Shear wave elastography to predict oesophageal varices, morbidity and mortality in chronic liver disease

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ABSTRACT

INTRODUCTION: In chronic liver disease (CLD), Fibroscan® (transient elastography) can be a useful "rule-out" test for oesophageal varices, but it is limited by body habitus. Shear wave elastography (SWE) is another non-invasive fibrosis test that is better suited for overweight subjects. We determined SWE's ability to predict oesophageal varices, morbidity and mortality in a predominantly overweight population.

METHODS: Subjects (n=1,120) with CLD who underwent SWE at Middlemore Hospital between 1 July 2015 and 30 June 2018 were identified. The diagnostic accuracy of SWE to rule out oesophageal varices in advanced hepatic fibrosis was assessed, as well as associations with morbidity and mortality.

RESULTS: Of 304 subjects with advanced fibrosis, 89 had endoscopic data and 18 had varices. Median body mass index was 28.2kg/m^2. Area under the receiver operating characteristic curve value for liver stiffness to predict varices was 0.74 and 0.80 when combined with serum albumin. Liver stiffness ≤12.4kPa and albumin ≥37g/L had a negative predictive value of 95%. There were 135 hospital admissions and 19 deaths. Liver stiffness correlated with hospital admissions (p=0.007) and independently predicted mortality.

CONCLUSIONS: Shear wave elastography could be a useful rule-out test for screening endoscopy in overweight populations with CLD.

Chronic liver disease (CLD) is a growing problem affecting approximately fifty million people worldwide.[1] Advanced fibrosis in CLD is associated with the development of oesophageal varices (OV).[1,2] Variceal bleeding is a life-threatening event associated with a mortality of 25% to 50% and occurs in up to 30% of cirrhotic patients within the first two years.[3] Routine endoscopic screening for OV in all cirrhotic patients is the widely accepted gold standard, but it is invasive and resource-intensive.[4] It is also not available outside specialised centres.

Elastography, which is based on the liver stiffness measurement (LSM), is an excellent tool for predicting liver fibrosis.[5] There are two main methods: transient elastography (TE, Fibroscan®) and shear wave elastography (SWE). Recently, FibroScan-based LSM has been shown to be useful in ruling out the presence of OV and has been incorporated into international guidelines for this purpose.[2,6–12] However, the reliability of FibroScan is significantly impaired in subjects with a high body mass index (BMI).[12] New Zealand has the third highest rate of adult obesity in the OECD, with 30.9% of adults being classified as obese.[13] Two-dimensional (2D) SWE is an ultrasound-based tool with the advantage of direct anatomical visualisation of the region of evaluation.[14,15] It is also more reliable in subjects with a higher BMI.[14] However, there is a paucity of data on the utility of SWE-derived LSM (SWE-LSM) for predicting the presence of OV, morbidity and mortality. There is also little data in Asia–Pacific populations with a high prevalence of Māori, Pasifika and Asian subjects.

The primary aim of this study was to determine the diagnostic accuracy of SWE-LSM to "rule out" the presence of OV in the real-world setting of a large tertiary centre with a predominantly overweight CLD population and a high prevalence of Māori, Pasifika and Asian subjects. Secondary aims included determining the

association of SWE-LSM with morbidity (hospital admissions) and mortality.

Methods

Study population

This retrospective cohort study analysed data from adult subjects who had a SWE scan performed at a tertiary hospital (Middlemore Hospital, Auckland, New Zealand) between 1 July 2015 and 30 June 2018. Inclusion criteria were: (a) a SWE scan performed for the indication of chronic liver disease, (b) a valid SWE-LSM result and (c) advanced fibrosis (LSM of ≥8.1kPa, ≥10.2 kPa for alcohol-related liver disease (ALD) and ≥9.2kPa for all other aetiologies).[16,17] All scans were performed by experienced operators (hepatology nurse specialists and gastroenterologists) using the Aixplorer® 2D SWE imaging system (SuperSonic Imagine, France).

