Article published online: 2024-05-16

Review Article

Magnetic Resonance Elastography of Liver: Current Status and Future Directions

Argha Chatterjee[1] Frank H. Miller[2] Emily Pang[3]

[1] Department of Radiology, Tata Medical Center, Kolkata, West Bengal, India
[2] Department of Radiology, Northwestern Memorial Hospital, Northwestern University Feinberg School of Medicine, Chicago, Illinois, United States
[3] Department of Abdominal Imaging, Vancouver General Hospital, University of British Columbia, Vancouver, British Columbia, Canada

Address for correspondence Argha Chatterjee, MD, PDCC, Department of Radiology, Tata Medical Center, Kolkata, West Bengal 700156, India (e-mail: arghachat84@gmail.com).

J Gastrointestinal Abdominal Radiol ISGAR

Abstract

Chronic liver disease (CLD) has been recently recognized as a major public health priority. Assessment of presence and degree of liver fibrosis is critical to the management of CLD and traditionally required a liver biopsy. However, biopsy has many limitations including the risk of complications and sampling error. Magnetic resonance elastography (MRE) has emerged as a noninvasive and highly accurate technique for evaluating liver fibrosis. In this comprehensive review, we will delve into the current uses and guidelines for the usage of MRE in CLD, highlighting its advantages and limitations.

Keywords
- liver fibrosis
- metabolic dysfunction-associated steatohepatitis
- quantitative imaging

Introduction

Chronic liver disease (CLD) is a global health concern with a significant impact on morbidity and mortality.[1] In India, it has been recently recognized as a major public health priority; India contributed to 18.3% of the global liver disease related deaths in 2015.[2] Different CLDs and cirrhosis accounted for 2.1% of all deaths in India in 2016.[3] Assessment of presence and degree of liver fibrosis is critical to the management of CLD. Biopsy is still the gold standard to diagnose and quantify liver fibrosis. However, it is invasive and associated with many limitations such as inherent disease heterogeneity, sampling error, inter- and intrareader variabilities, risk of complications, and potential challenges with patient compliance. Magnetic resonance elastography (MRE) has emerged as a noninvasive and highly accurate technique for evaluating liver fibrosis.[4] Liver fibrosis is a result of excessive extracellular matrix protein (such as collagen) deposition and leads to increased tissue stiffness. This stiffness can be quantified on MRE by utilizing a phase contrast technique to determine the velocity of shear waves that are propagated through liver tissue by a mechanical external driver; the measured shear wave velocity increasing as the tissue stiffness increases.[4] In this comprehensive review, we will delve into the current uses and guidelines for the usage of MRE in CLD, highlighting its advantages and limitations.

Technique of MRE

Basic Principle

Different pathological processes alter the viscoelastic properties of the tissue. It is shown that tissue elasticity varies over a much wider range of values in different pathologies than other commonly measured parameters such X-ray attenuation or T1 relaxation time.[5] This potentially offers accurate diagnosis and grading of pathological processes by the measurement of tissue elasticity. Elastography offers

DOI https://doi.org/10.1055/s-0044-1786812.
ISSN 2581-9933.

Thieme Medical and Scientific Publishers Pvt. Ltd., A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

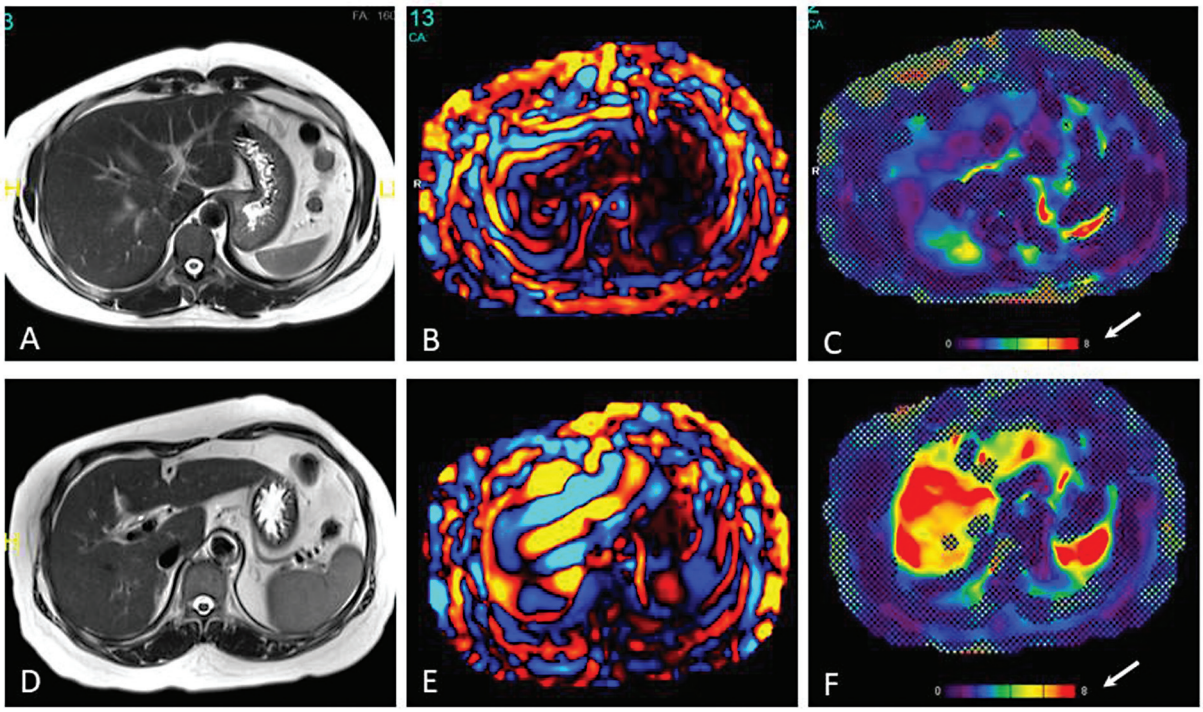

Fig. 1 Principle of MR elastography. (**A**) T2W image of a normal liver. (**B**) Wave map generated in the normal liver shows smaller waves and, hence, slower wave progression. (**C**) Corresponding stiffness map shows predominantly blue–purple color suggestive of low LSM. Notice the color scale at the bottom of the image (arrow). (**D**) T2W image of another patient shows cirrhotic liver. (**E**) Corresponding wave map shows larger waves suggesting faster wave propagation. (**F**) Corresponding stiffness map shows predominantly red–yellow color suggestive of high LSM. LSM, liver stiffness measurement; MR, magnetic resonance; T2W, T2-weighted.

