

The Clinical Value of Ultrasound Viscoelasticity Imaging for Non-Invasive Evaluation of Autoimmune Liver Diseases

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Abstract

Autoimmune liver diseases (AILD) primarily include autoimmune hepatitis, primary biliary cholangitis, primary sclerosing cholangitis, and overlap syndromes. The degree of liver fibrosis is closely associated with portal hypertension, decompensated liver function, and liver-related clinical outcomes; however, inflammatory activity, cholestasis, and biliary obstruction can all alter the mechanical properties of the liver, making it difficult to directly correlate a single liver stiffness measurement with histological fibrosis. Vibration-controlled transient elastography, point shear-wave elastography, and two-dimensional shear-wave elastography have accumulated substantial noninvasive evidence for fibrosis staging; meanwhile, shear-wave dispersion slope (SWDS) and the viscoelastic parameters derived from the model supplement mechanical information from the perspective of tissue energy dissipation. Available studies suggest that liver stiffness is sensitive to fibrosis, whereas dispersion or viscosity parameters may provide complementary information on inflammation and microstructural changes; nevertheless, direct evidence in autoimmune liver diseases is still derived mainly from small, single-center studies. Conventional elastography provides the fibrosis-focused clinical foundation, whereas SWDS and model-derived viscosity are the direct focus of the viscoelastic assessment discussed here. Building on an explanation of fibrosis staging and inflammation activity grading, this article discusses the principles of ultrasound viscoelastic imaging and its clinical applications in AILD, and explores the potential of combined multiparameter assessment.

Keywords

Autoimmune Liver Diseases, Ultrasound Elastography, Shear-Wave Dispersion, Viscosity, Liver Fibrosis

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1. Introduction

Autoimmune liver diseases (AILD) are a group of chronic liver and biliary diseases mediated by immune dysregulation. A global meta-analysis estimates that the annual incidence and prevalence of autoimmune hepatitis (AIH) are approximately 1.28 per 100,000 person-years and 15.65 per 100,000, respectively; the corresponding figures for primary biliary cholangitis (PBC) are approximately 1.76 per 100,000 person-years and 14.60 per 100,000, and for primary sclerosing cholangitis (PSC), approximately 0.87 per 100,000 person-years and 13.53 per 100,000 [1]-[3].

AIH is primarily characterized by elevated transaminases and immunoglobulin G levels, positive autoantibodies, and interface hepatitis; PBC primarily affects the intrahepatic small bile ducts, while PSC is characterized by multifocal inflammation, strictures, and fibrosis of both intra- and extrahepatic bile ducts [4]-[6]. Although the target tissues and clinical phenotypes differ among these diseases, persistent inflammation or bile-duct injury can drive fibrosis, thereby increasing the risk of portal hypertension, liver decompensation, and liver-related mortality.

Liver biopsy can simultaneously assess both the degree of inflammation and the extent of fibrosis; however, its invasive nature, sampling errors, and inter-observer variability limit its repeated use. Ultrasound elastography is easy to perform and allows for repeated measurements; it has become an integral part of the noninvasive assessment of chronic liver disease [7]-[9].

Liver stiffness is not determined solely by collagen content; active hepatitis, cholestasis, biliary obstruction, venous congestion, and food intake can all elevate measured values. If results are interpreted without considering the stage of treatment and the status of the biliary tract, a decrease in stiffness following resolution of inflammation may be misinterpreted as reversal of fibrosis, and elevated values associated with obstruction may be misinterpreted as structural progression. Shear-wave dispersion and viscosity parameters have garnered attention precisely because they aim to supplement the information on intratissue friction and microstructure that liver stiffness does not capture. Conventional VCTE, pSWE, and 2D-SWE provide the fibrosis-focused clinical foundation, whereas SWDS and model-derived viscosity are frequency-dependent viscoelastic measures. Because stiffness validation cannot be transferred directly to dispersion or viscosity, their evidence is evaluated separately [10].

