CLINICAL IMAGING

CT and MRI of Acquired Portal Venous System Anomalies

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Abstract

In this educational presentation, we offer an overview of acquired anomalies of the portal venous system explored by biphasic helical CT and MRI. Portosystemic collateral vessels, cavernous transformation of the portal vein, intrahepatic vascular shunts, aneurysms of the portal venous system, thrombosis of the portal venous system, and gas in the portal venous system will be discussed. For liver surgery and interventional procedures it is necessary to have a correct mapping of normal anatomy, variants, and different pathologies involving the portal venous system.

Key words

Acquired anomalies - portal venous system - helical computed tomography - magnetic resonance imaging MRI - MR angiography

Introduction

The portal venous system comprises all the veins draining the abdominal part of the digestive tract, including the lower esophagus but excluding the lower anal canal. Tributaries of the portal vein are the splenic, superior mesenteric, left gastric, right gastric, paraumbilical, and cystic veins.

Imaging evaluation of the portal venous system is usually performed with color Doppler ultrasonography, spiral computed tomography (CT), and magnetic resonance imaging (MRI) (1). Invasive techniques such as arterial portography, direct portography, and splenoportography may also be used, but in our days these methods are replaced by MR portography (1).

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Imaging details

Biphasic dynamic contrast material–enhanced helical CT is a useful tool for assessing abnormalities of the portal venous system. Most perfusion alterations are seen during the hepatic arterial phase (HAP), with normal attenuation in the portal venous phase (PVP).

Our protocol for biphasic helical CT of the abdomen is: we inject 100 mL of iodinated contrast material at a rate of 3–4mL/sec. HAP images are acquired 20–25 seconds after the start of the injection, and PVP images are acquired 25–35 seconds later.

MR evaluation was performed with a 1.5-T high-performance–gradient MR imaging system and a phased array torso coil. The following pulse sequences were used: T2-weighted fast spin-echo fat-suppressed sequence with a respiratory trigger, T1-weighted gradient echo sequence and 3D MR multiphase angiography fast spoiled gradient echo dynamic fat-suppressed sequence after the administration of contrast material.

Portal hypertension results from a relative or absolute obstruction of the splanchnic blood flow or, less commonly, from increased portal blood flow. Normal portal pressure is 5-10 mm Hg. Portal hypertension is present if portal venous pressure exceeds 10mmHg (1). Portal hypertension can be classified into presinusoidal, sinusoidal and postsinusoidal causes.

The obstruction of hepatoportal flow leads to the development of numerous collateral pathways from the high-pressure portal system to the low-pressure systemic circulation.

Portosystemic collateral vessels

The most common cause of portosystemic collateral vessels is portal hypertension. Other causes of their presence are splenic or splenomesenteric venous compression and obstruction due to neoplasms, pancreatitis, or surgery.

Coronary, esophageal, paraesophageal, and gastric collateral vessels

Esophageal varices are of major clinical importance

because they are a frequent source of gastrointestinal bleeding (1/3). Endoscopy is more sensitive than CT to the diagnosis of esophageal varices (Fig.1). Paraesophageal collateral vessels are located outside the walls of the esophagus (Fig.2). Gastric varices are located at the posterosuperior aspect of the gastric fundus (Fig.3) and may simulate a gastric neoplasm on nonenhanced CT scans. Most gastric varices drain into the esophageal or paraesophageal veins, but occasionally they drain into the left renal vein (1).

Paraumbilical collateral vessels

Paraumbilical collateral vessels are next in frequency; their extent was usually underestimated with conventional angiography alone until the advent of cross-sectional imaging. Numerous paraumbilical vessels can arise from the left portal vein in patients with cirrhosis (2,3). The most common pattern of drainage of paraumbilical veins is through the epigastric veins into the external iliac veins. Paraumbilical veins can also connect with subcutaneous vessels of the anterior abdominal wall, creating the caput medusae: a varicose dilatation of subcutaneous veins around the umbilicus (Fig.4).