noninvasive assessment of tissue elasticity.[6] In clinical use, measurement of liver elasticity is called liver stiffness measurement (LSM). Currently, liver stiffness evaluation with ultrasound and MRE are widely used in clinical practice. MRE is currently regarded as the most accurate noninvasive diagnostic tool for detection and staging of liver fibrosis.[7,8] MRE can be easily included within the routine magnetic resonance imaging (MRI) of the liver.[9]

MRE measures tissue stiffness by analyzing the propagation of shear waves through the liver. Waves travel faster in stiffer tissue; thus, in the case of the liver, the velocity of shear waves is directly proportional to the liver stiffness via the complex shear modulus (►**Fig. 1**). The most commonly used MRE pulse sequence is a phase contrast two-dimensional gradient-recalled echo (2D GRE MRE) sequence. The shear waves are generated mechanically using a passive driver that is connected to an active pneumatic mechanical driver stationed outside the MRI gantry. The passive driver is placed on the right lower chest wall of the patient, approximately overlying the liver (►**Fig. 2**). The active driver produces continuous acoustic vibrations at a fixed frequency of 60 Hz that are transmitted to the passive driver which then transmits the waves to the entire abdomen, including the liver. These acoustic waves produce microscopic shear waves within the tissue, which is imaged by the motion-sensitive MRE sequence as microscopic tissue displacement to produce a wave image. Typically, four slices of 5 to 10 mm thickness through the largest cross-section of the liver are obtained, avoiding the lung, dome of the liver, and the inferior portion of the liver, as slices through these regions of liver can result in a high LSM.

Image Interpretation

For every slice, the MRE sequence produces six sets of images. As with any phase contrast sequence, a *magnitude image* delineating the anatomy of the imaged section of upper abdomen and a *phase contrast* image showing propagating shear waves at the same level are immediately reconstructed. An inversion algorithm automatically processes these images to further produce gray-scale and colored stiffness maps which depict the shear modulus of the different regions of the liver. A confidence map is overlaid on the stiffness maps where regions with unreliable data are excluded. In addition, a colored wave map is also available (►**Fig. 3**). To obtain LSM data, a region of interest (ROI) needs to be drawn on the confidence map. The ROI needs to be as large as possible while avoiding the liver edge, artifacts, fissures, gallbladder fossa, large vessels, and regions of wave interference. The mechanical property measured with MRE is the magnitude of the complex shear modulus expressed in kilopascals (kPa). Arithmetic mean of the LSM value from the ROIs drawn on the four slices is reported as the mean LSM value.

Patient Preparation

- Fasting: About 4 to 6 hours of fasting before the examination is recommended. In patients with CLD, possibly

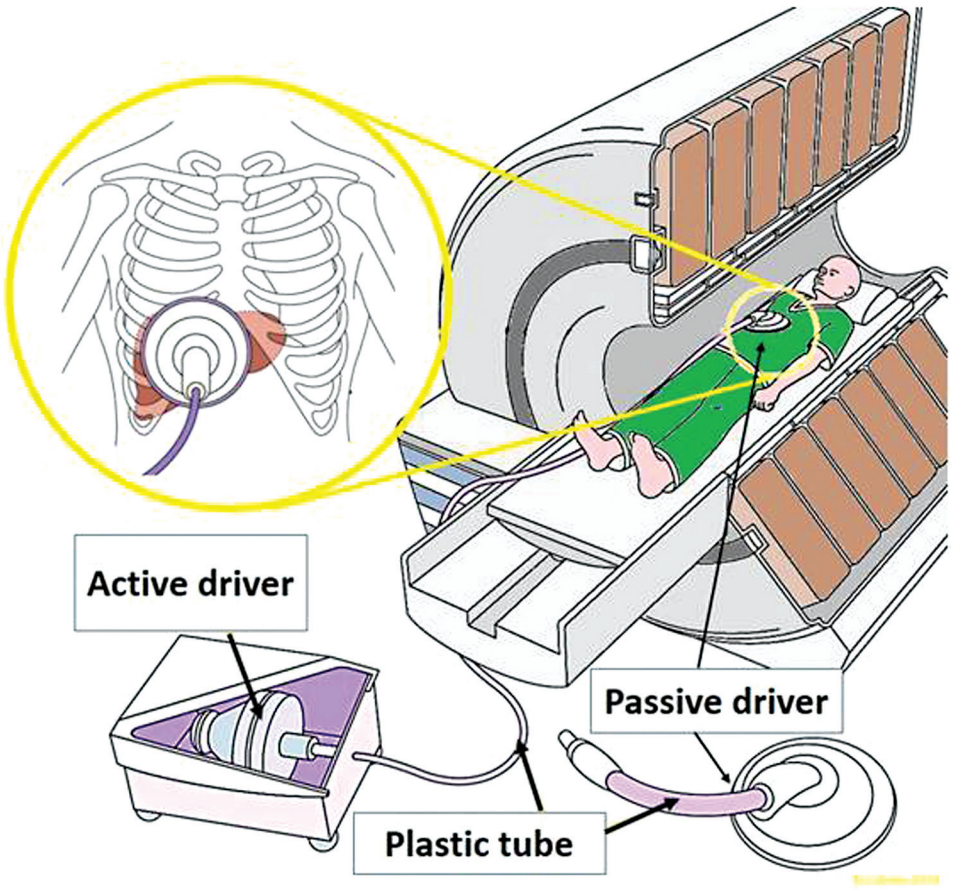

Fig. 2 *Diagram showing MR elastography setup*. The patient lies supine inside the MR gantry. The passive driver is placed on the patient's body, and it is connected to the active driver via a plastic tube. The active driver is placed outside the MR room. Inset, the passive driver is placed on the right lower anterior chest wall approximately covering the area of the liver. MR, magnetic resonance. (Image courtesy: David Botos.)

owing to high portal venous inflow after meals, spuriously high LSM may be obtained.[10]

- Breath-hold at end expiration: Clinical MRE sequences are acquired in breath-hold; the patient is instructed to hold her breath in end-expiration. The rationale for this is because the end-expiration position offers better reproducibility and additionally liver stiffness tends to increase with deep inspiration.
- If necessary MRE can be performed after gadolinium administration as the use of gadolinium-based contrast agents does not affect the stiffness measurements.[11]

