Everyday Practice

Evaluation of the liver using computed tomography scan

VAIBHAV JAIN, RAJU SHARMA, HARSH KANDPAL

INTRODUCTION

Lesions of the liver are a common clinical problem. Although ultrasonography is the first screening investigation in such patients, a computed tomography (CT) scan is often required for their evaluation. With the introduction of multi-detector row CT (MDCT), it is now possible to image larger volumes at narrower slice collimations; and image the lesions in various phases after administering a contrast medium. All these features result in improved detection and characterization of the lesion.[1] We describe the CT appearance of a spectrum of common conditions that are encountered by a clinician, and briefly discuss these in relation to recent advances in CT technology.

THE NORMAL ANATOMY

The liver is divided 'anatomically' by the falciform ligament into right and left lobes. However, with better understanding of the surgical anatomy, it is now divided into 8 segments, with each segment having a branch of the portal vein, hepatic artery and biliary duct in the centre; and a hepatic vein in the periphery. The most widely followed system is the one proposed by Bismuth–Couinad, which defines a transverse portal vein plane that intersects the liver at the point of bifurcation of the portal vein into right and left branches.[2] Segment I corresponds to the caudate lobe. The 'functional' left hepatic lobe is constituted by segments II to IV. Segments II and III lie lateral and to the left of the left hepatic vein. While segment II lies above the portal vein plane, segment III is below it. Between the left and middle hepatic veins lies segment IV that is divided by the portal vein plane into segments IVa (above) and IVb (below). Segments V to VIII comprise the functional right hepatic lobe. Segments V and VIII lie between the middle and right hepatic veins, with the portal vein separating segment V (below) from segment VIII (above). Similarly, segments VI (below) and VII (above) are separated by the portal vein plane, both of which lie lateral and to the right side of the right hepatic vein.

CT TECHNIQUE

Complete hepatobiliary evaluation requires intravenous administration of a contrast medium followed by multiphasic image acquisition. Two widely recognized phases after administration of the contrast medium are described for hepatic imaging. These are

1. Hepatic arterial phase (HAP): This starts approximately 20 seconds after administration of contrast and lasts for 10–15 seconds. It is ideal for detection of hypervascular liver lesions such as hepatocellular carcinoma (HCC), and for evaluation of the coeliac axis and its branches including the hepatic artery.
2. Portal venous phase (PVP): This starts 60–65 seconds after administration of contrast. The liver shows maximum and homogeneous contrast uptake during this phase. Therefore, this phase is used to characterize the majority of liver lesions.

Non-contrast CT is not necessary as a routine, and is used to detect the presence of fat, calcification or haemorrhage within the lesions. Delayed imaging is also done for specific indications, and is useful in cholangiocarcinomas, haemangiomas and HCC.

DIFFUSE LIVER DISEASES

Fatty liver (hepatic steatosis)

This refers to an excessive accumulation of triglycerides within the hepatocytes. On CT, fatty liver appears as a diffuse low attenuation on non-enhanced scans (Fig. 1). Normally, the liver attenuation is 6–10 Hounsfield units (HU) more than that of the spleen. This relationship is reversed in a fatty liver. The vascular structures may stand out against a background of low attenuation. However, the liver morphology and distribution of blood vessels are unaffected in the radiologically abnormal area. Sometimes the fatty change may not be uniform and only focal areas may be affected. Such areas of focal fatty change or sparing are usually seen in the vicinity of major vascular structures such as the portal vein, or the fissure for the ligamentum venosum and ligamentum

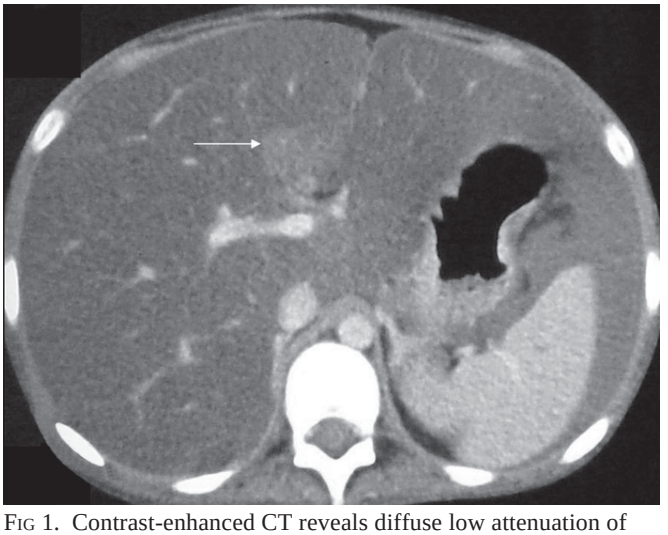

FIG 1. Contrast-enhanced CT reveals diffuse low attenuation of the liver suggestive of hepatic steatosis. The geographical area of increased density in segment IV (arrow) represents a focal area of relative fatty sparing.

All India Institute of Medical Sciences, Ansari Nagar, New Delhi 110029, India

VAIBHAV JAIN, RAJU SHARMA, HARSH KANDPAL
Department of Radiology

Correspondence to RAJU SHARMA; *raju152@yahoo.com*

teres (Fig. 1). They can mimic a focal liver lesion. However, the characteristic location and underlying normal morphology of the liver can help in making a correct diagnosis.

Cirrhosis

This is characterized by fibrosis as a result of hepatocellular injury and nodular regeneration of the liver. Although CT scan may be normal in up to 25% of cases of early cirrhosis,[3] in advanced disease, there is an overall decrease in liver volume with atrophy of the right lobe and relative hypertrophy of the left and caudate lobes. The liver surface becomes nodular, and the hepatic fissure and porta hepatis become wider with low periportal attenuation due to fibrosis (Fig. 2). The liver parenchyma becomes heterogeneous in attenuation on unenhanced scans as well as in the PVP due to altered hepatic perfusion.[4,5] With progressive fibrosis and ongoing repair, nodules are formed, which can be of relatively uniform size—micronodular cirrhosis (common in alcoholic, hepatitis C virus infection and biliary cirrhosis); or can be larger and more variable in size—macronodular cirrhosis (most common in hepatitis B virus infection). The regenerating nodules are usually isodense with the liver parenchyma in all phases of enhancement. Among other nodular lesions are the dysplastic nodules and HCC. There is no specific imaging feature on CT to differentiate a dysplastic nodule (which is a precursor to the development of HCC) from a regenerative nodule.[6] Ancillary features of portal hypertension such as ascites, splenomegaly and porto-systemic collaterals (commonly seen in the distal oesophagus, gastrohepatic ligament and perisplenic region) including recanalized paraumbilical veins can be seen.

Haemochromatosis

Primary haemochromatosis is an autosomal recessive disorder characterized by increased intestinal iron absorption and increased deposition of haemosiderin in the cytoplasm of parenchymal cells in the liver, pancreas, heart and other organs. Secondary haemochromatosis (also referred to as haemosiderosis) results from iron overload and is characterized initially by reticulo-endothelial cell iron deposition. Later, however, it may become pathologically and clinically indistinguishable from primary haemochromatosis.

In both types of haemochromatosis, the liver parenchyma shows a diffuse increase in attenuation and a value >70 HU is considered highly sensitive and fairly specific for the diagnosis (Fig. 3).[7] Portal venous branches and hepatic veins stand out as prominent low-attenuating structures against a hyperattenuating background. The spleen, pancreas, adrenals and lymph nodes also demonstrate increased attenuation in the primary type. In the secondary type, the spleen and bone marrow also show iron deposition due to accumulation of iron in the reticuloendothelial cells, with sparing of the parenchymal cells of the liver and pancreas.

Wilson disease

This is an autosomal recessive disorder of copper metabolism characterized by impaired copper excretion, which is deposited in the liver and basal ganglia of the brain. CT findings in the liver are non-specific, with changes of cirrhosis, which is usually of the macronodular type.

Focal hepatic lesions

Table I shows the appearance of various hepatic lesions in the non-contrast phase and in 3 post-contrast phases. To make a final imaging diagnosis of a lesion, its appearance on all 4 phases has to be taken into account. For example, a haemangioma is hypodense on non-contrast CT, shows peripheral globular enhancement in the arterial phase, gradual centripetal filling in the venous phase and homogeneous retention of contrast in the delayed phase. A simple cyst is hypodense and shows no enhancement in any of the post-contrast phases. Similarly, other focal lesions have typical appearances in various phases and this plays a crucial role in differentiating the lesions from each other. Hence, the enhancement characteristics of a lesion are important for making a diagnosis. In difficult situations, an ultrasound and magnetic resonance imaging may help in the characterization of a lesion.

