ORIGINAL ARTICLE

Quantification of Liver Stiffness Using Magnetic Resonance Elastography in Comparison with Transient Elastography and Noninvasive Fibrosis Score in Fatty Liver

Preethi Sharon M[1], Sameer Peer[2], Anjali Raj[3], Gourav Kaushal[4], Harmeet Kaur[5], Arvinder Wander[6], Sandeep Singh[7], Paramdeep Singh[8*]

Received: 11 October 2024; *Accepted:* 01 September 2025

Abstract

Background: The global incidence of fatty liver (FL) [alcoholic and nonalcoholic FL disease (NAFLD)] is increasing. Imaging-based elastography techniques, being noninvasive, may eliminate the need for more invasive techniques for the diagnosis and staging of liver fibrosis in FL disease.

Objective: Our study aims to address the gap in the current research by exploring the correlation between mean liver stiffness measurement (LSM) as obtained through magnetic resonance elastography (MRE) and transient elastography (TE), and two commonly used clinical scores, fibrosis-4 index (FIB-4) score and aspartate aminotransferase to platelet ratio index (APRI) score.

Materials and methods: In this hospital-based cross-sectional study, 62 patients diagnosed with FL on ultrasound were recruited. The patients were further subjected to MR liver elastography and TE, and LSM using both modalities was recorded. A history of diabetes mellitus and alcohol intake was taken. Moreover, noninvasive fibrosis scores such as FIB-4 and APRI were calculated using standard formulas.

Results: The correlation analysis revealed a strong positive correlation between LSM values obtained from MRE and TE ($r = 0.88$) (Cohen's $\kappa = 0.87$), a moderate correlation between MRE and FIB-4 score ($r = 0.44$), and weak positive correlations involving MRE and APRI ($r = 0.34$), TE and FIB-4 score ($r = 0.36$), and TE and APRI ($r = 0.29$). Additionally, significantly higher fat fractions were quantified [median (IQR)] in grade III FL [23.6 (15.9–29.5)] as compared to grades I [8.45 (2.25–13.9)] and grade II [13.1 (8.4–19.7)].

Conclusion: MRE shows a strong positive correlation with TE for LSM and stage of fibrosis. Our findings suggest that MRE could be a valuable tool in the diagnostic armamentarium of FLD.

Journal of The Association of Physicians of India (2025): 10.59556/japi.73.1276

Introduction

Fatty liver (FL) represents the common underlying factor in the two most prevalent and emerging determinants of chronic liver disease (CLD), that is, alcoholic liver disease (ALD) and nonalcoholic FL disease (NAFLD). The adoption of a westernized lifestyle and consumption of a high-calorie diet contribute to the development of NAFLD. The accumulation of an excessive amount of fat within hepatocytes leads to fatty change, often asymptomatic, and can be reversed. However, when conditions are conducive to inflammation and, more importantly, the development of fibrosis, it becomes an irreversible process.[1,2] Biopsy is considered the gold standard for examining liver fibrosis, but it is an invasive examination that may cause sampling error and other complications.[3] Hence, there is a need for the development of noninvasive tests (NITs). NITs are increasingly replacing biopsies, effectively addressing the limitations of the invasive procedure and gaining popularity in clinical practices. The two primary categories of NITs for liver fibrosis staging are imaging-based elastography and serum biomarkers. Biomarkers such as the aspartate aminotransferase to platelet ratio index (APRI) and fibrosis-4 index (FIB-4) scores have undergone extensive validation and are widely used due to their ease of accessibility. Furthermore, methods like transient elastography (TE), shear wave elastography (SWE), and magnetic resonance elastography (MRE) have demonstrated exceptional accuracy in detecting liver fibrosis.[4] Out of these techniques, MRE is considered the most accurate diagnostic tool.[5]

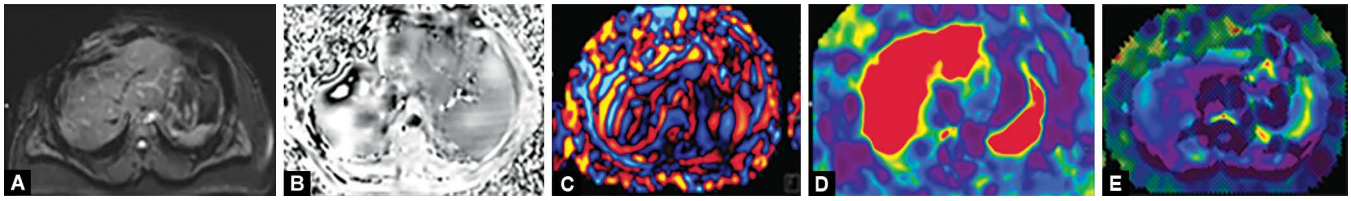
Magnetic resonance elastography utilizes magnetic resonance imaging (MRI) to calculate the mechanical properties of tissues quantitatively and is carried out using a source of vibration to generate mechanical low-frequency waves in tissues and analyze wave information to create images. Liver MRE is used to measure liver stiffness in assessing fibrosis or cirrhosis. Moreover, MRE offers the advantage of scanning a wide area of liver parenchyma while allowing the flexibility to select specific region of interest (ROI).[6–8] It allows for the detection of fibrosis and accurately stages the same.[9] MRE proves valuable in the early changes in liver stiffness, making it easier to identify individuals with FL who could potentially develop cirrhosis.[10]

