The use of magnetic resonance imaging (MRI) in the diagnosis of intra-abdominal diseases in the past has been limited because artifacts caused by respiratory, cardiac, and peristaltic motion significantly degraded the quality of images. As MRI technology has advanced, these limitations have been overcome.

Motion artifacts in $T_1$-weighted images can be controlled for spin echo sequences using a combination of respiratory gating, averaging, and presaturation pulses. However, with the introduction of breath-hold sequences, spin echo sequences in imaging of the abdomen are being replaced on up-to-date equipment. Multisec-

tion spoiled gradient echo sequences are primarily used for this purpose (e.g., fast low-angle shot [FLASH]), which allows $T_1$-weighted imaging of the entire upper abdomen in one single breath-hold of less than 20 seconds. Thus, dynamic imaging of the upper abdomen during contrast medium injection in the arterial and portal venous phase of liver perfusion is possible.

For $T_2$-weighted imaging, multishot fast spin echo (FSE) or so-called turbo spin echo (TSE) sequences are primarily used, which allow respiratory gated imaging of, for example, the liver in only a few minutes. Although, as reported in the literature, these sequences may show a slight loss of contrast between liver lesions and surrounding liver parenchyma, the higher image quality regarding motion artifacts more than compensates for this. Newer sequences allow $T_2$-weighted imaging of the upper abdomen in suspended respiration. Even non-breath-hold $T_2$-weighted studies using ultra-
fast single-shot sequences can be performed in patients who cannot follow breathing commands adequately.

For imaging of the bile ducts and the pancreatic duct in magnetic resonance cholangiopancreatography (MRCP), heavily $T_2$-weighted sequences should be applied because the fluid in the bile ducts and pancreatic duct is static and has a long $T_2$ time. Using this tech-
nique either in suspended respiration or with respiratory gating, two- and three-dimensional reconstructions of the biliary system can be acquired.

Chemical shift imaging of the abdomen is another technique that is of great interest. It allows detection of focal fatty infiltration in the liver (Fig. 11-1) as well as identification of benign adenocortical masses in imaging of the adrenal glands. Currently used chemical shift techniques include opposed-phase imaging featuring signal cancellation in voxels that contain both tri-
glyceride and water as well as frequency selective fat suppression techniques in which a narrow band excitation pulse is used to selectively saturate triglyceride protons without affecting water protons. Opposed-phase images are more sensitive in detecting small quantities of lipid (e.g., in focal fatty infiltration of the liver), whereas fat-suppressed images are better in identifying large quantities of lipid in a tissue (e.g., a lipoma).

The detection and differential diagnosis of focal liver lesions in patients with suspected primary malignant liver tumors or metastases are important challenges that have major clinical consequences. Although the accurate differentiation between benign lesions such as liver hem-
angioma or focal nodular hyperplasia (FNH) and malignant liver lesions is of great importance, the ability to localize lesions precisely, to determine the exact number of lesions present, and to ascertain the involvement, if any, of vascular or biliary structures may have a major impact on therapeutic management. MRI can be regarded as the most accurate method for the detection, differential diagnosis, and staging of benign and malignant liver tumors.

Although unenhanced MRI, using morphologic characteristics on $T_1$- and $T_2$-weighted images to differentiate between benign and malignant liver lesions, has high accuracy rates, it is widely accepted that the use of contrast can improve the detection as well as the differential diagnosis of focal liver lesions.

Contrast for MRI of the liver has generally been classified in one of two ways: as a nonspecific agent that distributes exclusively to the extracellular fluid space and that is effective for dynamic phase imaging of the liver for lesion detection and characterization (e.g., gadolinium [Gd]-DTPA, Gd-DTPA-BMA or Gd-HP-DO3A); or as a liver-specific agent that is targeted specifically to liver cells (i.e., hepatocytes, e.g., Gd-BOPTA, Gd-EOB-DTPA, or mangafodipir trisodium [Mn-DPDP]) or Kupffer cells (e.g., superparamagnetic iron oxide (AMI-25), magnetite) and that is effective in a more delayed phase for liver lesion detection.

Although liver-specific agents like Mn-DPDP and ferumoxides (AMI-25) are approved for clinical use in the United States, their actual utilization is very low.
Thus, the use of extracellular space agents for imaging of the liver is emphasized in this chapter.

The vascular anatomy of the liver is essential for the differential diagnosis of focal liver lesions and for liver surgery. Surgical anatomy divides the liver into nine segments, any of which can be resected individually or in combination with neighboring segments. If there is enough liver parenchyma left, no diminution of liver function should result. The falciform ligament divides the liver into the left and right lobes. The liver segments are differentiated by their vascular supply with the branches of the portal vein showing intrasegmental localization and the hepatic veins located intersegmentally.

Knowledge of the different perfusion phases of liver parenchyma and focal liver lesions is essential to understand the diagnosis and therapy of focal liver lesions. The liver parenchyma receives up to 80% of its blood supply from the portal vein; the remaining 20% is supplied by the common hepatic artery via the celiac trunk. In contrast to normal liver parenchyma, focal liver lesions are mainly fed by branches of the hepatic arteries.

Extracellular contrast in MRI is given intravenously and distributed initially within the intravascular compartment. In this use, application of the contrast by means of a power injector with a flow rate of at least 1 mL/sec is superior to a continuous infusion if dynamic imaging is to be performed.

The normal blood supply of the liver, characterized by perfusion via the hepatic artery and portal vein leads to a typical enhancement pattern after bolus contrast administration. With a delay of 25 to 30 seconds after starting contrast injection, hepatic arteries are contrasted, whereas venous and portal venous structures are still unenhanced and remain hypointense to surrounding liver tissue. The hypointense appearance of venous structures in the arterial phase scan should not be mistaken for focal liver lesions. In the arterial phase of liver perfusion, arterial hypervascularized liver lesions can easily be differentiated from hypointense, only slightly contrasted normal liver tissue. Hepatic areas with incidental regional hypervascularization, already known from computed tomography (CT) examination as areas of focal attenuation difference, must not be misinterpreted as hypervascularized focal lesions. Thus, a comparison with unenhanced and portal venous scans is always necessary. After initial arterial enhancement, a
homogeneous filling of venous and portal venous structures can be observed in the portal venous phase, 50 to 55 seconds after the beginning of contrast injection, also accompanied by increasing contrast enhancement of surrounding normal liver tissue. This phase of liver perfusion enables good differentiation of liver segments based on the identification of left, middle, and right liver vein. Furthermore, hypovascularized focal lesions can be clearly depicted because the entire liver now shows homogeneous contrast enhancement. Delayed-phase images 5 to 15 minutes after contrast administration show an enhancement of liver tissue in comparison with unenhanced images. At this time, the vessels may again appear hypointense or isointense in comparison with surrounding liver parenchyma. Liver lesions with a delayed, persistent enhancement can be detected and classified.

Classification of Focal Liver Lesions in MRI Based on Vascularization Patterns

Dynamic MRI examinations refer to a classification that describes perfusion patterns, and specifically the degree of tumor vascularization, without primary consideration of the origin of the tumor or tumor-like lesion.

Regarding perfusion patterns of focal liver lesions, three groups can be distinguished:

1. Hypervascular liver lesions
2. Hypovascular liver lesions
3. Lesions presenting delayed persistent enhancement

Figure 11-2 demonstrates the three different perfusion characteristics to which most focal liver lesions can be assigned. Figures 11-3 to 11-5 briefly summarize the lesion types that may be found in each of the three groups and give additional information for subdivisions.

All three groups in this classification include benign and malignant liver tumors as well as primary and secondary liver lesions. Additional differentiation is possible by comparing dynamic images with unenhanced T1- and T2-weighted scans and by analyzing internal lesion morphology based on contrast-enhanced images.

This radiologic classification does not consider the histopathologic differentiation of liver tumors because the degree of vascularization and not the histologic appearance is the basis for differentiation of focal liver lesions in dynamic MRI. Applying histopathologic criteria, focal liver lesions can be divided in different groups with regard to their differentiation and type (Fig. 11-6). Primary lesions can be subgrouped as mesenchymal, epithelial, or mixed as well as tumor-like lesions; secondary lesions mainly comprise metastatic and parasitic disease.

Some of the morphologic characteristics described in pathologic classifications may also be recognized in MRI of the liver (e.g., the central scar in focal nodular hyperplasia, regressive changes in hepatocellular carcinoma, and myxoid degeneration in a giant hemangioma). Nevertheless, the pathologic description in most cases does not reflect vascularization patterns that may be used for differential diagnosis in contrast-enhanced cross-sectional imaging (e.g., the centripetal filling of hemangioma or the arterial hypervascular rim in liver metastases of colorectal adenocarcinoma). Thus, classification of liver lesions in dynamic contrast-enhanced MRI uses morphologic criteria described for pathologic classifications as well as contrast kinetics, leading to the differentiation between arterial hypervascular and hypovascular lesions and lesions demonstrating delayed persistent enhancement without primary regard to origin or type of liver lesions. Figure 11-6 shows the clinically most

**FIGURE 11–2.** Classification of focal liver lesions in T1-weighted dynamic imaging.
FIGURE 11–3. Liver lesions that are hypervascular in the arterial phase. (SI, signal intensity; CMA, contrast medium administration.)

Cystic appearance
Hypervascular rim in arterial phase
Hypovascular in arterial and portalvenous phase, isointense in images 5 min post CM injection
Hypervascular rim with persistent enhancement, no central CM uptake
History of abdominal trauma or surgery

Sharply demarcated, no irregular regional vascularization
Cyst solitary/multiple
Caroli’s disease
Polycystic kidney disease
Washout sign 5–15 min after CM injection, T2w: Doughnut sign/ halo sign
Irregular areas of low SI in T1w images, increased SI or isointense in T2w images
High SI in T2w images (almost cystic appearance), occasionally gas formation
Liver abscess
Cystic appearance, tends to increase in size, dislocation of vessels, high SI in T2w images
Bilioma

Low SI rim, internal septations, daughter cysts
Echinococcal disease
Cystic appearance, irregular cyst wall with focal solid areas, internal septations
Cystic metastases (e.g., ovarian cancer or after chemotherapy)
Irregular borders, inhomogeneous SI in T1w and T2w images, hyperintense rim due to extracellular methemoglobin, with time increase of central SI
Liver hematoma/ rupture

FIGURE 11–4. Liver lesions that are hypovascular in the arterial phase. (CM, contrast medium; SI, signal intensity.)
### Infiltration along portal tracts, segmental biliary obstruction

± Known primary neoplasm

Nodular peripheral enhancement in arterial phase, centripetal filling

Irregular, partially nodular enhancement in arterial phase, irregular filling

Homogeneous, sometimes early enhancement, irregular shape

<table>
<thead>
<tr>
<th>Hypovascular with hypervascular peripheral enhancement in arterial phase</th>
<th>Hypo- or slightly hypervascular in arterial phase, low SI in T1w and high SI in T2w images, sometimes peripheral washout</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intrahepatic cholangiocellular carcinoma (CCC)</strong></td>
<td><strong>Metastases of leiomyosarcoma and gastrointestinal stromal tumors (GIST)</strong></td>
</tr>
</tbody>
</table>

| High SI in T2w images (light bulb appearance), in large lesions central areas may remain unenhanced after CM injection due to thrombosis/fibrosis, sharply demarcated |
|---|---|
| Hemangioma |

<table>
<thead>
<tr>
<th>Centrifugal or irregular filling, irregular borders</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hemangiosarcoma, hemangiopericytoma</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>High SI in T2w images, iso- or hyperintense in delayed-phase images</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peliosis hepatis</td>
</tr>
</tbody>
</table>

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**FIGURE 11–5.** Liver lesions that show a delayed persistent enhancement. (SI, signal intensity; CM, contrast medium.)

---

**TABLE 11–4.** Classification of focal liver lesions by histopathology.

<table>
<thead>
<tr>
<th>PRIMARY TUMORS</th>
<th>BENIGN LIVER TUMORS</th>
<th>MALIGNANT LIVER TUMORS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epithelial tumors</td>
<td>Hepatocellular adenoma</td>
<td>Hepatocellular carcinoma</td>
</tr>
<tr>
<td></td>
<td>Bile duct adenoma</td>
<td>Fibrolamellar carcinoma</td>
</tr>
<tr>
<td>Cholangiocellular</td>
<td>Biliary papillomatosis</td>
<td>Cholangiocellular carcinoma</td>
</tr>
<tr>
<td>Mesenchymal tumors</td>
<td>Hemangioma</td>
<td>Bile duct cystadenocarcinoma</td>
</tr>
<tr>
<td></td>
<td>Hemangioendothelioma (children)</td>
<td>Angiosarcoma</td>
</tr>
<tr>
<td></td>
<td>Angiomyolipoma</td>
<td>Hemangiosarcoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Malignant epithelioid hemangioendothelioma</td>
</tr>
<tr>
<td>Vascular tumors</td>
<td></td>
<td>Embryonal sarcoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rhadomyosarcoma</td>
</tr>
<tr>
<td>Mixed type</td>
<td></td>
<td>Hepatoblastoma</td>
</tr>
</tbody>
</table>

| SECONDARY TUMORS | Parasilic infections | Metastases |

| TUMOR-LIKE LESIONS | Cysts | |
|---|---|
| Focal nodular hyperplasia | |
| Regenerative nodules | |
| Mesenchymal hamartoma | |
| Peliosis hepatis | |
| Inflammatory pseudotumors | |

|---|---|

---

**FIGURE 11–6.** Classification of focal liver lesions by histopathology.
relevant primary and secondary hepatic lesions as well as tumor-like lesions, which must be distinguished from true neoplasms.

