Abdominal Radiology
https://doi.org/10.1007/s00261-026-05701-z

REVIEW

Two-dimensional shear wave elastography in Fontan-associated liver disease: a narrative review

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Received: 16 April 2026 / Accepted: 14 July 2026
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Abstract
Single ventricle congenital heart defects are rare conditions that require surgical palliation following birth. Often, such defects require multistep surgical procedures in different stages of life. The Fontan operation, considered the final procedure for single ventricle congenital heart disease repair, has been associated with long-term complications to the liver despite its cardiac benefit. Fontan-associated liver disease (FALD) is considered the main hepatological complication post-Fontan surgery. Liver biopsy has been the gold standard for diagnosis. Ultrasound-based shear wave elastography (SWE) has been investigated as a non-invasive adjunct for liver stiffness assessment alongside serological, imaging, and histopathological parameters. This narrative review summarizes the current evidence regarding the use of SWE as a method to assess liver stiffness in FALD, including current applications, technical challenges, and limitations associated with its clinical implementation. Particular emphasis is placed on the confounding influence of hepatic congestion, the lack of validated FALD-specific stiffness thresholds, and the need for standardized acquisition and interpretation protocols.

Keywords Fontan procedure · Liver diseases · Elasticity imaging techniques · Ultrasonography · Hepatic congestion

Introduction

Fontan-associated liver disease (FALD) is a long-term complication observed in patients who have undergone the Fontan procedure, also known as total cavo-pulmonary connection, a palliative surgical intervention for single-ventricle congenital heart defects [1, 2]. This procedure reroutes systemic venous blood directly into the pulmonary circulation, bypassing the heart, and results in chronically elevated central venous pressure (CVP). Over time, the new Fontan circulation is associated with long-term cardiac complications, such as arrhythmias, thrombosis, and heart failure, as well as non-cardiac complications, such as restrictive lung disease, protein-losing enteropathy, plastic bronchitis,

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Published online: 30 July 2026

thromboembolic events, and liver disease [3]. In light of these haemodynamic changes—which may result in hepatic congestion, fibrosis, cirrhosis, and an increased risk of hepatocellular carcinoma (HCC)—early and ongoing monitoring of liver function is essential [2, 4]. Liver biopsy remains the gold standard for fibrosis staging, but its invasiveness, sampling error (especially given the patchy fibrosis in FALD), and procedural risks (bleeding, pain, infection, even mortality) render it impractical for routine surveillance [4–6].

Non-invasive imaging modalities, such as transient elastography (TE) and magnetic resonance elastography (MRE), offer alternatives to biopsy. TE is operator-dependent and limited by small sampling volumes, while MRE is costly, time-consuming, and requires patients to be completely still, as well as not always accessible [7]. Two-dimensional shear wave elastography (2D-SWE) has therefore emerged as a practical non-invasive option for serial liver stiffness assessment, although interpretation in FALD remains challenging. It provides reproducible, real-time, quantitative stiffness maps (elastograms) using non-ionizing ultrasound and integrates seamlessly into standard ultrasound platforms [8, 9]. This review aims to summarize current evidence regarding 2D-SWE in FALD, with emphasis on clinical applications, technical challenges, interpretive limitations, and areas requiring further validation.

Methods

This narrative review was conducted using a targeted literature search of MEDLINE via PubMed/Ovid and Embase to identify relevant studies on 2D-SWE in FALD. The primary search focused on recent publications to capture current developments in the field. Search terms included combinations of "Fontan," "Fontan-associated liver disease," "FALD," "shear wave elastography," "two-dimensional shear wave elastography," "liver stiffness," "hepatic fibrosis," and "hepatic congestion."

Studies were selected based on their relevance to the application of 2D-SWE or related elastography methods for liver stiffness assessment in patients with Fontan circulation. Original research articles, comprehensive reviews, consensus statements, and guideline documents published in English were considered. Selected earlier publications were also included when they provided relevant background on Fontan physiology, FALD pathophysiology, elastography principles, or liver disease surveillance. Articles were excluded if they did not address Fontan circulation, FALD, liver stiffness assessment, or clinically relevant elastography methods.