2. Principles of Ultrasound Viscoelastic Imaging

2.1. Elasticity, Dispersion, and Viscosity of Tissue

Liver tissue is not an ideal elastic body, but rather a viscoelastic medium that exhibits both energy-storage and energy-dissipation characteristics. Shear wave speed (SWS) is primarily influenced by the shear modulus. Based on the assumptions that the tissue is homogeneous, isotropic, approximately incompressible, and purely elastic, some devices convert SWS to Young's modulus and express

liver stiffness in kPa. Thickening of the fibrous septa, collagen deposition, and remodeling of the hepatic lobule structure typically accelerate shear wave propagation; therefore, SWS or liver stiffness is relatively sensitive to fibrosis [11].

Shear-wave dispersion reflects frequency-dependent wave speed from energy dissipation. Evidence outside AILD links stiffness more closely to fibrosis, while dispersion or viscosity may also respond to necroinflammation and fluid changes [12]-[14]. In AIH, inflammation, edema, cholestasis, and obstruction may affect these outputs; dispersion is therefore not inflammation-specific.

2.2. Staging of Liver Fibrosis and Grading of Inflammatory Activity

Fibrosis staging describes scarring; activity grading describes necroinflammatory injury. METAVIR uses F0-F4 and A0-A3 [15], while Ishak uses a 0 - 6 fibrosis scale and the modified histological activity index [16]. They are not numerically interchangeable and do not capture all AILD lesions. **Tables 1-4** summarize both systems.

Disease-specific histological interpretation must also consider interface hepatitis, bile-duct injury, and cholestatic features [17]-[19].

Table 1. METAVIR liver fibrosis staging system.

Stage	Histopathological findings
F0	No fibrosis.
F1	Portal fibrosis without fibrous septa.
F2	Portal fibrosis accompanied by a few septa.
F3	Numerous fibrous septa, without cirrhosis.
F4	Cirrhosis.

Table 2. Ishak liver fibrosis staging system.

Stage	Histopathological findings
0	No fibrosis.
1	Fibrosis expands some portal tracts; short septa may be present.
2	Fibrosis expands most portal tracts; short septa may be present.
3	Most portal tracts are expanded by fibrosis, with occasional bridges between portal tracts.
4	Portal fibrosis with prominent bridging between portal tracts and between portal tracts and central veins.
5	Prominent bridging fibrosis with occasional nodules, indicating incomplete cirrhosis.
6	Probable or definite cirrhosis.

Table 3. METAVIR necroinflammatory activity grading system.

Activity grade	Histopathological interpretation
A0	No necroinflammatory activity.
A1	Mild necroinflammatory activity.
A2	Moderate necroinflammatory activity.
A3	Severe necroinflammatory activity.

Table 4. Ishak modified histological activity index for necroinflammatory grading.

Component	Score	Histopathological findings
A. Periportal or periseptal interface hepatitis	0	Absent.
	1	Mild (focal, few portal areas).
	2	Mild/moderate (focal, most portal areas).
	3	Moderate (continuous around <50% of tracts or septa).
	4	Severe (continuous around >50% of tracts or septa).
B. Confluent necrosis	0	Absent.
	1	Focal confluent necrosis.
	2	Zone 3 necrosis in some areas.
	3	Zone 3 necrosis in most areas.
	4	Zone 3 necrosis with occasional portal-central bridging.
	5	Zone 3 necrosis with multiple portal-central bridges.
C. Focal (spotty) lytic necrosis, apoptosis, and focal inflammation	6	Panacinar or multiacinar necrosis.
	0	Absent.
	1	One focus or less per 10× objective.
	2	Two to four foci per 10× objective.
	3	Five to ten foci per 10× objective.
D. Portal inflammation	4	More than ten foci per 10× objective.
	0	None.
	1	Mild, in some or all portal areas.
	2	Moderate, in some or all portal areas.
	3	Moderate/marked, in all portal areas.
Total modified HAI	4	Marked, in all portal areas.
	0 - 18	Sum of components A-D; fibrosis stage is reported separately.

Note: METAVIR and Ishak report fibrosis and inflammatory activity separately. METAVIR activity (A0-A3) is derived from interface hepatitis and lobular necrosis; Ishak mHAI sums four necroinflammatory components (0 - 18). The systems are not numerically interchangeable [15] [16].

Inflammation may improve rapidly after immunosuppression, whereas collagen remodels slowly; early stiffness decreases may reflect less inflammatory swelling, not fibrosis regression. Cholestasis or obstruction may sustain stiffness. Histology remains important for discordant findings or suspected overlap despite biopsy sampling variation [17] [18].