INFECTIOUS DISEASES OF THE LIVER

Liver abscess

A localized collection of pus in the liver with destruction of liver parenchyma results in an abscess. The common sources of pathogens include the biliary tract (most common), the portal vein, hepatic artery, direct extension from adjacent organs and trauma. The pathogens may be bacteria (*E. coli* being most common), *E. histolytica* and, uncommonly, fungi.

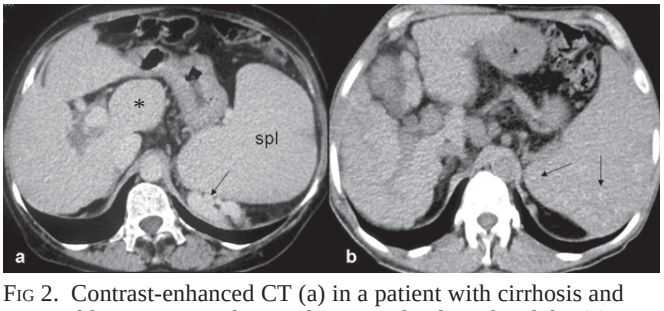

FIG 2. Contrast-enhanced CT (a) in a patient with cirrhosis and portal hypertension shows a hypertrophied caudate lobe (*), enlarged spleen (spl) and large lienorenal collaterals (arrow). In another patient with cirrhosis (b), diffusely nodular liver surface and high density, splenic Gamma Gandy bodies can be seen on a non-contrast CT (arrows).

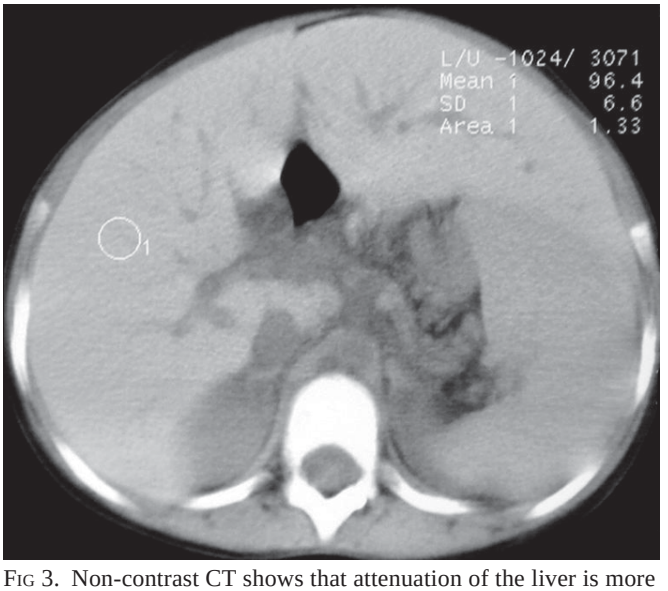

FIG 3. Non-contrast CT shows that attenuation of the liver is more than that of the spleen. The measured mean Hounsfield units value is 96, suggestive of a diagnosis of haemochromatosis.

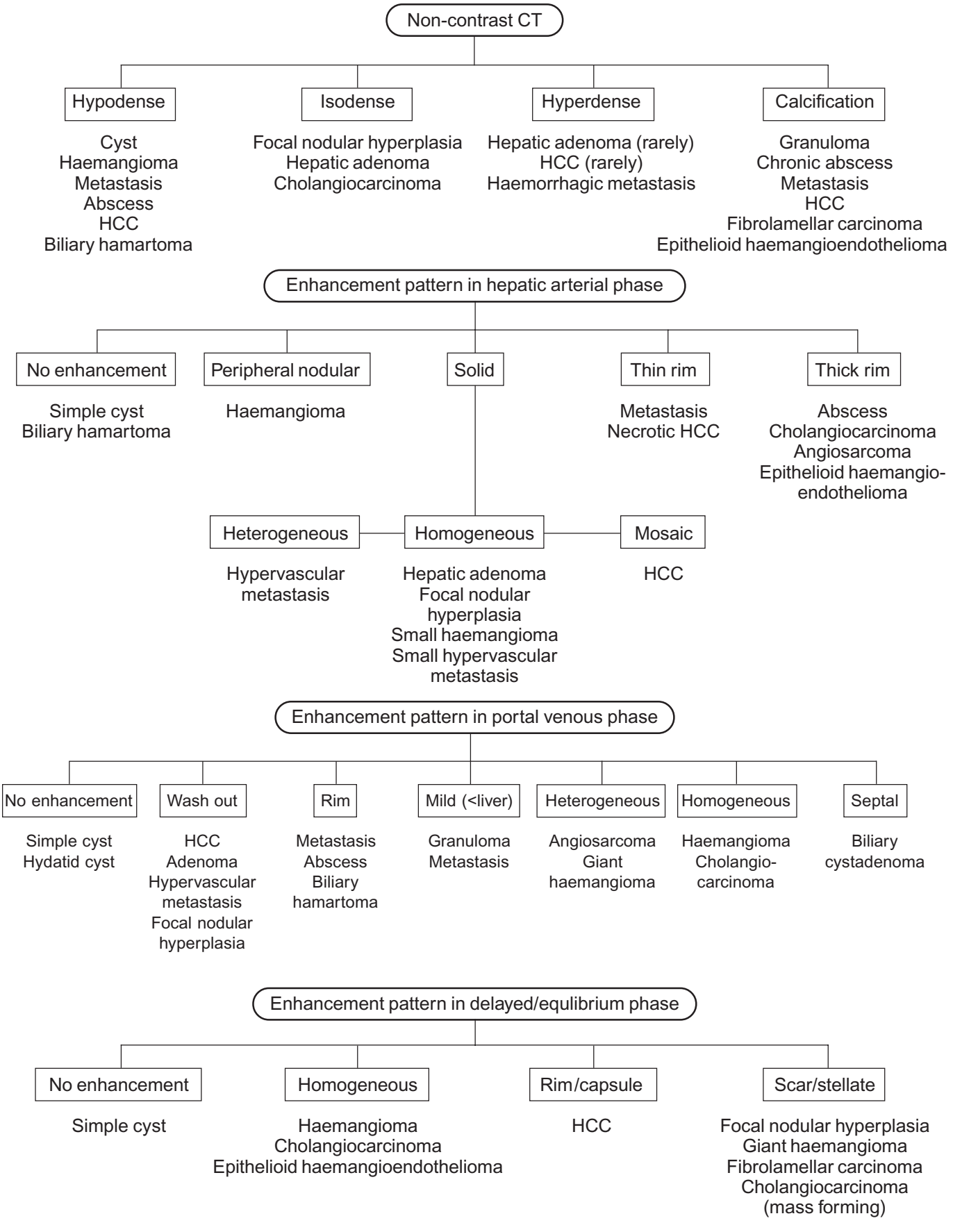

TABLE I. CT appearance of focal liver lesions in different phases (HCC hepatocellular carcinoma)

On contrast-enhanced CT, the lesion is single or multilocular, low attenuation (0–45 HU) mass, with or without a peripheral rim of enhancement.[8] Perilesional low attenuation due to oedema may give an appearance of a 'double target sign' (Fig. 4). The presence of gas within the lesion may indicate an infection by a gas-producing organism, or formation of an enteric communication. A 'cluster sign', in which multiple small abscesses are seen to aggregate or coalesce together, when seen, is helpful in differentiating them from other multiple focal lesions such as liver metastases. Extension/rupture of an abscess can occur, which can be confined to the perihepatic region, or may extend into the peritoneal cavity or pleura. The generation of multiplanar reformats (MPRs) is helpful in demonstrating such extensions, especially the site of diaphragmatic rupture in intrathoracic extension (Fig. 5).

Hydatid cyst

It is the most common parasitic infection of the liver in India. The cyst has 3 layers—outermost pericyst, middle laminated ectocyst and the innermost germinal layer (endocyst), which produces the ectocyst and the scolices. Although ultrasonographic features of hydatid disease are characteristic, a CT scan is often done.

