

CORRECTED VERSION | 13 September 2026

From FibroScan to 2D Shear Wave Elastography: Technological Evolution or Diagnostic Substitution in Liver Fibrosis Assessment?

Akib Arfee¹, Omair Ashraf Shah¹, Asif Iqbal², Afshana Bashir³, Aaqib Manzoor^{1*}

¹ Department of Radiodiagnosis and Imaging, Government Medical College Srinagar, India

² Department of Gastroenterology and Hepatology, Government Medical College Srinagar, India

³ Department of Pathology, Government Medical College Srinagar, India

Corresponding author: Aaqib Manzoor · akibmanzoor3721@gmail.com

Publication date: 29.12.2025. Corrected version: 13 September 2026.

Swiss Journal of Radiology and Nuclear Medicine · SJORANM GmbH · Rosenweg 3 · CH-6340 Baar · Switzerland

Abstract

Objective: To compare FibroScan and two-dimensional shear wave elastography (2D SWE) for liver fibrosis staging and describe the limits of the reported aggregate evidence.

Methods: This prospective single-center study enrolled 155 patients with chronic liver disease between October 2022 and November 2025. The report describes 136 patients with complete assessment. Separate operators performed FibroScan and 2D SWE. Multiple biopsy specimens were obtained per patient and examination and staged using METAVIR. Counts and stage-specific diagnostic measures were recalculated from the submitted contingency tables.

Results: Reported technical failure occurred in 18/155 FibroScan examinations (11.6%) and 8/155 2D SWE examinations (5.2%); 2D SWE succeeded in 11/18 patients with FibroScan failure. Within the respective tables, exact agreement with biopsy was 123/136 (90.4%) for FibroScan and 120/136 (88.2%) for 2D SWE. Intermethod agreement was 118/136 (86.8%), with linear weighted kappa 0.893. Different stages in multiple biopsy specimens account for the differing histological distributions. Specimen-to-measurement matching is undocumented, and elastography margins also differ between comparisons. These recalculations describe the reported cells; patient-level comparative estimates remain unvalidated.

Conclusion: The reported observations suggest potential complementary use of 2D SWE when FibroScan is unsuccessful. They do not establish diagnostic equivalence, superiority, or substitution. Specimen-level matching, reconciliation of elastography counts, and documented staging thresholds are needed for valid comparative conclusions.

Keywords: liver fibrosis; transient elastography; FibroScan; two-dimensional shear wave elastography; METAVIR; diagnostic accuracy

CORRECTED VERSION | 13 September 2026

Introduction

Fibrosis staging helps characterize chronic liver disease and informs prognosis and management. Fibrosis is dynamic, and studies of antiviral therapy have documented histological regression or longitudinal changes in liver stiffness [1, 2, 3]. Liver biopsy provides a histological reference but is invasive and subject to complications and sampling variability [4, 5, 6]. These limitations motivate the evaluation of non-invasive methods.

Transient elastography, including FibroScan, generates shear waves mechanically and estimates liver stiffness from their propagation. Two-dimensional shear wave elastography (2D SWE) uses acoustic radiation force within an ultrasound system and displays stiffness information alongside B-mode imaging [8]. Studies have evaluated SWE against biopsy, other fibrosis indices, and magnetic resonance elastography [7, 9, 10, 11]. These reference standards and patient populations are not interchangeable.

This study compared FibroScan and 2D SWE in patients undergoing liver fibrosis assessment. The corrected analysis accounts for multiple biopsy specimens per patient and distinguishes table-level calculations from comparisons requiring explicit linkage of specimens and elastography measurements.

Materials and methods

Study population

This prospective study was conducted at Government Medical College Srinagar, in collaboration with the departments of radiodiagnosis and imaging, gastroenterology and hepatology, and pathology, between October 2022 and November 2025. The study enrolled 155 patients with chronic liver disease scheduled for fibrosis staging. Exclusion criteria were heart failure, acute hepatitis, hepatic vascular thrombosis, focal liver lesions, prior interventions, or incomplete biopsy data. The source reports ethical approval and informed consent; an ethics committee identifier was not supplied.

The report describes 136 patients with complete assessment: 84 men (61.8%) and 52 women (38.2%), with a mean age of 52 ± 7 years. Disease etiologies, body mass index strata, and the full individual exclusion and examination-failure record were not available for this correction.

