## 66. Malignant Hepatic Masses

The most common primary malignant hepatic neoplasm is hepatocellular carcinoma (HCC), which occurs in a solitary, multifocal, and less common diffuse pattern. It is usually associated with liver cirrhosis of different causes such as chronic viral hepatitis or alcoholism. However, HCC may also arise in non-cirrhotic livers, where exposure with aflatoxins, nitrosamines and chemical carcinogens are discussed as underlying causes.
In liver cirrhosis, the pathogenesis of HCC usually follows a multistep process where the formation of a benign regenerative nodule is the result of recurrent focal inflammatory processes and scarring. With progression of disease these nodules may transform to low-grade and high-grade dysplastic nodules with the end-stage being early HCC. Diagnosis of this transformation from benign regenerative nodules to intermediate dysplastic nodules or malignant HCC is probably the most challenging topic in the work-up of chronic liver cirrhosis.
On unenhanced MRI the appearance of HCC is inconclusive, as it consists in principle of malignant hepatocytes with atypical growth pattern failing to form typical hepatic acini. Thus, in particular well-differentiated HCC may show signal intensities comparable to normal liver tissue. However, deposition of fat, calcification, or hemorrhage results in signal alterations of unenhanced T1WI and T2WI contributing to lesion conspicuity. For further evaluation, analysis of the enhancement pattern on dynamic contrast enhanced MRI is crucial. Based on their predominantly arterial blood supply HCCs typically demonstrate a strong and homogeneous contrast enhancement on arterial phase T1WI followed by an early wash-out in the portal-venous and equilibrium phases. Detection of enhancing pseudocapsules surrounding the nodule in the portal-venous and equilibrium phases further contributes to the diagnosis of HCC. In addition, DWI sequences may help to detect malignant transformation of dysplastic nodules into HCC showing higher hyperintense signals with increasing b-values and low signal intensities on corresponding ADC maps. In addition, the degree of dysplasia and malignant transformation appear to correlate with the degree of washout notable during dynamic CE studies, where arterially hypervascularized nodules without notable washout on further sequences may be considered more benign. The same holds true for the application of hepatobiliary contrast agents. Low-grade dysplastic nodules may contain functioning hepatocytes and normal biliary architecture resulting in notable accumulation of hepatospecific Gd-based contrast agents. Thus, lesion signal may be less hypointense compared to normal liver tissue, when compared to HCC containing non-functioning malignant hepatocytes and no normal biliary architecture. Thus, HCC is usually hypointense on the hepatobiliary phase T1WI after application of either Gd-BOPTA

(MultiHance) or Gd-EOB-DTPA (Primovist/Eovist). However, in some cases central necrosis, hemorrhage, and subsequent fibrosis of HCC may also result in late-phase enhancement of central parts of the lesion, which complicates the diagnosis.
In a cirrhotic liver, the major differential for a lesion enhancing in the arterial phase is a regenerative nodule, although these typically remain similar to parenchymal SI on interstitial phase images, enhance overall more homogenously, and may demonstrate hemosiderin deposition resulting in a low SI on T2WI based on T2* effects .

