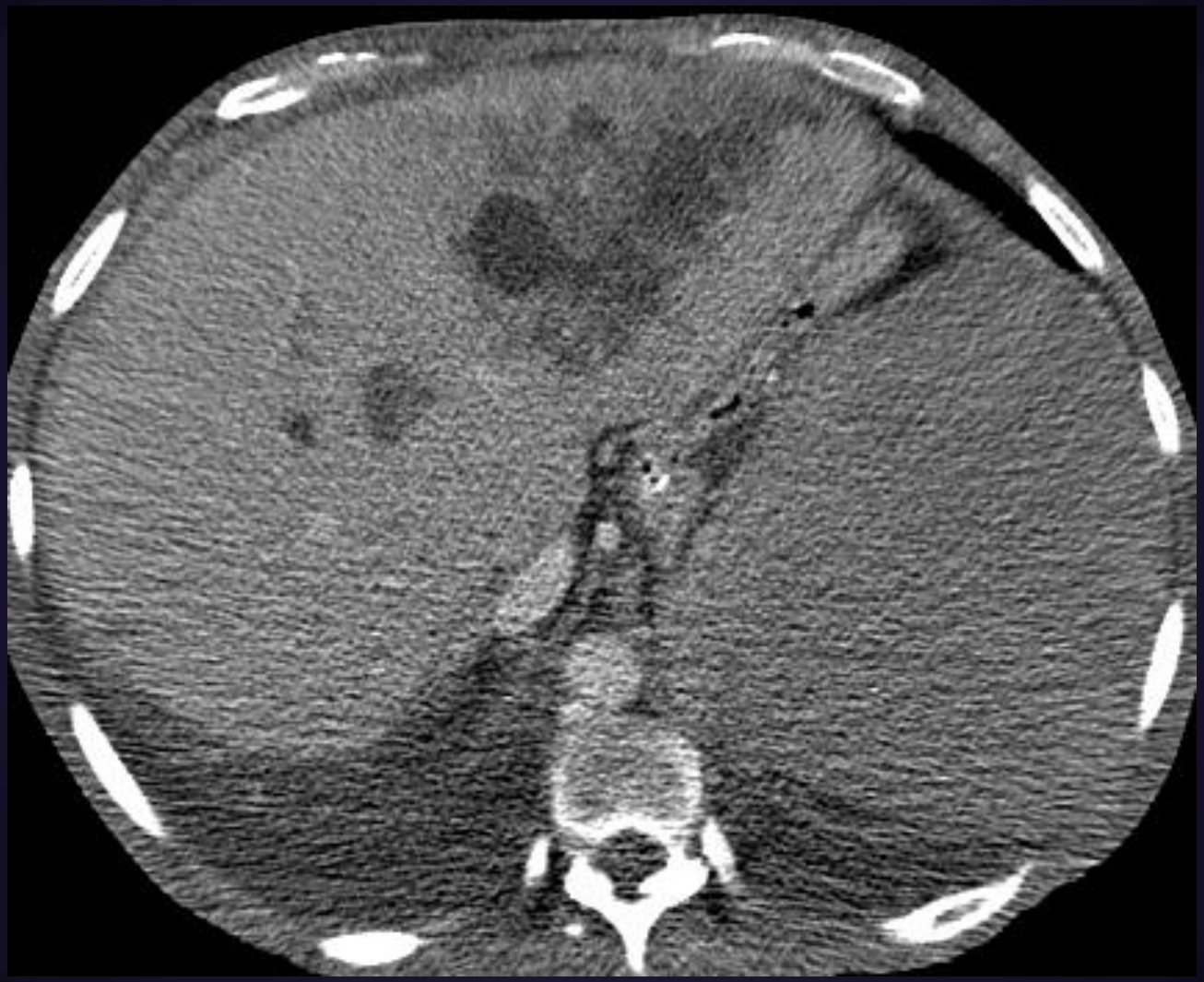
5. HEPATIC INFARCTION





Hepatic infarctions, atypical configuration.

5. HEPATIC INFARCTION



Hepatic infarction, atypical configuration.

