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Retrospective Study

Lung Ultrasound Surface Wave Elastography in Cardiogenic Pulmonary Edema: A Retrospective Pilot Study

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Abstract

Objective: This retrospective pilot study aimed to evaluate the feasibility of Lung Ultrasound Surface Wave Elastography (LUSWE) as a quantitative Point-of-Care tool for assessing cardiogenic pulmonary edema in patients with acute congestive heart failure.

Methods: A small cohort of 9 patients admitted for acute cardiogenic pulmonary edema underwent B-mode lung ultrasound and LUSWE assessment. Examinations were performed at admission and 2 days after initiating diuretic therapy. We retrospectively analyzed the total lung B-lines and lung surface wave speeds (at 150 and 200 Hz), correlating them with the patients' net fluid balance.

Results: Effective diuresis was achieved in the cohort (average net fluid balance of -2.3L). This was accompanied by a significant reduction in pulmonary edema on B-mode ultrasound (average decrease of 11 B-lines). Concurrently, LUSWE measurements demonstrated a statistically significant reduction in wave speed from admission to follow-up ($p < 0.001$).

Conclusion: This pilot study suggests that LUSWE can detect quantitative changes in superficial lung stiffness corresponding to diuresis-mediated reduction of edema. The technique shows promise as a noninvasive, quantitative adjunct for Point-of-Care assessment, warranting further investigation in larger, prospective studies.

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Abbreviation: Two-dimensional lung ultrasonography=2D-LUS, extravascular lung water=EVLW, acute congestive heart failure=ACHF, lung ultrasound surface wave elastography=LUSWE, Shear Wave Elastography=SWE, Acoustic Radiation Force=ARF.

Introduction

Two-dimensional lung ultrasonography (2D-LUS) has emerged as a clinically valuable diagnostic tool, offering advantages in feasibility, portability, and detection of cardiogenic pulmonary edema in patients presenting with undifferentiated dyspnea[1]. This modality has demonstrated lower accuracy than lung ultrasound in quantifying extravascular lung water (EVLW)[2]. Ultrasound reverberation artifacts, manifesting as vertical hyperechoic lines (termed B-lines), arise from interstitial thickening caused by engorged lymphatics or alveolar flooding due to overt cardiogenic pulmonary edema[3]. Quantitative assessment of B-line number and density via 2D-LUS exhibits a significant correlation with EVLW volume. This relationship has been investigated in studies involving both outpatients and hospitalized individuals with heart failure [4]. However, B-line analysis using 2D-LUS remains semi-quantitative, as it requires visual interpretation and is consequently operator-dependent[5]. Furthermore, methodologies for quantifying B-lines during 2D-LUS assessment of EVLW differ across studies. These include: (i) analyzing B-mode still frames displaying maximal B-line density; (ii) counting cumulative B-lines throughout a respiratory cycle; and (iii) calculating the lung zone percentage occupied by B-lines[6]. The utility of 2D-LUS in serial or longitudinal monitoring—for instance, in tracking pulmonary edema dynamics in hospitalized

heart failure patients—is constrained by its qualitative to semi-quantitative characteristics. Establishing a strictly quantitative bedside method for EVLW would empower clinicians to precisely gauge response to therapy and adjust treatment strategies based on objective data.

We have established a lung ultrasound surface wave elastography (LUSWE) method for assessing the stiffness of superficial lung tissue[7]. This noninvasive technique employs low-frequency harmonic vibrations produced by a handheld shaker applied to the subject's chest wall. An ultrasound transducer is positioned approximately 5 mm from the shaker within the same intercostal space to measure surface wave propagation across the lung surface in that region. The surface wave speed is derived from the phase shift of the wave over distance. Previous clinical validation has demonstrated that this technique distinguishes patients with interstitial lung disease from healthy controls based on statistically significant differences in lung surface wave speed. This study aims to clinically implement the LUSWE technique for quantitatively evaluating lung stiffness in pulmonary edema patients. We postulate that variations in pulmonary edema severity (clinically assessed as EVLW), measured by the established ultrasonographic biomarker B-lines (via 2D-LUS), will demonstrate a quantifiable association with LUSWE-derived measurements of superficial lung elasticity.

