INTRODUCTION
Cholangitis refers to bile duct inflammation which, untreated, ultimately destroys cholangiocytes, causing scarring and/or ductopenia. Subsequently, the accumulation of bile constituents will damage the hepatocytes, leading to fibrosis, end-stage biliary cirrhosis, and finally, liver failure.1,2 Cholangitis is classified as acute (AC) vs chronic (CC), according to its aetiology, or site of bile flow impairment.1,4 CC mandates that symptoms last at least 6 months, otherwise, it is labeled AC.1

Cholangitis may affect the intrahepatic ducts (IHD) and extrahepatic ducts (EHD). However, intrahepatic cholestasis can also originate at the hepatocyte level through impaired canalicular bile secretion caused by genetic defects, drugs, or inflammatory processes.5–7 Like many other chronic liver diseases, primary biliary cholangitis (PBC) and primary sclerosing cholangitis (PSC) may be characterised by clinically quiescent phases and intermittent bouts of bile duct inflammation or infection, i.e. true bacterial “cholangitis”.

Extrahepatic duct (EHD) cholangitis, involving the common hepatic (CHD) or common bile ducts (CBDs), can be intrinsic, e.g. strictures in PSC, cholangiocarcinoma (CCA), and stones, or extrinsic, e.g. lymphadenopathy, masses, or cysts.8,9 Clinical, serologic, and imaging features help to differentiate the various types of cholangitides.10

The typical features of AC, including fever, colicky upper abdominal pain, and tenderness, are most often due to cholecdocholithiasis.11 These symptoms may also follow interventions, e.g. biliary-enteric Anastomotic stenosis, or indwelling biliary stent malfunction.12 Non-bacterial, e.g. immune-mediated or viral cholangitides, typically evolve more slowly.13 As cholangitis differs between
immunocompetent and immunocompromised patients, the host’s immune status may be inferred from imaging. Viruses (e.g. CMV, HIV), either by direct invasion, immune-mediated cell destruction, or associated bacterial or opportunistic infection, may cause cholangitis, particularly in immunocompromised patients (Table 1).14

Laboratory tests help determine the severity and, sometimes, even the etiology of cholangitis. Elevated serum alkaline phosphatase (ALP) and γ-glutamyl transpeptidase (GGT) are indicative of cholestatic disease, with bilirubin increasing only in the late stages of fibroproliferative cholangiopathies, such as PBC and PSC, or when complicated by dominant/relevant strictures (e.g. PSC).15 Importantly, ALP and GGT may take 24–48 h to increase significantly and may still be normal in acute biliary obstruction, while a hepatitis profile, e.g. titer (e.g. E.coli, Klebsiella, Enterococcus, Enterobacter) RPC, Parasitic infection Viral (HBV, HCV, HEV, HSV CMV, EBV) AIDS-Cholangiopathy PSC IgG4 holangitis AIDS cholangiopathy Intra-arterial chemotherapy DILI

Table 1. Aetiology: acute and chronic cholangitis

| Etiology                  | Acute cholangitis | Chronic cholangitis |
|---------------------------|-------------------|---------------------|
| Obstruction               | Biliary stones    | Biliary strictures  |
|                           |                   | Anastomotic strictures post-OLT |
|                           |                   | NAS post-OLT         |
|                           |                   | Neoplasms (benign or malignant) |
| Infectious cholangitiides | Bacterial (E.coli, Klebsiella, Enterococcus, Enterobacter) | RPC |
|                           |                   | Parasitic infection  |
|                           |                   | Viral (HBV, HCV, HEV, HSV CMV, EBV) |
|                           |                   | AIDS-Cholangiopathy  |
| Immunologic               |                   | PSC                  |
|                           |                   | IgG4 holangitis       |
|                           |                   | AIDS cholangiopathy  |
| Toxic                     | Drugs             | DILI                |
| Ischaemic cholangiopathy  |                   | Intra-arterial chemotherapy |
| Congenital                | Secondary sclerosing cholangitis | |

DILI, drug induced liver injury; OLT, orthotopic liver transplantation; PBC, primary biliary cholangitis; PSC, primary sclerosing cholangitis; RPC, recurrent pyogenic cholangitis.