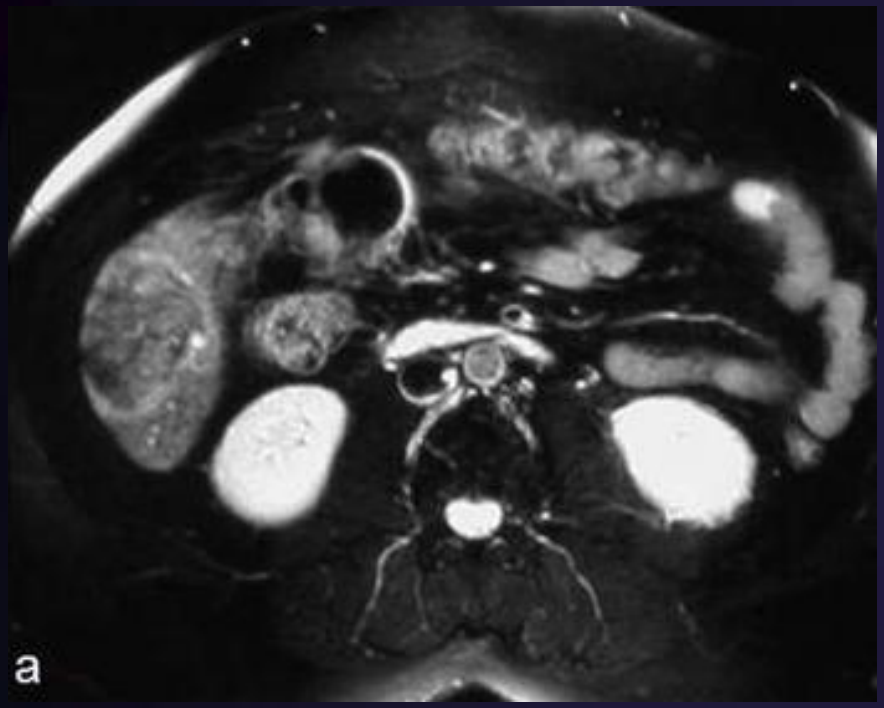
6. PELIOSIS HEPATIS

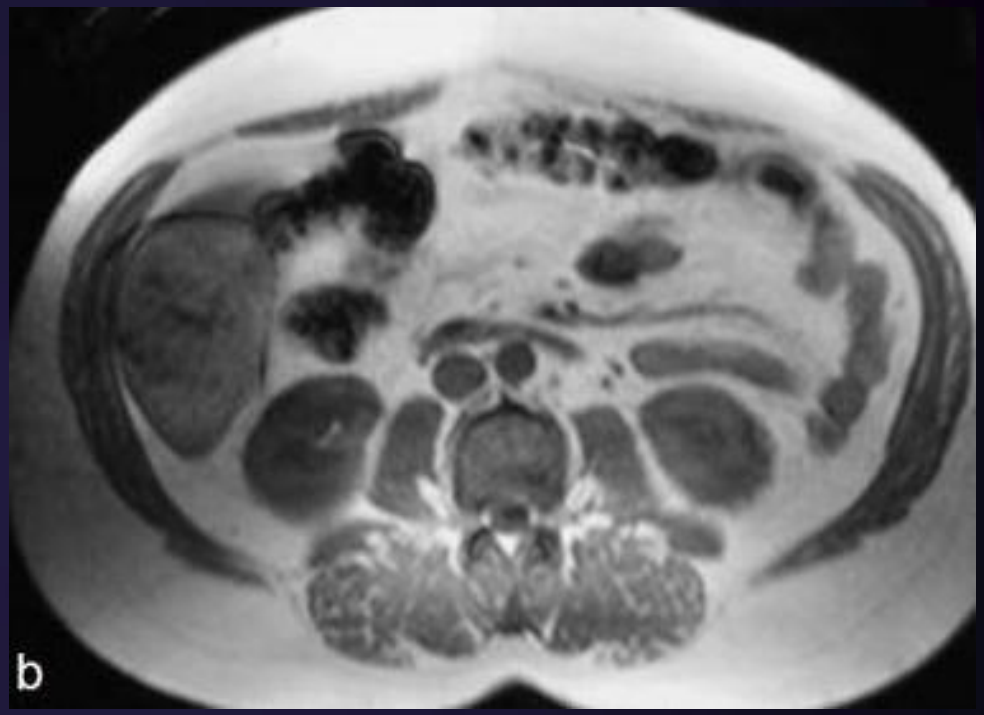
- Peliosis hepatis is an uncommon benign disorder of the liver, characterized by oval and irregularly shaped blood-filled spaces, which are lined either by hepatocytes or endothelial cells.
- These cavities communicate with the sinusoids, many of which are dilated.
- A number of theories have been postulated 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.

6. 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.
- Enhancement: Peripheral enhancement progressing centripetally or central enhancement progressing centrifugally – persists on delayed images
- 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.
- An important distinctive feature is that peliosis lacks mass efect.
- 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.

6. PELIOSIS HEPATIS





Peliosis hepatis.

7. PORTAL VEIN THROMBOSIS

- When acute the portal vein contents may be high in attenuation on precontrast images.
- Postcontrast images demonstrate a filling defect incompletely or completely filling the portal vein lumen.
- Enhancement of the wall of the portal vein can be identified with complete thrombosis, likely due to flow through dilated vasa vasorum.

