Hepatic Perfusion Disorders: Computer-Tomographic and Magnetic Resonance Imaging

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Abstract

The liver has a unique dual blood supply from the hepatic artery (25%) and the portal vein (75%). Helical computer tomography (CT) and also magnetic resonance imaging (MRI) are suitable techniques for hepatic imaging. Helical CT and MR angiography allow single breath-hold scanning without motion artifacts. This article illustrates helical CT and MRI findings of different types of hepatic perfusion disorders. Because of rapid image acquisition, three-phase (hepatic arterial phase, portal venous phase and parenchymal phase) CT or MR-angiography evaluation of the hepatic parenchyma is possible, improving perfusion disorders evaluation, tumors detection and characterization in a single study. We classified hepatic perfusion abnormalities in: portal disorders, arterial disorders, hepatic veins abnormalities, intrahepatic vascular communication, hepatic lesions and perfusion disorders and other causes. Differential diagnosis and pitfalls of these entities must be known for a correct diagnosis of focal hepatic lesions.

Key words

Hepatic perfusion disorders - spiral computed tomography (SCT) - magnetic resonance imaging (MRI) - MR angiography

Introduction

Hepatic blood supply comes from the hepatic artery - 25% of flow and the portal vein - 75% of the flow. The arterial and venous supplies to the liver are not independent systems (1). There can be several communications between the vessels, including transsinusoidal, transvasal, transtumoral, and transplexal (peribiliary) routes (2). Transient hepatic parenchymal enhancement (THPE) reflects a change in the normal dual blood supply of the liver. The causes of such perfusion disorders are portal vein obstruction, liver cirrhosis, hepatic neoplasms, hepatic trauma, hereditary hemorrhagic telangiectasia (HHT), hepatic vein obstruction, steal phenomenon by hyper-vascular tumors, inflammatory changes, aberrant blood supply, hepatic parenchymal compression, and other causes (2-4).

We present the spectrum of various hepatic perfusion disorders explored by spiral CT angiography and MR angiography.

J Gastrointestin Liver Dis
September 2006 Vol.15 No.3, 273-279
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Imaging techniques

Unenhanced and enhanced spiral CT of the upper abdomen (collimation: 5-7 mm; pitch: 1-1.5; overlap: 50%) were performed using a monoslice spiral CT. We injected 1.5 ml/kg of nonionic contrast material at a rate of 3-4 ml/sec and began the helical study 20-30 seconds after the start of injection. Another examination was performed 50-60 seconds after the contrast administration to visualize the venous phase, and a third one started 3 or 5 minutes after contrast infusion. MR imaging was performed on a 1.5-T field with a phased-array Torsopa and a respiratory gating; FSE (Fast Spin Echo) T2-weighted, FSPGR (Fast Spoiled Gradient Recalled) T1 breath hold before and after gadolinium injection and 3D FSPGR MR angiography multiphase (arterial, venous and late phase). We injected 0.1 mmol/kg Gd-DTPA at a rate of 2.5-3 ml/sec.

Classification of hepatic perfusion disorders

The classification is shown in Table I.

1. Portal disorders

a. Decrease of portal flow or portal obstruction. Causes of portal flow decrease are: tumoral invasion, compression or surgical ligature (9,10). Perfusion alterations are produced by: increases in arterial flow through transsinusoidal, transvasal, transtumoral, and especially transplexal

(peribiliary) routes to compensate for the diminished portal venous flow (functional APS) and decreased dilution of the contrast material by the non-enhanced portal venous flow. Triphasic helical CT and MR-triphasic angiography can demonstrate: decreased attenuation at CT and T1 hypointensity, T2 hyper/ hypointensity of the affected hepatic parenchyma on MRI images due to edema, depletion of hepatocytes, or fibrosis; transient high attenuation and enhancement during the arterial phase due to increased arterial flow, and portal vein thrombosis (bland or tumor thrombus) or compression (Fig.1).