Overview of 2D-SWE

2D-SWE is an ultrasound-based imaging technique that quantitatively assesses tissue stiffness by measuring the propagation speed of shear waves generated within the tissue. When an acoustic radiation force is applied, the resulting shear waves travel through the tissue, and their velocity correlates directly with tissue stiffness. By measuring the velocity of the induced shear wave, c_s (m/s), and if the density of the material can be estimated, ρ (kg/m^3), the Young's modulus, E (kPa), can be estimated as

$$E = 3\rho\, c_s^2 \tag{1}$$

In liver imaging, higher stiffness values (as expressed by Eq. 1) are indicative of fibrosis or cirrhosis, although liver congestion is a confounding factor [10]. Values may be reported in m/s or kPa; for consistency, all values in this article are reported in kPa. kPa stiffness values derived from SWE are not equivalent to and cannot be directly compared with kPa stiffness values derived from MRE.

Compared to earlier elastography methods, 2D-SWE provides real-time imaging with high spatial resolution and a larger field of view, see Fig. 1. Unlike TE, which provides a single-point measurement, 2D-SWE evaluates a broader region of interest, offering a more representative assessment of heterogeneous liver pathology.

In chronic liver disease, 2D-SWE has proven useful for staging fibrosis in conditions such as viral hepatitis, metabolic dysfunction-associated steatotic liver disease, and autoimmune hepatitis [11, 12]. Its potential utility in monitoring FALD stems from its ability to detect longitudinal changes in liver stiffness; however, whether these changes reflect fibrosis, congestion, or both remains a key limitation [13, 14].

FALD pathophysiology

The Fontan procedure reroutes systemic venous blood directly into the pulmonary circulation, bypassing the heart. It relies on elevated venous pressure to act as the driving force in lieu of the pumping of the heart. This creates chronically elevated CVP, reduced cardiac output, and lymphatic congestion. In the liver, these factors collectively lead to hepatic congestion, sinusoidal dilation, and fibrosis. Over time, persistent congestion and decreased hepatic perfusion contribute to hepatic parenchymal remodeling, fibrosis progression, and the development of cirrhosis. These structural changes also elevate the risk of HCC, even in the absence of viral hepatitis or metabolic liver disease [2, 3].

