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REVIEW

Use of liver stiffness measurements in acute decompensated heart failure: new applications of a non-invasive technique

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Abstract

Heart failure (HF) is a complex disease associated with multisystem organ failure, recurrent hospital admissions, and increased mortality. Acute decompensated heart failure (ADHF) increases central venous pressure (CVP) with resultant hepatic congestion, and this relationship has prognostic significance. The gold standard method of measuring CVP, right heart catheterization, is invasive and costly, prompting further investigation into more accurate non-invasive assessments in HF patients, including liver elastography. Liver elastography relies on imaging techniques to assess liver stiffness measurements (LSM), with high values equating to increased stiffness. While this was developed to assess fibrosis in liver disease, LSM also reflect increased CVP and hepatic congestion. Multiple studies involving ADHF patients, find that increased LSM are independently predictive of increased cardiac events, all-cause mortality, and worse post-operative outcome after both acute HF exacerbation and left ventricular assist device (LVAD) placement. In this review, we discuss the role of LSM as a surrogate for CVP and their applications in determining prognosis in both the ADHF and LVAD populations.

Keywords Heart failure; Liver stiffness measurement; Elastography; Mechanical circulatory support; Heart transplantation; Liver fibrosis

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Introduction

Heart failure (HF) is a complex disease associated with multisystem organ failure, recurrent hospital admissions, and increased mortality. Acute decompensated heart failure (ADHF) increases central venous pressure (CVP) with resultant hepatic congestion, and this relationship has prognostic significance.[1] The gold standard method of measuring CVP, right heart catheterization (RHC), is invasive and costly, prompting further investigation into more accurate non-invasive assessments in HF patients, including liver elastography. Liver elastography relies on imaging techniques to assess liver stiffness measurements (LSM), with high values equating to increased stiffness.[2] While this was developed to assess fibrosis in liver disease, LSM also reflect increased CVP and hepatic congestion.[3–7] Multiple studies involving ADHF patients, find that increased LSM are independently predictive of increased cardiac events, all-cause mortality, and worse post-operative outcome after both acute HF exacerbation and left ventricular assist device (LVAD) placement.[4–6,8–11] In this review, we discuss the role of LSM as a surrogate for CVP and their applications in determining prognosis in both the ADHF and LVAD populations.

Pathophysiology of congestive hepatopathy and liver stiffness

Congestive hepatopathy (CH) is the result of chronic passive venous congestion as CVP elevation in right-sided HF (RHF) is transmitted to the hepatic (central) veins of the liver. This

gives rise to pre-sinusoidal dilation, decreased hepatic artery blood flow, and decreased arterial oxygen saturation, which can ultimately result in irreversible congestive liver fibrosis and cardiac cirrhosis.[12] While CH is reversible with treatment of HF, baseline liver dysfunction has been associated with increased morbidity and mortality in HF patients, generally portending a poor prognosis.[13,14]

Histologically, CH is characterized by dilation of the lobular hepatic veins and hepatic sinusoids, perisinusoidal oedema, acinar steatosis and heterogeneous fibrosis.[15] True cardiac cirrhosis is relatively uncommon due to this heterogeneity, with many patients failing to demonstrate extensive fibrosis or regenerative nodule formation.[12,16]

CH is often clinically silent, although patients may experience right upper quadrant pain from stretching of the liver capsule, and exhibit a pulsatile liver on physical exam.[13] Bilirubin and gamma-glutamyl transferase levels may be elevated, particularly in patients with cardiac index (CI) < 1.5 L/min/m^2, with improvement after decongestion.[16] Ultrasound findings include hepatomegaly, IVC and hepatic vein dilation with diminished respiratory variation, and retrograde portal vein flow with phasic changes.[12,15] Cross-sectional CT similarly shows hepatomegaly and venous dilation, along with reflux of contrast into the IVC and hepatic veins in the arterial phase, delayed parenchymal enhancement in the venous phase, and, with chronic CH, hepatic venous shunting.[15]

