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Reporting standards for primary sclerosing cholangitis using MRI and MR cholangiopancreatography: guidelines from MR Working Group of the International Primary Sclerosing Cholangitis Study Group

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Abstract

Primary sclerosing cholangitis (PSC) is a chronic inflammatory disorder affecting the bile ducts and is characterized by biliary strictures, progressive liver parenchymal fibrosis, and an increased risk of hepatobiliary malignancies primarily cholangiocarcinoma (CCA). PSC may lead to portal hypertension, liver decompensation, and liver failure with the need for liver transplantation. Magnetic resonance imaging (MRI)/magnetic resonance cholangiopancreatography (MRCP) are considered the imaging standard for diagnosis and follow-up in patients with PSC. Currently, there are no universally accepted reporting standards and definitions for MRI/MRCP features. Controversies exist about the definition of a high-grade stricture and there is no widely agreed approach to their management. The members of the MRI working group of the International Primary Sclerosing Cholangitis Study Group (IPSCSG) sought to define terminologies and reporting standards for describing MRI/MRCP features that would be applied to diagnosis and surveillance of disease progression, and potentially for evaluating treatment response in clinical trials. In this extensive review, the technique of MRI/MRCP and assessment of image quality for the evaluation of PSC is briefly described. The definitions and terminologies for severity and length of strictures, duct wall thickening and hyperenhancement, and liver parenchyma signal intensity changes are outlined. As CCA is an important complication of PSC, standardized reporting criteria for CCA developing in PSC are summarized. Finally, the guidelines for reporting important changes in follow-up MRI/MRCP studies are provided.

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Key Points

- *Primary sclerosing cholangitis is a chronic inflammatory disorder affecting the bile ducts, causing biliary strictures and liver fibrosis and an increased risk of cholangiocarcinoma.*
- *This consensus document provides definitions and suggested reporting standards for MRI and MRCP features of primary sclerosing cholangitis, which will allow for a standardized approach to diagnosis, assessment of disease severity, follow-up, and detection of complications.*
- *Standardized definitions and reporting of MRI/MRCP features of PSC will facilitate comparison between studies, promote longitudinal assessment during management, reduce inter-reader variability, and enhance the quality of care and communication between health care providers.*

Keywords Sclerosing cholangitis · Biliary strictures · Cholangiocarcinoma · Definition · Terminology

Abbreviations

CCA	Cholangiocarcinoma
DS	Dominant stricture
EHD	Extrahepatic ducts
ERCP	Endoscopic retrograde cholangiopancreatography
IBD	Inflammatory bowel disease
IHD	Intrahepatic ducts
IPSCSG	International Primary Sclerosing Cholangitis Study Group
LD-PSC	Large duct primary sclerosing cholangitis
MRCP	Magnetic resonance cholangiopancreatography
MRI	Magnetic resonance imaging
PSC	Primary sclerosing cholangitis
SD-PSC	Small duct primary sclerosing cholangitis

Introduction

Primary sclerosing cholangitis (PSC) is a chronic idiopathic inflammatory disorder affecting intrahepatic and/or extrahepatic biliary ducts that is frequently associated with inflammatory bowel disease (IBD) [1]. PSC may lead to progressive hepatic fibrosis along with portal hypertension and the development of PSC-associated malignancies. Many patients are asymptomatic while some patients rapidly develop end-stage liver disease or cholangiocarcinoma (CCA) [2, 3].

A recent position statement from the International PSC Study Group (IPSCSG) recommended that magnetic resonance imaging (MRI) and magnetic resonance cholangiopancreatography (MRCP) should be the first diagnostic imaging modality in patients with suspected PSC [4]. MRI/MRCP is important in the surveillance of asymptomatic PSC patients for CCA, and is recommended in symptomatic patients prior to intervention, and for multicenter clinical and research trials [4–9]. MRI/MRCP may provide PSC prognostic information. For example, parenchymal abnormalities such as liver stiffness measured by elastography, hepatic dysmorphy, and delayed variable enhancement with use of hepatobiliary specific contrast agents are indicators of severity of liver fibrosis and predict adverse outcomes [10–12]. Other prognostic imaging parameters including the ANALI score (composite score of bile duct dilatation and parenchyma abnormalities), spleen size, and liver and spleen volumes have been also shown to predict outcome [10, 13–16].

Currently, there are no reporting standards/guidelines for MRI/MRCP findings in PSC. Controversies exist about the definition of severe or dominant strictures; a concept derived from endoscopic retrograde cholangiography (ERCP) but not widely used in routine clinical MRI/MRCP reports [17]. Guidelines are therefore needed for standardized reporting to promote better communication between healthcare providers and enhance the overall quality of care. In this paper, we present the proposed definitions and guidelines for standardized reporting of MRI/MRCP findings in PSC. Expert members of MR working group were tasked with development of a new consensus approach to reporting features of PSC aimed to standardize MRI/MRCP findings, assessment of disease severity and follow-up changes and diagnosis of cholangiocarcinoma.

Clinical manifestations of PSC

PSC has an adult male predilection, most commonly presenting in 4th to 5th decade though it can occur at any age including infants and children. The incidence is around 1 per 100,000 in Northern Europe and the USA. PSC is associated with IBD in up to 80% of patients with a predominantly ulcerative colitis-like phenotype [18–20]. Frequently, PSC is subclinical and discovered incidentally on imaging or laboratory tests during an evaluation of IBD. In general, symptomatic patients usually have advanced disease. With no effective medical therapy, the median transplant-free survival among those with PSC is approximately 15–20 years [7–9, 19].

The most common form is large duct PSC (LD-PSC). Variants include small duct PSC (SD-PSC) in 5–10% with a higher incidence in pediatric subjects [21] and PSC with concomitant autoimmune hepatitis in about 6–9% that is frequently corticosteroid responsive [22]. LD-PSC is typically visible on MRCP/ERCP as biliary strictures and dilatations. SD-PSC exclusively involves smaller ducts (< 100 microns) with normal cholangiography findings, and diagnosis therefore requires histological confirmation with liver biopsy [21, 23–25].

Management of PSC

Currently, the aim of PSC management is the early recognition and management of disease-related complications. Endoscopic dilatation and drainage of the severe stricture can be performed to ameliorate symptoms of a flow-limiting stricture and allow brushings and biopsy to evaluate for CCA. Careful selection of patients for endoscopic treatment is important, as it may precipitate intractable cholangitis [1]. Transplantation is indicated in patients with liver failure even without malignancies. However, some centers perform liver transplant in eligible cases of PSC with unresectable perihilar CCA with or without neoadjuvant chemoradiation. PSC can recur in up to 20–25% of patients within 5–10 years of transplantation, causing graft dysfunction and cholangitis [18].