In practice, the liver should be scanned before administration of an extracellular agent (unenhanced imaging) using both T1- and T2-weighted sequences and in the immediate postcontrast arterial (approximately 18–25 seconds after the start of contrast administration) and portalvenous phases (approximately 50–55 seconds after contrast administration) after bolus injection of the contrast using T1-weighted gradient echo breath-hold sequences covering the entire liver in a single breath-hold. Additional T1-weighted images should be acquired approximately 5 minutes after contrast injection.

Hypervascularized Liver Lesions

Hypervascularized liver lesions are characterized by strong contrast enhancement in the arterial phase scan (25–30 seconds after the start of contrast application) as a result of the blood supply being mainly from the hepatic arteries. Hypervascularized liver lesions may be sharply demarcated or may show a more diffuse appearance. Often lesions presenting with early arterial enhancement are isointense and barely detectable in the portalvenous and delayed-phase scans. Arterial hypervascularization is rarely seen as a result of arteriovenous shunting, such as in high-flow arteriovenous malformations (AVMs). However, in general, benign or malignant neoplasms are the main cause of arterial blood supply of focal liver lesions with the exception of inflammatory changes.

The most common primary malignant tumor of the liver presenting with arterial hypervascularization is hepatocellular carcinoma (HCC). This malignant neoplasm is mostly associated with alcoholic cirrhosis, long-standing chronic hepatitis B or C, and other forms of chronic parenchymal damage (e.g., hemochromatosis). Three different types of HCC can be distinguished by means of morphologic appearance in macroscopic pathology or histology as well as by imaging studies: the expansile (encapsulated) form, a more infiltrative variant, and the multifocal or diffuse type. All types of HCC typically show hypervascularization in the arterial phase scan. Depending on necrosis or hemorrhage in HCC, hypervascularization may range from homogeneous to inhomogeneous. In expansile or encapsulated HCC (Fig. 11-7), a hypointense pseudocapsule may be seen in the portalvenous phase scan consisting of compressed liver tissue, and lesions may appear single or multinodular. The infiltrative type (Fig. 11-8) shows irregular margins with partially hypervascular and hypovascular areas occasionally replacing most or all of one liver lobe. The diffuse type presents as several small tumors (<1 cm) at multiple sites in the liver, resulting in a more diffusely appearing hypervascularization in the arterial phase scan, isointense or slightly hypointense in portalvenous phase, and almost always associated with advanced signs of cirrhosis. On unenhanced images, depending on underlying parenchymal changes, HCC in patients with underlying cirrhosis typically appears hypointense on T1-weighted images and hyperintense on T2-weighted images. In patients with hemochromatosis, HCC appears hyperintense on T1- and T2-weighted images compared with the surrounding low-signal-intensity-liver tissue (resulting from excessive iron storage within the liver).

Fibrolamellar carcinoma (FLC) is a rare type of hepatocellular carcinoma and is found mainly in young patients without predisposing chronic liver disease. Early resection may result in long-term survival. FLC can resemble focal nodular hyperplasia (FNH). In the arterial phase, FLC shows marked enhancement with a central hypointense scar similar to the findings in FNH. In contrast to FNH, the central scar in FLC typically appears hypointense in T2-weighted imaging, and central calcification may be noted on CT in 30% of the patients. The central scar in FLC represents lamellar fibrosis ("true scar"); thus, in contrast to FNH, no delayed enhancement of the central scar is observed on delayed-phase imaging.

Liver cell (hepatocellular) adenoma (HCA) is the most important benign primary liver tumor predominantly arising in young female patients (15–45 years). The association with oral contraceptive steroid usage is well established. Even so, HCA may occur, albeit rarely, spontaneously. Incidence and complication rates seem to correlate with duration and dosage of oral contraceptives. Rare cases of malignant transformation are described. Histologically, HCA consists of benign hepatocytes arranged in sheets without acinar architecture, portal tracts, or bile ducts. Large adenomas may cause abdominal discomfort; they often appear with areas of necrosis, hemorrhage, or fatty degeneration. Thus, inhomogeneous signal intensity may be noted in unenhanced T1- and T2-weighted and contrast-enhanced MRI. The risk of rupture into the peritoneal cavity is high in liver cell adenomas larger than 10 cm, and resection should be seriously considered for this reason alone. Typically, these lesions are hypervascular on the arterial phase scan. Depending on the size of the lesion internal, hypovascular areas resulting from hemorrhage or necrosis may be present. On unenhanced T1- or T2-weighted images, small HCAs without necrotic areas or hemorrhage may appear isointense to surrounding liver tissue (Fig. 11-9). Larger HCAs with hemorrhage or necrosis may appear hyper- or hypointense (Fig. 11-10).

Regarding pathologic classification, focal nodular hyperplasia (FNH) represents a tumor-like benign liver lesion presenting with arterial hypervascularization. It is most often incidentally found in young, otherwise healthy individuals. Relation to oral contraceptives is less evident than for HCA, but oral contraceptive steroid usage may promote growth. On macroscopic tissue pathology examination, FNH shows fibrous septa and a central stellate scar. On histologic exam, abnormally arranged normal hepatocytes with Kupffer cells and primitive bile ductules without communication with ducts are present. No portal tracts or central veins can be found. Typically, the central scar of the lesion is hyperintense on T2-weighted scans and shows enhancement on delayed-phase images (Fig. 11-11), a relatively reliable sign for FNH in differential diagnosis from fibrolamellar carcinoma. In contrast to FLC, the "pseudoscar” in FNH consists of vessels, bile ducts, and Text continued on page 300
Figure 11–7. Encapsulated hepatocellular carcinoma (HCC) in a 34-year-old man with chronic hepatitis C and recently elevated alpha fetoprotein. A, On T2-weighted imaging the liver surface is bulged by an inhomogeneous, mostly isointense lesion in the left liver lobe. Whereas most of the lesion is hypointense on the T1-weighted unenhanced image, the center of the lesion has high signal intensity indicating internal hemorrhage (B). C, In arterial phase T1-weighted dynamic imaging, the lesion is markedly hypervascular with an early washout of contrast medium in the more central areas in the portalvenous phase (D). There is a persistent enhancing pseudocapsule (arrow) surrounding the lesion in the equilibrium phase (E), which is typically found in encapsulated HCC caused by compressed surrounding liver parenchyma.
FIGURE 11–8. Liver cirrhosis and hepatocellular carcinoma (HCC) in a 63-year-old man with long-standing chronic hepatitis B and C. A and B, Unenhanced T₂- and T₁-weighted images show liver cirrhosis with a humpy liver surface and an irregular nodular appearance of the liver parenchyma as a result of multiple macoregenerative nodules. There is a hyperintense lesion in the left liver lobe on T₂-weighted imaging (A), which shows arterial enhancement in the arterial phase (C, arrow) and early contrast washout in the portalvenous phase (D), consistent with HCC.
FIGURE 11–9. Small hepatocellular adenoma without degeneration in a 16-year-old boy with a history of Hodgkin’s lymphoma in complete remission, recurrent right upper quadrant pain, and a focal liver lesion on sonography. T₂- (A) and T₁-weighted (B) images hardly detect the round focal lesion in segment 8. It is almost isointense to surrounding parenchyma on T₂-weighted imaging and slightly hypointense on T₁-weighted imaging. There are no signs of hemorrhage or regressive changes. C, In the arterial phase, there is strong hypervascularization, and the lesion is isointense in the portalvenous phase (D).
Figure 11–10. Hepatocellular adenoma with large internal hemorrhage in a 34-year-old woman with right upper quadrant pain. A, In T₂-weighted imaging an inhomogeneous signal intensity with areas of high signal, visible also in the T₁-weighted image (B), is demonstrated. C, The fat-suppressed image again demonstrates high-signal-intensity areas, indicating hemorrhage. The lesion shows inhomogeneous strong arterial enhancement (D) with a slightly persistent elevated signal in the portalvenous phase (E).
FIGURE 11–11. Typical focal nodular hyperplasia (FNH) in a 27-year-old woman who underwent routine sonography, which detected a large focal lesion. A, On T₂-weighted imaging the lesion is slightly hyperintense to surrounding parenchyma, whereas on the T₁-weighted image (B) the lesion appears slightly hypointense. C, The typical arterial hypervascularization with a hypointense central scar is clearly visible in the arterial phase T₁-weighted dynamic image. D, In the portalvenous phase the central scar is still clearly visible. However, the central stellate scar displays enhancement in the equilibrium phase (E) typical of FNH.
edematous liver tissue. On T₁-weighted arterial phase imaging, FNH shows strong enhancement and the central scar appears hypointense. In the portalvenous phase, FNH is almost isointense to surrounding liver tissue (Fig. 11-12). The central scar in most cases still appears hypointense but demonstrates contrast uptake on delayed-phase images. On unenhanced T₁-weighted images, the lesion is typically isointense or slightly hypointense. T₂-weighted images depict FNH as isointense or slightly hyperintense lesions. However, approximately 30% of FNHs are atypical (Fig. 11-13) and do not show a central scar.

Hypervascular liver metastases represent secondary malignant liver lesions. The following primary tumors typically show arterial hypervascular liver metastases: functional islet cell tumors such as gastrinoma and insulinoma, nonhyperfunctioning islet cell tumors, hypernephroma, neuroendocrine pancreatic tumor, carcinoid (Fig. 11-14), malignant pheochromocytoma, gastrointestinal stroma tumor (GIST), uveal melanoma, pancreaticoblastoma, breast cancer, and other rare primary neoplasms. Depending on the primary tumor, hypervascular liver metastases may show extremely high signal intensity in T₂-weighted images (e.g., insulinoma, gastrinoma) comparable to the signal intensity found in a hemangioma. Especially with small metastases, differen-
FIGURE 11–13. Atypical focal nodular hyperplasia (FNH) and liver hemangioma in a 52-year-old woman with newly diagnosed breast cancer. Magnetic resonance imaging of the liver was performed for staging. On T₂-weighted imaging (A), only the high-signal-intensity lesion of the left liver lobe is visible, which is hypointense on unenhanced T₁ (B) and shows peripheral nodular enhancement (arrow) in dynamic imaging (C and D). There is a clump-like fill-in of the lesion 5 minutes after contrast administration (E), consistent with a hemangioma. The lesion of the right liver lobe can only be visualized clearly on T₁-weighted contrast-enhanced images (C and D). There is a strong enhancement in the arterial phase with a slight pooling of contrast in the portalvenous phase. In the equilibrium phase, the lesion is again isointense to surrounding liver tissue. F, In images acquired 1 hour after injection of a hepatocyte-directed contrast (0.05 mmol/kg Gd-BOPTA), the lesion is hyperintense compared with surrounding liver tissue, indicating a liver lesion that contains functioning hepatocytes. There is no otherwise typical central stellate scar. Despite its atypical appearance, the diagnosis of an FNH can be made because of the contrast media uptake of the liver cell-specific contrast medium and the homogeneous hypervascularization of the lesion in a patient without underlying liver cirrhosis. An adenoma of this size would show regressive changes. Metastases would show a more inhomogeneous, increased signal intensity in unenhanced T₂-weighted images.
Figure 11–14. Hypervascular liver metastasis from carcinoid tumor found during a follow-up study in a 72-year-old man with previous resection of a small bowel carcinoid. A. On the T$_2$-weighted image, a hyperintense liver lesion (arrow) with homogeneous signal intensity is visible. The lesion is hypointense on T$_1$ and shows a strong homogeneous hypervascularization in arterial phase dynamic imaging (C). There is an early washout of contrast in the central area, indicating arteriovenous shunting, which is often present in malignant liver lesions (D, portalvenous phase image).

Hypovascular Liver Lesions
Hypovascular liver lesions are characterized by reduced or missing perfusion of the lesion, resulting in a hypointense appearance of the lesion compared with surrounding liver tissue in all phases of the dynamic T$_1$-weighted liver examination. Lesions may show signs of peripheral hypervascularization or irregular segmental perfusion in the arterial phase as an indicator of malignant liver lesions (e.g., with metastases from colorectal carcinoma or cystic metastasis from ovarian cancer). Alternatively, no signs of irregular perfusion indicate benign liver tumors, as in simple hepatic cysts (Figs. 11-15 to 11-17) or post-traumatic lesions such as biloma. Benign primary, traumatic, parasitic, or inflammatory...
Figure 11–15. Simple liver cyst in a 34-year-old woman with incidental finding of a focal liver lesion on routine sonography. A, On T2-weighted imaging, a sharply demarcated lesion with high signal intensity can be noted in the left liver lobe. On T1-weighted unenhanced (B), dynamic (C and D) and delayed phase (E) images, the lesion appears hypointense without signs of perilesional enhancement or contrast media uptake of the lesion itself.
Liver hemangioma and simple liver cyst in a 32-year old man with chronic pancreatitis. A. Both the hemangioma and the cyst display high signal intensity, the so-called light bulb sign, on T2-weighted magnetic resonance imaging and are clearly demarcated and hypointense on the unenhanced T1-weighted image (B). As the main distinctive feature, the cyst remains hypointense throughout dynamic T1-weighted imaging, whereas the hemangioma displays typical peripheral nodular enhancement. In the arterial phase (C), there is no enhancement. Nodular enhancement is initially noted in the hemangioma in the portalvenous phase (D), with ongoing uptake of contrast media (arrow) in the equilibrium phase (E). Enhancement of the lesion was more characteristic at a slightly lower slice position (not shown).
hypovascular liver lesions include simple hepatic cysts, lipoma, biloma, liver abscess, echinococcal (Fig. 11-18) and amebic liver abscess, and others. Malignant lesions include primary liver tumors (e.g., juvenile hepatocellular carcinoma or bile duct cystadenocarcinoma) and metastases from most primary sites (e.g., colon, rectum, pancreas, lung), adenocarcinomas, small cell lung cancer, pharyngeal carcinoma, melanoma, cystadenocarcinoma of the pancreas and ovaries, liposarcoma, teratoma, breast cancer, cervical carcinoma, and secondary involvement of the liver in Hodgkin's lymphoma and NHL and others.