Newer Advances in MRE Technology

- A rectangular flexible and soft pneumatic passive driver has been developed recently that can cover a larger area of the liver and showed similar LSM compared with a conventional driver.[12] This is also more comfortable for the patient.
- A spin echo (SE) echo-planar imaging (EPI)-based MRE (SE EPI MRE) sequence is now available commercially that is faster and much less susceptible to liver iron deposition-related signal loss compared with standard 2D GRE MRE.[13,14] A nongated, free-breathing, single-shot, multislice 2D EPI MRE technique with a view-sharing-based reconstruction strategy was recently developed that can be used in patients who have difficulty with breath holds.
- The conventional MRE sequence is 2D MRE, meaning in the case of shear wave velocity measurements, it detects motion in one plane and assumes that shear waves are propagating through the axial plane of acquisition. However, this assumption may lead to overestimation of LSM close to the dome and inferior margin of liver where the waves travel more obliquely. A SE-based three-dimensional MRE sequence (3D MRE) sensitized to detect motion in all three planes can correct for this. It samples a larger tissue volume and has a lower failure rate.[15,16] LSM obtained by 3D MRE may be slightly lower than those obtained with conventional sequence.[17]

MRE Indications

- Hepatic fibrosis and CLD:
  - *Pathophysiology*: Liver fibrosis is the final common pathway to a multitude of hepatic injuries such as alcohol,

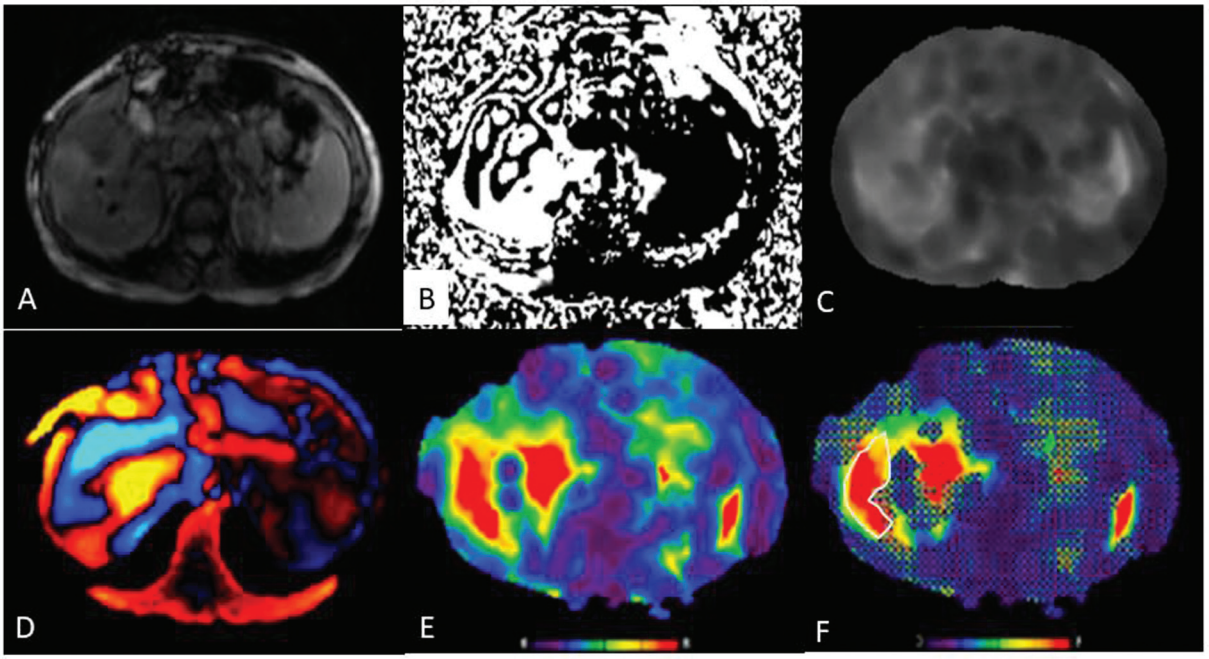

Fig. 3 Routine set of MRE images. (**A**) Magnitude image: Ensures proper passive driver position and provides anatomical image. (**B**) Phase image: Assessment of quality of wave progression and identification of areas of interference. (**C**) Grayscale stiffness map: Can be used to place the ROI for calculation of stiffness. (**D**) Wave image: Assessment of quality of wave progression and identification of areas of interference. (**E**) Color stiffness map: Global view of stiffness of the section on a color scale. (**F**) Confidence map: Areas where stiffness measurement is reliable. Used for measurement of LSM. LSM, liver stiffness measurement; MRE, magnetic resonance elastography; ROI, region of interest.

metabolic dysfunction-associated fatty liver disease (MAFLD, previously known as nonalcoholic fatty liver disease), viral infection, and other toxic and inflammatory insults. Once fibrosis is advanced, it can result in cirrhosis, which in turn can cause portal hypertension and is associated with an increased risk of hepatocellular carcinoma (HCC).

- *Imaging indications*: Liver fibrosis is the single most important factor that determines the clinical outcome in CLD. Early treatment of liver fibrosis is associated with better outcomes; therefore, early detection and gradation of liver fibrosis are crucial.[18,19] The current gold standard for the diagnosis of liver fibrosis is liver biopsy. Biopsy is associated with a small but definite risk of complications, such as pain and bleeding, and it is limited by sampling error and low intra- and interobserver agreements.[20] It also has limited patient acceptance, high cost, and is impractical for serial follow-up. Noninvasive tests measure surrogate markers for the presence of liver inflammation and/or fibrosis. A combination of direct and indirect biomarkers is used clinically in the form of indices such as FibroSure, Hepascore, aspartate aminotransferase-to-platelet ratio index and fibrosis-4 score which are most useful for distinguishing cirrhosis from early-stage fibrosis, but are not useful for diagnosis or differentiation of early stages of fibrosis.[21,22] Furthermore, biopsy or blood test cannot detect complications (portal hypertension, ascites, and HCC). Imaging with elastography offers simultaneous detection and semiquantitative assessment of liver fibrosis as well as detection of complications. MRE shows higher accuracy in detecting fibrosis compared with the semantic or anatomic features detected by conventional cross-sectional imaging.[23]