Data collection

Ethics approval was received from the Health and Disability Ethics Committee (Reference: 18/NTB/2). Subjects meeting the inclusion criteria were identified from an existing clinical database. Data collected included: (a) population characteristics (age, sex, ethnicity, body mass index, aetiology of liver disease and beta blocker use); (b) serum markers (serum biochemistry (alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma glutamyl transferase (GGT), alkaline phosphatase (ALP), bilirubin, serum albumin, sodium and creatinine), complete blood count (haemoglobin, platelet count) and prothrombin ratio; and (c) endoscopic findings (the presence and size of oesophageal varices (absent, small (<5mm) or large (>5mm))). Biochemical data were used to derive model for end-stage liver disease (MELD) scores. The maximum interval permitted between SWE and other findings was three months for biochemical parameters and 12 months for endoscopy. Subjects were followed for up to three years for number of all-cause hospital admissions, liver disease-related hospital admissions, infection-related hospital admissions and mortality.

Analysis

Data were analysed in two subgroups: (a) subjects with endoscopic data and (b) all subjects meeting the inclusion criteria over the study period. Statistical analyses were performed using IBM Statistical Package for Social Sciences (SPSS) version 25. The Shapiro–Wilk test was utilised to test for normality. Non-normally distributed variables were presented as medians with inter-quartile ranges and analysed using Spearman's rho, Mann–Whitney U, Kruskal–Wallis and Fisher's Exact tests. The utility of SWE to rule out the presence of OV was assessed using receiver operating characteristic (ROC) curves. The utility of SWE to predict morbidity and mortality was assessed using logistic and cox regression analyses. Statistical significance was defined by $p<0.05$.

Results

Population characteristics

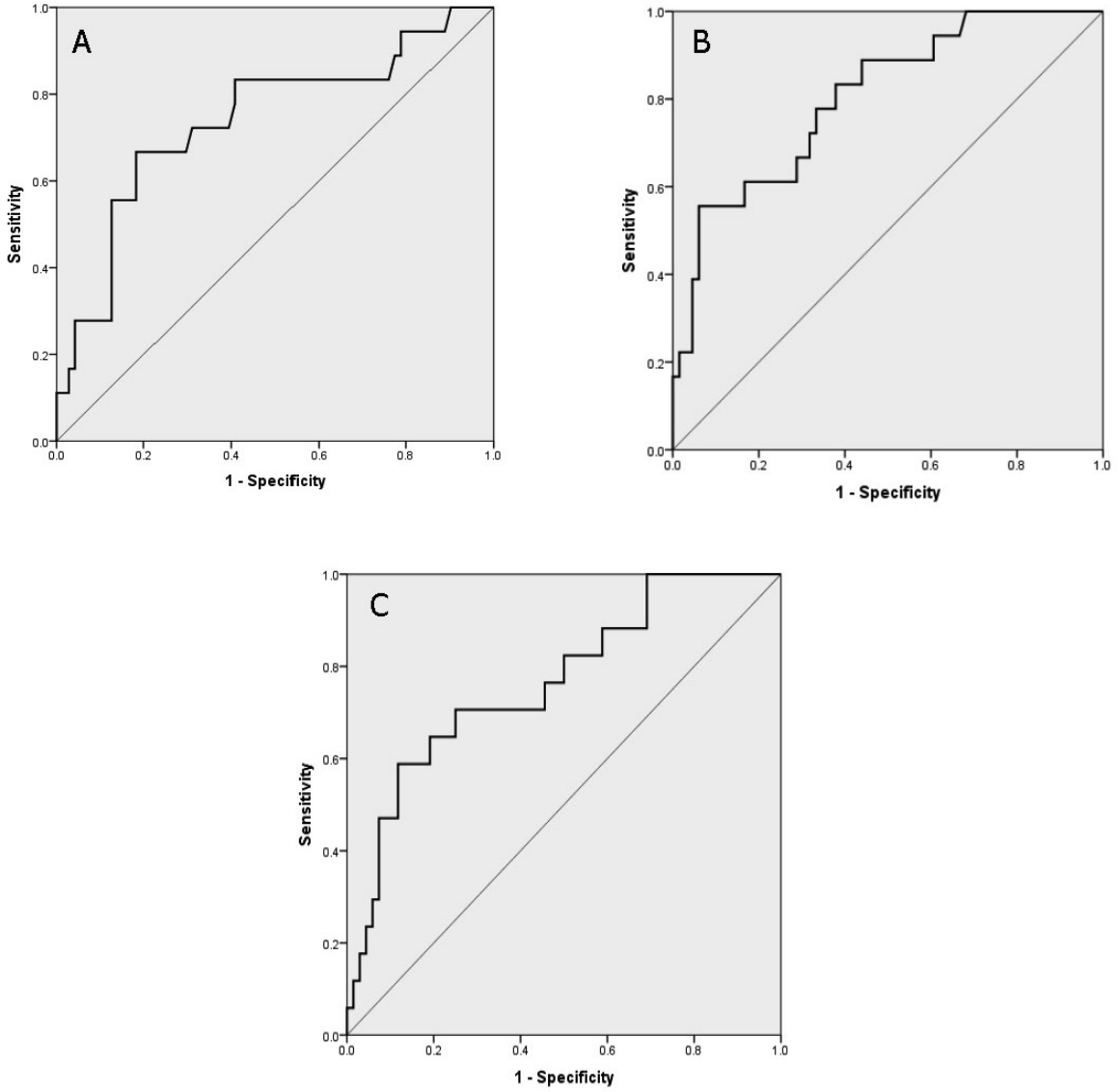
Of 1,120 subjects who underwent SWE scans within the study period, 304 subjects had advanced hepatic fibrosis and were included for analysis. Baseline characteristics of the total population are listed in Table 1. Overall, the most common aetiology was chronic hepatitis B (31.3%), followed by non-alcoholic fatty liver disease (24%) and chronic hepatitis C (23.7%). The median LSM score was 11.7kPa. The majority of subjects were overweight with 69.8% having a BMI >25. The median BMI was 28.2kg/m^2. Māori and Pasifika comprised 30.9% of the population, and Asian subjects accounted for 30.6%. Biochemical parameters indicated that the subjects had largely compensated liver disease.