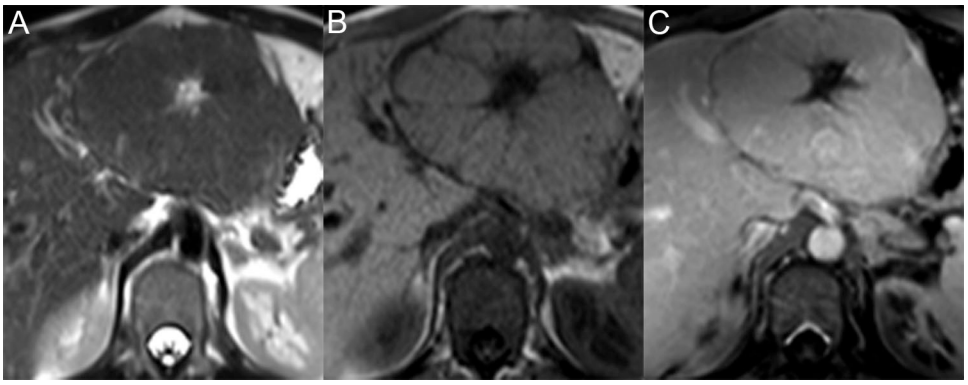

Fig. 66.1

Fibrolamellar carcinoma—a HCC variant found in young patients without cirrhosis—may be confused with focal nodular hyperplasia due to its central scar, which is hyper- and hypointense to hepatic parenchyma on the respective T2 and GRE T1WI of Fig. 66.1A,B. This lesion enhances on (C) arterial phase CE T1WI with sparing of the scar. With time, overall enhancement of this type of lesion fades, although progressive, if only partial, enhancement of the central scar occurs. The hypointense capsule surrounding this particular lesion is atypical for FNH, and subsequent biopsy revealed fibrolamellar carcinoma.
Cholangiocarcinoma (CCC) is an important differential consideration in the context of primary malignant hepatic tumors. In brief, CCC is usually hypovascularized, thus depicting hypointense on arterial and portal-venous phase T1WI. Desmoplastic reaction in the center of the nodules may result in some faint enhancement on later T1WI in the equilibrium phase. Larger centrally located masses with adjacent smaller satellite nodules and concomitant cholestasis are further typical signs. CCC is further discussed in Chapter 68.
Metastatic disease within the liver may demonstrate avid arterial phase contrast enhancement or more commonly enhance less than the surrounding parenchyma. Unlike HCC, however, such lesions do not possess functioning hepatocytes, and thus uniformly lack delayed enhancement with Gd-BOPTA (or Gd-EOB-DTPA) aiding lesion conspicuity on hepatobiliary phase T1WI.

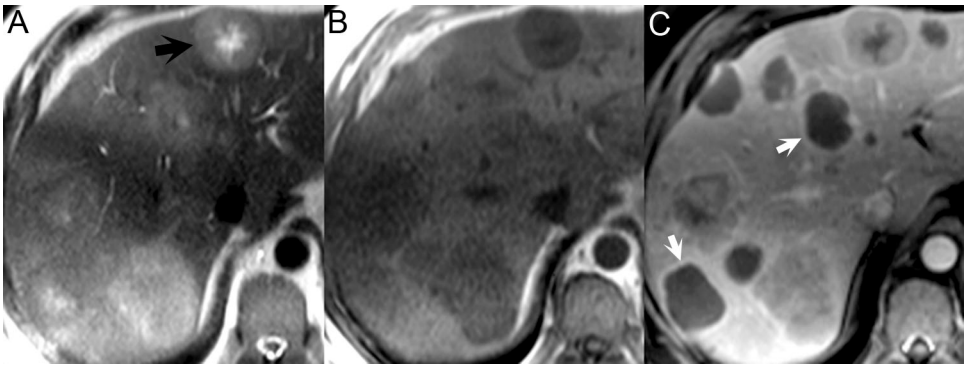


Fig. 66.2

Sources of metastatic disease may be distinguished, in part, by MR SI and enhancement characteristics. Neuroendocrine tumors appear as very bright lesions on T2WI and demonstrate avid arterial-phase enhancement, thus mimicking capillary hemangioma. Focal liver lesions of inhomogeneous high SI on T2WI which enhance avidly in the arterial phase include renal cell carcinoma, pheochromocytoma, breast cancer, and melanoma. The latter, along with ovarian cancer and hemorrhagic neoplasias, may have high SI on T1WI. A fatty liver is often present in metastatic disease, in some instances secondary to chemotherapy; thus lesions with high SI on T1WI may be better detected on out-of-phase imaging. Metastatic colon carcinoma is illustrated on the respective HASTE T2, pre-contrast T1, and early venous phase CE FS T1WI of Fig. 66.2A-C. Multiple, large lesions are evident, one of which demonstrates a characteristic donut sign (A, black arrow) on T2WI signifying central necrosis, a typical finding in adenocarcinoma. This necrotic area appears as central hyperintensity surrounded by moderate SI tumor. A halo sign may alternatively be present, referring to a high SI rim of viable cells surrounding rims of alternating decreased, correlating with mucin, necrosis, or fibrosis, and increased SI, representing liquefactive necrosis. The centrally necrotic area does not enhance on (C) FS post-contrast T1WI. Colorectal carcinoma, like most adenocarcinomas, is frequently hypovascular in the arterial phase, demonstrating only a rim of enhancement on arterial and (C, white arrows) early venous phase imaging with subsequent washout 5-15 minutes post-contrast. Cystic metastases of ovarian cancer may enhance similarly, but with internal septations and of course a cyst-like appearance on unenhanced images. Ill-defined rim enhancement persisting past the venous phase without central contrast uptake, may be seen with a pyogenic or amebic abscess, the latter preferentially involving the right hepatic lobe. Treated metastatic lesions may appear identical although a septate cystic appearance is rarely present, and clinical history should aid in distinction. Lymphoma is also hypovascular in the arterial and portal venous phases, but often iso- rather than hypointense to parenchyma in the interstitial phase. Metastases of leiomyosarcoma and gastrointestinal