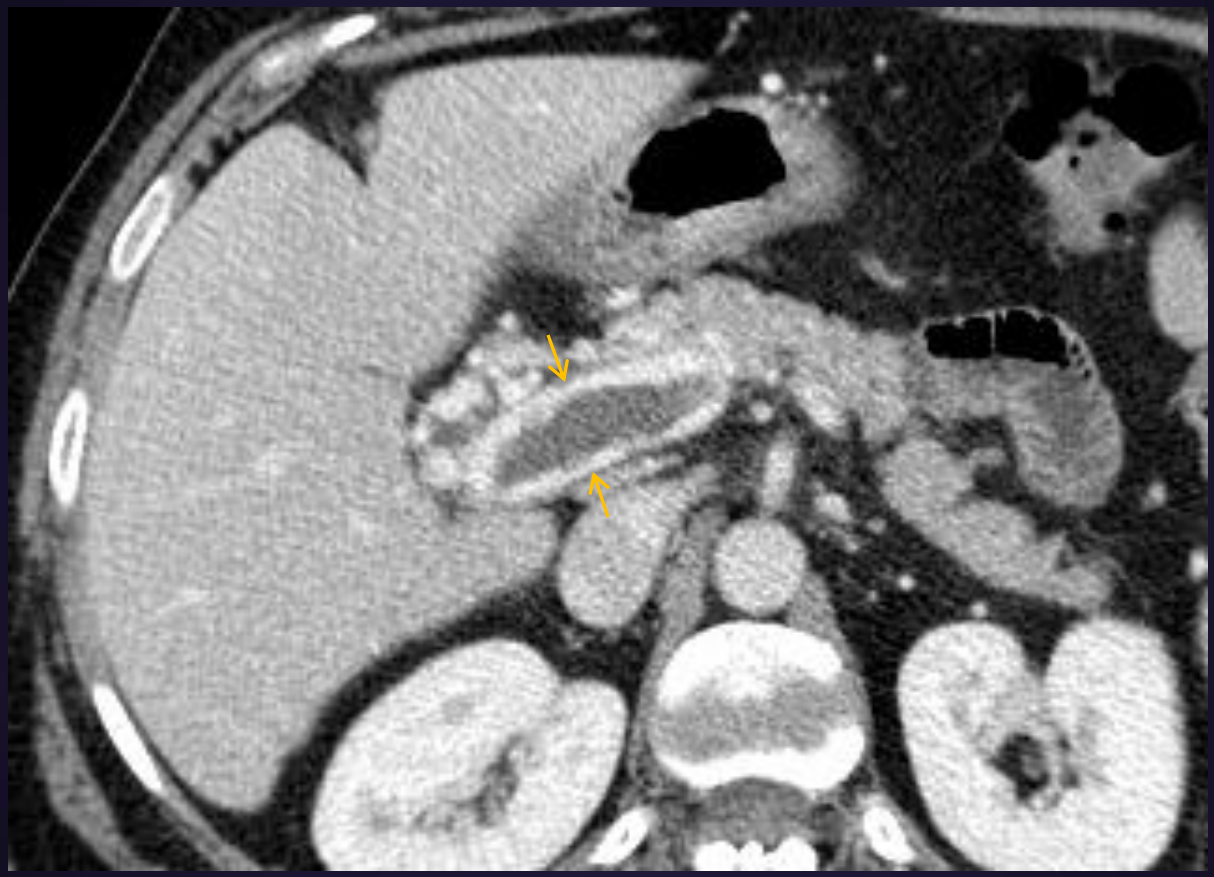
7. PORTAL VEIN THROMBOSIS

- Streaky or diffuse enhancement within the obstructed portal vein indicates the presence of tumor thrombus.
- With chronic portal vein thrombosis numerous small peri-portal collateral veins are often identified – cavernous transformation.

7. PORTAL VEIN THROMBOSIS

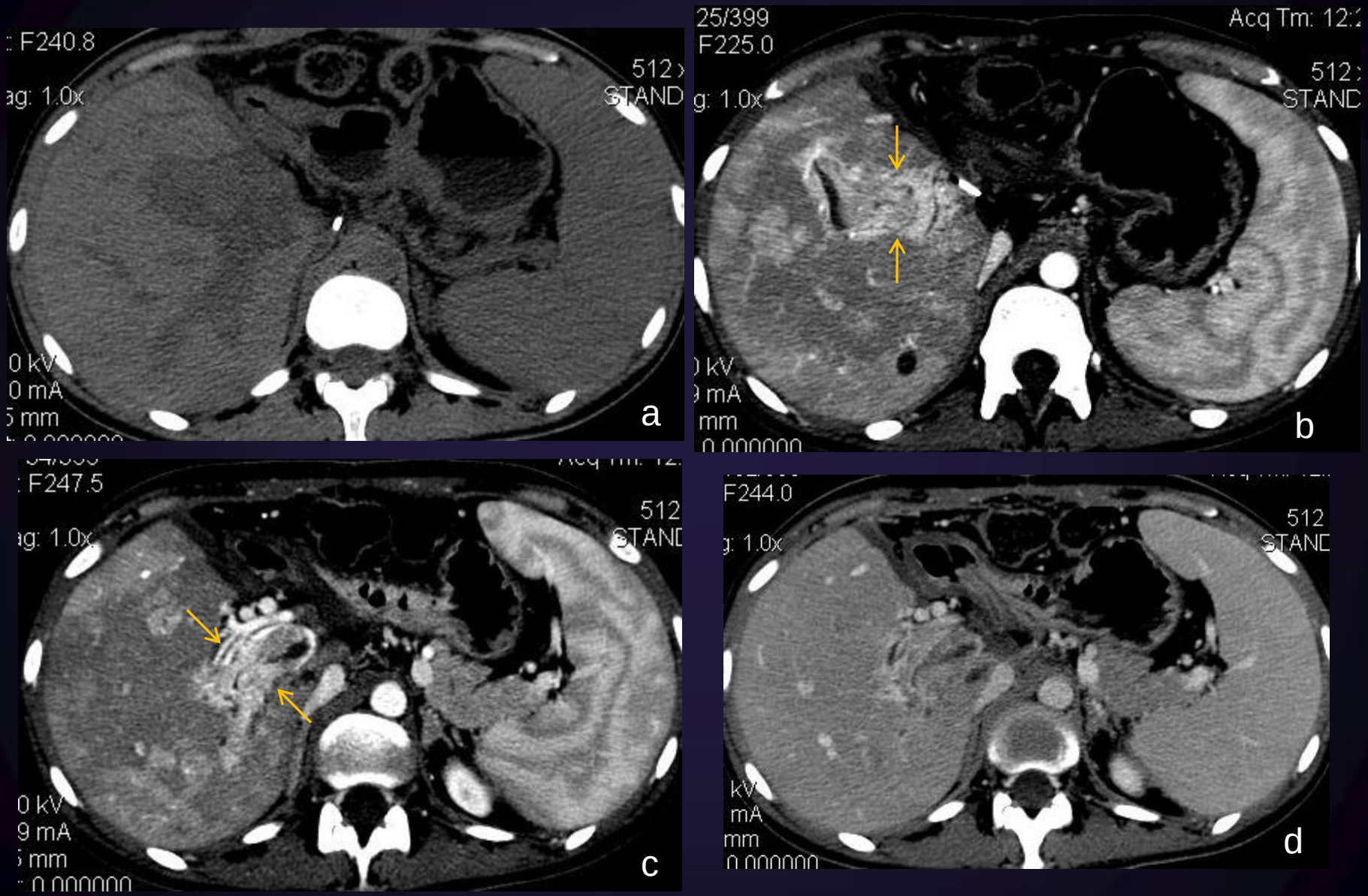
- Indirect CT signs of portal vein thrombosis are related to alterations in hepatic blood supply.
- Portosystemic collateral veins may develop as a consequence of portal venous hypertension.
- Decreased hepatic lobar attenuation on precontrast images is postulated to be due to hepatic glycogen depletion and increased hepatocyte fat content.
- Two flow-related phenomena that can be seen during dynamic contrast-enhanced imaging are THAD and diminished enhancement during the portal venous phase.

