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MR elastography in primary sclerosing cholangitis: correlating liver stiffness with bile duct strictures and parenchymal changes

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Abstract

Aim: To determine correlation of liver stiffness measured by MR Elastography (MRE) with biliary abnormalities on MR Cholangiopancreatography (MRCP) and MRI parenchymal features in patients with primary sclerosing cholangitis (PSC).

Methods: Fifty-five patients with PSC who underwent MRI of the liver with MRCP and MRE were retrospectively evaluated. Two board-certified abdominal radiologists in agreement reviewed the MRI, MRCP, and MRE images. The biliary tree was evaluated for stricture, dilatation, wall enhancement, and thickening at segmental duct, right main duct, left main duct, and common bile duct levels. Liver parenchyma features including signal intensity on T2W and DWI, and hyperenhancement in arterial, portal venous, and delayed phase were evaluated in nine Couinaud liver segments. Atrophy or hypertrophy of segments, cirrhotic morphology, varices, and splenomegaly were scored as present or absent. Regions of interest were placed in each of the nine segments on stiffness maps wherever available and liver stiffness (LS) was recorded. Mean segmental LS, right lobar (V–VIII), left lobar (I–III, and IVA, IVB), and global LS (average of all segments) were calculated. Spearman rank correlation analysis was performed for significant correlation. Features with significant correlation were then analyzed for significant differences in mean LS. Multiple regression analysis of MRI and MRCP features was performed for significant correlation with elevated LS.

Results: A total of 439/495 segments were evaluated and 56 segments not included in MRE slices were excluded for correlation analysis. Mean segmental LS correlated with the presence of strictures ($r = 0.18$, $p < 0.001$), T2W hyperintensity ($r = 0.38$, $p < 0.001$), DWI hyperintensity ($r = 0.30$, $p < 0.001$), and hyperenhancement of segment in all three phases. Mean LS of atrophic and hypertrophic segments were significantly higher than normal segments (7.07 ± 3.6 and 6.67 ± 3.26 vs. 5.1 ± 3.6 kPa, $p < 0.001$). In multiple regression analysis, only the presence of segmental

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Compliance with ethical standards

Conflict of Interest RLE and the Mayo Clinic have a financial interest in MRE technology and hold equity in Resoundant, Inc. All other authors declare that they have no conflict of interest for the above work.

strictures ($p < 0.001$), T2W hyperintensity ($p = 0.01$), and segmental hypertrophy ($p < 0.001$) were significantly associated with elevated segmental LS. Only left ductal stricture correlated with left lobe LS ($r = 0.41$, $p = 0.018$). Global LS correlated significantly with CBD stricture ($r = 0.31$, $p = 0.02$), number of segmental strictures ($r = 0.28$, $p = 0.04$), splenomegaly ($r = 0.56$, $p < 0.001$), and varices ($r = 0.58$, $p < 0.001$).

Conclusion: In PSC, there is low but positive correlation between segmental LS and segmental duct strictures. Segments with increased LS show T2 hyperintensity, DWI hyperintensity, and post-contrast hyperenhancement. Global liver stiffness shows a moderate correlation with number of segmental strictures and significantly correlates with spleen stiffness, splenomegaly, and varices.

Keywords

Segmental stiffness; Biliary stricture; Atrophy; Hypertrophy; Signal intensity; Hyperenhancement

Primary sclerosing cholangitis (PSC) is a chronic liver disease characterized by multifocal intra- and extrahepatic biliary strictures and a frequent association with inflammatory bowel disease [1–3]. Nearly half of patients with PSC are asymptomatic at the time of diagnosis [4] with variable rate of progression and variable associated complications including cholangitis, cholangiocarcinoma, cirrhosis, and liver failure. Currently, there is no effective medical therapy for PSC [5]; however, development of new therapies requires accurate, non-invasive biomarkers as surrogate endpoints for disease progression and therapy response [5–9]. There is an unmet need for accurate and preferably non-invasive biomarkers to evaluate stage of the disease, assess progression of disease, and to serve as surrogate endpoints in the design of clinical trials [5–9].

Imaging plays an important role in the diagnosis and management of PSC. MR cholangiopancreatography (MRCP) or endoscopic retrograde cholangiopancreatography (ERCP) is essential for diagnosis of PSC. High-grade and diffuse strictures of intrahepatic biliary tree on ERCP were found to be indicators of poor prognosis and correlated with disease severity [10–12]; however, ERCP is invasive and associated with complications. MRCP is now the standard non-invasive imaging test for diagnosis of PSC; however, association with biliary strictures on MRCP and clinical severity is yet to be determined [13, 14].

