Mohammed et al. Scr Med. 2026 Mar-Apr;57(2):255-65. DOI:10.5937/scriptamed57-65172

ORIGINAL ARTICLE

Role of Lung Ultrasound in Determining the Endpoint of Fluid Therapy in Patients With Septic Shock

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Abstract

Background/Aim: Fluid resuscitation in septic shock is essential, but determining the optimal volume remains challenging. This research aimed to evaluate the role of lung ultrasound (LUS) in assessment of endpoint of fluid therapy in septic shock.

Methods: This prospective observational research comprised 60 adults with septic shock admitted to critical care unit between May 2024 and December 2025. Fluid responsiveness was assessed by left ventricular outflow tract velocity–time integral (LVOT VTI) variation during passive leg raise; ≥ 10 % increase defined responders. LUS was performed using simplified, quantitative eight zone and qualitative eight zone protocols. Echocardiographic evaluation of left ventricular systolic and diastolic function, along with inferior vena cava collapsibility index (IVCCI), was also performed.

Results: Thirty-five patients (58.3 %) were fluid responders and 25 (41.7 %) non-responders. Non-responders exhibited lower IVCCI and higher prevalence of diastolic dysfunction, particularly grades II–III. The simplified LUS score (cut-off ≥ 11) showed excellent discrimination of fluid unresponsiveness [AUC 0.913 (95 % CI 0.844 – 0.983)]. The quantitative 8-zone score (cut-off ≥ 4) had good performance [AUC 0.834 (95 % CI 0.732 – 0.942)], while the qualitative protocol had the highest specificity for discriminating fluid unresponsiveness (sensitivity 76 %, specificity 85.5 %). Combining LUS with echocardiography improved identification of patients unlikely to benefit from further fluid.

Conclusion: LUS, particularly the simplified protocol, may serve as a bedside tool to predict fluid unresponsiveness and guide resuscitation endpoints in septic shock. Combined with echocardiography, it supports a multimodal, individualised approach that minimises overload risk.

Key words: Fluid therapy; Shock, septic; Ultrasonography.

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Citation:
Mohammed WSE, Gouda TE, Hafez AA, Elmansy M, Mousa SAE. Role of lung ultrasound in determining the endpoint of fluid therapy in patients with septic shock. Scr Med. 2026 Mar-Apr;57(2):255-65.

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Received: 25 February 2026
Revision received: 13 April 2026
Accepted: 13 April 2026

Introduction

Septic shock is a prevalent reason of death among critically ill cases. The pathophysiology of sepsis includes vasodilation, heightened microvascular permeability and capillary leaking, resulting in diminished intravascular volume. Fluids are crucial for enhancing cardiac output (COP) and organ perfusion.[1]

Determining the optimal volume of fluids remains challenging. Reliable instruments to improve preload whereas preventing complications of excessive fluid administration are needed. Guidelines suggest utilising dynamic measures instead of static indices to evaluate fluid responsiveness so that clinician could tailor fluid resuscitation to

enhance perfusion whereas avoiding fluid overload. These measures depend on preload-modifying procedures, including the passive leg raise test, followed by evaluations of COP alterations through pulse pressure change and stroke volume variation as alternative measures.[2]

Identifying responsiveness to fluids has to be balanced with the existence of fluid tolerance described as the degree to which a case can tolerate fluid administration without falling into organ dysfunction.[3] The coordinated assessment of these two variables could prevent and reverse acute organ dysfunction, hence imposing a new duty on intensivists: fluid responsibility.[4]

Recently, lung ultrasound (LUS) has emerged as a new non-invasive, rapidly available instrument to assess over hydration and may offer a valuable safety threshold to guide fluid therapy and to optimise volume status in critically diseased cases due to its ability to detect real time rise of extravascular lung water (EVLW). LUS isn't supposed to substitute invasive haemodynamic monitoring or alternative techniques for evaluating fluid responsiveness, but rather to assist intensive care clinicians through establishing a safety threshold for fluid resuscitation.[5]

This research aimed to evaluate the role of LUS in evaluation of endpoint of fluid therapy in septic shock.

Methods

This prospective observational research included 60 adult cases with septic shock admitted to medical Critical Care Unit at Specialised Medical Hospital, Mansoura University, Egypt between May 2024 and December 2025. Fluid responsiveness was assessed using left ventricular outflow tract velocity–time integral (LVOT VTI) variation throughout the passive leg raise (PLR) test. Patients with a VTI rise ≥ 10 % were classified as fluid responders, whereas those with < 10 % increase were considered non-responders.