Materials and Methods

In this hospital-based cross-sectional study, we recruited 62 patients diagnosed with FL who met the specified inclusion and exclusion criteria. Patients referred for abdominal ultrasound on clinical suspicion (age-group 18–65 years) were included. Pregnant women, individuals with a history of hemochromatosis, iron overload disease or storage disorders, ascites, claustrophobic patients, and those who have ferromagnetic substances, implants, or any established contraindication for MRI were excluded [details described in supplementary data (Table 1)]. The other variables considered, and their criteria are discussed in supplementary data.[11–15] Anthropometric measures such as height and weight were recorded, and body mass index (BMI) was calculated. A history of diabetes mellitus and alcohol intake was taken, considering known diabetics on medication, and according to standard measures of alcohol consumption, patients were classified as alcoholic or nonalcoholic. Furthermore, noninvasive fibrosis scores such as FIB-4 and APRI were calculated using standard formulae.

[1]Junior Resident; [2]Assistant Professor, Department of Radiodiagnosis; [3]Junior Research Fellow, Department of Neurosurgery; [4]Associate Professor, Department of Surgical Gastroenterology; [5]Associate Professor, Department of Radiodiagnosis; [6]Associate Professor, Department of Pediatrics; [7]Junior Research Fellow; [8]Additional Professor, Department of Radiodiagnosis, All India Institute of Medical Sciences, Bathinda, Punjab, India; *Corresponding Author

How to cite this article: M PS, Peer S, Raj A, *et al.* Quantification of Liver Stiffness Using Magnetic Resonance Elastography in Comparison with Transient Elastography and Noninvasive Fibrosis Score in Fatty Liver. J Assoc Physicians India 2025;73(12):64–70.



Figs 1A to E: Development of color elastogram from magnitude and phase sequences: (A) Magnitude image; (B) Phase image; (C) Wave image; (D) Color elastogram; and (E) Color elastogram with 95% confidence map

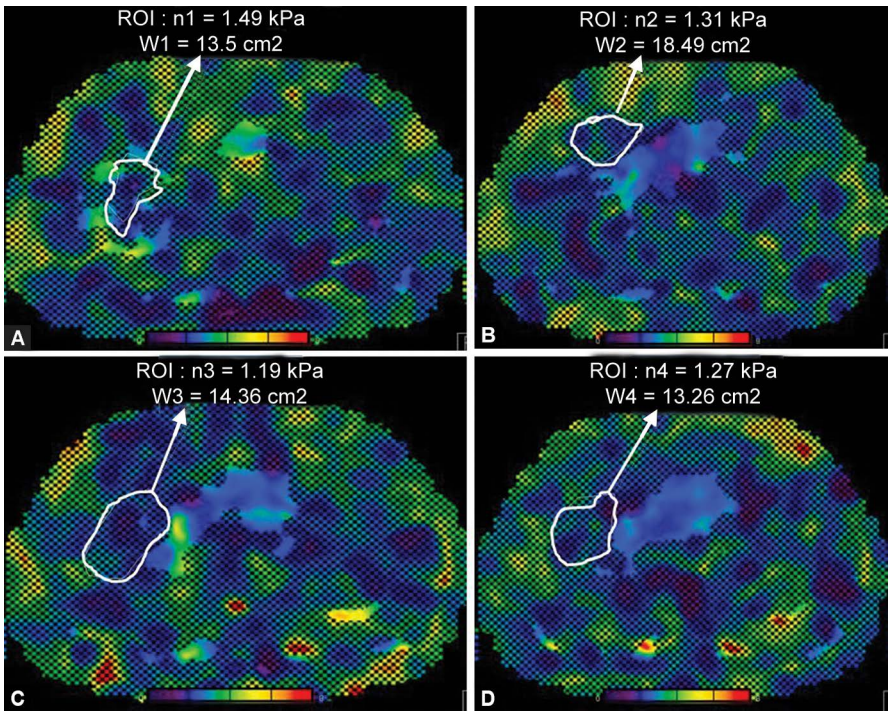


Fig. 2: Freehand ROIs drawn at various sections of the liver elastograms obtained, for calculating the mean liver stiffness

The data obtained from MRE, TE, and other NITs were compared, correlated, and subjected to further analysis.

Equipment used:

- Ultrasound machine: Alpinion E-Cube i7.
- Transducer: convex–C1-6T.

The liver was scanned in the supine position using a convex transducer. The grade of FL if seen was assigned according to the standard ultrasound grading described in supplementary data Table 2.[16]

- Siemens 3 Tesla Magnetom Skyra.
- Body coil.
- Resonant passive driver (rigid type) with elastic band to fasten the driver.
- Resonant active driver (placed outside the MRI room).

Patient Positioning

Patients were positioned in a supine posture. The passive driver was installed along the midclavicular line, placed above the lower right chest wall at the xiphisternum level. It was placed in direct contact with the body wall and fastened with an elastic band. The passive driver was then linked through a plastic tube to the active driver located outside the scanning room.

Sequences used: two-dimensional (2D) gradient-echo sequences with cyclic motion-encoding gradients (MEG) for MRE.

The generation of a color elastogram from the magnitude and phase sequences is represented in Figure 1.