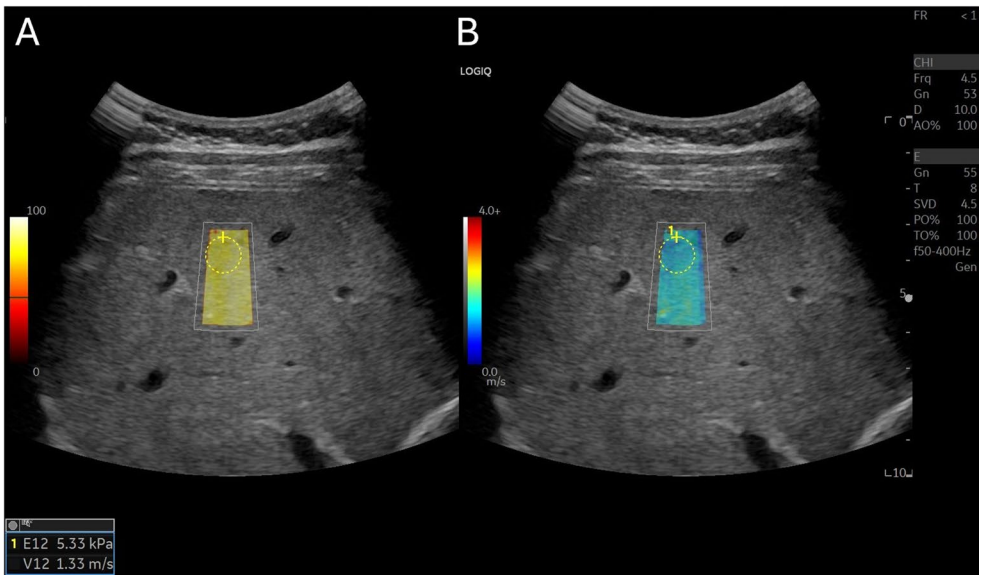

Fig. 1 Representative two-dimensional shear-wave elastography (2D-SWE) of the right hepatic lobe in a Fontan patient obtained on a GE LOGIQ E10 ultrasound system. **A** Quality map showing a homogeneous, high-confidence shear-wave signal within the rectangular region of interest (ROI). **B** Corresponding stiffness map over the same ROI; the colour bar indicates shear-wave speed (m/s), with measurements also displayed in kilopascals (kPa). Display format and colour scaling are vendor specific

Although the general course of FALD has been characterized, there is no consensus on its definition, with various descriptions encompassing different serological, histological, and clinical criteria. One widely accepted definition from combined pediatric cardiology and hepatology perspectives defines FALD as a broad spectrum of hepatic congestion, fibrosis, and cirrhosis resulting from the Fontan circulation. It further suggests that every patient with a Fontan circulation has some degree of FALD regardless of symptoms or the onset of abnormal liver tests, although it remains unclear when these patients will progress to clinically recognized liver disease [2, 5].

As a distinct form of congestive hepatopathy, FALD often develops with few clinical symptoms or is entirely asymptomatic. Its variable severity and the fact that hepatic fibrosis can progress somewhat independently of cardiac function increase the challenges in diagnosis and follow-up [14, 15]. The initial subclinical presentation may range from completely asymptomatic to subtle, nonspecific symptoms such as anorexia, fatigue, or weight loss. Moreover, the lack of correlation between these mild clinical features and laboratory or histological parameters makes it difficult to gauge FALD status and stage clinically [16].

Diagnosing FALD also presents challenges. Fibrosis progression is often insidious, with patients remaining asymptomatic until advanced stages. Serum biomarkers (e.g., liver enzymes and composite fibrosis scores) may not reliably reflect the degree of liver damage. Imaging techniques, including ultrasound, computed tomography, magnetic resonance imaging, and Doppler studies of hepatic blood flow can be used to assess structural changes and hemodynamics. However, these modalities often fail to capture subtle architectural changes [2, 5, 8]. Unlike other chronic liver diseases, where fibrosis progresses in a predictable manner, FALD exhibits a more heterogeneous pattern of fibrosis distribution, often sparing certain liver regions while severely affecting others [3]. Moreover, congestion itself can elevate liver stiffness measurements (LSM), complicating the interpretation of 2D-SWE values.

While some studies suggest that congestion-related stiffness may resolve with improved circulation, others indicate that persistent congestion contributes to progressive fibrosis [17]. Therefore, accurately interpreting LSM in Fontan patients requires consideration of both hemodynamic status and hepatic architecture.

Liver stiffness measurement in Fontan populations

There are multiple studies that have investigated the use of 2D-SWE in measuring LSM in Fontan populations, see Table 1. Kutty et al. published a seminal article comparing 41 Fontan patients (age 13.8 ± 6 years) to 65 age-matched controls, reporting a significant difference in LSM between the Fontan group and controls (15.6 ± 5.1 vs. 5.5 ± 0.9 kPa, respectively; $p < 0.0001$) [18]. This study found that increased LSMs correlated with greater degrees of histopathologic fibrosis. However, as mentioned previously, the increase in CVP and hepatic congestion can result in elevated LSMs, which complicates the staging of fibrosis. Schachter et al. attempted to compare 2D-SWE LSMs to liver biopsy using the congestive hepatic fibrosis (CHF) score. The CHF score is a histological grading system (0–4) for liver damage from chronic heart failure. SWE agreed with liver biopsy in 0% of cases: overestimating in 10 and underestimating in 4 cases [19]. Alsaied et al. also reported high LSMs in adult Fontan cohorts (18.8 ± 0.8 and 11.6 ± 1.0 kPa, respectively)

Table 1 Recent 2D-SWE studies of Fontan populations [18–26]