As the liver is surrounded by a non-elastic capsule, congestion and fibrosis lead to increased liver stiffness.[1] Assessing cardiac cirrhosis in HF patients is difficult, as the histologic features are disparate and may not correlate with HF severity or chronicity.[17] The Congestive Hepatic Fibrosis Score (CHFS) was developed for grading patterns of fibrosis on liver biopsy, ranging from Stage 1 (central zone fibrosis) to Stage 4 (cirrhosis).[18] On the validation study, higher stages were associated with higher right atrial pressure, which was assumed to indicate more severe HF.[18] However, subsequent studies using the CHFS reliably found evidence of congestion but had low interobserver agreement in diagnosing fibrosis.[19–21] Previous groups have also attempted to characterize fibrosis in CH by incorporating non-invasive testing (NIT) validated for chronic liver diseases, such as Fibrosis-4 score, FibroTest/FibroSURE, and hyaluronic acid, but the data in cardiac patients have shown poor correlation between NIT, LSM and actual liver fibrosis.[22] This is not surprising, because CH is a distinct pathophysiologic process in which quantifying fibrosis is difficult even on biopsy.[20] Translating the results of non-invasive imaging tests in HF patients is not expected to yield useful information about fibrosis. However, there seems to be some correlation to elevated CVP. LSM may therefore be a better surrogate of hepatic congestion in HF patients.

Non-invasive assessments of liver stiffness

Several non-invasive imaging modalities for assessing LSM have been studied for use in liver disease, including vibration-controlled transient elastography (VCTE), point shear wave elastography (pSWE, which includes acoustic radiation force impulse (ARFI) elastography and ElastPQ), and magnetic resonance elastography (MRE). Originally validated for the assessment of liver fibrosis in cirrhosis, both VCTE and pSWE use an ultrasound probe to send shear waves through the liver, and results are correlated with histologic METAVIR (Meta-analysis of Histological Data in Viral Hepatitis) fibrosis staging (*Table 1*). In VCTE, the velocity of this wave is converted into kiloPascals (kPa), which correlates to stiffness.[2] In ARFI, the shear waves are pulsed in a pre-determined vessel-free region of interest (ROI) on ultrasound, and the speed of displacement is calculated in meters per second (m/s) by the Virtual Touch software, with increased speed correlating to increased stiffness.[2] In contrast to the acoustic pulse used in ARFI, ElastPQ transmits an electronic voltage pulse to a transducer, where it is converted into an ultrasonic pressure wave and sent into the ROI, after which the Doppler frequencies are correlated to liver stiffness.[23] MRE requires placement of an external driver device onto the patient that generates mechanical shear waves, which are captured with a modified phase-contrast magnetic resonance sequence to create quantitative images of liver stiffness, called 'elastograms', that depict LSM in kiloPascals.[2] In the ADHF literature, VCTE and pSWE have been most commonly used, while MRE has only been evaluated by one group[24] and has been more extensively studied in the congenital heart disease (CHD) population.[25–27]

Table 1 Comparison of VCTE and ARFI for evaluation of liver fibrosis in patients with hepatitis C2

Modality	Brand	Stage ≤F2 (no significant fibrosis)[a]	Stage >F3 (advanced fibrosis/cirrhosis)[a]	Confounders
VCTE	FibroScan (Echosens)	<7 kPa	>15 kPa	Meal intake, obesity, ascites, steatosis, inflammation, passive congestion, extrahepatic cholestasis
ARFI	Siemens ACUSON S2000	<1.34 m/s	>2.2 m/s	Steatosis, inflammation, passive congestion, extrahepatic cholestasis

ARFI, acoustic radiation force impulse; VCTE, vibration-controlled transient elastography.
[a]Fibrosis as described in METAVIR staging guidelines.