Important clues for distinguishing between simple hepatic cysts and cystic or necrotic metastasis include a hypervascularized rim, hypervascularized areas in the arterial phase together with irregular internal lesion morphology, enhancing mural nodules or septations, and a solid tissue rim surrounding central liquefaction necrosis (Figs. 11-19 and 11-20). Biphasic contrast-enhanced MRI may very sensitively show these areas of irregular vascularization as a result of tumor infiltration in arterial phase images, which is occasionally the only sign of malignant liver tumor in small lesions. Nevertheless, additional delayed-phase images should be performed to demonstrate peripheral washout as a very specific sign of malignancy in focal liver lesions, especially in metastases of adenocarcinoma. This sign may be explained by the difference in vascularity between the periphery and the center of the lesion, which becomes rather ischemic or even degenerative and necrotic as the tumor outgrows its own blood supply. Histologic examinations have shown that the peripheral zone of metastases demonstrating washout phenomenon in images acquired approximately 5 minutes after contrast injection represents the growing tumor margin, whereas the center contains necrotic areas. Echinococcal disease (see Fig. 11-18) may show irregular hypervascularization as a result of inflammatory changes; however, in most cases no significant irregular blood supply is noticed in the arterial phase, and only cystic lesions with septations and daughter cyst are demonstrated. Hodgkin's lymphoma and NHL typically appear hypovascular in arterial and portalvenous phase scans. On delayed images lesions may appear isointense to surrounding liver tissue as a result of the diffuse periportal infiltration that results in a homogeneous late enhancement of the affected areas. Thus, this type of lesion can as well be classified as a tumor with a delayed persistent enhancement. In children, hepatoblastoma also has to be considered in the differential diagnosis of liver tumors because these primary liver lesions, found only in children and young adults, are also mainly hypovascular (Fig. 11-21).

On T₂-weighted imaging, hypovascular lesions may appear homogeneously hyperintense like hepatic cysts or may present irregular hyperintensity compared with surrounding liver tissue such as most solid metastases.
FIGURE 11–18. Echinococcal disease in a 36-year-old man with persistent right upper quadrant pain and blood eosinophilia. A, On T2-weighted imaging there is a septated, mainly hyperintense mass with sharp demarcation to surrounding parenchyma. B, on T1-weighted imaging, the mass is mainly hypointense with no enhancement after contrast administration (C). The perilesional liver parenchyma shows a slight increase in signal compared with surrounding normal liver because of compression of liver tissue. D, In another patient, a similar lesion is evident in the dorsal right liver lobe with typical septations.
FIGURE 11–19. Liver metastasis of colorectal carcinoma. A, On the T2-weighted image the liver lesion shows the so-called target sign, which is highly indicative of liver metastases. B, On T1-weighted unenhanced imaging, the lesion is hypointense with an even lower central signal intensity. In dynamic T1-weighted imaging, the lesion shows peripheral hypervascularization in the arterial phase (C) with slight contrast uptake of the lesion in the portalvenous phase sparing the central necrotic areas (D) and a peripheral washout sign in the equilibrium phase (E). This peripheral washout sign, which is characterized by a hypointense rim surrounding the lesion in images acquired approximately 5 to 10 minutes after contrast injection, is typically found in metastatic disease of the liver and so far has not been described in benign liver lesions. Unfortunately, it is not found in all liver metastases.
FIGURE 11–20. Liver metastasis from rectal adenocarcinoma with internal hemorrhage after chemotherapy in a 64-year-old man. A, $T_2$-weighted image shows a slightly hyperintense liver lesion (arrow) with a low signal intensity (SI) center. $B$, On the $T_1$-weighted image these central areas correspond with a slightly increased SI in the overall hypointense lesion. $C$, The periphery of the lesion has an irregular arterial contrast uptake, indicating an infiltrative growth. $D$, The central areas remain hypointense to surrounding parenchyma in the portalvenous phase. Note that the low SI of the central area in the $T_2$-weighted image is caused by hemorrhage during chemotherapy. Thus, the overall appearance of the lesion is rather atypical for a metastasis of an adenocarcinoma, and the clinical information about previous chemotherapy is essential for diagnosis.
FIGURE 11–21. Hepatoblastoma in a 16-month-old boy with abdominal pain and increased abdominal circumference. A and B, T2-weighted axial and coronal images show multiple inhomogeneously hyperintense liver lesions (arrows) with indistinct borders to surrounding liver parenchyma. C, The lesions are mainly hypointense on T1-weighted fat-saturated magnetic resonance imaging, but small areas of hemorrhage with high signal intensity can be detected, indicating internal hemorrhage. D, After contrast administration, most of the lesions remain hypointense. In contrast to hepatocellular carcinoma, hepatoblastomas found in pediatric patients are mainly hypovascular, although sometimes small areas of hypervascularization may be noted.
In metastases with central liquefactive necrosis, central areas may have increased signal relative to the more solid components of the metastases. This feature has been termed the “doughnut sign” (Fig. 11-22) and is most commonly identified in metastatic lesions of adenocarcinoma. In some metastatic lesions, a bright halo surrounding a less intense nodule is present on T2-weighted images, the “halo sign” (see Fig. 11-22), in which the peripheral halo represents viable tumor surrounding low-signal coagulative necrosis.

Lesions Presenting Delayed Persistent Enhancement

Focal liver lesions presenting delayed with persistent enhancement are characterized by a delayed uptake of contrast medium. Thus, lesions appear isointense or hyperintense on delayed-phase images, whereas most of the lesion is hypointense in the arterial or portalvenous phase. On scans 10 to 15 minutes after contrast administration, lesions may still demonstrate hyperintensity compared with surrounding liver tissue because of the beginning washout of contrast medium in normal liver and contrast pooling (delayed washout) in liver diseases.

The most common benign nonepithelial primary liver tumor is the hemangioma, a liver lesion typically presenting delayed persistent enhancement after contrast injection (see Figs. 11-13 and 11-16). In the arterial phase, as known from dynamic CT imaging, hemangiomas typically show a nodular peripheral enhancement (Fig. 11-23) with subsequent centripetal filling in the portalvenous and delayed-phase images. This classic appearance of hemangioma in dynamic imaging, together with the homogeneous high signal intensity in T2-weighted images, is highly specific for the diagnosis of hemangioma. Large or so-called giant hemangioma may have scar tissue, thrombosis, myxoid changes, or larger fibrotic areas (Fig. 11-24). Thus, lesions may not show complete fill-in on delayed-phase images, and inhomogeneous internal morphology may be noted on T1-weighted scans. Peliosis hepatitis is defined by the presence of cystic blood-filled spaces. In contrast to extreme sinusoidal dilatation, microscopy of peliosis hepatitis should show evidence of lysis of reticulin fibers supporting normal anatomy. Macroscopic peliosis hepatitis is usually induced by anabolic, estrogenic, or adrenocortical steroids. It also has been reported with a variety of chronic diseases such as malnutrition, leukemia, vasculitis, AIDS, and others. Because of the presence of dilated blood-filled spaces, peliosis hepatitis may mimic a hemangioma with high signal intensity in T2-weighted images and delayed enhancement. In the cases documented so far in our department, the main differences on imaging from a hemangioma were inhomogeneous margins, no nodular peripheral enhancement on arterial phase images, and sometimes irregular early enhancement in the arterial phase in parts of the lesion. Hemangiosarcoma, a malignant nonepithelial primary liver tumor, is the most common sarcoma arising in the liver. As a result of large cavernous, blood-filled spaces and rarely seen solid cellular masses, angiosarcomas show delayed persistent enhancement after intravenous contrast injection and high signal intensity in T2-weighted images, comparable to that for hemangioma. As an important difference to hemangioma, angiosarcomas show inhomogeneous enhancement in the arterial phase with early enhancement of central regions, sometimes resulting in rather centrifugal than centripetal filling as for hemangioma.
FIGURE 11–23. Hemangioma in a 38-year-old woman with a sonographic diagnosis of a large, focal liver lesion. A, On T₂-weighted images the lesion in the right liver lobe demonstrates a homogeneous high signal intensity with sharp, lobulated borders. B, on T₁-weighted unenhanced imaging, the lesion is hypointense with nodular peripheral enhancement in the arterial phase image (C). In the portalvenous phase (D) and the equilibrium phase (E), the lesion shows centripetal filling clearly indicating a hemangioma.
FIGURE 11–24. Giant hemangioma in a 34-year-old man with right upper quadrant pain. Because large hemangiomas often show areas of thrombosis or myxoid changes, the high SI of the lesion on T₂ (A) is inhomogeneous. B, on the T₁ weighted unenhanced image, the lesion shows an inhomogeneous hypointense signal. Nodular peripheral enhancement in the arterial phase (C), together with centripetal filling in the portalvenous (D) and the equilibrium phase (E) after injection of contrast medium, can be noted. In images acquired 15 minutes after contrast administration, (F) the central areas of the lesion still show no contrast media uptake because of thrombosis and myxoid changes (arrow). However, as a result of the postcontrast nodular peripheral enhancement that increases with time, the diagnosis of a giant hemangioma can be made.
Intrahepatic cholangiocellular carcinoma (CCC) represents a malignant epithelial primary liver tumor that typically appears hypointense in unenhanced arterial phase and portalvenous phase $T_1$-weighted images (Fig. 11-25). On $T_1$-weighted images, CCCs typically appear hyperintense. In cases without larger areas of necrosis or CCC with desmoplastic changes, delayed-phase images show a homogeneous uptake of contrast into the lesion. Thus, CCC may appear hyperintense in comparison to surrounding liver tissue in delayed-phase $T_1$-weighted images. Comparable findings concerning contrast dynamics can be made in metastases of leiomyosarcoma (e.g., of the uterus). Lesions smaller than 5 cm usually appear homogeneously hypointense on unenhanced $T_1$-weighted images and homogeneously hyperintense on $T_2$-weighted images and show delayed enhancement as

![Figure 11-25](image-url)

**Figure 11–25.** Intrahepatic cholangiocellular carcinoma in a 62-year-old woman with right upper quadrant pain and jaundice. A, The $T_2$-weighted image shows a large inhomogeneous hilar liver lesion with increased signal intensity and peripheral focal biliary dilatation (arrows). B, On $T_1$-weighted imaging the lesion demonstrates a hypointense signal, and irregular peripheral enhancement can be noted in arterial phase $T_1$-weighted imaging (C), indicating infiltrative growth of the lesion. D, In the portalvenous phase, initial contrast enhancement can be noted in the peripheral areas of the lesion, which increases with time. Thus, on the $T_1$-weighted image 5 minutes after contrast administration (E), the lesion shows almost complete contrast media uptake. F, This delayed enhancement is more obvious on the $T_1$-weighted fat-saturated image 15 minutes after contrast administration.
a result of enlarged extracellular space after intravenous contrast injection, thus appearing slightly hyperintense or isointense in delayed-phase images.

In addition to the classification made in dynamic imaging of the liver, focal liver lesions can be classified in T<sub>2</sub>-weighted imaging. Figure 11-26 gives a general overview of the signal intensity characteristics found in T<sub>2</sub>-weighted imaging of liver lesions.

Regarding vascular diseases of the liver, mainly portal thrombosis and Budd-Chiari syndrome have to be considered. Spin echo images (because of flow void) as well as flow-sensitive gradient echo images are useful in depicting patency of the main portal vein and its major branches. On contrast-enhanced images, thrombus material may be displayed as hypointense material in the portal vein. Indirect signs of portal thrombosis are increased arterial vascularization of affected liver lobes (in thrombosis of a major branch of the portal vein) as well as a generally reduced enhancement of the liver in the arterial and portal venous phases after contrast administration.

Budd-Chiari syndrome results from obstruction of the venous outflow from the liver, located in either the main hepatic veins or in small to intermediate-size veins, also known as veno-occlusive disease. In acute Budd-Chiari syndrome, perihilar ascites, enlargement of the liver, and inhomogeneous and delayed enhancement of the liver may be found (Fig. 11-27). The inhomogeneous enhancement is caused by the decreased portal venous perfusion of the liver caused by the increased vascular resistance. Peripheral liver tissue may show signs of severe hypovascularization. This may lead to atrophy of peripheral liver areas in chronic disease, whereas the more central parts may show collateral venous drainage. In the chronic stage of Budd-Chiari syndrome, hypertrophy of segment 1 can be found resulting from the separate venous drainage of this segment directly into the inferior caval vein. The same can be found for the lower liver segments 6 and 5, which often demonstrate accessory liver veins.

### SPLEEN

In spite of its important role in immunology, the spleen has always been of less interest for clinicians and radiologists compared with other parenchymal abdominal organs. However, as we know from macro- and microscopic disease, the spleen is involved in a wide variety of neoplastic and infectious diseases, although this involvement often cannot be visualized by noninvasive imaging modalities and thus remains undetected. MRI, with its great potential in soft tissue contrast and the possible imaging of splenic perfusion, seems to be a reliable tool for evaluating diffuse and focal splenic disease.