Diagnosis of PSC

Diagnostic criteria for PSC include cholangiographic findings of bile-duct strictures detected by means of either MRCP or ERCP and exclusion of causes of secondary sclerosing cholangitis [18, 20]. A normal alkaline phosphatase does not exclude PSC as it may be normal in up to 50% [26, 27].

SD-PSC is a histologic diagnosis as the cholangiogram is normal. SD-PSC is associated with an improved overall prognosis and reduced risk of hepatobiliary malignancies, particularly CCA, when compared to LD-PSC [28].

Cholangiography

The typical cholangiographic findings are multiple strictures with intervening dilatations of both intrahepatic and extrahepatic bile ducts. Isolated changes can involve only intrahepatic bile ducts (IHD) in about 20–30% [18] and less than 10% of involvement of only extrahepatic ducts (EHD) [29]. The ductal changes are not specific to PSC and must be interpreted in the context of clinical and laboratory data.

ERCP is invasive and carries risk of complications, including infection and perforation, as well as exposure to ionizing radiation. MRI/MRCP is safer, usually less costly, and has a similar diagnostic performance to detect PSC when compared

Table 1 Guidelines for MRI/MRCP technique for PSC. Adapted from Ref.4. Schramm et al. Hepatology 2017; 66 (5)

Preparation	Fasting for minimum 4 h MRI/MRCP should be performed before any biliary intervention
Field strength	At least 1.5 Tesla.
T2-weighted MRCP	3D MRCP (TSE or FSE) or 2D MRCP or Single shot MRCP 3D MRCP preferred over 2D MRCP and single shot MRCP. Orthogonal coronal plane covering maximum possible liver in anteroposterior direction for adequate evaluation of peripheral bile ducts. Thin slice (~1 mm) acquisition is recommended. Use of respiratory or navigator triggered sequences and breath-hold 3D MRCP for minimizing motion artifacts
Liver parenchyma	T2-weighted (T2W) axial T1-weighted (T1W) axial-GRE[1] Diffusion weighted imaging (DWI) axial T2-weighted coronal Post contrast dynamic T1-weighted GRE[1] (arterial, portal, and delayed phases)
Optional	MR elastography 20-min hepatobiliary phase if a hepatobiliary contrast is used

TSE turbo spin echo, *FSE* fast spin echo, *GRE* gradient echo

[1] Three-dimensional spoiled GRE sequences are preferred for dynamic T1W acquisitions. These sequences are available as liver acquisition volume acceleration (LAVA), T1W high resolution isotropic volume examination (THRIVE), and volume interpolated breath-hold examination (VIBE). These can be obtained with DIXON reconstructions (in-phase, opposed-phase, fat-only, and water-only) when available

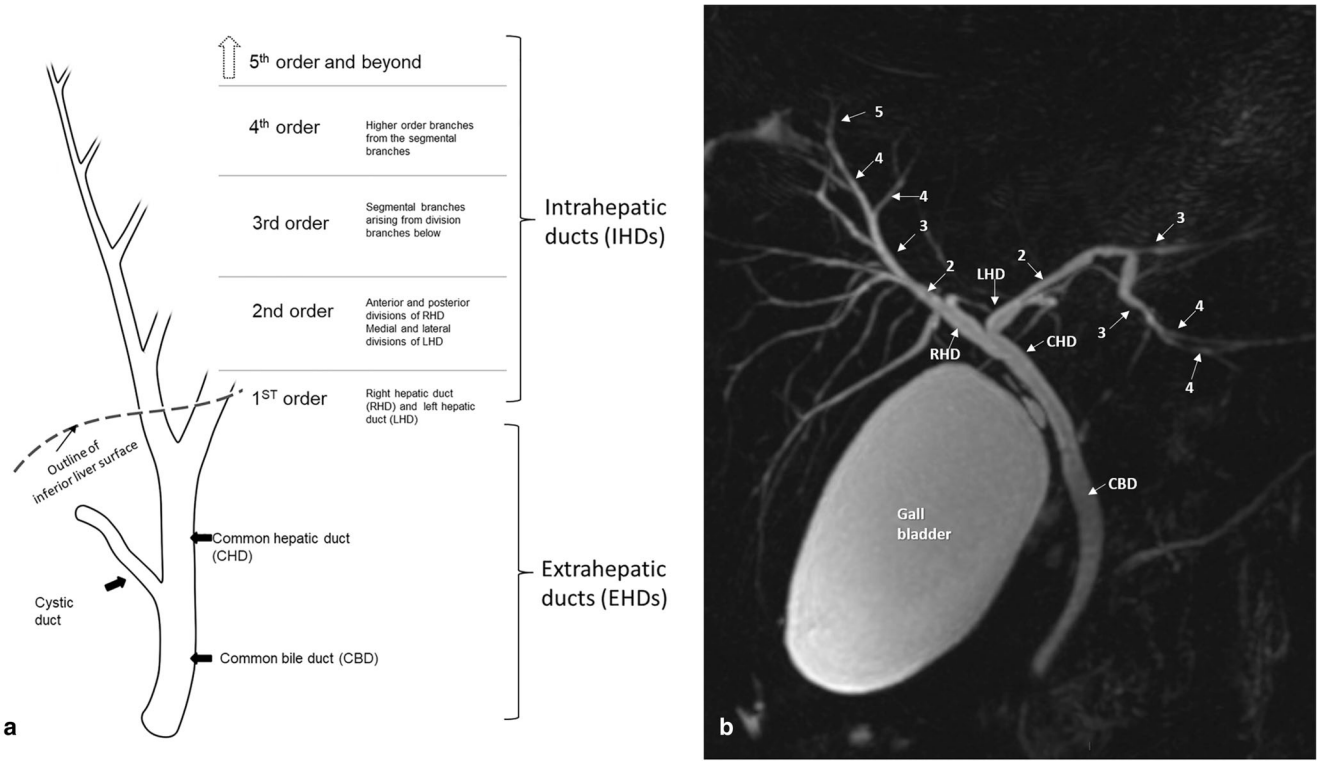

Fig. 1 **a** Schematic diagram showing biliary tree branching order. **b** Maximum intensity projection image of normal biliary tree. The number and arrows point to the branching order of ducts. Note that in this example, some 5th-order branches are also demonstrated

to ERCP [6]. The advantages of ERCP are its ability to perform therapeutic interventions and biliary brushings for cytology. ERCP also has higher resolution compared to MRCP which is particularly useful for evaluation of strictures in small peripheral ducts as in SD-PSC. However, ERCP may fail to visualize the entire biliary tree, and thus some severe intrahepatic strictures may not be observed. In the event ERCP is needed, a recent MRI/MRCP can provide a roadmap to the endoscopist by highlighting areas which should be targeted (e.g., strictures associated with a hepatic abscess) and others which contrast injection should be avoided (e.g., atrophic segments).