Liver fibrosis stage is predictive of transplant-free survival and time to liver transplant in PSC [6]. Elastography methods that measure liver stiffness (LS) noninvasively show excellent correlation with liver fibrosis stage when compared with biopsy [15]. Baseline LS can differentiate severe from non-severe liver fibrosis at the time of diagnosis. Both baseline and rate of progression of LS are strongly associated with clinical outcome in PSC. A recent study by Eaton et al. [16] demonstrated that LS measured with MR Elastography (MRE) accurately distinguishes the presence of cirrhosis and predicts patient outcomes in PSC.

Development of liver fibrosis is an important prognostic factor for liver-related events in the course of PSC [6]. Due to the heterogeneous and variable distribution of parenchymal changes in PSC, MRE may be better suited for providing an accurate assessment of liver

stiffness and other PSC-related complications when compared to ultrasound-based elastography techniques. There are limited data describing the relationship between cholangiographic and parenchymal features [13]. Liver function evaluated with hepatobiliary contrast has previously been shown to be heterogeneously distributed and correlates with biliary obstruction [14]. However, the association between LS with hepatic parenchymal signal intensity, parenchymal enhancement, and biliary tree features is not well understood. Improving our understanding of these imaging relationships is important to further our understanding of the disease process, and generate new hypotheses that could be used to develop future imaging surrogate markers that could predict clinically relevant outcomes. Therefore, we performed this study to determine the correlation between parenchymal signal intensity/enhancement, parenchymal atrophy or hypertrophy and biliary abnormalities on MRCP with LS evaluated with MRE.

Methods

This study was approved by our institutional review board with a waiver for informed consent for retrospective review of data. Patients with PSC between February 2007 and December 2013 who satisfied inclusion criteria were randomly selected for review.

Patient selection

A total of 55 patients with (a) clinically confirmed diagnosis of large duct PSC and who (b) underwent a diagnostic MRI with dynamic contrast enhanced sequences including a MRCP and MRE formed the study group. PSC patients with biliary intervention including drainage/stents prior to their MRI or diagnosed with cholangiocarcinoma at the time of their initial presentation were excluded.

MRI/MRCP/MRE

MRI with MRCP and MRE was performed on either a 1.5T or 3T clinical MR scanners (GE, Waukesha, WI) at our institution. MRI of the liver was performed with routine clinical liver MRI protocol that included fat suppressed T2 weighted (T2W), pre-contrast T1 weighted (T1W), in- and opposed phase T1W, diffusion-weighted imaging (DWI), and dynamic post-contrast (arterial, portal venous, and delayed phases) T1W sequences. MRCP was performed with heavily T2W MR sequences either using navigator respiratory triggered or breath-hold techniques. Coronal and radial single-shot fast spin echo (SSFSE) T2W sequences were also obtained whenever needed.

MRE was performed with conventional two-dimensional GRE (2D GRE MRE) sequence as described in the literature [17, 18]. Four 10-mm-thick slices were obtained through the largest cross section of the liver. The MRE acquisition was completed in 2 min. Parameters of the liver MRI, MRCP, and MRE protocol sequences are summarized in Table 1

Image evaluation

Two board-certified attending radiologists with 3 and 15 years of post-training experience in reviewing the abdominal MRI studies evaluated the MRI/MRCP/ MRE images in consensus and blinded to laboratory findings.

Common bile duct (CBD), right main duct (RMD), and left main ducts (LMD) and segmental ducts (SD) of all nine Couinaud segments were evaluated for strictures, dilatation, wall thickening (> 2 mm), and hyperenhancement. For the study purpose, the entire length of the common duct from hilar confluence to the level of duodenal papilla was considered as one duct. Strictures were scored as present or absent and graded as 0 for no stricture, 1 for $< 25\%$, 2 for $25\text{--}50\%$, 3 for $50\text{--}75\%$, and 4 for $75\text{--}100\%$ of the lumen narrowing. Bile duct wall hyperenhancement was considered present or absent on each dynamic contrast-enhanced images including arterial phase, portal venous phase, and delayed phase (3–5 min). Dilatation was considered to be present when segmental ducts were > 4 mm, RMD/LMD were > 7 mm in and common bile duct was > 10 mm in diameter. Parameters were based on a simplified system by Ruiz et al. [14].