A hydatid cyst appears as a well-defined, unilocular or multilocular, round-to-oval cystic mass, with attenuation similar to water (Fig. 6). Daughter cysts can be seen in the periphery or floating freely inside the mother cyst. Detachment of the ectocyst from the pericyst results in the visualization of hyperdense curvilinear 'membranes' floating inside the low attenuation cyst. The calcification of a cyst wall or septae can be easily seen on CT. Post-contrast, there may be enhancement of the cyst wall and internal septations. The most common complications include rupture and infection. Cyst rupture can be contained within the liver, or can communicate with the biliary radicles or directly into the peritoneal or pleural cavities (Fig. 7). An infected cyst appears more 'solid' on CT, but ultrasonography may show internal echogenic foci, debris and thickened septae.

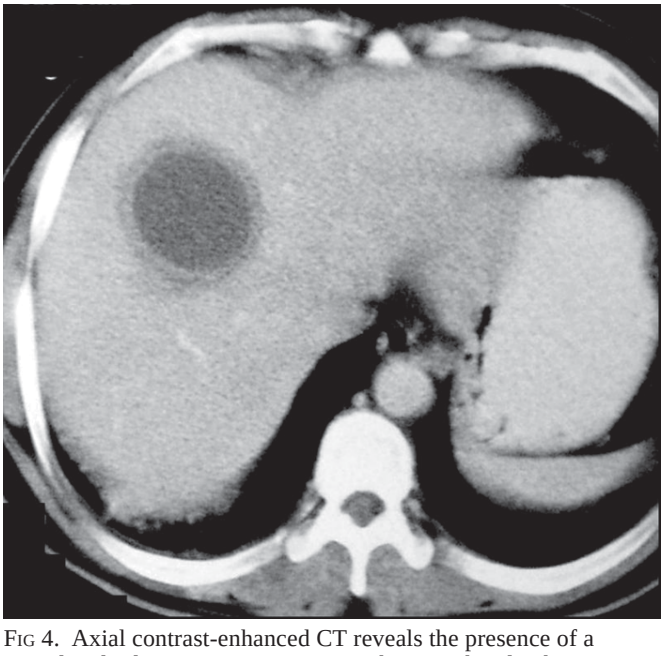

FIG 4. Axial contrast-enhanced CT reveals the presence of a unilocular low-attenuation mass with a peripheral enhancing rim in the right lobe of the liver, suggestive of a liver abscess. The characteristic 'double target sign' due to the presence of a low attenuation peripheral rim as a result of perilesional oedema is also seen.

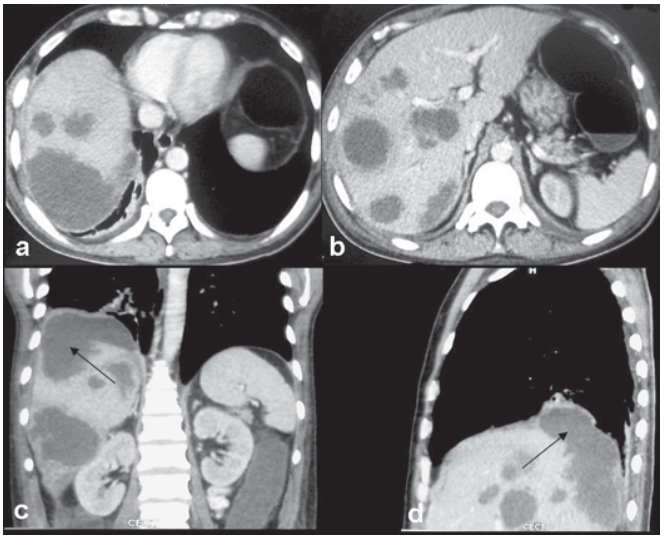

FIG 5. Axial (a, b), coronal (c) and sagittal (d) multiplanar reformat images in a patient show multiple hepatic abscesses with one in segment 8 showing rupture. The site of rupture and the intrathoracic extension is better seen on the coronal and sagittal reformat (arrows) than axial images alone.

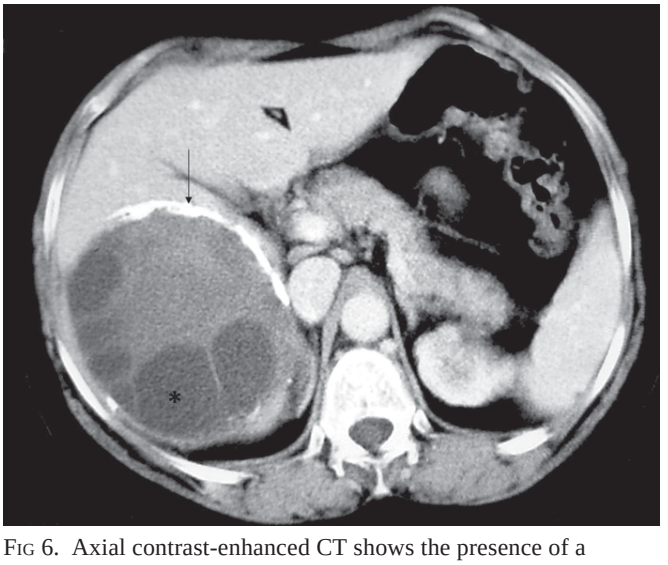

FIG 6. Axial contrast-enhanced CT shows the presence of a characteristic hydatid cyst with solid areas and multiple daughter cysts (asterisk) inside. The wall of the cyst is partially calcified (arrow).

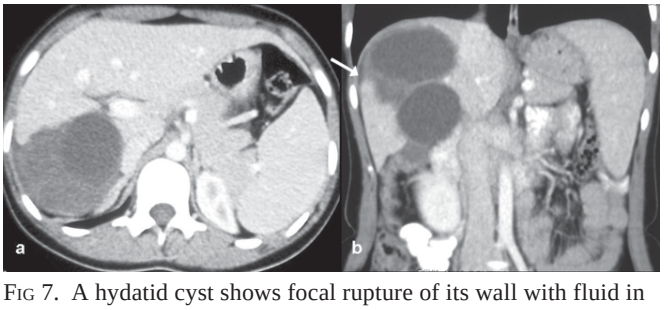

FIG 7. A hydatid cyst shows focal rupture of its wall with fluid in the perihepatic space (arrow).

Tuberculosis

Although the incidence of hepatic involvement in disseminated miliary tuberculosis is high at autopsy, radiological manifestations in the liver are not that common. The most common finding is hepatomegaly. Occasionally, CT scan may show multiple small hypodense nodules (Fig. 8). Localized tubercular abscess or large nodule formation in non-miliary cases of tuberculosis is exceedingly rare. Calcific specks may be seen in long-standing or healed granulomatous disease. The presence of associated lymphadenopathy is common, with or without rim enhancement on post-contrast CT scans.

BENIGN LIVER LESIONS

The common and clinically important lesions include:

1. *Lesions of hepatocyte origin*: hepatocellular adenoma, focal nodular hyperplasia and nodular regenerative hyperplasia.
2. *Lesions of the bile duct epithelium*: simple hepatic cysts, polycystic liver disease, biliary hamartoma, biliary cystadenoma.
3. *Lesions of the mesenchymal cells*: haemangioma, lipomatous tumours.

Hepatocellular adenoma

It is a rare solid primary liver tumour which is related to the use of oral contraceptives in women and anabolic steroids in men. It may also occur in association with glycogen storage disease, with an incidence as high as 40% in type I (Von Gierke) disease. These tumours are mostly solitary, but can be multiple (hepatic adenomatosis). Histologically, adenomas contain slightly atypical hepatocytes without any bile duct cells or Kupffer cells.