SWE to predict the presence of OV

Eighty-nine subjects had endoscopies within the defined time-period. Of these, 18 had OV. Out of the 18 subjects with OV, 14 had grade 1 varices, two had grade 2 varices and two had grade 3 varices. Baseline demographics of the endoscopy population are listed in Table 2. The subjects with OV were older and had a higher prevalence of alcohol-related liver disease. Other baseline, clinical and biochemical characteristics were similar. No subjects had a bleeding event following a SWE-LSM.

Subjects with OV had a significantly higher median SWE scores when compared to subjects without OV (21.5kPa vs 13.5kPa, p=0.002, Table 2). This finding was not affected by interval between SWE and endoscopy (p=0.10) and differences in beta-

Figure 1: Diagnostic performance of SWE-LSM for prediction of OV. (A) SWE-LSM (AUROC 0.74, p=0.02), (B) SWE-LSM and serum albumin (AUROC 0.80, p<0.001) and (C) SWE-LSM and platelet count (AUROC 0.77, p=0.01).



Abbreviations: SWE-LSM (shear wave elastography-derived liver stiffness measurement), OV (oesophageal varices), AUROC (area under the receiver operating characteristic curve).

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Table 1: Population characteristics of the total population (n=304).

Descriptor	Value
Population characteristics	
Median age [years (IQR)]	58 (50–66)
Sex [n (%)]	
Male	214 (70.4)
Female	90 (29.6)
Ethnicity [n (%)]	
European	107 (35.2)
Māori	41 (13.5)
Pacific	53 (17.4)
Asian	93 (30.6)
Other	10 (3.3)
Aetiology [n (%)]	
Hepatitis B virus	95 (31.3)
Hepatitis C virus	72 (23.7)
Non-alcoholic fatty liver disease	73 (24.0)
Alcohol	30 (9.9)
Other	34 (11.2)
Median BMI [(IQR)]	28.2 (24.3–32.3)
BMI<25 [n (%)]	91 (31.2)
BMI 25-30 [n (%)]	86 (29.5)
BMI>30 [n (%)]	115 (39.4)
Beta-blocker use [n (%)]	43 (14.1)
SWE	
Median SWE-LSM [kPa (IQR)]	11.7 (10–15)
Fibrosis stage [n (%)]*	
F3 equivalent	189 (62.2)
F4 equivalent	115 (37.8)
Biochemistry [median (IQR)]	
ALT (U/L)	52 (34–103)
AST (U/L)	46 (36–87)
ALP (U/L)	92 (68–152)
GGT (U/L)	86 (38–171)
Total bilirubin (µmol/L)	10 (8–15)
Albumin (g/L)	36 (32–39)
Prothrombin ratio	1.0 (0.95–1.1)
Platelet count (x10^9/L)	199 (151–246)
Haemoglobin (g/L)	133 (116–152)
Sodium (mmol/L)	139 (137–141)
Creatinine (µmol l/L)	85 (73–99)
MELD Score	7 (6–9)

Abbreviations: IQR (interquartile range), n (number), BMI (body mass index), SWE (shear wave elastography), LSM (liver stiffness measurement), kPa (kilopascal). *Fibrosis stage is based on Metavir equivalent; advanced fibrosis is ≥F3. ALT (alanine aminotransferase), AST (aspartate aminotransferase), ALP (alkaline phosphatase), GGT (gamma glutamyl transferase), MELD (model of end-stage liver disease).