The spleen develops during the fifth week of gestation as a mesenchymal proliferation of the dorsal mesogastrium. It is located intraperitoneally and follows the axis of the 10th rib in the left upper quadrant. Ligamentous fixation includes the gastrosplenic ligament with the arteria and vena gastrica brevis and arteria gastroepiploica sinistra and the phrenicosplenic ligament with the splenic artery and vein. Traumatic rupture of those fixating structures may lead to splenic torsion or the so-called wandering spleen. Accessory spleens most often develop in the omentum majus or gastroplenic ligament (in 10% of individuals).

A physiologic spleen in adults is about 11 cm long, 7 cm wide, and 4 cm thick with a weight of approximately 150 g. It is surrounded by a fibrous capsule. Microscopically, it consists of the red and white pulp. The red pulp is mainly built of the splenic sinuses, whereas the white pulp comprises the lymphoid elements. Typically, T lymphocytes are found in the periafferent T-cell-rich lymphoid sheaths around central arteries. Primary and secondary B-cell follicles are located at the periphery of the T-cell zone. Hemodynamically, a fast compartment with early passage of the blood from the arteries to the sinus can be distinguished from a slow compartment, where the in-flowing blood remains for up to hours in the mantle zone before reaching the
ABDOMEN

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FIGURE 11–27. Budd-Chiari syndrome in a 38-year-old woman with ascites and enlarged liver on sonography. A, T₂-weighted imaging shows perihepatic and perisplenic ascites. The liver surface appears rounded, and liver segment 1 is hypertrophied. B, On T₁-weighted imaging, the liver shows homogeneous signal intensity. C and D, On T₁-weighted dynamic imaging, the hepatic veins cannot be identified. There is an inhomogeneous peripheral hypervascularization (arrow), indicating an increased arterial blood supply. Overall, the perfusion of the liver appears delayed because of the increased vascular resistance. In an image acquired 5 minutes after contrast injection (E), peripheral areas of the liver appear hypointense, indicating early necrosis. Note as well the enlarged spleen.

sinusoidal structures. The normal passage of blood is via the splenic artery to central arteries and arterioles to capillaries, which lead to the sinusoids, where the blood is drained by small veins to the splenic vein into the portalvenous circulation. There are no afferent lymphatic vessels to the spleen. Splenic function consists of maturation of reticulocytes, elimination of defective erythrocytes, and some pooling function for thrombo-
cytes. During the first 4 months of gestation, there is additional physiologic hematopoiesis.

Because sonographic evaluation of the spleen is quite unreliable, most splenic diseases are detected by CT and MRI, often incidentally. Once a focal lesion is found, further evaluation has to be made by means of imaging because fine-needle biopsy of the spleen in contrast to hepatic biopsy is rarely performed.
Evaluation of splenic disease in imaging relies considerably on the evaluation of splenic perfusion. The inhomogeneous contrast enhancement of the red and white pulp during the early arterial phase results in a high detection rate of even small focal lesions. They present as a gap in the normal perfusion pattern, either as a hypoperfused area or a homogeneously enhancing small lesion.

The use of unenhanced MRI in the detection of focal lesions is restricted because splenic and tumorous tissue in a large number of cases demonstrates similar signal intensities. Imaging of contrast dynamics offers an additional approach to detect splenic disease. Bolus administration of extracellular Gd-based contrast agents leads to an early irregular, arciform pattern of enhancement of the spleen within the first 25 to 30 seconds after contrast administration. A second perfusion phase is characterized by its homogeneous peak enhancement about 60 seconds after contrast injection. Imaging of focal lesions depends primarily on the contrast between a hypointense lesion and the arciform-enhancing splenic tissue. In the second phase of perfusion, the signal intensities become more alike as contrast slowly distributes in the extracellular compartment of the lesion. Thus, early imaging of the spleen after contrast administration is crucial for lesion detection. Figure 11-28 gives an overview for classification of splenic tumors, diagnosed on contrast-enhanced MRI. Intravenous administration of superparamagnetic iron oxide (SPIO) particles represents a second approach to increase the contrast between the normal parenchyma and focal lesions. Iron particles are taken up only by reticuloendothelial cells and thus lead to a loss of signal intensity in normal splenic tissue, whereas focal lesions remain unaffected. In contrast to the use of the extracellular contrast agents, the time window is much longer because of the intracellular uptake of iron in the mononuclear phagocytic system.

The spleen may be affected by many different diseases. Often, as in infectious disease, the involvement remains clinically relatively silent, and in trauma it may even be life threatening. Splenic malformations include accessory and ectopic splenic tissue as well as agenesis of the spleen. Agenesis of the spleen is very rare, often seen in combination with cardiovascular malformations, known as Ivemark’s syndrome. Most children die of overwhelming postsplenectomy infections (Streptococcus pneumoniae, malaria). In contrast, accessory spleens are seen in up to 10% of individuals. An ectopic spleen can be found intrathoracically (agenesis of the diaphragm) or in an umbilical herniation. In splenogonadal fusion, fibrous tissue connects the spleen and the gonads; heterotopic splenic tissue on the gonads can be found as well, but only left sided. In acute trauma of the upper quadrants, the spleen is the most affected parenchymal organ, often in combination with rib fractures. Trauma may result in subcapsular hematoma without discontinuity of the splenic capsule, or in more severe cases in a laceration, which may lead to intraperitoneal loss of blood. In up to 85% of splenic trauma, CT or MRI may demonstrate perisplenic blood clots, thus pointing to the diagnosis. Often there is a delay of weeks between acute trauma and onset of symptoms in splenic rupture. Post-

![Figure 11-28. Splenic disease.](image-url)
FIGURE 11–29. Splenic regeneration after splenectomy because of a laceration in a 56-year-old with a history of trauma. Routine sonography demonstrated a new lesion in the left upper abdomen. A, On $T_2$, a round lesion with sharp borders and a signal intensity comparable to that of the liver is noted. It is homogeneously hypointense on $T_1$-weighted (B) and $T_1$-weighted fat-saturated (C) images without signs of necrosis or hemorrhage. After injection of a gadolinium chelate, the lesion shows the typical arciform enhancement pattern like a normal spleen in the early dynamic phase (D) with a homogeneous enhancement in a later acquisition (E). In general, the differential diagnosis includes splenic artery aneurysm as well as a tumor of the pancreatic tail.

traumatic findings include splenic torsion with traumatically ruptured ligaments and splenosis (Fig. 11-29). Splenosis after laceration or rupture of the spleen represents splenic tissue that is seeded out intraperitoneally and may be incidentally found without causing any primary symptoms.

With regard to inflammation, the spleen reacts in all different bacterial infections, with the exception of peritonitis; thus, peritoneal mesothelium has a reticulo-histiocytic function. Often the changes are more diffuse and do not allow an etiologic classification (splenitis). The spread of the infectious agents most often occurs hematogenously. An acute focal perisplenitis may arise in splenic infarctions. Specific infections such as tuberculosis, syphilis, or brucellosis and yersiniosis may lead to a more granulomatous infiltration resembling sarcoidosis. Abscess formation is most often associated with bacterial endocarditis, trauma, or hemoglobinopathies; it may be found in patients with hemorrhagic hereditary telangiectasia resulting from AVMs of the lung. In this
context, patients are predisposed to brain abscess and other visceral abscesses formations. Granulomas typically develop in patients with sarcoidosis or Wegener's granulomatosis. Granulomas may be as large as 5 mm and are typically seen throughout the spleen as glioma lesions that are hyperintense on contrast-enhanced images and often also have low signal intensity on unenhanced T₁-weighted images.

Disorders of splenic or portal circulation may result in splenomegaly or splenic infarctions. Splenomegaly caused by hyperemia is found with elevated pressure of the venous circulation such as in cardiac insufficiency or portal hypertension. Thrombosis of the splenic vein also leads to a splenomegaly, which may be caused by torsion, compression, or thrombus formation in coagulation or lymphoproliferative disorders as well as in acute pancreatitis. Infarctions are typically anemic and caused by emboli (endocarditis, arteriosclerosis) or thrombus formation (CML, sickle cell anemia).

With regard to neoplasms, the spleen is most often secondarily involved in Hodgkin's lymphoma and NHL with an incidence of up to 60% to 100% in small lymphocytic NHL. In Hodgkin's disease (HD), up to one third of patients have splenic involvement at staging laparotomy. In patients with the lymphocyte-predominant type of HD, the incidence seems to be somewhat lower. Unfortunately, increased splenic size does not invariably stand for infiltration. The spleen may also have a normal size. Typically, in disease, randomly distributed fleshy nodules are found, which may be confluent, but isolated small (1 mm) foci or mililiary small nodules may be present as well, which cannot be diagnosed by means of imaging. Because liver or bone marrow infiltration is rare without splenic involvement, this diagnostic test seems to be a more reliable prognostic factor than imaging.

In NHL as well, the spleen may have a normal size despite infiltration. Involvement may be depicted as mililiary small nodules typically found in follicular lymphoma, large solitary nodules, or a diffuse infiltration of the splenic parenchyma without evidence of a focal lesion, which may be seen in diffuse large cell lymphoma. A sign on imaging indicating diffuse infiltration of the spleen in HD and NHL is the missing inhomogeneous enhancement of the spleen in images 25 to 30 seconds after contrast administration. This rather homogeneous enhancement may also be found in cases of circulation disorders. Rare cases of primary splenic lymphoma are described in literature. This entity should be applied only to lymphoma involving the splenic parenchyma or the splenic lymph nodes without evidence of involvement of other sites. Its incidence is as low as 1% of all lymphomas, and most of them are NHL. In all cases of splenic involvement by lymphoid cells, mononucleosis is the main differential diagnosis, especially in younger patients.

Splenic hemangioma is the most common benign splenic tumor. Comparable to hepatic hemangioma, the size may vary from 3 mm up to several centimeters (Fig. 11-30). On rare occasions, a so-called giant hemangioma (>10 cm) may be found. Symptoms may result from consumption coagulopathy, rupture, or thrombocytopenia, but most patients remain asymptomatic. Splenic hemangiomas usually have high signal intensity on T₁-weighted images comparable to the findings in liver hemangiomas. If no degeneration or thrombosis is present, they demonstrate a homogeneous delayed, persistent enhancement on images after injection of a Gd chelate. If other organs such as liver or skin are involved as well, diffuse angiomatosis has to be considered in the differential diagnosis.

Most hemangiomas are cavernous with large blood-filled spaces lined by endothelium and separated by fibrous septae or splenic pulp. Larger hemangiomas may show regressive changes such as infarction, thrombosis, or fibrosis. Hemangiomas have to be distinguished from splenic peliosis, which is typically of a more diffuse nature and is found in patients with wasting disease or under medication with anabolic steroids. Splenic lymphangioma is a rare disease and most often occurs in diffuse lymphangiomatosis with involvement of other organ systems. It is usually found in the capsular or trabecular areas of the spleen, where most of the lymphatics are located, and may present as a focal nodule as well as a multicentric lesion. On T₁-weighted images, lymphangioma appears similar to a hemangioma. However, there is no enhancement of the lesions after Gd injection, and typically multiple septations can be demonstrated.

Hemangioendothelioma and hemangiopericytoma are rarely found in the spleen. The diagnosis of hemangioendothelioma is typically made in cases of hemangioma with cellular atypia and mitoses, representing a borderline tumor between hemangioma and angiosarcoma. Angiosarcoma of the spleen is another rare primary splenic tumor, occurring most often in older patients presenting with abdominal pain or even rupture of the spleen. Symptoms like coagulopathy or thrombocytopenia may be found in larger tumors. Metastatic spread most often involves the liver, lung, and bone, and the prognosis is grave. On macroscopic exam, there are typically multiple ill-defined nodules with large areas of hemorrhage or necrosis. The classic differential diagnosis in imaging is hemangioma. The irregular borders and the infiltrative growth pattern of an angiosarcoma favor the diagnosis of a malignant vascular lesion. However, there may be an overlap in the imaging appearance with multiple hemangiomas and peliosis hepatis. Biopsy in suspected angiosarcoma of the spleen should be extensive because angiosarcoma may have spots of benign appearance that may be misinterpreted as hemangioma. Inflammatory pseudotumor of the spleen is a reactive lesion sometimes associated with prior bacterial infection. It typically presents as a solitary nodule with areas of hemorrhage or necrosis, thus mimicking infiltration by a lymphoid or even a solid tumor. Definitive diagnosis in most cases is possible only by histology.

