Liver

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24.1 Anatomy

The liver consists of a right and left lobe, which are divided by the gallbladder fossa and the falciform ligament. For clinical practice, the division of the liver into **segments** is important. In this context, the **classification according to Couinaud and Bismuth** is most commonly used (Fig. 24.1). In this classification, segments are separated by the right, middle, and left hepatic veins in one plane and by the portal vein in a perpendicular plane. The caudate lobe is defined as S1. Table 24.1 presents typical values regarding the anatomy and physiology of the liver.

The liver is supplied by both the hepatic artery (15–20%) and the portal vein (75–80%). The portal vein originates posterior to the pancreatic head/body where the superior mesenteric vein and the splenic vein unite and enter the porta hepatis along the hepatoduodenal ligament. The hepatic artery is a branch of the celiac trunk and also enters the liver via the hepatoduodenal ligament. The central veins of the hepatic lobules consecutively unite and finally form the right, middle, and left hepatic veins. These veins enter the inferior vena cava below the diaphragm.

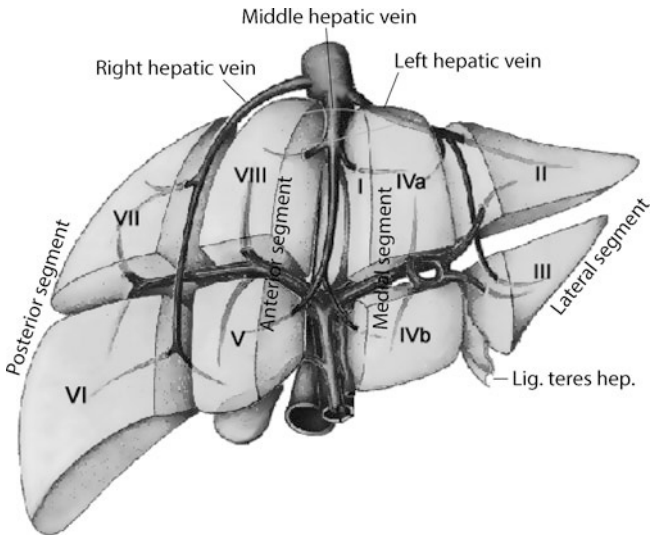

Fig. 24.1 Classification of liver segments according to Coinaud and Bismuth

Table 24.1 Liver: typical values

Weight	1,500 g
Volume	1,700 ml
Diameter	12 ± 3 cm (midclavicular line)
Attenuation (computed tomography)	~ 60 HU
Diameter (portal vein)	< 15 mm (extrahepatic)
Diameter (celiac trunk, superior mesenteric artery)	< 6 mm
Diameter (inferior vena cava)	< 15 mm
Diameter (hepatic veins)	< 10 mm
Blood flow (hepatic artery)	500 ml/min
Blood flow (portal vein)	1,000 ml/min
Pressure (portal vein)	5–10 mmHg (portal hypertension > 12 mmHg)
Pressure (hepatic veins)	2–4 mmHg

24.2 Imaging modalities

24.2.1 Ultrasound

Despite increasing use of computed tomography (CT) and magnetic resonance (MR) imaging, in most cases ultrasound remains the first imaging modality in the evaluation of focal and diffuse liver disease, owing to its availability, low cost, and noninvasiveness. Disadvantages are operator dependence and limitations regarding reproducibility and comparability. Because of limited examination conditions, evaluation of the entire liver may not be possible. In recent years, **contrast-enhanced ultrasound** has entered the clinical routine, especially for evaluating focal liver lesions. Similar to CT and MR imaging, it can differentiate between hypovascular and hypervascular tumours. A typical feature of many malignant tumours is a substantial washout of contrast material, whereas benign lesions tend to show longer-lasting plateaus of enhancement.

24.2.2 Computed tomography

Modern multidetector CT (MDCT) scanners allow imaging of the entire liver in a very short time. Therefore, even in uncooperative patients, high-quality images can be acquired. Depending on the indication, different multiphase protocols can be used (Table 24.2).

Potential **indications** for acquiring a native scan include the following:

- Haemorrhage
- Fibrotic changes
- Calcifications
- Changes in density (e.g., haemochromatosis, steatosis)

Contrast phases in liver imaging (e.g., 90 ml lomeron 400, flow 3 ml/s)

- Arterial phase: 25 s p.i.
- Late arterial phase: 35–45 s p.i.
- Portal venous phase: 70 s p.i.
- Vascular equilibrium phase: 3–5 min p.i.
- Parenchymal equilibrium phase: 10–15 min p.i.

24.2.3 Magnetic resonance imaging

MR imaging is performed at 1.5 T and 3.0 T using body phased-array coils. A typical sequence protocol consists of an axial na-

Table 24.2 CT imaging protocols

Portal venous phase	Hypovascular lesions, metastases Follow-up imaging Inflammatory processes
Late arterial–portal venous phase	Primary staging of tumours with hypervascular metastases Gastrointestinal stromal tumours (GIST) Neuroendocrine tumours Depiction of vessels, e.g., posttransplantation
Native–late arterial–portal venous phase	Hepatocellular carcinoma (HCC), e.g., after transarterial chemoembolisation
Late arterial–portal venous–delayed phase	HCC, cholangiocarcinoma, haemangioma

tive T1w gradient echo (GRE) sequence (e.g., FLASH), axial and coronal T2w HASTE/TSE sequences, an axial diffusion-weighted single-shot echo-planar imaging (ssEPI) sequence, and an axial fat-saturated T1w 3D GRE sequence (e.g., VIBE/THRIVE) performed repetitively after intravenous application of the contrast agent. In most cases, nonspecific extracellular gadolinium (Gd) chelates such as Gd-DTPA are used (0.1 mmol/kg body weight). Whereas the use of superparamagnetic iron oxide particles (SPIOs) has consistently declined in recent years, Gd-EOB-DTPA represents a hepatocyte-specific contrast agent that is increasingly used, such as for the detection and characterisation of focal liver lesions. Gd-EOB-DTPA is taken up by hepatocytes and subsequently eliminated via the kidneys (50%) and bile (50%). Imaging during the hepatobiliary phase (20 min p.i.) allows differentiation of lesions containing functioning hepatocytes (benign and low-malignant hepatocellular lesions) from lesions without intact hepatocytes (e.g., metastases). Because of higher costs and longer scanning time, hepatocyte-specific contrast agents are used only for particular indications at most institutions.

Diffusion-weighted MR imaging (DWI) is increasingly used in hepatic imaging, mainly in the detection and to some degree the characterisation of focal liver lesions. DWI can be performed as a breath-hold or respiratory-triggered sequence. Usually, ssEPI sequences are used. Using DWI, focal liver lesions, even those < 10 mm, can be detected with high contrast-to-noise ratio (CNR), and due to the black-blood effect, small lesions can be differentiated from vessels. Diffusion of water protons can be quantified by calculating apparent diffusion coefficient (ADC) values. In most cases, benign lesions such as cysts and haemangiomas, owing to their relative hypocellularity, show higher ADC values than malignant lesions such as hepatocellular carcinoma (HCC) or metastases that are hypercellular in most cases.

24.3 Disorders and variants of liver perfusion

24.3.1 Portal venous hypertension

Portal venous hypertension is defined as a difference of more than 5 mmHg between blood pressure in the portal vein and the inferior vena cava. In most cases it becomes clinically evident when > 12 mmHg. Reasons for portal hypertension are divided into extrahepatic/presinusoidal, intrahepatic, and posthepatic/postsinusoidal (Table 24.3).

Table 24.3 Aetiology of portal venous hypertension

Extrahepatic/presinusoidal	Portal vein thrombosis Arterioportal fistula Splenic vein thrombosis Occlusion of superior mesenteric vein
Intrahepatic	Congenital liver fibrosis Idiopathic liver fibrosis Primary biliary cirrhosis Felty syndrome Sarcoidosis Wilson's disease Schistosomiasis Toxic liver fibrosis (copper, arsenic)
Intrahepatic/sinusoidal	Cirrhosis Sclerosing cholangitis
Posthepatic/postsinusoidal	Occlusion of hepatic veins/Budd–Chiari syndrome

Typical imaging findings in patients with portal venous hypertension are dilatation of the portal vein (diameter > 15 mm) and splenic/superior mesenteric vein (diameter > 10 mm). In addition, portosystemic collateral vessels, such as oesophageal varices or recanalisation of the umbilical vein, may be seen. Furthermore, ascites or splenomegaly can result from increased portal venous blood pressure. In MR imaging, multiple small (diameter of a few millimetres) nodules that are hypointense on T1-weighted and T2-weighted sequences (particularly GRE sequences) may be seen; these are the so-called **Gamna–Gandy bodies**.

24.3.2 Portal vein thrombosis

Portal vein thrombosis is one of the common reasons for prehepatic portal venous hypertension. It can be secondary to invasion by a tumour (as in patients with HCC) but can also occur in inflammatory or myeloproliferative diseases, coagulopathies, or liver transplant recipients. In the acute phase, the thrombosis within the portal vein is hypoechoic on ultrasound. Over time and along with organisation, the thrombus becomes subsequently hyerechoic; collateral vessels may form; and **cavernous transformation** (Fig. 24.2) and even calcification may occur. In CT imaging an intraluminal filling defect can be seen. In contrast to thrombus formation by apposition, tumour thrombi show moderate to strong enhancement of contrast material. In MR imaging, subacute thrombotic material can be hyperintense in both T1-weighted and T2-weighted sequences with gradual loss of signal over time.