Acute cholangitis
Acute bacterial cholangitis (AC) is the most common biliary tree affliction, mainly caused by calculi in middle-aged obese females.27 Less often, stricture(s) or extrinsic duct compression, or rarely, bile duct cysts or duodenal diverticula, cause gradual obstruction.28 Seldom does intraductal pressure exceed 25 mm H2O. However, if this threshold is crossed, cholangitis occurs due to bile reflux into veins and lymphatics.9 According to the Tokyo Guidelines, clinical presentation, laboratories, and imaging are necessary for a diagnosis of AC.29 Charcot’s triad is seen in 70%.13 However, diagnosing AC in underlying chronic cholestasis is challenging since symptoms may be atypical and criteria are not universal.30 On MRI, periportal tracking and high-signal-intensity (SI), wedge-shaped segmental cholangitis, due to lymphatic inflammation may be visible even in asymptomatic patients, preceding the increase in cholestatic labs (Figure 1). Furthermore, liver enzymes may remain at baseline even when E. coli and other gut bacteria are present, especially if limited to smaller liver segments.31 Haematogenous spread of bacteria to the liver is a critical pathogenic factor for cholangitic liver abscesses (Figure 2). However, as cholangitis spreads, systemic inflammatory response syndrome (SIRS) turns into sepsis, with or without multiorgan failure, increasing mortality risk to 50%.32 Urgent imaging to determine the site of the obstruction, should be followed by ERCP or percutaneous biliary drainage within 24 h.29,34 In patients with AC refractory to treatment,
| Disease entity                     | Main imaging features | Other diagnostic tests | Associated conditions | Treatment                                      |
|-----------------------------------|-----------------------|------------------------|-----------------------|------------------------------------------------|
| Acute Cholangitis                 | CBD stone, bile duct dilatation | Gallstones | ERCP or PTC drainage |
| Acute Suppurative Cholangitis     | Pus within bulging bile duct(s) | ERCP or PTC drainage |
| Oriental Cholangitis              | IHD stones without gallstones, Segmental liver atrophy | Possible eosinophilia | Clonorchis sinosis or Ascaris lumbricoides | Praziquantel or albendazole/ivermectin |
| Fasciola Hepatica                 | Tunnels and caves R liver lobe | Possible eosinophilia | | Trichlubendazole |
| Hepatic Schistosomiasis           | Cirrhosis             | Possible eosinophilia | | Praziquantel |
| Echinococcus Granulosus           | Hydatid cysts         | Possible eosinophilia | | Albendazole, PAIR (if no biliary tree contact) or surgery |
| AIDS Cholangiopathy               | PSC-like pattern      | Low CD4 counts, Cryptosporidium, CMV as additional causative factors | HAART-resistance | ERCP |
| Post-transplant Cholangiopathy    | Anastomotic biliary stricture | | ERCP |
|                                    | Multiple NAS, intraductal casts, intrahepatic biloma | | ERCP or re-transplantation |
| Secondary Sclerosing Cholangitis   | PSC-like pattern      | Iatrogenic (e.g., surgery, TACE), SSC-CIP (SSC- in critically ill patients) | Liver transplant | |
|                                    |                       |                        | Toxins | Discontinue exposure |
|                                    |                       |                        | Langerhans Cell Histiocytosis | Steroids |
|                                    |                       |                        | Mastocytosis | Steroids, Imatinib, Ilotinib or Dasatinib |
| PSC                               | IHD and/or EHD strictures | Elevated ALP, Possible atypical pANCA | IBD | No established medical therapy, UDCA frequently used, Liver transplant |
| PBC                               | No visible IHD or EHD dilatation on MRI, splenomegaly | Elevated ALP, Positive titers AMA, Positive titers ANA-PBC specific | Hashimoto’s Sjogren’s, Celiac disease | UDCA, Second line: obeticholic acid, fibrates |
| IgG4 Sclerosing Cholangitis        | Solitary IHD or EHD stricture | IgG4 levels > 4x normal | Autoimmune pancreatitis, Salivary gland and/or retroperitoneal inflammation | Steroids, azathioprine, Second line: Rituximab |
| DILI                              | PSC-like pattern or PBC-like pattern | Chemotherapy, Drugs, Anabolic steroids | | Discontinue drugs/steroids |
| Stauffer Syndrome                 | Non-specific           | Paraneoplastic syndrome | Treat underlying malignancy | Steroids, cyclosporine |
| Graft Versus Host Disease          | Non-specific           | Gastrointestinal symptoms | | |
| Intrahepatic Cholestasis of Pregnancy | Not usually imaged beyond ultrasound | | UDCA anti-pruritic drugs |