Table I Classification of hepatic perfusion disorders

1. *Portal disorders*
   a. Decrease of portal flow or portal obstruction
   b. Arterio-portal shunts
2. *Arterial disorders*
   a. Decrease or disparition of arterial blood flow
   b. Increase of arterial blood flow
   c. Hepatic artery aneurysm
3. *Hepatic veins abnormalities*
   a. Budd-Chiari syndrome
   b. Veno-occlusive disease
   c. Right sided heart failure
   d. Pericardial disease
   e. Fibrosing mediastinitis
4. *Intrahepatic vascular communication*
   a. Porto-hepatic shunts
   b. Arterio-hepatic shunts
5. *Hepatic lesions and perfusion disorders*
   a. Liver cirrhosis
   b. Hepatic neoplasms
   c. Hepatic trauma
   d. Hereditary hemorrhagic teleangiectasia
   e. Steal phenomenon by hypervascular tumors
   f. Inflammatory changes
6. *Other causes*
   a. Hepatic parenchymal compression
   b. Hepatic fibrosis
   c. Aberrant blood supply

Causes of portal vein thrombosis are infectious processes (sepsis) neoplasms that invade or compress portal system (hepatoma, pancreatic cancer), hypercoagulation and mieloproliferative disorders. Spiral CT and MRI demons-trate: decreased CT attenuation and hypointensity T1/hyperintensity T2 of the affected liver parenchyma; transient high attenuation at HAP; portal vein thrombosis (bland or tumoral thrombus) or compression at PVP.

b. Arterio-portal shunts. APS is an organic or functional communication between a hepatic arterial branch and the portal venous system, resulting in redistribution of arterial flow into a focal region of portal venous flow (Fig.2). The shunt can occur by several routes: through a macroscopic fistula (usually iatrogenic); transsinusoidal (between microscopic interlobular arterioles and portal venules); transvasal (due to a tumor thrombus); transtumoral (via a draining vein from a hypervascular tumor); transplexal (peribiliary). The causes of APS include hepatic neoplasms such as HCC, hemangioma, and cholangiocarcinoma; hepatic trauma or interventional procedures (Fig.3), liver cirrhosis (2,5). The helical CT and MRI findings of APS are as follows: early enhancement of the peripheral portal vein branches during the arterial phase and before the main portal vein is enhanced; enhancement of the peripheral portal vein branches and main portal vein without enhancement of the superior mesenteric and splenic veins; transient, peripheral, wedge-shaped hepatic parenchymal enhancement usually with a straight margin during the arterial phase (THPE) (2,6-8). Portal phase spiral CT and MRI permit confirmation that the parenchymal lesion has the same attenuation with the surrounding liver.

2. Arterial disorders

a. Decrease or absence of the arterial flow occurs in: arterial stenosis; thrombosis; hepatic infarction.

b. Increase of arterial flow is caused by: hepatic artery aneurysm; Rendu Osler Weber disease;

c. Hepatic artery aneurysm in a context of atherosclerosis; inflammatory pattern; trauma or fibrodysplasia.

Spiral CT and MR 3D angiography with MIP and MPR reconstructions are able to characterise these abnormalities.

Hepatic infarction causes are: systemic disease; HELLP syndrome; post liver transplantation. Spiral CT demonstrates a triangular hypoattenuating area on enhanced HAP and PVP (Fig.4). Sometimes there are hemorrhagic zones visible on nonenhanced scans.

On MRI, the lesions are hypo-T1 and hyperintensity T2 zones with no enhancement after gadolinium injection.

3. Hepatic veins abnormalities

Occlusion of the hepatic veins results in increased sinusoidal pressure and reverses the pressure gradient between the sinusoidal and portal veins (2). The portal vein becomes a draining vein and there is an increase in hepatic arterial flow, resulting in a functional APS, as occurs in liver cirrhosis. Hepatic vein occlusion can be secondary to right-sided heart failure, pericardial disease, Budd-Chiari syndrome, or mediastinal fibrosis. In such cases, arterial CT or MR images demonstrate transient hepatic enhancement in the area of obstructed hepatic venous drainage. The vertex of the wedge-shaped hyperattenuating area points to the inferior vena cava in hepatic vein obstruction. In Budd Chiari syndrome, CT and MRI evidence morphologic and perfusion disorders: atrophy of the liver periphery and hypertrophy of central zones (caudate lobe). After injection: heterogeneous or mosaic pattern; small IVC; absence of hepatic veins enhancement (Fig. 5); portal hypertension may be present.