Methods

Study Population and Baseline Assessments

A retrospective analysis included 9 adults hospitalized for acute congestive heart failure (ACHF) with radiographic pulmonary edema. ACHF diagnosis was based on composite clinical and imaging criteria. Exclusion criteria comprised a history of interstitial lung disease or pulmonary fibrosis, requirement for mechanical or non-invasive ventilation, or suboptimal ultrasound windows due to severe obesity.

Patient baseline characteristics are summarized in **Table 1**. The cohort (mean age 73.7±6.5 years; 77.8% male) had a mean left ventricular ejection fraction of 48±6%. To assess pulmonary status, two-dimensional lung ultrasonography (2D-LUS) and lung ultrasound surface wave elastography (LUSWE) were performed at admission (establishing baseline) and repeated within the first 1-2 hospital days. Net fluid balance, reflecting diuretic response, was obtained from electronic medical records.

Table 1. *Baseline characteristics of patients hospitalized with cardiogenic pulmonary edema.*

Baseline characteristics of patients	
Age, years	73.7±6.5
Male sex (n)	7
Hypertension (n)	7
Diabetes Mellitus (n)	4
Coronary Artery Disease (n)	2
Chronic Kidney Disease (n)	2
B-type Natriuretic Peptide (BNP), pg/mL	477±192
Left Ventricular Ejection Fraction, %	48±6

B-Line Quantification Protocol Using 2D-LUS

B-line assessment employed a validated semi-quantitative approach scanning 28 intercostal spaces (28-zone protocol) per established methodology[8]. Bedside examinations were performed using the Mindray M9 ultrasound system (Mindray, Shenzhen, China) equipped with a high-frequency linear transducer. Standardization across this pilot study mandated linear transducers for both B-line quantification and LUSWE measurements.

Experienced sonographers conducted all scans. Patients were positioned supine with 45-60° head elevation during bedside image acquisition. Each intercostal space was systematically interrogated with optimization of near-field pleural line visualization. After pleural line identification, 4-6 second cine loops were captured to document lung sliding dynamics. Consistent with published criteria, B-lines were defined as discrete comet-tail artifacts originating from the pleura and extending to the screen's inferior margin without attenuation.

An independent reviewer blinded to LUSWE results performed offline analysis. For each zone, the frame demonstrating maximal B-line density within the cine loop was identified per prior studies. The cumulative B-line score (representing extravascular lung water) was derived by summing counts from the maximally affected frame across all 28 zones. Identical bedside reassessment was performed within 1-2 days using the standardized 28-zone protocol, enabling quantification of interval B-line changes.

LUSWE Methodology

Lung Ultrasound Surface Wave Elastography (LUSWE) was developed as a noninvasive modality for quantifying pulmonary surface wave propagation velocity. The

technique employs a function generator to produce a 0.1-second harmonic vibration signal, which is amplified through an audio amplifier and transmitted to a handheld mechanical actuator. This actuator delivers precisely controlled vibrations via a 3-mm diameter indenter positioned within an intercostal space, generating tissue displacements typically below 10 μm with applied forces <1N. The resulting sub-micron mechanical stimulus is perceptible as minimal cutaneous vibration without causing patient discomfort during bedside implementation.

The ultrasound transducer was positioned with a 5-mm offset from the mechanical actuator within the same intercostal space to quantify surface wave propagation. During testing, the actuator's indenter maintained an approximate 30° angle relative to the transducer. Crucially, neither angular orientation nor excitation location influences wave speed measurements, as these parameters depend solely on local tissue elasticity (**Figure1**).