ALP, Alkaline phosphatase; CBD, common bile duct; DILI, drug-induced liver injury; EHD, Extrahepatic bile duct; ERCP, endoscopic retrograde cholangiopancreatography; HAART, highly active antiretroviral therapy; HBV, Hepatitis B virus; HCV, Hepatitis C virus; IBD, Inflammatory bowel disease; IHD, intrahepatic bile duct; NAS, Nonanastomotic strictures; PBC, primary biliary cholangitis; PSC, primary sclerosing cholangitis; PTC, Percutaneous transhepatic cholangiography; TACE, Transcatheter arterial chemoembolisation; UDCA, Ursodeoxycholic acid.
contrast-enhanced CT (CE-CT) or MRI (CE-MRI) should be done to evaluate the liver parenchyma for abscess(es).\(^{35}\) MRCP is ideal for complete localisation and characterisation of duct pathology.\(^{19,36}\)

Normal IHDs are small in calibre and only faintly seen on CE-CT or CE-T1-weighted MRI, but can be seen more clearly on T2-weighted imaging and hepatobiliary CE-MRC.\(^{23}\) Using a hepatobiliary contrast agent, we can visualise the biliary tree and its patency and function (Figure 1). In 85% of acute cholangitis, the CBD appears smooth with symmetric wall thickening, and infrequently, with IHD wall enhancement as well. Liver parenchymal enhancement occurs in the arterial-phase only in approximately 60%, delayed-phase only in nearly 15%, and both phases in one-third. On T2-weighted images, approximately 70% have parenchymal wedge-shaped or periductal high SI\(^{37}\) (Figure 1). Diffusion-weighted imaging (DWI) is even more sensitive to cholangitis,\(^{38}\) and can distinguish it from perfusion defects, such as transient hepatic intensity defects (THID).\(^{39}\) Although both show arterial-phase enhancement, only cholangitis has a DWI correlate (Figure 1). Furthermore, cholangitis appears hypointense on the 20 min hepatobiliary-phase (HBP) of gadoxetic acid-enhanced MRI due to oedema and fibrosis in acute and chronic cholangitis, respectively\(^{40}\) (Figure 1).

Portal vein thrombosis and/or hepatic abscess(es) are complications in bacterial cholangitis.\(^{41,42}\) Pus within the bile duct(s) \(i.e.\) low SI on heavily T2-weighted images and moderate SI on fat-suppressed T1-weighted images, or a bulging, enhancing bile duct papilla that exceeds 1 cm (specificity 86%) is diagnostic of acute suppurative cholangitis, which complicates AC in approximately 60% and warrants immediate drainage.\(^{43}\)

**Chronic cholangitis**

**Infectious**

Recurrent pyogenic cholangitis (RPC), previously known as Oriental cholangiohepatitis, is typically seen in patients who reside in or who have immigrated from Southeast Asia. It is characterised by IHD and EHD strictures and dilatation with pigment stone formation, usually due to parasites, \(e.g.\) Clonorchis sinensis or Ascaris lumbricoides.\(^{44}\) Delayed diagnosis leads to chronic bile stasis with subsequent hepatolithiasis, segmental liver atrophy, and cholangiocarcinoma (CCA). The most helpful imaging findings are the presence of large IHD stones in the absence of gallbladder calculi, which occur in 80%, sparing of the EHDs, and high-protein content calculi that appear bright on T1- and dark on T2-weighted sequences.\(^{37}\) Either due to bowel gas reflux or superinfection with gas-forming bacteria from the gut (\(e.g.\) Escherichia coli), pneumobilia is also common.

Other common hepatobiliary parasites endemic to Asia, Egypt, and Africa and, infrequently, Europe, include Schistosomiasis. Echinococcosis and, \(Fasciola hepatica\) are universal. On imaging, parasites cause filling defects within the bile ducts, and sometimes blockage of the ducts leading to upstream dilatation.\(^{46}\)


**Figure 3.** 57-yr-old female with *Fasciola hepatica* infection of the liver. (A) Axial DWI shows a subcapsular tubular area of hyperintense signal in segment 6 (arrow). (B) Axial HASTE with fat sat shows no visible correlate (arrow). (C) and (D) axial and (E) coronal portal venous phase images post-gadoxetic acid injection show the extent of the subcapsular lesion, which appears as “tunnels and caves” (arrows). (F) Axial image, 20 min post-gadoxetic acid injection, shows lack of lesion enhancement (arrow). DWI, diffusion-weighted imaging.