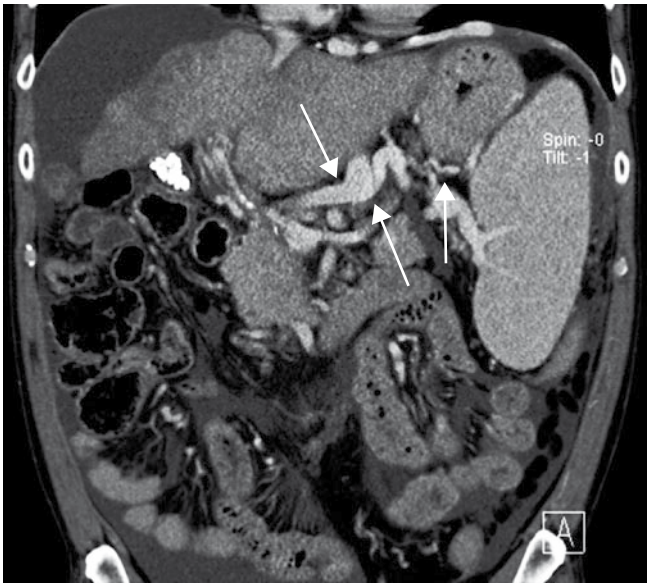

Fig. 24.2 Cavernous transformation in a patient with portal vein thrombosis. Note formation of multiple collateral venous vessels in the porta hepatis (*arrows*)

24.3.3 Occlusion of hepatic veins/Budd–Chiari syndrome

Hepatic venous outflow can be impaired on any level, from small postsinusoidal veins (veno-occlusive disease, such as secondary to chemotherapy or radiation therapy) to the inferior vena cava. Whereas primary Budd–Chiari syndrome can be observed preferentially in women in the Middle East and Asia and is caused by formation of venous membranes or septae, in Europe and the USA it is more commonly seen in patients with coagulopathies or vascular injury or inflammation, or secondary to obstruction by a tumour.

In imaging studies, the hepatic veins are partially or completely occluded by thrombotic material. Hepatomegaly and ascites are also seen. In the arterial phase, a speckled enhancement (mosaic pattern) may be observed, especially in the periphery, with more normal enhancement in the central parts of the liver and the caudate lobe, which may be hypertrophic (Fig. 24.3). In later phases, a so-called flip-flop pattern may be seen, with central washout of contrast material and delayed peripheral enhancement.

24.3.4 Right-sided heart insufficiency

Secondary to reduced function of the right side of the heart, increased pressure in the inferior vena cava and hepatic veins, hepatomegaly, and relative hypoechogenicity may be observed on abdominal ultrasound. In addition, the hepatic veins may show diameters >10 mm close to their confluence with the inferior vena cava and not change substantially in diameter when measured during inspiration and expiration.

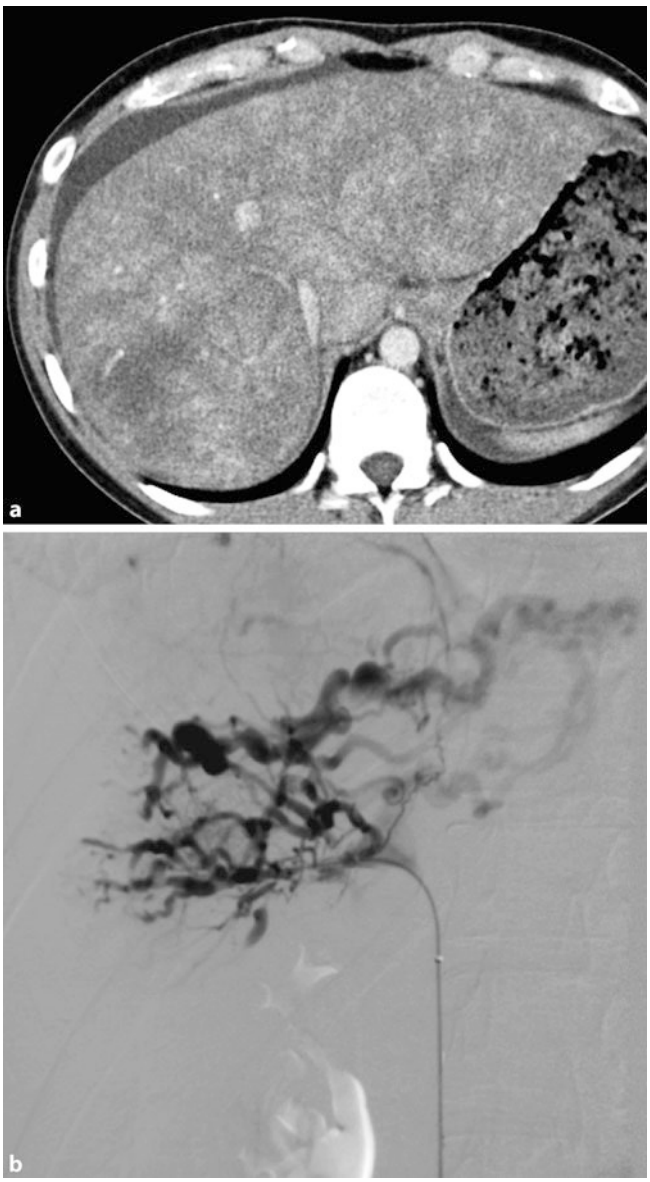

Fig. 24.3a,b Budd–Chiari syndrome. Computed tomography shows inhomogeneous, patchy enhancement with a mosaic pattern within the liver. The hepatic veins are not visible (**a**). Cavography shows numerous small venous vessels (spider web) but not the hepatic veins (**b**)

24.3.5 Hereditary haemorrhagic telangiectasia (Osler–Weber–Rendu disease)

Hereditary haemorrhagic telangiectasia (HHT) is inherited in an autosomal dominant way. It is characterised by the presence of numerous vascular malformations that may be found in different organ systems. When involving the liver, telangiectasias as well as arterioportal and arteriovenous shunts may be seen.

24.3.6 Peliosis hepatis

Peliosis hepatis is characterised by the presence of intrahepatic cavities filled with blood communicating with hepatic sinusoids. Among other causes, it is associated with hormone-producing

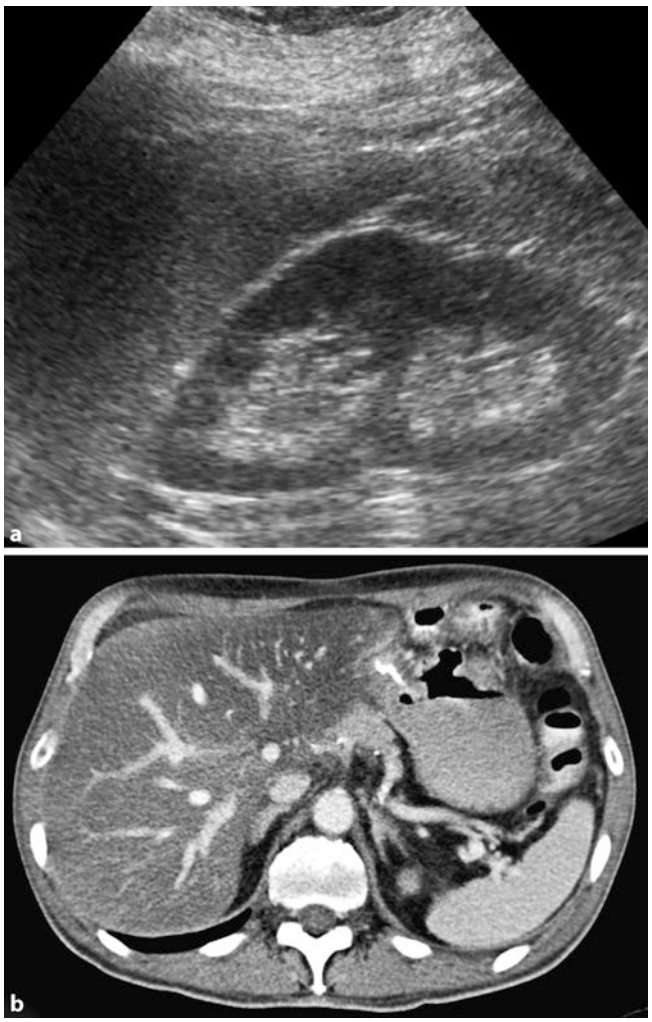

Fig. 24.4a,b Hepatic steatosis. The liver is hyperechogenic on ultrasound, and areas of the liver distant from the transducer are less well visualised (**a**). It is hypodense on CT compared to muscles and spleen (**b**)

hepatic tumours, oral contraception, chronic infections, diabetes mellitus, and chronic renal failure. In imaging studies, cavities with strong arterial enhancement with a slow decrease of enhancement in delayed phases can be seen. Ruptures may cause intrahepatic or subcapsular haematomas.

24.4 Diffuse liver disease

24.4.1 Liver steatosis

Steatosis of the liver can be age-related, food-related, or the consequence of intoxication (alcohol, drugs, chemotherapy), infection (viral hepatitis), disorders of metabolism (diabetes mellitus, haemochromatosis, Wilson's disease), or systemic disorders. In most cases, diffuse fatty infiltration of the liver results in hepatomegaly that may become obvious as hypoechogenicity and rounded liver contours on abdominal ultrasound. Increased echogenicity of the liver can be objectified by comparing it with the echogenicity of the right liver. In advanced hepatic steatosis,

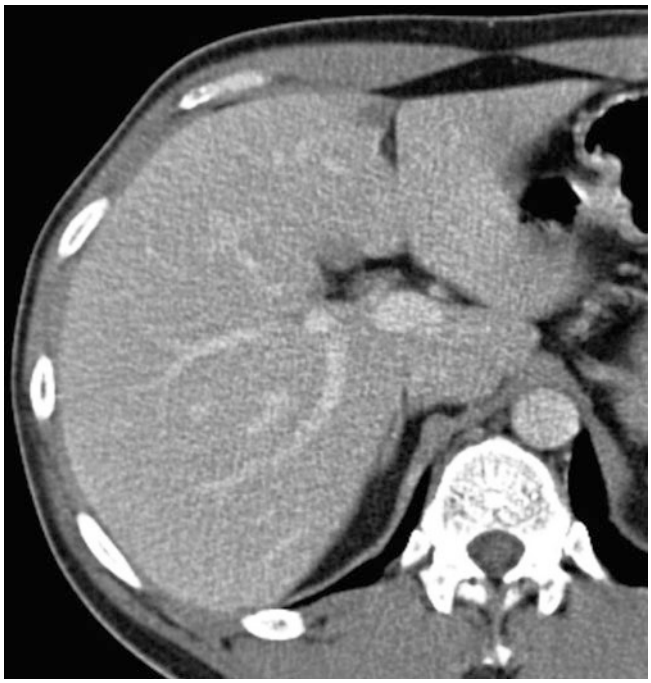

Fig. 24.5 Focal hepatic steatosis. Computed tomography shows a focal hypodensity adjacent to the falciform ligament

parts of the liver distant from the transducer become less well visualised (Fig. 24.4). On unenhanced CT images, a decrease of 15 HU per 10% increase in hepatic fat content can be seen. Focal steatosis can typically be observed in the caudate lobe, adjacent to the falciform ligament (Fig. 24.5) or the gallbladder fossa. In rare cases, ringlike or focal lesions can be mimicked by focal fatty infiltration.