Epithelial cysts of the spleen are typically solitary tumors with an epithelium lining. They are mostly asymptomatic lesions but may sometimes present as a mass lesion (Fig. 11-31) or may be complicated by rupture or bacterial superinfection. Uncomplicated cysts show homogeneous high signal intensity on T₁-weighted images, and there is no enhancement after injection of
FIGURE 11–30. Splenic hemangioma in a 38-year-old man with a gastric ulcer. Routine sonography demonstrated a large splenic lesion. Comparable to hepatic hemangiomas, splenic hemangiomas have high signal intensity on T₂-weighted magnetic resonance images (A) and are hypointense or even isointense to normal splenic tissue on unenhanced T₁-weighted images (B). C–E, The lesion shows peripheral nodular enhancement on dynamic T₁-weighted images. F, Delayed persistent enhancement is visible 20 minutes after contrast administration. Note the typical stripelike appearance (arrow) of normal splenic parenchyma in the early dynamic image (C).
FIGURE 11–31. Splenic cyst in a 16-year-old girl. A large splenic lesion was found on routine sonography, and there was no history of trauma. A, T2-weighted magnetic resonance imaging shows a large hyperintense cyst with sharp demarcation from the surrounding parenchyma. After injection of contrast, the early dynamic T1-weighted image (B) shows typical inhomogeneous enhancement of remaining splenic tissue with homogeneous enhancement in a later dynamic image (C).

contrast. Parasitic cysts may, as with hepatic disease, arise in infection with echinococcus, presenting as unilocular or multilocular disease with septated cystic lesions that may be ill defined from surrounding splenic tissue. In contrast to true epithelial cysts, false cysts or pseudocysts arise after trauma, the result of liquefaction of a hematoma. They typically lack an endothelial lining, and the fibrous wall may show calcifications. Patient history is the most important information in diagnosis of posttraumatic pseudocysts because imaging findings may be similar to true epithelial cysts.

In cases of splenic metastases, the spleen may be involved by tumor growth in the vicinity (stomach, pancreas, kidney) or by hematogenous spread. There is great variability in the reported frequency of splenic metastases. Regardless, the most common primaries are malignant melanoma and breast and lung cancer. Involvement may be present as a solitary nodule (Fig. 11-32) as well as miliary lesions or diffuse replacement of splenic parenchyma. Frequency of splenic metastases seems to increase with greater long-term survival of cancer patients. On MRI usually solid, hypovascular (in comparison with splenic tissue) tumors are identified that may show central necrosis. On T2-weighted images, a solid metastasis may be either hyper- or isointense; however, if central necrosis is present, a central high-signal-intensity area may be identified.

PANCREAS

The normal pancreas on conventional T1-weighted spin echo images is of intermediate signal intensity similar to liver and hypointense compared with surrounding retroperitoneal fat. With fat suppression, the relative signal intensity of the pancreas on T1-weighted images increases substantially so that normal pancreatic tissue is the brightest soft tissue structure in the upper abdomen and can be clearly demarcated from surrounding structures (Fig. 11-33). On T2-weighted images, the pancreas is isointense or slightly hyperintense to the liver. The fat surrounding the pancreas is typically hyperintense compared with the pancreas on T2-weighted non-fat-suppressed images. The pancreatic duct as well as the common bile duct in the pancreatic head show a T2 value that is much longer than that of adjacent soft tissue and thus is displayed with high signal intensity on T2-weighted images or may even be depicted exclusively as in MRCP. Concerning contrast dynamics, the pancreas enhances before the liver after bolus injection of extracellular distributed Gd chelates. As for the liver, it is important to image the pancreas in a dynamic fashion after contrast administration. This approach improves lesion detection and provides, on the basis of enhancement characteristics, information regarding differential diagnosis. Because of the central position of the pancreas...
FIGURE 11–32. Splenic metastases in a 36-year-old woman with a clinical history of breast cancer and an enlarged spleen with a focal lesion on routine follow-up sonography of the upper abdomen. A, The T₂-weighted image shows an inhomogeneous, hyperintense splenic lesion (arrow). There are signs of central necrosis on T₁-weighted contrast enhanced (B) and T₁-weighted fat-suppressed contrast enhanced (C) images. On contrast-enhanced images after intravenous injection of a gadolinium chelate, the lesion is primarily hypovascular.

FIGURE 11–33. Normal pancreas on T₁-weighted fat-suppressed images. A and B, The pancreatic parenchyma is best visible on T₁-weighted fat-suppressed images because the retroperitoneal fat remains dark and the parenchyma can well be appreciated due to its homogeneous high signal intensity. Note the close relationship of the pancreatic tail to the kidney and splenic hilum and the clear distinction of the pancreatic head from the neighboring duodenum (arrow).
in the upper abdomen, special problems may arise for MRI. Artifacts from respiration, peristalsis, and vascular pulsation may obscure fine detail of the pancreatic parenchyma; thus, different strategies should be applied to improve the diagnostic quality of pancreatic MRI. These include signal averaging, presaturation, antiperistaltic medication, and in particular the use of fast breath-hold imaging techniques. Oral contrast material may be used in addition to achieve better differentiation of adjacent bowel loops.

With regard to their pathologic classification, pancreatic tumors can be categorized as tumors of the exocrine or endocrine pancreas, tumor-like lesions, and secondary tumors. Figure 11-34 gives an overview of the pathologic classification of pancreatic tumors. For the differential diagnosis of pancreatic lesions on MR imaging, a classification that reflects the imaging characteristics should be used. For this reason, pancreatic lesions may first be subdivided into solid, cystic, or complex masses and may then be subdivided into hyper- or hypovascular tumors as well as solitary or multiple lesions (Fig. 11-35).

The differential diagnosis of pancreatic adenocarcinoma is an important diagnostic challenge in imaging of pancreatic masses. Whereas adenocarcinomas of the head of the pancreas frequently come to attention at a rather small size as a result of obstruction of the bile duct producing jaundice (Fig. 11-36), tumors of the pancreatic body and tail tend to grow insidiously, presenting with almost no or ill-defined late symptoms and a large tumor size at initial presentation (Fig. 11-37). Pancreatic carcinoma typically has decreased signal intensity on T₁-weighted images. However, on T₂-weighted images, pancreatic carcinoma may be isointense or only slightly higher in signal intensity than the normal pancreas. Only in large tumors with high-signal-intensity central necrosis does T₂-weighted imaging help in the diagnosis of pancreatic carcinoma. On T₁-weighted images with fat suppression, pancreatic carcinoma has substantially decreased signal intensity compared with the high signal intensity of normal pancreatic tissue. Thus, this imaging sequence is used principally for the diagnosis of small pancreatic carcinomas. One major problem in imaging of pancreatic carcinoma are those cases that present together with pancreatitis, because in these cases the signal intensity of pancreatic parenchyma on T₁-weighted images is lowered. Thus, the contrast between pancreatic carcinoma and inflammatory pancreatic tissue is decreased.

The classic sign of pancreatic carcinoma in the pancreatic head is the “double-duct” sign, with obstruction of the common bile duct as well as the pancreatic duct...
Islet cell tumors
Inflammatory pseudotumors
Hypervascular metastasis
Pancreaticoblastoma

Solid

Hypervascular

Solitary lesion
Multiple lesions

Hypovascular

Metastasis
Lymphoma
Multiple myeloma

Serous microcystic neoplasm (microcystic adenoma)
Mucinous cystic neoplasm
Congenital retention cyst
Parasitic cyst
Pseudocyst in chronic pancreatitis
Endometriosis

Lymphangioma
Mucinous cystic neoplasm
Cystic transformation (von Hippel-Lindau)
Pseudocysts in chronic pancreatitis
Endometriosis

Teratoma
Mucin hypersecreting carcinoma
Solid and papillary epithelial neoplasm
Endometriosis

Cystic

Solitary lesion
Multiple lesions

Hemangioma (delayed enhancement)

Lymphoma

Multiple myeloma

Ductal adeno—CA
Anaplastic—CA
Hypovascular metastasis
Lymphoma

FIGURE 11–35. Classification of pancreatic lesions in magnetic resonance imaging. (CA, carcinoma.)

FIGURE 11–36. Pancreatic adenocarcinoma of the pancreatic head in a 70-year-old man with weight loss and abdominal pain. A, On T2-weighted imaging, a small tumor of the pancreatic head with slightly elevated signal compared with normal pancreatic parenchyma can be identified. B, T1-weighted imaging shows early infiltration of the peripancreatic fat; however, there seems to be an intact fat plane between the tumor and the superior mesenteric vessels. C, T1-weighted fat-suppressed imaging again demonstrates the lower signal intensity of the carcinoma (arrow) compared with normal parenchyma.
FIGURE 11–37. Pancreatic adenocarcinoma in a 73-year-old man with weight loss and increasing back pain of several weeks duration. A and B, Axial T2-weighted images show significant atrophy of the pancreatic tail with dilatation of the pancreatic duct (arrow) as well as a large tumor of the pancreatic head with infiltration of the peripancreatic fat. The tumor is very inhomogeneous with central areas of high signal intensity. On T1-weighted (C) and T1-weighted fat-suppressed (D) images, encasement of the superior mesenteric vessels can be clearly visualized. The unsharp borders with infiltration of peripancreatic fat and retroperitoneal structures are best displayed on the T1-weighted fat-suppressed image. E, On early-phase T1-weighted dynamic imaging, after contrast administration the tumor is hypovascular and shows inhomogeneous enhancement of peripheral areas.

(Fig. 11-38). One should always look for an obstructed pancreatic duct as an indirect sign of pancreatic carcinoma. Typically, in a case of pancreatic carcinoma, the obstructed and dilated duct is surrounded by atrophic pancreatic tissue without signs of chronic pancreatitis such as pseudocyst formation or inflammatory changes in the surrounding peripancreatic fat. For contrast-enhanced MRI of the pancreas, bolus injection of Gd chelates with early dynamic imaging using breath-hold T1-weighted sequences is the most reliable technique for detection of pancreatic carcinoma. Typically, pancreatic carcinoma is hypointense precontrast and also hypovascular (and thus remaining hypointense) during the first 1 to 2 minutes after contrast administration when normal pancreatic tissue maximally enhances. In images 2 to 5 minutes after contrast injection, leakage of contrast to the extracellular fluid compartment of the tumor reduces the contrast between pancreatic carcinoma and normal pancreatic tissue. Thus, pancreatic carcinoma may become isointense. In staging of pancreatic carcinoma, local as well as remote spread of tumor has to be considered concerning resectability of the tumor. Because of the special lymphatic drainage of the pancreas and the lack of a capsule surrounding the organ, pancreatic carcinoma can metastasize to almost any lymph node group of the upper abdomen. Metastases may occur in the liver or to the peritoneum; either finding makes the tumor unresectable. Another area of special interest concerning staging of pancreatic carcinoma is the involvement of vessels of the upper abdomen, specifically encasement of the superior mesenteric artery, superior mesenteric vein, or portal vein. Figure 11-39 gives an overview concerning resectability criteria for ductal adenocarcinoma of the pancreas on MRI.
FIGURE 11–38. Pancreatic adenocarcinoma of the pancreatic head in a 74-year-old man with sudden onset of painless jaundice and weight loss. A, The axial T2-weighted image shows dilatation of the choledochal as well as the main pancreatic duct (arrows), the so-called double-duct sign. The pancreatic tail shows parenchymal atrophy without signs of acute or chronic pancreatitis, which is rather common in pancreatic carcinoma. B, Magnetic resonance cholangiopancreatography confirms obstruction of the common bile duct and pancreatic duct. C, On the T1-weighted fat-suppressed unenhanced image, a low-signal-intensity (SI) lesion in the pancreatic head can be identified with small areas of remaining high SI pancreatic tissue in the more medial aspect of the pancreatic head. D, The T1-weighted contrast-enhanced image demonstrates a hypovascular area in the pancreatic head close to the papillar region consistent with pancreatic adenocarcinoma.
In imaging of islet cell tumors of the pancreas, distinction has to be made between tumors that produce hormonally active peptide (functioning islet cell tumors) and those tumors that do not produce peptides (nonfunctioning islet cell tumors). Because of the production of hormonally active peptides and the resulting clinical symptoms, functioning islet cell tumors typically are much smaller at presentation. In distinction, nonfunctioning tumors are often larger (Fig. 11-40) and clinical symptoms that lead to presentation include mass effect and metastases. Generally, islet cell tumors of the pancreas show high signal intensity on $T_2$-weighted and low signal intensity on $T_1$-weighted fat-suppressed images. After intravenous bolus injection of Gd chelates, they show very early enhancement. Thus, imaging of islet cell tumors in the early arterial phase is of great importance. Functioning endocrine pancreatic neoplasms have to be considered in patients with classic multiple endocrine neoplasia type I (MEN I). An increased incidence of functioning pancreatic neoplasms also has to be considered in von Hippel-Lindau disease, in which other findings like pancreatic cysts may also be evident. Because of the potential malignancy and multicentricity of endocrine pancreatic neoplasms and the lack of pathologic findings that can clearly prove nonmalignant tumors, preoperative imaging is very important in the evaluation of patients with an assumed pancreatic endocrine neoplasm.

Insulinoma of the pancreas is the most frequent islet cell tumor; 90% of these tumors are benign, and in the majority of cases patients have solitary neoplasms. Gastrinomas, the most frequent islet cell tumor associated with MEN I, however, are multicentric in 20% to 40% of cases and malignant in up to 60%. Gastrinomas as well as insulinomas are hypointense on $T_1$-weighted fat-suppressed images and hyperintense on $T_2$-weighted sequences. However, gastrinoma may show larger cystic areas in contrast to insulinoma. Rare functioning islet cell tumors include glucagonoma, VIPoma, and somatostatinoma; each has a distinct clinical presentation. As a reminder, approximately one third of pancreatic tumors are classified as insulinoma, one third are other functioning islet cell tumors, and one third are nonfunctioning islet cell neoplasms.

True pancreatic cysts are rare and typically occur in combination with a systemic disease such as von Hippel-Lindau disease. Other congenital diseases that may be associated with true pancreatic cysts are cystic fibrosis and polycystic kidney disease. True pancreatic cysts have uniform high signal intensity on $T_2$-weighted images, have a thin wall, lack contrast enhancement, and are not associated with chronic pancreatitis.