**Figure 4.** 34-yr-old female with a hepatic hydatid infection. (A) Axial T2-weighted HASTE fat sat show a 6.2 cm cystic lesion with floating, undulating membranes within it due to detached endocysts (daughter cysts) in segment 7 (arrow). (B) Coronal HASTE and (C) Axial DWI (b 300 s/mm²) again show the “water-lily sign” of this Echinococcal cyst (arrow). (D) Axial and (E) coronal T1-weighted GRE, 20 min post-gadoxetic acid injection (HBP), show no enhancement within the cyst. (F) Axial and (G) coronal images obtained three hours after injection show enhancement of the lesion (arrow), indicating its communication with the biliary tree. Caveat: Delayed images, i.e. 2–3 h after gadoxetic acid injection to determine whether the patient was a candidate for PAIR must be obtained. If no connection to the bile ducts is found, then PAIR can be performed. DWI, diffusion-weighted imaging; HBP, hepatobiliary-phase; PAIR, puncture-aspiration-injection-reaspiration.

*Fasciola hepatica* has a predilection for the right subphrenic space where it can form low-attenuation liver lesions, called “tunnels and caves,” sometimes complicated by hemorrhage and capsular retraction (Figure 3). *Hepatic schistosomiasis* has non-specific imaging findings of cirrhosis.45

In *Echinococcus granulosus*, the liver, followed by the lungs, are the most commonly involved organs. Once suspected, MRCP, and T2-weighted MR sequences can give the extent of biliary tree involvement. Like CT, MRI can determine the content and grade of the cysts. A hypointense peripheral rim surrounding a hydatid cyst on T2-weighted images is thought to be due to fibrosis or calcification. However, wall calcification is better seen on CT. Percutaneous drainage has replaced surgical decompression in many instances of parasitic disease.46 Morbidity, mortality, and recurrence have been shown to be greater the more radical the surgery. Cyst diameters > 7.5 cm are associated with a high risk of bile duct communication.47 Gadoxetic acid-enhanced MRC in the delayed phase, i.e. 2–3 h after injection, is recommended to exclude communication between the cyst and the biliary system before percutaneous ultrasound or CT-guided PAIR (puncture-aspiration-injection-reatpiration) is performed (Figure 4).

**In immunocompromised patients**

Immunocompromised patients prone to opportunistic infections often develop biliary stricture(s) that lead to cholestatic liver impairment. AIDS cholangiopathy occurs primarily in those with very low CD4 counts, drug-resistant HIV or lack of access to antiretroviral therapy.48 *Cryptosporidium parvum*, and CMV, the most common inciting pathogens, may cause peri-ampullary stenosis (isolated finding in 10%), “beading” of the IHD resembling sclerosing cholangitis, or segmental EHD strictures with/ out IHD involvement, thought to be secondary to ischaemia.48

Liver transplant recipients are also prone to cholangitis, typically from biliary infection, chronic rejection, ischaemia, drugs, or anastomotic stricture.49 Because the biliary tree cholangiocytes (epithelial cells of the bile duct) are supplied by the peribiliary plexus, which comes from the hepatic artery, it is prone to hypoxia compared to the hepatocyte, which receives a dual blood supply, i.e. the hepatic artery and the portal vein.50 Anastomotic or non-anastomotic strictures are best detected by MRCP, which has a sensitivity and specificity of 95%. Non-anastomotic stenoses (NAS) are characterised by intraductal casts and/or intrahepatic biloma formation, requires retransplantation in over 10%51 (Figure 5). The presence of bile duct-wall necrosis, with spillage of secretions into the liver parenchyma, portends a poor prognosis. On the contrary, if an isolated anastomotic stricture is suspected, ERCP is done so that balloon dilatation and/or stenting can follow, if necessary.

**Immune-mediated**

Autoimmune diseases, plus some drugs and toxins, have been implicated in the development of chronic cholangitis.5 Because these patients have no or mild symptoms early on, i.e. fatigue and pruritus, cholangiocyte and/or hepatocyte damage may already be advanced when they present with elevated cholestatic liver enzymes.52 What unifies the group of sclerosing cholangitides is their imaging appearance, which resembles that of PSC.
but an unknown cause, we will start with SSC. The imaging findings of sclerosing cholangitis, because PSC is a diagnosis of exclusion, comprising primary (PSC) and secondary sclerosing cholangitis (SSC). Because PSC is a diagnosis of exclusion, comprising those patients with imaging findings of sclerosing cholangitis, but an unknown cause, we will start with SSC. The imaging features of SSC are identical to those of PSC, including multiple short-segment strictures with intervening normal-calibre or slightly dilated segments, which results in a beaded appearance (Figure 6).

