65. Benign Hepatic Masses

Pulse sequences acquired for abdominal MRI typically consist of fast acquisition schemes such as single-shot turbo spin echo (i.e. HASTE) and gradient echo schemes such as FLASH or VIBE with acquisition times equal to or below 20 sec. These fast imaging techniques typically permit acquisition of one image stack within a single breath hold, limiting any artifacts due to respiration or bowel motion. In particular, with the advent of more powerful gradients, multielement/multichannel coil technologies allowing for parallel imaging and view sharing acquisition schemes, MR sequences with high temporal and spatial resolution and acquisition times on the order of seconds have become feasible. In patients unable the follow breathing commands, alternative techniques such as radial k-space sampling (with compressed sensing), respiratory gating, or sequences with an increased number of acquisitions allow for diagnostic abdominal MRI with free respiration.
Unenhanced sequences acquired for abdominal MRI typically include T2WI with (i.e. STIR) or without fat-saturation (i.e. HASTE) and diffusion-weighted images (DWI). In particular the latter help to detect lesions/areas with increased fluid content based on edema (associated with inflammation or malignancy) or increased cell density (i.e. neoplasm). In addition, T1WI with and without fat-saturation and opposed-phase sequences contribute to further characterize focal abdominal lesions or diffuse organ pathologies. Here, in particular Dixon fat water separation techniques are of interest allowing for four different image contrasts within one breath hold acquisition.
Moreover, analyzing the vascular characteristics of abdominal lesions is indispensable for accurate diagnosis. For this purpose, dynamic acquisition of T1WI sequences covering the abdomen (or at least the organ or lesion of interest) are acquired after injection of Gd-based contrast agents. Here, typical enhancement patterns in arterial, portal-venous or equilibrium phase images markedly help to identify important lesion characteristics and classify pathologies. In addition, applying contrast agents with biliary excretion (such as Gd-BOPTA or Gd-EOB-DTPA) allows for hepatobiliary phase imaging contributing to differentiate hepatic lesions or to depict pathologies of the biliary tract. Finally, sequences performed 10 to 30 min after contrast injection display renal excretion of the gadolinium chelates, thus providing MR images of the urinary tract.
Hepatic cysts are the most common entity encountered in the liver and appear as sharply demarcated, non-septated, hyperintense lesions on T2WI (FS T2WI, Fig. 65.1A). Simple cysts are hypointense on T1WI and do not enhance on post-contrast T1WI, as seen in Fig. 65.1B—VIBE breath hold images wherein a solitary, hypointense hepatic cyst is present. Occasionally hemorrhage into a cyst increases its SI on T1WI and T1WI with fat

suppression, adding mucinous cysts of foregut origin—a lesion usually involving the superficial liver and expanding its margin—to the differential. Peribiliary cysts are usually considered as acquired hepatic cysts and are typically located adjacent to the main intra- and extrahepatic bile ducts. Underlying causes of peribiliary cysts include chronic liver disease affecting the biliary system as well as obstructive jaundice and recurrent ascending cholangitis.

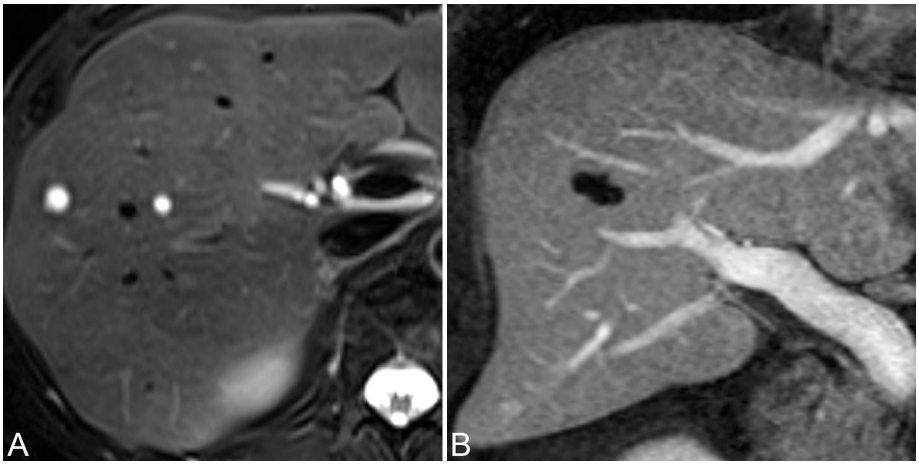

Fig. 65.1

In addition, some infectious or neoplastic diseases may manifest with cystic lesions within the liver and need to be taken into consideration. For example, hepatic metastases from mucinous bowel cancer or mucinous pancreatic cystadenocarcinoma may be cystic with increased SI of the content on T1WI.