Histological evaluation

Ultrasound-guided percutaneous right-lobe biopsies used 18-gauge needles, with a reported core length of 2 cm. More than one biopsy specimen was obtained per patient at each examination. Specimens were fixed, paraffin-embedded, and stained with hematoxylin–eosin, Azan, and Gitter stains. Two experienced pathologists, blinded to imaging data, evaluated the samples and resolved disagreements by consensus.

Fibrosis was staged using METAVIR: F0, no fibrosis; F1, portal fibrosis without septa; F2, portal fibrosis with few septa; F3, numerous septa without cirrhosis; and F4, cirrhosis. Different specimens from the same patient could receive different METAVIR stages. This explains the differing biopsy-stage distributions in Tables 1 and 2. The tabulations do not document the number of specimens per patient or which specimen was used for each elastography comparison. FibroScan in Table 3 was a comparator, not a histological reference.

CORRECTED VERSION | 13 September 2026

Elastography techniques

FibroScan examinations used an Echosens FibroScan Expert system. Patients were supine with the right arm maximally abducted. An M or XL probe was selected according to body habitus. Ten valid measurements were obtained through the right midaxillary line while avoiding large vessels. The stated validity criterion was an interquartile range (IQR)/median ratio below 30%; median liver stiffness was recorded in kilopascals (kPa).

2D SWE used an Esaote MyLab X90 system with a convex 1–6 MHz probe and B-mode guidance. Measurements were obtained in the right liver lobe, at least 2 cm below the capsule and away from vascular structures. The source reports 9–12 valid measurements with IQR/median below 30%. The system converted stiffness measurements to kPa.

Separate experienced operators performed the two techniques and were blinded to the other elastography result and to biopsy findings. The source does not specify the interval between examinations or the stiffness thresholds used to assign METAVIR-equivalent stages.

Statistical analysis

The original analysis used SPSS version 24. For this correction, each contingency table was recalculated independently without altering its 25 interior counts. Row and column totals, percentages, exact agreement, and directional stage disagreements were obtained directly from those counts. Multiple specimens from the same patient were not assumed to be independent patients. All three reported tables total 136, but specimen-to-measurement linkage is not given and the elastography margins differ between tables. No common paired dataset was reconstructed.

For each stage in Tables 1 and 2, that exact stage was treated as positive and all other stages as negative. Sensitivity was $TP/(TP + FN)$, specificity $TN/(TN + FP)$, positive predictive value (PPV) $TP/(TP + FP)$, negative predictive value (NPV) $TN/(TN + FN)$, and accuracy $(TP + TN)/N$. These are one-versus-rest stage measures, not threshold measures for significant fibrosis ($\geq F2$) or advanced fibrosis ($\geq F3$).

Linear weighted kappa for Table 3 used agreement weights $1 - |i - j|/4$ for stages F0–F4. The point estimate is reproducible, but the method underlying the original confidence interval was not documented; that interval is not retained. Pearson chi-square assesses association, not agreement or equivalence. No equivalence margin, valid paired comparison, or inferential claim of superiority is included in the corrected analysis.

Results: reported examination failures

FibroScan failed in 18 of 155 examinations (11.6%), reportedly mainly because of obesity or ascites. 2D SWE failed in 8 of 155 examinations (5.2%), reportedly mainly because of steatosis or motion artefacts. Of the 18 patients with FibroScan failure, 11 (61.1%) were successfully assessed by 2D SWE. These are source-reported summaries; the overlapping failure categories were not independently verified.

CORRECTED VERSION | 13 September 2026

Results: staging and examination duration

Table 1 contains 123 exact FibroScan–biopsy stage matches (90.4%), four lower-stage assignments (2.9%), and nine higher-stage assignments (6.6%). **Table 2** contains 120 exact 2D SWE–biopsy matches (88.2%), five lower-stage assignments (3.7%), and 11 higher-stage assignments (8.1%). These totals correct the previous statement that 2D SWE understaged only three patients.

Table 3 contains 118 exact intermethod matches (86.8%). Its linear weighted kappa is 0.893. Its Pearson chi-square value is 371.22 (16 degrees of freedom), but 16 of 25 expected counts are below five. The statistic is retained only as a reproducible table calculation; it does not confirm diagnostic equivalence.

The recalculated stage-specific measures appear in **Table 4**. Across stages, sensitivity ranges from 84.0% to 97.4% for FibroScan and 80.0% to 95.0% for 2D SWE within their respective tables. Accuracy ranges from 94.9% to 97.8% and from 93.4% to 97.1%, respectively. Differences in sampled biopsy stages and the absence of a documented matching rule prevent these table-level values from establishing a valid paired ranking of the methods.