Secondary sclerosing cholangitis (SSC)
The most frequent chronic cholestatic disease is sclerosing cholangitis (SC), due to bile duct inflammation, obliterative fibrosis, and stricture formation that can end in liver cirrhosis. SC can be classified as primary (PSC) and secondary sclerosing cholangitis (SSC). Because PSC is a diagnosis of exclusion, comprising those patients with imaging findings of sclerosing cholangitis, but an unknown cause, we will start with SSC. The imaging features of SSC are identical to those of PSC, including multiple short-segment strictures with intervening normal-calibre or slightly dilated segments, which results in a beaded appearance (Figure 6).

Causes linked to SC include infection, immune-mediated injury (suggested by elevated IgG4 serum levels and/or lymphocytic infiltrates at histology), ischaemia, toxins, recurrent pancreatitis, Langerhans cell histiocytosis, and mastocytosis, as well as untreated mechanical biliary obstruction that progresses over time. However, iatrogenic causes are among the most frequently reported, including surgical trauma or ischaemic injury after orthotopic liver transplantation (OLT). Recently, a largely unrecognised new form of SSC, also believed to be due to ischaemia and/or systemic inflammation, has been observed in intensive care unit patients. Referred to as SSC-CIP (critically ill patients), they typically have sepsis, shock, trauma, and burns. The hallmark of SSC-CIP is the early formation of biliary casts. Sometimes the diagnosis can be made by portable ultrasound, especially if patients are unstable. Biliary casts may be overlooked early but as the mixture of inspissated bile and sloughed biliary mucosa hardens, it can appear as linear highly echogenic debris within alternating dilated and strictured bile ducts. Diagnosis often requires ERCP which may also allow attempts to remove biliary casts and debris. Rapid progression to liver cirrhosis, with a median survival of 13 months without OLT, is their typical fate. Recently, post-COVID-19 cholangiopathy, with clinical and histologic features similar to SSC, was reported in three patients. It is thought to be due to direct viral cholangiocyte injury, with possible overlapping pathogenetic hypoxia and cytokine storm. Therapeutic options for most forms of SSC are limited. Patients who do not undergo OLT have significantly reduced survival compared to PSC patients.

Primary sclerosing cholangitis (PSC)
Primary sclerosing cholangitis (PSC) is a rare, chronic, progressive immune-mediated inflammatory disease predominantly in middle-aged males, of whom over 70% also have inflammatory bowel disease (IBD). Usually asymptomatic, the diagnosis is made by elevated alkaline phosphatase (26 months), screening MRCP for stricture detection, and the absence of any...
Figure 7. 42-yr-old male with chronic inflammatory bowel disease and advanced PSC. (A) Axial DWI \( (b = 300 \text{ s/mm}^2) \) shows a wedge-shaped peripheral area of increased SI in segment 6 (arrow) of the right liver lobe. (B) Axial T1 GRE, arterial phase post-gadoxetic acid injection, shows wedge-shaped enhancement in segment 6 (arrow). (C) Axial and D) coronal T1 GRE, 20 min post-gadoxetic acid injection (HBP), show absent enhancement in the wedge-shaped area of cholangitis (arrow) indicative of early PSC. Strong uniform liver enhancement and timely contrast excretion indicate the preservation of liver function. DWI, diffusion-weighted imaging; HBP, hepatobiliary-phase; PSC, primary sclerosing cholangitis; SI, signal intensity.

Figure 8. 26-yr-old male with chronic inflammatory bowel disease and advanced PSC. (A) Coronal MRCP T2-weighted MIP shows multiple segmental strictures alternating with dilated segments of the more central biliary tree, the so called “beads-on-a-string” appearance, and the absence of bile ducts peripherally; in other words, an incipient form of a “pruned tree.” (B) Axial HASTE fat sat shows dilatation of predominantly the central intrahepatic bile ducts. (C) Axial DWI \( (b = 300 \text{ s/mm}^2) \) shows biliary dilatation and faint areas of segmental cholangitis (arrow). (D) Axial and E) coronal T1 GRE, 20 min post-gadoxetic acid injection (HBP), show uniformly decreased enhancement of the liver (thin arrow) relative to the right kidney (asterisk) and mild enhancement, i.e., contrast retention, in the portal vein (thick arrow) indicating absent hepatobiliary excretion (i.e., FLIS = 0). A few months later, the patient received a liver transplant. HBP, hepatobiliary-phase; MIP, maximum intensity projection; MRCP, magnetic resonance cholangiopancreatography; PSC, primary sclerosing cholangitis.