stromal tumors may occasionally show delayed, persistent enhancement, as may hemangioendotheliomas or hemangiosarcomas. The latter can be distinguished from benign hemangiomas by its irregular borders and heterogeneous internal appearance secondary to hemorrhage.

Not yet mentioned in detail, but critical for clinical MR imaging of the liver, is scan time. Although there are many different ways to achieve shorter breath-hold scans, doing so will improve patient compliance and lead to markedly higher diagnostic quality in abdominal MR. Today imaging sites may use a scan time as long as 20 seconds to image the liver, during which the patient needs to hold their breath. For many patients this is much too long. Illustrated in Fig. 66.3 are high quality very short breath-hold scans, obtained with one of several different approaches available today. In this instance, an ultrashort breath-hold Dixon VIBE sequence has been used for improved arterial phase imaging, resulting as well in overall improved image quality due to better patient compliance. (A) Precontrast, (B) first, and (C) third arterial phase images, together with a (D) 20-minute delayed hepatocellular phase image, after injection of gadoxetic acid are presented in an elderly man with metastatic rectal adenocarcinoma. Acquisition time for each image series was 4.8 seconds, with scans acquired at 3 T using a 60-channel body coil. A new small metastasis along the dorsal margin of the liver (black arrow) demonstrates prominent early arterial enhancement, with some wash out by (C) the late arterial image, and no uptake in the delayed phase. Also visualized at this level are a hemangioma (white arrow) and a larger metastasis previously treated by microwave ablation (asterisk). Note the absence of respiratory artifacts due primarily to the short scan time, as compared with more conventional 15 to 20 seconds VIBE acquisitions often acquired.

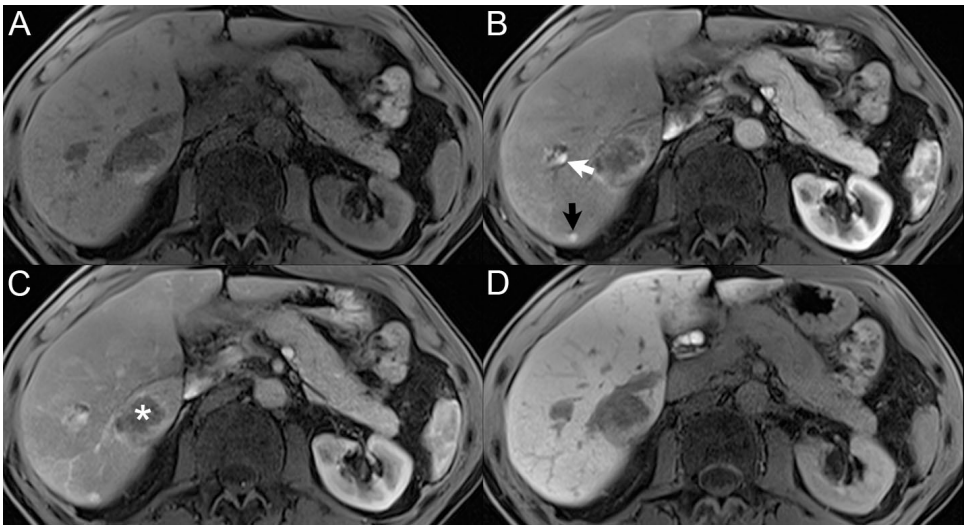


Fig. 66.3

(Adapted with permission from Invest Radiol 2017;52:1)