7. PORTAL VEIN THROMBOSIS



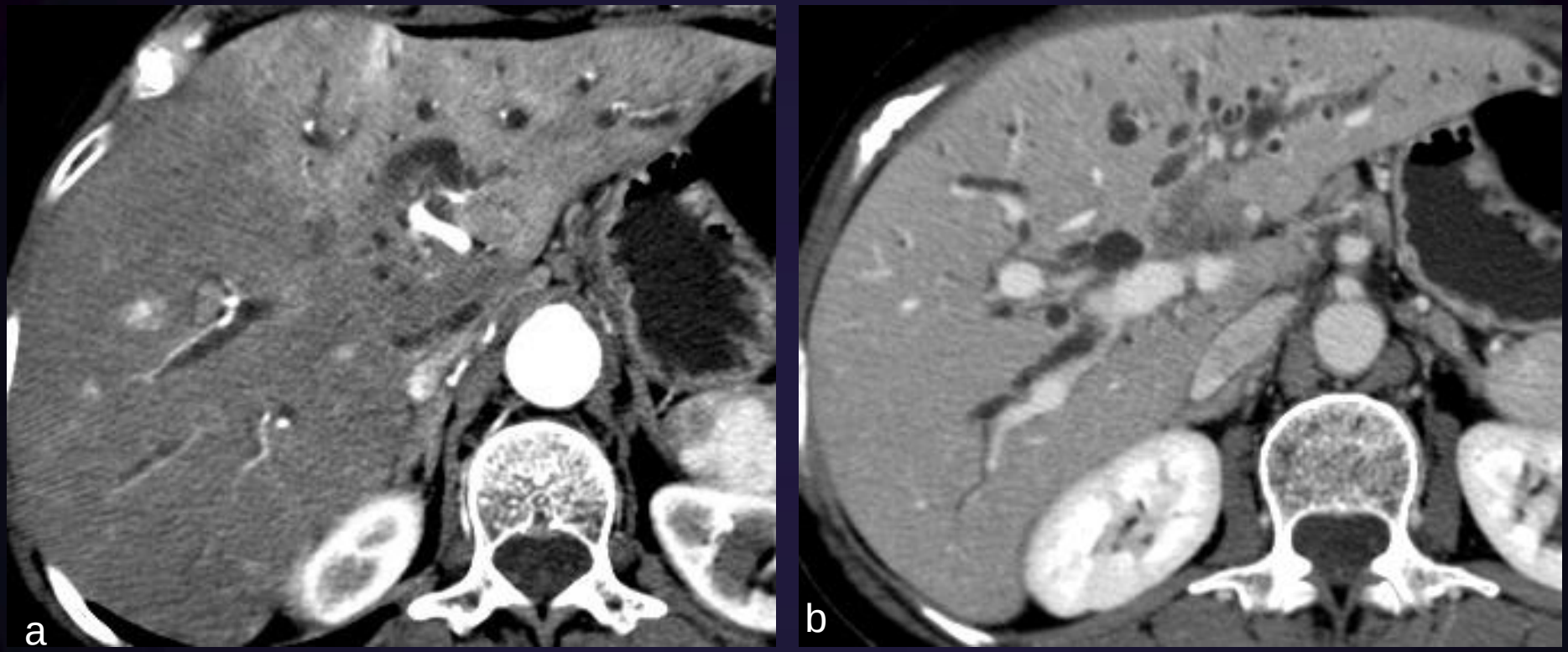
Bland portal vein thrombus.