4.Intrahepatic vascular communication

a. Porto-hepatic shunts may be congenital or acquired (posttrauma, portal hypertension).

b. Arterio-hepatic shunts are found in Rendu-Osler-Weber disease, in hepatic benign and malignant tumors (Figs. 6,7). Hereditary hemorrhagic telangiectasia (Osler-

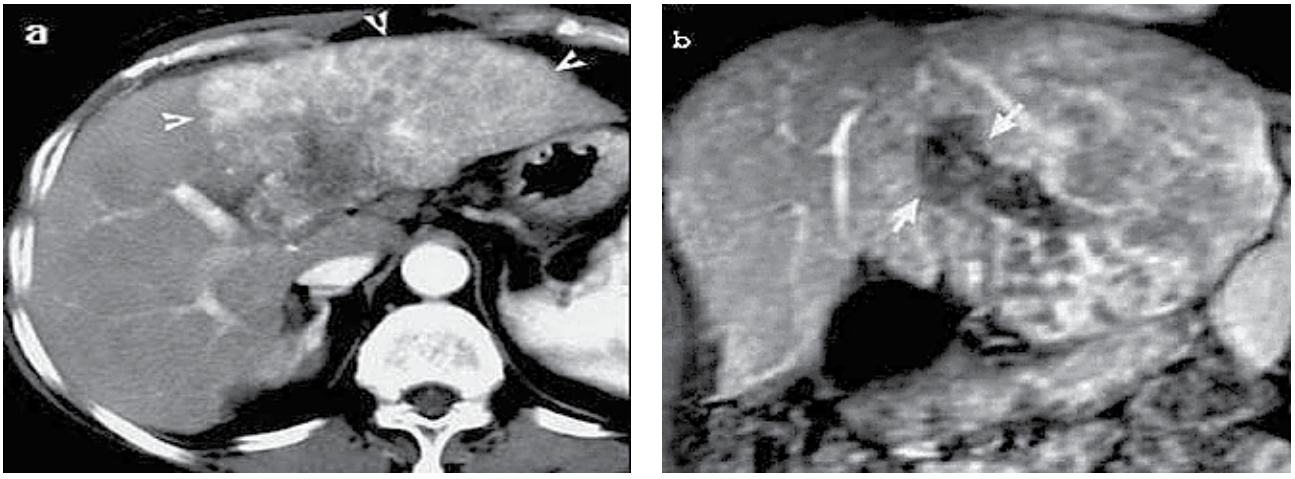

Fig.1 Portal vein obstruction and hepatocarcinoma. (a) arterial helical: total obstruction of left portal vein associated with transient hepatic parenchymal enhancement. (b) coronal reconstruction after 3D MR angiography: better visualization of left portal thrombosis and better delineation of the diffuse type of hepatocarcinoma.

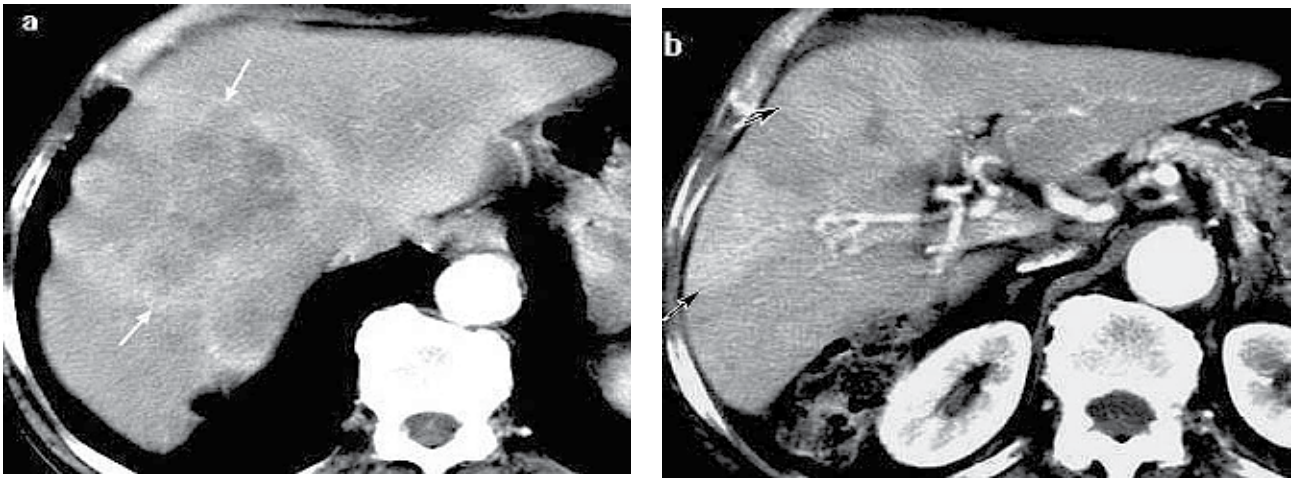

Fig.2 Arterio-portal shunt and hepatocarcinoma. (a), (b)- arterial helical CT: polycyclic hepatic mass (white arrows) and distal peripheral wedge-shaped hepatic parenchymal enhancement (black arrows).