On non-contrast CT, adenomas are isodense to the liver parenchyma. They may contain hyperdense areas corresponding to haemorrhage and hypodense areas due to presence of necrosis or fat (Fig. 9). The lesions show prominent intense enhancement in the HAP reflecting hypervascularity of the lesion. Rapid-contrast washout in the PVP results in this lesion becoming isodense to the liver, and even hypodense on delayed phase.[5,8]

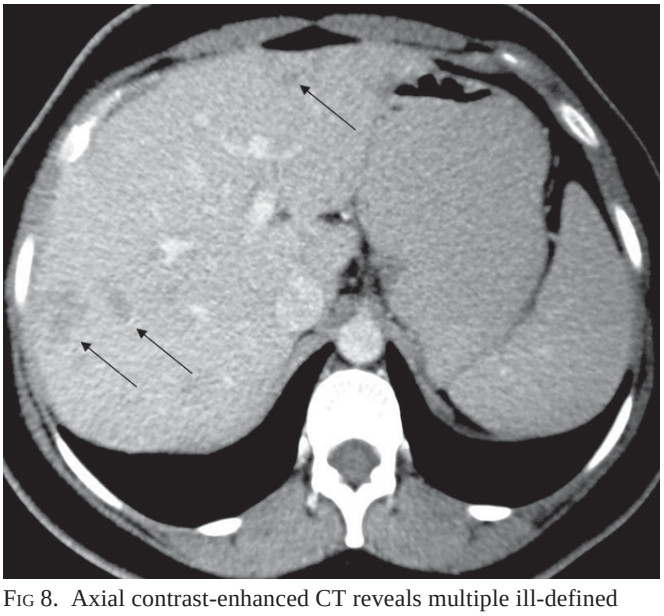

FIG 8. Axial contrast-enhanced CT reveals multiple ill-defined hypodense areas in both lobes of the liver (arrows). Needle biopsy of these lesions revealed the presence of caseating epithelioid granulomas suggestive of tuberculosis.

Focal nodular hyperplasia (FNH)

It is the second most common benign liver tumour after haemangioma. It is usually solitary, <5 cm in size, and is most commonly seen in middle-aged women in their third to fifth decades of life. It contains hepatocytes, bile duct elements, Kupffer cells and fibrous tissue. It is non-encapsulated, and contains a central scar comprising myxoid and vascular tissue, surrounded by hyperplastic hepatocytes.

Focal nodular hyperplasia appears isodense to the liver on unenhanced CT, and may be apparent only due to the mass effect on adjacent structures. It shows intense enhancement in the HAP (Fig. 10) and one or more enlarged feeding arteries may be seen in a peripheral, septal or central location within the lesion. The

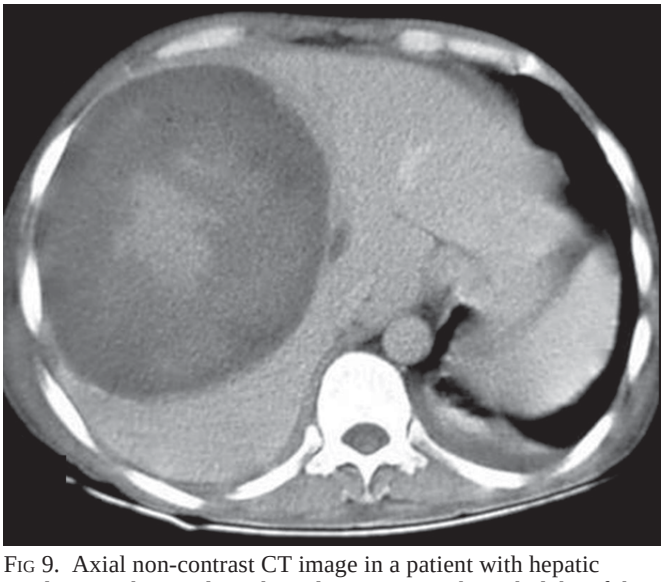

FIG 9. Axial non-contrast CT image in a patient with hepatic adenoma shows a large hypodense mass in the right lobe of the liver. The hyperdense areas within the mass represent areas of haemorrhage.

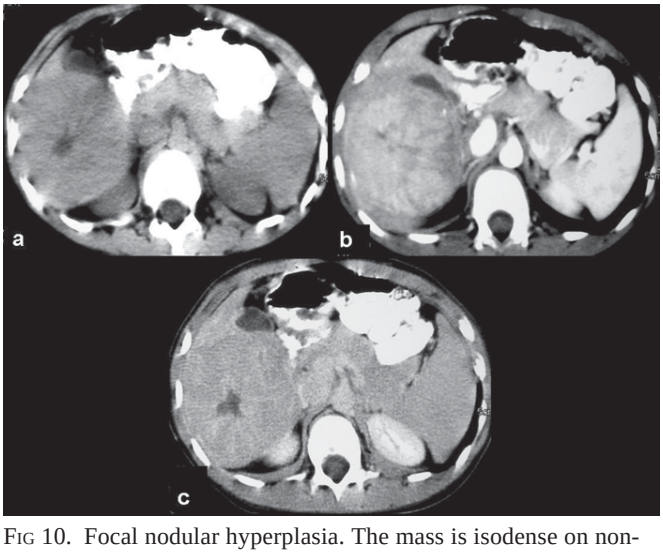

FIG 10. Focal nodular hyperplasia. The mass is isodense on non-contrast images (a) and shows intense enhancement in the arterial phase (b) with rapid washout and isodensity to the liver on the venous phase (c) of enhancement. A central hypodense non-enhancing stellate scar is also seen.

feeding artery usually divides into multiple small penetrating branches, resulting in a characteristic reticular or net-like pattern of enhancement in the HAP.[4] The lesion shows rapid washout in the PVP, becoming isodense to the liver. Several small anomalous sinusoids and draining veins may be seen surrounding the lesion which drain into a hepatic vein; and, unlike HCC, portal venous drainage is unusual for FNH. A pseudocapsule is rarely seen as a hypodense rim in the HAP and hyperdense in the PVP. It is more common in larger lesions, and is composed of dilated surrounding vessels or sinusoids, or compressed hepatic parenchyma. A central scar is characteristic of FNH. Linear septations may be seen radiating from the central scar peripherally to the surface of the lesion, resulting in the lesion being lobulated. The scar is hypodense on non-contrast, HAP and PVP, and shows typical enhancement on delayed (5–10 minutes) scans.

Nodular regenerative hyperplasia

This is characterized by the presence of multiple regenerative nodules in the liver without any fibrosis. Therefore, it is also known as non-cirrhotic nodular hyperplasia. The CT appearance varies from a completely normal scan due to the presence of multiple slightly hypodense nodules ranging from few millimetres to several centimetres in size, which may cause a bulge on the surface of the liver.[5] It needs to be differentiated from adenomatous hyperplastic nodules (AHN; dysplastic nodules), which is a premalignant condition seen in cirrhotic livers.

Simple liver cysts

Liver cysts are developmental lesions that do not communicate with the biliary tree. A simple cyst is a cyst of bile duct origin, defined as a solitary, unilocular cyst, which has an epithelial lining of a single layer of cuboidal cells. Ultrasonography is highly accurate in detecting these as echo-free, thin-walled lesions. On CT scan, they appear as well-defined, round-to-oval, water attenuation (0–10 HU) lesions with smooth and thin walls (Fig. 11). There are no internal solid areas and there is lack of enhancement on any phase after contrast administration. An infected or haemorrhagic cyst may show internal septations or peripheral enhancement.

Polycystic liver disease

It is a spectrum of fibro-polycystic disease of the liver, ranging from polycystic liver disease on one end to congenital hepatic fibrosis on the other. On CT, polycystic liver disease is characterized by the presence of multiple, variable-sized simple cysts that may show haemorrhage or calcification. It is often associated with adult autosomal dominant polycystic kidney disease (Fig. 12). Congenital hepatic fibrosis is characterized by the presence of increased periductal fibrosis and aberrant bile duct proliferation. The CT features of congenital hepatic fibrosis include morphological variations of the liver (i.e. hypertrophy of the left lateral segment, normal size or hypertrophy of the left medial segment and atrophy of the right lobe), features of portal hypertension such as varices and splenomegaly, associated ductal plate malformations such as Caroli disease, choledochal cyst and biliary hamartomas, and renal abnormalities such as polycystic renal disease and renal calcifications.[9]

Biliary hamartoma

These originate from embryonic bile ducts that fail to involute and are also known as von Meyenburg complexes.[10] They are generally asymptomatic and detected incidentally. Non-enhanced CT images show multiple, low-attenuating, cystic lesions distributed throughout both lobes of the liver. They usually do not show any enhancement. The most characteristic feature that differentiates them from simple liver cysts is that hamartomatous cysts are <1.5 cm in diameter.[10]

Biliary cystadenoma

It is a rare, slow-growing, cystic tumour of middle-aged women, which is considered premalignant. Although the majority are intrahepatic (~85%), a few extrahepatic lesions have also been reported.[11] The CT appearance is of a solitary, cystic mass with a thick capsule and multiple internal septations that are thin and show contrast enhancement.[11] They may contain a mural nodule; however, polypoid pedunculated excrescens are more common in biliary cystadenocarcinomas.