24.4.2 Liver cirrhosis

Liver cirrhosis is among the ten most common causes of death in the Western world. In addition to the risk of lethal bleeding from oesophageal varices, the risk of developing HCC is substantially increased in patients with hepatic cirrhosis. Chronic alcohol abuse and viral hepatitis are the most common conditions leading to liver cirrhosis. On imaging studies, rounded liver contours along with changing ratios between the sizes of the right and left liver lobes and hypertrophy of the caudate lobe may be initial signs of cirrhotic changes (Fig. 24.6). Later on, decreased organ volume; an irregular, nodular surface; and an inhomogeneous increase in echogenicity can be seen. Signs of portal venous hypertension such as recanalisation of the umbilical vein may be encountered. Peripheral vessels may be rarified, and nodular changes (regenerative nodules, dysplastic nodules, HCC) can be seen.

24.4.3 Primary biliary cirrhosis

Primary biliary cirrhosis (PBC) is a rare cause of chronic liver disease. Presumably due to an autoimmune process, cholestasis

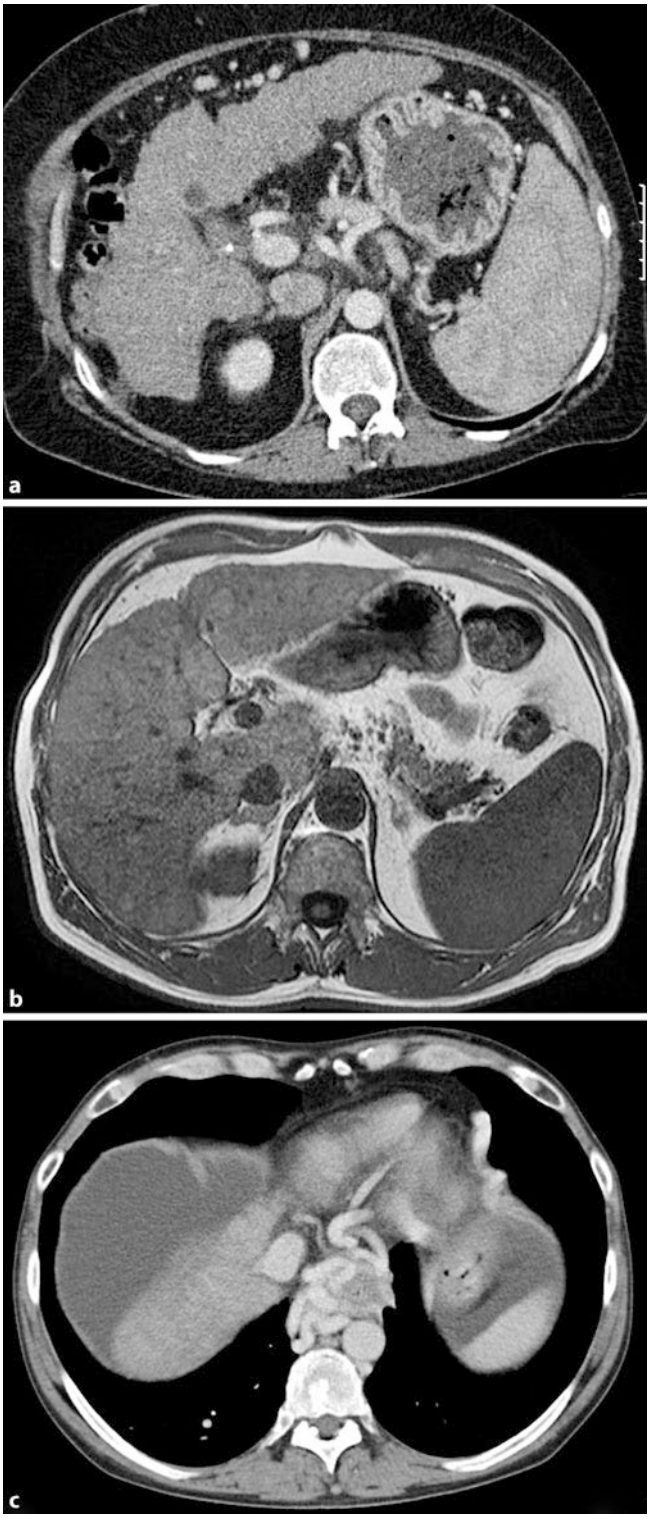

Fig. 24.6a–c Liver cirrhosis. A small liver with a nodular surface is seen (**a**). In addition, hypertrophy of the caudate lobe can occur (**b**). Secondary changes including ascites and formation of venous collateral vessels, such as oesophageal varices in this case, are often seen (**c**)

and inflammatory changes affecting the small intrahepatic bile ducts are triggered, finally resulting in liver cirrhosis. The risk of developing HCC is only moderately elevated. Middle-aged women are those mainly affected. Associations with other autoimmune disorders have been established. In imaging studies, morphology of the liver is comparable to that of cirrhosis from other causes. The liver surface remains comparably smooth. Portal hypertension and hilar lymphadenopathy are quite common.

24.4.4 Haemochromatosis and haemosiderosis

Haemochromatosis and haemosiderosis are different entities regarding the pathophysiology and clinical presentation of patients. **Haemochromatosis** is inherited in an autosomal recessive manner. Hepatic iron overload is mainly the result of increased intestinal iron absorption. Iron deposits are found within hepatocytes as well as in other organs including the pancreas, heart, and skin ("bronze diabetes"), but not the spleen. Liver function finally deteriorates, and the risk of developing HCC is increased up to about 30%. In contrast, **haemosiderosis** results from iron overload (e.g., from blood transfusions). Iron is stored in the cells of the reticuloendothelial system (Kupffer cells in the liver) as well as in the spleen and bone marrow.

In native CT imaging, iron overload results in hyperdensity of the liver (> 70 HU, rarely up to 140 HU). MR imaging is well suited for diagnosing and monitoring iron overload. The liver is relatively hypointense on T1-weighted and T2-weighted sequences (Fig. 24.7). Cirrhotic changes and concomitant extrahepatic alteration can be visualised on both CT and MR imaging.

24.4.5 Other diffuse liver diseases

On histopathological analysis, the liver and spleen are involved in 25–75% of patients with **sarcoidosis**. However, hepatic involvement is rarely clinically relevant. On imaging, hepatosplenomegaly and/or small disseminated nodular changes may occasionally be visible. Concomitant abdominal lymphadenopathy is common.

Hepatomegaly may be the only finding in patients with hepatic involvement in **amyloidosis**. Rarely, hypodense areas with reduced uptake of contrast material can be seen in the liver and spleen.

In patients with **Wilson's disease,** the liver is hyperdense on CT imaging. On MR imaging, the T1 signal may be reduced. Later on, cirrhotic changes along with hypointense focal lesions can be observed.

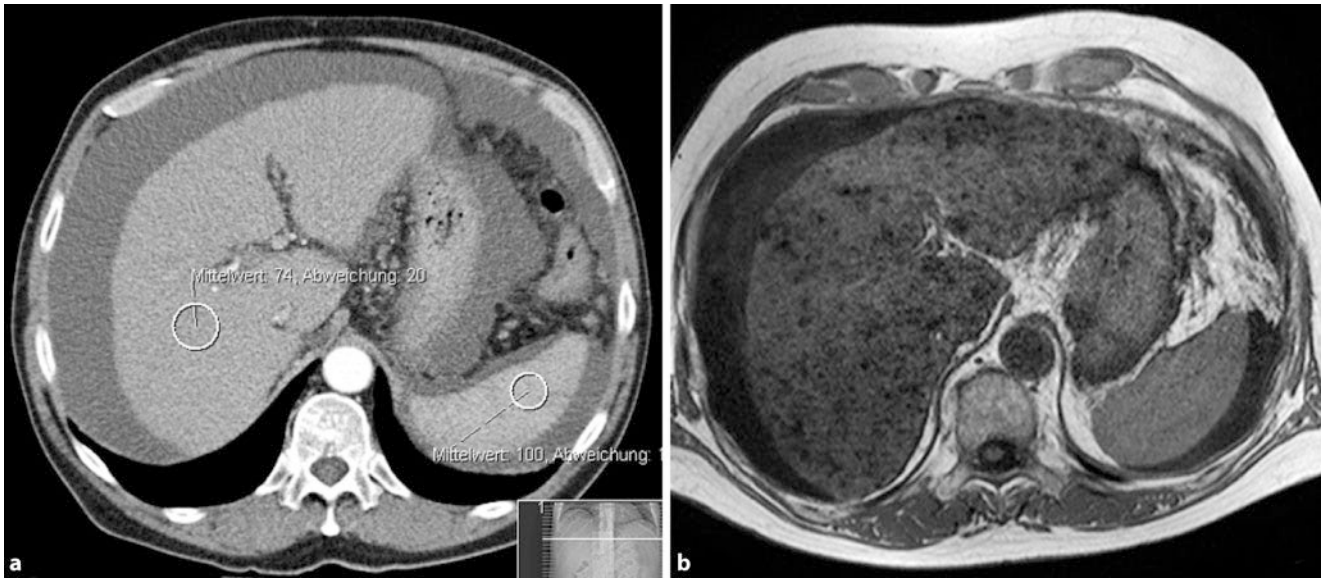

Fig. 24.7a,b Haemochromatosis. Liver density is increased to > 70 HU on CT (**a**). In T1-weighted MR imaging, focal hypointensities due to iron deposition are seen (**b**)

24.5 Focal liver disease: cystic and dysontogenetic changes

24.5.1 Liver cysts

Simple liver cysts can be seen in about 7% of the population; women are affected more often than men. Liver cysts can occur as single or multiple lesions and generally are clinically asymptomatic. They do not show malignant transformation. On ultrasound, simple cysts show no echogenicity, no solid mural nodules, and typically increased dorsal echogenicity. On CT imaging there is no enhancement, and the content of cysts shows densities between 0 and 15 HU. On MR imaging the signal intensity parallels that of water in all sequences: hypointense in T1-weighted images and markedly hyperintense in T2-weighted images, with no uptake of contrast material. Rarely, hyperdense and hyperintense (T1) contents can be seen within the cyst, pointing to a post-haemorrhage status or protein-rich fluid within the cyst. In these cases, the lack of contrast enhancement is crucial for making the correct diagnosis (Fig. 24.8).