  Liver stiffness increases with increased stage of fibrosis.[24] MRE is especially accurate in the diagnosis of severe fibrosis (grades 3 and 4).[24,25] There is no consensus on cutoff values for diagnosis and staging of liver fibrosis. A widely used guideline for approximate cutoff values is given in ►**Table 1**. MRE shows 98% sensitivity and 99% specificity for the diagnosis of liver fibrosis using a cutoff value of 2.93 kPa. MRE can distinguish between mild (F0–F2) and clinically significant fibrosis (F3–F4) with 86 to 91% sensitivity and 80 to 85% specificity.[26]
- *Viral hepatitis*: Detection and staging of fibrosis in patients with chronic hepatitis B and C are important as the presence of advanced fibrosis is an indication for

Table 1 General guideline for interpretation of LSM[27]

Mean LSM	Fibrosis stage
< 2.5 kPa	Normal
2.5–3.0 kPa	Normal or inflammation
3.0–3.5 kPa	Stages 1–2 fibrosis
3.5–4.0 kPa	Stages 2–3 fibrosis
4.0–5.0 kPa	Stages 3–4 fibrosis
> 5.0 kPa	Stage 4 fibrosis or cirrhosis

Abbreviation: LSM, liver stiffness measurement.

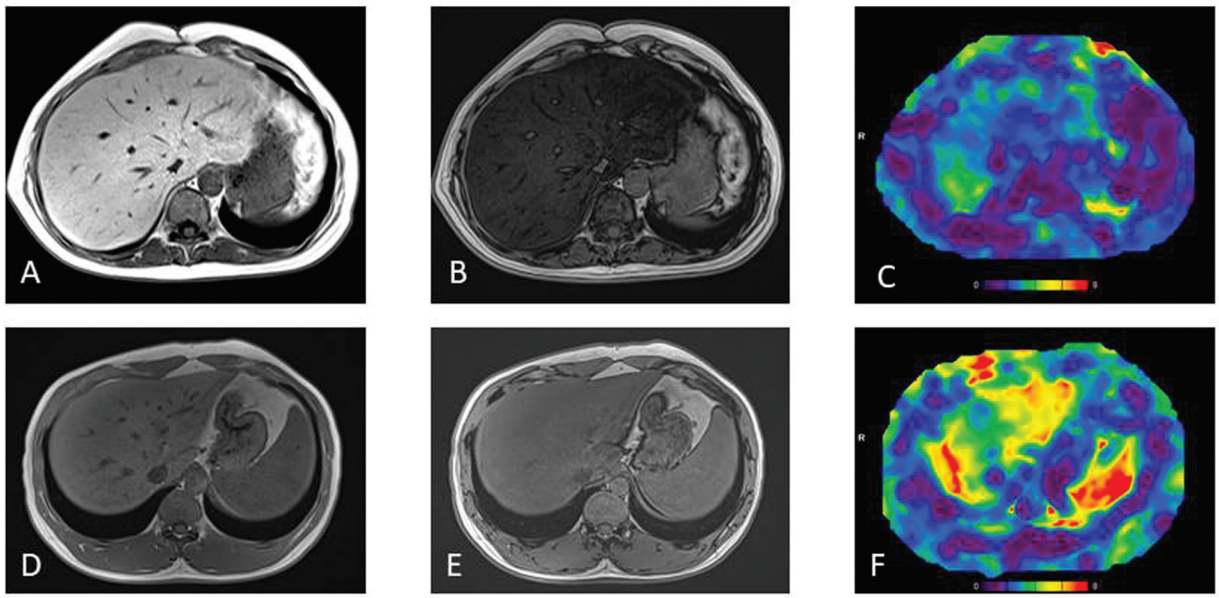

Fig. 4 *Metabolic dysfunction-associated fatty liver disease (MAFLD) versus metabolic dysfunction-associated steatohepatitis (MASH).* (**A, B**) In-phase (**A**) and opposed (**B**) phase T1-weighted images of a 43-year-old man shows severe fat deposition in liver (~50%). (**C**) Stiffness map shows normal liver stiffness of 2.6 kPa. These findings are suggestive of MAFLD. (**D, E**), In-phase (**D**) and opposed (**E**) phase T1-weighted images in a 32-year-old man shows mild fat deposition (12%). (**F**) However, stiffness map shows high liver stiffness measurement (5.1 kPa) suggesting advanced fibrosis indicating advanced MASH.

starting antiviral therapy and performing cancer surveillance.[28] MRE can predict disease progression in such patients.[29] LSM is useful in follow-up by documentation of regression of fibrosis.[19]

- *MAFLD*: MAFLD is currently the most common cause of CLD.[30] MAFLD is a spectrum of disease with metabolic-associated fatty liver (MAFL) at one end to metabolic dysfunction-associated steatohepatitis (MASH) at the other end which is associated with increased risk of mortality.[31,32] MAFL alone does not cause elevated liver stiffness but MASH increases LSM; 20 to 40% of patients with MASH progress to advanced fibrosis.[33] Early diagnosis of inflammation/fibrosis helps diagnose MASH. Approximately 30 to 60% patients of biopsy-proven MASH have normal alanine transaminase levels.[34] magnetic resonance (MR)-based hepatic fat fraction assessment is inaccurate in the presence of advanced fibrosis.[35] Therefore, simple fat quantification is not sufficient at all stages of MAFLD. Distinction between MAFL and MASH is crucial for risk stratification (►**Fig. 4**). MRE has high sensitivity and specificity for distinguishing MAFL from MASH, and MAFL from advanced fibrosis with area under the curve–receiver operating characteristic curve ranging from 0.92 to 0.96.[36–39] MRE outperforms clinical prediction rules in the diagnosis of advanced fibrosis in MAFLD.[40] The role of MRE in MAFLD is twofold, distinguishing MAFL from MASH and identification of patients with advanced fibrosis.
- *Other etiologies*: MRE can detect fibrosis in other etiologies of CLD such as primary sclerosing cholangitis, autoimmune hepatitis, or sarcoidosis[41] (►**Fig. 5**).
- *Follow-up*: As fibrosis is the major determinant of outcome in CLD, assessment of progression in patients on follow-up is important (►**Fig. 6**). MRE is well suited for longitudinal follow-up of patients with CLD.[41] A 20% change in mean LSM under identical conditions is a significant change in the LSM during longitudinal assessment.[42]
- *Clinical trials*: Several trials in MAFLD have used MRE-derived liver stiffness combined with proton density fat fraction as surrogate end points.[43,44]