Haemangioma

It is the most common benign neoplasm of the liver, is mesodermal in origin and composed of blood-filled cavernous vascular spaces lined by a single layer of flat endothelium. In the HAP, these show peripherally enhancing nodules, and an attenuation value within those nodules that is similar to the abdominal aorta, is considered diagnostic of the lesion. They show a progressive centripetal fill in the PVP and delayed phases, appearing hyperdense to the liver

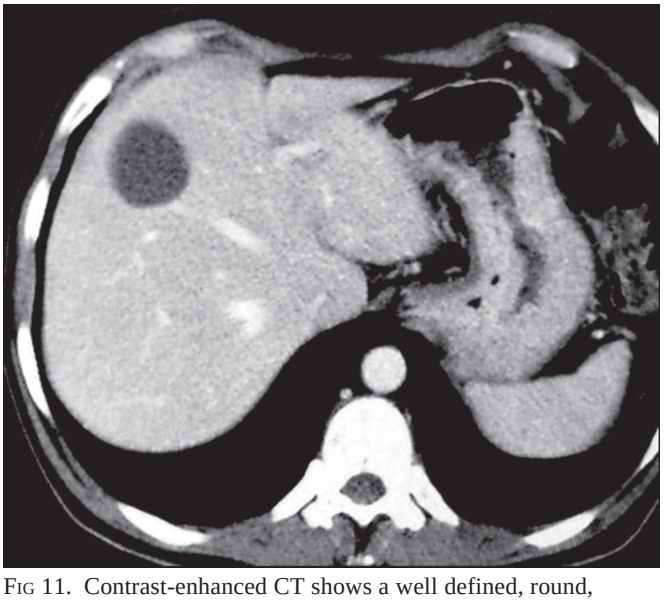

FIG 11. Contrast-enhanced CT shows a well defined, round, unilocular, low-density, non-enhancing lesion suggestive of a simple cyst in the right lobe of the liver.

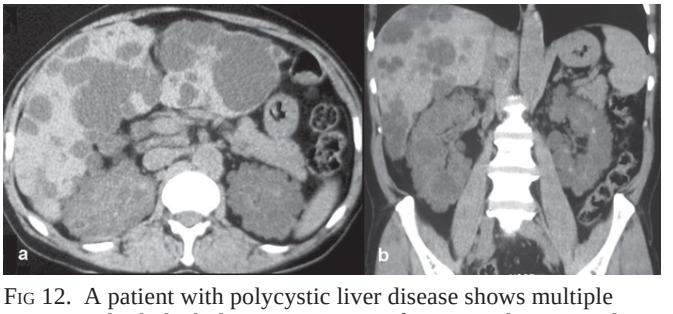

FIG 12. A patient with polycystic liver disease shows multiple cysts in both the kidneys suggestive of associated autosomal dominant polycystic kidney disease.

(Fig. 13). Smaller lesions show a complete fill in; however, larger lesions may have a central non-enhancing area composed of fibrotic scar tissue. Some atypical enhancement patterns may also be seen in haemangiomas.[4] Complete hyalinization may result in non-enhancement of a haemangioma even on delayed images due to obliteration of vascular sinusoids. Small haemangiomas may also show a rapid enhancement, becoming hyperdense in the HAP and PVP, when they become difficult to distinguish from hypervascular metastases. The presence of transient peritumoral enhancement favours a diagnosis of haemangioma.[13] Giant haemangiomas are defined as lesions 4–6 cm in diameter, and may even replace an entire lobe of the liver. These may be symptomatic causing abdominal discomfort from liver enlargement and capsular distension.

MALIGNANT LIVER LESIONS

Primary malignant hepatic tumours include a wide variety of neoplasms of hepatocellular, cholangiocellular and mesenchymal origin. Secondaries from an extrahepatic malignancy are by far the most common hepatic neoplasm.

Hepatocellular carcinoma

HCC is the most common primary liver malignancy. CT scan is the most common investigation for HCC, and MDCT has enabled accurate multiphasic evaluation, leading to improved detection and characterization of the lesions.[4,15] On non-contrast CT, most HCCs are seen as solitary or multiple, low-attenuating lesions. In HAP images, most HCCs are hypervascular and show intense, homogeneous enhancement throughout the tumour (Fig. 14). The reason for this is that most HCCs receive a majority of their blood supply from the hepatic artery, whereas the normal liver receives approximately 80% supply from the portal vein.[16] Larger tumours show a heterogeneous or mosaic pattern of enhancement and may contain areas of necrosis and haemorrhage. Most of the lesions show a pseudocapsule or fibrous septae (80%), seen as a hypodense rim in the HAP.[5] In the PVP, HCCs show rapid washout of contrast and appear hypodense to isodense to the liver parenchyma. HCCs frequently invade the portal vein (~40% cases), hepatic veins and IVC (~15% cases)[2] and these are seen as hypodense filling defects within the enhancing vein (Fig. 15). A tumour thrombus can be differentiated from a bland thrombus by the presence of enhancement within the thrombus in the HAP (Fig. 16). The presence of portal vein invasion is a useful diagnostic clue for HCC as <8% of malignant portal vein thrombi are due to other malignancies.[15]

Extrahepatic spread is rare; the lung is the most common site followed by lymph nodes in the periportal and peripancreatic location. Delayed phase images may show enhancement of the pseudocapsule and fibrous septae in the lesion. High resolution

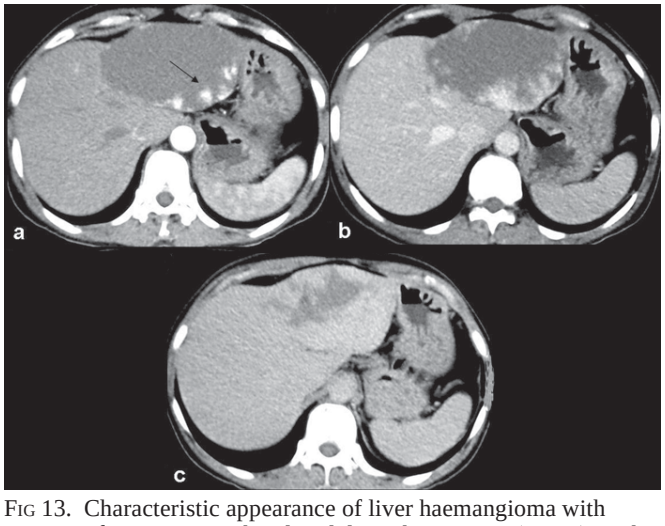

FIG 13. Characteristic appearance of liver haemangioma with areas of intense, peripheral nodular enhancement (arrow) on the arterial phase (a) that show progressive centripetal enhancement in the venous (b) and delayed (c) phases. The mass shows incomplete fill-in with a central hypodense area representing a fibrotic scar.

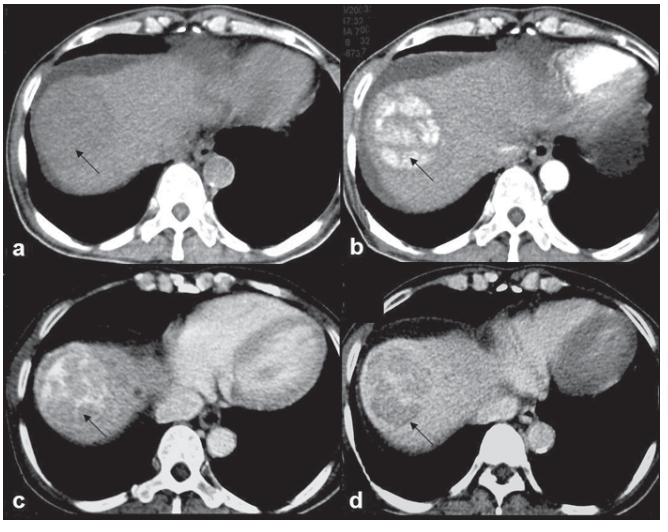

FIG 14. A patient with cirrhosis shows a hypodense mass in the right lobe of the liver on non-contrast image (a). The mass shows an intense, mosaic pattern of enhancement on the arterial phase (b), rapid washout on the venous phase (c), and an enhancing pseudocapsule on delayed images (arrow-d), characteristic of a hepatocellular carcinoma.