Reported examination duration was 5.3 ± 2.2 minutes for FibroScan and 5.4 ± 2.5 minutes for 2D SWE. A reproducible comparison test was not supplied, so no claim of a statistically non-significant difference is retained. Figures 1–3 illustrate the three source-reported cases; they do not establish subgroup performance.

Discussion

The submitted tables show frequent exact stage agreement within each comparison. More than one biopsy specimen was taken per patient and examination, and differences in the stages assigned to these specimens explain the differing histological distributions. Thus, the biopsy margins should not be interpreted as one identical stage assignment per patient in both comparisons. The specimen-selection rule and linkage to elastography measurements remain undocumented; the elastography margins also differ. Agreement percentages and weighted kappa alone cannot establish that 2D SWE can replace FibroScan.

2D SWE permits B-mode visualization and selection of a measurement region. This may be useful when an examination with another technique is unsuccessful, as suggested by the reported 11 successful SWE examinations after FibroScan failure. The present data do not justify stating that SWE is unaffected by obesity or ascites, or that it is superior in either subgroup. No stratified denominators or validated subgroup comparisons were supplied.

Earlier elastography literature includes etiology-specific guidance and ultrasound consensus recommendations [12, 13]. Comparative work by Osman et al. [14] provides relevant context, but populations and definitions of failure differ. Castéra et al. [15] reported technical failure in 3.1% of 13,369 examinations and unreliable measurements in 15.8%; these are distinct outcomes and should not be described as a 20% technical failure rate.

Evidence from alcohol-related disease, intermethod concordance studies, and hepatitis B cohorts further illustrates the need to preserve disease context and reference-standard definitions [16, 17, 18, 19]. Measurements also require attention to reproducibility and system differences [20]. The illustrative cases and short examination times in this report do not demonstrate cost-effectiveness or clinical interchangeability.

Conclusion

The reported aggregate observations support further evaluation of 2D SWE as a complementary technique for liver stiffness assessment, including after unsuccessful FibroScan examinations. The present tables do not establish equivalence, superiority, or diagnostic substitution. Documented specimen matching, reconciled elastography counts, and staging thresholds are necessary before stronger conclusions are warranted.

CORRECTED VERSION | 13 September 2026

Table 1. FibroScan versus biopsy

Biopsy stage	F0	F1	F2	F3	F4	Total
F0	23	1	0	0	0	24
F1	1	21	2	1	0	25
F2	1	0	21	1	1	24
F3	0	0	1	21	3	25
F4	0	0	1	0	37	38
Total	25	22	25	23	41	136

Columns: FibroScan. Interior counts are unchanged from the submitted table; margins were recalculated.

Table 2. 2D SWE versus biopsy

Biopsy stage	F0	F1	F2	F3	F4	Total
F0	20	3	0	0	0	23
F1	1	19	3	0	0	23
F2	0	2	20	3	0	25
F3	0	0	0	23	2	25
F4	0	0	0	2	38	40
Total	21	24	23	28	40	136

Columns: 2D SWE. Interior counts are unchanged from the submitted table; margins were recalculated.

Table 3. 2D SWE versus FibroScan

2D SWE stage	F0	F1	F2	F3	F4	Total
F0	20	2	1	0	0	23
F1	2	18	2	1	0	23
F2	1	1	20	1	1	24
F3	0	1	1	22	2	26
F4	0	0	1	1	38	40
Total	23	22	25	25	41	136

Columns: FibroScan. Interior counts are unchanged from the submitted table; margins were recalculated.

Limitations

Multiple biopsy specimens per patient explain the different histological distributions. However, specimen counts, the rule for selecting a reference specimen, and patient–specimen–measurement linkage are not documented. The differing elastography margins also require reconciliation. Additional limitations include the single-center design, incomplete participant-flow documentation, unavailable etiologic and body-habitus strata, absent device-specific staging thresholds, an unspecified interval between imaging and biopsy, and no interobserver reproducibility analysis. Biopsy sampling variability also remains relevant. Reanalysis of the underlying paired records is required to validate comparative diagnostic estimates.