3. Clinical Applications of Ultrasound Viscoelastic Imaging

3.1. Vibration-Controlled Transient Elastography

Vibration-controlled transient elastography (VCTE) uses a surface probe to generate low-frequency mechanical vibrations and tracks shear-wave propagation using pulse-echo techniques. Two meta-analyses demonstrated good overall performance for staging significant fibrosis, advanced fibrosis, and cirrhosis in patients with AIH, PBC, or PSC, although disease composition, treatment status, histological systems, and diagnostic thresholds varied substantially among the included studies [20] [21]. VCTE assesses fibrosis severity within these diseases but does not establish the underlying autoimmune liver disease diagnosis.

In AIH, Mahmud *et al.* found that VCTE correlated with histological fibrosis stage, although probe selection and body habitus affected technical success [22]. Interpretation is also strongly influenced by treatment phase. Hartl *et al.* reported that liver stiffness was closely associated with inflammatory activity during the first 3 months of treatment, whereas its relationship with fibrosis became more reliable after at least 6 months of immunosuppression [23].

Accordingly, baseline elastography in AIH should not delay indicated immunosuppression or be interpreted as an unconfounded fibrosis stage. ALT, AST, and IgG should be recorded at each examination, and biochemical flares warrant cautious interpretation. Although liver stiffness may decrease after sustained biochemical remission, an early reduction more likely reflects improvement in inflammatory activity and does not by itself demonstrate collagen regression [24].

In PBC, Corpechot *et al.* reported VCTE cutoffs of 8.8, 10.7, and 16.9 kPa for F ≥ 2 , F ≥ 3 , and F4 fibrosis, respectively. An annual increase in liver stiffness exceeding 2.1 kPa was also associated with adverse outcomes, supporting the value of longitudinal rather than isolated measurements [25]. A reliable baseline examination near diagnosis and UDCA initiation may therefore support risk assessment after biliary obstruction has been excluded. Follow-up may coincide with biochemical response assessment at 6 or 12 months [5]. However, these intervals have not been validated specifically for SWDS or viscosity, and changes during UDCA treatment should not automatically be interpreted as antifibrotic effects.

In PSC, VCTE can distinguish severe fibrosis and cirrhosis and may provide prognostic information [26] [27]. However, strictures, cholangitis, cholestasis, and asymmetric biliary dilatation create substantial spatial heterogeneity. A technically valid right-lobe measurement may therefore not represent other affected segments. Serial examinations should use documented, comparable sites, and the results should be interpreted together with cholangiography, biochemical find-

ings, and the timing of biliary interventions [6].

When biliary obstruction or acute cholangitis is present, definitive fibrosis interpretation should be deferred until clinical and biochemical stabilization. Liver stiffness may decline rapidly after biliary drainage, but the available evidence does not establish a universal waiting interval before repeat assessment [28].

Across AILD subtypes, VCTE provides rapid and reproducible measurements but lacks real-time two-dimensional grayscale localization. Ascites, obesity, narrow intercostal spaces, and focal biliary abnormalities may increase the failure rate or introduce sampling bias. Results should therefore be interpreted in conjunction with liver biochemistry, immunoglobulin G, cholestatic markers, biliary imaging, and treatment status.

3.2. Point and Two-Dimensional Shear-Wave Elastography

Shear wave elastography (SWE) includes point shear wave elastography (point SWE, pSWE) and two-dimensional shear wave elastography (two-dimensional SWE, 2D-SWE). Both techniques allow for the selection of sampling regions under conventional two-dimensional ultrasound guidance; pSWE generates SWS within a single small region, while 2D-SWE produces a larger color elastography image, enabling observation of the distribution of measured values within the region and avoidance of blood vessels, bile ducts, and artifacts. Li *et al.* included 51 patients with AILD; the area under the curve (AUC) for 2D-SWE in identifying METAVIR $F \geq 2$ was 0.781, with a sensitivity of 83.3% and a specificity of 72.7% when using a cutoff of 9.15 kPa. Liver stiffness was found to be associated with both fibrosis and necroinflammation, suggesting that a single measurement is still subject to confounding by inflammation [29].