Echinococcal cysts (E. granulosis) are characterized by a hypointense fibrous capsule, which may enhance. Multiple daughter cysts are classically contained within the capsule, with extrinsic cysts termed satellite lesions. Hepatic alveolar echinococcosis (E. multilocularis) is more aggressive, demonstrating involvement (and thus enhancement) of the heart, lungs, and their serosa. Further cystic lesions may arise from the biliary system and include peribiliary cysts (as discussed above), biliary hamartoma or biliary cystadenoma. For further information please see chapter 68.

Hemangiomas are the most common neoplastic hepatic lesion. A lobulated giant hemangioma demonstrating typical hyperintensity on HASTE T2WI (which is less than for a water containing cyst) is illustrated in Fig. 65.2A. Hemangiomas are less lobulated when they are smaller in size, with all varieties generally exhibiting homogenously low SI on T1WI. Several enhancement patterns have been described. Intense and uniform enhancement in the arterial phase imaging with diminishing enhancement on subsequent images is characteristic for capillary hemangiomas. In distinction, peripheral, nodular, and

discontinuous arterial-phase enhancement, typical for a cavernous hemangioma, is demonstrated in Fig. 65.2B—a breath hold VIBE T1WI obtained one minute following contrast injection. Enhancement then progresses centripetally and is homogeneous throughout the lesion on equilibrium phase images.

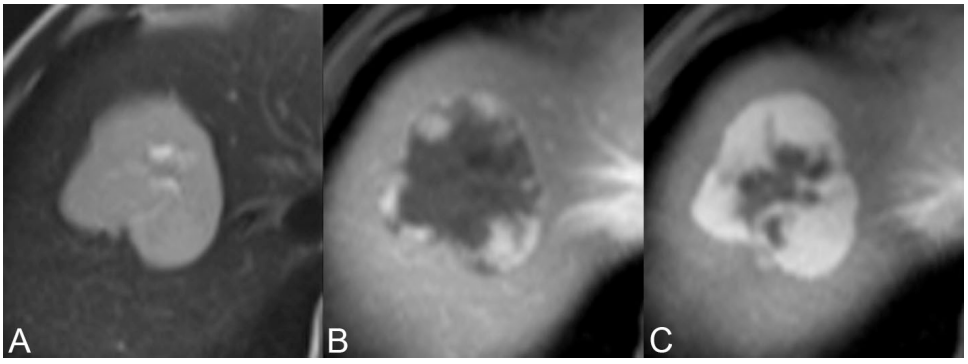

Fig. 65.2

The T1WI (10 minutes post-contrast) in Fig. 65.2C demonstrates an area of central scar with spared enhancement, a pattern frequently found in giant hemangiomas. In general, biliary phase enhancement (not illustrated) is absent in hemangiomas, thus showing a hypointense signal compared to surrounding normal liver parenchyma on T1WI. Important differential diagnostic considerations regarding hepatic hemangiomas include hemangioendothelioma and angiosarcoma. However, these entities are, in general, more ill-defined with concomitant signal alteration and contrast enhancement of adjacent liver tissue, and expedited contrast kinetics in the lesion including pathologic washout, revealing their malignant character.

Focal nodular hyperplasia (FNH), nodular regenerative hyperplasia (NRH) and hepatic adenoma are probably the most frequent and important benign focal liver lesions to be addressed besides the aforementioned hemangioma. As these lesions consist of benign hepatic cells (yet atypically arranged) lesion conspicuity is less apparent on precontrast sequences as compared to the aforementioned cystic or angiomatous lesions. But still there are typical findings on precontrast and contrast-enhanced imaging clearly characterizing these entities.

After hemangioma FNH is considered the second most common benign tumor of the liver. Development of FNH is most likely based on a preexisting vascular malformation in the liver subsequently triggering a localized hyperplastic growth of hepatocytes. As a consequence, FNH is frequently found in association with hepatic hemangioma.