24.5.2 Biliary hamartomas

Biliary hamartomas are congenital benign biliary malformations with cystic components. They do not cause symptoms and do not show malignant transformation. In addition, they do not communicate with the biliary system. On imaging, numerous small cyst-like lesions disseminated throughout the liver can be seen. There is no uptake of contrast material.

24.5.3 Mesenchymal hamartomas

Mesenchymal hamartomas are very rare. They can grow rapidly and tend to become clinically evident during early childhood (peak between 15 and 22 months of age). Most commonly they occur in the right liver lobe as large lesions with mixed density containing cystic and solid components, the latter showing moderate, inhomogeneous enhancement after administration of contrast material.

24.6 Focal liver disease: inflammatory/infectious changes

24.6.1 Tuberculosis

When hepatic involvement occurs in patients with tuberculosis, disseminated, micronodular ("miliary") changes are usually seen, often in association with mild hepatomegaly. On imaging studies, the liver may appear homogeneous or show disseminated small nodules. These are hypodense in CT imaging (35–45 HU). In addition, calcification, regional lymphadenopathy, and ascites may be present.

24.6.2 Hepatic abscesses

In Western countries, hepatic abscesses are rare and most commonly caused by pyogenic bacteria (85%). In contrast, in countries with low hygienic standards, infections with Entamoeba histolytica and fungi (e.g., *Candida* species, *Aspergillus* species) are more frequently seen. The right liver lobe is involved more frequently, and multiple abscesses may occur, particularly in bacterial and fungal abscesses.

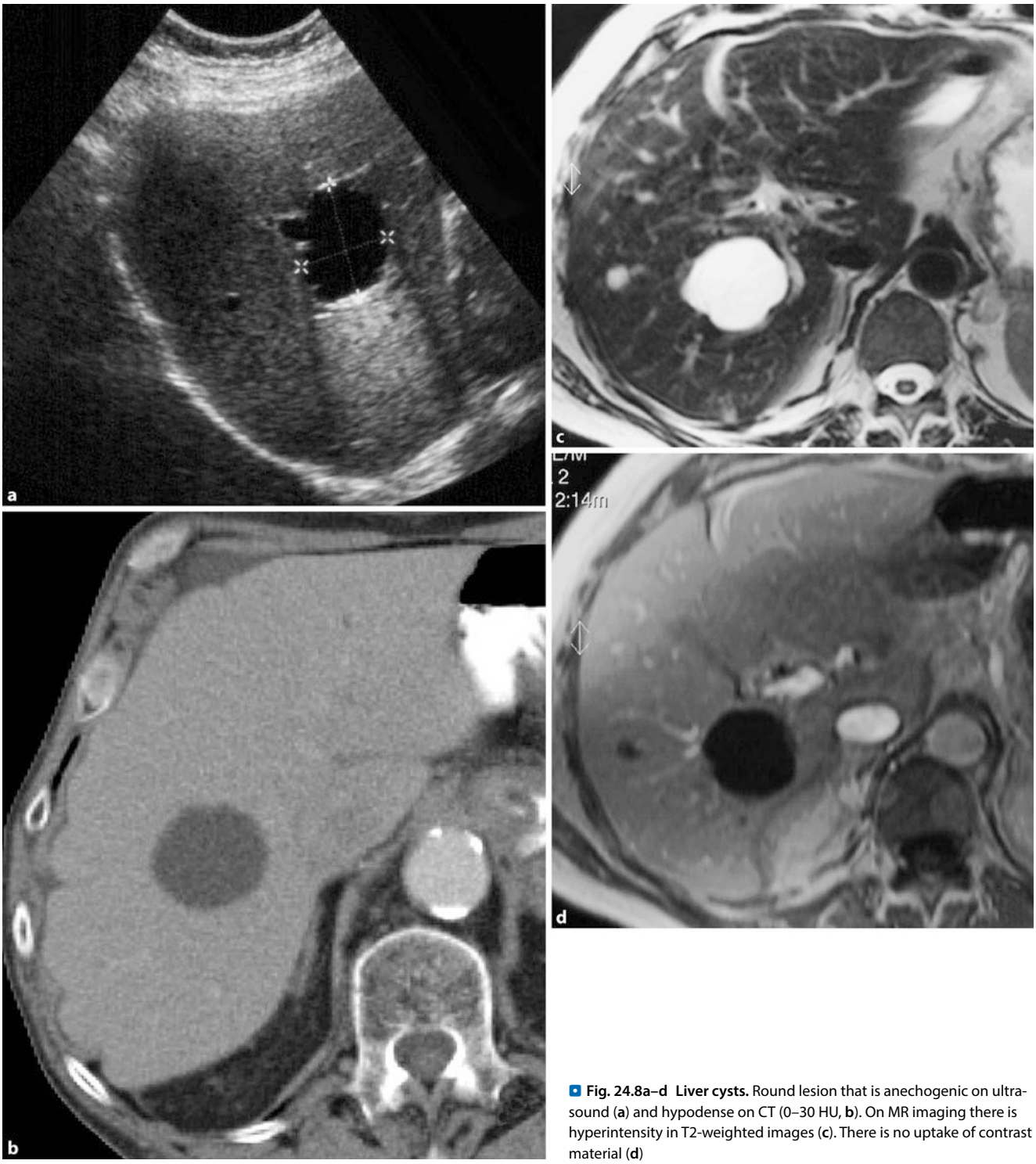

Fig. 24.8a–d Liver cysts. Round lesion that is anechogenic on ultrasound (**a**) and hypodense on CT (0–30 HU, **b**). On MR imaging there is hyperintensity in T2-weighted images (**c**). There is no uptake of contrast material (**d**)

Pyogenic hepatic abscesses

Pyogenic hepatic abscesses are most commonly seen in older patients with a weakened immune system. Typical bacteria are *E. coli*, *Staphlyococcus aureus,* enterococci, *Klebsiella*, and anaerobe species. In more than 50% of cases, more than one infectious agent can be found. In most cases, the hepatic infection occurs via the biliary system, such as in patients with cholangitis, followed by portal venous entry of bacteria (e.g., appendicitis, diverticulitis, pancreatitis). Haematogenous spread to the liver can occur in patients with pneumonia, pyelonephritis, or sepsis.

In imaging studies, initially an oedematous area that is hypoechogenic on ultrasound and hypodense/T1-hypointense/T2-hyperintense on CT/MR imaging (about 30 HU) can be seen. After 10–15 days, a central hypoechogenic part and a hyperechogenic periphery can be differentiated. The latter, corresponding to the abscess capsule, reveals a ringlike enhancement of contrast material on CT/MR imaging and surrounds the necrotic, often

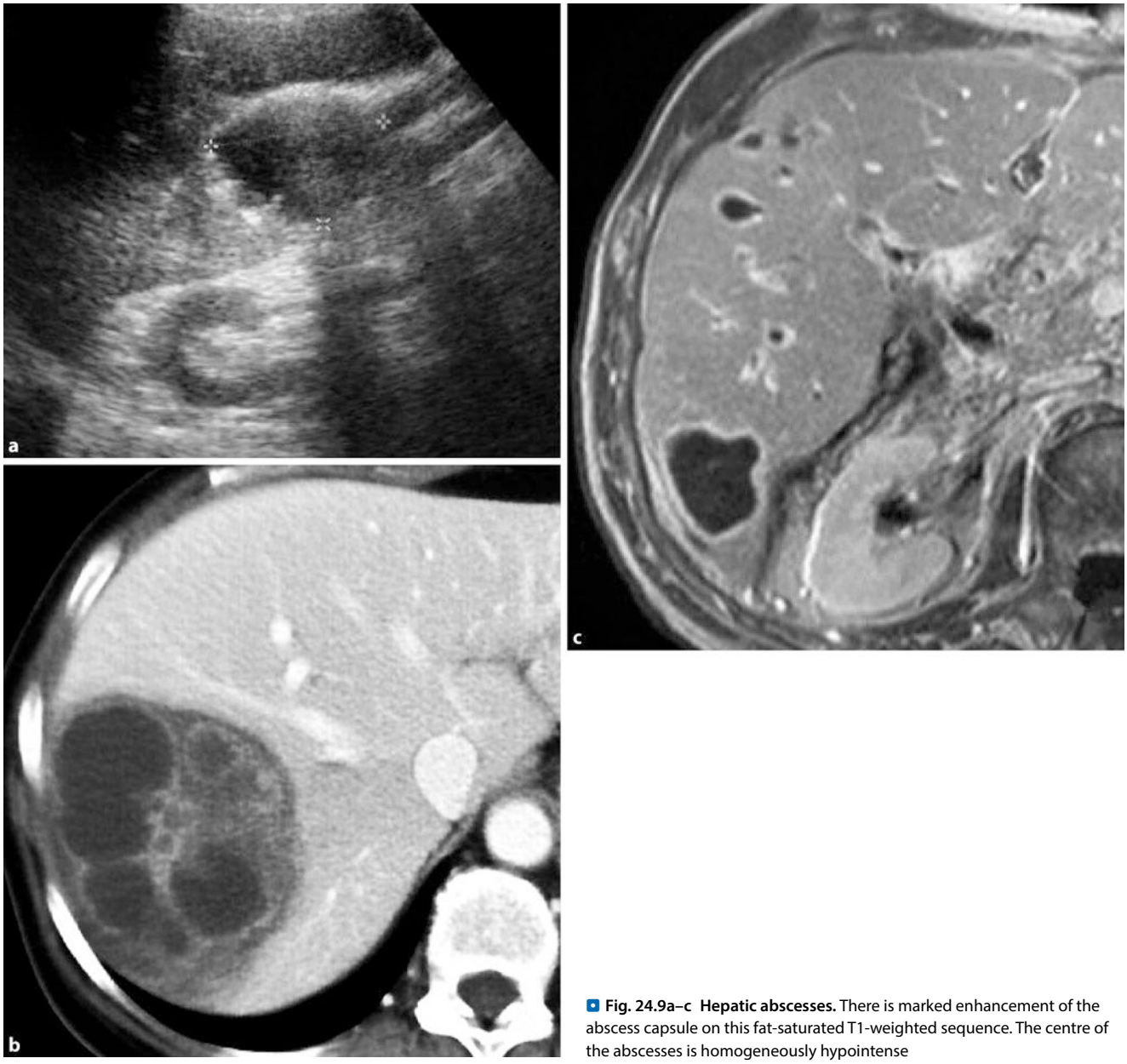

Fig. 24.9a–c Hepatic abscesses. There is marked enhancement of the abscess capsule on this fat-saturated T1-weighted sequence. The centre of the abscesses is homogeneously hypointense

liquid centre of the abscess (Fig. 24.9). If the capsule of the abscess is surrounded by a second, oedematous, hypodense zone, a so-called double-target sign is seen. Inclusions of gas are rarely seen, most commonly in anaerobic infections.