Parenchymal signal intensity and enhancement for each Couinaud liver segments I to III, IVA, IVB, and V to VIII were assessed. Signal intensity and enhancement of a segment was determined qualitatively by majority ($> 50\%$) of parenchyma showing that feature in relation to adjacent hepatic parenchyma particularly to the central regenerative parenchyma [19]. The signal intensity on T2W and DWI was graded as iso/hypo-intense or hyperintense with hyperintensity considered as abnormal. Parenchymal enhancement on post-contrast-enhanced arterial, portal, and delayed T1W images were graded as iso- or hyperintense. Segments were also evaluated for atrophy (reduction in volume, crowding of biliary ducts and intrahepatic vessels) and hypertrophy (enlarged lobe with separation of vessels/fissures) and graded as present or absent. Signs of cirrhosis (surface nodularity, caudate lobe enlargement, volumetric changes of atrophy and hypertrophy) and portal hypertension (splenomegaly, varices, and ascites) were also noted as present or absent.

LS with MRE was also evaluated for all nine segments by placing regions of interest (ROI) on each segment wherever available and mean values were recorded. Our study population included patients from 2007 to 2013 when confidence maps, which are now routinely available for ROI placements were not available. Hence, we did not use confidence maps to guide placing the ROIs for this study. The ROIs were oval or round shaped and at least 100 mm^2 in size and placed in all nine Couinaud segments wherever available (Fig. 1). Lobar stiffness was calculated by averaging LS of the individual segments: right lobe LS was derived from segments V to VIII and the left lobe LS was derived from I to III, and IVA and IVB. Global stiffness was obtained by averaging mean values from all available segments. We used the magnitude images from the MRE sequence and matched it to closest possible section on T2W, DWI, pre- and post-Gd-enhanced sequences. After drawing the ROI on the magnitude images, they were copy pasted on to the gray-scale stiffness maps that provided the liver stiffness measurements. The color stiffness maps were not used to guide ROI placement (Fig. 1). Laboratory data collected included serum alkaline phosphatase (ALP) and total bilirubin (TB). Model end liver data (MELD) scores and Mayo Risk scores were also computed [20].

Statistical evaluation

Correlation between LS and MRI/MRCP features were evaluated using Spearman rank correlation analysis. Non-parametric one-way analysis of variance (ANOVA) was used to

test for any significant differences between means when correlations were significant and post hoc test was done with Student-Newman-Keuls test. Multiple regression analysis was performed for correlation between continuous variables AST level, total bilirubin, alkaline phosphatase (ALP), MELD, Mayo Risk Score, and LS with liver parenchymal signal, enhancement characteristics, and ductal findings. All statistical analysis was performed on MedCalc Statistical Software version 16.4.3 (MedCalc Software bvba, Ostend, Belgium).

Results

A total of 55 livers and 439 out of 495 possible liver segments were evaluated (Table 2). Fifty-six liver segments were excluded as those segments were not included on at least one of the 4 MRE slices or the segments were atrophic and too small to accurately measure LS. All MRI sequences were available for evaluation of parenchymal signal intensity and morphological abnormalities. The caudate lobe and segment II and segment IVB were the most common liver segments not included in the MRE sections and hence not available for evaluation. Hypertrophy of the segment was most commonly present in caudate lobe and atrophy most commonly seen in right lobe segments VI-VIII.

Bile duct abnormalities

Evaluation of biliary tree including extrahepatic and intrahepatic segmental bile ducts was possible in all 55 patients using a combination of MRCP and coronal/axial SSFSE images. Strictures were present in 20 CBDs, 33 RMDs, 36 LMDs, and 141 segmental ducts. The grades of strictures, ductal dilatation, wall thickening, and enhancement are detailed in Table 3. Severe strictures were the most common grade of strictures in all the bile ducts.

Liver parenchyma features

T2W hyperintensity was present in 141 segments, DWI hyperintensity in 97 segments, arterial phase hyperenhancement in 140 segments, portal venous phase hyper-enhancement in 71 segments, and delayed phase hyperenhancement in 82 segments. Seventy-one segments showed atrophy and 93 segments showed hypertrophy and no volumetric change in the remaining 275 segments. Spearman rank correlation analysis showed significant correlation between segmental T2 hyperintensity, DWI hyperintensity, arterial phase hyperenhancement, portal phase hyperenhancement, and delayed phase hyperenhancement (all $p < 0.001$) (Table 3). Segmental strictures showed significant correlation with T2 hyperintensity and arterial phase hyperenhancement only. However, segmental ductal dilatation showed significant correlation with the T2 hyperintensity and hyperenhancement in all 3 phases. Segmental ductal wall thickening and enhancement correlated with T2 hyperintensity only. Segmental strictures, dilatation, and duct wall thickening and enhancement correlated significantly (all $p < 0.001$). Chi-square analysis showed significant association of the presence of segmental strictures with T2W hyperintensity ($p < 0.001$), and arterial phase hyperenhancement of parenchyma ($p < 0.001$) of the segments, whereas DWI hyperintensity ($p = 0.09$), portal venous phase ($p = 0.54$), and delayed phase enhancement ($p = 0.22$) did not show significant association.