MRI/MRCP technique for PSC

MRI/MRCP should be the first diagnostic imaging modality in patients with suspected PSC for diagnosis as well as detection and characterization of complications including severity of biliary obstruction (4). Although the diagnostic workup for

Table 2 Image quality of MRI/MRCP for the evaluation of PSC

Quality	Artifacts[1]	Blurring[2]	Biliary tree delineation[3]
Excellent	None	None	Third-order* branches and beyond
Good	None or artifacts do not overlap biliary tree	Blurring of few ducts without affecting stricture assessment	Second-order branches and some third branches
Fair	Artifacts overlap biliary tree without affecting assessment	Moderate blurring of ducts affecting stricture assessment in <50%	First-order branches and some second-order branches
Poor	Artifacts overlap biliary tree rendering evaluation difficult	Severe blurring of biliary tree affecting stricture assessment in >50%	Incomplete biliary tree

*See Fig. 1

[1] Only artifacts (wrap around, phase shift, truncation, etc.) that superimpose the biliary tree should be considered for evaluation. Artifacts occurring at the edges of anatomical body without any impact on biliary tree depiction should not be considered

[2] Motion blurring due to respiration or patient motion that causes blurring of outline of biliary ducts and impacting the evaluation of strictures. Mild motion blur without impacting the evaluation of ductal lumen may not be considered

[3] Biliary tree includes EHD, IHD up to fourth order, and gallbladder when present

Table 3 Guidelines for reporting imaging findings on MRI/MRCP for primary sclerosing cholangitis

Quality of MRCP

Excellent/good/fair/poor (see description in Table 1)

Biliary strictures

Fixed, focal, or diffuse narrowing of bile duct which may or may not cause upstream dilatation. Strictures may involve a part (segmental or band) or entire length of the duct(s)

Present/absent

Number- single or multiple

Distribution

Localized to segment(s), lobe(s), or diffuse involvement

Severity of stricture (ref 3)

Diameter of the narrowed duct compared to normal or expected diameter of the duct. Avoid measuring severity at branching points. High-grade strictures can be variable in length and may cause minimal upstream dilation if there is another upstream stricture impeding biliary flow.

Low grade (< 75% luminal narrowing)
High grade (> 75% luminal narrowing)

Location of high-grade stricture(s)—describe the most severe high-grade stricture if present

Extrahepatic, intrahepatic, or both

If intrahepatic- mention the segment(s) involved

If extrahepatic- mention the location (CBD, CHD, RHD, LHD). Additional description of upper, mid, or distal CBD may be added

Length of the longest stricture

Measure the longest stricture length using the best possible image(s) that depicts the stricture including base images of MRCP, multi-planar reconstruction (MPR), or maximum intensity (MIP) projections. Depending on plane and length, multiple measurements may need to be added for full length of the stricture.

Bile duct dilatation-general

Present/absent

CBD > 6 mm in patients with gallbladder intact and > 10 mm in post cholecystectomy patients

> 4–6 mm for CHD, RHD, and LHD (reported normal size of extrahepatic ducts is variable and depend on the modality, age group studied and post cholecystectomy status). Measurement with higher specificity (> 6mm) is preferred (*)

> 3 mm for second-order branches and beyond

Additional description of the upstream dilatation in relation to high-grade strictures described

Duct ectasia/sacculation (> 10mm dilatation of IHD)

Present/absent

Duct wall thickening and enhancement

Measure the duct wall thickness as a single wall extending from biliary lumen to either peribiliary space or adjacent hepatic parenchyma and perpendicular to the lumen in the best image possible. Any wall thickness > 2 mm is abnormal (ref 3)

Present/absent

Mention the phase (arterial/portal/delayed) of maximal enhancement

Location of the duct wall thickening

Focal/segmental involving one or multiple ducts (see under CCA)
Diffuse duct wall thickening involving one or multiple ducts

Hepatolithiasis

Present/absent

Location

Single/multiple

Size—measure the largest dimension of the largest stone

Choledocholithiasis

Present/absent

Single/multiple

Size—measure the largest stone and report

Table 3 (continued)

Cholangiocarcinoma (CCA)
- Definite or possible (see Table 4 for criteria of perihilar CCA)
- Location—specify the duct or ducts involved
  - Perihilar/distal/intrahepatic
- Size—measure in mm the extent of the mass. At least one dimension (longest) should be reported from the sequence that demonstrate the mass best.
- Post gadolinium enhanced delayed phase images are most useful; however, DWI and HBP images can also be useful
  - Radial diameter of the mass should be measured where it is thickest and perpendicular to the orientation of the duct involved.
- Vessel involved?
  - Yes/no
  - Report which vessel and length of vessel involvement
  - When CCA involves only one lobe, involvement of hepatic artery and portal vein supply to contralateral lobe should be specifically mentioned
  - Extent of involvement
  - • Abutment—vessel contact < 180 degrees-without narrowing
  - • Abutment with narrowing
  - • Encasement—vessel contact > 180 degrees without obliteration
  - • Encasement with obliteration
- Associated parenchymal atrophy or hypertrophy
- Invasion into the liver parenchyma (loss of fat plane between the mass and adjacent liver parenchyma)
  - Present/absent
- Suspicious lymph nodes (see below portal lymphadenopathy)
  - Round morphology, central necrosis, restricted diffusion compared to other lymph nodes, and hyperenhancement
  - Present/absent
- Intrahepatic metastases
  - Present/absent

Liver parenchyma
- Parenchyma signal—evaluate liver parenchyma signal on all available sequences including T2- or T1-weighted and diffusion weighted imaging
  - Abnormal/normal
  - If abnormal describe the sequence and if present in other sequences
  - Distribution of abnormal signal
  - Segmental, lobar, or diffuse
- Parenchyma atrophy and hypertrophy
  - Atrophy is seen as loss of volume of a segment or lobe with crowding of the biliary ducts.
  - Hypertrophy is enlargement of the lobe or segment
  - Present/absent
  - If present—specify which segment(s) or lobe(s)
- Abnormal liver parenchyma enhancement
  - Present/absent
  - If present, mention the phase—arterial, portal, delayed, or hepatobiliary if available
  - Distribution—segmental, lobar, or diffuse and if they correspond to parenchyma signal abnormality seen before contrast
- Cirrhotic morphology
  - Present/absent