Splenorenal collateral vessels

Collateral vessels from the splenic hilum to the left renal vein are fairly common. They are desirable spontaneous shunts in portal hypertension because they are not associated with gastrointestinal bleeding . A common feature depicted at cross-sectional imaging is an enlarged left renal vein and dilatation of the inferior vena cava at the level of the left renal vein in the presence of a splenorenal shunt (Fig.5).

Mesenteric collateral vessels

Inferior mesenteric collateral vessels are less frequent than the collateral vessels mentioned earlier but are of great importance because of their association with rectal bleeding. The portal venous system (superior hemorrhoidal vein) and the systemic venous circulation (middle and inferior hemorrhoidal veins) connect via the hemorrhoidal plexus (3,4,5). Mesentericorenal collateral vessels (Fig.6) between the superior mesenteric vein and the right and left renal veins are the least frequent mesenteric shunts.

Other collateral pathways

Rare collateral pathways have been reported, including pleuropericardial-peritoneal, splenoazygos, intrahepatic, and from the coronary or splenic veins to the inferior pulmonary vein (Fig.7) or to diaphragmatic veins. Portoportal and portosystemic collateral vessels also develop if there is occlusion of the splenic vein. In occlusion of the superior mesenteric vein, mesenteric varices and mesentericorenal collateral vessels develop (1).

Cavernous transformation of the portal vein

Cavernous transformation of the portal vein consists of formation of venous channels within and around a previously stenosed or occluded portal vein that act as portoportal collateral vessels (1,6,7).

On contrast-enhanced CT scans, a characteristic beaded appearance (mass of veins) at the porta hepatis is the most frequent finding (Fig.8). Intrahepatic extension of the cavernous transformation and involvement of intrahepatic branches with a normal-appearing main portal vein have also been described (Fig.9).

Portosystemic shunts

Direct communication between a portal vein and a hepatic vein is uncommon. The most frequently reported intrahepatic portosystemic shunt occurs between the right portal vein and the inferior vena cava. Acquired conditions of intrahepatic portosystemic collateral vessels are seen in cirrhotic patients and trauma (1,8-10).

Helical CT scans obtained during the PVP show a communication between a portal vein branch and the hepatic vein, as well as early and asymmetric enhancement of the hepatic vein.

Arterioportal shunts

Acquired arterioportal shunts may be found in cirrhosis, post liver biopsy, trauma, or liver neoplasms and consist of a communication between the hepatic artery and the portal venous system. They appear as small, wedge-shaped, peripheral or subcapsular areas of increased attenuation with early portal venous filling on HAP CT scans or hepatic arteriograms and demonstrate normal attenuation during the PVP (Fig.10).

Arteriosystemic shunts

The rarest form of an intrahepatic shunt is a communication between the hepatic artery (or other systemic arteries) and the hepatic veins. Such shunts have been reported in hepatocarcinoma, and large hemangiomas (11,12).

Contrast-enhanced CT performed during the HAP shows increased asymmetric and early enhancement of a hepatic vein (Fig.11).

Aneurysms of the portal venous system

Aneurysms of the portal vein were once thought to be extremely rare but nowadays are well documented and not unusual. They represent only 3% of all aneurysms of the venous system (Fig.12). Although aneurysms of the portal venous system may be present in patients with liver disease, an overwhelming majority of patients do not have portal hypertension or chronic liver disease (1,3).

Thrombosis of the portal venous system

Portal vein thrombosis occurs in various clinical settings, with the most common being liver cirrhosis. Venous system thrombosis occur in infectious diseases (eg, sepsis, cholangitis, pancreatitis), neoplasms, hypercoagulable states, myeloproliferative disorders, surgery, and embolism from a thrombus located in the superior mesenteric or splenic vein. Nonenhanced CT may show focal high attenuation in the portal, superior mesenteric, or splenic vein and venous enlargement when thrombosis is acute. Chronic venous thrombosis can manifest as linear areas of calcification within the thrombus (1,3).