Segmental LS

Segmental LS significantly correlated with the presence of strictures and there was no significant correlation with segmental ductal dilatation, thickening and/or enhancement. Significant correlation was present between grade of strictures and mean segmental LS (Table 4). Segmental LS was significantly higher when strictures (6.86 vs. 5.20 kPa, $p < 0.001$) or ductal enhancement (6.40 vs. 5.50, $p = 0.02$) were present (Fig. 2). Further analysis showed significant differences in LS between grade 2 strictures and other grade of strictures. When grouped as $< 50\%$ and $> 50\%$ strictures, there were significant differences in mean LS (6.5 vs. 5.5 kPa, $p = 0.01$). Segmental LS also correlated with atrophy and hypertrophy of the segments. LS of atrophic (Fig. 3) and hypertrophic segments (Figs. 4, 5) were significantly higher LS than normal segments (7.07 and 6.67 vs. 5.10 kPa, $p < 0.001$).

Segmental LS and parenchymal changes

Segmental LS correlated positively with T2W hyperintensity and DWI hyperintensity hyperenhancement in arterial portal venous and delayed phases (Table 4). Segmental LS was significantly higher when segments showed T2W hyperintensity (Fig. 2) (7.20 vs. 4.96 kPa, $p < 0.01$), DWI hyperintensity (7.40 vs. 5.30 kPa, $p < 0.01$), portal venous phase hyperenhancement (7.20 vs. 5.50 kPa, $p < 0.001$), and delayed phase hyperenhancement (6.94 vs. 5.50 kPa, $p = 0.001$). Segmental LS was higher when arterial hyperenhancement (Fig. 2) was present but did not reach statistical significance (6.24 vs. 5.50 kPa, $p = 0.05$).

With multiple regression analysis, the presence of strictures ($p = 0.001$) and T2W signal hyperintensity ($p = 0.009$) showed significant correlation with segmental LS.

Lobar LS

The mean $\pm$ SD right lobe LS was 6.03 ± 3.7 kPa (range 1.97–16.40 Pa) and the mean $\pm$ SD left lobe LS was 5.30 ± 3.0 kPa (range 1.72–13.60 kPa) with no significant differences. The left lobe LS significantly correlated with the presence of LMD stricture ($r = 0.35$, $p < 0.01$), but there was no significant correlation with LMD dilatation thickening or enhancement (Table 4).

There was a significant correlation between mean left lobe LS and LMD stricture grade ($r = 0.41$, $p = 0.02$). The mean left lobe LS was significantly higher when LMD stricture was present (6.00 vs. 3.90 kPa, $p = 0.02$). On multiple regression analysis, only LMD stricture significantly correlated with left lobe LS. There was no significant correlation between right lobe LS with RMD stricture, dilatation, wall thickening, or enhancement (Table 4). However, there was some significant correlation with right lobe LS and grade of RMD stricture. Also the mean right lobe LS was higher when RMD stricture was present but did not reach statistical significance (6.76 vs. 4.92 kPa, $p = 0.07$).

Global LS

Global LS ranged from 2.01 to 14.1 kPa with a mean $\pm$ SD of 5.64 ± 3.3 kPa. Global LS positively correlated with CBD stricture, number of segmental strictures (Fig. 1), spleen stiffness, splenomegaly, and varices and negatively correlated with CBD dilatation. There was no significant correlation between LS and CBD wall enhancement or wall thickening

(Table 2). Non-parametric analysis did not show significant differences between global LS and different grades of CBD stricture ($p = 0.05$) or when stricture present or absent ($p = 0.07$). Similarly, there were no significant differences in global LS with increasing number of segmental strictures ($p = 0.09$). In addition, there was no correlation between LS and ALP, AST, TB, MELD score, and Mayo Risk score. Global LS was significantly higher when splenomegaly (7.70 vs. 3.90 kPa, $p = 0.02$) or varices (9.46 vs. 4.6 kPa, $p < 0.001$) were present. In multiple regression analysis, only splenomegaly and varices showed significant correlation with global LS. A LS of > 4.00 kPa predicted splenomegaly with accuracy of 0.83, sensitivity of 92%, and specificity of 70%. Similarly, a LS > 6.27 kPa predicted varices with an accuracy of 90%, sensitivity of 100%, and specificity of 76.7%. Mayo risk score and MELD score did not demonstrate any significant correlation with the presence of ductal enhancement, number of ducts showing enhancement, or number of segmental ductal strictures.