Generic formula for calculating the weighted arithmetic mean (AMw) of the mean liver stiffness (*n*) for ROIs drawn on four images (Fig. 2), with an ROI size of w pixels:

$$AMw = (n1w1 + n2w2 + n3w3 + n4w4) \div (w1 + w2 + w3 + w4)$$

Where:

- n1, n2, n3, and n4 were the mean liver stiffness values measured on four elastograms.
- w1, w2, w3, and w4 are the sizes of the ROIs drawn on each of the four elastograms.

Siemens liver lab sequences for quantification of fat in the liver.

- T1 VIBE—enhanced Dixon technique (eDIXON).
- T1 VIBE—quantitative Dixon technique (qDIXON).

Guidelines for interpreting liver stiffness through the application of MRE are provided in supplementary data (Table 3).[17]

- Transient elastography: Echosens FibroScan 502 Touch.

The grading of liver fibrosis in both alcoholic and nonalcoholic patients is determined by liver stiffness measurements (LSMs) obtained from TE, as detailed in supplementary data Table 4.[18]

The workflow employed in the study is depicted in Figure 1 of the supplementary data.

Statistical Analysis

Statistical analysis was conducted using SPSS version 23.0. Continuous variables were expressed as the mean and standard deviation (SD), reported as mean ± SD, and compared using the independent *t*-test. For variables not following a normal distribution, the median and interquartile range were used, with comparisons made *via* the Mann–Whitney *U* test. Categorical variables were presented as percentages and analyzed using the Chi-squared test. When comparing continuous variables across more than two groups, the Kruskal–Wallis test was applied (if they were not following a normal distribution), and further pairwise analysis was done using Tukey's test. Interrater reliability between MRE LSM and FibroScan LE was done using Cohen's kappa. Correlation analysis was done between MRE vs TE, MRE vs scores, TE vs scores, and *r*-values calculated. Variables with p-value < 0.05 were considered statistically significant.

Results

This study included 62 patients (40 males and 22 females) with a diagnosis of FL on ultrasound and further subjected to MR liver

elastography and TE, and measurement of LSM using both modalities was recorded. The mean age of the patients was 47.5 ± 10.52 years. Additionally, about 40% of the population was alcoholic and 17.74% was diabetic.

Mean Liver Stiffness Measurement from Magnetic Resonance Elastography

The mean LSM obtained through MRE was 3.39 ± 1.53 in males and 3.08 ± 1.15 in females. Based on grading of liver stiffness measurement obtained through MR elastography, 25.8% had stage 1–2 fibrosis, 14.5% had stage 2–3 fibrosis, 8.1% had stage 4 fibrosis/cirrhosis, and 4.8% had stage 3–4 fibrosis.

Mean Liver Stiffness Measurement in Alcoholic vs Nonalcoholic and Diabetic vs Nondiabetic

The mean LSM obtained through MRE for alcoholic and nonalcoholic, and diabetic and nondiabetic patients is shown in Table 1. Based on adjusted standardized residuals, stage 2–3 fibrosis was seen as significantly higher in diabetic patients, and normal and normal/inflammation were seen as significantly higher in nondiabetic patients (Table 2 and 3).

Correlation Analysis

A strong correlation was noted between LSM obtained with MRE and TE (R = 0.883). Good correlation was noted between LSM values obtained with MRE and Fib-4 (R = 0.446) (Fig. 3). Weak correlation was noted between LSM measurements obtained with MRE and APRI score (R = 0.34), and also LSM obtained with TE and Fib-4 score (R = 0.36) (Fig. 4). Also, a weak correlation was noted between LSM measurement obtained with TE and APRI score (R = 0.298) (Fig. 5).

Subgroup Analysis

Median FIB-4 and APRI Scores across Different Grades of Liver Stiffness

Table 1: Mean LSM (MRE) in alcoholics vs nonalcoholics and diabetics vs nondiabetics

LSM MRE	*Alcoholic (N = 16)*	*Nonalcoholic (N = 46)*	*p-value*	*Diabetic (N = 11)*	*Nondiabetic (N = 51)*	*p-value*
Mean ± SD	4.04 ± 1.82	3.02 ± 1.15	0.012	3.69 ± 1.43	3.19 ± 1.4	0.314

Table 2: Distribution of stage of LSM (MRE) in alcoholic/nonalcoholic and diabetic/nondiabetic

LSM stage		*Alcoholic (N = 16)*	*Nonalcoholic (N = 46)*	*p-value*	*Diabetic (N = 11)*	*Nondiabetic (N = 51)*	*p-value*
Normal	Number (percentage)	1 (6.3)	15 (32.6)	0.111	1 (9.1)	15 (29.4)	0.046
Normal/inflammation		2 (12.5)	11 (23.9)		0 (0)	13 (25.5)	
Stage 1–2 fibrosis		5 (31.5)	11 (23.9)		5 (45.5)	11 (21.6)	
Stage 2–3 fibrosis		4 (25)	5 (10.9)		4 (36.4)	5 (9.8)	
Stage 3–4 fibrosis		1 (6.3)	2 (4.3)		0 (0)	3 (5.9)	
Stage 4/cirrhosis		3 (18.8)	2 (4.3)		1 (9.1)	4 (7.8)	