Study	Age at evaluation (years)	Time since Fontan (years)	Scanner model	Sample size	Liver stiffness measurement (kPa)	Reported format
Kutty et al. (2014)	13.8 ± 6.3	11 ± 6	SuperSonic Imagine Aixplorer	41	15.6 ± 5.1	Mean ± SD
Schachter et al. (2018)	26.4 ± 7.5	24.7 ± 6.2	GE Healthcare, LOGIQ E9	14	11.5 ± 3.3	Mean ± SD
Alsaied et al. (2020)	27.7 ± 8.2	19.3 ± 5.7	Canon Aplio 500 & i800	23	18.8 ± 0.8	Mean ± SD
Koizumi et al. (2021)	17 (12–25)	15.3 (9.7–21.7)	GE Healthcare, LOGIQ E9	43	11.6 ± 1.0	Mean ± SD
Mareș et al. (2022)	10 ± 3.8	4.2 ± 2.3	GE Healthcare, LOGIQ S8	13	4.9 ± 1.0	Mean ± SD
Gill et al. (2023)	22 (18–29)	18.6	GE Healthcare, LOGIQ E9	25	8.1 (7.0–10.3)	Median (IQR)
Bolia et al. (2024)	13.9 (6–21)	8.3 (2.1–18.7)	Not stated in published full text	48	15.4 (9.5–38.7)	Median (IQR)
Derinkuyu et al. (2024)	19.1 (14.2–23.9)	15.0 (10.2–20.6)	Aplio 500 (n = 56), Aplio i800 (n = 45), Mindray Resona 7 (n = 18)	119	14.8 (10.9–19.8)	Median (IQR)
Lo Yau et al. (2025)	17 (13–27)	14 (9–23)	Philips EPIQ 5	29	11.6 (8.3–13.4)	Median (IQR)

[20, 21]. In a purely pediatric study (ages 10 ± 3.8), Mareș et al. observed that Fontan patients had higher LSMs than healthy controls (4.9 kPa vs. 3.9 kPa, $p = 0.0049$), but not to the same extent as adult Fontan patients, suggesting that longer time post-Fontan may be associated with progressively higher LSMs [22].

The relationship between time since Fontan and LSM remains uncertain. Gill et al. did not find a significant correlation between time post-Fontan and LSM ($r = 0.19$, $p = 0.19$), while Bolia et al. found a weak positive correlation ($r = 0.31$, $p = 0.0002$), suggesting that further study is needed to clarify fibrosis progression over time [23, 24]. A recent study by Derinkuyu et al. found that baseline LSMs were significantly higher in patients who subsequently developed Fontan circulatory failure during follow-up ($p < 0.001$), highlighting the potential utility of 2D-SWE in post-Fontan surveillance and risk stratification [25]. Another recent study by Lo Yau et al. found a weak correlation between 2D-SWE values and fibrosis severity on biopsy (polychoric correlation = −0.021), with elastography again tending to overestimate fibrosis degree; the authors noted that because FALD is patchy, a biopsy may underestimate the overall fibrosis compared to a larger liver volume [26, 27]. A systematic review by Seol et al. concluded that liver biopsy results in FALD showed only a weak correlation ($r = 0.36$) between Fontan duration and fibrosis severity, whereas SWE and MRE demonstrated stronger correlations ($r = 0.53$–0.74) with biopsy-confirmed fibrosis severity in FALD [28].

Clinical interpretation

LSM values across studies show a broad range, from as low as ~4.9 kPa in younger Fontan patients to values exceeding 30 kPa in advanced cases. This variation highlights the importance of interpreting LSM in the context of patient-specific factors, including age, time since Fontan, and fibrosis severity. Figure 2 illustrates liver stiffness versus time since Fontan for the studies in Table 1 [18–26]. Table 2 provides a focused summary of studies that applied fibrosis cutoffs or reported threshold-based decision points relevant to the interpretation of liver stiffness in FALD.