Discussion

This study evaluated the relationship between hepatic parenchymal changes, biliary features, and liver stiffness in patients with PSC at a segmental, lobar, and global level. Parenchymal changes within the liver included hyperintensity on T2W and DW images as well as hyperenhancement on post-contrast images.

Our study shows correlation between segmental biliary ductal abnormalities with parenchymal changes and LS. Segmental strictures and not segmental dilatation correlated with LS and parenchymal changes. CBD strictures were not significantly associated with global LS suggesting that segmental strictures are associated with parenchymal changes and therefore parenchymal stiffness. The lack of a strong correlation between liver stiffness and other biliary changes (dilatation and enhancement) likely reflect underlying fibrosis as the cause rather than a clinically significant increase in liver stiffness from biliary obstruction.

We also found that both atrophy and hypertrophy caused significantly higher LS in affected hepatic segments. Segmental atrophy in the setting of biliary obstruction caused by PSC is likely seen as a late finding on MRI and the expected fibrotic changes in these segments likely account for the elevated LS. Perhaps the finding of increased LS in hypertrophic segments is counterintuitive. However, segmental hypertrophy seen on a morphologic level is likely secondary to liver regeneration in response to injury and there is likely to be both fibrosis and hyperplasia in these segments which could explain the increased LS.

At the lobar level, stricture of the LMD correlated with increased LS in the left hepatic lobe; however, the presence of RMD stricture was not associated with statistically significant increase in LS in the right hepatic lobe. This may be due to the heterogeneous distribution of PSC in the biliary tree in both small and large ducts. Perhaps the segmental strictures are the dominant contributor to LS rather than the larger ducts such as RMD and LMD.

Alternatively, proportion of atrophic and hypertrophic segments with elevated LS could be affecting the correlation. In our study, the proportion of atrophic and hypertrophic segments in the right and left lobe were significantly different (30% vs. 45%). These findings

highlight the complex relationship of liver stiffness with parenchymal changes, signal abnormalities, and biliary changes.

Global LS significantly correlated with secondary signs of portal hypertension which suggests a role of LS for an overall severity assessment as currently used in other chronic liver diseases. Global LS did not significantly correlate with any of the laboratory tests or MELD and Mayo risk score. Earlier studies have shown variable association of parenchymal changes and LS with Mayo risk score [13, 21]. The possible explanation for our study results could be due to different population and also smaller number of patients included in our study. Previous studies have attempted to correlate MRCP and MRI findings with clinical severity scores and showed variable correlation with clinical severity scores. In one study, Petrovic et al. [22] studied 47 patients for the extent of anatomical changes in biliary tree and degree of peribiliary enhancement and compared them to Mayo Risk score. Similar to our study, they demonstrated no significant relationship between anatomical and enhancement changes, and Mayo Risk score. Bader et al. [13] studied 52 patients with PSC and correlated imaging pattern of liver parenchyma, presence and grade of intrahepatic biliary ductal dilatation, atrophy and abnormal signal intensity of post-gadolinium enhancement of the parenchyma with Child class, Child-Turcotte-Pugh score, and MELD scores and found no significant correlation with the score.

In another study, Ni Mhuirheartiagh et al. [23] studied 60 patients and showed that the presence of arterial peribiliary enhancement in patients with PSC is associated with higher Mayo risk scores and may suggest poorer prognosis. They postulated that arterial peribiliary enhancement could be attributed to on-going biliary inflammation and therefore a worse prognosis. In addition, they hypothesized that delayed peribiliary hyper-enhancement might reflect a more chronic or fibrotic form of PSC and may not affect survival as disease is likely more stabilized in these patients. In our study, we evaluated the periductal enhancement as present or absent in any of the three phases and we did not demonstrate any significant correlation. Our study results support the findings of previous studies by Petrovic et al. and Bader et al. that biliary ductal abnormalities and parenchymal signal abnormalities do not correlate with clinical severity scores such as MELD and Mayo Risk score. The possible reasons are due to complex changes occurring in PSC and the biliary ductal abnormalities do not necessarily reflect the overall liver dysfunction as normal segments can compensate for liver function except in advanced cases. However, the studies are not uniform in the imaging features evaluated and therefore a larger study is required for evaluation of these abnormalities.