7. PORTAL VEIN THROMBOSIS



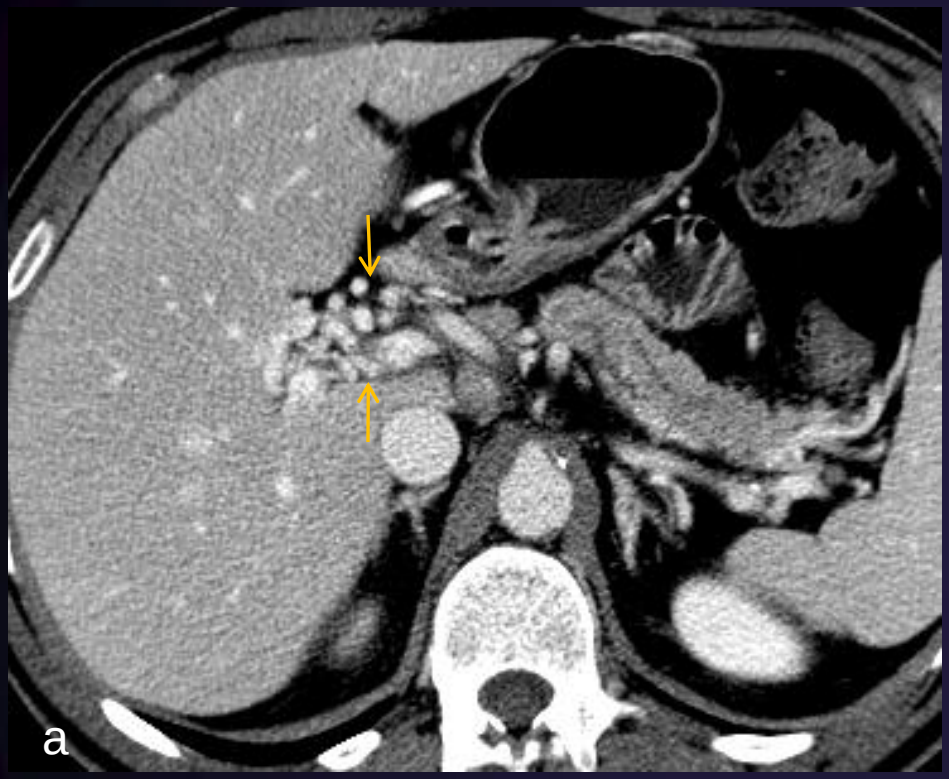
Tumoral thrombus in a patient with recurrent HCC after left hemihepatectomy.

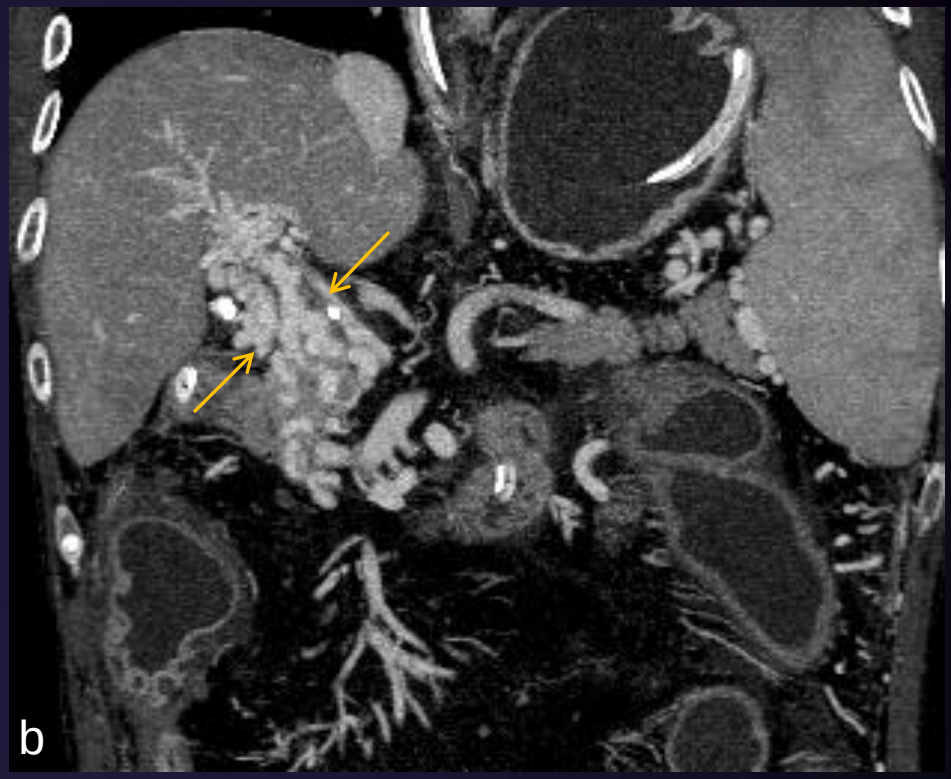
7. PORTAL VEIN THROMBOSIS



THAD due to left portal vein thrombosis in a patient with cholangiocarcinoma.

7. PORTAL VEIN THROMBOSIS





Chronic cavernous transformation of the portal vein.