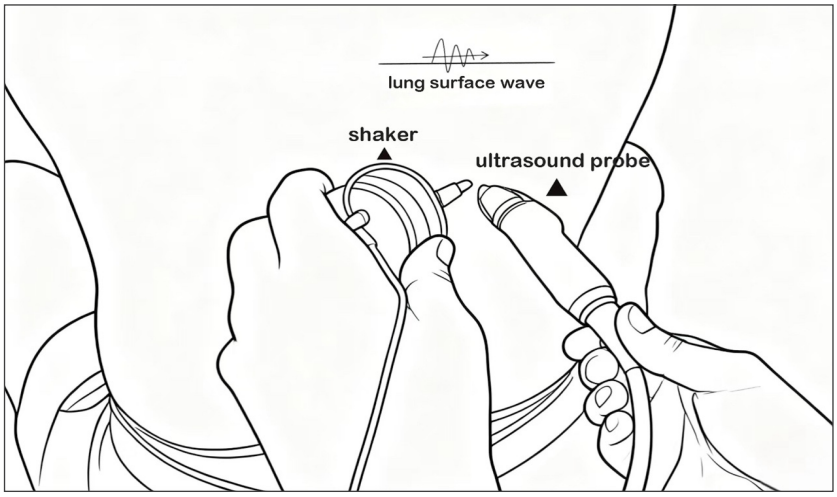


Figure 1. Ultrasound probe (5-mm offset from mechanical shaker in same intercostal space) and shaker indenter (≈30° angle to probe) for surface wave propagation quantification, with wave speed dependent solely on local tissue elasticity (unaffected by angular orientation/excitation location).

Multi-frequency vibrations (150, and 200 Hz) were generated using 0.1s duration signals (15cycles at 150 Hz; 20 cycles at 200 Hz). Local wave speeds at each frequency were calculated via phase gradient analysis. Triplicate measurements per frequency/location ensured data robustness.

LUSWE assessments were conducted at six bilateral thoracic locations:

Right/left 2nd intercostal space (mid-clavicular line)

Right/left 3rd intercostal space (anterior axillary line)

Right/left 4th intercostal space (mid-axillary line)

designated as R1/L1, R2/L2, and R3/L3 respectively.

2D-LUS guidance ensured precise anatomical localization of intercostal spaces and pleural lines[9]. Synchronized with 2D-LUS evaluations, the complete LUSWE protocol required approximately 30 minutes per patient.

At each location, surface wave speeds were quantified across two frequencies (150, 200 Hz) with triplicate measurements per frequency, yielding 54 datasets per subject. Crucially, wave speed calculations derived from direct pulmonary ultrasound data analysis exhibited independence from excitation parameters (location/amplitude).

Wave Propagation Analysis

Surface wave propagation was assessed within subpleural lung parenchyma (≤3 mm depth below the pleural line), enabling direct quantification of pulmonary surface wave velocity. Prior evidence suggests minimal interference from pleural fluid layers on these measurements[10]. Using our noninvasive ultrasound methodology, harmonic wave dynamics at designated locations were captured through phase-shift analysis of propagating waves, permitting calculation of surface wave speed.

Results

LUSWE measurements revealed significant differences in surface wave propagation velocities between baseline and post-diuresis follow-up across all thoracic locations and frequencies ($P < 0.001$). At baseline, the mean wave speeds (mean ± SD) in the right hemithorax at 150 Hz were 5.4 ± 0.5 m/s (R1), 5.7 ± 0.4 m/s (R2), and 5.2 ± 0.6 m/s (R3), while at 200 Hz, they were 6.3 ± 0.6 m/s (R1), 6.6 ± 0.5 m/s (R2), and 6.8 ± 0.7 m/s (R3). Corresponding values in the left hemithorax at 150 Hz were 5.6 ± 0.5 m/s (L1), 5.3 ± 0.4 m/s (L2), and 5.8 ± 0.6 m/s (L3), with 200 Hz measurements yielding 6.5 ± 0.6 m/s (L1), 6.2 ± 0.5 m/s (L2), and 6.4 ± 0.7 m/s (L3) (**Table 2**).