Progression of liver fibrosis is the single most important determinant of outcome in primary sclerosing cholangitis as they can predict development of portal hypertension and other complications. Surrogate markers for liver fibrosis are therefore essential for the management. MRE may be useful in detection of early fibrosis in PSC especially when there are no other morphological signs of disease [24]. Previous studies have shown the utility of MRE in the assessment of the presence or absence of cirrhosis and patient outcomes in PSC. Liver stiffness measurement at baseline and longitudinal changes has been shown to be useful biomarkers for prognostication of these patients [16, 21].

There are some limitations to our study. The relatively small number of patients included in this study over a wide range of clinical stages does limit the evaluation of relationship between LS and changes occurring in the disease over the course of time. As the study was performed with a 2D MRE technique, several segments had to be excluded from analysis. 3D MRE technique may be useful to assess the changes in all segments and global changes in LS. Future studies are required to establish our findings of correlation of segmental LS with strictures. Confirmation of findings would help understand the pattern of progression of the disease and a method to correlate with liver stiffness and therefore severity of the disease. We did not have a reference standard such as histology for confirmation of our findings; however, liver biopsy is not routinely performed for diagnostic or management in PSC patients. However, we used the standard MRI sequences and MRE is the most accurate method to evaluate liver stiffness. In this study, we did not use confidence maps as our cases were recruited from 2007 to 2013 when confidence maps were not available for clinical purposes. Use of confidence maps in cases performed with 2D MRE may limit the number of segments available for evaluation. The use of 3D MRE may help in the future to evaluate more liver parenchyma when potentially more liver volume will be assessed.

In summary, our preliminary study shows low correlation between segmental liver stiffness with segmental parenchymal signal changes and the presence of segmental ductal strictures suggesting a possible role for MRE for evaluation of disease severity. Global LS is useful to predict varices and splenomegaly. Future studies are required to confirm our findings and also show its utility in clinical follow-up.

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Ethical approval This is a retrospective study and an Institutional Review Board approval was obtained for review of patient data.

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Fig. 1.

Example illustrating ROI placement and correlation with routine MRI images. MRE magnitude images (**A, D**) through top and middle part of the liver and corresponding stiffness maps in gray scale (**B, E**). Regions of interest (dotted circles) have been drawn in each of the nine Couinaud segments in the gray-scale stiffness maps. T2W axial image (**C**) corresponding to the magnitude image (**A**) and DWI axial image (**F**) corresponding to magnitude image (**C**) used for correlation of signal intensity.

Fig. 2.

Increased global liver stiffness with multiple segmental strictures. A 20-year-old female with PSC: Radial single-shot spin echo sequence image (A) shows multiple strictures of intrahepatic ducts, right and left main ducts, and intrahepatic ducts in both lobes. Corresponding axial sections on T2W (B), DWI (C), arterial Phase (D), and delayed phase (E) images. T2W and DWI images do not show any significant parenchymal signal abnormalities except a subtle patchy hyperintensity on T2W images. Heterogeneous hyperenhancement of the parenchyma in the arterial and delayed phases. Note dilated ducts in both lobes. MRE stiffness map (0–20 kPa scale) (F) shows diffusely stiff liver with heterogeneous distribution of stiffness in both right and left lobes and a mean stiffness of 12.8 kPa consistent with cirrhosis.

Fig. 3.

Atrophic lobe showing increased stiffness. A 60-year-old male with PSC. MRCP maximum intensity projection (MIP) image (**A**) showing multiple strictures of both intrahepatic and extrahepatic ducts with a tight long stricture (arrow) affecting posterior right lobe.

Corresponding level sections showing hyperintensity (arrows) on T2W (**B**) and DWI (**C**) images in the posterior right lobe that is mildly atrophic with compensatory hypertrophy of the left lobe. Hyperenhancement is observed in the same lobe in the arterial (**D**) and delayed (**E**) phases. Focally increased liver stiffness is observed on MRE (**F**) in the same lobe. Note mildly increased liver stiffness in the rest of the liver.

Fig. 4.

Hypertrophied caudate lobe showing increased stiffness. A 65-year-old with PSC and cirrhosis with portal hypertension. MRCP 3D MIP (**A**) showing multiple intrahepatic biliary strictures and right and left main hepatic ducts. Axial T2W image (**B**) and DWI image (**C**) shows normal signal intensity of the right anterior lobe and hypertrophied caudate lobe (*). Arterial phase image (**D**) shows diffuse hyperenhancement of liver except caudate lobe. Delayed image (**E**) shows normal signal intensity in the right anterior lobe and caudate lobe. MRE (**F**) shows diffuse increased liver stiffness throughout the liver including caudate lobe. Incidental finding of multiple intraductal papillary mucinous neoplasms of the pancreas.