Advantages of VCTE include low cost, point-of-care use in the clinic and a wealth of validated data as compared with ARFI; although it is confounded by obesity and ascites due to lack of direct visualization of the ROI.[28] Advantages of pSWE include higher rates of reliable measurements and less interference by obesity and ascites, as the ROI can be visualized beforehand; although it is hindered by the need for the Virtual Touch software to be downloaded onto the ultrasound machine and review by a radiologist.[28] A meta-analysis comparing VCTE and ARFI in liver disease found a similar predictive value for fibrosis and cirrhosis, but ARFI may have higher rates of reliable measurements.[29] However, no head-to-head comparisons have been done in the HF population. Further comparison is described in *Table 1*. Various conditions confound LSM, including steatosis, hepatitis, and extrahepatic cholestasis.[30,31]

While MRE has not been extensively studied in the ADHF population, in the non-alcoholic fatty liver disease literature the larger assessment area of MRE has exposed the heterogeneity of liver fibrosis throughout the liver, and this heterogeneity would be expected in the ADHF population also.[32,33] ARFI visualizes an ROI prior to LSM capture, and while both ARFI and VCTE use standard anatomic locations for assessment, the validity of an isolated LSM may be misleading in a liver of heterogenous stiffness given the much smaller assessment area. However, liver biopsy, the gold-standard for fibrosis staging, is similarly bound by this limitation.

Liver stiffness measurements have expanded into assessing the development of fibrosis after the Fontan procedure, in which systemic venous return is diverted to the pulmonary arterial system and the morphologic right ventricle assumes responsibility of arterial perfusion, which invariably leads to congestive heart failure, chronic passive hepatic congestion, and cirrhosis over time.[34] Higher LSM may be associated with unfavourable Fontan haemodynamics and hepatic fibrosis on liver biopsy and ultrasound.[19,20,35] However, there is a paucity of data from large series correlating LSM with the presence of fibrosis; particularly of advanced stage fibrosis.[34]

Lebray *et al*. noted elevated LSM in a HF patient without clinical or pathologic evidence of cirrhosis, positing passive congestion as a confounder of LSM.[35] Millonig *et al*. was the first to correlate FibroScan LSM and central venous pressure by clamping the IVC in Landrace pigs, finding a linear stepwise increase in LSM as intravenous hydrostatic pressure increased ($r = 1$, $P < 0.01$).[36] Jalal *et al*. similarly found a correlation between LSM and CVP in the CHD population.[37] This relationship between elastography and passive congestion has the focus of LSM as a marker of hepatic congestion in the setting of HF.

Liver stiffness measurements as a surrogate marker for central venous pressure

Multiple studies have demonstrated elastography to be a non-invasive surrogate for CVP as measured by RHC (*Table 2*). Yoshitani *et al*. found a positive relationship using ARFI, with CVP being an independent predictor for increased LSM on multivariate analysis.[7] Nishi *et al*. and Potthoff *et al*. both studied HF patients after LVAD placement using FibroScan and ARFI, respectively, and also found a linear relationship between pre-operative CVP and pre-operative LSM.[4,6] Outside of the HF population, Jalal *et al*. also found a correlation between CVP on RHC and LSM on FibroScan in adult and paediatric patients with congenital heart disease.[37]

These studies consistently found RHC and LSM having a baseline correlation, establishing LSM as a non-invasive tool to assess hydrostatic pressure. Few studies compared correlations in CVP with other laboratory and echocardiographic indicators of elevated pressure alongside LSM. Taniguchi *et al*. found FibroScan had improved sensitivity and accuracy in detecting RAP >10 mmHg as compared with IVC measurements on echocardiography.[3] Jalal *et al*. found LSM to have a better correlation with CVP than brain natriuretic peptide (BNP).[37]

Elastography can demonstrate decongestion in acute decompensated heart failure

Inadequate decongestion on discharge for ADHF is associated with increased morbidity and mortality.[38] Nevertheless, many patients are discharged with residual congestion due to the

Table 2 Correlation between invasively measure central venous pressure and liver stiffness measurements