Signs of portal hypertension including splenomegaly, portosystemic collaterals, and ascites
- Present/absent for each

Portal lymphadenopathy (lymph nodes > 1 cm in short axis)—the size and morphology of the lymph nodes are not specific for presence or absence of cholangiocarcinoma and cannot differentiate from reactive lymph nodes due to ongoing PSC
- Present/absent

Gallbladder
- Present/absent
- If present
  - Normal/distended.
- Report other abnormalities when present

CBD common bile duct, *CHD* common hepatic duct, *RHD* right hepatic duct, *LHD* left hepatic duct

*Reference (3): Ruiz A, et al. Hepatology 2014;59(1):242-25 and (60) Pavlovic T et al. Croat Med J 2020; 61:239-45

PSC can be performed using only MRCP, it is preferable to have a comprehensive evaluation of the liver and entire abdomen with contrast-enhanced MRI [4]. The recommended guidelines for MRI/MRCP technique for PSC are summarized in Table 1. Ideally, MRI/MRCP of patients with suspected PSC should be assessed by radiologists experienced with PSC [4].

The order of biliary tree branches is illustrated in Fig. 1. In normal individuals, it is common for only central IHDs (up to second-order branches) to be adequately visualized on a

Table 4 Definitions of MRI criteria for early stage perihilar cholangiocarcinoma in PSC. The criteria should be applied only in PSC patients. Other biliary diseases that show periductal soft tissue thickening including IgG4 disease and recurrent pyogenic cholangitis should be excluded

Criteria	Definition
Definite	• Perihilar mass with progressive enhancement on delayed phase imaging with or without intraductal luminal mass lesions or invasion into surrounding liver parenchyma or • Focal periductal soft tissue thickening with vascular narrowing or encasement or periductal soft tissue thickening that progressively enhances to maximum on delayed phase imaging
Possible	• Stricture with periductal thickening but without delayed phase enhancement or vascular narrowing (or encasement) or • Progressive lobar atrophy secondary to worsening perihilar stricture. or • Ill-defined delayed enhancement of ductal wall without distinct mass.

Adapted from Reference (7) Eaton et al. Hepatology 2020. doi: 10.1002/hep.31575

standard MRCP [30, 31]. While segmental ducts (third order) are often seen, non-dilated subsegmental ducts (fourth order) are typically not visualized on a standard 3D MRCP with 1-mm resolution. Therefore, when many fourth-order IHDs are visualized, the ducts should be evaluated for disease and downstream strictures (32). Conversely, non-visualization of third- and fourth-order ducts should not be interpreted as pruning or severe disease.

Quality of MRCP

The quality of MRCP obtained may be evaluated based on the depiction of EHDs and IHDs, gallbladder and absence of artifacts and motion blurring. An excellent MRCP study for evaluation of PSC would depict up to third-order biliary ducts with no artifacts over the biliary tree and no blurring of the ducts. The proposed classification of quality

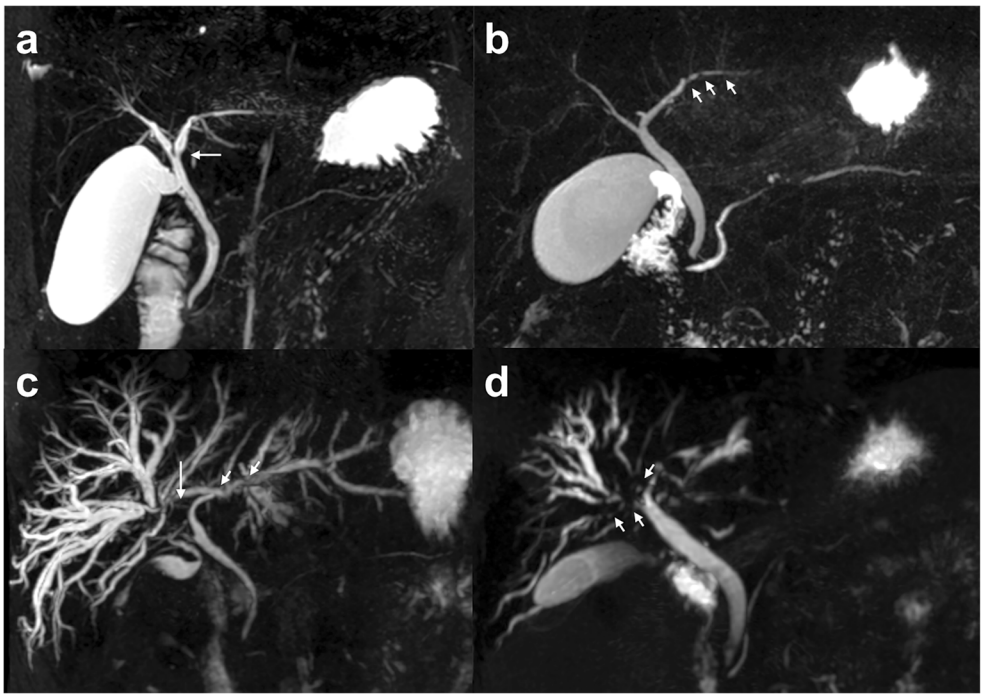

Fig. 2 Maximum intensity projection (MIP) images from MRCP of different PSC patients showing strictures of various grades. **a** Low-grade (<75% lumen narrowing) strictures involving the extrahepatic portions of the right and left hepatic ducts (white arrow). **b** Low-grade stricture involving the left hepatic duct and extending into subsegmental duct in another patient (short arrows) in addition to several other subtle strictures (not shown). **c** High-grade (>75% lumen narrowing) stricture involving the right hepatic duct (white arrow) in addition to low-grade strictures in the left second- and third-order intrahepatic duct (short arrows). Follow-up ERCP and biopsy were negative for cholangiocarcinoma. **d** Multiple high-grade strictures involving the first- and second-order intrahepatic ducts (short arrows) in a patient with PSC and chronic ulcerative colitis