Fig. 5.

Hypertrophied lobes showing only mildly increased stiffness. A 55-year-old male with PSC. MRCP image (A) showing multiple intrahepatic bile duct strictures and lobar duct strictures. Axial T2W (B) and DWI (C) images showing mild peripheral hyperintensity with hypertrophy of inferior left lobe (*) and caudate lobes. There is peripheral hyperenhancement in post-contrast-enhanced arterial (D), and delayed (E) phases with no hyperenhancement in hypertrophied segments (*). MRE (F) showing increased stiffness more in the peripheral right lobe and only mildly increased stiffness in hypertrophied caudate and left lobe (*).

Table 1.

Liver MRI, MRCP, and MRE protocol parameters at 1.5T and 3T scanners

Field strength	Sequence	Plane	Mode	FOV (mm)	TR (ms)	TE (ms)	ST/gap (mm)	Matrix	NEX
1.5T	SSFSE	Coronal	2D	440	2000	85	5/0	384 288 *	1
	SSFSE	Axial	2D	360–400	2000	85	5/2	256 224 *	1
	3DMRCP RT	Coronal	3D	340	RT	Min	1.6/0	320 320 *	1
	3D MRCP BH	Coronal	3D	400	1880	956	3/0	256 160 *	1
	T2FSE	Axial	2D	360–400	RT	>85	5–6/1	320 224 *	2
	DWI	Axial	2D	400	RT	Min	6/1	128 128 *	1
	In/Opp	Axial	2D	360–400	125–200	Min Full	5/1	256 192 *	1
	LAVA-pre	Axial	3D	360–400	3.8	Min	3–4/0	256 224 *	1
	LAVA post [^]	Axial	3D	360–400	3.8	Min	3–4/0	256 224 *	1
	2D-GREMRE	Axial	2D	420	50	18–23	10	192 128 *	1
3T	SSFSE	Coronal	2D	440	2000	85	5/0	384 288 *	1
	SSFSE	Axial	2D	360–400	2000	85	5/2	384 256 *	1
	3DMRCP RT	Coronal	3D	340	RT	655	1.6/0	320 320 *	1
	3D MRCP BH	Coronal	3D	400	1900	800	3/0	256 160 *	.5
	T2FSE	Axial	2D	360–400	RT	> 85	5–6/1	320 320 *	2
	DWI	Axial	2D	400	RT	Min	6/1	128 128 *	1
	In/Opp	Axial	2D	360–400	110–250	Min Full	5/1	256 192 *	1
	LAVA-pre	Axial	3D	360–400	4	Min	3–4/0	320 256 *	1
	LAVA post [*]	Axial	3D	360–400	4	Min	3–4/0	320 256 *	1
	2D-GREMRE	Axial	2D	420	50	15–18	10	256 64 *	1

* All scans were performed on GE scanners

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FOV Field of view, *TR* time to repeat, *TE* time to echo, *ST* slice thickness, *NEX* number of excitations, *SSFSE* single-shot fast spin echo, *3D* three dimensional, *MRCP* magnetic resonance cholangiopancreatography, *RT* respiratory triggered, *BH* breath hold, *FSE* fast spin echo, *DWI* diffusion-weighted imaging; *In Opp* in-phase and opposed phase sequence, *LAVA* liver acquisition with volume averaging;

✓ acquired in late arterial phase (35 s), portal venous phase, (65–70 s) and delayed phases (3–5 min); *2D-GRE/MRE* two-dimensional gradient recalled echo magnetic resonance elastography

Table 2.

Bile duct features on MRCP and MRI in 55 patients with PSC

Duct	Common bile duct	Right main duct	Left main duct	Segmental ducts
Strictures				
0: none	20	33	36	140
1: 0–25%	3	3	7	22
2: 25–50%	4	2	–	10
3: 50–75%	1	8	12	43
4: 75–100%	12	20	17	66
Dilatation	11	6	10	131
Duct wall thickening	7	4	5	120
Duct wall enhancement	29	24	25	120

Table 3.