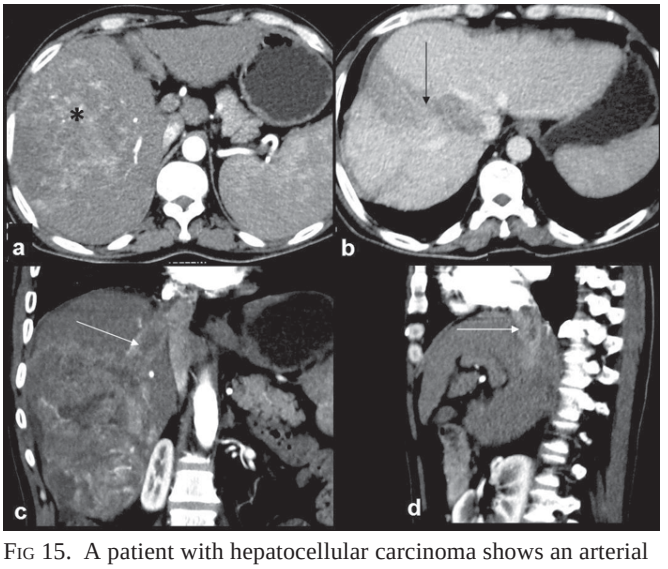

FIG 15. A patient with hepatocellular carcinoma shows an arterial enhancing mass in the right lobe of the liver (asterisk-a). The venous phase axial image (b) reveals a thrombus in the middle hepatic vein with extension into the inferior vena cava (arrow). The presence of arteriovenous shunting and enhancing tumour thrombus is better appreciated on coronal (c) and sagittal (d) maximum intensity projection images (arrows in c, d).

MPRs made possible by MDCT are particularly useful in detecting small subcapsular lesions at the diaphragmatic surface of the liver, and help in planning surgical resection.[16]

Fibrolamellar HCC

This is a tumour of young adults without underlying cirrhosis or other risk factors. On non-contrast CT, fibrolamellar HCC is usually a large (5–20 cm), well-defined, lobulated, hypodense lesion. The radiological clue to the diagnosis of fibrolamellar HCC is the central fibrous scar that may be hypodense with typical central stellate calcification in up to 55% of cases (Figs 17 and 18). Fibrolamellar HCC shows prominent heterogeneous enhancement in the HAP and PVP, and becomes more homogeneous in delayed images (Fig. 17). Haemorrhage and necrosis are rare, and small satellite lesions may be seen in 10%–15% of cases. Regional adenopathy occurs in 50%–70% (Fig. 18) and distant metastases are rare. Delayed images may show the typical appearance of a fibrolamellar HCC with an enhancing scar within a hypodense tumour parenchyma.[4,14,17]

Epithelioid haemangioendothelioma

This is a rare tumour of vascular origin, developing almost exclusively in adults, with a slight preponderance in women, and has a better prognosis. Pathologically, multiple nodular lesions are present, which are typically distributed in the liver periphery, with a tendency to coalesce. Non-contrast CT shows the presence of multiple, peripheral, low-attenuation lesions, which may coalesce to form a more diffuse pattern. Calcification may be seen in up to 30% of cases. Peripheral enhancement surrounding the hypodense fibrous core is seen on post-contrast images (Fig. 19). The frequently associated findings of adjacent retraction of the liver capsule and hypertrophy of the normal liver, though non-specific, suggest the possibility of this diagnosis.[18]

Angiosarcoma

Although rare, hepatic angiosarcoma is the most common sarcoma

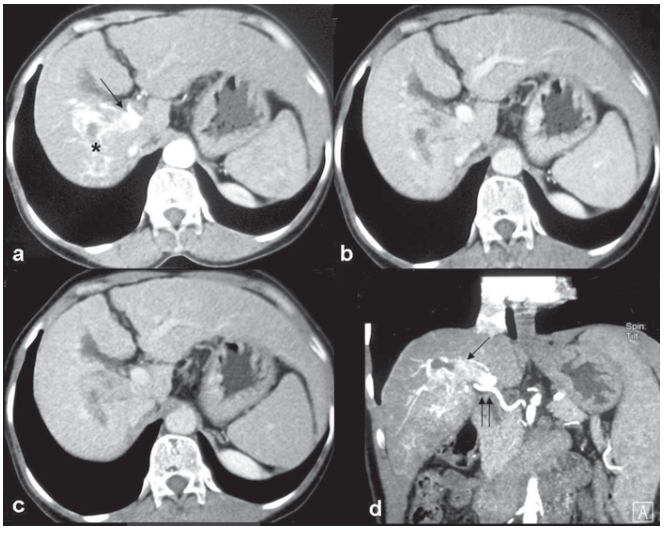

FIG 16. Arterial (a), venous (b) and delayed (c) phase images reveal an arterial enhancing hepatocellular carcinoma (asterisk) with portal venous shunting and enhancing tumour thrombus (arrows). Portal venous shunting and tumour thrombus are much better seen on the arterial phase maximum intensity projection images (arrow in d) that also show a hypertrophied hepatic artery (double arrows) supplying the tumour.

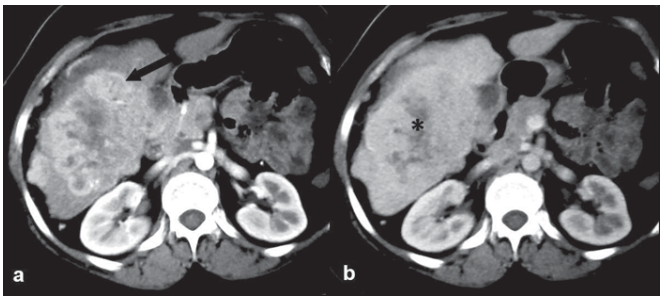

FIG 17. Axial CT images show an intensely enhancing mass (solid arrow) in the arterial phase (a) with rapid washout in the venous phase (b). The mass shows an irregular non-enhancing scar (asterisk) and focal hepatic capsular retraction. Histopathology revealed a fibrolamellar hepatocellular carcinoma.

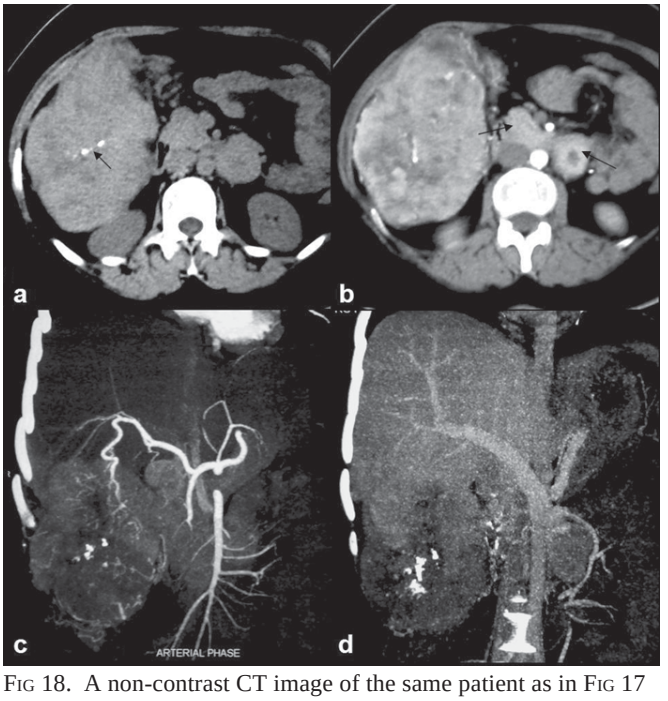

FIG 18. A non-contrast CT image of the same patient as in FIG 17 (a) shows presence of calcification within the scar. Arterial phase image (b) at a different level shows an intensely enhancing mass as well as similar morphology retroperitoneal lymph nodes (arrows) suggestive of nodal metastases. Maximum intensity projection images in the arterial (c) and venous (d) phases demonstrate the relationship of the mass with branches of the hepatic artery and portal vein.