Table 3: Distribution of LSM measured using TE vs alcoholic/nonalcoholic and diabetic/nondiabetic

TE stage		*Alcoholic (N = 16)*	*Nonalcoholic (N = 46)*	*p-value*	*Diabetic (N = 11)*	*Nondiabetic (N = 51)*	*p-value*
F0–F1	Number (percentage)	4 (25)	27 (58.7)	0.076	1 (9.1)	30 (58.8)	0.025
F2		8 (50)	9 (19.6)		6 (54.5)	11 (21.6)	
F3		2 (12.5)	4 (8.7)		2 (18.2)	4 (7.8)	
F4		2 (12.5)	6 (13)		2 (18.2)	6 (11.8)	

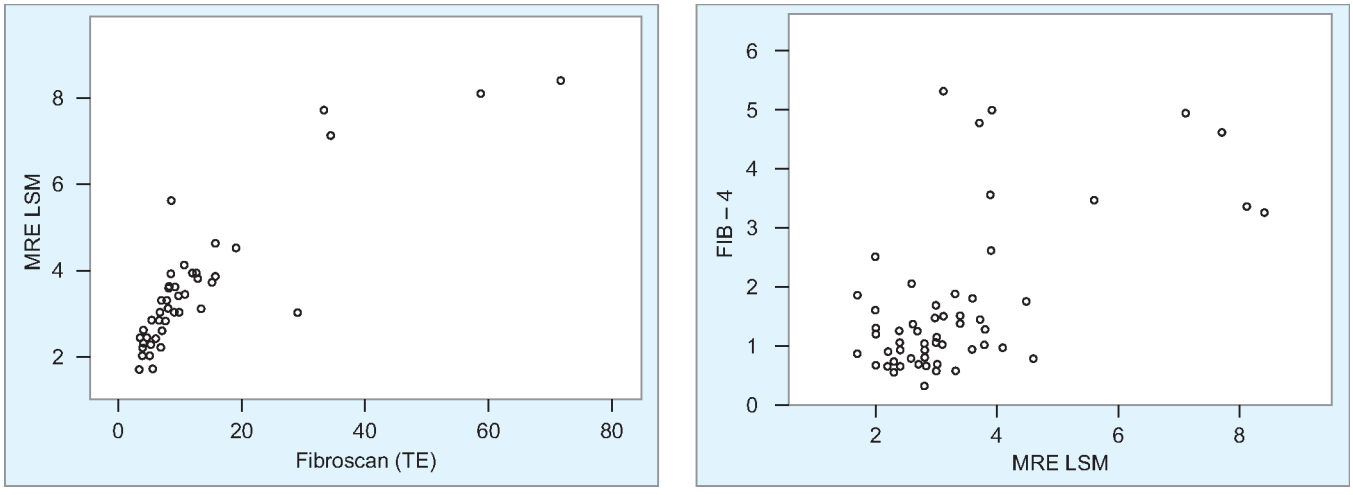


Fig. 3: Correlation between MRE vs TE [scatter plot showing very strong correlation (R = 0.883) between LSM values obtained through MRE and TE] and correlation between MRE vs FIB-4 [scatter plot showing moderate correlation (R = 0.446) between MRE LSM and FIB-4 score]

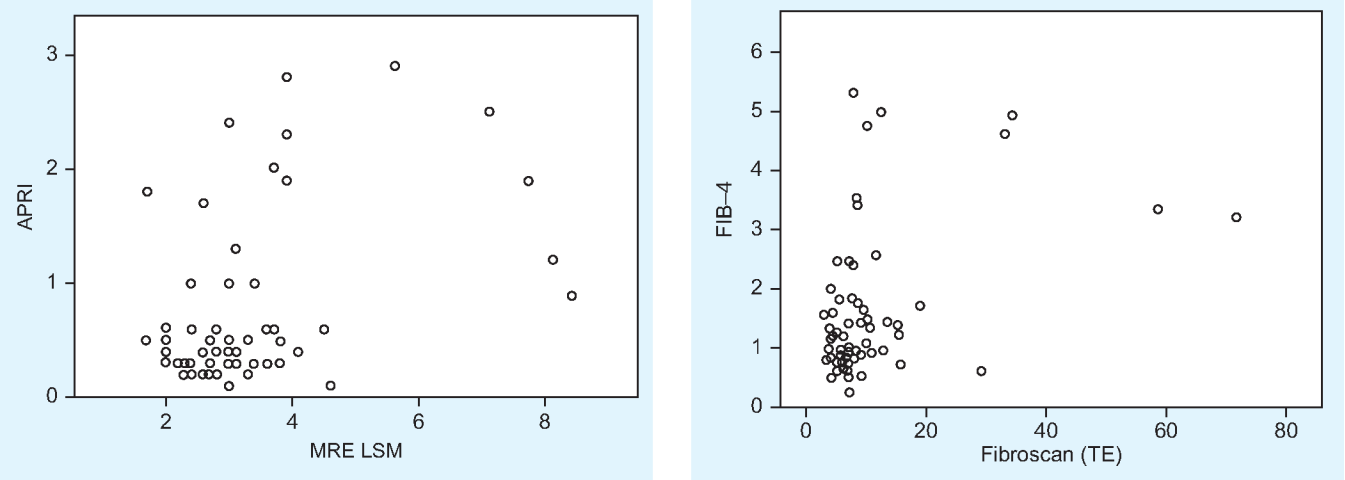


Fig. 4: MRE vs APRI [scatter plot showing weak correlation (R = 0.34) between MRE LSM and APRI score] and correlation between TE vs FIB-4 [scatter plot showing weak correlation (R = 0.36) between TE LSM and FIB-4 score]