Table 2: Population characteristics of the endoscopy population (n=89).

Characteristic	Endoscopy population (n=89)	OV absent (n=71)	OV present (n=18)	p
Population characteristics				
Median age [years (IQR)]	61 (53–70)	57 (51–67)	67 (57–71)	0.04*
Sex [n (%)]				
Male	55 (61.8)	47 (66.2)	8 (44.4)	0.11
Female	34 (38.2)	24 (33.8)	10 (55.6)	0.11
Ethnicity [n (%)]				
European	39 (43.8)	30 (42.3)	9 (50.0)	0.43
Māori	13 (14.6)	10 (14.1)	3 (16.7)	1.00
Pacific	12 (13.5)	10 (14.1)	2 (11.1)	1.00
Asian	21 (23.6)	19 (26.8)	2 (11.1)	0.12
Other	4 (4.5)	2 (2.8)	2 (11.1)	1.00
Aetiology [n (%)]				
Hepatitis B virus	18 (20.2)	16 (22.5)	2 (11.1)	0.35
Hepatitis C virus	11 (12.4)	10 (14.1)	1 (5.6)	0.45
Alcohol	19 (21.3)	10 (14.1)	9 (50.0)	0.002**
Non-alcoholic fatty liver disease	27 (30.3)	24 (33.8)	3 (16.7)	0.25
Other	14 (15.7)	11 (15.5)	3 (16.7)	1.00
Median BMI [(IQR)]	28.7 (24.5–33.5)	28.2 (24.3–33.4)	30.3 (26.0–35.0)	0.37
Beta-blocker use [n (%)]	19 (21.3)	14 (19.7)	5 (27.8)	0.52
SWE				
Median SWE score [kPa (IQR)]	14.3 (11.1–18.4)	13.5 (11.0–16.6)	21.5 (14.8–33.7)	0.002**
Fibrosis stage [n (%)]				
F3 equivalent	37 (41.6)	34 (47.9)	3 (16.7)	0.80
F4 equivalent	52 (58.4)	37 (52.1)	15 (83.3)	0.80
Median interval between SWE and endoscopy [days (IQR)]	105 (29–200)	110 (39–191)	54 (7–220)	0.10
Biochemistry [median (IQR)]				
ALT (U/L)	34 (24–53)	34 (21–82)	63 (22–141)	0.85
AST (U/L)	41 (26–59)	43 (30–56)	59 (34–160)	0.17
ALP (U/L)	111 (79–167)	99 (74–165)	110 (78–265)	0.21
GGT (U/L)	108 (53–204)	82 (35–223)	151 (27–865)	0.32
Total bilirubin (micromol/L)	12 (8–21)	9 (7–25)	14 (10–36)	0.09
Albumin (g/L)	36 (31–39)	34 (32–40)	25 (21–32)	0.001**
Prothrombin ratio	1 (1–1.1)	1.0 (1–1.2)	1.1 (1.10–1.2)	0.11
Platelet count (x10^9/L)	185 (128–254)	206 (128–264)	146 (73–192)	0.01*
Haemoglobin (g/L)	130 (112–146)	119 (102–138)	122 (105–142)	0.08
Sodium (millimol/L)	139 (137–141)	140 (137–142)	137(136–139)	0.27
Creatinine (micromol/L)	84 (67–98)	89 (71–106)	80 (62 –93)	0.43
MELD Score	7.5 (6–10)	8.0 (6.0–9.5)	8.0 (6.8–11.5)	0.55

Abbreviations: OV (oesophageal varices), p (probability value), IQR (interquartile range), n (number), BMI (body mass index), SWE (shear wave elastography), LSM (liver stiffness measurement), kPa (kilopascal), F3 (fibrosis stage 3), F4 (fibrosis stage 4), ALT (alanine aminotransferase), AST (aspartate aminotransferase), ALP (alkaline phosphatase), GGT (gamma glutamyl transferase), MELD (model of end-stage liver disease). *p<0.05, **p< 0.0.

blocker use between the two subgroups (p=0.52). Only two of 18 subjects had variceal banding.