CORRECTED VERSION | 13 September 2026

Table 4. Recalculated one-versus-rest stage measures

Method / stage	Sensitivity %	Specificity %	PPV %	NPV %	Accuracy %
FibroScan / F0	95.8	98.2	92.0	99.1	97.8
FibroScan / F1	84.0	99.1	95.5	96.5	96.3
FibroScan / F2	87.5	96.4	84.0	97.3	94.9
FibroScan / F3	84.0	98.2	91.3	96.5	95.6
FibroScan / F4	97.4	95.9	90.2	98.9	96.3
2D SWE / F0	87.0	99.1	95.2	97.4	97.1
2D SWE / F1	82.6	95.6	79.2	96.4	93.4
2D SWE / F2	80.0	97.3	87.0	95.6	94.1
2D SWE / F3	92.0	95.5	82.1	98.1	94.9
2D SWE / F4	95.0	97.9	95.0	97.9	97.1

Each exact stage is positive; all other stages are negative. PPV: positive predictive value; NPV: negative predictive value. Values describe Tables 1 and 2 independently. They are not validated paired comparisons and do not represent $\geq$ F2 or $\geq$ F3 threshold performance.

Distributions across the reported comparisons

Distribution (F0 / F1 / F2 / F3 / F4)	Table A	Table B
Biopsy: Table 1 vs Table 2	24 / 25 / 24 / 25 / 38	23 / 23 / 25 / 25 / 40
FibroScan: Table 1 vs Table 3	25 / 22 / 25 / 23 / 41	23 / 22 / 25 / 25 / 41
2D SWE: Table 2 vs Table 3	21 / 24 / 23 / 28 / 40	23 / 23 / 24 / 26 / 40

Multiple biopsy specimens per patient and examination explain the different histological distributions. The specimen-selection rule is not documented. The differing FibroScan and 2D SWE margins remain to be reconciled; multiple biopsy specimens alone do not explain those margins. Interior counts are unchanged.

Declarations

Ethics and consent: The authors report ethical approval, informed consent, and written consent for publication. No committee name or approval number was provided in the source material. Identifying patient report fields are excluded from the reproduced FibroScan report excerpts.

Author contributions: The source states that all authors contributed to study conception or design and manuscript drafting or revision, approved the final manuscript, and accepted accountability for the work.

Competing interests and funding: The authors declared no competing interests and stated that no funding was required for the study.

Data and correction scope: This correction uses the reported aggregate tables and supplied case figures. The correction incorporates the clarification that multiple biopsies were taken per patient and examination. Calculations are conditional on the submitted cells; specimen matching and the differing elastography counts could not be reconciled without the underlying records.