Primary biliary cholangitis (PBC)

Initially called primary biliary cirrhosis (PBC), the name was recently changed to group it among the cholangitides, and to raise awareness that cirrhosis can be prevented by treatment, particularly during earlier stages. PBC predominantly affects females over 40 years of age. Of autoimmune origin, it usually occurs with conditions such as Hashimoto’s thyroiditis, Sjogren’s disease, or celiac disease. Although the antimicrobial antibody (AMA) test is not specific for PBC, over 90% of PBC patients will be AMA-positive and another 30% positive for the PBC-unique antinuclear antibodies (ANA, directed against gp210 or sp100, usually allowing a diagnosis without liver biopsy). On ultrasound or cross-sectional imaging, there are no characteristic findings in early PBC, other than occasional porta hepatitis or gastroduodenal ligament lymphadenopathy. Although the main role of MRI is to exclude other causes of IHD or EHD risk factors for SSC. Although 90% of patients have classic PSC, involving the entire biliary tree, 5% have exclusively small-duct pathology, i.e. involving the peripheral IHD, which has no imaging correlate, or overlap syndrome, which describes concurrent autoimmune hepatitis (AIH) and PSC. Only in these two instances a liver biopsy is necessary for diagnosis. Periductal onion-skin-like fibrosis on histology confirms the diagnosis, though this finding is seen in only 10–20%, likely due to the performance of blind biopsy within unevenly distributed disease. Gadoxetic acid-enhanced MRI and DWI can detect early or subtle PSC, potentially guiding the biopsy (Figure 7).

Due to the lack of established drug therapy for PSC, OLT is the only curative treatment, although many centers use Ursodeoxycholic acid (UDCA) despite an unproven survival benefit in PSC. However, annual CA 19–9 and MRCP are recommended for CCA, and ultrasound for gallbladder cancer surveillance. Furthermore, in PSC patients with IBD, the risk of colon cancer increases fourfold over patients with solely IBD which justifies (bi)annual surveillance colonoscopy. Serial LFTs determine the timing of MRCP and MRI to screen for the development of a dominant stricture (DS), the severity of which serves as a prognosticator. Functional imaging with gadoxetic acid-enhanced MRI is the non-invasive method of choice. The appearance of gadoxetic acid within the expected 20 min of injection, i.e. the HB phase image, indicates that the stricture under evaluation is of no functional significance. When excretion exceeds 20 min, we consider the possibility of a DS, warranting dilatation with ERCP. Caveat: If morphologic features of advanced cirrhosis are present, delayed excretion could be due to chronically impaired function. Endoscopic biopsies (to exclude CCA) and dilatation with and without short-term stenting of such DS is essential to PSC management. In addition, parenchymal progression to liver cirrhosis can be followed on serial MRIs, both dynamic and DWI. In advanced PSC, gadoxetic acid-enhanced MRI aids pre-OLT evaluation and post-OLT follow-up, including graft injury and disease recurrence (Figure 8). Recently, Bastati et al introduced the so-called FLIS score as a semiquantitative method with which to predict survival in chronic liver disease and liver transplant patients.

Primary sclerosing cholangitis.
Figure 9. 34-yr-old male with advanced primary biliary cholangitis. (A) and (B) Axial DWI (b = 300 s/mm²) show perportal tracking (arrow), (A) and increased lymph nodes in the hepatoduodenal ligament (arrow), (B). (C) Axial T1 GRE, arterial phase post-gadoxetic acid injection, shows no significant abnormalities. (D) Axial MR-Elastography map shows significantly increased liver stiffness values, approximately 3.5 kPa compatible with Stage 3 fibrosis. (E) Axial and (F) and (G) coronal T1 GRE, 20 min post-gadoxetic acid injection (HBP), show inhomogeneous uptake with multiple high SI intraparenchymal nodules compatible with RNH. Prompt hepatobiliary excretion (arrow) indicates preserved liver function. Increased longitudinal diameter of the spleen is due to splenomegaly (asterisk). HBP, hepatobiliary-phase; RNH, regenerative nodular hyperplasia.