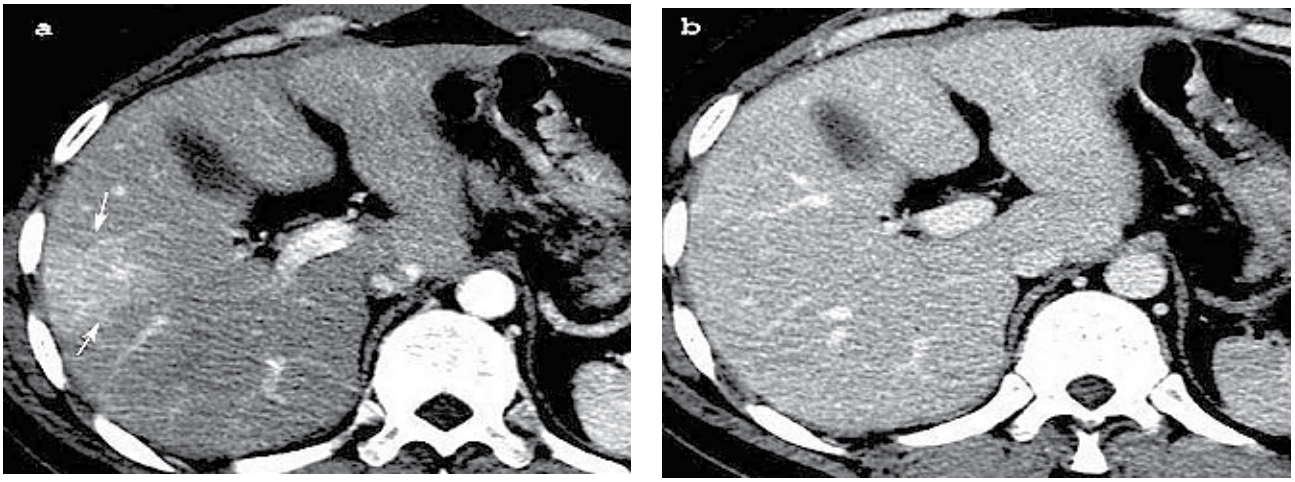

Fig.3 Transient hepatic parenchymal enhancement (THPE) post iatrogenic arterio-portal shunt. (a)-arterial helical CT and (b) portal venous phase; triangular hyperattenuating area (long arrows) on enhanced hepatic arterial phase with normal aspect on portal phase.

Weber-Rendu disease) is a vascular disease with autosomal dominant transmission characterized by multiple telangiectasies, dilated vascular channels with arteriovenous communications. The hepatic perfusion changes, including widened and tortuous hepatic arteries, telangiectasies, and arteriovenous and portovenous fistulas, involve the

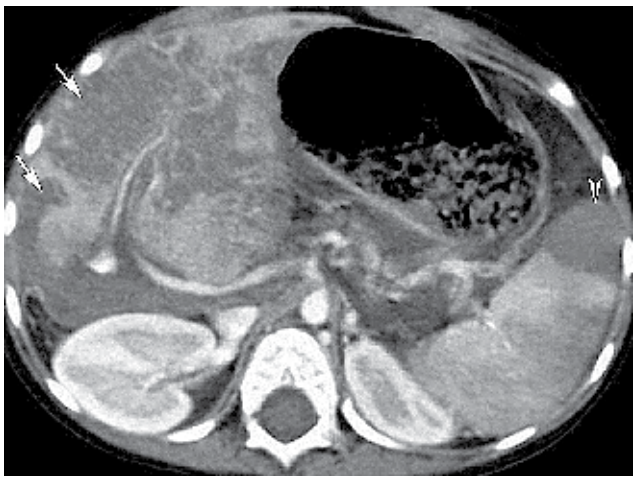

Fig.4 Hepatic infarction - multiple triangular hypoattenuating areas on enhanced hepatic arterial phase (long arrows); splenic infarction (small arrowhead).

simultaneous enhancement of hepatic arteries and veins (11).