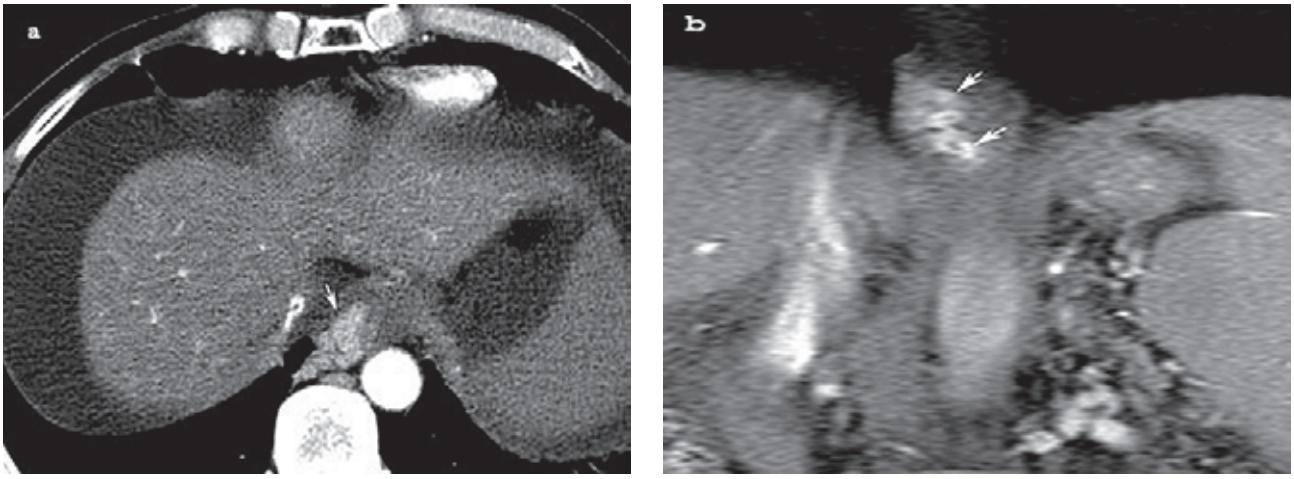

Fig.1 Esophageal varices (arrows). a - CT postcontrast evaluation, b- MRI evaluation T1 postGd enhancement.

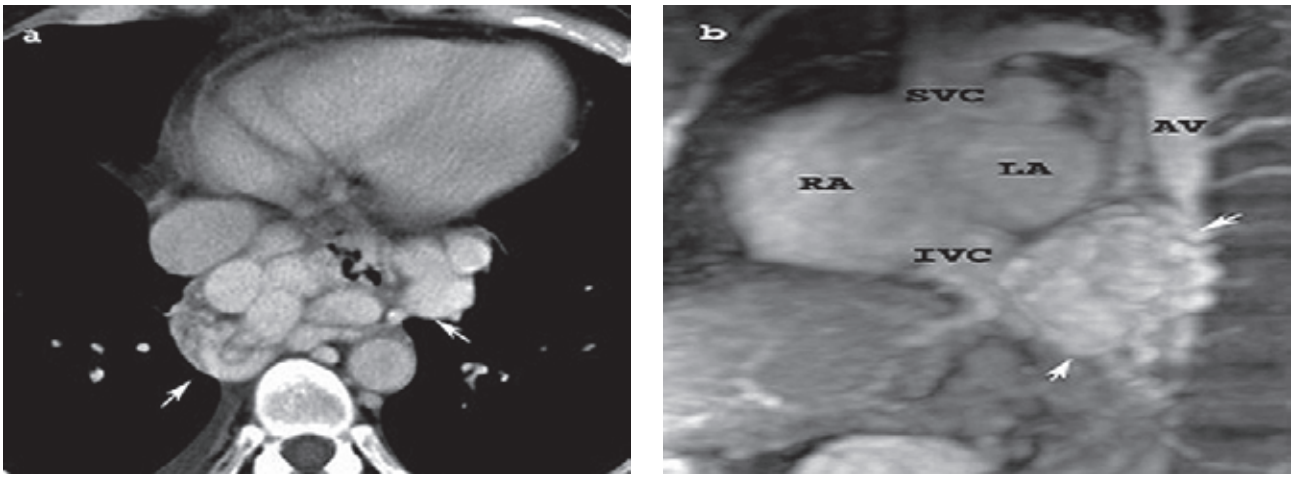

Fig.2 Paraesophageal varices (arrows). a - CT postcontrast evaluation, b- MRI evaluation - sagittal MIP reconstruction; RA-right atrium, LA-left atrium, IVC-inferior vena cava; SVC-superior vena cava, AV-azygos vein.