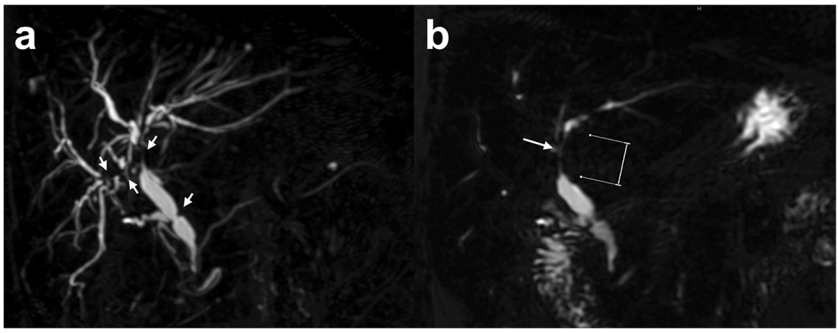

Fig. 3. Maximum intensity projection image (**a**) and a single coronal MRCP image (**b**) in a patient with ulcerative colitis and PSC, and history of gallbladder carcinoma incidentally found at cholecystectomy for symptomatic gallstones. Multiple biliary strictures involving the intrahepatic and extrahepatic ducts (short arrows). The longest stricture involved the left hepatic duct and a second-order branch (arrow) and measured 21mm in length (calipers, b). Findings were stable on follow-up for 8 years

of MRCP is outlined in Table 2. Every attempt should be made to obtain at least a good quality MRCP. In case of low-quality (poor or fair) MRCP even after multiple attempts, one should consider using another scanner platform (1.5T vs. 3T or 3T vs.1.5T) or at a more experienced MR scanning facility to ensure a high-quality MRCP is obtained. When MRI/MRCP is suboptimal but clinical suspicion of PSC remains high, a repeat MRCP is recommended within 3 months preferably at a facility experienced with such scanning.

MRI/MRCP findings in PSC

The main recommendations and reporting terminologies for reporting MRI/MRCP in PSC are summarized in Table 3

Bile ducts

Biliary stricture

Biliary strictures are a hallmark of PSC. Strictures may or may not cause upstream biliary dilation and may involve only a part (band or segmental) or entire length of the duct. This applies to both IHDs and EHDs. The strictures should be reported as present/absent (Table 2).

The distribution of the strictures should be discussed, noting single or multiple strictures as described in Table 3. Special attention must be directed to progression of the severity of a previously demonstrated stricture and/or increasing upstream dilatation as these findings may indicate CCA. This is further discussed under the CCA section.

Severity of stricture

Severity of the stricture should be assessed whenever possible on the best quality sequence. Classification into low grade (< 75% luminal narrowing) and high grade (> 75% luminal narrowing) may be reported [3]. Factors that should be considered include the diameter of the involved segment compared to the normal or expected diameter of that segment, the length of the stricture (described below), and any upstream dilation. The extent of strictures can be described as localized (segmental/lobar) or diffuse when the entire biliary tree is involved [3]. High-grade strictures may have clinical implications as biliary intervention may be required to relieve obstruction and evaluate for CCA.

Over 50% of patients with PSC will develop focal high-grade strictures referred to as dominant stricture (DS) during the course of disease [32]. Patients with DS may be asymptomatic or present with abnormal liver function tests, abdominal pain, and/or cholangitis [33]. DS is defined in the ERCP literature arbitrarily as intraluminal diameter < 1.5 mm in the common bile duct and < 1 mm in the hepatic ducts within 2 cm of bifurcation [34, 35]. ERCP is performed with pressure injection, while distensibility of the strictures is not assessed on MRCP. Thus, findings on MRCP may not correlate precisely to DS on ERCP. Also, additional strictures peripheral to a non-traversed or poorly filled DS on ERC may be detected on MRCP. Bile duct wall thickening, a valuable feature in the assessment of stricture, is easily seen with MRCP and not possible on ERCP. Severe strictures in PSC may lack prognostic value as there is lack of correlation between biochemical cholestasis and degree of stenosis [32, 36]. Furthermore, the term DS can be confusing, as it creates a distinction among different strictures when such differences might not be present. Even though severe strictures are associated with morbidity, clinical perceptions and practices vary widely among clinicians who manage these lesions [32]. Therefore, we recommend against using the term DS with MRCP. Rather, we advise grading strictures as low or high grade (Fig. 2), mentioning the location and features of the