Hepatic abscess caused by Entamoeba histolytica

In most cases these abscesses are unilocular with a predilection for the right liver lobe. Imaging findings for these abscesses are similar to those for hepatic abscesses caused by bacteria.

24.6.3 Hepatic echinococcosis

Hepatic involvement in patients infected with ***Echinococcus granulosus*** is characterised by formation of unilocular or multilocular cystic lesions, occasionally with the aspect of branching cysts ("cyst in cyst"). The capsule of the cysts may be thick. In addition, solid septations and calcifications within the cyst walls are often seen.

In contrast, in patients infected by ***Echinococcus multilocularis*** an infiltrative intrahepatic spread is common, resulting in ill-defined hypodense areas within the liver on CT imaging. Necrotic areas and inhomogeneous enhancement of contrast material may be seen. In these patients, differentiation from hepatic malignancies may be impossible by imaging.

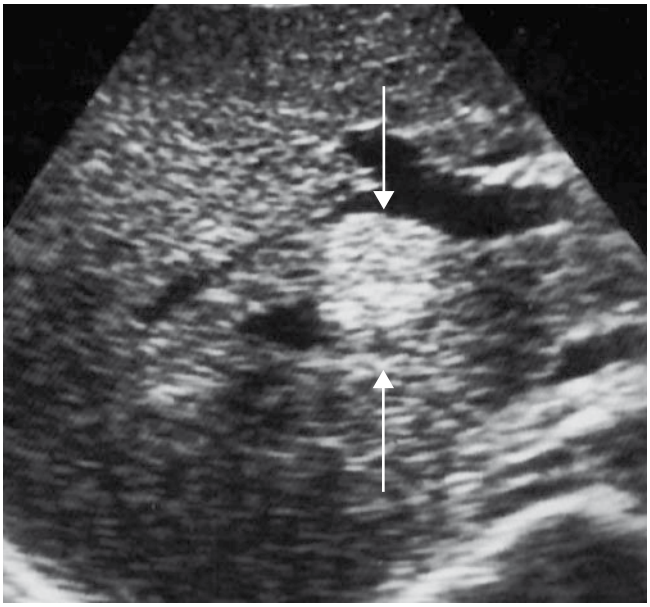

Fig. 24.10 Haemangioma. Ultrasound shows a homogeneously hyperechogenic lesion

24.7 Focal liver disease: benign/tumour-like lesions

24.7.1 Haemangioma

Haemangiomas are incidentally diagnosed in 5–7% of the population. They are more common in women. Rarely, they are associated with syndromes such as Kasabach–Merritt syndrome.

On ultrasound, haemangiomas characteristically are homogeneously hyperechogenic, well-defined focal lesions (Fig. 24.10). However, atypical morphologic features such as hypoechogenic areas may be seen, especially in larger haemangiomas with central areas of thrombosis or hyalinisation or in patients with hepatic steatosis. Haemangiomas larger than 10 cm are called giant haemangiomas. After the administration of contrast material, a centripetal mode of contrast enhancement is observed in all imaging modalities (Fig. 24.11), with a typical nodular peripheral enhancement in the early phases. Haemangiomas do not show a washout of contrast material in delayed phases. Small capillary haemangiomas may already show rapid

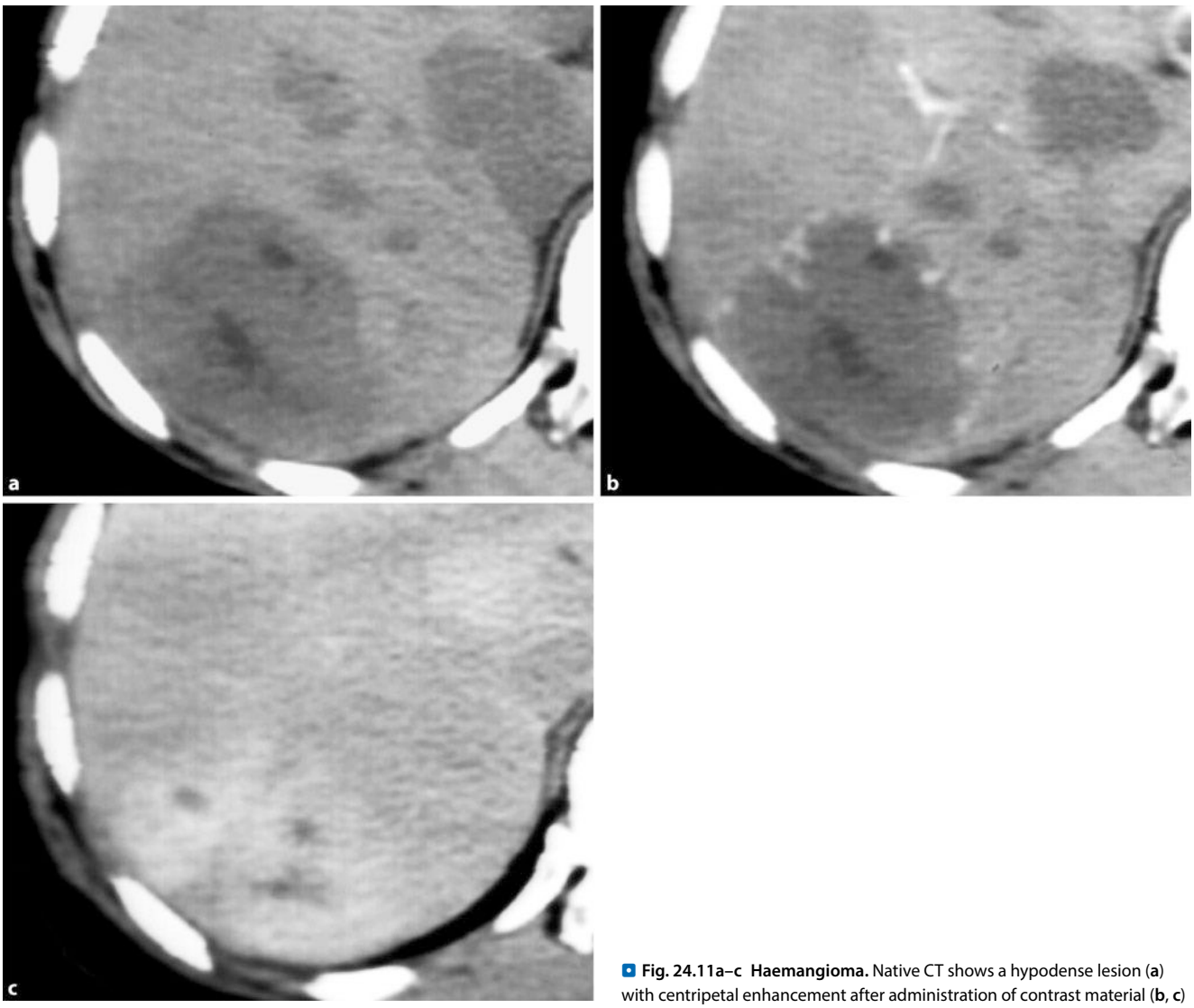

Fig. 24.11a–c Haemangioma. Native CT shows a hypodense lesion (**a**) with centripetal enhancement after administration of contrast material (**b**, **c**)

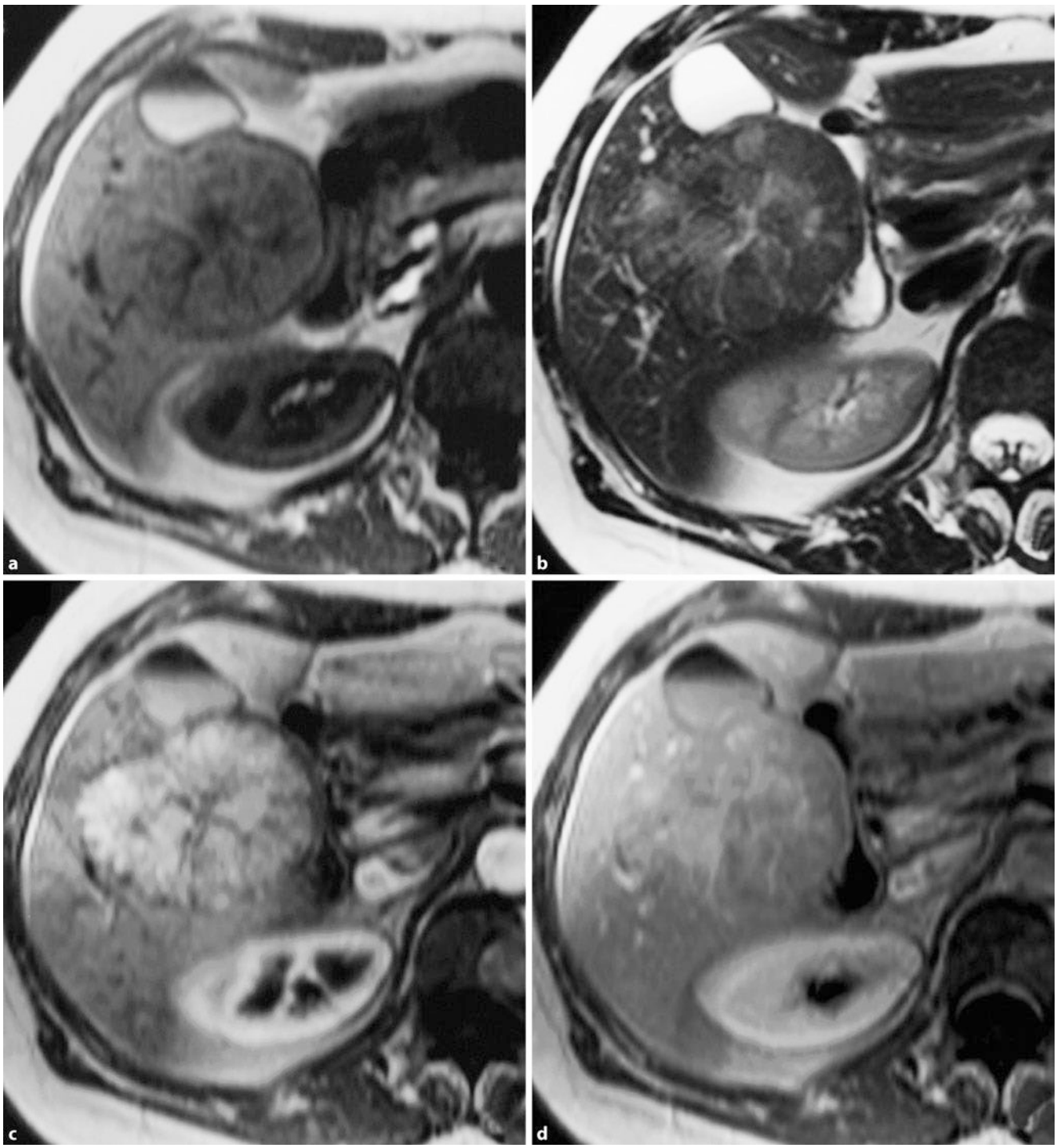

Fig. 24.12a–d Focal nodular hyperplasia. The lesion is hypointense on T1-weighted (**a**) and hyperintense on T2-weighted MR images (**b**) with a central scar. There is marked enhancement in the arterial phase (**c**). The central scar shows enhancement in the delayed phase (**d**)