As noted earlier, 2D-SWE LSMs in Fontan patients often overestimate the degree of fibrosis, likely due to hepatic congestion. Consistent with this, Terashi et al. found a significant correlation between ultrasound-based liver stiffness and CVP in Fontan patients ($r = 0.78$, $p < 0.001$), suggesting that 2D-SWE measurements should be interpreted primarily as an index of venous pressure rather than a direct measure of fibrosis severity [29].

Longitudinal data suggest a potential role for serial elastography. In a study of 39 Fontan patients, Talwar et al. obtained LSMs over one year at 3-month intervals following the Fontan procedure [30]. Significant changes were observed between nearly all time points (except between 3 and 6 months), with the largest increase occurring between the pre-Fontan baseline and the 12-month post-Fontan measurement (13.9 ± 6.2 vs. 22.8 ± 9.1 kPa, respectively; $p = 0.001$). The authors acknowledged that both congestion and fibrosis may contribute to these LSM increases, and they suggested that serial 2D-SWE monitoring could facilitate earlier therapeutic interventions to reduce right-sided pressures and to monitor fibrosis progression. As 2D-SWE is a non-invasive and cost-effective modality, it may be particularly well-suited for routine postoperative follow-up in this population.

Clinical implications

From a clinical standpoint, 2D-SWE may serve as an adjunctive tool for the long-term surveillance of Fontan patients. Incorporating routine LSMs into yearly follow-up visits could allow for the earlier detection of advanced hepatic changes [31]. As FALD appears to be highly prevalent in