Table 2. *Baseline Mean Wave Speeds Measured by Lung Ultrasound Surface Wave Elastography (LUSWE) Across Different Thoracic Locations and Frequencies (m/s, mean±SD).*

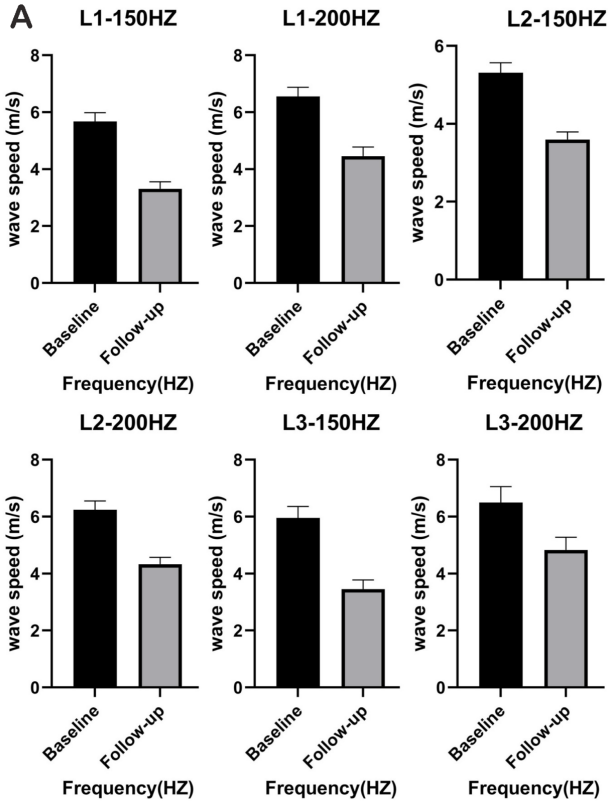
Thoracic Location	Frequency (150 Hz) Mean Wave Speed (m/s)	Frequency (200 Hz) Mean Wave Speed (m/s)
R1	5.4 ± 0.5	6.3 ± 0.6
R2	5.7 ± 0.4	6.6 ± 0.5
R3	5.2 ± 0.6	6.8 ± 0.7
L1	5.6 ± 0.5	6.5 ± 0.6
L2	5.3 ± 0.4	6.2 ± 0.5
L3	5.8 ± 0.6	6.4 ± 0.7

Following diuresis, all follow-up wave speeds decreased significantly. In the right hemithorax, 150 Hz velocities were 3.2 ± 0.4 m/s (R1), 3.5 ± 0.3 m/s (R2), and 3.7 ± 0.5 m/s (R3), with 200 Hz values of 4.5 ± 0.5 m/s (R1), 4.2 ± 0.4 m/s (R2), and 4.8 ± 0.6 m/s (R3). Left hemithorax follow-up measurements at 150 Hz were 3.3 ± 0.4 m/s (L1), 3.6 ± 0.3 m/s (L2), and 3.4 ± 0.5 m/s (L3), while 200 Hz velocities were 4.4 ± 0.5 m/s (L1), 4.3 ± 0.4 m/s (L2), and 4.6 ± 0.6 m/s (L3) (**Table 3**).