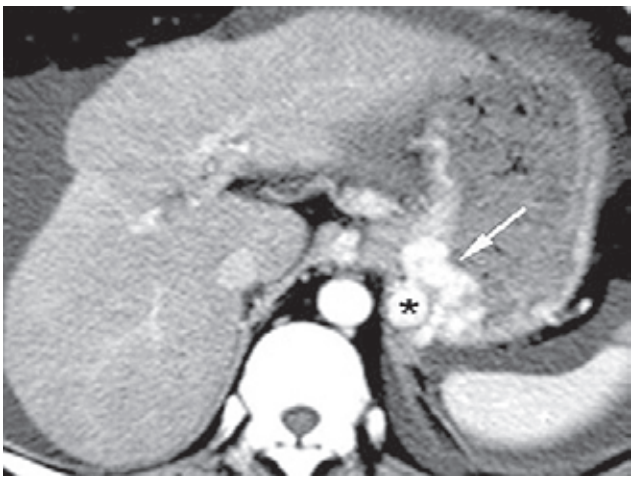

Fig.3 Gastric varices (arrow) - CT postcontrast evaluation.

Contrast-enhanced helical CT demonstrates a filling defect partially or totally occluding the vessel lumen (Figs.9,10). Rim enhancement of the vessel wall may also be seen and is presumed to be due to normal flow in the vasa vasorum. Indirect signs of portal vein thrombosis are the presence of cavernous transformation of the portal vein and the presence of portosystemic collateral vessels and arterioportal shunts. Care must be taken to avoid confusion of the "pseudothrombus image" with a true portal vein thrombus. The pseudothrombus appearance occurs during the HAP in the main portal vein lumen and is due to mixed flow from the enhanced splenic vein return and the nonenhanced superior mesenteric vein return. Two types of perfusion anomalies have been reported in portal vein thrombosis (1,11):

a) a transient hepatic attenuation difference during the late HAP: this anomaly appears as an increase in the attenuation of segments poorly perfused by the portal vein and is due to an increase in arterial inflow.

b) diminished enhancement during the PVP due to locally decreased portal vein perfusion.

Gas in the portal venous system

Traditionally, the presence of gas in the portal venous system was interpreted as an ominous sign in the clinical setting of mesenteric ischemia in adults or necrotizing enterocolitis in infants. It was a surgical emergency, with a mortality rate of 75–90%. Nowadays, mortality rates