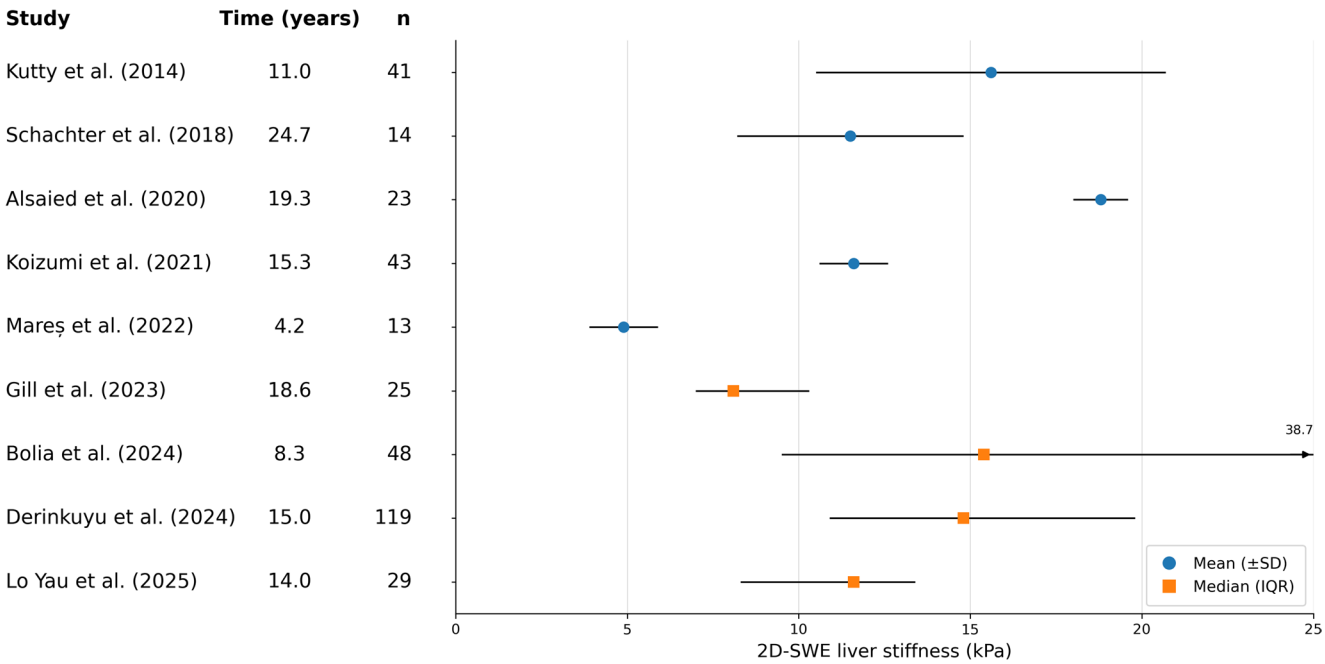

Fig. 2 Study-level summary of 2D-SWE liver stiffness in Fontan cohorts. Circles show mean LSM (whiskers, ±SD); squares show median LSM (whiskers, IQR). Time since Fontan and sample size are shown. Values are extracted as reported; no pooling was performed. For truncated whiskers, arrows indicate values extending beyond the x-axis limit [18–26]

Table 2 Reported elastography thresholds and practical interpretation in Fontan-associated liver disease [19, 24–26]

Study	Reported threshold or decision point	Interpretation for FALD
Schachter et al. (2018)	Conventional GE LOGIQ E9 fibrosis thresholds	Poor biopsy agreement; conventional chronic liver disease cutoffs should not be directly applied to FALD
Bolia et al. (2024)	LSM ≥ 20 kPa; annual increase ≥ 1 kPa/year	Exploratory monitoring decision points; not diagnostic of advanced fibrosis
Derinkuyu et al. (2024)	> 13.4 kPa for follow-up VAST score ≥ 1; >13.5 kPa for Fontan circulatory failure	Outcome-derived risk stratification thresholds; require external validation
Lo Yau et al. (2025)	METAVIR-based categories; >14.5 kPa also assessed	Poor biopsy correlation; congestion and heterogeneous fibrosis remain major confounders

FALD, Fontan-associated liver disease; LSM, liver stiffness measurement; VAST, varices, ascites, splenomegaly, and thrombocytopenia

The thresholds listed are study-specific thresholds, exploratory decision points, or conventional fibrosis cutoffs applied within individual studies. They should not be interpreted as validated FALD-specific stiffness cutoffs

long-term Fontan survivors, regular non-invasive monitoring is appealing; however, LSM values should be interpreted alongside clinical status, laboratory markers, hemodynamics, and other imaging findings [5]. A rising LSM trend in an otherwise asymptomatic patient might prompt more detailed evaluation, while a stable, low LSM could provide reassurance.

2D-SWE may also aid in risk stratification and management decisions. For instance, patients with markedly elevated LSMs could be evaluated more intensively for Fontan circulatory failure or referred for consideration of heart transplant before severe complications arise [25]. The ability to track liver stiffness non-invasively means that interventions can be objectively followed for effect. Elastography findings should always be interpreted in the context of the overall clinical picture, but they can add impetus to multidisciplinary discussions. In cases where liver disease is advanced, identifying high-risk patients is critical for planning [5]. Additionally, recognizing when a Fontan patient has likely developed cirrhosis would prompt initiation of HCC surveillance according to standard guidelines for cirrhosis (e.g., ultrasound and alpha-fetoprotein every 6 months) [5].

Future work/directions

Ongoing research is aimed at overcoming limitations and refining the use of elastography in FALD. One priority is refining stiffness interpretation. Multi-center collaborations are necessary to assemble larger Fontan cohorts, derive externally validated FALD-specific stiffness thresholds, and determine how these should be used in clinical

decision-making. One such effort is the recently published FONLiver study, in which Téllez et al. pooled data from multiple centers to develop a risk score that combines LSM with platelet count to predict severe fibrosis in adult Fontan patients [8, 32]. This FONLiver risk score demonstrated superior accuracy (AUROC ~0.81) for severe fibrosis compared to using stiffness or laboratory indices alone. Such multiparametric indices represent a promising direction. By combining SWE with serum biomarkers, clinicians may better identify which patients truly have advanced disease.