Study	Modality	Sample size (*n*)	Study population	LSM[a]	Measured CVP (mmHg)	Correlation (*r*)	*P* value
Taniguchi *et al*.[3]	FibroScan	31	Decompensated HF	8.5 (5.3–12.0) kPa	9.0 (5.0–12.0)	0.95	<0.001
Nishi *et al*.[4]	FibroScan	30	LVAD recipients	13.3 ± 13.0 kPa	8.8 ± 6.9	0.515	<0.01
Kashiyama *et al*.[5]	FibroScan	55	LVAD recipients	12.7 ± 13.1 kPa	7.4 ± 5.0	0.52	<0.01
Potthoff *et al*.[6]	ARFI	28	LVAD recipients	2.50 ± 0.92 m/s	14.0 ± 6.0	0.793	0.001
Yoshitani *et al*.[7]	ARFI	38	Decompensated HF	2.03 ± 0.91 m/s	11.8 ± 5.4	0.636	0.014

ARFI, acoustic radiation force impulse; CVP, central venous pressure; HF, heart failure; kPa, kilopascals; LSM, liver stiffness measurements; LVAD, left ventricular assist device; m/s, meters per second.
[a]Values are expressed as mean ± standard deviation or median (interquartile rage).

lack of an objective measurement of HF.[39] Several studies evaluated BNP as a prognostic factor in HF and showed benefit in BNP-guided HF treatment.[40–42] However, after an acute exacerbation, BNP normalization does not follow a predictable pattern. As RHC is invasive, several studies have turned to serial LSM to assess decongestion after ADHF.

Various studies have compared admission and discharge LSM in patients admitted for ADHF (*Table 3*). All but one of the six studies found a significant decrease in LSM after diuresis. A study that compared LSM in patients with normal cardiac function, stable left HF (LHF), stable RHF, and ADHF, found that all HF groups had significantly higher LSM than controls, with the ADHF group also having significantly higher LSM than the stable LHF group (median 11.2 vs. 4.7 kPa, $P = 0.01$).[6] Median LSM also correlated significantly with N-terminal pro B-type natriuretic peptide (NTproBNP), RAP and right ventricular pressure on echocardiogram.[44] Alegre *et al*. similarly saw significantly higher FibroScan LSM in ADHF patients as compared with stable biventricular HF patients.[45] LSM significantly decreased in the ADHF group after diuresis, reaching comparable levels to the stable HF groups [median 8.2 (5.1–11.2) kPa vs. median 6.5 (5.0–10.8) kPa)].[45]

When compared with other non-invasive markers of CH, LSM more accurately demonstrated decongestion. Yoshitani *et al*. compared total bilirubin, AST, ALT, and GGT before and after diuresis and found no significant change, whereas body weight, LSM, and BNP all significantly decreased.[7] While CVP was shown to be an independent predictor for changes in LSM, other markers of decongestion (body weight, BNP, and PCWP) did not correlate with changes in LSM despite significant improvement.[7] Two studies found LSM and NTproBNP were both significantly reduced after sufficient diuresis.[45] In a cross-sectional study, Hopper *et al*. demonstrated that increased LSM correlated with increased bilirubin, GGT and alkaline phosphatase in LHF, RHF, and ADHF groups.[44] This was also the only study that showed correlation of median LSM and NTproBNP ($r = 0.24$, $P = 0.01$), but failed to show a significant change in LSM after adequate diuresis.[44] Together, these data confirm the known clinical course of CH, in which liver markers vary and are typically unreliable despite larger shifts in body fvolume, and support LSM as a superior tool.

The inability to determine accurate reference ranges to assess for adequate decongestion is a major limitation. While most studies show a significant decrease in LSM after diuresis, a standard has not been established, and it is unclear if residual abnormalities in LSM reflect congestion or underlying fibrosis. As such, there may be discordance between liver stiffness and congestion in heart failure patients. Changes in LSM with therapy may be more useful than the absolute value of any particular measurement, as it is not possible to distinguish if high values represent congestion or fibrosis. Future studies should focus on individualized use of LSM to establish baseline diuresis goals for each patient prior to safe discharge.