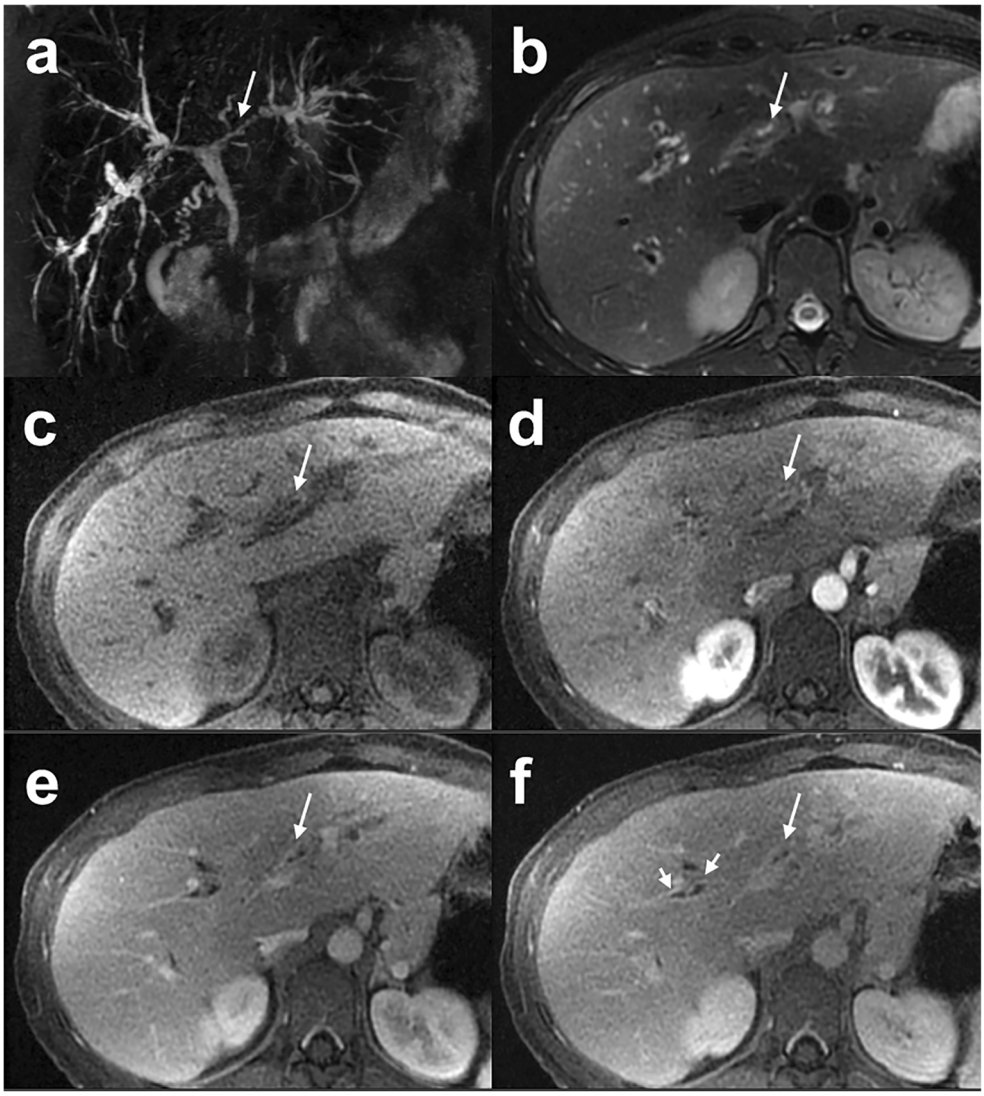

Fig. 4 MRCP (**a**) and representative axial T2-weighted (**b**), pre contrast (**c**), post contrast enhanced arterial phase (**d**), portal venous phase (**e**), and delayed phase (**f**) T1-weighted images in a patient with primary sclerosing cholangitis. Multiple strictures involving predominantly intrahepatic ducts are seen on the MRCP image. Note the longest stricture involving the left hepatic duct (arrow, **a**). Thickening of the left hepatic duct seen as hypointensity with surrounding hyperintensity on T2-weighted image (arrow), isointensity on pre contrast T1-weighted image, and enhances in the arterial phase with maximum enhancement in the delayed phase. The inflammatory wall thickening is diffuse and measured 3.6mm in maximum dimension with no associated mass. Note also enhancing mildly thickened walls of the right hepatic ducts best seen in delayed phase (arrow heads). ERCP brushings and biopsy were negative for cholangiocarcinoma. Patient received a deceased donor liver transplant 4 years later for progressive liver disease

strictures outlined in Table 3, and if there are concerns for CCA (defined in Table 4).

Length of stricture

The length of the longest stricture may be useful information for any planned interventions. The length of the most severe stricture(s) on any sequence that clearly delineates the length should be reported (Fig. 3). When a stricture extends from a more central duct into a single or multiple IHDs, the measurement should include the total length of the stricture choosing the longest and most severe stricture course.

The presence of biliary stents can make it difficult to fully appreciate strictures and thus measure their length. Additional clues including wall enhancement, increased wall thickness, and any residual dilatation may be useful when stents are present. If the reader is not confident about the stricture length, a disclaimer can be added to explain the uncertainty related to the stent.

Dilatation

The degree of biliary dilatation should be discussed, either as a general description or with specific examples particularly of the greatest degree of dilatation. The degree of IHD dilatation may serve as a prognostic marker as demonstrated in a few publications [3].

Asymmetrical dilatation of the duct causing outpouching of a part of the duct leads to sacculations, also known as ectasia. The importance of detecting sacculation alone on MRCP is uncertain, although a recent study showed that cystic dilation of IHDs results in unfavorable clinical course and a significant prognostic factor likely due to intractable super added infection [37].