Inclusion criteria: Cases more than eighteen years old of either sex diagnosed as septic shock. Septic shock has been defined according to sepsis -3 definition: Sepsis with persisting hypotension needing vasopressors to preserve mean arterial pressure (MAP) not less than 65 mm Hg and serum lactate concentration > 2 mmol/L (18 mg/dL) in spite of suitable volume resuscitation.[6]
Exclusion criteria: Patients less than 18 years old, non-septic origin of shock state (cardiogenic, obstructive, hypovolemic), heart failure (ejection fraction less than 50 %) and valvular heart disease, atrial fibrillation, patients with known lung pathology (pulmonary fibrosis and chronic obstructive pulmonary disease (COPD), persistent pleural effusion, pneumonectomy), end stage kidney disease and patients on haemodialysis, patients on chemotherapy due to malignancy, increased intra-abdominal pressure as in pregnancy and portal hypertension, or cases refused to sign the consent form. All cases have been subjected to history taking, physical examination and laboratory investigations. APACHE II and SOFA scores were recorded on admission.

Transthoracic echocardiography

Echocardiographic readings were the average measurements of two qualified operators who were blind to clinical data. Each operator readings were the average of three consecutive measurements. All examinations were done using *Vivid T8* (*GE Healthcare*, Chicago, US) ultrasound machine using the 3Sc-RSphased array probe sector with frequency range of 1.7- 4 MHz. Echocardiographic examination followed standardised protocol depending on the recommendations of the American and European Societies of Echocardiography. This involved evaluation of cardiac dimensions and ejection fraction using M-mode as well as comprehensive valvular assessment.

Measurement of LVOT VTI variability using PLR test

In optimal apical five chamber view, pulsed wave Doppler (PWD) gate placed in the centre of the LVOT, about 5 mm from the aortic valve in the direction of the apex. VTI measurement started from the baseline at the left-most part of the negative deflection, trace distally along the outermost edge of the waveform, extended around the trough and returned to the baseline at the right-most part of the waveform, average of three successive VTI measurements were calculated.[7]
Using an automatic bed elevation method, the patient's trunk has been lowered from the semi recumbent to the supine position, whereas the lower limbs were raised to a 45° angle and kept in this position for two min then in the apical five-chamber view, VTI measurement was repeated.[8]

VTI variation = (VTI max –VTI min) / [(VTI max +VTI min) /2] x 100.[9] Accordingly, enrolled patients were classified into two groups: Fluid responders: VTI increased ≥ 10 % during the test and fluid non-responders: VTI increased < 10 % during the test.[8]

Evaluation of diastolic function (DD)

Through measurement of the following parameters: mitral inflow E wave velocity, A wave velocity, E/A ratio and using tissue Doppler to evaluate e′ velocity , E/ e′ ratio also measurement of left atrial volume index (LAVi) and peak tricuspid regurge (TR) velocity.[10] Patients were classified based on 2016 ASE / EACVI guidelines for assessment of diastolic dysfunction in cases with normal systolic left ventricle ejection fraction (LVEF), utilising the following cut-offs: lateral e′ < 10 cm/s, lateral E/e′ ratio > 13, tricuspid regurgitation (TR) velocity > 2.8 m/s and LAVi > 34 mL/m^2). If a patient fulfilled more than 50 % of the evaluated parameters, the diagnosis of diastolic dysfunction was established.[11] Grade I DD was assigned if E/A ratio was < 0.8 along with E velocity not more than 50 cm/s, Grade II DD was assigned if E/A ratio was < 2 or E/A ratio < 0.8 along with E velocity above 50 cm/s with at least two criteria: lateral E/e′ ratio > 13, LAVi > 34 mL/m^2 and TR velocity > 2.8 m/s. Grade III DD was assigned if E/A ratio > 2.[12]

Assessment of inferior vena cava collapsibility index (IVCCI)

IVCCI was determined as: IVCCI (%) = [(Dmax – Dmin) / Dmax] × 100, where D max is the maximal expiratory diameter and Dmin is the minimal inspiratory diameter.[8]

Lung ultrasound

Three different protocols for quantification of B lines and assessment of EVLW were used. All findings were the simultaneous observation of specialised radiologist and intensivist who were blind to clinical data using *GE Health Care, Vivid T8* ultrasound machine using 4C-RS curved array probe with frequency range of 1.8 – 6 MHz. Cases were scanned although in supine or semi recumbent position.