In 103 AIH patients, Xing *et al.* reported AUCs of 0.84, 0.84, and 0.94 for significant fibrosis, severe fibrosis, and cirrhosis; performance exceeded APRI and FIB-4 [30]. Sound touch elastography also discriminated fibrosis in AILD, although cutoffs were platform-dependent [31]. In 49 AIH and 41 PBC patients, pSWE showed an AIH $F \geq 2$ AUC of 0.70 and acceptable PBC discrimination [32].

In PSC, B-mode guidance can avoid visible ducts and vessels, but 2D-SWE still samples only part of the liver. Serial studies should use consistent segments, depths, and protocols; additional valid sites may be reported separately when heterogeneity is relevant. Persistent discordance with biochemistry or cholangiography requires specialist assessment [6] [9].

3.3. Acoustic Radiation Force Impulse-Based Elastography

ARFI generates tissue displacement and shear waves with focused ultrasound. In 85 autoimmune or cholestatic liver disease patients, velocity correlated with Ishak stage ($r = 0.510$); a 2.04 m/s cutoff for Ishak ≥ 5 yielded an AUC of 0.872, 90.0% sensitivity, and 74.7% specificity [33]. The mixed cohort and few advanced cases limit disease-specific thresholds. ARFI is an excitation mechanism, not a viscosity measure.

3.4. Shear-Wave Dispersion and Viscosity Assessment

Shear-wave dispersion imaging reports SWDS or model-derived viscosity. Evidence outside AILD suggests that dispersion may provide complementary tissue information, but these findings cannot be directly extrapolated to AILD [34]. Direct evidence in AILD remains limited to small and heterogeneous cohorts, with platform variation, incomplete follow-up, and limited histological validation (Table 5). Therefore, validated disease-specific thresholds for SWDS or viscosity have not yet been established.

Table 5. Direct evidence for dispersion and viscosity measurements in autoimmune liver diseases.

Study	Modality/ parameter	AILD subtype and sample	Reference/ outcome	Cutoff	AUC or association	Main limitation
Soh 2022 [35]	2D-SWE and SWDS Aixplorer/Aplio i800	AIH 69 total; 42 baseline SWDS	METAVIR and treatment-associated change	Stiffness: 8.2/12.2/14.3 kPa for $\geq F2/\geq F3/F4$	Stiffness AUC: 0.903/0.815/0.854; SWDS decreased after steroids	AUCs belong to stiffness, not SWDS. Two platforms and incomplete follow-up.
Hazzan 2025 [36]	SWE and viscosity-related output SuperSonic Mach30	AIH 25 total	Scheuer fibrosis and mHAI	No diagnostic cutoff reported in abstract	Viscosity: $r = 0.58$ with fibrosis; $r =$ 0.62 with mHAI	Small single-center cohort; correlations do not establish specificity.
Schulz 2022 [37]	2D-SWE and SWDS Aplio i800	PBC 22 total	Laboratory and noninvasive comparators; no biopsy reference	No histology-validated cutoff	No histological AUC; SWDS associated with ALP	Exploratory cross-sectional association; inflammation and fibrosis not disentangled.

SWDS is the shear-wave dispersion slope, usually expressed in (m/s)/kHz; mHAI is the modified histological activity index; ALP is alkaline phosphatase. Model-derived viscosity is expressed in Pa-s and is not interchangeable with SWDS.

An increased SWDS should not be interpreted as inflammation-specific because fibrosis, fluid redistribution, and tissue architecture may also affect shear-wave dispersion [10].

In 148 AIH-PBC overlap patients, including 82 followed for more than 1 year, stiffness correlated with Scheuer stage and declined with biochemical remission, illustrating combined hepatitic and cholestatic influences. Interpretation should record immunosuppression and UDCA timing. Reduced stiffness does not prove histological resolution or justify treatment withdrawal, and AIH-PBC evidence cannot establish AIH-PSC or universal overlap thresholds [38].

3.5. Multiparametric Clinical Framework and Research Priorities

Figure 1 summarizes a three-step framework that confirms acquisition quality, identifies reversible confounders, and integrates elastography with laboratory and biliary imaging findings. Within this framework, stiffness remains the principal fibrosis-related measure, whereas dispersion and viscosity are considered complementary. Stiffness is better established for fibrosis and prognosis; dispersion and viscosity remain complementary.