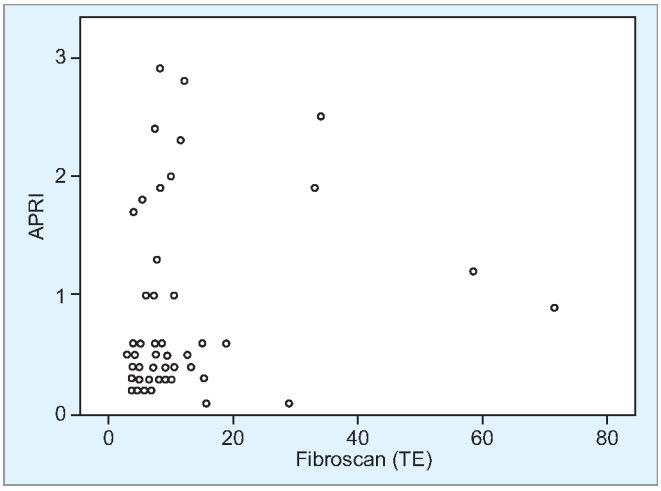


Fig. 5: Correlation between TE vs APRI [scatter plot showing weak correlation (R = 0.298) between TE LSM and APRI score]

Table 4: FIB-4 vs MRE scores pairwise

Pairs	*p-value*
Normal vs normal/inflammation	1
Normal vs stage 1–2 fibrosis	1
Normal vs stage 2–3 fibrosis	0.177
Normal vs stage 3–4 fibrosis	1
Normal vs stage 4/cirrhosis	0.007
Normal/inflammation vs stage 1–2 fibrosis	0.717
Normal/inflammation vs stage 2–3 fibrosis	0.054
Normal/inflammation vs stage 3–4 fibrosis	1
Normal/inflammation vs stage 4/cirrhosis	0.002
Stage 1–2 fibrosis vs stage 2–3 fibrosis	1
Stage 1–2 fibrosis vs stage 3–4 fibrosis	1
Stage 1–2 fibrosis vs stage 4/cirrhosis	0.211
Stage 2–3 fibrosis vs stage 3–4 fibrosis	1
Stage 2–3 fibrosis vs stage 4/cirrhosis	1
Stage 3–4 fibrosis vs stage 4/cirrhosis	0.335

Measurement Obtained through Magnetic Resonance Elastography

In addition, on subgroup analysis, a significant difference in FIB-4 score was seen between normal vs stage 4 fibrosis/cirrhosis, and normal/inflammation vs stage 4 fibrosis/cirrhosis of MRE LSM grading (Table 4), and a significant difference in APRI score was seen between normal vs stage 4 fibrosis/cirrhosis, and normal/inflammation vs stage 4 fibrosis/cirrhosis of MRE LSM grading (Table 5). Furthermore, the median FIB-4 score and APRI score across different grades of fibrosis were obtained through MRE summarized in Table 6.

Median FIB-4 and APRI across Different Grades of Liver Stiffness Measurement Obtained through Transient Elastography

In the subgroup analysis, a significant difference in FIB-4 score was seen between F0 and F1 vs F2 stages of TE LSM grading (Table 7). On the contrary, a significant difference in APRI score was seen between F0–F1 vs F2 and F0–F1 vs F3 stages of TE LSM grading in the case of APRI and TE subgroup analysis (Table 8).

Median FIB-4 score and APRI score across different grades of fibrosis were obtained through TE (Table 9).

The proton density fat fraction (PDFF) values obtained through quantitative MRI sequences were significantly different in grade I FL and grade III FL determined on ultrasonography (USG) (Table 10).

Correlation analysis revealed a very strong correlation between LSM values obtained through MRE and TE, a moderate correlation between MRE LSM and FIB-4 score, and a weak correlation between MRE LSM and APRI score,

Table 5: APRI vs MRE scores pairwise

Pairs	*p-value*
Normal vs normal/inflammation	1
Normal vs stage 1–2 fibrosis	1
Normal vs stage 2–3 fibrosis	0.472
Normal vs stage 3–4 fibrosis	1
Normal vs stage 4/cirrhosis	0.044
Normal/inflammation vs stage 1–2 fibrosis	1
Normal/inflammation vs stage 2–3 fibrosis	0.08
Normal/inflammation vs stage 3–4 fibrosis	1
Normal/inflammation vs stage 4/cirrhosis	0.007
Stage 1–2 fibrosis vs stage 2–3 fibrosis	0.783
Stage 1–2 fibrosis vs stage 3–4 fibrosis	1
Stage 1–2 fibrosis vs stage 4/cirrhosis	0.076
Stage 2–3 fibrosis vs stage 3–4 fibrosis	1
Stage 2–3 fibrosis vs stage 4/cirrhosis	1
Stage 3–4 fibrosis vs stage 4/cirrhosis	0.326