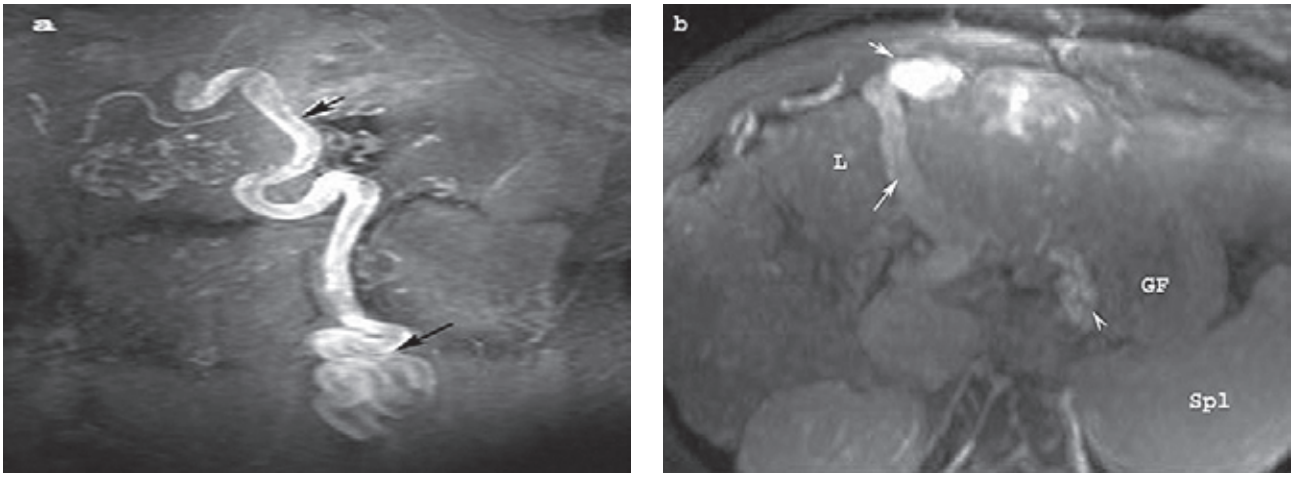

Fig.4 Umbilical vein recanalisation - MRI evaluation. a- coronal MIP reconstruction (black arrows), b- axial MIP reconstruction (white arrows). L-liver, Spl-spleen, GF-gastric fornix.

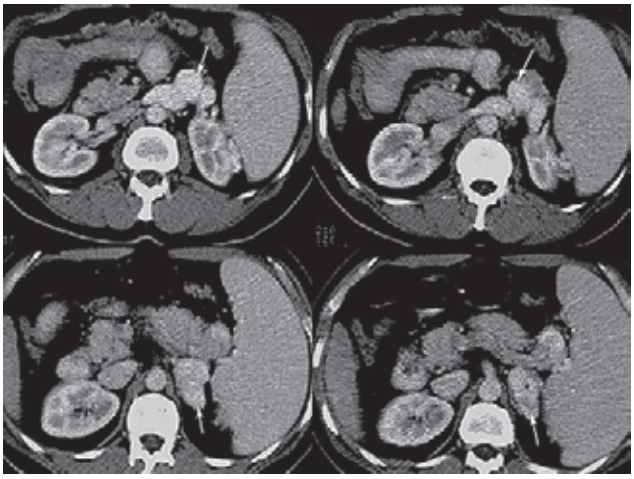

Fig.5 Portal hypertension. Spleno-renal shunt (arrows) - CT evaluation.

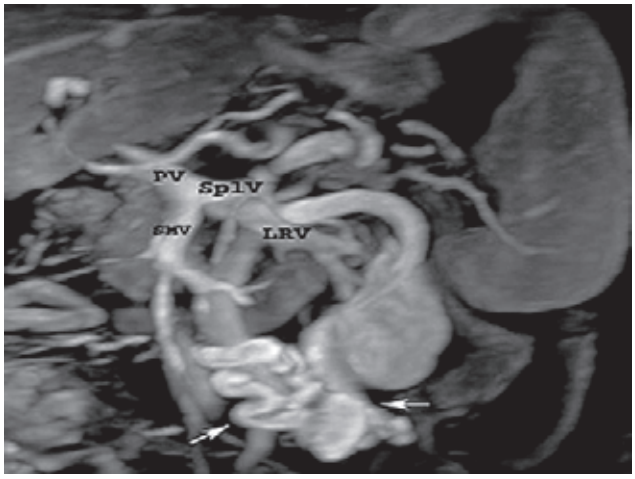

Fig.6 Meseterico-renal collateral vessels (arrows) - MRI- postGd MIP reconstruction. SMV-superior mesenteric vein, PV-portal vein, LRV-left renal vein, Spl V- splenic vein.