The area under the receiver operating characteristic curve (AUROC) for SWE-LSM to predict OV was 0.74 (p=0.02, Figure 1A). Using LSM alone, a cut-off of ≤10.7kPa had a sensitivity of 89% and ruled out OV with a negative predictive value of 97% (Figure 1A). Serum albumin, platelet count and age were also significantly different between subjects with and without OV (Table 2). Therefore, they were individually incorporated with LSM to improve the diagnostic performance of the model. Combining LSM with serum albumin improved the diagnostic accuracy (AUROC 0.80, p<0.001, Figure 1B). On logistic regression, a SWE score of ≤12.4kPa combined with an albumin level ≥37g/L had a sensitivity of 88% and a negative predictive value of 95% for OV. The addition of platelet count (a common biochemical surrogate for portal hypertension) and age did not improve diagnostic performance over that of LSM (Figure 1C).

SWE-LSM and morbidity and mortality

Three hundred and four subjects were followed-up over a period of up to three years (median 695 days). There were 137 all-cause admissions, 35 liver-disease related admissions and 48 infection-related admissions (Table 3). The SWE-LSM weakly correlated with the number of all-cause hospital admissions (Spearman's rho, r=0.2, p=0.007) but more strongly correlated with hospital admissions related to liver disease (r=0.5, p<0.001) and infection (r=0.6, p=0.008). SWE-LSM also correlated with serum biochemical markers of liver disease severity (prothrombin ratio, bilirubin, low albumin) as well as low platelet count and low haemoglobin (p<0.001).

The cumulative three-year survival for the 384 subjects was 0.8. There were 19 deaths. On multivariate cox regression, SWE-LSM (p=0.04) and age (p=0.03) were both independent predictors of mortality (Figure 2).

Discussion

Non-invasive predictors of significant portal hypertension are needed to rationalise which patients with advanced liver disease should be selected for endoscopic assessment. Current standard practice is that all patients with cirrhosis, and probably advanced fibrosis, should have a screening endoscopy. Elastography (both transient elastography (Fibroscan) and more recently SWE) has an established role in staging hepatic fibrosis.[15,18] Our findings add to the emerging data showing that SWE can also be used to confidently exclude the need for invasive endoscopy in these high-risk patients.

The AUROC for SWE as a diagnostic test to predict varices was 74%, which is considered "good performance" for a diagnostic test. For a rule-out test, the negative predictive value is important. An excellent negative predictive value could be achieved using a 10.7kPa cut-off. The diagnostic performance of the model was improved by the addition of serum albumin. If the serum albumin was ≥37g/L, the SWE cut off could be increased to ≤12.4 kPa while retaining a high sensitivity and an excellent negative predictive value of 95%. This would confidently exclude the need for endoscopy in a greater proportion of patients. Platelet count, which is often considered a biochemical surrogate for portal hypertension, did not improve the accuracy of the model.

On logistic regression analysis, SWE-LSM was an independent predictor of survival over a three-year period. This finding is also supported by a recent study by Trebicka et al, who showed that SWE-LSM combined with MELD scores predicted mortality.[21] The SWE-LSM had a weak but significant association between the number of all-cause hospital admissions and a stronger association for liver-related and infection-related admissions in particular. To our knowledge, this is the first study to demonstrate longitudinal correlation between SWE-derived LSM, morbidity and mortality in subjects with advanced liver disease.