Liver stiffness measurements as a prognostic tool in acute decompensated heart failure

İçen *et al*. found LSM on ElastPQ increased with increasing NYHA class, supporting elastography's ability to discern higher-risk patients.[46] Four groups used elastography to predict prognosis in ADHF, and found LSM to be an independent predictor for adverse events (*Table 4*). Saito *et al*. used FibroScan to categorize ADHF patients into low LSM (<8.8 kPa) and high LSM (≥8.8 kPa) groups based on the median LSM on admission, with primary endpoints of death from cardiovascular disease (CVD) and readmission for HF after a median follow up of 153 days. In the 40% of patients with cardiac events (11 CVD deaths and 31 HF readmissions), the high LSM group had significantly higher rates of composite events ($P = 0.001$) and readmission ($P = 0.022$). High LSM were the only independent risk factor for cardiac events, and not echocardiographic and serologic data.[8]

Omote *et al*. used ARFI to assess prognosis in patients admitted for ADHF, segregating patients into low and high LSM groups. Adverse events were observed more frequently in the high LSM group compared with the low LSM group.

Table 3 Changes in liver stiffness measurements after intervention

Study	Modality	Intervention	Sample size (n)[a]	LSM before intervention[b]	LSM after intervention[b]	P value
Millonig *et al*.[38]	FibroScan	Diuresis	10	40.7 (6.1–51.3) kPa	17.8 (3.3–33.2 kPa)	0.004
Colli *et al*.[43]	FibroScan	Diuresis	27	8.80 (5.92–11.90) kPa	7.20 (5.2–11.30) kPa	0.003
Hopper *et al*.[44]	FibroScan	Diuresis	8	11.2 (6.7–14.3) kPa	9.5 (7.3–21.6) kPa	>0.09
Alegre *et al*.[45]	FibroScan	Diuresis	9	14.7 (8.3–18.8) kPa	8.2 (5.1–11.2) kPa	0.008
Soloveva *et al*.[10]	FibroScan	Diuresis	149	12.2 (6.3–23.6) kPa	8.7 (5.9–14.4) kPa	<0.001
Yoshitani *et al*.[7]	ARFI	Diuresis	14	2.37 ± 1.09 m/s	1.27 ± 0.33 m/s	<0.001
Potthoff *et al*.[6]	ARFI	LVAD placement	23	1.88 (0.92–3.72) m/s	1.43 (0.93–3.67) m/s	<0.001

ARFI, acoustic radiation force impulse; kPa, kilopascals; LSM, liver stiffness measurements; LVAD, left ventricular assist device; m/s, meters per second.
[a]Sample sizes may differ from *Table 2* due to variability in obtaining post-intervention LSM.
[b]Values are expressed as mean ± standard deviation or median (interquartile range).

Table 4 Summary of LSM as an independent risk factor for adverse events

Study	Modality	Sample size (*n*)	Cut-off for high LSM group	Composite endpoints	Number of events (*n*)	Mean follow-up time (days)	Independent risk factor(s) for event
Saito *et al*.[8]	FibroScan	105	≥8.8 kPa (on admission)	Death from CV disease, readmission for HF	42	153	LSM
Omote *et al*.[9]	ARFI	70	≥1.50 m/s (on admission)	All-cause death, worsening HF	26	272	SBP
							LSM
Taniguchi *et al*.[11]	FibroScan	171	>6.9 kPa (on discharge)	Cardiac death, readmission for HF	41	203	LSM
Soloveva *et al*.[10]	FibroScan	149	>13 kPa (on admission) ≥5 kPa (on discharge)	All-cause death, heart transplant, HF readmission	71	289	LSM at discharge

ARFI, acoustic radiation force impulse; CV, cardiovascular disease; HF, heart failure; kPa, kilopascals; LSM, liver stiffness measurements; m/s, meters per second; SBP, systolic blood pressure.

Systolic BP and high LSM were independently associated with increased risk of adverse events on multivariate analysis.[9]

Taniguchi *et al*. evaluated discharge FibroScan LSM to assess for outcomes, separating the patients into tertiles, with cut-offs at 4.6 and 6.9 kPa, corresponding to estimated RAP of 4.6 and 7.1 mmHg, respectively. The highest tertile had significantly more cardiac events and was an independent risk factor for worse outcome on multivariate analysis. ROC curve analysis determined LSM > 10.1 kPa (sensitivity of 0.73, specificity of 0.90) as the optimal cut-off for predicting short-term cardiac events.[11] LSM increased the C-statistic model of age, sex, eGFR, and BNP from 0.704 to 0.844 (P = 0.006).[11] This study validates LSM as an independent risk factor for adverse events, enhances current prognostic models for HF, and defines a cut-off value for determining increased risk, increasing its clinical applicability in risk-stratification in HF.