CORRECTED VERSION | 13 September 2026

Figure 1

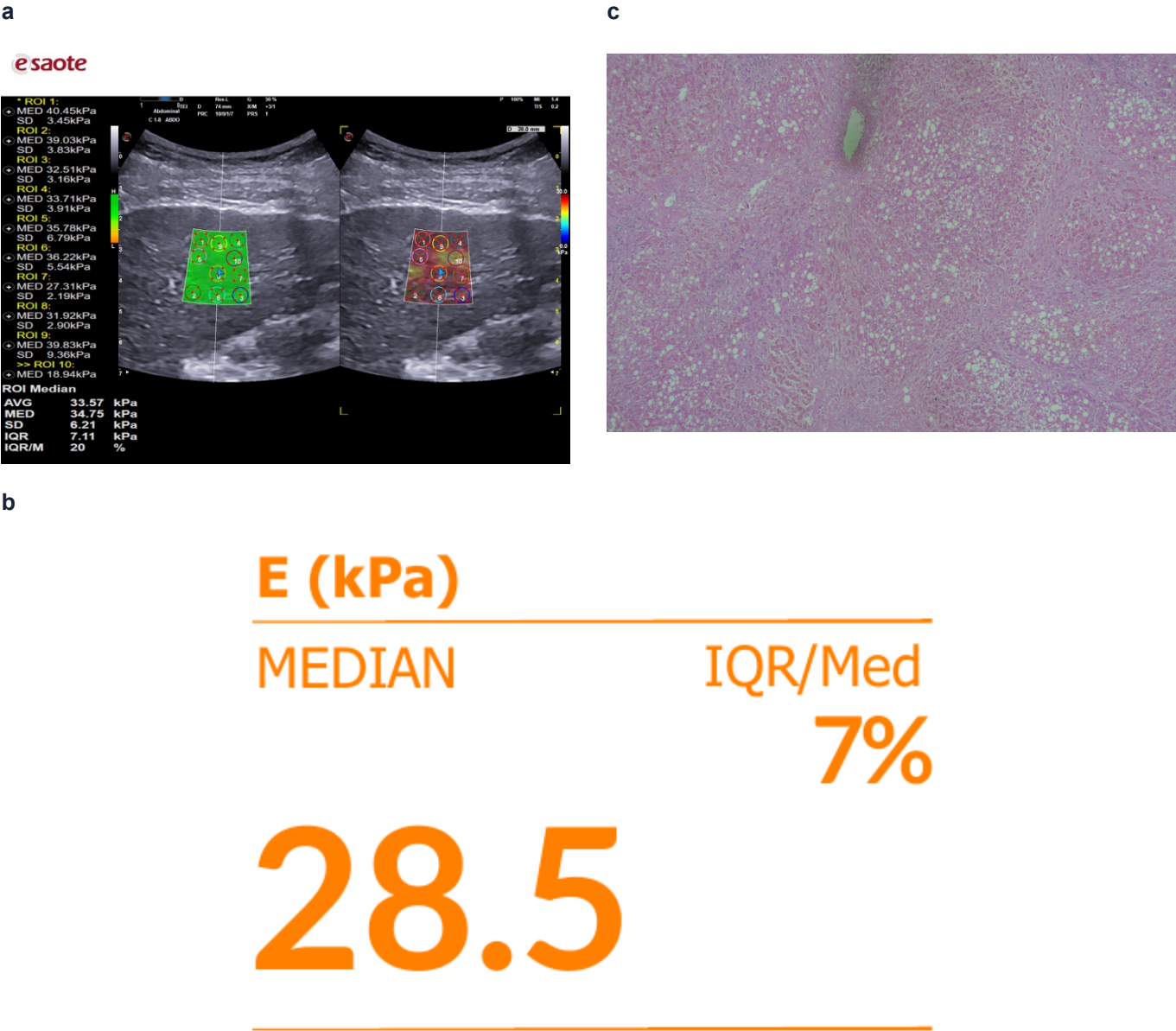


Figure 1. A 52-year-old man with chronic liver disease, normal subcutaneous tissue thickness, and no ascites. (a) The 2D SWE display gives a median stiffness of 34.75 kPa, correcting the 34.57 kPa transcription in the source narrative; the source assigned F4. (b) FibroScan report excerpt showing 28.5 kPa, assigned F4 in the source. (c) Histology (hematoxylin–eosin, low power) was described as cirrhosis, METAVIR F4, with fibrous septa, regenerative nodules, and macrovesicular steatosis.

Stage labels are retained as reported for these cases; device-specific cutoffs were not supplied and the displayed values should not be used as universal staging thresholds. FibroScan panels reproduce the measurement region only, excluding patient identifiers, QR codes, and administrative details.