- *Focal liver lesions*: Malignant liver tumors have a significantly higher mean LSM than benign tumors, fibrotic liver and normal liver[45] (►**Fig. 7**). Research into whether LSM may be able to distinguish between benign and malignant liver tumors is still at an early stage.
- LSM with MRE can be used for risk prediction for development of HCC in patients with CLD.[46] In patients with HCC and undergoing treatment, a LSM > 5.5 kPa is also predictive of early recurrence of HCC following treatment.[47]
- *Portal hypertension*: Patients with advanced fibrosis are at risk of developing portal hypertension. One of the most important complications of decompensated cirrhosis is esophageal variceal bleeding which has a high mortality rate. Several studies have shown the feasibility and utility of MRE in prediction of esophageal varices, either with LSM or spleen stiffness measurements.[48,49] Alternatively, MRE can also be used to rule out high LSM and liver fibrosis in cases of isolated massive splenomegaly (►**Fig. 8**). The absence of risk factors for CLD preserved liver function and mild to moderately elevated LSM (< 5 kPa) is useful information to suspect noncirrhotic portal hypertension.[50,51] High LSM has been shown to be associated with high risk of decompensation in patients with advanced liver fibrosis.[52] Baseline LSM can, therefore, be

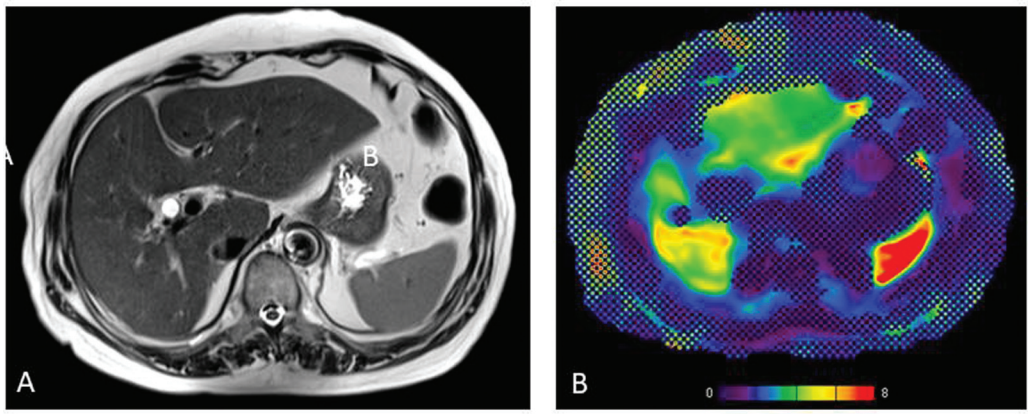

Fig. 5 *Autoimmune hepatitis in a 63-year-old woman.* (**A**) T2-weighted axial image shows liver enlargement with rounding of liver margin. (**B**) Confidence stiffness map shows liver stiffness measurement of 4.1 kPa suggesting advanced fibrosis. Biopsy showed moderate interface hepatitis with periportal fibrosis. Serum immunoglobulin G level and serum antinuclear antibody level were elevated.

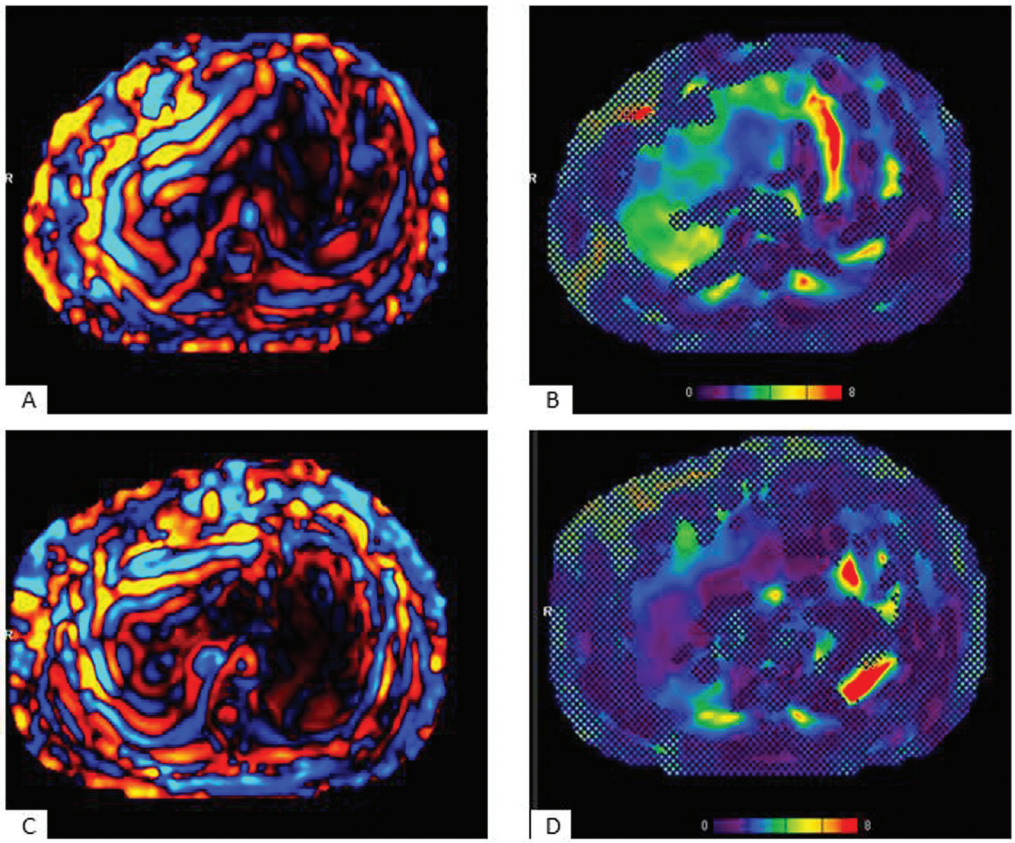

Fig. 6 *Follow-up of MASH in a 40-year-old man after Saroglitazar treatment.* (**A**) Wave image at diagnosis shows larger waves. (**B**) Confidence stiffness map at diagnosis shows LSM of 3.5 kPa. (**C**) Wave image after two years of treatment shows smaller waves throughout the liver. (**D**) Corresponding confidence stiffness map shows LSM of 1.9 kPa, a 45% reduction in stiffness. LSM, liver stiffness measurement; MASH, metabolic dysfunction-associated steatohepatitis.

used for close monitoring of patients with advanced fibrosis who are at higher risk of decompensation.