Soloveva *et al*. evaluated FibroScan LSM on admission and prior to discharge, finding a significantly higher probability of negative outcomes associated with LSM >13 kPa on admission and ≥5 kPa at discharge. Discharge LSM independently predicted HF readmission and were associated with worse composite endpoints and all-cause death. Post-mortem liver biopsy was performed in seven patients, and discordance was found between LSM and histologic grading of fibrosis in 4 patients, confirming poor correlation between LSM and liver pathology.[10]

Soloveva *et al*. and Saito *et al*. both reported elevated total bilirubin and no significant difference in BNP in the higher LSM groups, whereas Taniguchi *et al*. found the opposite.[8,10] Soloveva *et al*. and Taniguchi *et al*. both found discharge LSM to have a high probability of predicting adverse events, supporting LSM as a clinically relevant tool to assess adequate decongestion prior to discharge.[10,11] As elastography is not able to differentiate residual congestion from underlying fibrosis, the driving factors behind LSM as a prognostic tool requires further investigation. However, this distinction may not be as important as its applicability in risk-stratifying patients for more urgent transplant referral and advanced interventions. Future directions should include further elaboration of optimal timing of LSM for prognostic purposes and determining appropriate cut-off values for stratifying increased risk.

Liver stiffness measurements as a prognostic tool after left ventricular assist device placement

As LVADs become increasingly common therapy in advanced heart failure, so has awareness of ensuing RHF which is a common complication and marker of poor survival. RHF after LVAD is attributed to the inability of the failing right heart to handle enhanced left-sided cardiac output, excessive

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left-ward shift of the interventricular septum, and altered haemodynamics worsening tricuspid regurgitation.[47] This typically occurs within 2 weeks of LVAD placement and is associated with increased ICU needs and overall poor prognosis.[48] No single marker or risk algorithm has significant predictive value for post-LVAD complications, although measurements such as NTproBNP, CVP, pulmonary artery pulsatility index (PAPi), right ventricular stroke work index (RVSWI), and the CVP to pulmonary capillary wedge (CVP/PCWP) ratio are commonly used to evaluate need for RVAD implantation and tricuspid valve replacement prior to surgery.[47–49] As advanced heart failure is typically characterized by biventricular disease with elevated CVP, three groups have evaluated elastography as an additional prognostic tool.

Potthoff *et al*. first characterized ARFI LSM after LVAD placement, finding a positive correlation between LSM and CVP, and significant decreases in LSM post-operatively, indicating improvement in CVP. LSM were significantly higher among the 5 patients that died compared with survivors, positioning LSM as a possible prognostic tool.[6]

Using FibroScan to assess LVAD candidates, Nishi *et al*. found LSM were significantly higher in patients who also required RVADs. ROC analysis identified a cut-off ≥7.0 kPa for increased RVAD need. Patients with major adverse events (MAEs: mortality, postoperative bleeding, cerebrovascular events and infection) had significantly higher LSM than those without (22.4 ± 17.4 vs. 8.0 ± 5 kPa, $P < 0.05$), and MAEs were particularly greater in patients with LSM ≥12.5 kPa (80% vs. 25% in LSM < 12.5 kPa).[4] While multiple markers of CH were evaluated, including pre-operative haemodynamic measurements, BNP, and transaminases, LSM were the only independent risk factor for MAEs.[4]