CORRECTED VERSION | 13 September 2026

Figure 2

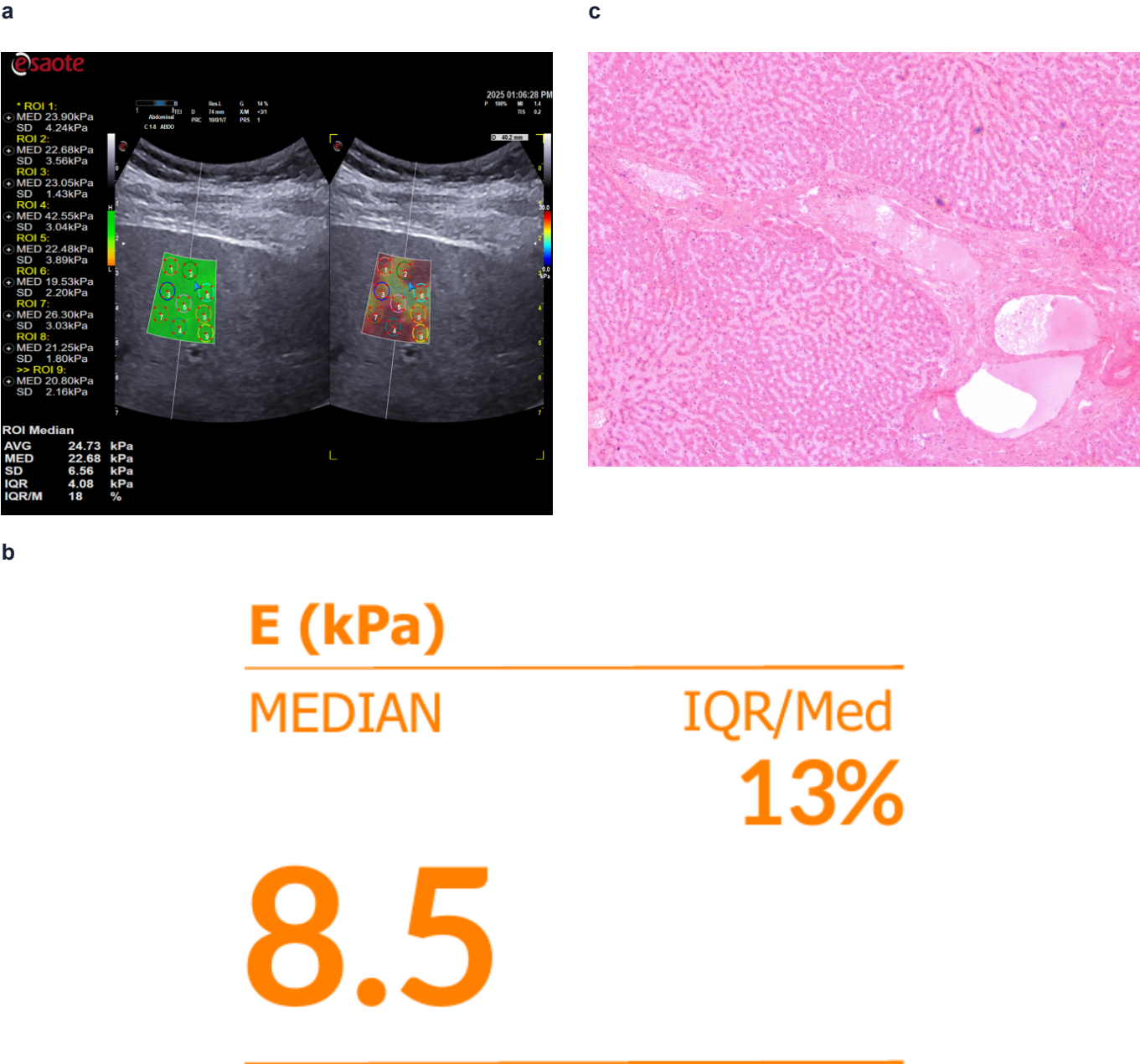


Figure 2. A 48-year-old woman with increased subcutaneous tissue thickness. (a) The 2D SWE display gives a median stiffness of 22.68 kPa, assigned F4 in the source. (b) FibroScan report excerpt showing 8.5 kPa, assigned F2 in the source. (c) Histology was described as bridging fibrosis, METAVIR F3. This discordant case is illustrative and does not establish comparative accuracy in obesity.

Stage labels are retained as reported for these cases; device-specific cutoffs were not supplied and the displayed values should not be used as universal staging thresholds. FibroScan panels reproduce the measurement region only, excluding patient identifiers, QR codes, and administrative details.