Table 3. *Mean Wave Speeds at Follow-Up Following Diuresis Measured by Lung Ultrasound Surface Wave Elastography (LUSWE) Across Different Thoracic Locations and Frequencies (m/s, mean ± SD).*

Thoracic Location	Frequency (150 Hz) Mean Wave Speed (m/s)	Frequency (200 Hz) Mean Wave Speed (m/s)
R1	3.2 ± 0.4	4.5 ± 0.5
R2	3.5 ± 0.3	4.2 ± 0.4
R3	3.7 ± 0.5	4.8 ± 0.6
L1	3.3 ± 0.4	4.4 ± 0.5
L2	3.6 ± 0.3	4.3 ± 0.4
L3	3.4 ± 0.5	4.6 ± 0.6

Figure2A&B presents comparative analysis of pulmonary surface wave velocities across all assessment sites in 6 subjects at baseline versus follow-up for excitation frequencies of 150 Hz and 200 Hz. Follow-up LUSWE assessments revealed statistically significant reductions in wave propagation speeds relative to baseline values ($p<0.001$) at each frequency and thoracic location. Longitudinal analysis revealed significant reductions ($p<0.001$) in mean pulmonary surface wave velocities across all frequencies (150, 200 Hz) among the 6 subjects when comparing follow-up to baseline measurements.



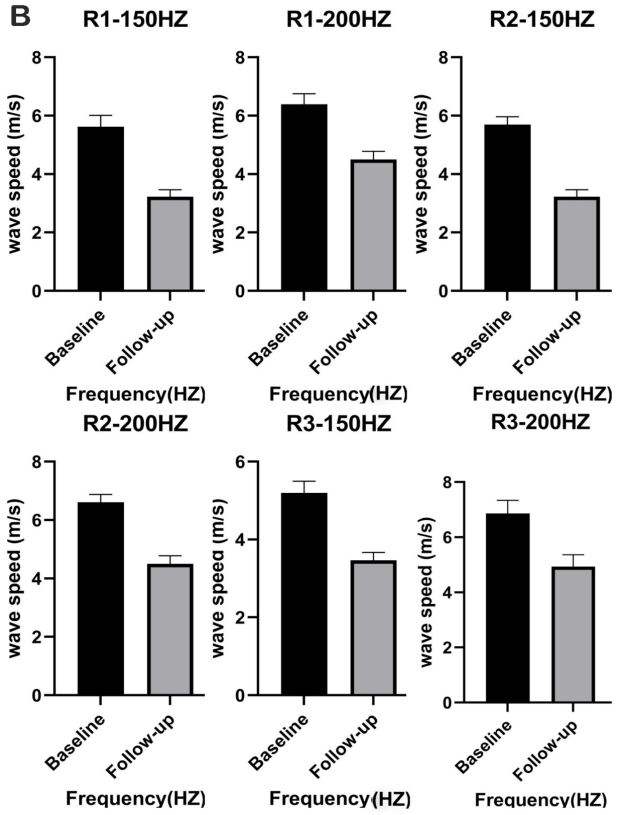


Figure 2 (A&B). Comparative analysis of pulmonary surface wave velocities (150 Hz, 200 Hz) across all thoracic sites in 6 subjects at baseline vs. follow-up. Follow-up LUSWE assessments showed statistically significant reductions in wave propagation speeds relative to baseline ($p<0.001$) at both frequencies and all locations, confirmed by longitudinal analysis.

2D-LUS evaluation revealed a significant reduction in total B-line counts from baseline to follow-up assessment, with a mean decrease of 11 using the 28-zone protocol. Concurrently, diuretic therapy achieved a mean net fluid balance shift of -2.3 liters. Crucially, the magnitude reduction in pulmonary surface wave velocity exhibited a significant inverse correlation with B-line reduction.

Discussion

This pilot study investigates the clinical feasibility of implementing a novel LUSWE technique for the quantitative bedside assessment of pulmonary edema in patients experiencing ACHF. To validate the LUSWE measurements, 2D-LUS—an established semi-quantitative modality for evaluating ELVW, indicative of cardiogenic pulmonary edema—was employed concurrently.