5. Hepatic lesions and perfusion disorders

Causes are represented by: inflammatory changes, hepatic neoplasms, liver cirrhosis, hereditary hemorrhagic telangiectasia, hepatic trauma, steal phenomenon by hypervascular tumors. Local inflammation can cause hyperemia (Fig.9) of the hepatic artery and stoppage of regional portal venous flow, as in case of hepatic abscess, acute cholecystitis, cholangitis, and iatrogenic conditions (2). Liver cirrhosis alters normal hepatic blood flow dynamics, resulting in increased arterial flow and decreased portal venous flow to the liver. Hypertrophy of the peribiliary plexus and direct anastomosis in the peripheral area of the liver are the major routes of APS. Abnormal permeability of the sinusoid has also a role in the development of arterioportal communications in the cirrhotic liver (2). The hypervascular tumor can "steal" arterial blood from the surrounding

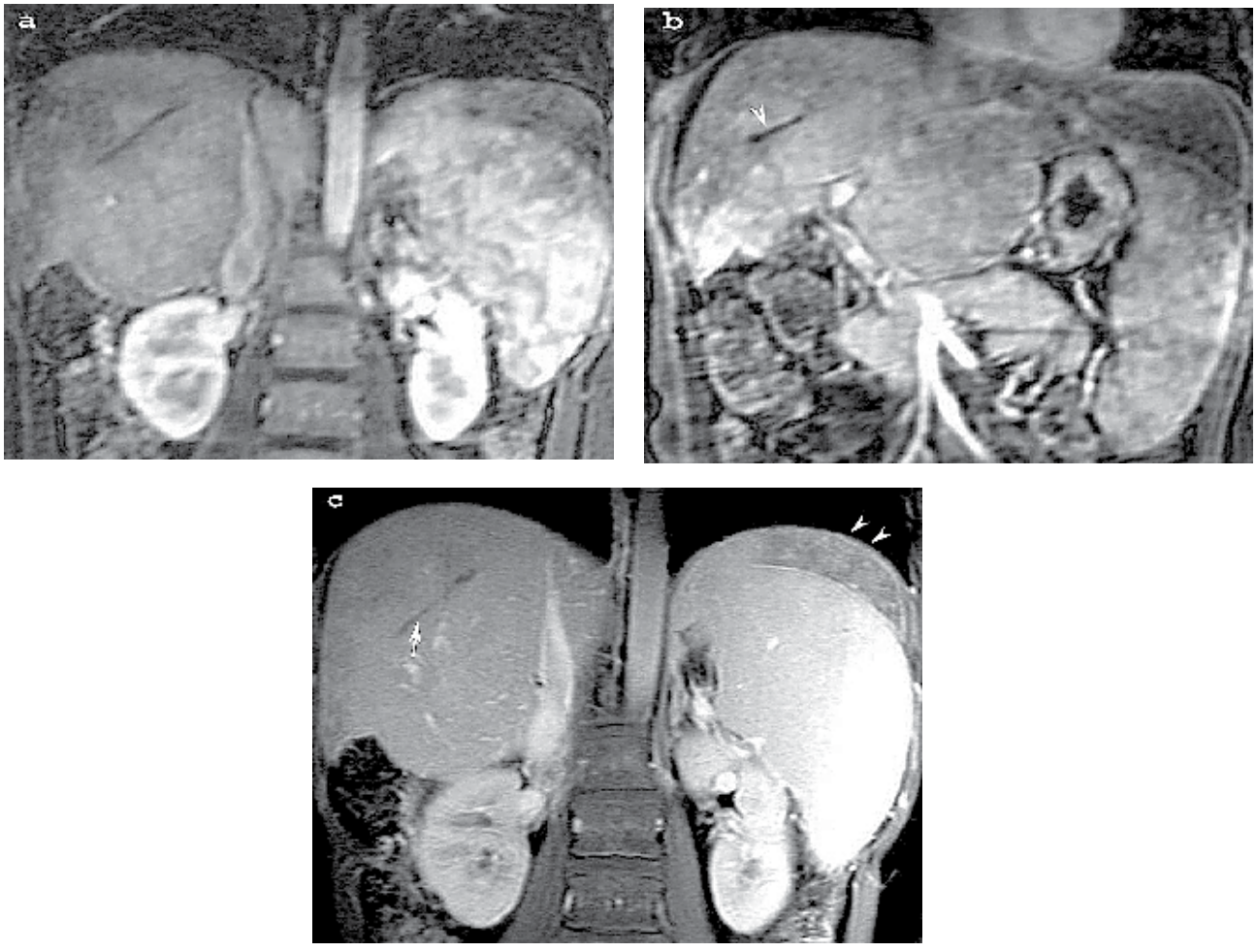

Fig.5 Budd-Chiari syndrome. (a), (b) MIP reconstruction after 3D MRA with Gd-DTPA Obstruction of right and left hepatic veins. Heterogeneous and mosaic pattern of the hepatic parenchymal enhancement in arterial and portal phase. (c) T1 FSPGR late parenchymal phase: thrombosis of right hepatic vein (long arrows); small inferior vena cava; left hepatic lobe infarction (arrow head).

extrahepatic and intrahepatic vessels (Fig.8). Helical CT and better, multislice CT demonstrate hepatic involvement as arterial dilatation and tortuosity, hepatomegaly, hepatic vein dilatation, and, when arteriovenous shunts are present, parenchyma (Fig.10), which then appears hypoattenuating on arterial phase images relative to the contralateral lobe of the liver (2,10).