enhancement of the whole lesion in the arterial phase. In addition, marked hyperintensity on T2-weighted images is another typical feature of haemangiomas.

24.7.2 Focal nodular hyperplasia

After haemangiomas, focal nodular hyperplasias (FNHs) are the second most common benign liver tumour, most frequently encountered in women between 20 and 50 years of age. Oral contraceptives may lead to a growth of preexisting FNHs; de novo development of FNHs, however, seems not to occur.

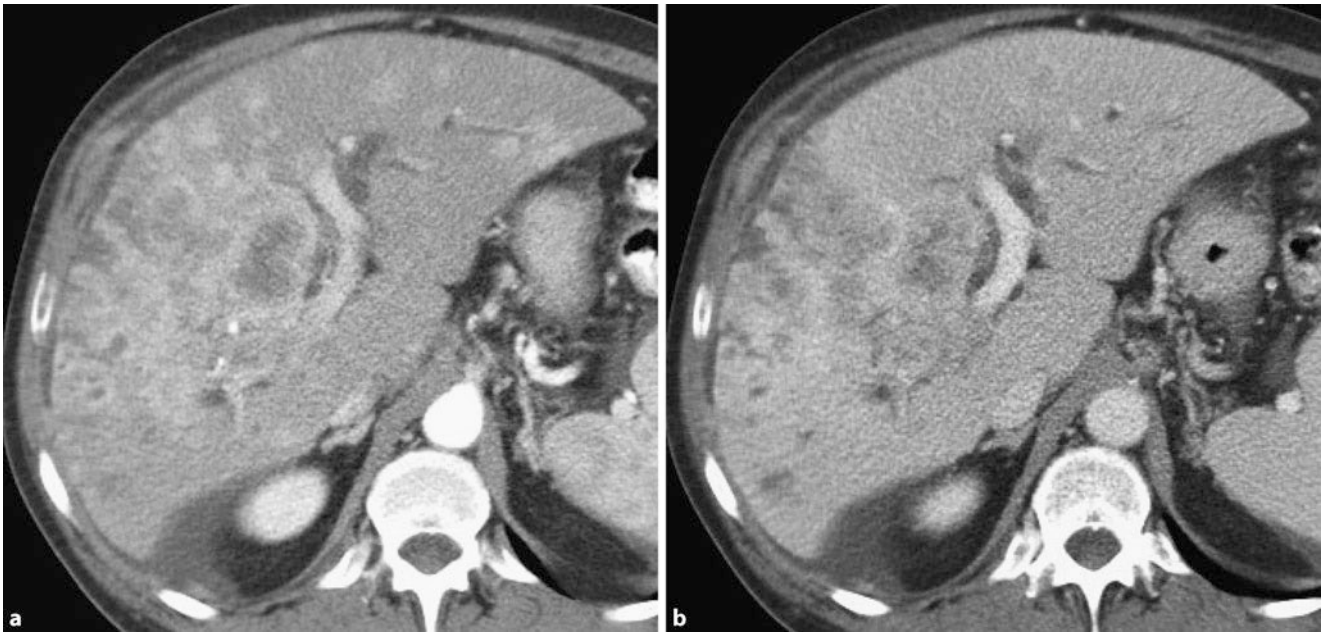

Fig. 24.13a,b HCC. Multiple lesions that are hypervascular in the arterial phase (**a**) and show rapid washout in the portal venous phase (**b**) are seen

Diagnosing FNHs by ultrasound can be difficult because characteristic features such as a central scar are usually not well visualised. Lesions may be hypoechogenic, isoechogenic, or hyperechogenic compared to surrounding liver parenchyma. On native CT and MR imaging, FNHs may also be difficult to detect. Rarely (< 1%), calcifications can be seen. On MR imaging a central scar may be visible in 35–50% of cases. After administration of contrast material, marked arterial enhancement with a rapid washout and isodensity/isointensity in the portal venous phase are typical features (Fig. 24.12). In delayed contrast phases there may be a slight to moderate uptake of the central scar. In addition, hyperintensity of FNHs compared to the surrounding liver is frequently seen in the hepatobiliary phase (20 min after administration of Gd-EOB-DTPA).

24.7.3 Hepatocellular adenoma

Hepatocellular adenomas (HCAs) are rare benign tumours of the liver, most often seen in young women using oral contraceptives. In addition, they may be seen in patients with metabolic diseases or in individuals consuming anabolic steroids. Patients are at risk for spontaneous bleeding. In addition, malignant transformation can occur with certain types of HCAs.

Using ultrasound, HCAs cannot be differentiated from other focal liver lesions such as FNHs, metastases, or abscesses. On CT and MR imaging, inhomogeneous densities/signal intensities secondary to intralesional haemorrhage may be seen. Hyperintensity on native T1-weighted images owing to high intralesional fat content can sometimes be observed. After administration of contrast material, HCAs typically show only weak or moderate arterial enhancement with inhomogeneous enhancement in the portal venous phase and relatively rapid washout.

24.8 Focal liver disease: malignant hepatic tumours

24.8.1 Hepatocellular carcinoma

About 80–90% of the primary malignancies of the liver are HCCs. Most commonly, men are affected during their fifth to seventh decades. As the tumours initially are asymptomatic, they are often diagnosed in advanced stages when causing upper abdominal pain, fever, weight loss, and ascites. The incidence of HCC is variable depending on the incidence of hepatitis B and C infections. Thus, in Japan, South Korea, China, and parts of Africa, high incidences of up to 150/100,000 per year are common. In Western Europe, the USA, and Australia, the reported incidences are lower (1–3/100,000 per year).

The main risk factor for developing HCC is liver cirrhosis, which is present in 60–90% of patients at the time of diagnosis. The most common etiologies are alcohol abuse, hepatitis B and/or C infection, and exposure to aflatoxin. In general, the risk of developing HCC in a patient with cirrhosis is about 3–5%.

In most cases, HCC develops gradually from regenerative nodules and low-grade and high-grade dysplastic nodules. With the grade of dysplasia, the perfusion percentage by the hepatic artery increases. HCCs can grow as unifocal or multifocal masses or as a diffuse infiltrative type. Angioinvasive growth and intratumoural haemorrhage are common in HCC.

On ultrasound the detection of HCC is often difficult due to nodular changes in cirrhotic liver. In most cases, it is inhomogeneously hyperechogenic with areas of hypoechogenicity secondary to haemorrhage and necrosis. On CT imaging a biphasic protocol is obligatory if HCC is suspected. Arterial hypervascularisation and early washout in the portal venous phase are typical for HCC. As on ultrasound, hypovascular areas, such as

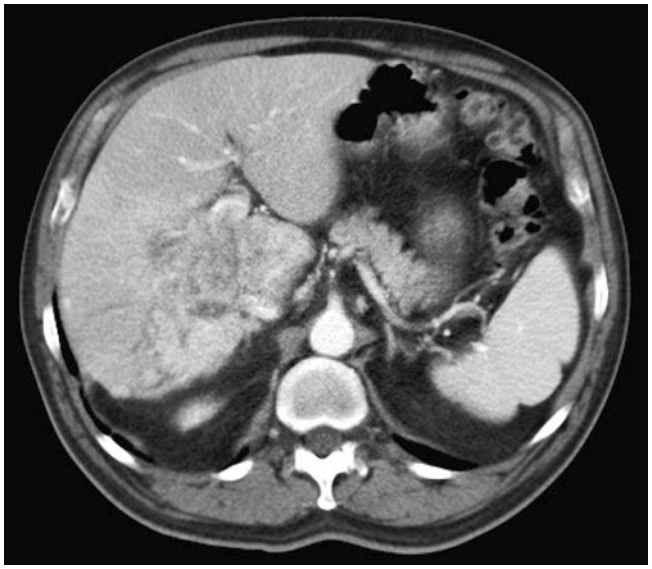

Fig. 24.14 Cholangiocarcinoma. An ill-defined hypodense lesion with dilatation of the peripheral bile ducts in the right liver lobe is seen. There is infiltration of the portal vein, which is less common for cholangiocarcinoma than for HCC

secondary to necrosis, may be seen, in particular in larger tumours (Fig. 24.13).

The appearance on MR imaging can be variable. In most cases HCC are hypointense on T1-weighted sequences and hyperintense on T2-weighted sequences. On contrast-enhanced sequences, especially in the arterial and portal venous phase or, importantly, similar to CT imaging, strong arterial enhancement and rapid portal venous washout are characteristic findings. In the hepatobiliary phase, HCC does not show an uptake of Gd-EOB-DTPA in most cases.

24.8.2 Cholangiocarcinoma

Following HCC, cholangiocarcinoma (CC) is the second most common malignant primary tumour of the liver. Women are affected more often than men. Among others, potential risk factors include exposure to Thorotrast, primary sclerosing cholangitis, choledochal cysts, and familial polyposis coli. Intrahepatic, hilar, and peripheral CCs are differentiated (hilar and peripheral CC are discussed in ▶ Chap. 25).