A FNH is illustrated in Figure 65.3A, demonstrating immediate, avid arterial phase (30 seconds post-contrast) enhancement with sparing of its central scar. Enhancement aids in

detection of these lesions as they may be essentially isointense to the liver on unenhanced MR sequences (including DWI). On portal-venous and equilibrium phase images lesion contrast consecutively gets less apparent as enhancement of liver parenchyma increases while FNH demonstrates a slow but continuous washout of contrast material (Fig. 65.3B, 1 minute post-contrast). On delayed hepatobiliary phase images (C) 3 hour after administration of Gd-BOPTA, FNH are typically iso- to hyperintense compared to adjacent normal liver tissue, demonstrating biliary retention of contrast—attributable to the malformed biliary ducts within FNH. Typical FNHs are also associated with a central stellate scar correlating with fibrotic tissue. This scar does not enhance (A) on early arterial phase images but does so later on portal-venous (B) – at about 1 minute post-contrast injection – and equilibrium phase images. If the central scar is not enhancing and concomitant retraction of the liver capsule is present fibrolamellar carcinoma (FLC) is an important differential diagnosis, which needs to be ruled out (FLC will not be iso- or hyperintense on hepatobiliary phase images after application of liver specific contrast agents). If a central stellate scar is not present but all other image characteristics are typical, the lesion may be referred to as atypical FNH.

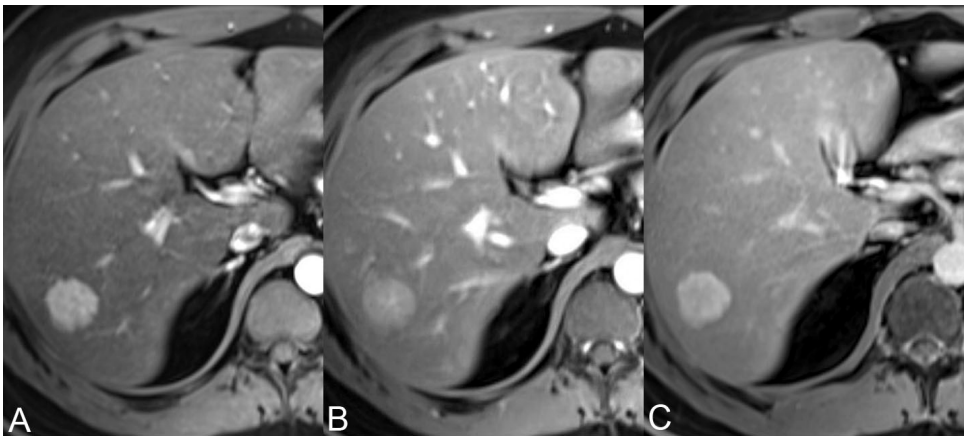

Fig. 65.3

Hepatic adenomas may appear similar to FNH on unenhanced MR images with the exception of substantial fat content which leads to characteristic signal dropout on out-of-phase GRE T1WI compared to in-phase images. On dynamic contrast enhanced T1WI hepatic adenomas typically demonstrate a strong and homogenous uptake of contrast material in the arterial phase followed by a slow washout, all of which comparable to the enhancement characteristics of FNH. However, as biliary ducts are absent within hepatic adenomas, enhancement with hepatospecific agents such as Gd-BOPTA is absent during hepatobiliary phase imaging, thus here adenomas typically are hypointense compared to

normal adjacent liver tissue. While FNH usually do not require any follow-up examinations or treatment, symptomatic or hepatic adenomas exceeding 5 cm in diameter usually are referred to surgical resection due to the risk of rupture and hemorrhage, in particular for lesions that are subcapsular in location.

Development of NRH is usually based on alteration of hepatic circulation, for instance disturbance of small portal veins or hepatic arteries in association with inflammation such as primary biliary cirrhosis or fatty liver disease. Here, the size and distribution of the vascular territory affected implies the size and distribution of the induced NRH. Associated disorders potentially triggering NRH include myelo- or lymphoproliferative syndromes, vasculitis, rheumatologic disease, as well as different hepatotoxic medications including chemotherapy and steroids. On unenhanced MRI NRH usually are iso- to hyperintense on T1WI and iso- to hypointense on T2WI. On dynamic CE T1WI NRH show a homogeneous but faint enhancement in the arterial phase and become more isointense in portal-venous or equilibrium phase as enhancement of the surrounding liver parenchyma increases. Some larger NRH may demonstrate a small hypointense rim probably consistent with an area of decreased blood supply. As NRH consist of functioning hepatocytes with abnormal biliary drainage, the SI on T1WI in the hepatobiliary phase after application of hepatospecific contrast agents is usually iso- to hyperintense.