Abdominal trauma and interventional procedures some-

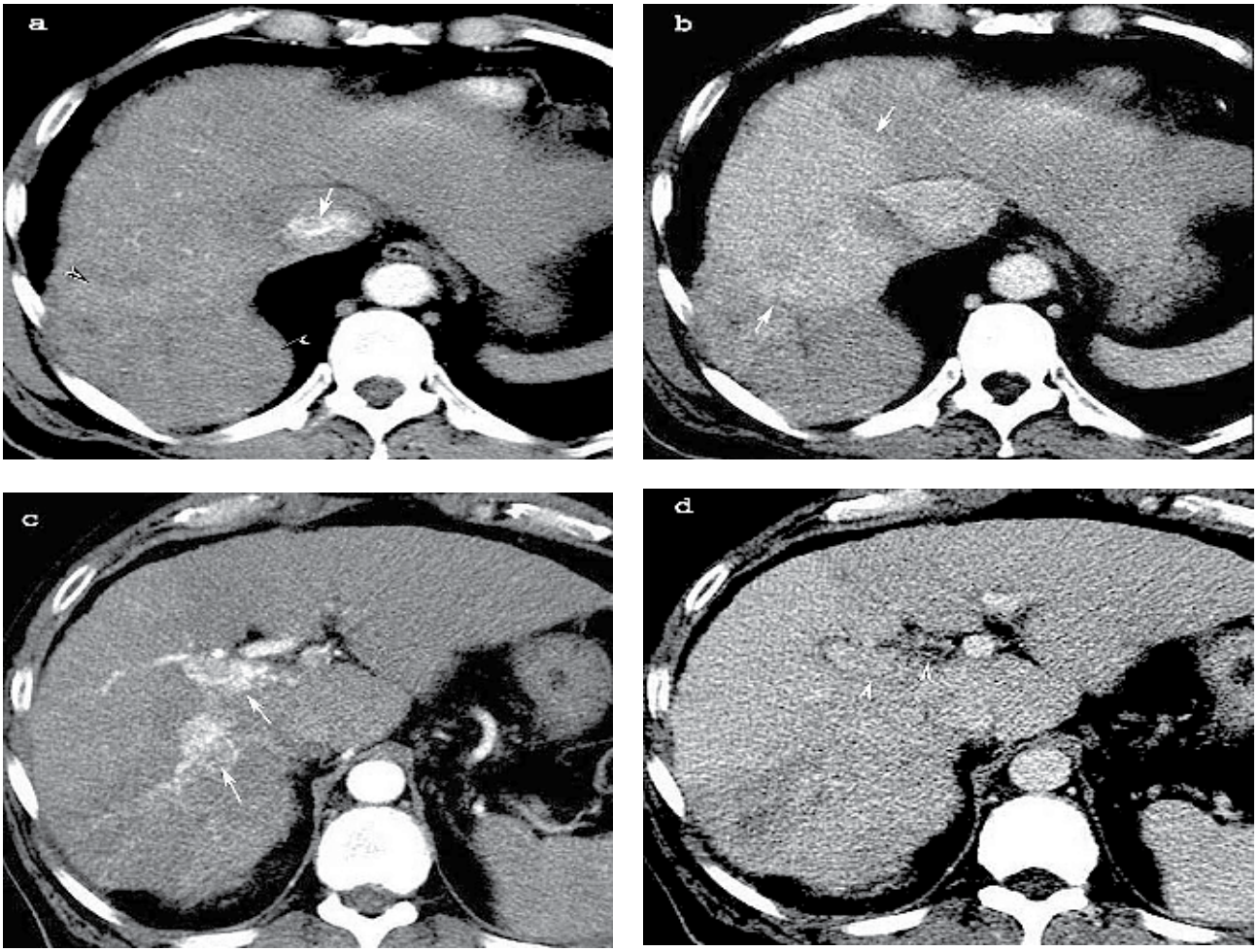

Fig.6 Right hepatocarcinoma, arterio-hepatic shunts (long white arrows) and right portal venous thrombosis with extension of the thrombus into the portal trunk (white arrow head). a, c - arterial and b, d - portal spiral CT phase.