Figure 10. 22-yr-old male presented with acute abdominal pain and clinical suspicion of pancreatitis. He had elevated serum enzymes in the course of IgG4-related sclerosing cholangitis. (A) Axial DWI (b = 300 s/mm²) shows multiple, wedge-shaped, peripheral areas of increased SI in both liver lobes (arrows) consistent with segmental cholangitis. (B) Axial and (C) coronal T1 GRE, 20 min post-gadoxetic acid injection (HBP), show poor enhancement of the areas corresponding to high SI on DWI (arrows), which confirmed regional liver impairment due to segmental cholangitis. Timely excretion (thick arrow) indicates that function was preserved. (D) Axial DWI (b = 300 s/mm²) shows a diffusely thickened high-signal-intensity area, corresponding to the oedematous pancreatic head (arrow) due to AIP in the spectrum of IgG4 disease. AIP, autoimmune pancreatitis; DWI, diffusion-weighted imaging; HBP, hepatobiliary-phase; SI, signal intensity.

cholangitis, lymphadenopathy and periportal tracking, defined as linear high-SI on T2-weighted images that parallel the bile duct walls, are frequent findings. Histology (when obtained) confirms that this “tracking” corresponds to active inflammation in the portal tracts, meaning that its presence can be used to assess disease activity. The fact that periportal tracking is absent in late-stage disease supports that it is a marker of acute inflammation. Additional imaging findings include signs of portal hypertension, which may also be seen in pre-cirrhotic stages, possibly caused by a pre-sinusoidal block at the level of the portal tract, granuloma formation, ductal proliferation, and pronounced portal fibrosis. Interestingly, splenomegaly is much greater in the early stages of PBC than in end-stage disease. Although lymphadenopathy has been seen in 80% of PBC patients on CT, neither it, periportal tracking, nor splenomegaly are unique to PBC. The diagnosis of PBC relies on elevated cholestatic liver enzymes and the presence of AMA and/or PBC-specific ANA. Diagnostic liver biopsy is necessary when these two antibodies are absent.

UDCA is the mainstay of therapy in these patients since disease is primarily limited to the small IHDs. Biochemical response to UDCA after 12 months, and various scores (e.g. GLOBE, UK-PBC score) are important prognostic factors that guide therapy with initiation of second line therapy (obeticholic acid, fribates) in incomplete responders to UDCA. The liver volume (LV) to splenic volume (SV) ratio is an important prognosticator in PBC. A low LV/SV ratio is associated with a significantly poorer outcome in PBC patients. Moreover, the LV/SV ratio was found to be significantly lower in PBC patients who developed symptoms than in those who remained asymptomatic. Furthermore, spleen and liver stiffness measurements, obtained by transient elastography, are excellent markers for advanced disease, i.e. advanced fibrosis or cirrhosis (Figure 9).

Again, OLT is the only curative treatment for end-stage PBC. However, on average, 20% of these patients will have clinical manifestations of recurrent PBC, although the rate of histological recurrence is likely higher. As AMA titers remain positive post-OLT, liver histology is necessary to diagnose recurrent PBC. In contrast to PSC, it is rather uncommon for recurrent PBC to lead to graft failure since continuing UDCA therapy significantly reduces the risk of recurrence.

As in PSC, MRI plays an important role in following biliary cirrhosis, excluding acute mechanical obstruction should LFTs suddenly rise. Although overlap syndrome, i.e. simultaneous AIH and PBC, could explain such laboratory results, PBC patients are subject to cholecolithiasis as in other middle-aged females. Furthermore, HCC must be considered, especially in males. Surveillance is part of PBC management.

IgG4-related sclerosing cholangitis
IgG4-related sclerosing cholangitis is one manifestation of this systemic immune-mediated inflammatory entity that can affect multiple organs over time. Although serology may show elevated titers, this is not sufficient for diagnosis since elevated serum IgG4 levels can also occur with malignancies, such as...
Figure 11. 75-year-old psoriatic female with drug-induced cholestasis after Ixekizumab. (A) Axial DWI ($b = 300$ s/mm$^2$) shows mildly increased SI diffusely in the liver due to oedema and periporal tracking (arrow). (B) Axial T1 GRE, arterial phase post-gadoxetic acid injection, is unremarkable. (C) Axial MR-Elastography map shows significantly increased liver stiffness values, approximately 4.5 kPa, indicative of diffuse oedema due to DILI. (D), (E) Axial and F) coronal T1 GRE, 20 min post-gadoxetic acid injection (HBP), show markedly reduced contrast media uptake (thin arrow), (E) relative to the right kidney (asterisk), (E) and absent hepatobiliary excretion (thin arrow), (F) indicating very poor liver function. The patient recovered weeks after drug cessation, indicating that DILI was transient and reversible. DILI, drug-induced liver injury; DWI, diffusion-weighted imaging; HBP, hepatobiliary-phase.