MRE versus Ultrasound Elastography

Ultrasound-based elastography techniques for assessment of liver fibrosis are extensively used in routine clinical practice. The most commonly used ultrasound elastography techniques are transient elastography (FibroScan) and 2D shear wave elastography. Both of these methods are recommended for diagnosis, follow-up, and surveillance in cases with liver fibrosis.[53] There are several advantages of MRE over ultrasound. MRE assesses a much larger portion of the liver compared with ultrasound elastography which only assesses

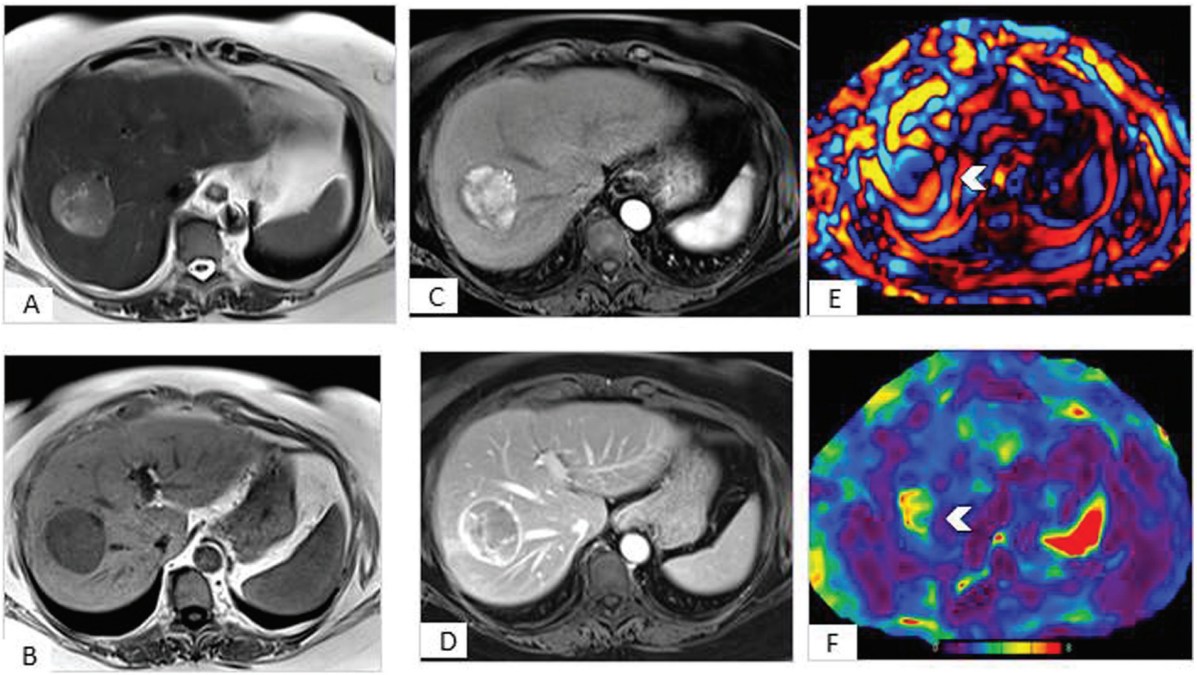

Fig. 7 **Hepatocellular carcinoma in a normal liver.** (**A**) Axial T2 HASTE image shows intermediate signal intensity mass in right lobe with low signal intensity in T1W image (**B**). (**C**) Postcontrast late arterial phase image shows hyperenhancement, and portal phase (**D**) shows washout with capsular enhancement. (**E**) MRE wave map shows normal amplitude background waves with distortion at the level of the mass (arrowhead). (**F**) Stiffness map shows focally raised stiffness within the mass (arrowhead). HASTE, half-Fourier single-shot turbo spin echo; MRE, magnetic resonance elastography; T1W, T1-weighted.