On ultrasound, the morphology of CC is comparable to that of HCC. On CT and MR imaging, weak to moderate enhancement is seen in the arterial as well as the portal venous phase. Capsular retraction and coarse calcifications are typical findings. Moderate enhancement may persist in the equilibrium phase. Most CCs are hypointense in T1-weighted sequences and show mixed signal intensities in T2-weighted sequences. Dilatation of the intrahepatic bile ducts peripheral to the tumour is common (Fig. 24.14), whereas angioinvasion is a less common feature. In the hepatobiliary phase, CC does not show an uptake of Gd-EOB-DTPA.

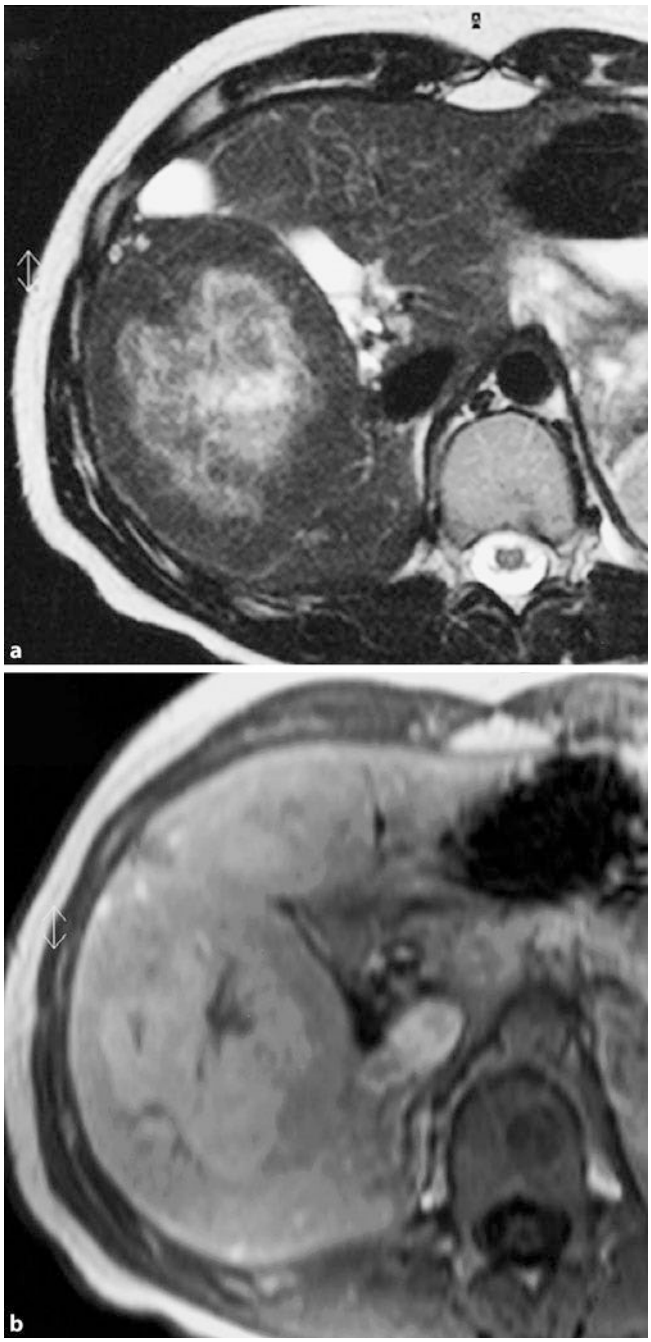

Fig. 24.15a,b Fibrolamellar carcinoma. The lesion shows an inhomogeneous, mostly hyperintense signal on the T2-weighted sequence (**a**). In the portal venous phase there is moderate enhancement with hypointensity of the central scar (**b**)

24.8.3 Fibrolamellar hepatocellular carcinoma

Fibrolamellar hepatocellular carcinoma (FLC) accounts for only about 2% of malignant liver tumours. It develops in noncirrhotic livers, and patients are substantially younger than those with HCC. Most often solitary, well-demarcated liver lesions are seen that show moderate peripheral enhancement on CT and MR imaging. Occasionally, a central scar may be present (Fig. 24.15).

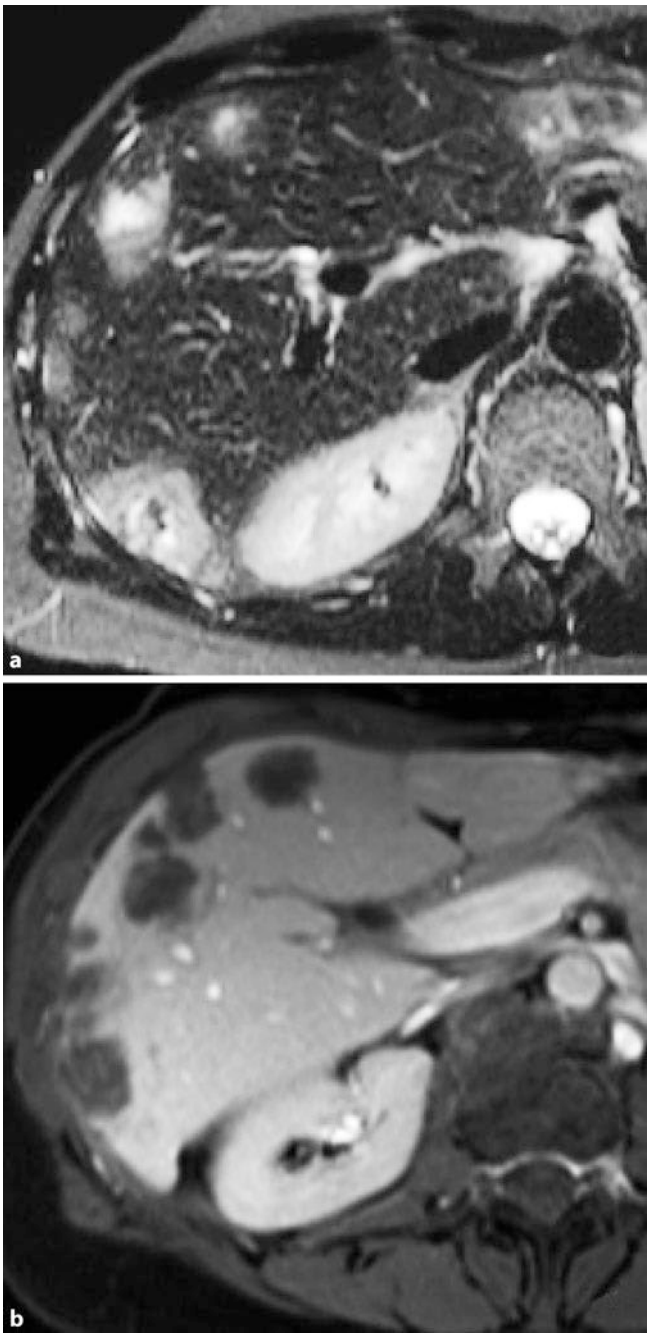

Fig. 24.16a,b Epithelioid haemangioendothelioma. Multiple lesions are located in the periphery of the liver. They are moderately hyperintense in the T2-weighted sequence (**a**) and show peripheral enhancement (**b**)

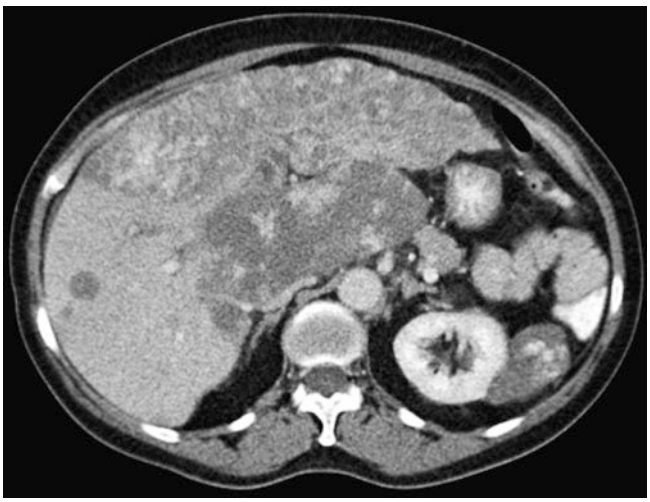

Fig. 24.17 Angiosarcoma. Multiple nodules are seen in the right and left liver lobe and the caudate lobe. Lesions show peripheral nodular enhancement

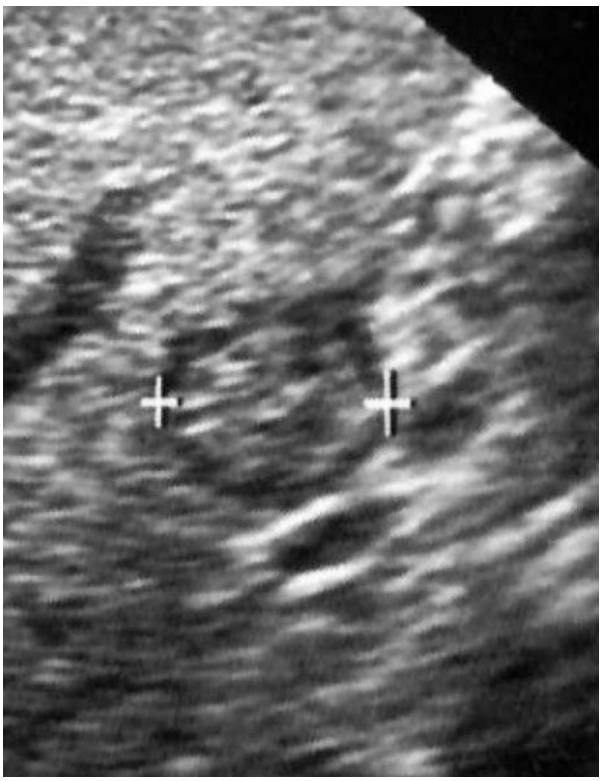

Fig. 24.18 Metastases. The lesion is almost isoechogenic to the liver on ultrasound but is surrounded by a hypoechogenic halo

24.8.4 Epithelioid haemangioendothelioma

Epithelioid haemangioendotheliomas are rare tumours with low malignant potential. In most cases, multilocular small lesions are seen in the periphery of the liver (Fig. 24.16). Capsular retraction and calcification of lesions may occur. On CT and MR imaging, tumours show weak or moderate peripheral enhancement.