Another area of active research is the development of advanced elastography techniques that may improve differentiation between fibrosis and congestion. Shear wave dispersion (SWD) measures the frequency-dependent variation in shear wave speed, providing an estimate of tissue viscosity in addition to elasticity. In theory, dispersion may reflect inflammatory activity or fluid content in the liver, rather than static collagen stiffness [33]. Early research in other congestive hepatopathies indicates that combining SWE with dispersion analysis provides a more nuanced assessment. For example, viscosity metrics appear to increase with edema and sinusoidal obstruction [34]. Applying SWD in Fontan patients could therefore help separate predominantly congestive changes from established fibrosis and improve diagnostic precision.

Similarly, strain elastography is being revisited. This older technique measures tissue deformation under mechanical stress [35]. Recent studies suggest that the strain-derived liver fibrosis index (LFI) might better reflect fibrosis in Fontan livers, where shear wave speed is elevated by congestion [36]. In a 2025 study, Imoto et al. found that although 2D-SWE shear wave velocity did not differ between Fontan patients with and without portal hypertension, the LFI from strain elastography was significantly higher in those with advanced FALD [36]. Based on these findings, the authors proposed a two-step strategy: initial screening with platelet count (platelets $< 185 \times 10^3/\mu L$ indicating high risk), followed by strain elastography with an LFI threshold of ~2.2 in patients with normal platelet counts to detect advanced disease. This combined strategy achieved 100% sensitivity for advanced FALD in their cohort. Future studies may refine such multimodal algorithms. For example, integrating SWE, strain imaging, and biochemical markers may improve early identification of high-risk Fontan patients.

Prospective longitudinal studies are still needed to clarify the relationship between LSM changes and clinical outcomes. It remains uncertain whether reducing a patient's LSM through medical or surgical interventions ultimately translates into a better prognosis, such as lower rates of cirrhosis or HCC. The field would also benefit from interventional trials in which elastography explicitly guides management. For instance, future studies should determine whether serial SWE can contribute to decisions regarding Fontan revision or transplantation assessment.

Additionally, standardization efforts are underway. Consensus groups (such as the European Association for the Study of the Liver (EASL), World Federation for Ultrasound in Medicine and Biology (WFUMB), American Heart Association (AHA) and Society of Radiologists in Ultrasound (SRU)) continue to update guidelines for elastography performance and reporting [8, 12, 37, 38]. Ensuring that all centers use consistent acquisition protocols and report results uniformly will facilitate easier data comparison and the definition of universal cutoffs. Finally, complementary imaging modalities beyond ultrasound may also aid diagnosis. MRE, although expensive, provides whole-liver stiffness maps and may be refined to distinguish fibrosis once confounding factors are accounted for. Likewise, contrast-enhanced ultrasound for focal lesion characterization and 4D flow MRI for hemodynamic assessment are being explored in the comprehensive evaluation of Fontan patients [13]. Together, these multidisciplinary approaches may allow 2D-SWE and its related innovations to be fully leveraged to improve long-term liver health and overall outcomes in Fontan patients.

Conclusion

As the Fontan population continues to grow, effective surveillance strategies for FALD are increasingly important. 2D-SWE is a promising non-invasive method for assessing liver stiffness in these patients; however, interpretation remains challenging because liver stiffness may reflect both fibrosis and venous congestion. In addition, validated FALD-specific stiffness thresholds are lacking, limiting the use of 2D-SWE as a stand-alone marker of fibrosis severity.

At present, 2D-SWE is best considered as part of longitudinal, multimodal surveillance alongside clinical assessment, laboratory markers, Fontan hemodynamics, and complementary imaging. Further prospective multicenter studies are needed to standardize acquisition, validate clinically meaningful thresholds, and clarify the prognostic role of 2D-SWE in FALD.

Acknowledgements This study was supported by the School of Medicine, Royal College of Surgeons in Ireland – Medical University of Bahrain.

Author contributions Author Contributions: Mohamed.A: Conceptualization; Formal analysis; Writing—original draft. J.Bl: Conceptualization (supporting); Writing—original draft (supporting); Writing—review & editing (equal). H.A: Image acquisition; Writing—review & editing. Maryam.A, Z.H, H. M. I, N.K, K.D: Writing—review & editing.

Data availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interest The authors declare no competing interests.

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