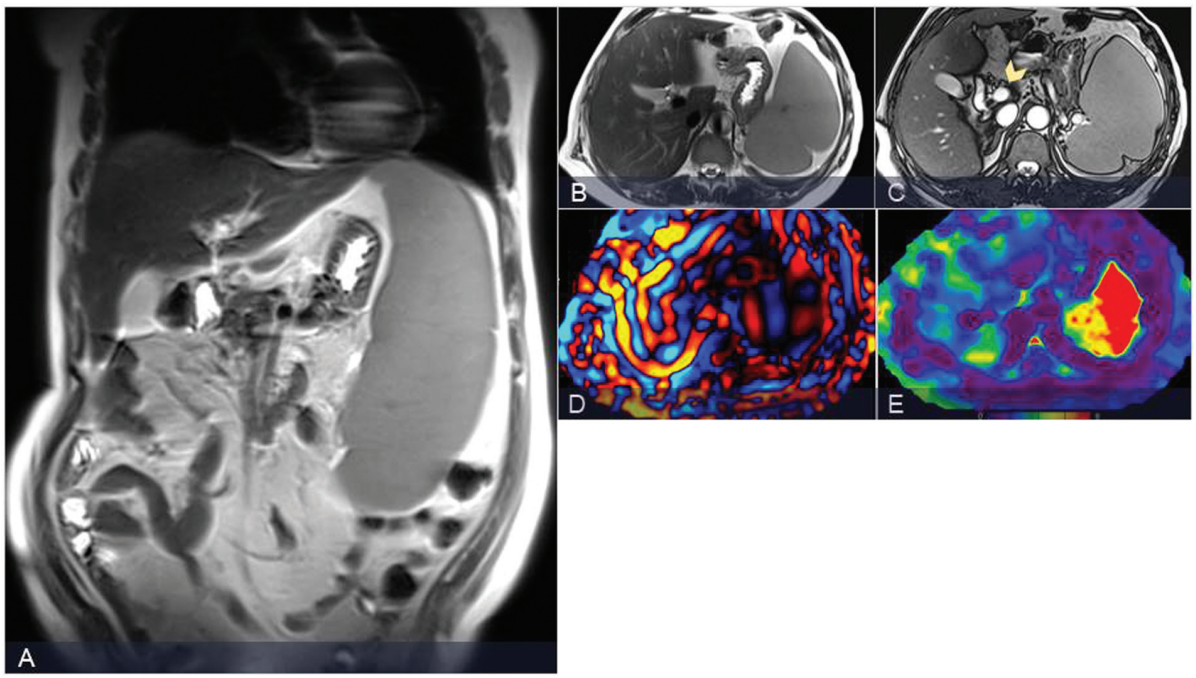

Fig. 8 **New-onset massive splenomegaly.** (**A**) Coronal T2W MR shows massively enlarged spleen with differential diagnosis of CLD and myeloproliferative disease. (**B**) Axial T2 HASTE shows normal liver morphology. (**C**) Axial TRuFI image shows patent portal vein (arrowhead). (**D**) MRE wave image shows normal amplitude waves. (**E**) Stiffness map shows normal LSM of 2.4 kPa suggesting absence of chronic liver disease. Bone marrow biopsy later proved myelofibrosis which explained the splenomegaly. CLD, chronic liver disease; HASTE, half-Fourier single turbo spin-echo; LSM, liver stiffness measurement; MR, magnetic resonance; MRE, magnetic resonance elastography; T2W, T2-weighted.

only a small ROI. MRE has shown a higher technical success rate (94%) than transient elastography/FibroScan (84%).[54] MRE showed better accuracy than acoustic radiation force impulse in the diagnosis of early stage of liver fibrosis.[55] MRE is less affected by presence of obesity, ascites, and anatomical variations.[56] MRE has also shown higher sensitivity and specificity in detection of fibrosis, especially early fibrosis, than ultrasound-based methods.[26,57] It is to be noted that LSM values

Table 2 Ultrasound elastography versus magnetic resonance elastography

Factor	Ultrasound elastography	Magnetic resonance elastography
Volume of tissue examined	Smaller	Larger
Presence of ascites	May affect measurement	Does not affect measurement
Presence of obesity	May affect measurement	Does not affect measurement
Detection of early fibrosis	Less accurate	More accurate
Technical success rate	Lower (up to 84%)	Higher (up to 94%)
Global evaluation of liver for other pathologies (such as hepatocellular carcinoma surveillance)	Limited, especially in presence of high liver fat content	Superior, not affected by degree of fat deposition or fibrosis

obtained by ultrasound and MRE are not comparable. Ultrasound-based shear wave elastography reports the Young's modulus (E in kPa) and/or shear wave velocity (cm/s). MRE reports complex shear wave modulus (G in kPa). Roughly, $E = 3G$, but this conversion is based on tissue property assumptions which may not be accurate (►**Table 2**).[58]

Newer Applications of MRE

MRE has been successfully utilized in the evaluation of CLD and its complications in the pediatric population.[59] MRE can be performed on liver transplant recipients as a part of surveillance for recurrence of fibrosis.[60–62] MRE can also be used in the evaluation of potential donors for detection of occult fibrosis.[63] Passive venous congestion due to increased central venous pressure can cause congestive hepatopathy which is particularly significant in patients with congenital single ventricle disease who have been treated successfully with Fontan surgery. Even after surgery, these patients can develop venous congestion termed as Fontan-associated liver disease (FALD) which in turn causes increased liver stiffness. MRE cannot distinguish between congestion and fibrosis as a cause of elevated LSM; however, it is shown that LSM detected by MRE is independently associated with central venous pressure[64] and correlates with MELD score in FALD.[50]

Limitation

- *Lack of optimal cutoff*: There is no consensus for optimal cutoff for the diagnosis of different stages of fibrosis for different etiologies of CLD. The cutoff for the diagnosis of clinically significant fibrosis may differ depending on the etiology of CLD.[65]

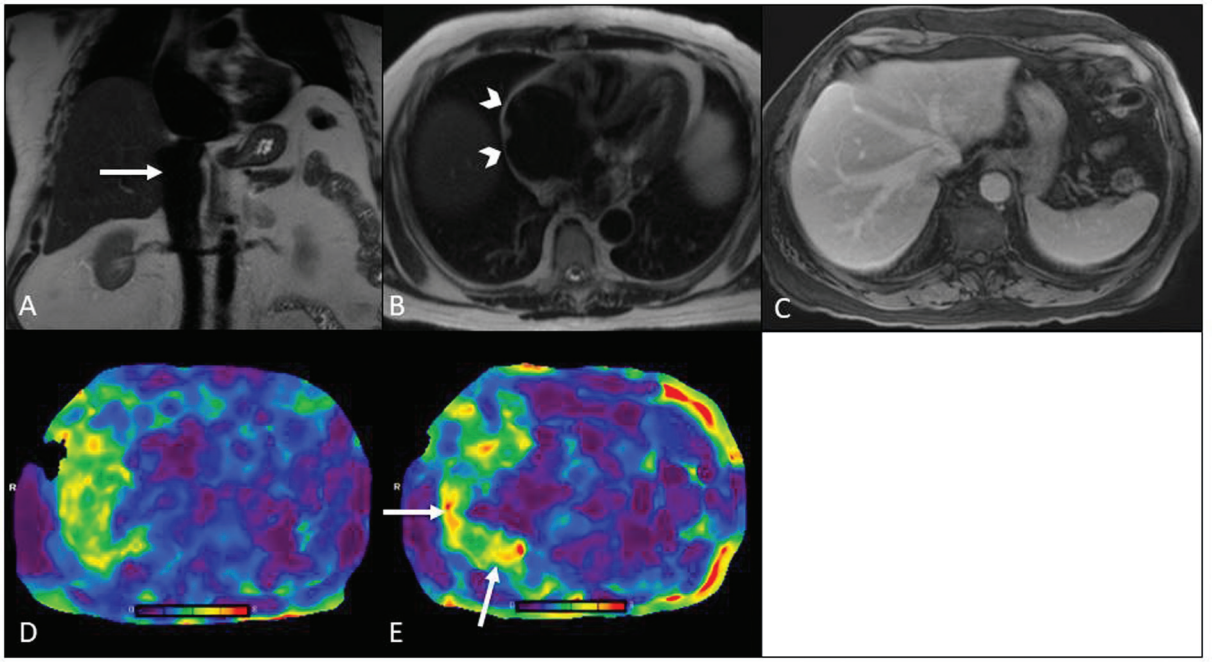

Fig. 9 Passive venous congestion causing elevated stiffness. A 70-year-old man with tricuspid regurgitation. T2W (**A, B**) and postcontrast T1W (**C**) images show dilated IVC (arrow in A), right atrium (arrowheads in **B**) and dilated hepatic veins (**C**). MRE (**D, E**) shows elevated stiffness (mean 3.9 kPa). Notice predominantly peripheral increase of stiffness (arrows). Biopsy showed no fibrosis. IVC, inferior vena cava; MRE, magnetic resonance elastography; T1W, T1-weighted; T2W, T2-weighted.