The term pancreatic pseudocyst refers to a collection of fluid consisting of pancreatic juice without a true cyst lining. This means that the fluid is kept within a fibrous capsule. Pancreatic pseudocysts are typical complications of pancreatic inflammation or trauma. Normally, in acute pancreatitis they are rather ill defined and in most of the cases resolve spontaneously within 3 to 4 weeks. However, chronic pseudocysts (Fig. 11-41) may occur within the pancreatic parenchyma. True neoplasms of the pancreas that appear cystic include microcystic adenoma, which is also referred to as serous cystadenoma, as well as mucinous cystic neoplasms, which include a spectrum of neoplasms characterized by the production of mucus. In microcystic adenoma, typically multiple, lobulated cysts with thin walls and thin internal septations can be found (Fig. 11-42) that show almost no contrast enhancement after injection of Gd chelates. However, in rare cases, some solid areas within the cystic tumor may be found that can show marked enhancement after contrast injection. Very rare tumors that should be mentioned in the context of pancreatic tumors are solid and papillary epithelial neoplasm, anaplastic carcinoma, metastases, and pancreaticoblastoma, a malignant pancreatic tumor predominantly found in children.
FIGURE 11–40. See legend on opposite page
FIGURE 11–41. Pancreatic pseudocysts in a 42-year-old man with a history of chronic pancreatitis. A–C, T₂-weighted images show marked dilatation of the pancreatic duct (arrow) and formation of pseudocysts in both the pancreatic head and tail. There is little remaining pancreatic tissue; however, no solid tumor can be identified.
FIGURE 11–42. Microcystic adenoma of the pancreatic head in a 65-year-old woman with upper abdominal pain and tumor of the pancreatic head on sonography. A and B, On T₂-weighted axial and coronal images, a large multicystic tumor of the pancreatic head can be identified with thin-walled septa. The coronal image shows no dilatation of the common bile duct (arrow). T₁-weighted (C) and T₁-weighted fat-suppressed (D) images show low signal intensity (SI) of the cystic tumor with depiction of high SI normal pancreatic tissue in the more medial aspect of the pancreatic head (arrow). E, After contrast administration no significant enhancement is seen.
ADRENAL GLAND

In clinical practice, CT is the preferred method of adrenal lesion imaging because of its low cost, greater availability, and higher spatial resolution compared with MRI. This is especially the case for localization of small functioning adrenocortical tumors as in Cushing’s and Conn’s syndromes. However, MRI is recommended for characterization of incidental adrenal masses, pheochromocytoma, localization and preoperative evaluation of adrenal carcinoma, and imaging of patients with known hypersensitivity to iodinated contrast agents or poor renal function.

MRI of the adrenal glands is usually performed in both the axial and coronal planes with 5- to 8-mm sections. With large masses, coronal as well as sagittal views may be helpful for differentiation of adrenal from hepatic or renal lesions. Imaging protocols should include T1- and T2-weighted sequences before and after injection of Gd chelates. Chemical shift MRI using in- and opposed-phase breath-hold imaging and fat-suppressed sequences are important MRI techniques available for identifying lipid-containing adrenal adenomas.

Chemical shift imaging in this context takes advantage of the fact that protons in water and lipid precess at different frequencies within a magnetic field. Thus, by choosing a time to echo appropriate for the magnetic field strength, images can be obtained in which lipid and water are maximally in-phase or out of phase. Using this imaging technique, tissue that contains both water and fat, such as an adrenal adenoma that contain lipids, shows reduction of signal intensity on opposed-phase images when compared with the in-phase sequences. Alternatively, the other types of lesions in the differential diagnosis of an adrenal mass, including specifically metastases, pheochromocytoma, or adrenal carcinoma, do not contain sufficient lipid to demonstrate signal loss on opposed-phase images. However, 20% of adrenal adenomas only display a slight decrease in signal intensity on opposed-phase images, which does not allow a specific diagnosis of adrenal adenoma.

On conventional spin echo images, the adrenal glands have homogeneous low signal intensity compared with the surrounding perinephric fat. They are isointense or hypointense to liver on T1- and T2-weighted images. Applying fat suppression, the adrenal glands are hyperintense to both the liver and the adjacent fat. For imaging of the adrenal glands, both axial and coronal images should be acquired, and the slice thickness should not exceed 5 mm when a large mass is not present.

In imaging of adrenal masses in general, functional adrenal disorders have to be distinguished from adrenal masses with normal function. Functional adrenal disorders include disorders that arise from the adrenal cortex, such as primary hyperaldosteronism, Cushing’s disease, and adrenocortical carcinoma, as well as adrenomedullary disorders, such as pheochromocytoma and neuroblastoma.

In primary hyperaldosteronism, typical laboratory findings are elevated plasma aldosterone levels and increased plasma renin activity. Clinical symptoms include hypertension, hypokalemia, fluid retention, weakness, and cardiac arrhythmia. Approximately 79% to 90% of patients with primary hyperaldosteronism have a benign aldosterone-producing adenoma, so-called Conn’s syndrome, and can be treated by surgical resection. However, in 10% to 30%, diffuse adrenal hyperplasia accounts for primary hyperaldosteronism in which medical treatment is the preferred therapeutic option. Rarely, extensive aldosterone production can be found in adrenocortical carcinoma.

In primary hyperaldosteronism that is caused by diffuse adrenocortical hyperplasia, the adrenal glands may appear normal on imaging studies. Bilateral nodules may be found with a diameter of up to 2 to 3 cm. If this diffuse nodularity of the adrenal gland is missed because of one dominating large nodule, this nodule may be mistaken for an adrenal adenoma.

Endogenous causes of Cushing’s syndrome include cortisol-producing adenoma (20%), adrenocortical carcinoma (10%), and adrenocortical hyperplasia resulting from increased corticotropic (ACTH) production (70%). In the majority of patients with increased ACTH production and Cushing’s syndrome, adrenocortical hyperfunction is caused by a pituitary adenoma. In 10%, ectopic production of ACTH is found associated with neoplasms (e.g., bronchial carcinoid and tumors of the pancreas or thyroid). In general, cortisol-producing adenomas of the adrenal gland are easily demonstrated because they are usually larger than 2 cm in diameter. The majority of such adenomas are isointense to liver on T1-weighted spin echo images. To confirm the diagnosis of a lipid-containing adenoma, opposed-phase images should be acquired to show the reduction of signal intensity in the opposed-phase image. In adrenal hyperplasia associated with Cushing’s syndrome, up to 50% of patients have normal-appearing adrenal glands, whereas in the rest of patients there is diffuse unilateral or bilateral enlargement. Occasionally, the glands have a macronodular appearance.

Primary adrenocortical carcinoma is a rare malignancy that shows hormonal hyperfunction in up to 50% of patients. Depending on the hormone that is mainly produced, clinical findings include Cushing’s syndrome, feminization or virilization, and rarely primary hyperaldosteronism. Typically, large masses are found that have an inhomogeneous enhancement after injection of Gd chelates as a result of central necrosis (Fig. 11-43). In 30% of cases, calcifications may be noted on CT. Primary adrenocortical carcinoma typically is hypointense compared with the liver on T1-weighted images and has high signal intensity on T2-weighted images. In opposed-phase images, some small areas of the tumor may show signal reduction. However, the majority of the lesion does not show a signal intensity decrease in opposed-phase images. The homogeneous reduction of signal characteristic of adrenal adenomas is not observed.

Pheochromocytomas are neuroendocrine tumors that arise within the adrenal medulla or from paraganglionic tissue. In approximately 85% to 90% of cases, a paraganglioma arises in the adrenal gland; this is called a pheochromocytoma. These tumors are usually hormonally
Adrenal carcinoma in a 48-year-old man with weight loss. An inhomogeneous, partially hypointense adrenal mass is identified on the T2-weighted image (A) with areas of high signal intensity (SI) on the T1-weighted in-phase (B) sequence. The areas of high SI cannot be suppressed on the T1-weighted fat-saturated image (C), indicating hemorrhage. No obvious drop of SI in the solid portions of the lesion are noted in the opposed phase image (D). There is inhomogeneous contrast uptake of the lesion on the T1-weighted fat-saturated image (E), indicating degenerative changes, consistent with a malignant lesion.
active, producing both norepinephrine and epinephrine. Thus, symptoms such as sustained or paroxysmal hypertension, headache, sweating, and attacks of anxiety may be found resulting from transient elevation of catecholamine levels. Typical syndromes that are associated with pheochromocytoma are MEN IIa and MEN IIb, neurofibromatosis, and von Hippel-Lindau disease. Laboratory findings consistent with a pheochromocytoma include elevated levels of catecholamines and metabolites, including norepinephrine, vanillylmandelic acid, and metanephrine. In such cases (when a pheochromocytoma is assumed to be present), MRI is performed for screening of the adrenal glands. Adrenal pheochromocytomas are usually larger than 3 cm and typically have very high signal intensity on T2-weighted images (Fig. 11-44). Sometimes, however, this high signal intensity may be reduced because of calcification or hemorrhage. In opposed-phase images, they do not show signal intensity loss compared with the in-phase image.

Adrenomedullary hyperfunction in children in most cases is caused by neuroblastoma. Pheochromocytomas are very rare in childhood. Neuroblastoma represents the most common extracranial malignant tumor in children. Most neuroblastomas arise from the adrenal gland, but they can develop from virtually any tissue of neural crest cell origin. Neuroblastomas are usually poorly defined tumors in which calcifications can be found (in contrast with a nephroblastoma, which is the principal differential diagnosis in childhood). Neuroblastomas show very early and extensive encasement of the vessels of the upper abdomen and are rather large at presentation (Fig. 11-45). Different stages of maturation exist, including ganglioneuroblastoma and benign ganglioneuroma, which, in contrast to neuroblastoma, do not show the early encasement of vessels of the upper abdomen (Fig. 11-46) and show a more expansile growth pattern and distinct borders. Because hemorrhagic neuroblastoma in neonates may present as traumatic adrenal

![Figure 11-44](image-url)

**Figure 11-44.** Pheochromocytoma in a 38-year-old woman with flushing and episodes of extremely elevated blood pressure. **A,** A large high-signal-intensity (SI) mass of the right adrenal can be identified on the T2-weighted image. **B,** The lesion shows lower SI than normal liver tissue on T1. **C,** Inhomogeneous but almost complete enhancement of the lesion, indicating a solid tumor rather than a cystic lesion, can be noted on the T1-weighted fat-suppressed contrast-enhanced image.
Neuroblastoma in a 4-year-old boy with abdominal pain and a palpable abdominal mass. A and B, T2-weighted images show a large inhomogeneous mass of the left upper abdomen that displaces the kidney. The T2-weighted images also show areas of necrosis within the tumor. Comparison of pre- (C) and post-contrast (D) fat-suppressed T1-weighted images reveals peripheral enhancement with large parts of the lesion remaining hypointense. Typical for neuroblastoma is the extensive encasement of abdominal vessels which in this case reaches the contralateral renal hilum.
Ganglioneuroblastoma. This 5-year-old boy presented with a large palpable mass of the right upper abdomen. A and B, T2-weighted images show a large, well-defined mass displacing the right kidney. The coronal image (A) demonstrates the adrenal origin of the lesion. C, There is no sign of encasement of vessels of the upper abdomen in the T1-weighted fat-suppressed unenhanced image as typically found in neuroblastoma. The pancreatic head (arrow) is displaced to the left. D, The lesion shows inhomogeneous enhancement on the T1-weighted fat-suppressed contrast-enhanced image, which is more indicative of ganglioneuroblastoma rather than ganglioneuroma.

In contrast to hyperfunctioning masses of the adrenal glands, nonhyperfunctioning adenomas or so-called incidentalomas are common. In most cases, these lesions are detected in imaging studies of the upper abdomen performed for other reasons. In spin echo imaging, nonhyperfunctioning adrenal adenomas have a signal intensity similar to the liver on T1- and T2-weighted sequences, whereas adrenal carcinoma and metastases are typically hyperintense on T2-weighted images. The signal intensity can be variable, however, because of hemorrhage, necrosis, or calcification. The most promising method to characterize adrenal adenomas is chemical shift imaging, in which up to 80% of adenomas show an obvious signal intensity reduction (Fig. 11-47) in opposed-phase images as compared with in-phase images.

Metastases to the adrenal glands are rather common. Overall, the adrenals are the fourth most common involved organ in metastatic disease. Typical primary tumors are lung cancer, breast cancer, and thyroid and colon cancer as well as malignant melanoma. In MRI adrenal metastases are seen as rounded or oval masses that are hyperintense to the liver on T2-weighted images (Fig. 11-48). Chemical shift imaging is also very sensitive for the detection of adrenal metastases. However, this technique does not allow differentiation from benign nonhyperfunctioning adenomas because both types of lesions fail to show signal reduction on opposed-phase images.