The advancement of elastography techniques has provided critical tools for assessing tissue mechanical properties. While conventional Shear Wave Elastography (SWE) utilizes the Acoustic Radiation Force (ARF) of ultrasound to generate tissue motion, its application is contraindicated in vulnerable organs. Specifically, the relatively high-intensity ultrasound fields employed by ARF-SWE carry a risk of inducing tissue damage, such as alveolar hemorrhage in the lungs, as documented in prior studies[11]. Furthermore, prolonged exposure to high-intensity ultrasound pulses may potentially lead to detrimental effects on the ultrasound system itself, including significant voltage drops and damage to probe elements. In contrast, LUSWE presents a fundamentally different and safer paradigm for wave excitation. This technique circumvents the use of acoustic radiation force entirely. Instead, it induces surface waves through the application of a gentle, external mechanical vibration on the skin surface. Consequently, the role of the diagnostic ultrasound system is restricted solely to the passive detection of wave propagation, thereby eliminating the risk of ultrasound-induced bioeffects during the wave generation phase [12]. This enhanced safety profile renders LUSWE suitable for clinical assessment of delicate organs, including the lung and the eye[13]. Beyond its superior safety, LUSWE also demonstrates advantages in wave generation efficacy. Although SWE relies on a short-duration ultrasonic “push” pulse, the mechanical vibration source utilized in LUSWE is capable of producing a more powerful and robust wave field[14]. The combination of this stronger excitation capacity with its inherent safety makes LUSWE a particularly promising technique for quantitative elasticity assessment in sensitive organs, such as the eyes.

In the current clinical implementation, surface wave speeds are quantified at two distinct frequencies: 150 Hz and 200 Hz. The signal amplification is calibrated individually for each frequency to optimize measurement quality. The wave motion generated at 150 Hz exhibits a higher amplitude

compared to those at higher frequencies. Although waves of 200 Hz provide a shorter wavelength and thus enhanced spatial resolution, they are also subject to more pronounced attenuation over propagation distance. The selection of this specific frequency range was guided by a balance among key factors, including wave motion amplitude, spatial resolution, and attenuation characteristics. Based on the wave speeds measured at these frequencies, the material's elastic modulus (μ_1) and viscous modulus (μ_2) can be derived by applying the Voigt viscoelastic model, utilizing a nonlinear least-squares fitting algorithm for parameter estimation[15]. While validated clinical imaging techniques for lung mass density analysis are currently lacking, Zhang et al. recently established deep neural network models based on LUSWE to assess lung mass density in interstitial lung disease patients[16]. Separately, Zhou and colleagues pioneered a methodology correlating lung mass density with computed tomography Hounsfield units[17]. This article exclusively reports wave speed data; however, future work will develop methodologies to assess mass density in cardiogenic pulmonary edema patients through analysis of lung viscoelasticity using wave speed measurements.

Diuretic therapy constitutes the gold standard for managing symptomatic pulmonary edema in acute congestive heart failure, effectively facilitating EVLW clearance[18]. Our longitudinal assessment further revealed a significant reduction in B-line density from baseline to follow-up examinations, consistent with diuretic administration. This finding reflects a global decrease in EVLW burden, corroborating prior validations of 2D LUS B-lines as quantitative biomarkers for monitoring EVLW resolution[19].

A fundamental limitation of relying exclusively on 2D LUS for detecting and monitoring EVLW alterations stems from the modality's quasi-quantitative assessment framework, which inherently introduces subjectivity and measurement variability. Specifically, this approach necessitates systematic scrutiny of each intercostal space recording to identify the exact frame containing the maximal B-line density. Subsequent summation of B-line counts across all zones to compute the composite 28-zone score constitutes a labor-intensive process. Crucially, result accuracy exhibits significant operator dependency, generating substantial inter-observer discrepancies that compromise diagnostic consistency.

Integrating 2D-LUS with LUSWE, as implemented in this investigation, enables the establishment of a robust quantitative framework for assessing superficial pulmonary stiffness. This approach may facilitate the derivation of an automated EVLW quantification index, providing patient-specific biomarkers for precise temporal monitoring of therapeutic efficacy (hourly/daily resolution). For instance, combined 2D-LUS/LUSWE protocols could be integrated into the clinical management of hospitalized and ambulatory heart failure cohorts, detecting both overt and subclinical pulmonary edema to optimize diuretic regimens.