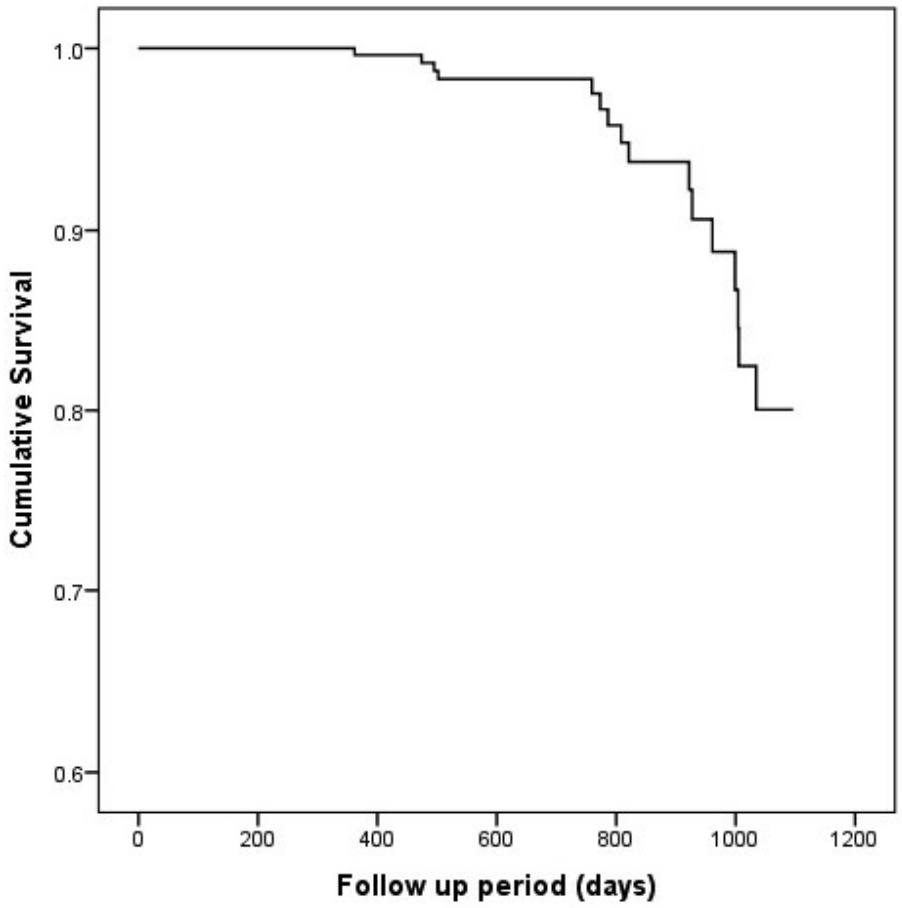
Although there are more data for the role of Fibroscan in predicting OV, the technical success rate of Fibroscan is limited by an increased BMI.[19–22] In comparison, the reproducibility of SWE is not affected by BMI. Our study population's high BMI reflects the rising obesity epidemic, and in that real-world context, SWE may be a more suitable tool. Therefore, although we had a relatively small sample size (89 with endoscopic

Table 3: Correlation of SWE-LSM with morbidity.

Hospital admissions	n	Spearman's rho	p
All-cause	137	0.2	0.007**
Liver disease-related	35	0.5	<0.001**
Infection-related	48	0.6	0.008**

Abbreviations: n (number), p (probability value). * p<0.05, ** p< 0.01.

Figure 2: Kaplan–Meier survival curve for the entire cohort and multivariate cox regression model showing the significant predictors of mortality.



Predictors	Exp (B)	p
SWE score (kPa)	1.02	0.04*
Age (years)	1.06	0.03*

Abbreviations: SWE (shear wave elastography), kPa (kilopascals).

data from 304 subjects) and it was a single centre study, these first New Zealand data for the use of 2D SWE as a diagnostic tool for predicting varices is still relevant. Studies in other populations are scarce. Most are retrospective with similar sample size. An Italian study suggested a cut-off of 13.2kPa (sensitivity 95%, but a poor AUROC 0.58), and an Asian study suggested a cut-off of 13.9kPa (AUROC 0.88).[22,23]

The 20% prevalence of OV limited our ability to assess SWE-derived LSM's clinical utility by restricting the number of parameters that could be incorporated into our model, which may explain why the addition of platelet count did not offer a significant benefit. Sample sizes for individual aetiologies and grades of OV were also not large enough to facilitate specific analyses or to be incorporated into our model. However, it was noted that ALD had a significantly higher proportion of OV compared to other aetiologies. Although data for active drinking were not specifically recorded, it was noted that those with OV had a trend towards a higher AST and GGT, raising the possibility that active hepatitis could have contributed to the risk. Nonetheless, clinical, endoscopic and biochemical data were collected using stringent criteria, and time-dependant confounders were accounted for, despite the study's limitations and retrospective nature.

In conclusion, SWE-LSM could be a useful test to identify patients with CLD and advanced fibrosis who do not need to undergo endoscopy to screen for varices. This could be particularly relevant to populations with a high prevalence of overweight subjects. In addition, SWE-LSM is predictive of morbidity and mortality. These findings warrant validation in larger prospective studies.

Competing interests:
Nil.
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