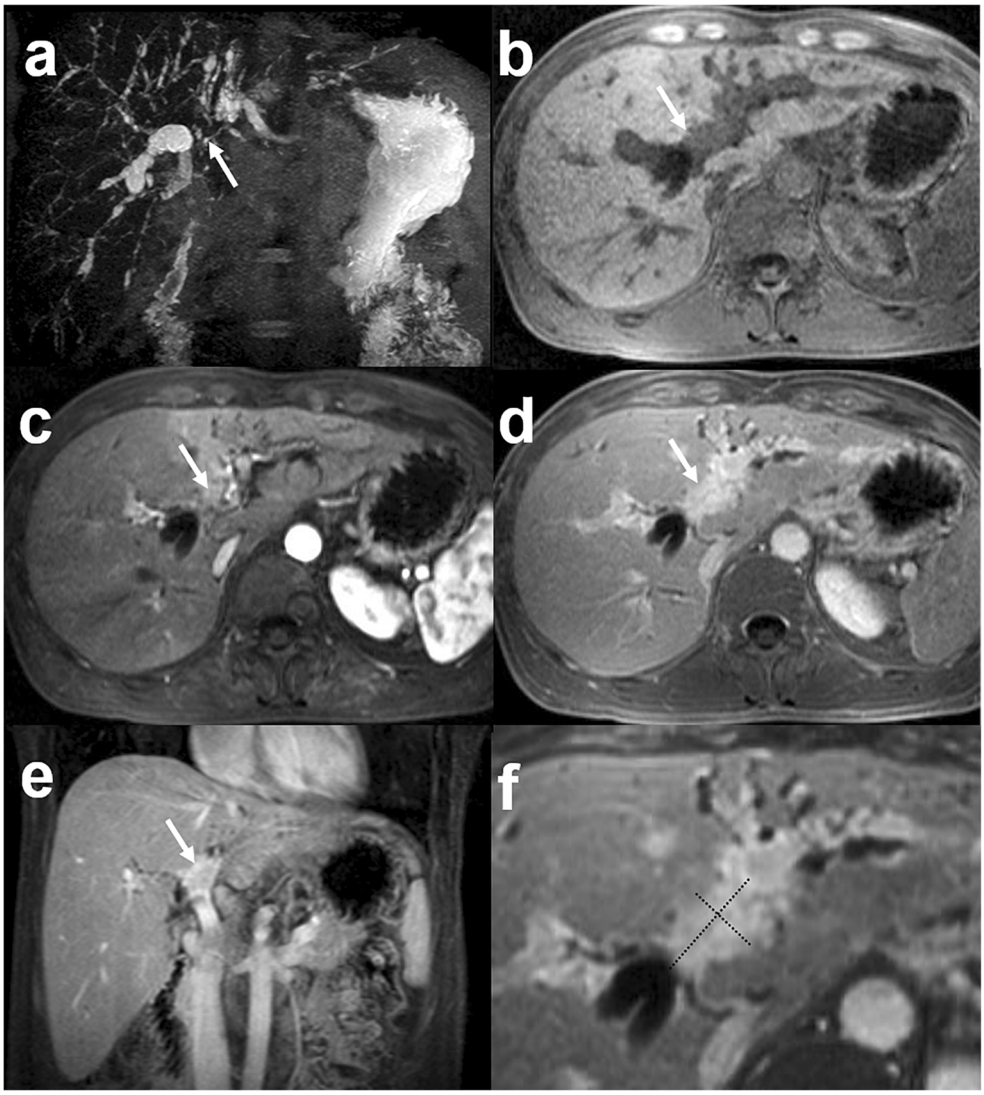

Fig. 5 Perihilar cholangiocarcinoma in a patient with PSC. MRCP (**a**) showing a severe stricture involving the hilum (white arrow) with more involvement of the left hepatic duct. Note multiple segmental strictures throughout the biliary tree. Pre contrast T1-weighted image (**b**) showing a hypointense mass in the hilum (white arrow) which shows heterogeneous enhancement in arterial phase (**c**) and complete enhancement in the delayed phase (**d**). Post contrast coronal T1-weighted image showing the hilar mass centered more on the left duct (**e**). Magnified view (**f**) of post contrast image showing radial measurement of the mass drawn perpendicular (line with end arrows) to the expected left hepatic duct course (dashed line). The mass measures 19 mm × 31 mm. ERCP performed after MRI was positive for cholangiocarcinoma. Patient received neo-adjuvant chemoradiation followed by deceased donor orthotopic liver transplantation

Duct wall thickening and hyperenhancement

Bile duct wall thickening may represent ongoing inflammation and/or fibrosis and in some cases CCA. Thickened bile duct walls are most conspicuous and easily measured on enhanced images, preferably in the equilibrium (delayed) phase (Fig. 4). While normal bile ducts are usually either imperceptible or very thin, any wall thickness > 2 mm should be considered abnormal, regardless of location [3]. Enhancement should be assessed in all phases available, with the phase of maximal enhancement stated. Our recommendation is to report the maximal wall thickness and associated hyperenhancement and any strictures. Any focal nodular thickening or focal thickening with associated portal vein narrowing should raise the suspicion of CCA. Bile ducts can demonstrate diffusion restriction in the setting of inflammation and tumor [38].

Hepatolithiasis

Hepatolithiasis, defined as biliary stones proximal to the confluence of the left and right hepatic ducts, is associated with cholestasis and biliary strictures and is an important observation due to the increased risk of cholangitis and symptomatic biliary obstruction [39]. Although MRCP and T2-weighted are most useful for detecting hepatolithiasis, T1-weighted images may be helpful in differentiating iso or hyperintense stones from pneumobilia [40]. In-phase images may show blooming relative to the opposed-phase images from pneumobilia. The presence and location of hepatolithiasis should be reported.

Cholangiocarcinoma in PSC

Cholangiocarcinoma (CCA), the most common PSC-associated malignancy, is the leading source of mortality in

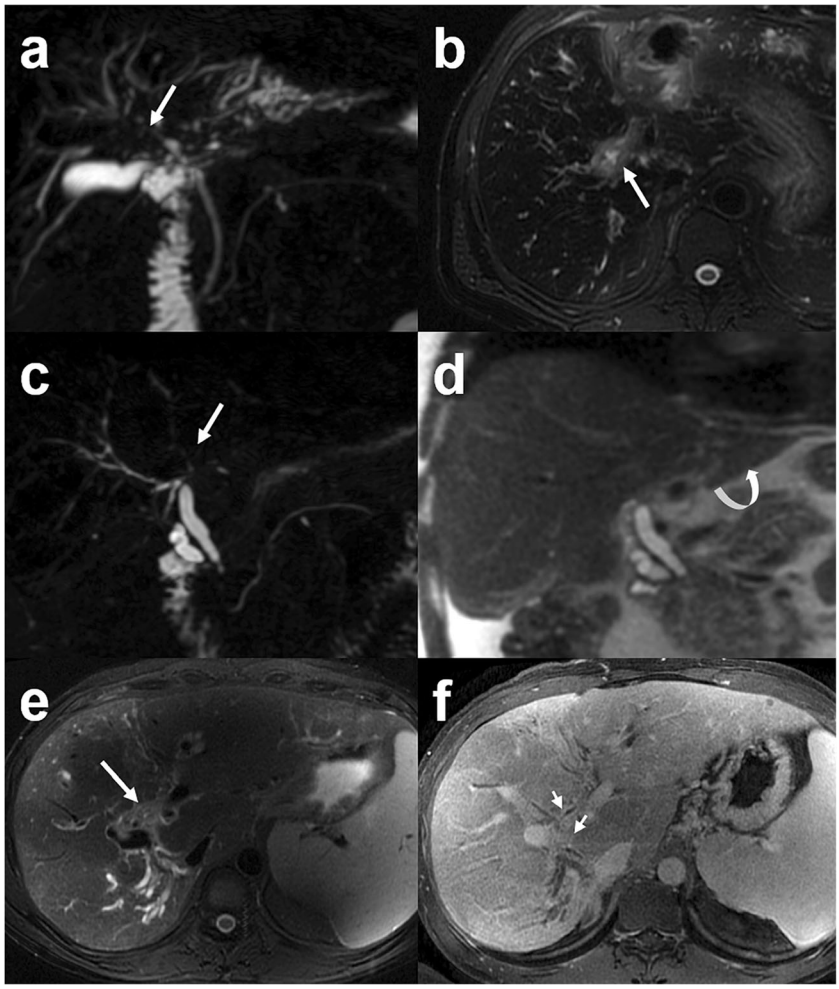

Fig. 6 Examples showing possible imaging features of cholangiocarcinoma in different patients with PSC. Top row (**a**, **b**): MRCP (**a**) showing a high-grade stricture of the right hepatic duct (arrow) with associated periductal thickening (arrow) shown on axial T2-weighted image (**b**) without vascular involvement. ERCP with brushings cytology and fluorescence in situ hybridization (FISH) was negative for malignancy. Patient was lost to follow-up. Middle row (**c**, **d**): MRCP (**c**) showing high-grade stricture with cutoff of left hepatic duct (arrow) and associated left lobe atrophy (curved arrow) shown on coronal T2-weighted image (**d**). ERCP brushings and FISH were negative for cholangiocarcinoma. Patient is currently on follow-up. Bottom row (**e**, **f**): Axial T2-weighted image (**e**) showing irregular thickening of the ducts (white arrow) that shows ill-defined delayed enhancement (short arrows, **f**) without any distinct mass. ERCP brushings were negative for cholangiocarcinoma. Four months later, the patient received a deceased donor orthotopic liver transplant for progressive liver disease. The explant showed no evidence of cholangiocarcinoma.

this population [19]. Anatomically, the majority of CCAs in PSC are perihilar (Fig. 5), though distal (below the cystic duct) or intrahepatic CCA (arising beyond second-order IHDs) can also occur. Among PSC patients, 10–20% will develop CCA regardless of the degree of fibrosis [20, 28]. Approximately, one-third of all CCAs are detected within 1 year of establishing a PSC diagnosis with an annual incidence thereafter of 0.5–1% [28, 41]. However, CCA is rare among children and in those with SD-PSC [19, 28].