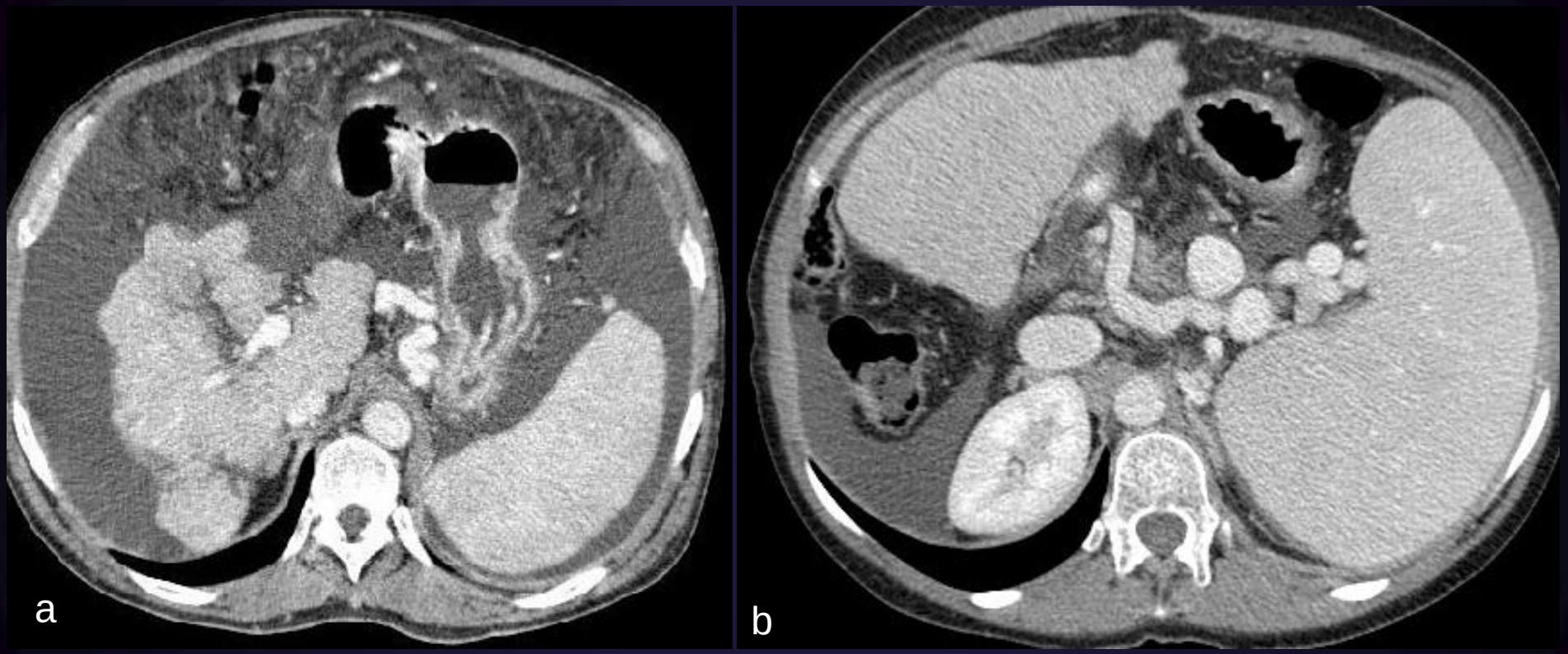
8. PORTAL HYPERTENSION

- 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, congenit, neoplastic).
- The pressure increase induces the formation of portosystemic collaterals which are usually dilated pre-existing vessels, but active angiogenesis is also seen

8. PORTAL HYPERTENSION

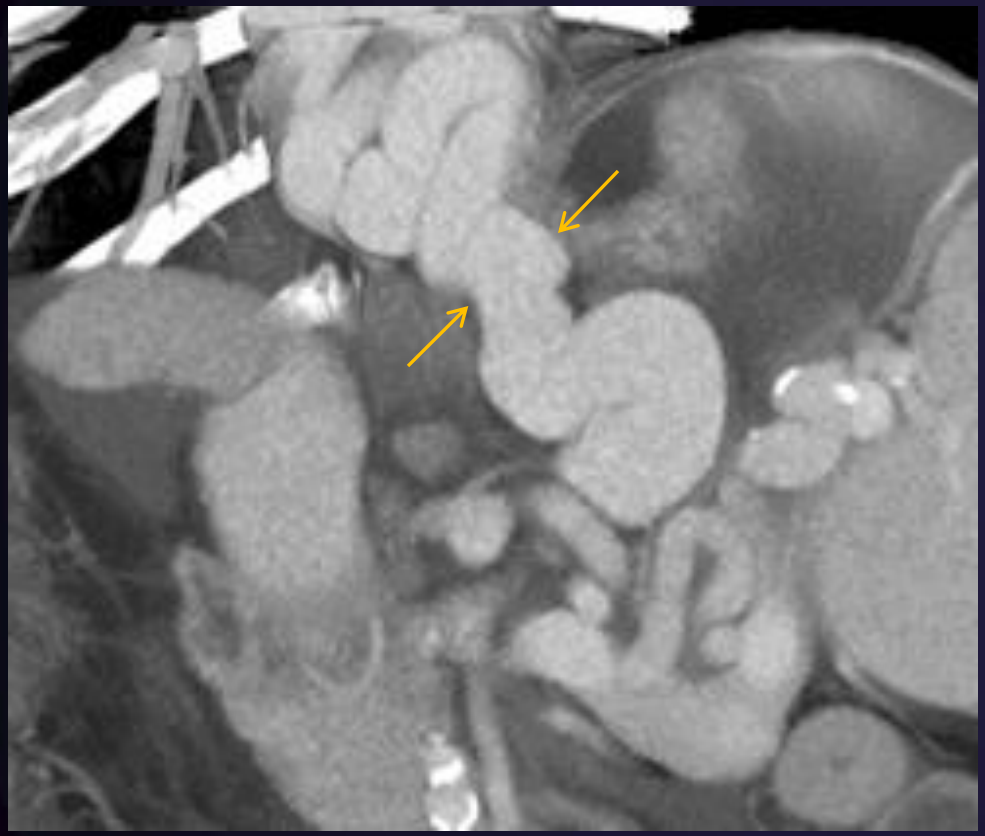
- CT is able to demonstrate the various collaterals.
- Common collateral pathways include:
  - the left gastric, posterior gastric and short gastric veins to the esophageal and paraesophageal veins;
  - gastrosplenic, gastrorenal and splenorenal shunts to the left renal vein
  - several pathways including the omental, paraumbilical, hemorrhoidal and retroperitoneal (Retzius) veins.
- An increased diameter of portal veins (>13 mm) and the superior mesenteric (>10 cmm) with development of paraumbilical collaterals (> 3mm) are highly specific.