CORRECTED VERSION | 13 September 2026

Figure 3

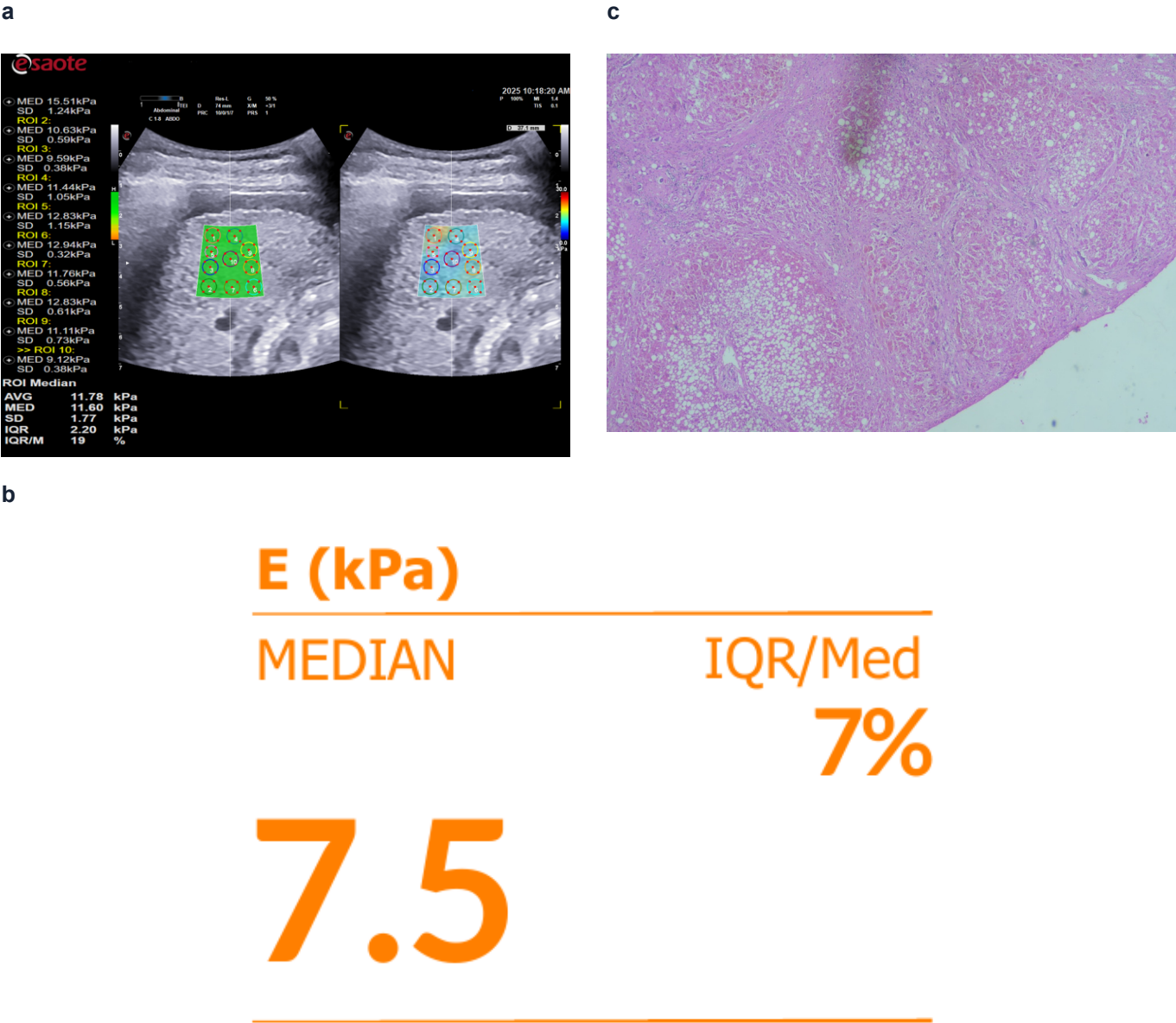


Figure 3. A 65-year-old man with chronic liver disease and ascites. (a) The 2D SWE display gives a median stiffness of 11.6 kPa, assigned F3 in the source. (b) FibroScan report excerpt showing 7.5 kPa, assigned F2 in the source. (c) Histology (hematoxylin–eosin, low power) was described as cirrhosis, METAVIR F4. Both elastography stage assignments were below the reported histological stage.

Stage labels are retained as reported for these cases; device-specific cutoffs were not supplied and the displayed values should not be used as universal staging thresholds. FibroScan panels reproduce the measurement region only, excluding patient identifiers, QR codes, and administrative details.