24.8.5 Angiosarcoma

Angiosarcoma, a rare liver tumour, occurs after exposure to Thorotrast, vinyl chloride, or inorganic arsenic and usually affects elderly patients. MR imaging shows multiple well-perfused nodules with mixed signal intensities (Fig. 24.17). After administration of contrast material, nodular, lacunar peripheral enhancement (similar to that seen in haemangiomas) may be observed, but with no centripetal enhancement.

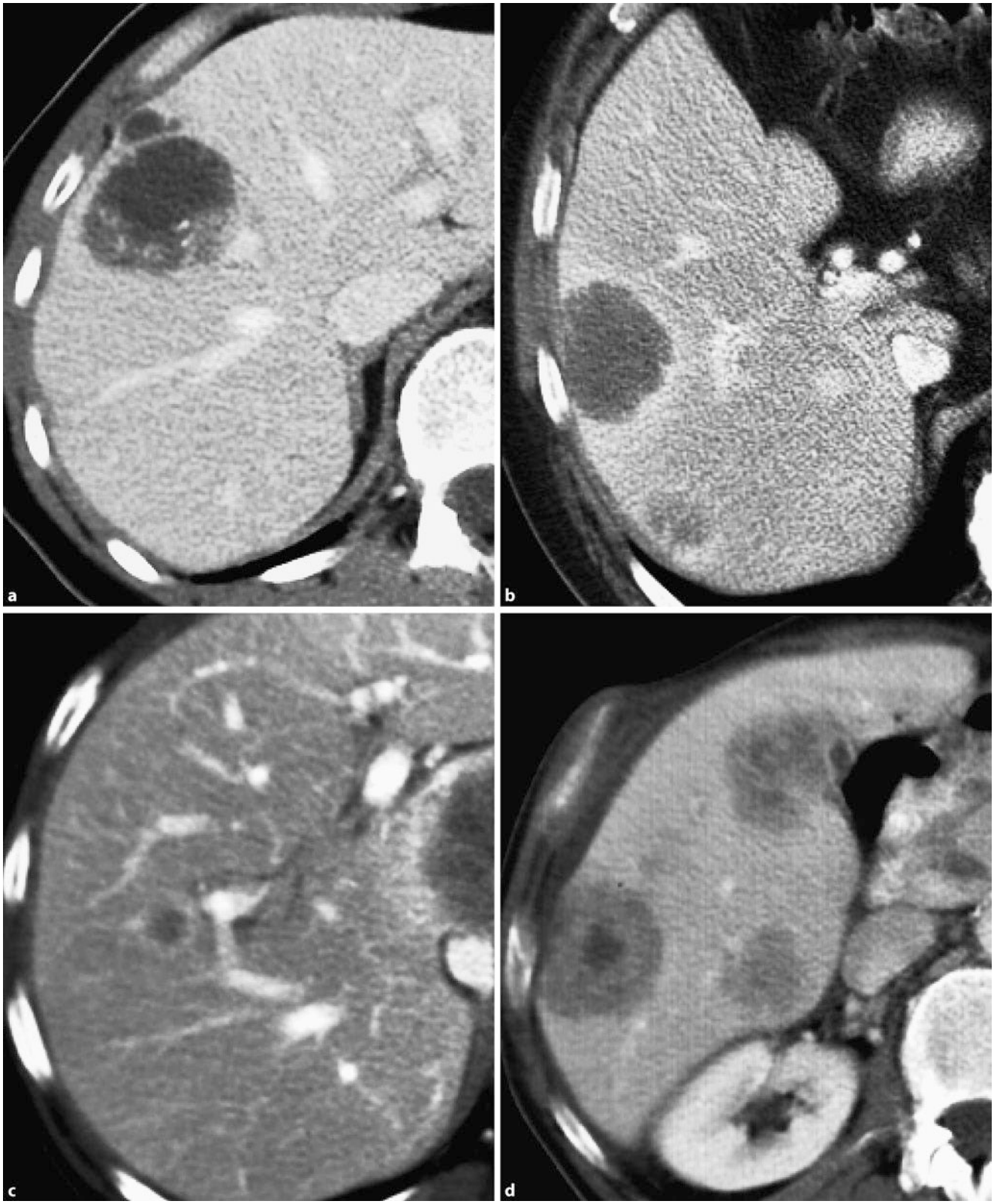

Fig. 24.19a–d Metastases. Different morphologies of liver metastases: cystoid (**a**), hypodense (**b**), ringlike enhancement (**c**), target lesions (**d**)

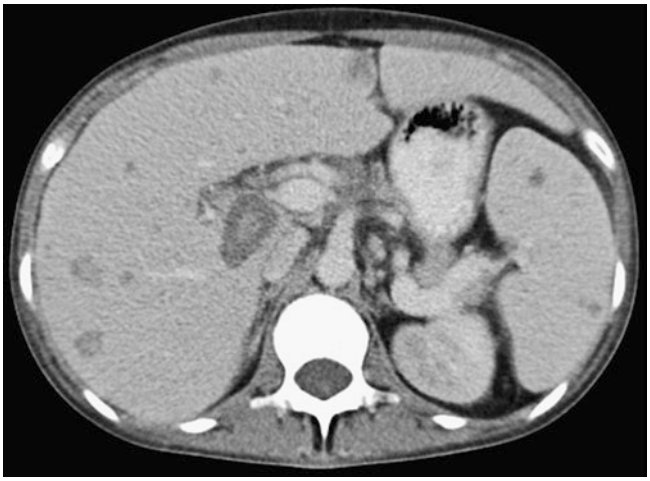

Fig. 24.20 Lymphoma. Note several ill-defined hypodense lesions in the liver and moderately enlarged spleen

24.8.6 Metastases

Metastases account for the majority of malignant liver tumours (about 90%). Haematogenous spread occurs via the portal vein in tumours of the gastrointestinal tract or via the hepatic artery in tumours of the lung, breast, or kidney, for example. In most cases more than one lesion of different sizes are seen, with a predilection for the right liver lobe. Morphology on imaging studies is very variable. On ultrasound, metastases may be hypoechogenic, isechogenic, or hyperechogenic to the liver. A hypoechogenic halo around the lesion is quite characteristic for metastases (Fig. 24.18). On native CT images, most metastases are hypodense. They are hypointense on T1-weighted images and hyperintense ion T2-weighted images in most cases. Due to melanin, metastases from malignant melanoma may be hyperintense on T1-weighted images. After administration of contrast material, metastases often show a peripheral ringlike enhancement or a so-called target sign (Fig. 24.19). In the hepatobiliary phase, metastases do not show an uptake of Gd-EOB-DTPA.

24.9 Hepatic involvement in systemic disorders

24.9.1 Lymphoma

Involvement of the liver in patients with lymphoma is common. In most cases hepatic involvement remains invisible in imaging studies, or only hepatomegaly but no focal lesions are seen. In cases of nodular infiltration, small disseminated nodules may be seen throughout the liver. These usually are hypoechogenic on ultrasound and hypodense on native CT images. On CT and MR imaging, these nodules are markedly hypovascular in most cases and are hypodense/hypointense in the portal venous phase. Splenomegaly and associated enlargement of abdominal lymph nodes are common (Fig. 24.20).

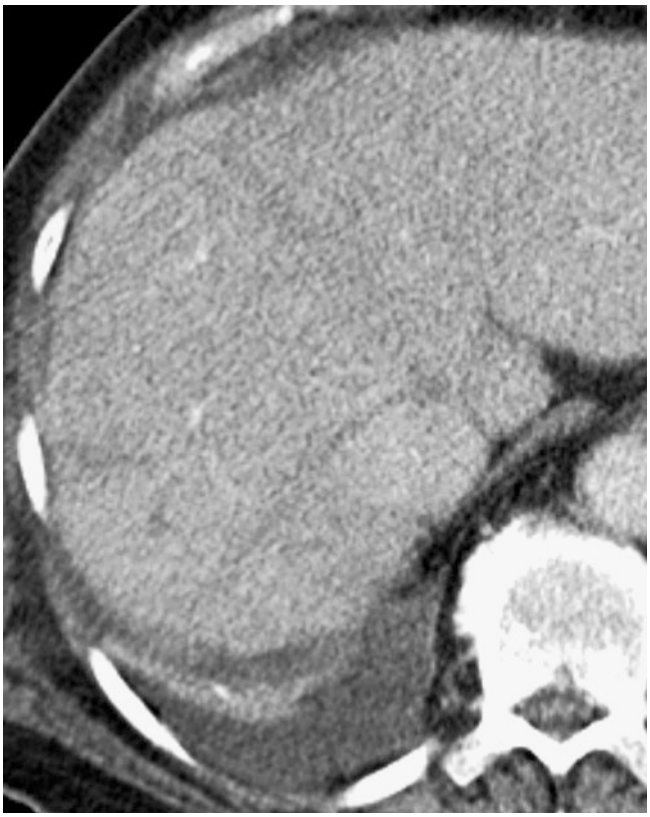

Fig. 24.21 Hepatic laceration is visible as a bandlike hypodensity oriented from the periphery to the hilum of the liver

24.9.2 Posttransplant lymphoproliferative disorder

Posttransplant lymphoproliferative disorder (PTLD) occurs in 2–5% of patients with transplanted organs. Often following an Epstein–Barr virus infection, lymphoid hyperplasia to the point of lymphoma development can be seen. In PTLD extranodal involvement is common. On imaging studies, singular or multiple hypodense hepatic nodes (1–4 cm); diffuse, geographic hepatic infiltration; or a mass in the porta hepatis infiltrating the liver along the bile ducts with associated lymph node enlargement can be seen.

24.10 Liver trauma

The liver is involved in 15–20% of blunt abdominal injuries. Concomitant haemoperitoneum is seen in 80% of cases. CT imaging plays a central role in the acute diagnosis of severely injured patients.

Signs of hepatic injury may be discrete. Occasionally, only relatively small amounts of hyperdense, free intraabdominal fluid and periportal hypoattenuation may be seen. Intraparenchymal and subcapsular haematomas are characterised by areas of inhomogeneous hypodensity within the parenchyma or adjacent to the capsule of the liver (Fig. 24.21). Lacerations of the liver are seen as bandlike or branching, ill-defined hypodensities, often oriented from the periphery to the hilum of the liver.