Correlation between signal intensity of liver segments and bile duct abnormalities

Feature	T2 hyperintensity	DWI hyperintensity	Arterial phase enhancement	Portal phase enhancement	Delayed phase enhancement	Segmental duct stricture	Segmental duct dilatation	Segmental duct thickening/enhancement
T2 hyperintensity		0.568 (< 0.001)	0.370 (< 0.001)	0.540 (< 0.001)	0.511 (< 0.001)	0.214 (< 0.001)	0.144 (0.002)	0.175 (< 0.001)
DWI hyperintensity	0.568 (< 0.001)		0.213 (< 0.001)	0.482 (< 0.001)	0.379 (< 0.001)	0.084 (0.078)	-0.038 (0.428)	0.031 (0.522)
Arterial phase enhancement	0.370 (< 0.001)	0.213 (< 0.001)		0.363 (< 0.001)	0.362 (< 0.001)	0.199 (< 0.001)	0.175 (< 0.001)	0.096 (0.045)
Portal phase enhancement	0.540 (< 0.001)	0.482 (< 0.001)	0.363 (< 0.001)		0.774 (< 0.001)	0.049 (0.305)	0.185 (< 0.001)	-0.089 (0.062)
Delayed phase enhancement	0.511 (< 0.001)	0.379 (< 0.001)	0.362 (< 0.001)	0.774 (< 0.001)		0.083 (0.082)	0.243 (< 0.001)	0.047 (0.326)
Segmental duct stricture	0.214 (< 0.001)	0.084 (0.077)	0.199 (< 0.001)	0.049 (0.305)	0.083 (0.082)		0.519 (< 0.001)	0.343 (< 0.001)
Segmental duct dilatation	0.144 (0.002)	-0.038 (0.428)	0.175 (< 0.001)	0.185 (< 0.001)	0.243 (< 0.001)	0.519 (< 0.001)		0.216 (< 0.001)
Segmental duct thickening/enhancement	0.175 (< 0.001)	0.031 (0.522)	0.096 (0.045)	-0.089 (0.062)	0.047 (0.326)	0.343 (< 0.001)	0.343 (< 0.001)	

Spearman correlation analysis; *p* values in parenthesis

Table 4.
Segmental, lobar, and global liver stiffness and its correlation with MRI and MRCP features

Liver stiffness	Feature/parameter	Spearman correlation rho (95% CI)	p
Segmental	T2 hyperintensity	0.38 (0.30 to 0.46)	<0.001
	DWI hyperintensity	0.30 (0.21 to 0.38)	<0.001
	Arterial phase hyperenhancement	0.12 (0.03 to 0.21)	0.01
	Portal phase hyperenhancement	0.28 (0.19 to 0.37)	0.28
	Delayed phase hyperenhancement	0.26 (0.17 to 0.25)	<0.001
	Segmental atrophy	0.21 (0.12 to 0.30)	<0.001
	Segmental hypertrophy	0.18 (0.9 to 0.27)	0.001
Right lobe	Segmental duct stricture (present/absent)	0.18 (0.09 to 0.30)	0.001
	Segmental duct stricture grade	0.17 (0.08 to 0.26)	<0.001
	Segmental duct dilatation	0.07 (−0.02 to 0.16)	0.14
	Segmental duct wall thickening/enhancement	0.06 (−0.03 to 0.16)	0.18
	RMD stricture (present/absent)	0.26 (−0.01 to 0.49)	0.06
	RMD stricture grade	0.27 (0.01 to 0.50)	0.047
	RMD dilatation	−0.21 (−0.45 to 0.05)	0.11
Left lobe	RMD wall thickening	−0.04 (−0.31 to 0.22)	0.74
	RMD wall enhancement	−0.05 (−0.31 to 0.21)	0.70
	LMD stricture (present/absent)	0.35 (0.09 to 0.56)	0.01
	LMD stricture grade	0.41 (0.16 to 0.61)	0.02
	LMD dilatation	−0.06 (−0.32 to 0.21)	0.64
	LMD wall thickening	0.10 (−0.17 to 0.36)	0.45
	LMD wall enhancement	−0.06 (−0.32 to 0.21)	0.66
Global (whole liver)	CBD stricture	0.31 (0.05 to 0.53)	0.02
	CBD stricture grade	0.34 (0.08 to 0.55)	0.01
	CBD dilatation	−0.36 (−0.57 to −0.10)	<0.001
	CBD wall thickening	0.13 (−0.14 to 0.38)	0.35
	CBD wall enhancement	−0.03 (−0.30 to 0.23)	0.80
	Splenomegaly	0.56 (0.35 to 0.72)	<0.001
	Varices	0.58 (0.37 to 0.73)	<0.001

$p < 0.05$ if P values considered interval. $CI95\%$ confidence interval.

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