Simplified lung ultrasound protocol was performed. Examination of the four intercostal spaces (ICSs) was conducted: ICS between third and fourth ribs and ICS between sixth and seventh ribs to the right and left of the sternum and among the midclavicular and parasternal line. The number of single and confluent B lines was documented for each ICS and a score varying from 0-8 per ICS was assigned as follow: 0 = No B-line; 1 = One B-line/ICS; 2 = Two B-lines/ICS; 3 = Three B-lines/ICS; 4 = Four B-lines/ICS; 5 = Five B-lines/ICS; 6 = Confluent B-lines > 50 % ICS; 7 = Confluent B-lines > 75 % ICS; 8 = Confluent B-lines 100 % ICS. The total LUS score ranged from 0 to 32.[13] B-lines were defined as hyperechoic, vertical artifacts result from the pleural line, moves in a synchronous manner with lung sliding, attaining the screen bottom and maybe fading or obliterating A-lines.[8]

Quantitative 8 zones LUS score

The chest wall was partitioned into eight regions (four in each hemithorax: 2 anterior and 2 lateral). The anterior chest wall was defined from the sternum to the anterior axillary line (AAL) and separated into lower and upper sections (from the clavicle to 3rd ICS and from the 3rd ICS to the diaphragm). The lateral zone was among the posterior axillary line and AAL and was subdivided into upper and basal halves.[14] The existence of B-lines and their maximal coalescence has been recorded. Each zone has been evaluated on a scale from zero to three based on the most severe finding seen, as follows: Normal (A-lines or < 3 B-lines per rib space) = 0; Multiple well-separated B-lines (≥ 3 per rib space) = 1; Multiple coalescent B-lines = 2; Consolidation = 3. The highest values were documented in each zone and a cumulative total was determined, resulting in a total LUS that varied from 0 to 24.[15, 16]

Qualitative 8 zone LUS protocol

Chest wall was divided into eight zones as previously described. Existence of three or more B lines in a single ICS was considered a positive zone. Abnormal pattern was identified when there were 2 or more positive zones in each hemithorax.[14] Representative images of different LUS patterns used for score calculation are shown in Figure 1.

Lung ultrasound and echocardiography were performed for study assessment purposes only and did not dictate fluid administration decisions, which followed standard septic shock management protocols. All measurements were performed after all patients had received initial volume resuscitation with 30 mL/kg of intravenous crystalloid (normal saline), in accordance with the international guidelines of Surviving