A rare benign neoplasm of the adrenal gland that contains both fat and myeloid elements is adrenal myelolipoma. In MRI of adrenal myelolipoma, it is important to look for lipomatous tumor or a solid tumor that contains foci of fat that can readily be identified in.
FIGURE 11–47. Nonhyperfunctioning adrenal adenoma in a 35-year-old woman with incidental detection of an adrenal mass. Imaging studies show a round, sharply demarcated, homogeneous lesion of the left adrenal gland. It has a signal intensity comparable to the liver in T₂-weighted (A) and T₁-weighted in-phase (B) images (arrows). C, The signal of the lesion decreases significantly in opposed-phase imaging. The early (D) and delayed (E) enhancement after injection of an extracellular gadolinium chelate is homogeneous comparable to normal adrenal tissue.
FIGURE 11–48. Adrenal metastases in a 72-year-old man with bronchial adenocarcinoma. T$_2$-weighted (A) and T$_1$-weighted in-phase (B) images show a large, poorly marginated, inhomogeneous mass of the right adrenal gland (arrow). Some high-signal-intensity (SI) areas can be identified in the periphery of the lesion that represent adjacent retroperitoneal fat rather than a fat-containing lesion. C, No obvious decrease in SI is noted in the opposed-phase image. D, After contrast administration the T$_1$-weighted fat-suppressed image demonstrates inhomogeneous enhancement of the lesion with irregular margins.
fat-suppressed images (Fig. 11-49). If only small foci of fat mainly in the periphery of an adrenal lesion are found in a patient with known malignancy, the differential diagnosis includes metastases, and biopsy must be performed to prove the diagnosis.

Another rare entity of the adrenal glands are cysts; most are endothelial. Pseudocysts may also be found, which develop after hemorrhage of the adrenal gland. The imaging appearance of adrenal cysts on MRI depends on the content of the cyst; however, most adrenal cysts are hypointense on T1-weighted images and have a very high signal intensity on T2-weighted sequences. If hemorrhage is present, an increase of signal intensity in T1-weighted images and a decrease in T2-weighted images can be seen.

KIDNEY

Because of its wide distribution and low cost, ultrasoundography is generally the method of choice for screening renal masses. Nevertheless, both MRI and CT are superior to ultrasonography for morphologic assessment. In distinction to ultrasound, MRI and CT permit simultaneous assessment of macroscopic changes and evaluation of functional derangements. In the 1980s, CT was the method of choice for characterization of suspected malignant renal lesions primarily detected using ultrasonography. At that time, MRI of the kidneys did not play an important role. In the interim, studies have shown MRI to be helpful in the detection and staging of renal cancer. With the development of new pulse sequences, including fast spin echo sequences with motion compensation and gradient echo sequences as well as the use of dynamic imaging, the role of and indication for MRI in the work-up of renal lesions have changed. Dynamic and delayed postcontrast images are considered to be the most helpful for lesion identification and characterization (i.e., determination of the lesion extent). In comparison with CT, MRI has been shown to be statistically superior for correct characterization of benign renal lesions regarding infiltrative versus expansive masses, whereas there is only a slight difference between these two methods concerning detection and differential diagnosis of renal masses. MRI is also considered to be superior for differential diagnosis of lesions that are equivocal on CT, especially in the differentiation between complicated cysts and cystic or hypovascular renal cell carcinoma (RCC). MRI is considered superior to CT in the evaluation of perirenal and vascular involvement, as well as the involvement of perinephric fat and adjacent organs. For evaluation of the renal vein and the inferior vena cava (regarding tumor thrombus formation), MRI again has advantages over CT.

Thus, today MRI is the imaging technique of choice for preoperative and postoperative tumor staging. Indeed, small solid masses, less than 1 cm in diameter, may be visible using MRI but not be visible with ultrasonography or CT.

Renal cell carcinomas are the most frequent malignant neoplasms of the kidney. Histologically, these tumors belong to the parenchymal malignant tumors. Moreover, there are many other malignant and benign tumors that can affect the kidney. With an incidence of 2% to 3% of all neoplasms, solid tumors of the kidney are rather rare. Most of the lesions that arise in the kidney are benign mesenchymal cysts and almost all are asymptomatic, whereas approximately 90% of solid renal lesions are classified as malignant.

From a histopathologic perspective, renal tumors may be divided into four groups:

1. Parenchymal tumors
2. Mesenchymal tumors
3. Tumors of the renal pelvis
4. Secondary tumors

Parenchymal tumors include both benign and malignant tumors. Adenomas, nephroblastomatosis, and multiloculated or uniloculated cystic kidney diseases are examples of benign renal masses. Renal cysts may be divided into genetic cystic diseases, obstructive cystic disease, acquired cystic disease, and cysts associated with systemic diseases. Within these entities, the age at presentation, renal function impairment, localization, and appearance of the cysts vary considerably. Autosomal-recessive polycystic kidney disease (ARPKD), also called type I cystic kidney disease according to Potter, is the most common heritable cystic disease in infancy and childhood, appearing unilateral and bilateral as well as uniloculated or multiloculated. Its incidence is 1 in 20,000 births, and it accounts for about 5% of all end-stage renal disease in the United States. In ARPKD the liver often is affected by congenital hepatic fibrosis, which may be helpful in the differential diagnosis.

Acquired cystic disease is most often caused by uremia. Etiologically, it has to be differentiated from obstructive cystic diseases, including multicystic dysplasia and cystic dysplasia. In rare cases, renal cysts are diagnosed in systemic diseases such as tuberous sclerosis and von Hippel-Lindau disease.

In addition to strictly benign and malignant parenchymal masses, there are also borderline types such as oncocytoma. Oncocytomas in general are benign masses but should be considered to be borderline because they have potential for malignant transformation.

Both RCC as well as nephroblastoma (Wilms’ tumor) represent malignant parenchymal tumors. RCC is the most common malignant tumor of the human kidney, and it is important to note that this group of tumors is very heterogeneous concerning histopathology. Approximately 6% to 7% of RCCs are impossible to classify by histology, and special molecular genetic studies are required for proper characterization.

Angiomyolipoma, hemangioma, lipoma, reninoma, and capsuloma are examples of benign mesenchymal tumors affecting one or both kidneys, whereas renal sarcoma is a quite common malignant mesenchymal tumor.

Tumors of the renal pelvis are another group of entities that may affect the human kidney. Transitional cell carcinoma is the most frequent malignant genitourinary tumor in adults and the second most common tumor of the kidney. This mesenchymal tumor develops from transitional epithelium and generally affects the human
Figure 11-49. Adrenal myelolipoma in a 47-year-old man with adrenal enlargement. There is a large solid lesion of the right adrenal gland, which has areas of fat on T₂- (A) and T₁-weighted (B) images. The fat signal can be suppressed on the T₁-weighted fat-saturated (C) and opposed-phase images (D).
Continued. **E–G.** Large areas of the tumor show homogeneous enhancement on dynamic imaging; some small regions of internal hemorrhage or degenerative changes remain hypointense. **H.** The T₁-weighted contrast-enhanced coronal image clearly demonstrates the adrenal origin of the lesion. The presence of fat and solid tissue within an adrenal lesion makes the diagnosis of myelolipoma most likely.
However, transitional cell carcinoma also occurs in the renal pelvis. Because there may be secondary involvement of the kidney in transitional cell carcinoma of the bladder or ureter, these tumors are considered as secondary (as opposed to primary) kidney tumors.

Secondary renal lesions may be benign (e.g., in case of parasitic infection such as echinococcal disease or a granulomatous tumor). Secondary malignant neoplasms include lymphomas, leukemia, and metastases. Renal involvement in patients with known lymphoma ranges from 30% to 60%.

Finally, tumors exist that cannot definitely be distributed to a specific group such as the malignant rhabdoid tumors (typically found in children and carrying a bad prognosis). These tumors have in common a distinct so-called rhabdoid cytologic feature and are further characterized by variable histologic and immunohistochemical patterns.

Figure 11-50 summarizes the pathologic classification of renal masses. The problems concerning specific pathologic characterization of renal masses reflect the difficulties in radiographic examination and characterization.

The basic MRI approach for imaging of the kidneys includes T1-weighted spin echo sequences acquired in the transverse plane analogous to CT scanning. Additionally, T2-weighted sequences should be performed. For lesions located at the upper or lower pole of the kidney, images in the coronal plane may give important additional information.

Postcontrast scans should be acquired in a dynamic fashion (after intravenous bolus injection of a Gd chelate) during the various phases of contrast enhancement. It should be noted that some lesions are only clearly visible in the early arterial phase, whereas others are better depicted in images acquired approximately 1 to 2 minutes after contrast injection. In cases of suspected disease involving the vasculature, additional gradient echo images or contrast-enhanced magnetic resonance angiography (MRA) can be performed for further evaluation. To evaluate perirenal or pararenal extension of tumors, fat-suppressed T1-weighted images after contrast injection are useful because they provide much better contrast between fat and solid contrast-enhancing lesions.

In the normal kidney, there is good differentiation (precontrast) between the cortex and the medulla on T1-weighted images; the cortex shows a higher signal intensity. Fat in the renal sinus has high signal intensity both on T1- and T2-weighted images; the vessels of the hilum are seen as flow voids. On T2-weighted images, the boundary between the cortex and medulla is less clearly seen. However, on unenhanced images cysts are best detected with T2-weighted sequences. In dynamic imaging of the kidney after bolus injection of a Gd chelate, an increase of signal intensity in the renal cortex can be observed 5 to 10 seconds after the start of contrast injection. An increase of signal intensity in the medulla starts at approximately 20 to 30 seconds. In images between 2 and 3 minutes postinjection, a homogeneous increase of signal intensity in the kidney can be observed.

### Classification of Renal Masses in MRI

On MRI, renal masses are classified with regard to their general imaging appearance (i.e., solid or cystic masses) and growth patterns (i.e., expansile vs. an infiltrative growth) (Fig. 11-51). In addition to imaging findings, the patient’s age, sex, and medical history should always be considered in the differential diagnosis. The prevalence of malignant tumors in childhood differs markedly from those of adults; thus, tumors of the kidney in childhood are discussed separately.

![Pathologic classification of renal tumors.](Figure 11–50)
Simple cysts of the kidney are the most frequent renal masses in adults; most cases are detected incidentally. With simple cysts, MRI has a minor role in evaluation because these lesions can be clearly characterized by ultrasonography or CT. However, in those lesions that cannot be categorized confidently on CT or ultrasonography, MRI provides important additional information. Simple cysts in MRI have low signal intensity on T1-weighted images and homogeneous high signal intensity on T2-weighted scans. After contrast injection, no increase in signal intensity is observed. Fat-suppressed T1-weighted images after contrast have a very high sensitivity for detection of small cysts.

Complicated cysts account for approximately 5% of renal masses. Complicated cysts are characterized by irregular borders, septations, and internal hemorrhage, or they may contain a high concentration of protein or calcium in suspension as well as wall calcification. The main differential diagnosis in complicated cysts is necrotic neoplasm or cystic RCC. These lesions can be indistinguishable from complicated, non-neoplastic cysts. Although the signal intensity of complicated cysts in T1-weighted images is variable and determined by the fluid composition, the high signal intensity in T2-weighted images in most cases is maintained. However, some cysts with hemoglobin degradation products may show decreased signal intensity both on T1- and T2-weighted sequences. Sometimes layering can be demonstrated. On T1-weighted images, complicated cysts often present as hyperintense masses, and susceptibility artifacts with the application of fat suppression further increase the signal intensity of the cyst fluid. In polycystic kidney disease (Fig. 11-52), multiple cysts of variable signal intensity can be depicted representing cysts with hemorrhage and infection. However, a clear differentiation between infected and hemorrhagic or even malignant cysts cannot be made accurately on MRI, CT or ultrasonography.

A benign neoplastic lesion that also presents as a cystic tumor is multilocular cystic nephroma. This tumor is composed of multiple noncommunicating cysts with a fibrous stroma and a fibrous capsule. Typically, no solid areas can be detected. However, differential diagnosis versus cystic RCC (Fig. 11-53) in the majority of cases is not possible by means of imaging findings.

The most frequent renal mass of mesenchymal origin is angiomyolipoma. Pathologically, angiomyolipoma is a hamartomatous tumor composed of vessels, smooth muscle fibers, and lipocytes in different proportions. Angiomyolipoma may present spontaneously or as bilateral or multilocular tumors in combination with tuberous sclerosis. The MRI appearance of angiomyolipoma depends on the histologic composition of the tumor. The signal intensity may be dominated by fat, leading to a high signal intensity on both T1- and T2-weighted images. The fat content can be proven by applying fat suppression (Fig. 11-54). In those tumors in which smooth muscle predominates differentiation from malignant tumors (e.g., RCC) may not be possible by means of imaging studies.

**Figure 11–51.** Magnetic resonance imaging classification of renal masses.
FIGURE 11–52. Adult polycystic kidney disease in a 45-year-old man with a history of nephrectomy of the left kidney as a result of renal cell carcinoma. A–D, T2-weighted axial, sagittal, and coronal images show a diffuse cystic transformation of the right kidney, which displaces the liver and reaches down into the pelvis. The cysts have different signal intensities indicating simple and complicated renal cysts with hemorrhage of different age. Note the multiple hepatic cysts.
FIGURE 11–53. Cystic renal cell carcinoma in a 38-year-old man with hematuria. Sonography demonstrated a renal tumor. A, T2-weighted imaging shows a mainly cystic tumor of the left kidney (arrow) with internal septations. B, On the T1-weighted image the tumor displays low signal intensity. Fatty components and hemorrhage can thus be excluded. However, the septations show strong contrast uptake on the T1-weighted fat-suppressed contrast-enhanced image (C), and solid areas are visible. Differential diagnosis includes benign multilocular cystic nephroma; however, solid areas favor the final histologic diagnosis of a cystic renal cell carcinoma.
Figure 11-54. Angiomyolipoma in a 58-year-old woman with a renal tumor on routine sonography. A, The lower pole of the left kidney is mainly replaced by a hyperintense tumor (on T2) with thin, hypointense septations. B, The signal remains almost isointense to perirenal fat on T1. C, On the fat-suppressed T1-weighted image, the tumor displays low signal intensity and is sharply demarcated from the adjacent renal parenchyma by a thin capsule. D, After contrast administration, the majority of the tumor does not seem to show any contrast uptake on the T1-weighted contrast-enhanced image. However, the T1-weighted fat-suppressed contrast-enhanced image clearly demonstrates contrast enhancement of septations and small solid areas (E).
Another benign tumor that can be diagnosed by means of MRI is lipoma of the kidney, although this tumor is rather rare. Other benign neoplasms that show no typical characteristics on MRI are hemangioma, lymphangiomatosis, and juxtaglomerular tumors. In these tumors, biopsy has to be performed for definitive diagnosis.