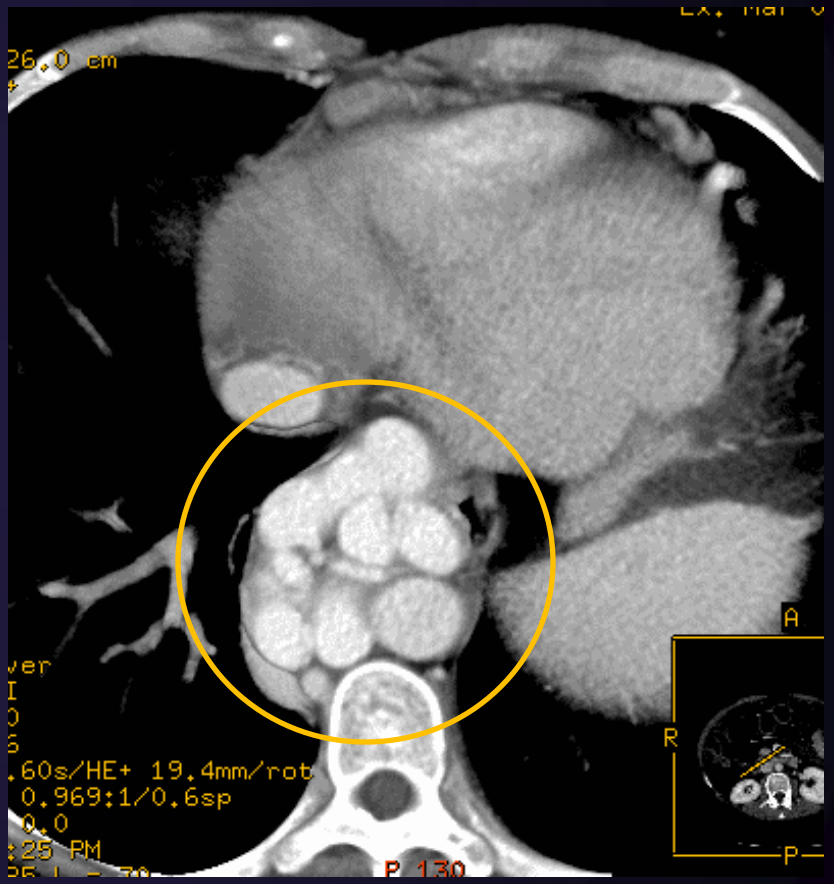
8. PORTAL HYPERTENSION



Contrast-enhanced CT of common collateral pathways in portal hypertension.