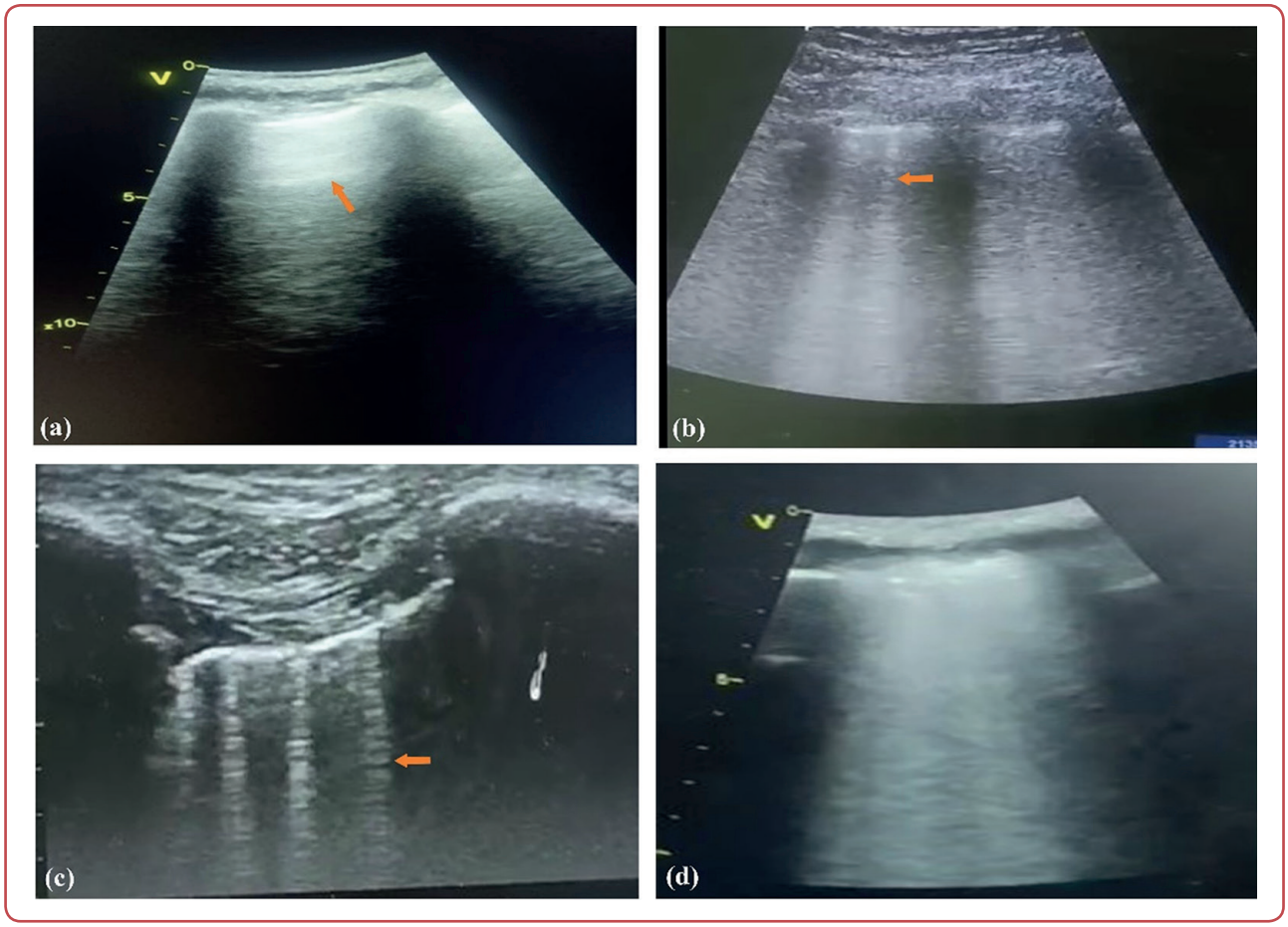

Figure 1: Lung ultrasound images
(a) Normal lung ultrasound (A-profile): A-lines are visible, no B-lines present, lung sliding normal. Arrow points to A line. (b) Two separate B-lines: Each B-line is hyperechoic, vertical, arises from the pleural line and moves with lung sliding (arrow points to B line). (c) Four B-lines within a single intercostal space: B-lines may indicate mild interstitial oedema. (d) Multiple coalescent B-lines: coalescent B-lines represent severe extravascular lung water.

Sepsis Campaign. Lung US scores were compared between responders and non-responders as well as all demographic, haemodynamic and echocardiographic data. All cases were followed up for ICU length stay and in hospital mortality.

Statistical analysis

Information has been examined utilising *IBM SPSS Statistics* version 27.0 and *MedCalc Statistical Software* version 20. Categorical informations were expressed as percentages and frequencies and the quantitative information received normality testing via the Shapiro-Wilk test. Normally distributed information was represented as mean ± standard deviation (SD) and non-normally distributed information as median with interquartile range. Comparisons between two groups utilised Fisher's exact, Chi-square, or Fisher-Freeman-Halton exact tests for categorical data and independent-samples t-test or U-test for quantitative variables. The diagnostic performance was evaluated utilising the area under the receiver operating characteristic curve (AUC). Univariate and multivariate logistic regression analyses have been done to determine determinants of outcomes, with outcomes presented as odds ratios (OR) and 95 % confidence intervals (CI). Spearman's correlation coefficients were classified as low (0.1–0.3), moderate (0.3–0.5), or high (> 0.5) based on their ability to evaluate correlations between numerical and ordinal variables. The Cochran-Armitage test was utilised to assess correlations between dichotomous and ordinal information. A p-value under 0.05 has been deemed statistically significant.

Results

Of the 60 patients enrolled, 35 were fluid responders and 25 were non-responders. There was insignificant variance in age between responders and non-responders, but male sex was significantly higher among non-responders. Comorbidities, sepsis causes, APACHE II score, SOFA score and mortality showed no significant differences between both groups. Non responders had a significantly higher baseline VTI, along with significantly lower VTI variation and lower IVC-

Table 1: Baseline clinical characteristics, echocardiographic parameters and lung ultrasound scores in fluid of responders (N = 35) and non-responders (N = 25)