Table 7: FIB-4 vs TE scores subgroups

Pairs	*p-value*
F0–F1 vs F2	0.015
F0–F1 vs F3	0.114
F0–F1 vs F4	0.077
F2 vs F3	1
F2 vs F4	1
F3 vs F4	1

Table 8: APRI vs TE scores subgroups

Pairs	*p-value*
F0–F1 vs F2	0.039
F0–F1 vs F3	0.026
F0–F1 vs F4	0.703
F2 vs F3	1
F2 vs F4	1
F3 vs F4	1

Table 6: MRE vs FIB-4 and APRI scores

Scores		*Normal*	*Normal/inflammation*	*Stage 1–2 fibrosis*	*Stage 2–3 fibrosis*	*Stage 3–4 fibrosis*	*Stage 4/cirrhosis*	*p-value*
FIB–4	Median (IQR)	0.96 (0.64–1.49)	0.83 (0.69–1.22)	1.44 (0.99–1.79)	1.77 (1.11–4.14)	0.93 (0.75)	3.44 (3.28–4.77)	0.001
APRI		0.35 (0.3–0.57)	0.3 (0.2–0.45)	0.35 (0.3–0.87)	0.6 (0.4–2.15)	0.4 (0.1)	1.9 (1.05–2.7)	0.003

Table 9: TE vs scores

Scores		*F0–F1*	*F2*	*F3*	*F4*	*p-value*
FIB-4	Median (IQR)	0.91 (0.64–1.25)	1.65 (0.95–1.95)	1.43 (1.25–3.18)	2.47 (0.87–4.29)	0.003
APRI		0.3 (0.2–0.5)	0.5 (0.3–1.6)	0.8 (0.475–2.42)	0.75 (0.15–1.72)	0.005

Table 10: PDFF vs grades of FL

PDFF	*Grade I*	*Grade II*	*Grade III*	*p-value*
Median (IQR)	8.45 (2.25–13.9)	13.1 (8.4–19.7)	23.6 (15.9–29.5)	0.001

Table 11: Correlation analysis

Correlation	*p-value*	*r-value*
MRE vs TE	<0.001	0.883
MRE vs FIB-4	<0.001	0.446
MRE vs APRI	0.007	0.34
TE vs FIB-4	0.004	0.364
TE vs APRI	0.019	0.298

Table 12: Interrater reliability between MRE and TE

	Cohen's kappa	*p-value*
MRE LSM vs TE LSM	0.363	<0.001

TE and FIB-4 score, as well as TE and APRI score (Table 11).

The interrater reliability between MRE LSM and TE LSM across different grades of fibrosis showed a Cohen's kappa value of 0.363, indicating fair agreement (Table 12).

Table 13: Level of agreement between MRE and TE for fibrosis detection

Fibrosis detected by MRE and TE	*Fibrosis not detected by both MRE and TE*	*Fibrosis detected by MRE only*	*Fibrosis detected by TE only*
30	28	3	1

Percentage of agreement = 93.54%; Cohen's κ = 0.871; almost perfect agreement

Our data shows an almost perfect agreement (93.54%) between MRE and TE for the measurement of LSM (Cohen's κ of 0.871) with a strong positive correlation. The interrater reliability between MRE and TE across various stages of fibrosis showed a fair agreement (Table 13).

Discussion

Fibrosis is the result of injury to hepatocytes, which may be incited by various etiologies. Hepatocyte injury leads to a cascade of events, mediated by various intermediate compounds such as free radicals and inflammatory cytokines, which culminate in cell death and subsequent fibrosis.[19] The stiffness of the liver undergoes a change during this process of hepatocyte injury and ultimately fibrosis. An inflamed liver parenchyma is stiffer than a normal parenchyma. With the progression of inflammation into fibrosis, liver stiffness progressively increases. While cirrhosis represents an irreversible end-stage liver parenchymal damage because of fibrosis, the earlier stages of liver inflammation could be potentially reversible. This is especially important in the setting of FL disease, whereby an intervention (such as alcohol cessation, modification of lifestyle, etc.) may halt the progression of steatosis into steatohepatitis and ultimately cirrhosis.[20] This underscores the

need for early diagnosis of FL disease. While biopsy may be considered the "gold standard" for establishing a diagnosis of FL disease, steatohepatitis, and cirrhosis, it is an invasive procedure and exposes patients to risks such as postprocedure hemorrhage and pain. Also, sampling error, as well as the subjective nature of the interpretation of biopsy specimens, may lead to false negative results and may not be particularly useful in the early stages of the disease. Due to these potential limitations, there has been growing interest in developing NITs for early detection of the stage of liver fibrosis.[21]