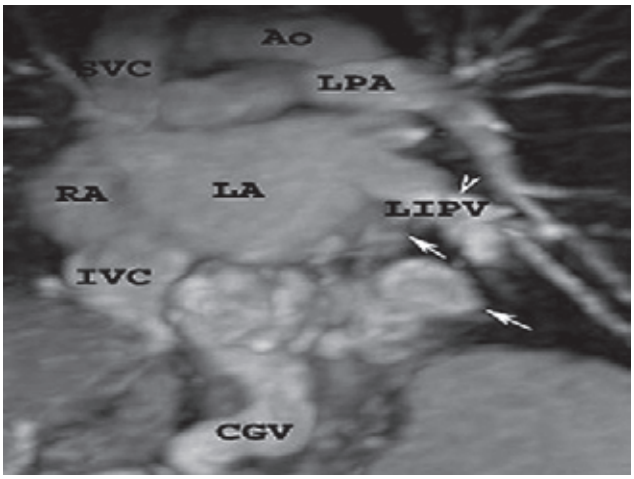

Fig.7 Portal hypertension. Communication with LIPV-left inferior pulmonary vein (arrow). LPA- ;eft pulmonary artery, Ao-aorta.

associated with portal venous system gas have declined to 29%–43%. This decline is due not to improved therapy but rather to new and better imaging techniques, which have allowed recognition of an increasing number of causes of gas in the portal venous system (1). The accessibility to CT units has increased the sensitivity for detection of portal venous system gas. Reported causes of portal venous system gas are necrotizing pancreatitis, abdominal abscess, intestinal obstruction, perforated gastric ulcer or carcinoma, diverticulitis, inflammatory bowel disease, abdominal trauma, ingestion of a caustic agent, enema administration, colonoscopy, gastrostomy tubes, and liver transplantation.

Intrahepatic portal vein gas should be differentiated from aerobilia. The distribution of hepatic gas in patients with aerobilia is central, around the portal hilum, and does not extend to within 2 cm of the liver capsule (Fig.12).

Gas in the mesenteric vein branches should be differentiated from pneumoperitoneum.

Conclusion

Biphasic helical CT is a useful tool for evaluation of

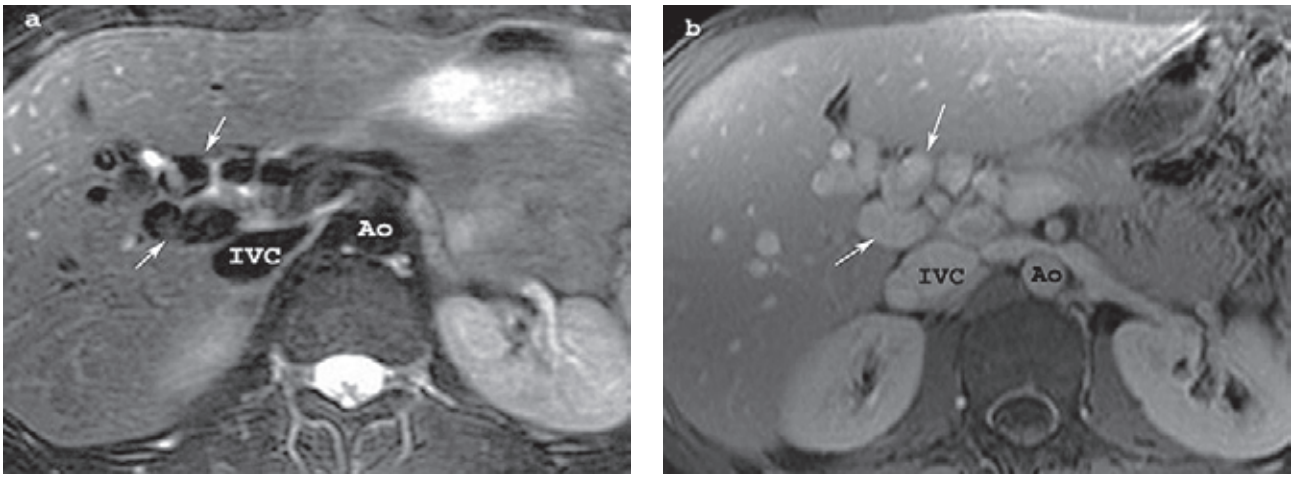

Fig.8 Portal cavernoma (arrows). MRI evaluation, a-T2 weighted, b-T1 postGd enhancement.