CORRECTED VERSION | 13 September 2026

References

1. Marcellin P, Gane E, Buti M, et al. Regression of cirrhosis during treatment with tenofovir disoproxil fumarate for chronic hepatitis B: a 5-year open-label follow-up study. *Lancet*. 2013;381(9865):468–475. [https://doi.org/10.1016/S0140-6736\(12\)61425-1](https://doi.org/10.1016/S0140-6736(12)61425-1)
2. Carrión JA, Navasa M, García-Retortillo M, et al. Efficacy of antiviral therapy on hepatitis C recurrence after liver transplantation: a randomized controlled study. *Gastroenterology*. 2007;132(5):1746–1756. <https://doi.org/10.1053/j.gastro.2007.03.041>
3. Martinez SM, Foucher J, Combis JM, et al. Longitudinal liver stiffness assessment in patients with chronic hepatitis C undergoing antiviral therapy. *PLoS One*. 2012;7(10):e47715. <https://doi.org/10.1371/journal.pone.0047715>
4. Seeff LB, Everson GT, Morgan TR, et al. Complication rate of percutaneous liver biopsies among persons with advanced chronic liver disease in the HALT-C trial. *Clin Gastroenterol Hepatol*. 2010;8(10):877–883. <https://doi.org/10.1016/j.cgh.2010.03.025>
5. Stotland BR, Lichtenstein GR. Liver biopsy complications and routine ultrasound. *Am J Gastroenterol*. 1996;91(7):1295–1296. <https://pubmed.ncbi.nlm.nih.gov/8677980/>
6. Regev A, Berho M, Jeffers LJ, et al. Sampling error and intraobserver variation in liver biopsy in patients with chronic HCV infection. *Am J Gastroenterol*. 2002;97(10):2614–2618. <https://doi.org/10.1111/j.1572-0241.2002.06038.x>
7. Zytoon AA, Elsakhawy MM, Algamal RM, Ali ZA. Real-time shear wave elastography for assessing liver fibrosis in patients with chronic hepatitis C. *Menoufia Med J*. 2018;31(2). <https://www.menoufia-med-j.com/journal/vol31/iss2/>
8. Shiina T, Nightingale KR, Palmeri ML, et al. WFUMB guidelines and recommendations for clinical use of ultrasound elastography: Part 1: Basic principles and terminology. *Ultrasound Med Biol*. 2015;41(5):1126–1147. <https://doi.org/10.1016/j.ultrasmedbio.2015.03.009>
9. Guibal A, Renosi G, Rode A, et al. Shear wave elastography: An accurate technique to stage liver fibrosis in chronic liver diseases. *Diagn Interv Imaging*. 2016;97(1):91–99. <https://doi.org/10.1016/j.diii.2015.11.001>
10. Tada T, Kumada T, Toyoda H, et al. Utility of real-time shear wave elastography for assessing liver fibrosis in patients with chronic hepatitis C infection without cirrhosis: comparison of liver fibrosis indices. *Hepatol Res*. 2015;45(10):E122–E129. <https://doi.org/10.1111/hepr.12476>
11. Song P, Mellema DC, Sheedy SP, et al. Performance of 2-dimensional ultrasound shear wave elastography in liver fibrosis detection using magnetic resonance elastography as the reference standard: a pilot study. *J Ultrasound Med*. 2016;35(2):401–412. <https://doi.org/10.7863/ultra.15.03036>
12. European Association for the Study of the Liver. EASL clinical practice guidelines: management of hepatitis C virus infection. *J Hepatol*. 2011;55(2):245–264. <https://doi.org/10.1016/j.jhep.2011.02.023>
13. Barr RG, Ferraioli G, Palmeri ML, et al. Elastography assessment of liver fibrosis: Society of Radiologists in Ultrasound consensus conference statement. *Radiology*. 2015;276(3):845–861. <https://doi.org/10.1148/radiol.2015150619>
14. Osman AM, El Shimy A, Abd El Aziz MM. 2D shear wave elastography (SWE) performance versus vibration-controlled transient elastography (VCTE/fibroscan) in the assessment of liver stiffness in chronic hepatitis. *Insights Imaging*. 2020;11(1):38. <https://doi.org/10.1186/s13244-020-0839-y>
15. Castéra L, Foucher J, Bernard PH, et al. Pitfalls of liver stiffness measurement: a 5-year prospective study of 13,369 examinations. *Hepatology*. 2010;51(3):828–835. <https://doi.org/10.1002/hep.23425>
16. Pavlov CS, Casazza G, Nikolova D, et al. Transient elastography for diagnosis of stages of hepatic fibrosis and cirrhosis in people with alcoholic liver disease. *Cochrane Database Syst Rev*. 2015;(1):CD010542. <https://doi.org/10.1002/14651858.CD010542.pub2>
17. O'Hara S, Hodson S, Hernaman C, Wambeek N, Olynyk J. Concordance of transient elastography and shear wave elastography for measurement of liver stiffness. *Sonography*. 2017;4(4):141–145. <https://doi.org/10.1002/sono.12122>
18. Foucher J, Chanteloup E, Vergniol J, et al. Diagnosis of cirrhosis by transient elastography (FibroScan): a prospective study. *Gut*. 2006;55(3):403–408. <https://doi.org/10.1136/gut.2005.069153>
19. Zeng J, Zheng J, Huang Z, et al. Comparison of 2-D shear wave elastography and transient elastography for assessing liver fibrosis in chronic hepatitis B. *Ultrasound Med Biol*. 2017;43(8):1563–1570. <https://doi.org/10.1016/j.ultrasmedbio.2017.03.014>
20. Ryu H, Ahn SJ, Yoon JH, Lee JM. Reproducibility of liver stiffness measurements made with two different 2-dimensional shear wave elastography systems using the comb-push technique. *Ultrasonography*. 2019;38(3):246–254. <https://doi.org/10.14366/usg.18046>