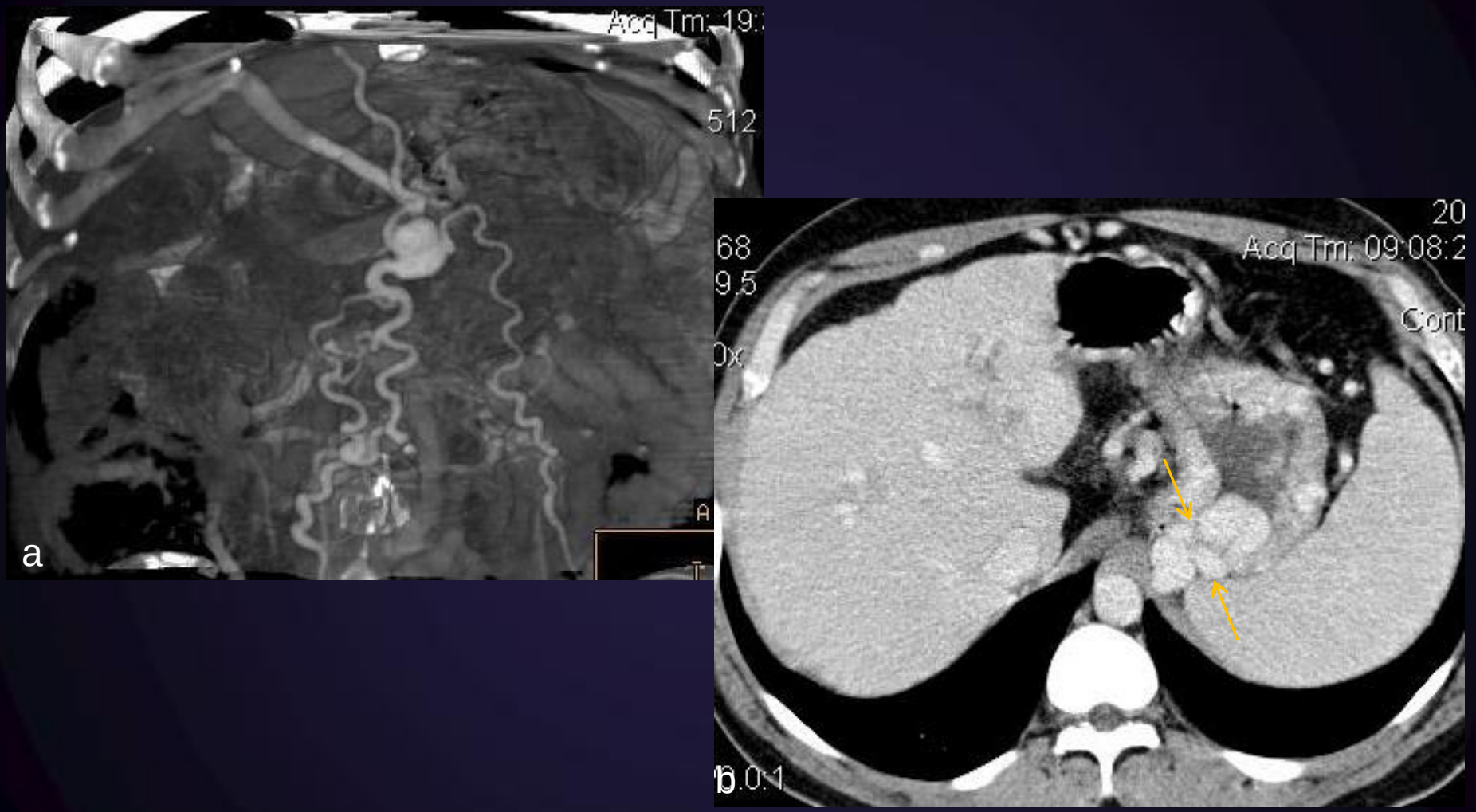
8. PORTAL HYPERTENSION





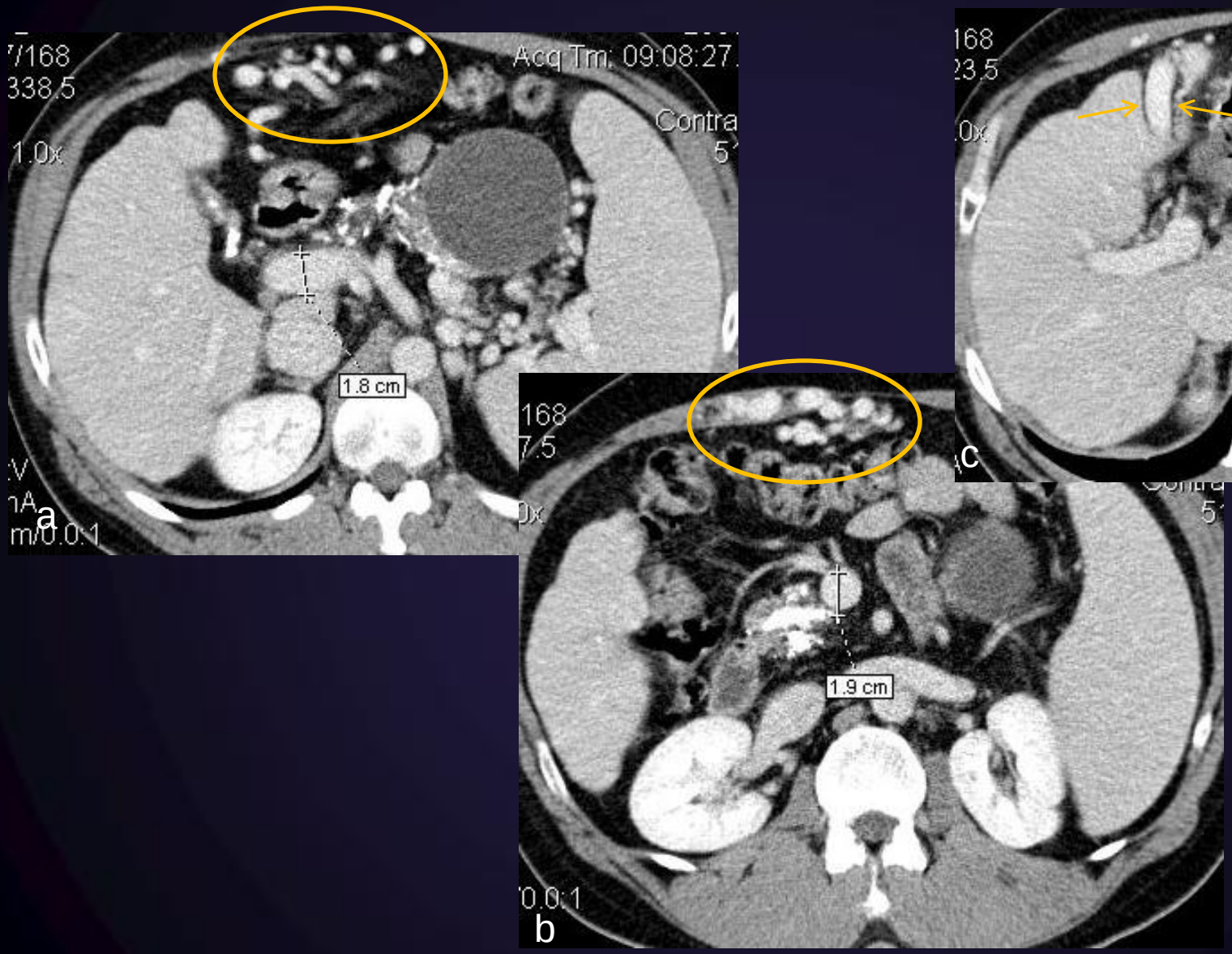
Contrast-enhanced CT of common collateral pathways in portal hypertension.

8. PORTAL HYPERTENSION



Contrast-enhanced CT in portal hypertension.

8. PORTAL HYPERTENSION



CONCLUSIONS

•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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