MRI/MRCP with contrast is the preferred noninvasive imaging study. Standardized reporting criteria of features definite for CCA and indeterminate stricture possibly due to CCA have been codified and studied [7] (Table 3). Definite imaging criteria for CCA (Table 4) alone have an excellent specificity (98%) but suboptimal sensitivity (58%) among those with early-stage perihilar CCA. However, suspicious strictures (Fig. 6) for CCA (Table 4) should prompt clinicians to proceed with an ERCP for biliary brushings. Combining both definitive and possible imaging criteria for perihilar CCA raise the sensitivity of perihilar CCA detection to 89% while maintaining a satisfactory specificity (86%) [7].

Description of tumor should be reported in detail including the length and radial diameter (perpendicular to the course of bile duct of the involved bile duct (s)) of the tumor, length of ducts involved, and any vessels involved for assessing for

resectability [42]. The individuals with localized perihilar CCA over 3 cm in radial diameter are generally excluded from liver transplantation due to recurrence risk [43]. Indistinct margins between the mass and adjacent liver should be described, as this suggests hepatic invasion.

Parenchymal atrophy and/or hypertrophy are nonspecific findings and can be seen in the setting of PSC or CCA, among other conditions [44]. Presence of intrahepatic metastases, lymphadenopathy, and peritoneal disease including peritoneal nodules, thickening, and free fluid should be mentioned. There are no reliable size criteria for differentiating malignant from reactive lymph nodes [45].

Hepatic parenchymal changes

Signal intensity changes on non-contrast-enhanced sequences

The parenchymal changes in PSC are often heterogeneous, with peripheral wedge-shaped areas of inflammation and/or fibrosis and central regenerative hypertrophy [46]. The relationship between signal intensity changes and biliary strictures is complex [47, 48]. Peripheral, wedge-shaped areas of T2-weighted hyperintensity, T1-weighted hypointensity, and diffusion restriction can be seen with inflammation and altered vascular/lymphatic flow. However, confluent fibrosis may show similar signal intensity changes and often shows crowding of vessels, volume loss, and capsular retraction [47, 49, 50]. Parenchymal signal intensity changes may be present in the absence of typical MRCP findings particularly in SD-PSC or in early LD-PSC when MRCP findings are equivocal, though the specificity for PSC is unclear [24, 25].

Atrophy and hypertrophy of the segments

As the disease progresses and destruction of the bile ducts continues, fibrosis and associated parenchymal atrophy develop. Relatively unaffected areas of the liver undergo compensatory hypertrophy, with macroregenerative nodules in the caudate lobe seen more commonly in PSC than with other causes of cirrhosis [46, 49]. It is important to differentiate the typical atrophy and regenerative hypertrophy seen in PSC from CCA-related atrophy (see CCA section).

Parenchymal enhancement after intravenous gadolinium chelate administration

Arterial phase hyperenhancement has been shown to correlate with active hepatic inflammation or compensatory arterial hyperperfusion related to portal venous branch narrowing or obstruction [51, 52]. Delayed phase enhancement, on the other hand, has been shown to correlate with increasing fibrosis [53, 54]. Any abnormal enhancement should be described as focal, segmental, lobar, or diffuse. Please see additional considerations during hepatobiliary phase in *supplement.*

Table 5 Additional comments on follow-up MRI/MRCP or when previous MRI/MRCP is available in patients with PSC

Feature	Description
Biliary strictures	• Stable
	• Worsening in length/narrowing/upstream dilatation
	• New strictures—describe
Bile ductal wall thickening and enhancement	• Increased thickening—measure and mention the size.
	• Change in enhancement.
	• Is thickening suspicious for CCA (see Table 3)
Parenchymal changes	• Development of heterogeneity of signal or enhancement
	• Morphological changes from prior study particularly—nodularity, lobular outline, atrophy, and hypertrophy of lobes
Portal hypertension	• New development of collaterals or splenomegaly as compared to prior when it was absent
	• Progressive splenomegaly
	• Worsening ascites
Focal liver lesions	• New lesions in the liver—describe the lesion
	• Use LIRADs if liver is cirrhotic morphology
MR elastography (Optional)	• Report changes in liver stiffness if any
Hepatobiliary phase (if available)	• Development of new region of liver without uptake and/or without biliary excretion

Cirrhosis and portal hypertension

PSC can eventually lead to cirrhosis and portal hypertension [20]. Liver stiffness is a surrogate for hepatic fibrosis and may be quantified using shear wave elastography or magnetic resonance elastography which in turn is associated with clinical outcome [10, 55–57]. The presence or absence of signs of portal hypertension should be discussed, and focal enhancing lesions in cirrhotic liver should raise the possibility of HCC or intrahepatic CCA.

Gallbladder

A brief mention of the gallbladder is warranted, including its presence or absence, enlargement, shrinkage, or normal status, as well as a description of any abnormalities. Patients with PSC have an increased risk of gallbladder carcinoma, so careful evaluation should be performed to assess for any growing polyps, focal thickening, or mass [58, 59].

Additional features with hepatobiliary contrast agents and MR elastography are described in *supplement*.

Follow-up MRI/MRCP

In a follow-up study, the report should mention what has changed since prior studies with regard to biliary strictures and liver parenchyma (Table 5). Note should be made regarding worsening or improving biliary strictures, development of new strictures, change in duct wall thickness or enhancement, any strictures suspicious for CCA, and portal hypertension features.

Summary

Herein, the Imaging working group of the IPSCSG has proposed new reporting standards for specific features of PSC at MRI/MRCP. Application of these standardized features can enhance the quality of care across centers by aiding in an early diagnosis, prompt recognition of PSC-related complications, and in longitudinal assessment.

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Methodology
- retrospective
- observational

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