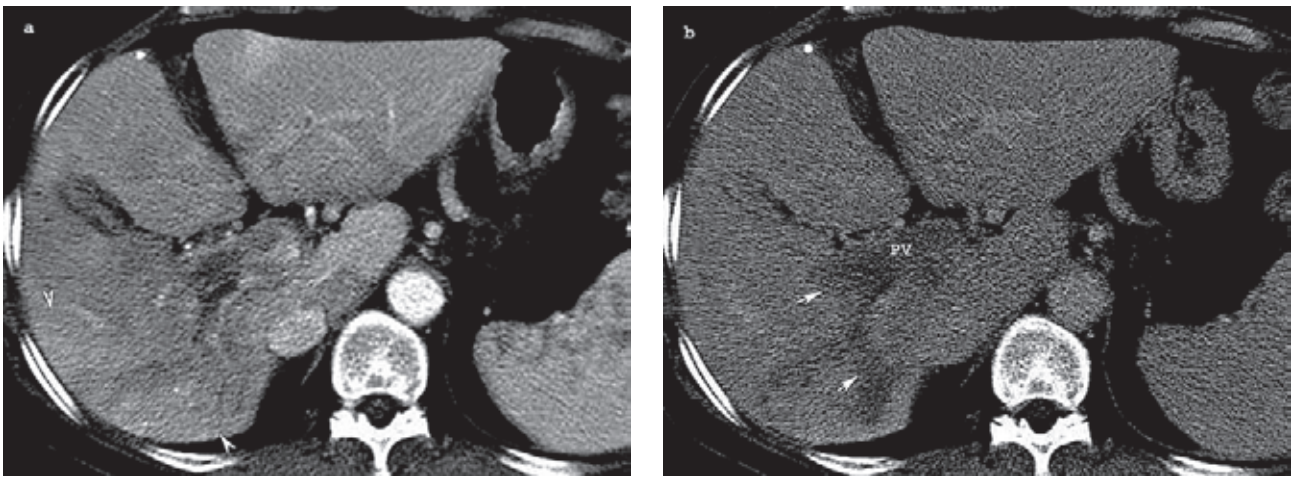

Fig.9 Portal thrombosis (short arrows) - CT evaluation a- arterial phase: peripheral hepatic perfusion disorders (arrows head), b-portal phase.

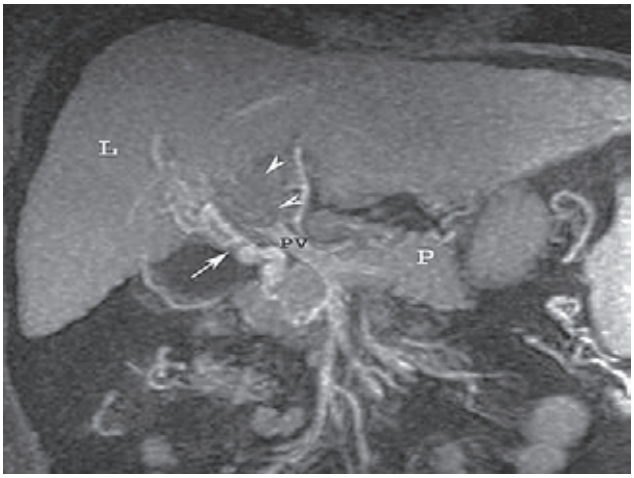

Fig.10 Portal cavernoma (long arrows). Portal vein thrombosis (arrows head)= MRI postGd evaluation. PV-portal vein. L-liver, P-pancreas.

perfusion disorders of the liver associated with portal venous system pathologies. MR imaging can aid in diagnosis and evaluation of these conditions and in particular cases is complementary to helical CT evaluation.