Kashiyama *et al*. evaluated VCTE LSM in 55 patients after LVAD placement, with endpoints of heart transplantation, death, or LVAD removal.[5] Univariate analysis identified dilated phase of hypertrophic cardiomyopathy aetiology, pre-operative LSM, LVEF, left ventricular diastolic dimension (LVDD), RVSWI, and CVP/PCWP ratio as predictive for RHF after LVAD placement. In multivariate analysis, all but LVEF and CVP/PCWP were predictive, with the combination of these factors having the highest predictive value (sensitivity 100%, specificity 64%, $P < 0.01$).[5] A pre-operative cut-off LSM ≥ 12.8 kPa was predictive of RHF. Pre-operative LSM and LVEF were independent risk factors for RVAD need, with a cut-off LSM ≥ 14.0 kPa for increased likelihood, far higher than the cut-off previously established.[4,5] Prior studies failed to find a significant correlation between LSM and liver function, although this group found a correlation between LSM and total bilirubin level.[5] While this does not obviate liver fibrosis from impacting LSM, it highlights the prognostic ability of elastography as an independent risk factor for adverse events following LVAD placement, and as a tool to augment existing predictors of poor outcome. This study also demonstrated the potential for non-invasive risk calculators, based on a subset of imaging and serologic tests, as an alternative to invasive procedures. However, this is the only published study assessing this relationship, and further research is needed.

Limitations of elastography and future directions

Expanding study of LSM into larger trials is limited by several factors. Although VCTE (FibroScan) is readily accessible and requires minimal training for use, it is primarily employed by hepatologists and gastroenterologists, while cardiologists are generally unaware of its applications for the ADHF population. While ARFI is similarly accessible, it requires interpretation by radiologists, most of whom may not have much experience in the context of heart failure patients. In terms of study design, sample sizes are limited by the exclusion criteria, which eliminates patients with known liver disease, ascites, and obesity, especially for VCTE. Due to ethical considerations in obtaining RHC and liver biopsy in these patients, more accurate assessments of CVP changes and fibrosis are limited. Due to this limitation, a single LSM value cannot differentiate reversible congestion from irreversible fibrosis.

The ability to quantify hepatic decongestion is a key strength of LSM that warrants further evaluation. Multiple studies reviewed here have demonstrated LSM are a surrogate for CVP, as LSM decrease linearly with CVP after adequate diuresis. While a single measurement will not accurately demonstrate the severity of ADHF, the change in LSM on admission and after diuresis is easily calculated and has significant value in the management of the individual patient. As with the chronic liver disease population, it will be necessary to further define the relationship between CVP and LSM in a multi-centre study with a larger number of patients. Focusing on the change in LSM will also allow more accurate comparisons of successful treatment of ADHF across different facilities using different LSM modalities.

The other strength of LSM is risk stratification in the ADHF and transplant populations, with multiple studies establishing their own optimal cut-off LSM for increased risk. These cut-offs should be validated in a larger multi-centre trial to further explore the application of LSM as a prognostic marker both alone and in conjunction with existing markers of disease severity. However, cut-off values cannot be extrapolated between the ADHF and transplant populations, as they represent two clinically distinct entities.

As LSM are a new area of study in the HF population, head-to-head comparisons of the various LSM modalities have not been established as they were for the liver disease population and warrants larger trials.[2,50] As such, a LSM obtained

by one imaging modality cannot be directly compared with a LSM obtained by another modality.

Conclusion

The HF population is a high-risk group subject to significant morbidity and mortality. Identifying the highest risk patients remains a challenge, and further research into enhanced assessments is critical. Liver elastography is emerging as a non-invasive surrogate for evaluating the increased venous congestion characteristic of heart failure, with several groups reporting novel use as a prognostic tool in both the ADHF and LVAD populations. The existing data illuminate three key applications: (i) assessment of adequate venous decongestion prior to discharge; (ii) prognosis after an acute exacerbation; and (iii) risk stratification for determining right ventricular support needs before LVAD placement. Elastography is prognostic both alone and in conjunction with existing markers which, given that it is relatively inexpensive and easy to use, has vast potential. While further research is needed to fully illustrate its practical applications, liver elastography shows great promise as a novel tool to improve outcomes in the management of heart failure population.

Conflict of interest

The authors report no financial conflict of interest related to this submission.

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