This study was intended to answer a specific research question as to how well the LSM measured with MRE correlates with that obtained by TE, FIB-4 score, and APRI score. Our data suggests a very strong correlation between LSM obtained by MRE and TE, with an *r*-value of 0.88. TE uses a probe to measure the velocity of shear waves to determine the LSM, while MRE uses an acoustic driver vibrating at a particular frequency to transmit shear waves into the liver parenchyma synchronized with a phase-contrast pulse sequence to determine the displacement of tissue at the microlevel and create an elastogram map.[7] While TE uses about 10 measurements to improve the precision of measurements of liver stiffness, it suffers from a few limitations, such as not being able to map the measurement to the exact site from which the signal is measured, and limitations of measurement in obese patients.[22] While MRE and TE measure the same fundamental tissue property (i.e., stiffness), they differ in the basic methods by which the measurements are acquired. This may explain, in part at least, the variation in absolute measurements of LSM obtained from TE and MRE, as suggested by our data as well as previous studies. Our study shows a mean LSM of 3.28 ± 1.4 kPa using MRE, indicating stage one to two fibrosis, while the mean stiffness value of 10.72 ± 11.8 kPa using TE indicates severe scarring. However, since the tissue property being measured by both these modalities is the same (i.e., LSM), it is prudent to expect a good positive correlation between MRE and TE regarding the measurement of LSM. Indeed, our data shows an almost perfect agreement (93.54%) between MRE and TE for the measurement of LSM (Cohen's κ of 0.871) with a strong positive correlation. The interrater reliability between MRE and TE across various stages of fibrosis showed a fair agreement. It has been shown in previous studies[7] that MRE has a higher sensitivity and specificity for the determination of the stage of hepatic fibrosis as compared with TE. Thus, MRE may become an accurate screening tool for early diagnosis of fibrotic changes in the liver parenchyma. MRE, in addition, offers an advantage of coverage of the whole of the liver parenchyma as well as exact mapping of LSM of various regions of the parenchyma. This may be useful in the planning of biopsies from the regions showing higher LSM and thus improving the diagnostic yield of biopsies.

As far as the correlation between MRE, TE, and other noninvasive scores, that is, FIB-4 and APRI, is considered, only a moderate correlation was found between MRE and FIB-4, while a weak correlation was noted between MRE and APRI, TE and FIB-4, and TE and APRI scores. On comparing these scores across different grades of LSM measured using MRE, a statistically significant difference was found between normal and normal/inflammation vs stage 4 fibrosis/cirrhosis. Similarly, on comparing these scores across different grades of LSM measured using TE, a significant difference was found between F0–F1 vs F2 and F3. The noninvasive fibrosis scores have been specifically designed for hepatitis C-related liver parenchymal disease[23]; however, these have been used in NAFLD also. These scores have been shown to have a good negative predictive value for very low scores and a good positive predictive value for very high scores. However, these scores may not be indicative of the stage of hepatic fibrosis in the intermediate score range. Since these scores are dependent on AST and ALT levels in the mathematical formula, the scores may be normal in settings where there may not be transaminitis in the face of established fibrosis. This underscores the limitations of these noninvasive fibrosis scores and may thus explain the finding of our study, which suggests a rather moderate to weak correlation between these scores, TE, and MRE.

As a secondary objective of our study, we quantified the fat fraction of the liver using MRI-based sequences. We found a significantly higher mean fat fraction in patients with grade III FL (23.6) as compared with grade I and grade II FL (as stratified using ultrasound). PDFF values were significantly different between grade I and grade III of FL determined through ultrasound. This indicates the feasibility of the use of MRI as a tool for early detection and quantification of liver fat, a distinct advantage over ultrasound-based qualitative stratification.

Another interesting finding of our study is that the mean LSM measured using MRE is significantly higher in alcoholics (4.04 ± 1.82) compared to nonalcoholics (3.02 ± 1.15). The same was found to be true for LSM measured using TE, with median LSM values significantly higher in alcoholics (8.45) compared to nonalcoholics (6.8). This shows a higher prevalence of fibrosis in alcoholics compared to nonalcoholics in our study. This may indicate an accelerated progression of steatohepatitis and fibrosis in alcoholics as compared to nonalcoholics.

In LSM measured through MRE, stage 2–3 fibrosis was seen to be significantly higher in the diabetic population, while initial grades of stiffness such as normal and normal/inflammation were seen higher in the nondiabetic population. Similarly, in LSM measured through TE, the F0–F1 stage of fibrosis was significantly higher in nondiabetics while median LSM values were significantly raised in the diabetic population. This also highlights diabetes as an important risk factor for the progression of fibrosis in FL patients.

Limitations

We did not have any histopathological confirmation of the stage of hepatic fibrosis and predictive analysis could not be performed for each of the noninvasive methods of quantification of LSM.

Conclusion

Magnetic resonance elastography shows a strong positive correlation with TE for LSM and the stage of fibrosis. A moderate to weak correlation was noted between MRE, TE, and NIF scores. MRI-derived FF values increase with increasing grade of FL. MRE could be a useful screening tool for the detection of liver fibrosis in FLD, comparable to the established noninvasive method of TE.

Ethical Approval

Approval was obtained from the Institute Ethics Committee under IEC letter no. 283.

Consent for Publication

As the corresponding author, I confirm that I have read and understood the publication policies of the Journal of the Association of Physicians of India and that the manuscript titled "Role of Magnetic Resonance Elastography as a Quantitative Tool for the Assessment of Liver Stiffness in Fatty Liver Disease in Comparison with Transient Elastography and Noninvasive Fibrosis Scores" complies with these policies. On behalf of all authors, I provide my consent for the publication of this manuscript in the Journal of the Association of Physicians of India.

Author Contributions

The authors confirm their contributions to the paper as follows:

Dr Preethi Sharon M: Conceptualization, methodology, data collection, writing original draft, and data analysis.

Dr Paramdeep Singh: Conceptualization, methodology, data collection, writing original draft, and data analysis.