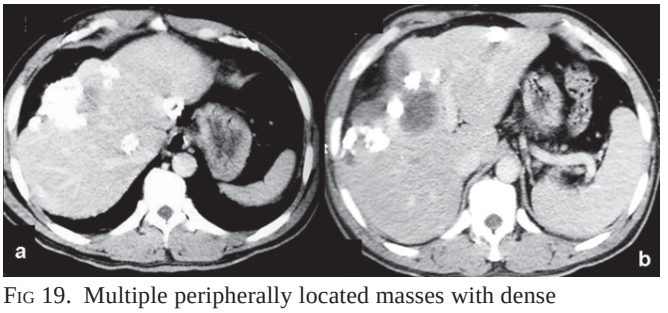

FIG 19. Multiple peripherally located masses with dense calcification are seen in both lobes of the liver. The masses are hypodense with peripheral enhancement and are associated with hepatic capsular retraction. These findings are indicative of an epithelioid haemangioendothelioma.

of the liver, accounting for about 2% of all primary liver neoplasms. On non-contrast CT, angiosarcoma is usually hypodense. When due to thorotrast exposure, there is a typical high metallic density in the background liver, abdominal nodes and the spleen (small and shrunken). Marked peripheral enhancement is seen after contrast administration which may show peripheral spread and inhomogeneity on delayed images, thus making differentiation from a liver haemangioma difficult.[14] In the absence of stigmata of thorotrast exposure, the imaging findings are non-specific and can mimic metastases. Splenic metastases and the presence of haemoperitoneum favour a diagnosis of angiosarcoma.[19]

Intrahepatic (peripheral) cholangiocarcinoma

This accounts for 10% of all cholangiocarcinomas and arises in the small intrahepatic ducts. Based on its growth characteristics, cholangiocarcinoma can be mass forming, periductal infiltrating and intraductal growing.[20] Peripheral intrahepatic cholangiocarcinomas are usually mass forming. On multiphasic CT examination, these tumours show a thin rim-like or thick band-like peripheral enhancement during the HAP or PVP, with progressive and concentric filling in the delayed phase.[21] Delayed images are important for diagnosis as some cholangiocarcinomas are apparent only in this phase. The associated findings include capsular retraction, and dilatation and thickening of the peripheral intrahepatic bile ducts (Fig. 20).

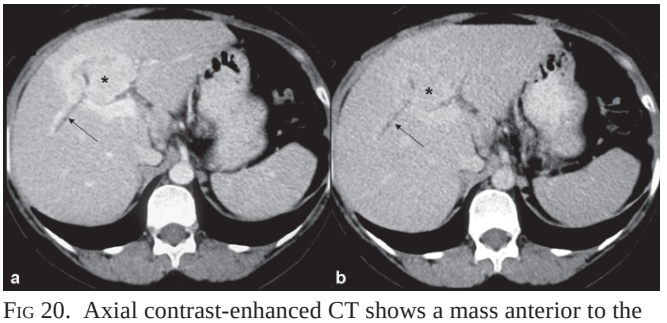

FIG 20. Axial contrast-enhanced CT shows a mass anterior to the portal vein (asterisks) that shows enhancement in the venous (a) as well as delayed (b) phases. Associated biliary ductal dilatation (arrows) suggests the possibility of an intrahepatic cholangiocarcinoma.

Secondary liver malignancies

Liver is the second most common site for metastases after the regional lymph nodes. The colon, breast, pancreas, lung, stomach and sarcomas are the most common primaries which metastasize to the liver.[4] As aggressive treatment regimens including liver resection are offered to patients with some types of liver metastases, an accurate knowledge of the size, number and location of lesions is important.[2,22] Accurate knowledge of the feeding vessels and veins is not only important for segmental resection, but also plays a vital role in planning minimally invasive techniques such as radiofrequency ablation.[4]

On non-contrast CT, most metastases are hypodense to the liver parenchyma. Calcification may be seen in metastases from mucinous gastrointestinal tract carcinomas, ovarian, renal and thyroid carcinomas, and in chemotherapeutically treated hypervascular lesions (e.g. carcinoid metastases). Most metastases are hypovascular, and after contrast administration, they may be seen as rim-enhancing lesions during the HAP (Fig. 21) and become hypoattenuating during the PVP. A majority of lesions are well defined and round-to-oval in shape. However, larger lesions are more irregular and heterogeneous, with ill-defined margins. Only 10%–15% of metastatic lesions are hypervascular, commonly from primaries such as renal cell carcinoma, thyroid carcinoma, islet cell tumours, carcinoids and melanoma. Such lesions show moderate to intense enhancement during the arterial phase, and hence multiphasic CT examination is more useful for the detection of such lesions (Fig. 22). Multiplanar volume rendering and maximum intensity projection techniques allow for evaluation of feeding vessels, adjacent major veins and vascular variants with an accuracy similar to, or even better than, digital angiography.[4,23,24] A summary of the salient features of various focal liver lesions is given in Table II.

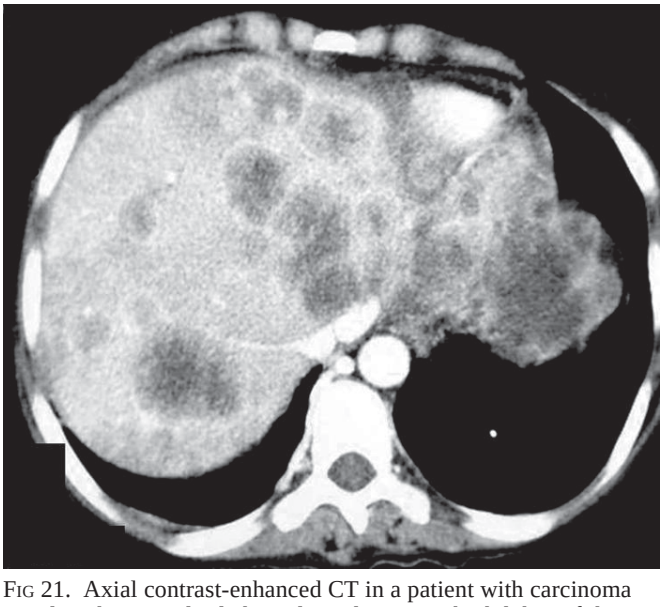

FIG 21. Axial contrast-enhanced CT in a patient with carcinoma colon shows multiple hypodense lesions in both lobes of the liver suggestive of hypovascular metastases.

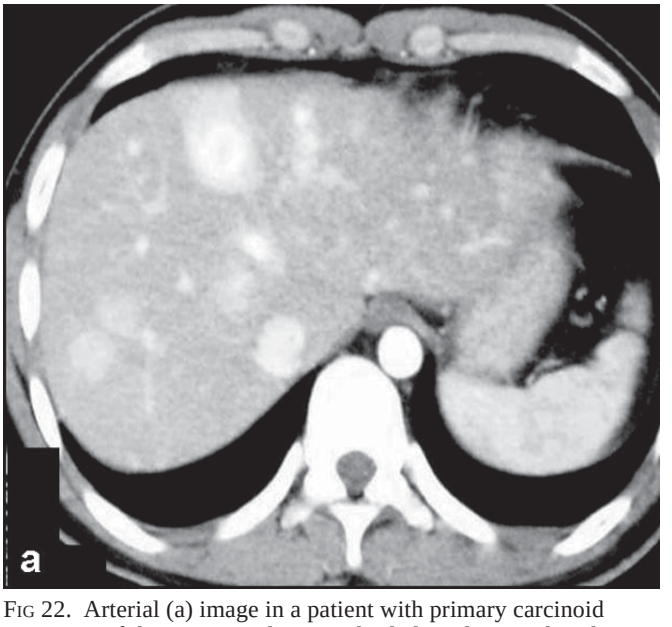

FIG 22. Arterial (a) image in a patient with primary carcinoid tumour of the pancreas shows multiple liver lesions that show intense arterial enhancement of hypervascular metastases.