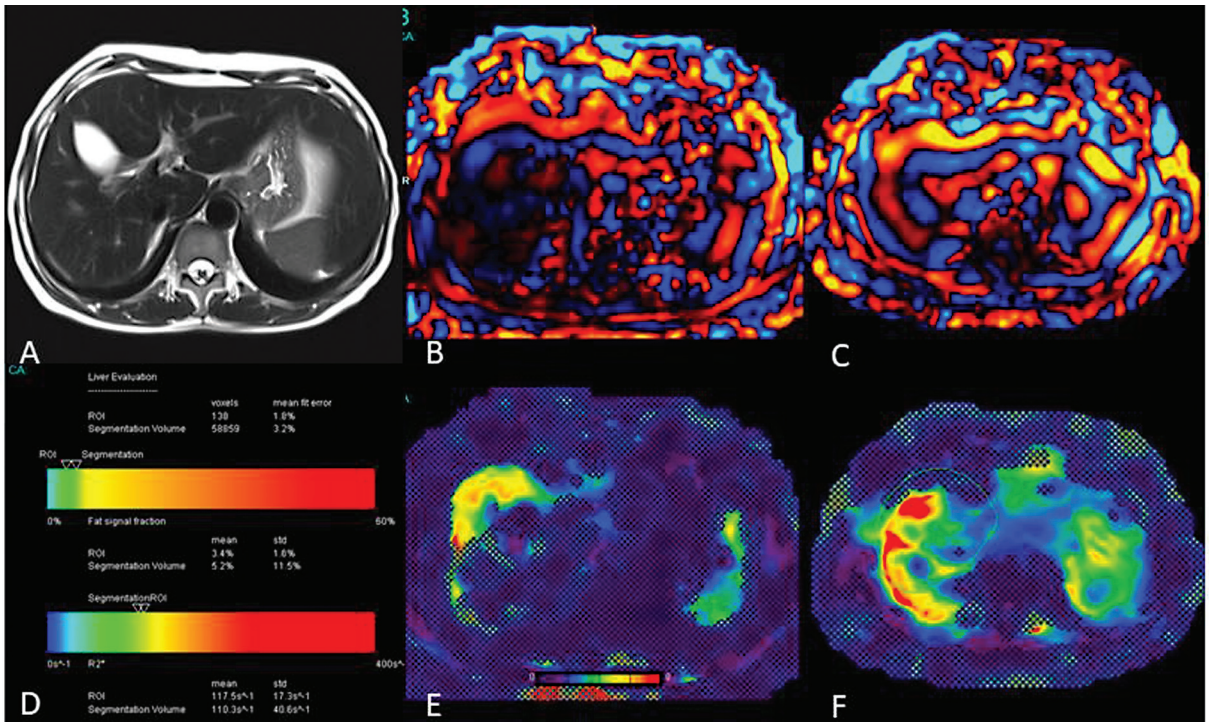

Fig. 10 **Liver iron overload and value of SE MRE.** (**A**) Liver shows low parenchymal signal in T2W images and shows high R* value (corresponding to liver iron content of ~ 2 mg/g of dry weight of liver) in qDIXON image (**B**). (**C, D**) Wave map (**C**) and confidence map (**D**) of GRE MRE sequence show signal loss and inadequate study. (**E, F**) Wave map (**E**) and confidence map (**F**) of SE MRE shows high liver stiffness. GRE, gradient-recalled echo; MRE, magnetic resonance elastography; SE, spin echo; T2W, T2-weighted.

- *Confounders of LSM*: LSM with MRE is a surrogate marker for liver fibrosis in CLD. As mentioned earlier, in the appropriate clinical context, increased LSM is suggestive of liver fibrosis and it is proportionate to the stage of liver fibrosis. However, other liver pathologies may cause elevated LSM independent of liver fibrosis, especially inflammation, biliary obstruction, and hepatic venous congestion. Inflammation is especially important as in most cases with early stages of CLD, inflammation may coexist. Furthermore, CLD may be complicated by acute hepatitis or acute flare of CLD.[41] Therefore, LSM should be interpreted with caution in the presence of acute inflammation and possibly avoided. In the presence of inflammation, especially in a liver with minimal to mild fibrosis, MRE can potentially overdiagnose fibrosis stage.[66,67] However, this limitation is also present with other noninvasive imaging-based techniques such as transient elastography and ultrasound shear wave elastography. Acute biliary obstruction and cholestasis can cause elevated LSM.[68] Passive venous congestion and congestive hepatopathy (post-Fontan surgery, congestive cardiac failure, or constrictive pericarditis) can cause increased liver stiffness.[69] Rarely, elevated LSM can be caused by diffuse infiltration such as metastases or amyloidosis.[41] Therefore, MRE should be interpreted in conjunction to the available clinical information and other MR sequences (►**Fig. 9**).
- *Iron overload*: As MRE sequence is based on GRE, susceptibility artifact from liver iron overload is the most common cause of technical failure of MRE.[14,65] A low T2* value of liver parenchyma can predict technical failure of MRE.[70] However, using a SE-based MRE or a GRE sequence with low echo time can increase the technical success rate[71] (►**Fig. 10**).

Conclusion

MRE has revolutionized the assessment of liver fibrosis in CLD, offering a noninvasive, accurate, and reproducible method for staging and monitoring fibrosis. With clear guidelines for its usage, the technique has become an integral part of clinical practice, aiding in the diagnosis, treatment, and follow-up of CLD patients. As technology continues to advance and more data become available, the role of MRE in CLD management is likely to expand, further improving patient care and outcomes.

Funding
None.

Conflict of Interest
None declared.

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