Chemotherapy-induced sclerosing cholangitis (CISC) is a frequent, potentially fatal complication of hepatic arterial infusion chemotherapy, occurring in up to 50% of patients being treated for hepatobiliary malignancies. The resulting biliary stricture may result in progressive cholangitis, unless recognised and treated early.

In athletes and body builders, anabolic steroid use should be considered if an otherwise healthy adult presents with unexplained cholestasis. The spectrum of imaging findings is non-specific, ranging from chronic hepatitis to vascular injury to bile duct damage. Although, anabolic-induced cholestasis starts with pure hepatocanalicular cholestasis, severe ductopenia, known as vanishing duct syndrome, may be seen on histology in chronic cases. As this is not specific for anabolic steroids, the diagnosis similar to that seen in PBC, and eosinophils may suggest a drug aetiology. A few drugs, such as the fluorodeoxyuridines and 5-fluorouracil, can cause large-duct injury (e.g. the common hepatic or perihilar intrahepatic bile ducts) with features resembling PSC. Therefore, diagnosis primarily rests on a careful medication history and histologic findings.

MR is the most sensitive imaging modality for DILI, particularly when DWI and gadoxetic acid-enhanced T1-weighted sequences are obtained. There may be evidence of periporal tracking, reduced uptake, and absent excretion of gadoxetic acid in the hepatobiliary phase as small ducts disappear, i.e. vanishing duct syndrome (Figure 11). CT and ultrasound show non-specific findings of liver injury, including hepatomegaly and heterogeneously enhancing parenchyma. When injury is severe, fibrosis and cirrhosis can be seen.

In athletes and body builders, anabolic steroid use should be considered if an otherwise healthy adult presents with unexplained cholestasis. The spectrum of imaging findings is non-specific, ranging from chronic hepatitis to vascular injury to bile duct damage. Although, anabolic-induced cholestasis starts with pure hepatocanalicular cholestasis, severe ductopenia, known as vanishing duct syndrome, may be seen on histology in chronic cases. As this is not specific for anabolic steroids, the diagnosis similar to that seen in PBC, and eosinophils may suggest a drug aetiology. A few drugs, such as the fluorodeoxyuridines and 5-fluorouracil, can cause large-duct injury (e.g. the common hepatic or perihilar intrahepatic bile ducts) with features resembling PSC. Therefore, diagnosis primarily rests on a careful medication history and histologic findings.

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Chemotherapy-induced sclerosing cholangitis (CISC) is a frequent, potentially fatal complication of hepatic arterial infusion chemotherapy, occurring in up to 50% of patients being treated for hepatobiliary malignancies. The resulting biliary stricture may result in progressive cholangitis, unless recognised and treated early.

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is presumptive, based on improvement following cessation of steroids. However, if severe enough, the patient can present in acute liver failure. 95

Transient intrahepatic cholestasis of pregnancy (ICP), in 0.2% of females, usually occurs during the second and third trimesters. Typically treated with antipruritics, these patients almost never come to imaging. We present a case where ICP was incidentally noted in a patient who underwent MRI for unexplained hyperemesis gravidarum in week 24 of pregnancy and elevated LFTs to exclude mechanical obstruction. The final diagnosis was anti-phospholipid syndrome (Figure 12).

Stauffer syndrome should be kept in mind in the cancer patient with unexplained cholestasis. The underlying malignancy is the clue to the imaging diagnosis. Interleukin six causes cholestasis with unexplained cholestasis. The underlying malignancy is the phospholipid syndrome (Figure 12).

Graft versus host disease (GVHD) may cause hepatocyte and/or cholangiocyte necrosis and cell death in allogenic more often than autologous stem cell transplant recipients. Since imaging features and histologic inflammatory infiltrates are nonspecific, the clue to the diagnosis is the presence of concurrent skin and intestinal findings, as well as the timing of elevated serum LFTs. GVHD typically starts within weeks of transplantation, even sooner without immunosuppression. 96

SUMMARY

Imaging plays a crucial role in the diagnosis of acute and chronic cholangitis. Ultrasound is usually the initial investigation. CT is helpful in the evaluation of trauma, oncologic, or post-operative complications. Conventional T2-weighted MRCP is the most helpful diagnostic modality, despite some shortcomings. DWI and gadoxetic acid-enhanced MRC can overcome these limitations, further detecting liver parenchymal damage, which is an indicator of early biliary disease, and predicting survival.

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