Magnetic resonance cholangiopancreatography (MRCP) allows for the depiction of high SI static fluid in the biliary and pancreatic ducts, which has a long T2 time, against surrounding structures of low SI on images acquired with long echo times. Typically, a series of conventional T1 and T2WI are acquired in axial and coronal planes (allowing precise evaluation of surrounding structures) followed by thick slab (4-5 mm), oblique coronal acquisitions in various planes adapted to the course of the hepatobiliary or the pancreatic duct using a single-shot 2D acquisitions with long echo times. A 3D scan with isotropic spatial resolution—to allow for reconstruction in any desired plane—is then acquired, typically with navigator echoes under free respiration of the patient compensating for motion artifact. MIP images are then constructed. Recently introduced sparse imaging techniques can substantially reduce acquisition times in this application. CE-MRCP may also be performed utilizing agents with biliary excretion such as Gd-BOPTA or Gd-EOBDTPA, acquiring T1WI 3D-FLASH or VIBE sequences.

MRCP is a valuable imaging technique for planning or even negating the need for invasive ERCP (endoscopic retrograde cholangiopancreatography) using ionizing radiation. The normal gallbladder wall appears as a low SI thin line on T2WI and shows a homogeneous faint enhancement after contrast agent application. The SI of bile itself increases with fasting on T1WI due to increasing bile salt concentrations. Cholelithiasis is a common incidental finding on MR. A gallstone appears hypointense on T1 and T2WI due to a lack of mobile protons within its internal lattice. Stones with high protein concentration may appear hyperintense on T1WI. Multiple low SI gallstones are present on the thick-slab MRCP image of Fig. 68.1A. A gallstone may be distinguished from a polyp by its dependent location and lack of enhancement, thus additional imaging with the patient in the prone position may aid to differentiate these two entities. Both MRCP and axial T2WI Fig. 68.1A,B, respectively, also demonstrate a solitary common bile duct stone at the level of the ampulla (white arrow) with a faint but characteristic high SI meniscus present superiorly. Pneumobilia may mimic gallstones but would be dependent in location in the opposite direction than gravity, in distinction to gallstones. Acute cholecystitis is sensitively detected by wall-enhancement and enhancement of reactive adjacent hepatic parenchyma. Gallbladder wall thickening and pericholecystic fluid are well-detected on T2WI. A shrunken gallbladder with only mild, homogeneous enhancement of a thickened wall typifies chronic cholecystitis, while wall calcification—hypointense on T2WI—is associated with an increased risk of gallbladder carcinoma. Carcinoma appears as a heterogeneously enhancing gallbladder lumen mass often with associated wall thickening,
local invasion, and lymphadenopathy. In distinction, adenomyomatosis demonstrates high SI cystic spaces (which do not enhance post-contrast) within a thickened gall bladder wall on T2WI. Intrahepatic (peripheral) cholangiocarcinomas should not be confused with HCC as they are typically hypovascularized on dynamic CE T1WI showing centrally located delayed enhancement due to their fibrous composition and desmoplastic reaction.

The extrahepatic cholangiocarcinoma (white arrow) in Fig. 68.2A demonstrates a typically moderate to low SI appearance on GRE T1WI. The coronal T2WI in Fig. 68.2B localizes this lesion near the common bile duct (white arrows). (C) Thick-slab MRCP images, which suppress the mass’s SI due to the long TE, allow better biliary visualization, specifically of the shoulder sign, a finding characterized by abrupt narrowing of the otherwise dilated common bile duct at the level of tumor. Ductal wall thickening (> 5 mm) also suggests

*Runge, von Tengg-Kobligk, Heverhagen*
cholangiocarcinoma, with wall irregularity signifying infiltration. So-called Klatskin
tumors occur at the confluence of the hepatic ducts. Primary sclerosing cholangitis,
illustrated in Fig. 68.3A,B, is an important predisposing condition to cholangiocarcinoma.
(A) Thick-slab MRCP demonstrates multifocal areas of stricture and dilatation within the
intrahepatic biliary system. Fibrosis of peripheral ducts lends a pruned appearance to the
intrahepatic biliary tree. Characteristic periportal inflammation and edema appear as high
SI on T2WI (Fig. 68.3B), whereas thickened, enhancing ductal walls may be seen on CE
T1WI. Focal wedge-shaped regions of hepatic parenchymal hyperintensity may arise
secondary to edema and inflammation. Similar involvement exclusive to the intrahepatic
biliary system suggests primary biliary cirrhosis with hepatic cirrhosis mimicking both
diagnoses. Parenchymal and wall findings are similar with infectious cholangitis but biliary
dilatation is more diffuse and additional small abscesses adjacent to the peripheral bile
ducts are often present.
Biliary hamartomas are benign cystic lesions distinguished by their small size (< 1.5 cm)
and multiplicity. Thin, peripheral contrast enhancement reflecting nearby compressed
hepatic parenchyma may occur, an appearance potentially mimicking that of abscess or
metastatic disease. Unlike the latter, progressively central enhancement is not seen and
surrounding tissue involvement is sparse.
Biliary cystadenoma is a rather rare neoplasm of the biliary tract mostly located within or in
part within the liver. Yet, any site of occurrence along the bile ducts is possible. These
lesions typically exceed 10 cm in diameter and are found in middle-aged women presenting
with abdominal mass and pain. As malignant transformation may occur in up to 15% of
cases biliary cystadenoma is considered a pre-malignancy and usually surgically resection
is pursued. On MRI a solitary large multiloculated cystic mass with thin peripherally
located septa is seen with water isointense signal on T2WI and T1WI. However,
hemorrhage, bile, serous or mucinous content results in signal alteration on unenhanced
sequences. Solid nodules or more contrast avid septa may be indicators of malignant
transformation.
Choledochal cysts and cystic dilatation of the bile duct represent congenital malformation of the biliary tract usually diagnosed in the first decade of life and essentially not based on other acquired causes such as inflammation or biliary obstruction by stones or tumors. Size and localization of these lesions are further characterized by the Todani or Miyano classification. Caroli’s disease (Todani type V or Miyano Type F) represents a rare subtype of biliary tree anomaly showing communicating fusiform and saccular dilatation of the intrahepatic bile ducts. MRI and MRCP are important to depict the communication of the dilated duct segments with the main biliary tree, usually with the SI of normal bile. However, signal alteration may occur in case of sludge, debris or stone formation within affected duct segments.

![Fig. 68.3](image-url)