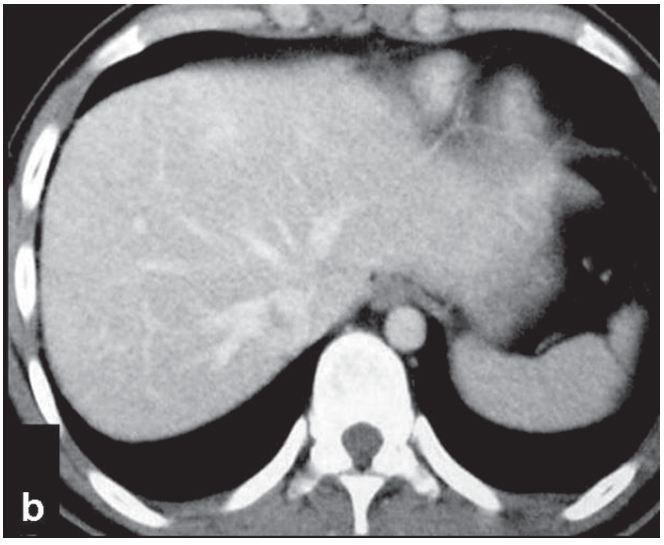

FIG 22. Venous phase (b) images in a patient with rapid venous washout suggestive of hypervascular metastases. Such hypervascular lesions can be missed if only a contrast-enhanced CT that is acquired in the venous phase is performed.

CONCLUSION

CT scan is a widely available and robust modality for evaluation of the liver and remains the modality of choice for many liver disorders. Advances such as multislice CT allow multiphasic scanning of the liver, further enhancing its ability to detect and characterize liver lesions.

TABLE II. Summary of CT imaging features of focal liver lesions

Focal liver lesion	Salient imaging features
Cysts	Anechoic (on US), hypodense (0–20 HU), no CE, may be associated with polycystic kidney disease
Hydatid cyst	Single/multiloculated, daughter cysts, membranes, hydatid sand
Liver abscess	Hypodense, thick rim enhancement, ± gas
Tuberculoma	Hypodense (solitary/multiple), associated with lymphadenopathy, splenic lesions, healed lesions show calcification
Hepatic adenoma	Isodense, intense arterial phase enhancement, capsule present
Focal nodular hyperplasia	Spoke-wheel pattern (colour Doppler US), isodense, intense arterial phase enhancement, scar in delayed phase, no capsule
Biliary hamartoma	Multiple, hypodense, < 1.5 cm in size, rim CE
Haemangioma	Hyperechoic (on US), triad: peripheral nodular arterial enhancement, gradual centripetal filling, density similar to aorta in all post-contrast phases
Cystadenoma	Hypodense, thick capsule, septae which may enhance; mural nodule or solid areas in cystadenocarcinoma
Hepatocellular carcinoma	Cirrhosis, CE in arterial phase, washout in venous phase, capsule in delayed phase, arterioportal shunting, vascular invasion
Fibrolamellar carcinoma	Young women, non-cirrhotic, calcification present, CE in arterial phase, occasional CE of scar in delayed phase
Haemangioendothelioma	Multiple nodules, arterial enhancement, persistent CE in delayed phase, calcification, capsular retraction
Angiosarcoma	History of exposure to thorotrast, peripheral CE in arterial phase, associated with splenic lesions, haemoperitoneum
Cholangiocarcinoma	Isodense, intrahepatic biliary radicle dilatation, atrophy, ±rim enhancement in HAP/PVP, CE in delayed phase, capsular retraction
Metastasis	Solitary/multiple, rim CE in HAP

CE contrast enhancement US ultrasonography HU Hounsfield units

REFERENCES

1 Foley WD, Mallisee TA, Hohenwalter MD, Wilson CR, Quiroz FA, Taylor AJ. Multiphase hepatic CT with a multirow detector CT scanner. *AJR Am J Roentgenol* 2000;**175:**679–85.

2 Gazelle GS, Saini S, Mueller PR (eds*). Hepatobiliary and pancreatic radiology—Imaging and intervention.* Stuttgart:Georg Thieme Verlag; 1998.

3 Dodd GD 3rd, Baron RL, Oliver JH 3rd, Federle MP. Spectrum of imaging findings of the liver in end-stage cirrhosis: Part II. Focal abnormalities. *AJR Am J Roentgenol* 1999;**173:**1185–92.

4 Kamel IR, Liapi E, Fishman EK. Liver and biliary system: Evaluation by multidetector CT. *Radiol Clin North Am* 2005;**43:**977–97.

5 Berry M, Suri S, Chowdhury V, Mukhopadhyay S (eds). *Diagnostic radiology: Gastrointestinal and hepatobiliary imaging.* Second edition. New Delhi:Jaypee Brothers Medical Publishers; 2004.

6 Imai Y, Murakami T, Yoshida S, Nishikawa M, Ohsawa M, Tokunaga K, *et al.* Superparamagnetic iron oxide-enhanced magnetic resonance images of hepatocellular carcinoma: Correlation with histological grading. *Hepatology* 2000;**32:**205–12.

7 Venbrux AC, Friedman AC. Diffuse hepatocellular diseases, portal hypertension, and vascular diseases. In: Friedman AC, Aachman AH (eds). *Radiology of the liver, biliary tract and pancreas.* St Louis:Mosby Year Book; 1994:49–167.

8 Kawamoto S, Soyer PA, Fishman EK, Bluemke DA. Nonneoplastic liver disease: Evaluation with CT and MR imaging. *Radiographics* 1998;**18:**827–48.

9 Zeitoun D, Brancatelli G, Colombat M, Federle MP, Valla D, Wu T, *et al*. Congenital hepatic fibrosis: CT findings in 18 adults. *Radiology* 2004;**231:**109–16.

10 Mortele KJ, Ros PR. Cystic focal liver lesions in the adult: Differential CT and MR imaging features. *Radiographics* 2001;**21:**895–910.

11 Palacios E, Shannon M, Solomon C, Guzman M. Biliary cystadenoma: Ultrasound, CT, and MRI. *Gastrointest Radiol* 1990;**15:**313–16.

12 Jang HJ, Choi BI, Kim TK, Yun EJ, Kim KW, Han JK, *et al.* Atypical small hemangiomas of the liver: 'Bright dot' sign at two-phase spiral CT. *Radiology* 1998;**208:**543–8.

13 Kim T, Federle MP, Baron RL, Peterson MS, Kawamori Y. Discrimination of small hepatic hemangiomas from hypervascular malignant tumors smaller than 3 cm with three-phase helical CT. *Radiology* 2001;**219:**699–706.

14 Fernandez MP, Redvanly RD. Primary hepatic malignant neoplasms. *Radiol Clin North Am* 1998;**36:**333–48.

15 Yu SC, Yeung DT, So NM. Imaging features of hepatocellular carcinoma. *Clin Radiol* 2004;**59:**145–56.

16 Kopp AF, Heuschmid M, Claussen CD. Multidetector helical CT of the liver for tumor detection and characterization. *Eur Radiol* 2002;**12:**745–52.

17 McLarney JK, Rucker PT, Bender GN, Goodman ZD, Kashitani N, Ros PR. Fibrolamellar carcinoma of the liver: Radiologic–pathologic correlation. *Radiographics* 1999;**19:**453–71.

18 Lyburn ID, Torreggiani WC, Harris AC, Zwirewich CV, Buckley AR, Davis JE, *et al.* Hepatic epithelioid hemangioendothelioma: Sonographic, CT and MR imaging appearances. *AJR Am J Roentgenol* 2003;**180:**1359–64.

19 Buetow PC, Buck JL, Ros PR, Goodman ZD. Malignant vascular tumors of the liver: Radiologic–pathologic correlation. *Radiographics* 1994;**14:**153–66.

20 Liver Cancer Study Group of Japan. *The general rules for the clinical and pathological study of primary liver cancer.* 4th ed. Tokyo:Kanehara; 2000.

21 Choi BI, Lee JM, Han JK. Imaging of intrahepatic and hilar cholangiocarcinoma. *Abdom Imaging* 2004;**29:**548–57.

22 Paley MR, Ros PR. Hepatic metastases. *Radiol Clin North Am* 1998;**36:**349–63.

23 Sahani D, Saini S, Pena C, Nichols S, Prasad SR, Hahn PF, *et al*. Using multidetector CT for preoperative vascular evaluation of liver neoplasms: Technique and results. *AJR Am J Roentgenol* 2002;**179:**53–9.

24 Freeny PC, Nghiem HV, Winter TC. Helical CT during arterial portography: Optimization of contrast enhancement and scanning parameters. *Radiology* 1995;**194:**83–90.