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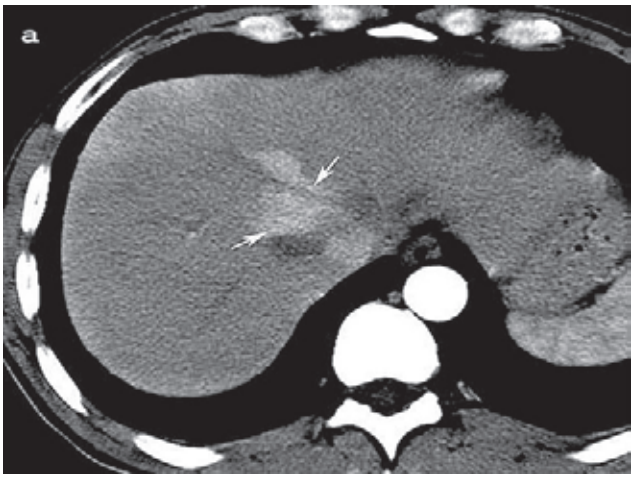

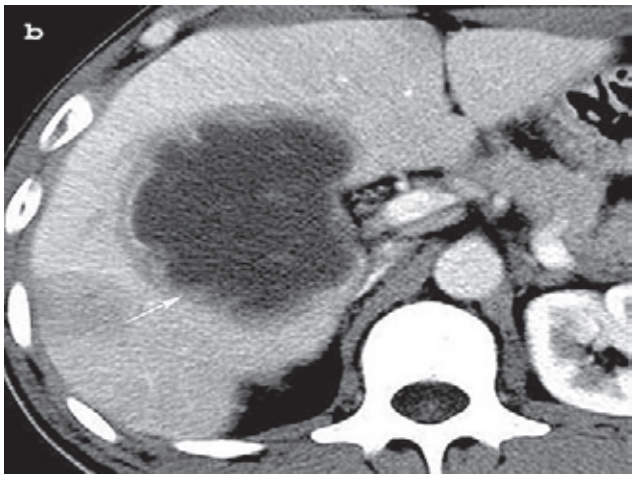

Fig.11 Arterio-hepatic shunts - CT evaluation: a-arterial phase: early enhancement of middle hepatic vein and inferior vena cava (arrows), b-portal phase: huge necrotic hepatic tumor (long white arrow).

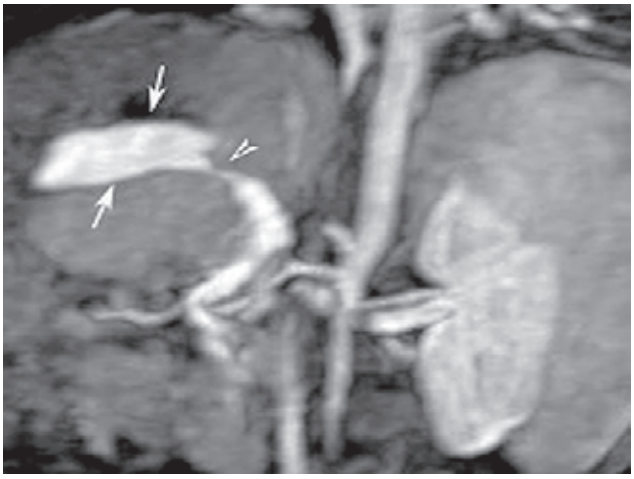

Fig.12 Pseudoaneurysm of portal vein - MRI- coronal MIP reconstruction poststenotic (arrows head) saccular dilatation of portal vein (arrows).

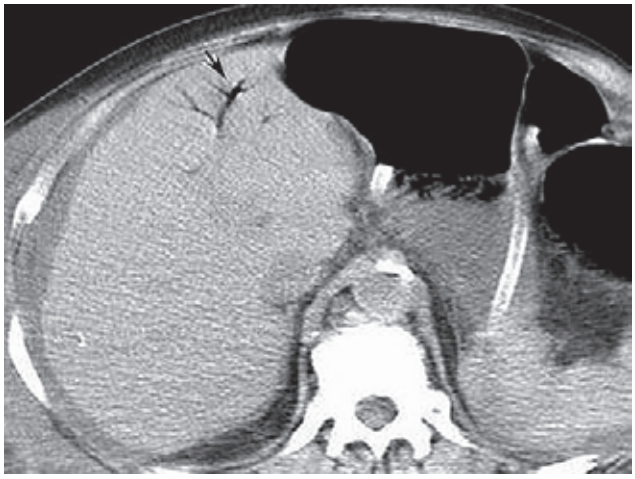

Fig 13 Portal air (black arrow)- CT evaluation.

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