Within inpatient settings, daily multimodal ultrasonographic assessments permit individualized titration of decongestive therapy based on quantitative changes in biomechanical and sonographic parameters. This patient-centric methodology—where each subject serves as their own biological control—objectively documents diuresis-induced improvements in pulmonary compliance.

For outpatient management, 2D-LUS/LUSWE deployment shows significant translational potential. Empirical evidence demonstrates that escalating subclinical B-line burden (reflecting occult EVLW accumulation) predicts heightened hospitalization and mortality risks in both ambulatory and hospitalized heart failure populations[20]. Preemptive surveillance using this dual-modality approach could trigger early therapeutic intervention prior to symptom manifestation.

Although current protocols require approximately 30 minutes per assessment, prior clinical studies in interstitial lung disease confirm LUSWE's feasibility in large-scale research contexts [21]. We project substantial reductions in examination duration through ongoing technical refinements of the LUSWE methodology.

In the present investigation, EVLW reduction was inferred from decreased cumulative B-line counts concurrent with effective diuresis, evidenced by significant body weight reduction. The validity of B-line quantification as an EVLW biomarker is substantiated by strong linear correlations demonstrated in gravimetric porcine models and corroborated in human studies against the invasive gold standard of transpulmonary thermodilution[22].

This study aims to advance a noninvasive, portable platform for monitoring EVLW. Crucially, lung ultrasound B-lines demonstrate superior sensitivity over chest radiography in detecting EVLW [23]. However, while sonographic B-lines exhibit reasonable correlation with EVLW volume, their diagnostic utility is constrained by operator-dependent visual interpretation and substantial inter-observer variability[24]. The qualitative-to-semi-quantitative nature of B-line quantification limits its reliability for longitudinal assessments, particularly in serial evaluations of pulmonary edema burden across inpatient and outpatient heart failure populations.

Our investigation revealed significant attenuation of lung

surface wave velocity following diuretic intervention, with post-admission reductions correlating strongly with decreased B-line indices and negative fluid balance. These LUSWE-derived biomechanical changes reflect enhanced pulmonary compliance, directly corresponding to EVLW clearance mediated by decongestive therapy.

Limitation

As a proof-of-concept investigation, this study presents notable methodological attributes and inherent limitations that merit rigorous consideration. The enrollment was restricted to a limited cohort, excluding subjects with pre-existing interstitial lung disease, or mechanical ventilation requirement. Notably, 2D-LUS evaluation of fibrotic pulmonary parenchyma demonstrates persistent B-line artifacts—clinically designated as "fixed" or "dry" B-lines—which remain unresponsive to decongestive therapy.

Furthermore, while EVLW accumulation may originate from inflammatory processes or transudative mechanisms, our protocol specifically targeted transudative pathophysiology. The differential diagnostic potential of LUSWE in distinguishing inflammatory versus transudative EVLW etiologies constitutes an active research trajectory currently under investigation.

Conclusion

The lung surface wave speed was assessed at bedside through a novel LUSWE method integrated with conventional 2D-LUS. Marked variations in wave speed across three frequencies were noted between baseline and follow-up assessments. We propose that these changes reflect diminished lung stiffness, likely resulting from a reduction in EVLW following diuretic treatment. The observed decrease in wave speed aligned with other indicators of EVLW reduction, such as decreased B-line presence and effective fluid elimination via diuretics.

Conflicts of Interest: The authors report no conflicts of interest.

Ethical statements: All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1964 and later versions. This study was approved by the Ethics Committee of Zhuji People's Hospital.

Author contributions

Conceptualization: XiangYu Chen.

Data curation: XiangYu Chen.

Writing – original draft: Ling Wang, XiangYu Chen.

Writing – review & editing: Ling Wang, XiangYu Chen.

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