As mentioned previously, oncocytoma historically was considered a benign tumor. However, since reports about malignant foci in oncocytomas, this tumor is now classified as a low-grade or borderline renal neoplasm. Typically, these tumors are well circumscribed and may show a central star-shaped scar composed of fibrous tissue. If a central scar exists, it is often depicted with low signal intensity on T1-weighted images. The tumor itself on T1- and T2-weighted images has a signal intensity similar to the renal parenchyma and often can only be identified because of mass effect. Differentiation between oncocytoma and RCC is difficult because central necrosis or hemorrhage may occur, and there are no reliable imaging findings that can clearly differentiate between these two entities. However, if oncocytoma is favored in the differential diagnosis, resection of the tumor should be performed by means of nephron-sparing surgery.

The most important malignant tumor of the kidney is RCC. About 50% of RCCs have cystic areas as a result of internal hemorrhage, necrosis, or even primary cystic growth (see Fig. 11-53). Improvements in imaging studies significantly increased the early detection of asymptomatic RCC; however, no specific morphologic finding exists that specifically allows the diagnosis of RCC. The signal intensity characteristics of RCCs are variable (Figs. 11-55 to 11-57). They may have areas of high and low signal intensity both on T1- and T2-weighted images. There are even reports about macroscopic foci of fat within RCC. Thus, the finding of fat in a solid renal tumor does not exclude RCC. Detection of RCC is improved with dynamic imaging after bolus injection of Gd chelates. However, there are no specific features on enhanced scans that reliably allow diagnosis or exclusion of RCC. In general, RCC has to be considered in all solid masses and even in cystic lesions showing small solid areas (Fig. 11-58).

An important role of imaging studies in RCC is the staging of tumor spread to establish prognosis and treatment planning. Hematogenous spread of RCC commonly involves lung, mediastinum, liver, brain, bones, and adrenal glands and even the contralateral kidney. Infiltration of renal veins and the inferior vena cava as well as tumor thrombus formation are common findings. Lymphatic spread involves the para-aortic lymph nodes. In imaging of para-aortic lymph nodes, it is important to note that the size of the lymph nodes does not necessarily correlate with metastatic spread of RCC. Reports exist of lymph nodes with a diameter of greater than 2 cm that histologically proved to be inflammatory changes.

For evaluation of tumor thrombus formation in the inferior vena cava, coronal imaging using gradient echo sequences as well as contrast-enhanced MRA have proven to be very useful. Another important issue in RCC with invasion of the renal vein is imaging of the right atrium to allow proper planning of surgery.

The second most common malignancy of the kidney in adulthood is transitional cell carcinoma. More than 40% of transitional cell carcinomas arise in the renal pelvis and have associated urothelial neoplasm in the ipsilateral ureter or bladder. Typically, these tumors show an infiltrative growth pattern that is clearly depicted by MRI. In patients with hydronephrosis caused by tumor growth, the tumor is typically depicted as a solid mass in the urine-filled renal pelvis.

Primary lymphoma of the kidney is very rare because of the lack of lymphoid tissue within the kidney. In most cases, the involvement of the kidney is secondary (metastatic). In MRI focal renal lymphoma is depicted as any other solid mass of the kidney. However, the lack of necrosis and the rather homogeneous signal intensity of the tumor on unenhanced and contrast-enhanced images favor the diagnosis of lymphoma. On unenhanced images, lymphoma typically has the same signal intensity as surrounding renal parenchyma. On contrast-enhanced studies in the early phase up to 1 to 2 minutes after contrast injection, lymphoma appears as a hypovascular mass with subsequent delayed homogeneous enhancement.

If multifocal renal tumors are detected, renal metastases have to be considered in the differential diagnosis. Metastases are the most common multifocal renal neoplasms and arise from primary tumors from the lung, colon, melanoma, breast, and others. Existence of a known extrarenal malignancy suggests the diagnosis. Imaging studies are not able to differentiate metastases from primary malignant tumors of the kidney (e.g., RCC).

**Renal Masses in Childhood**

The most common malignant neoplasm of the kidney in childhood is nephroblastoma, also called Wilms' tumor. These tumors are generally detected because of their huge mass at clinical presentation. T1-weighted images typically demonstrate a large, inhomogeneous hyperintense tumor that often is septated (Fig. 11-59). The main differential diagnosis is neuroblastoma, which, in contrast to nephroblastoma, is often combined with lymph node enlargement and early encasement of abdominal vessels. Metastases from lymphoma and leukemia may also affect the kidneys of children. This differential diagnosis is especially important because these two tumors account for about half of the cancer cases of children in the United States.

Nephroblastomatosis is a very rare tumor that generally does not appear in adults. Pathologically, these tumors represent nephrogenic blastema that usually regresses by the age of 4 months. If this regression does not occur, so-called nephroblastomatosis with the potential to develop into Wilms’ tumor may persist. T1-weighted images show hypointense, partially irregular lesions often located in both kidneys. On T2-weighted images, the tumors demonstrate high signal intensity comparable with the intensity of the renal parenchyma (Fig. 11-60). Most of these tumors have a distinct
Renal cell carcinoma in a 68-year-old man with recurrent episodes of hematuria. A. The coronal T2-weighted image shows a round tumor of the upper pole of the left kidney (arrow), which shows an inhomogeneous but almost isointense signal to the renal cortex. On T1-weighted (B) and T1-weighted fat-suppressed (C) images, the tumor again is isointense to the kidney with small streaks of low signal radiating to the center of the lesion. After contrast administration, there is an inhomogeneous contrast enhancement of the tumor demonstrated on T1-weighted contrast-enhanced (D) and T1-weighted fat-suppressed contrast-enhanced (E) images, clearly indicating a solid renal tumor.
Figure 11–56. Renal cell carcinoma in a 64-year-old woman with hematuria. She had a history of nephrectomy after left-sided renal cell carcinoma. A, On the T2-weighted axial image, a large hyperintense tumor (arrow) is demonstrated in the renal pelvis of the right kidney. The tumor is isointense to the renal parenchyma on the T1-weighted image (B) and shows inhomogeneous enhancement with internal septations on T1-weighted contrast-enhanced (C) and T1-weighted fat-suppressed contrast-enhanced (D) images. Note the marked low signal intensity of the liver on T1-weighted images resulting from iron overload after multiple blood transfusions.
FIGURE 11–57. Bilateral renal cell carcinoma in a 59-year-old man with macrohematuria. A and B, On T2-weighted images there are inhomogeneous, partially hyperintense renal tumors (arrows) in both kidneys. The large left-sided tumor invades the perirenal fat and the renal pelvis as shown on the T1-weighted unenhanced image (C). The tumors enhance strongly after contrast injection and display large areas of necrosis as well as ill-defined borders (D and E: T1-weighted fat-suppressed axial and coronal contrast-enhanced images).
FIGURE 11–58. Multiple cysts and renal cell carcinoma in a 45-year-old man with von Hippel-Lindau disease. A, The T₂-weighted image shows multiple cystic lesions of both kidneys; one lesion at the lower pole of the left kidney demonstrates internal septations. B, On the T₁-weighted unenhanced image the lesions are hypointense. C, The T₁-weighted fat-suppressed contrast-enhanced image demonstrates no contrast enhancement in the majority of lesions, consistent with simple cysts. However, there is inhomogeneous enhancement with demonstration of solid areas in the lesion at the lower pole of the left kidney (arrow), indicating a solid tumor that proved to be a renal cell carcinoma on surgery.
FIGURE 11–59. Bilateral nephroblastoma in a 2-year-old girl with abdominal discomfort and hematuria. A–C, Axial and coronal T₂-weighted images show large bilateral renal tumors with obstruction of the renal pelvis and hydronephrosis of the left kidney (arrow). The tumors have inhomogeneous signal intensity with a partially cystic appearance. Encasement of vessels of the upper abdomen is not present, in distinction from a neuroblastoma. D, On the T₁-weighted unenhanced image, the tumors are isointense to the renal medulla. E and F, The tumors are hypointense on T₁-weighted fat-suppressed contrast-enhanced images, and remaining renal parenchyma can be clearly identified by the strong contrast enhancement.
FIGURE 11–60. Nephroblastomatosis in a 2-year-old girl. Several renal masses were demonstrated on routine sonography. The T₂-weighted coronal image shows two high-signal-intensity tumors (arrows) of the left kidney with distinct borders. The tumors are confined to the kidney and have a round to oval shape.

border, and only a few are characterized by a slightly invasive growth. Because of its expansile character, nephroblastomatosis is often combined with a grossly dilated renal pelvis. The key to diagnosis is the more oval shape of nephroblastomatosis in contrast to the round appearance in nephroblastoma. Because of its morphologic characteristics, mature teratoma of the kidney is quite easy to classify. These tumors generally consist of solid and cystic parts. High signal intensity on T₁- and T₂-weighted images is typical for the fat contents of these tumors, which can be proven by fat suppression. Mature tumors show no infiltrative growth. The multicystic appearance of the tumor together with the finding of fat leads to the correct diagnosis. As with adults, it is important to emphasize that diffuse enlargement of both kidneys is highly suspicious for lymphoma (Fig. 11-61). Because of patchy diffuse hypointense infiltration of the kidney on T₁- and T₂-weighted images, it is often not possible to visualize the physiologic border between renal cortex and medulla. Contrast administration generally shows decreased contrast uptake of the tumor. Nevertheless, in the equilibrium phase, homogeneous enhancement may be seen.

In summary, MRI is the imaging modality of choice for the diagnosis of kidney tumors. Lesion morphology, both in regard to tissue consistency and lesion growth pattern, is clearly seen. Unfortunately, the macroscopic appearance of lesions does not correlate well with their pathologic classification. That is why correlation between MRI findings and pathologic classification is difficult and requires much experience. It is important also to recognize that some tumors may have many different morphologic appearances. It bears repeating that RCC accounts for almost 90% of all malignant kidney tumors.

FIGURE 11–61. Diffuse leukemic infiltration of the kidney in a 6-year-old boy with newly diagnosed acute lymphatic leukemia. T₁-weighted (A) and T₂-weighted (B) images show generalized enlargement of both kidneys with loss of the corticomedullary junction. C. After contrast injection, early T₁-weighted dynamic imaging depicts the leukemic infiltration as focal areas of hypointensity, whereas the residual parenchyma shows strong enhancement.
in adults and presents as a chameleon that may mimic many other benign and malignant conditions. It may appear uniloculated or unilateral/bilateral and multiloculated. It may have solid or cystic components or a combination. In regard to growth patterns, RCC can be infiltrative or expansile. Furthermore, areas of fatty degeneration may be found. Because of the high prevalence of RCC and its mimicking character, this diagnosis should always be considered in the differential diagnosis of kidney tumors.

**Imaging in Renal Transplantation**

In renal transplantation, the differentiation of acute tubular necrosis from rejection is critical. Basic knowledge of the appearance of a normal kidney is necessary to appreciate the changes in failing transplants. Healthy transplants should have the same signal characteristics as normal kidneys. On T₁-weighted images, the renal cortex should be of intermediate signal intensity, differentiating it from the triangle-shaped areas of lower intensity representing the medullary pyramids. In most normal kidneys, the corticomedullary junction is visible. The loss of corticomedullary differentiation on T₁-weighted images is the most consistent finding in acute or chronic renal transplant rejection, but the loss of corticomedullary differentiation is not specific for rejection. Relative or complete loss of corticomedullary differentiation may occur in hydronephrosis, glomerulonephritis, nonspecific renal failure, renal artery stenosis, pyelonephritis, and infiltrative tumor. Various degrees of loss of corticomedullary differentiation can also be seen in acute tubular necrosis. Cyclosporine toxicity has not been definitely associated with this finding. Perinephric fluid collections, such as postoperative urinomas, seromas, and lymphoceles, are typically depicted with a low intensity on T₁-weighted sequences and intermediate or high signal intensity on T₂-weighted images. This appearance is similar to other simple fluids. A perinephric abscess is typically heterogeneous and higher in intensity than other fluid collections on T₂-weighted images. Another common finding is gas formation within the abscess and secondary changes in the adjacent kidney, which are best demonstrated on Gd-enhanced images. Hematoma characteristics on MRI vary with age. Chronic hematomas have low signal intensity on T₁-weighted sequences and low to intermediate signal intensity on T₂-weighted images, sometimes similar to seromas, urinomas, and lymphoceles. Acute hematomas can be homogeneous in appearance and of intermediate to high signal intensity on T₁-weighted sequences and lower signal intensity on T₂-weighted images. In subacute hematomas, different components may be visible, with a central, low-intensity area surrounded by a high signal intensity rim on T₁-weighted images. Thus, in the early postoperative period, in most cases, MRI is able to differentiate hematomas from other fluid collections. Imaging after contrast administration allows evaluation of the perfusion of renal transplants and implementation of contrast-enhanced MRA makes possible the evaluation of transplant arteries.