Dr Sameer Peer: Conceptualization, validation, interpretation, supervision, and editing.

Ms Anjali Raj: Visualization, writing and editing, and interpretation.

Dr Gourav Kaushal: Data curation, conceptualization, and validation.

Dr Arvinder Wander: Data curation, conceptualization, and validation.

Dr Harmeet Kaur: Writing, reviewing, methodology, and data analysis.

Mr Sandeep Singh: Validation and editing of the manuscript.

All authors reviewed the results and approved the final version of the manuscript.

Data Availability

We confirm that the data supporting the findings of this study are available within the article and/or its supplementary materials. Additional data, if required, can be obtained from the corresponding author upon reasonable request.

Supplementary Material

Supplementary files are available with author. Please connect with author for the supplementary content.

Orcid

Sameer Peer https://orcid.org/0000-0002-7674-5004
Arvinder Wander https://orcid.org/0009-0002-4949-9706
Sandeep Singh https://orcid.org/0000-0001-5993-1270

References

1. Mitra S, De A, Chowdhury A. Epidemiology of non-alcoholic and alcoholic fatty liver diseases. Transl Gastroenterol Hepatol 2020;5:16.
2. Bedogni G, Nobili V, Tiribelli C. Epidemiology of fatty liver: an update. World J Gastroenterol 2014;20(27):9050–9054.
3. Venkatesh SK, Yin M, Takahashi N, et al. Non-invasive detection of liver fibrosis: MR imaging features vs. MR elastography. Abdom Imaging 2015;40(4):766–775.
4. Thanapirom K, Suksawatamnuay S, Tanpowpong N, et al. Non-invasive tests for liver fibrosis assessment in patients with chronic liver diseases: a prospective study. Sci Rep 2022;12(1):4913.
5. Guglielmo FF, Venkatesh SK, Mitchell DG. Liver MR elastography technique and image interpretation: pearls and pitfalls. Radiographics 2019;39(7):1983–2002.
6. Mariappan YK, Glaser KJ, Ehman RL. Magnetic resonance elastography: a review. Clin Anat 2010;23(5):497–511.
7. Liang Y, Li D. Magnetic resonance elastography in staging liver fibrosis in non-alcoholic fatty liver disease: a pooled analysis of the diagnostic accuracy. BMC Gastroenterol 2020;20(1):89.
8. Venkatesh SK, Yin M, Ehman RL. Magnetic resonance elastography of liver: technique, analysis, and clinical applications. J Magn Reson Imaging 2013;37(3):544–555.
9. Serai SD, Obuchowski NA, Venkatesh SK, et al. Repeatability of MR elastography of liver: a meta-analysis. Radiology 2017;285(1):92–100.
10. Reddy JK, Rao MS. Lipid metabolism and liver inflammation. II. Fatty liver disease and fatty acid oxidation. Am J Physiol Gastrointest Liver Physiol 2006;290(5):G852–G858.
11. Riestra-Candelaria BL, Rodriguez-Mojica W, Jorge JC. Anatomical criteria to measure the adult right liver lobe by ultrasound. Sonography 2018;5(4):181–186.
12. Weir CB, Jan A. BMI Classification Percentile and Cut Off Points. 2023 Jun 26. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024.
13. Petroni ML, Brodosi L, Marchignoli F, et al. Moderate alcohol intake in non-alcoholic fatty liver disease: to drink or not to drink? Nutrients 2019;11(12):3048.
14. Li Y, Chen Y, Zhao Y. The diagnostic value of the FIB-4 index for staging hepatitis B-related fibrosis: a meta-analysis. PLoS One 2014;9(8):e105728.
15. De Matteis C, Cariello M, Graziano G, et al. AST to platelet ratio index (APRI) is an easy-to-use predictor score for cardiovascular risk in metabolic subjects. Sci Rep 2021;11(1):14834.
16. Goel A. Diffuse hepatic steatosis (grading). Radiology Reference Article. Radiopaedia.org [Internet]. Radiopaedia. Available from: https://radiopaedia.org/articles/diffuse-hepatic-steatosis-grading.
17. Venkatesh SK, Ehman RL. Magnetic resonance elastography of liver. Magn Reson Imaging Clin N Am 2014;22(3):433–446.
18. de Lédinghen V, Vergniol J. Transient elastography for the diagnosis of liver fibrosis. Expert Rev Med Devices 2010;7(6):811–823.
19. Shao M, Wang Y, Dong H, et al. From liver fibrosis to hepatocarcinogenesis: role of excessive liver H_2O_2 and targeting nanotherapeutics. Bioact Mater 2022;23:187–205.
20. Bataller R, Brenner DA. Liver fibrosis. J Clin Invest 2005;115(2):209–218.
21. Afdhal NH. Fibroscan (transient elastography) for the measurement of liver fibrosis. Gastroenterol Hepatol (N Y) 2012;8(9):605–607.
22. Venkatesh SK, Wang G, Lim SG, et al. Magnetic resonance elastography for the detection and staging of liver fibrosis in chronic hepatitis B. Eur Radiol 2014;24(1):70–78.
23. Lee YS, Lee JE, Yi HS, et al. MRE-based NASH score for diagnosis of nonalcoholic steatohepatitis in patients with nonalcoholic fatty liver disease. Hepatol Int 2022;16(2):316–324.