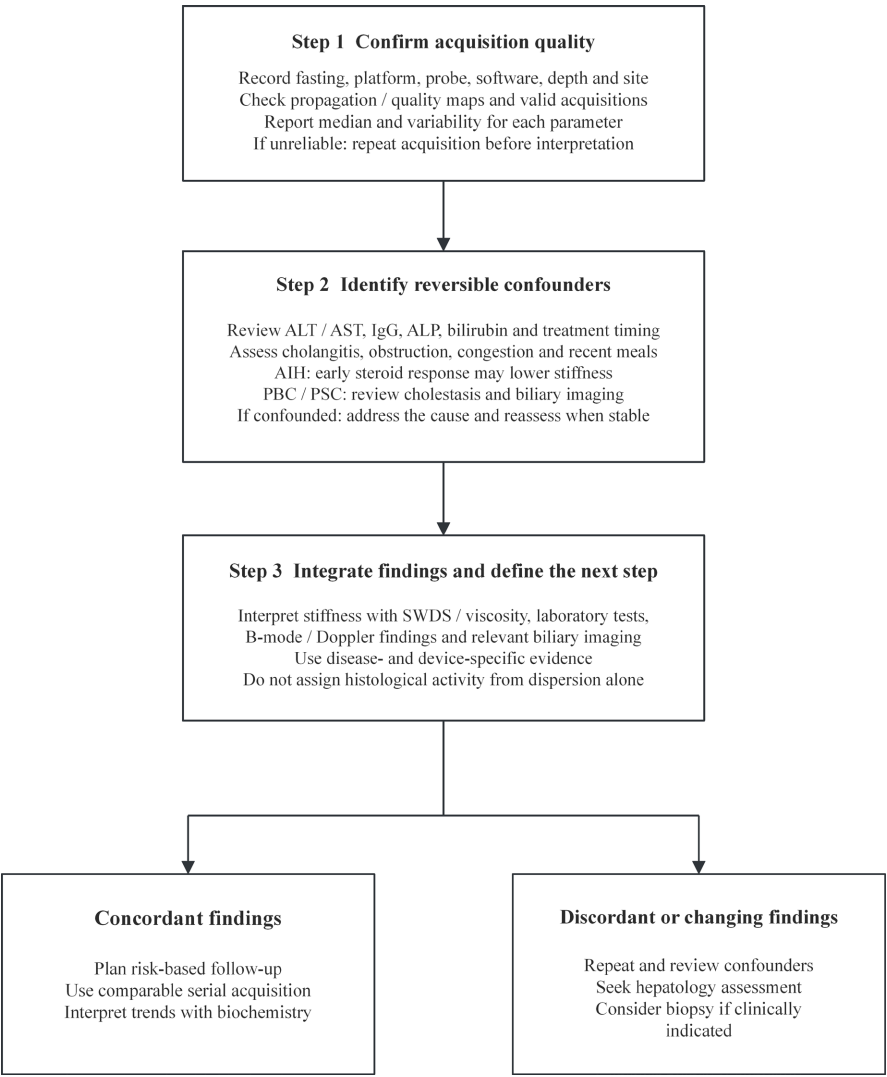


Figure 1. Proposed three-step framework for multiparametric ultrasound assessment in AILD.

Future multicenter prospective studies should prespecify AILD subgroups and standardize fasting, acquisition, quality control, treatment timing, and blinded histopathology when available. Serial work should separate short-term inflammatory or cholestatic effects from long-term fibrosis and outcomes, test incremental value beyond stiffness and routine laboratories, and validate results across ven-

dors [36] [37]. Thresholds should remain disease- and device-specific.

4. Summary and Perspectives

Ultrasound viscoelastic technology offers a repeatable, noninvasive approach to mechanical assessment of AILD. The evidence linking VCTE to fibrosis and outcomes is relatively well-established, while pSWE and 2D-SWE enhance sampling control through the use of two-dimensional imaging; ARFI should be understood as an intratissue excitation mechanism commonly used in the latter two techniques. SWDS and model viscosity complement liver stiffness by providing insights into energy dissipation; however, their inflammatory specificity and clinical added value have not yet been fully established and require prospective validation with standardized acquisition, disease-specific treatment timing, and independent clinical or histological endpoints.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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