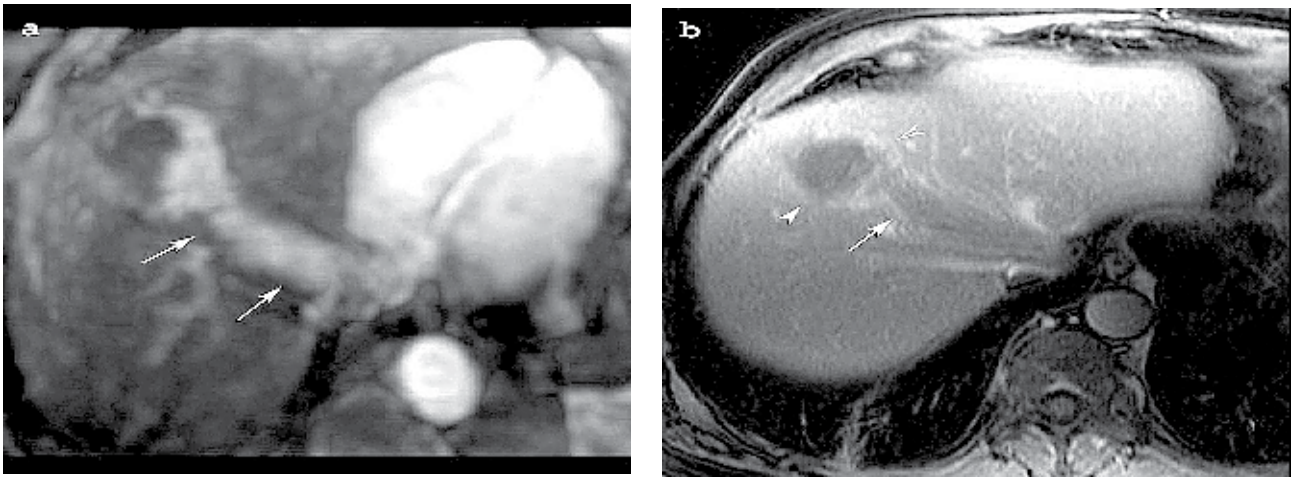

Fig.7 Hepatocarcinoma with arterio-hepatic shunt and tumoral thrombosis of middle hepatic vein and inferior vena cava. (a)-MIP arterial phase: tumoral thrombus (long arrows); (b) T1 FSPGR portal phase-tumoral hepatic nodule (arrow head).

times cause an organic communication between the arterial and portal venous systems or produce functional APSs due to portal vein injury, such as portal vein thrombosis. CT and MRI: high attenuation area on HAP, image returns to normal in PVP.

6. Other causes of hepatic perfusion disorders are: hepatic parenchymal compression (ribs-pseudolesions in segments V and VI); perihepatic peritoneal lesions; aberrant blood supply; portal hypertension; liver fibrosis. Rib compression, perihepatic peritoneal implants, pseudomyxo-

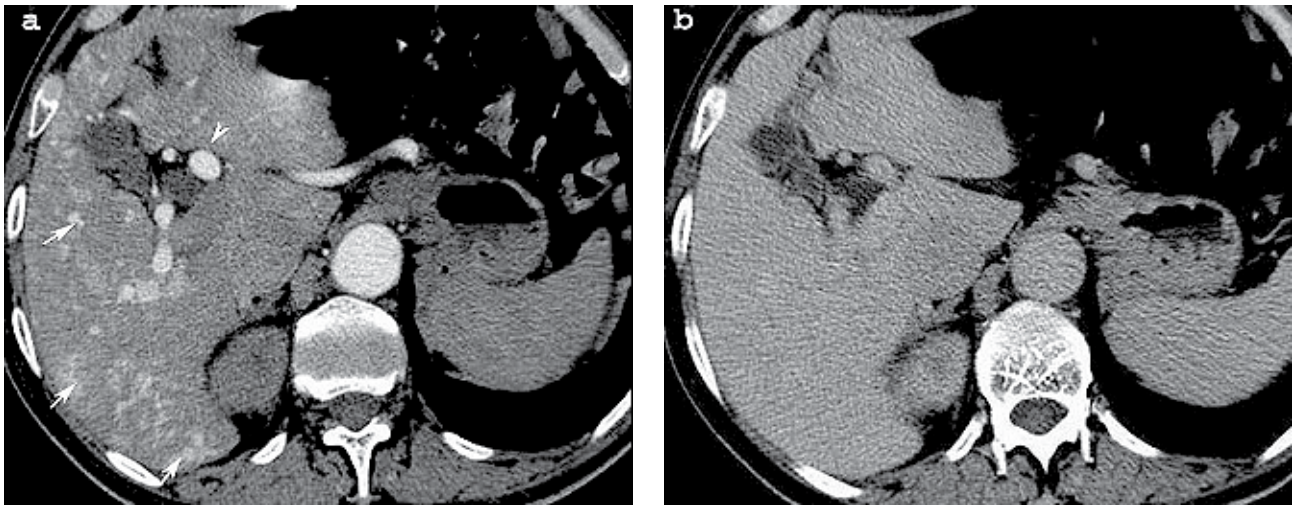

Fig.8 Hereditary hemorrhagic telangiectasia. a - CT arterial phase; b - portal venous phase: tortuous hepatic arteries and arteriovenous and portovenous fistulas.

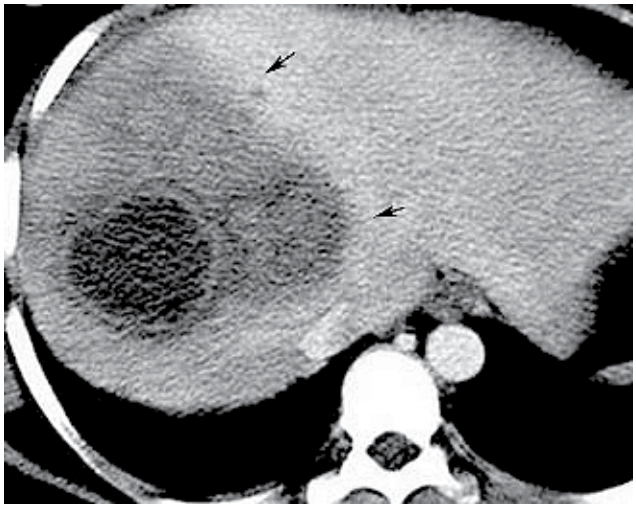

Fig.9 Hepatic abscess and adjacent hyperemia. enhanced helical CT: high-attenuation area around the abscess.

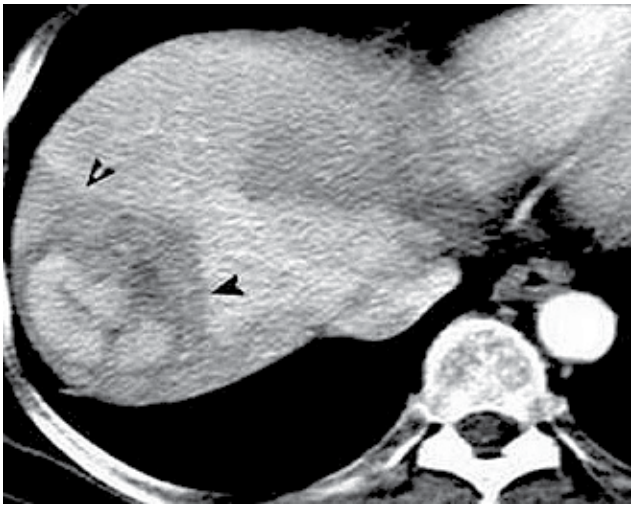

Fig.10 Hepatocarcinoma with steal phenomenon. Arterial phase: hypoattenuating zone surrounding the tumor (short arrows).

ma peritonei, and perihepatic fluid collections are examples of potential focal hepatic parenchymal compression that can result in an area of decreased portal venous perfusion. When third degree hepatic inflow tracts (capsular veins, accessory cystic vein, or aberrant right gastric vein) are present, systemic venous blood drains into the hepatic sinusoids. Common locations are adjacent to the gallbladder fossa, anterior to the porta hepatis, adjacent to the falciform ligament, and in the subcapsular area. Confluent fibrosis in liver cirrhosis can decrease portal venous flow and slightly increase arterial flow, resulting in an irregular and ill-defined hyperattenuating area on arterial phase images that becomes isoattenuating on PVP images.

Conclusions

The knowledge concerning physiology and pathophysiology of liver hemodynamics is crucial to diagnose perfusion disorders and to avoid the false-positive diagnosis of liver pseudolesions. Spiral CT and MR-angiography with its ability for three-phase contrast-enhanced studies, can demonstrate alterations in the dynamics of hepatic blood flow. Combined use of SCT in arterial phase and portal phase is considered the most sensitive imaging technique to detect focal hepatic lesions and pseudolesions caused by hemodynamic changes. In particular cases, MRI using specific contrast enhancement offers more information regarding the characterization of liver mass associated with hepatic perfusion abnormalities.

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