Characteristic	Non-responders	Responders	p-value
Age (years)	66.0 ± 9.3	65.1 ± 9.7	0.720
Sex N (%)			
Female	7 (28.0)	20 (57.1)	0.025
Male	18 (72.0)	15 (42.9)	
Comorbidities N (%)			
Diabetes mellitus	11 (44.0)	17 (48.6)	0.726
Hypertension	8 (32.0)	19 (54.3)	0.087
Ischaemic heart disease	2 (8.0)	6 (17.1)	0.449
Chronic kidney disease	1 (4.0)	5 (14.3)	0.386
Cerebrovascular accidents	3 (12.0)	6 (17.1)	0.722
Malignancy	6 (24.0)	7 (20.0)	0.711
Cause of septic shock N (%)			
Pneumonia	6 (24.0)	8 (22.9)	0.575
Cholangitis	3 (12.0)	9 (25.7)	
Cellulitis	4 (16.0)	3 (8.6)	
Pyelonephritis	7 (28.0)	7 (20.0)	
Acute pancreatitis	2 (8.0)	1 (2.9)	
Acute osteomyelitis	1 (4.0)	5 (14.3)	
Infected bed sores	2 (8.0)	2 (5.7)	
SOFA	8 (6 – 11)	10 (8 – 13)	0.059
APACHE II	14 (11 – 19)	15 (14 – 23)	0.136
ICU length stay (days)	7 (4.5 – 13.5)	7 (5 – 9)	0.582
In-hospital mortality N (%)	14 (56.0)	23 (65.7)	0.445
Baseline VTI (cm)	21.7 (17.4 – 22.7)	16.5 (13.9 – 20.1)	0.001
VTI variation after PLR (%)	3.6 (1.6 – 6.8)	14.5 (11.8 – 19.6)	< 0.001
Diastolic dysfunction (DD)			
No DD	6 (24.0)	18 (51.4)	0.004
Grade 1 DD	6 (24.0)	13 (37.1)	
Grade 2 DD	10 (40.0)	4 (11.4)	
Grade 3 DD	3 (12.0)	0 (0.0)	
IVC collapsibility index	27 (15.5 – 40.5)	50 (43 – 60)	< 0.001
Simplified LUS score (0- 32)	15 (11 – 16)	6 (4 – 10)	< 0.001
Quantitative 8 zone LUS score (0-24)	6 (4.0 – 8.5)	1 (0 – 3)	< 0.001
Qualitative 8 zone LUS (abnormal pattern)			
Yes	19 (76.0)	5 (14.3)	< 0.001
No	6 (24.0)	30 (85.7)	

Notes: Abnormal pattern = 2 or more positive zones in each hemithorax. Test of significance for categorical data was Chi-square test (c), Fisher's exact test (F) or Fisher-Freeman-Halton exact test (FFH). The test of significance for numerical data was independent-samples t-test for age and U-test for all other numerical data. Data expressed as mean (± SD), median (25th, 75th percentile), or absolute numbers N (%); LUS: lung ultrasound; VTI: velocity–time integral; ICU: intesive care unit; PLR: passive leg raise test;

CI compared to responders. Non-responders also exhibited a higher prevalence and more severe grades of diastolic dysfunction (Table 1).

Cochran-Armitage test of trend illustrated a significant association between increasing diastolic dysfunction grade and a higher proportion of non-responders ($p < 0.001$) (Figure 2).

Both simplified LUS score and quantitative 8 zone LUS scores were significantly higher in non responders than responders. Also, the presence of abnormal pattern detected by qualitative interpretation of 8 zone LUS protocol was significantly more frequent among non-responders than responders (Table 1). Simplified LUS score showed a strong negative association with VTI variation and a moderate negative association with IVCCI, alongside a moderate positive association with diastolic dysfunction grade. Quantitative 8-zone LUS score exhibited comparable correlations, including a significant negative association with VTI variation and IVCCI and a weaker yet statistically significant positive association with diastolic dysfunction grade (Table 2).

Receiver operating characteristic (ROC) curve analysis has been done to recognise an optimal cutoff value for LUS scores to detect fluid unresponsiveness. The study revealed that simplified LUS score at cutoff ≥ 11 significantly discriminated non-responders from responders (AUC [95 % CI] = 0.913 [0.844 – 0.983], $p < 0.001$) and quantitative 8 zone LUS score at cutoff ≥ 4 significantly discriminated non-responders from responders (AUC [95 % CI] = 0.834 [0.732 – 0.942], $p < 0.001$). Also, qualitative LUS score for presence of abnormal pattern showed good diagnostic performance for discriminating non responders (Table 3). IVCCI at a cut-off ≤ 42 % was a significant discriminator of non-responders, with an AUC of 0.889 (95 % CI 0.781–0.955, $p < 0.001$), sensitivity of 85.7 % and specificity of 88 % (Figure 3).

Univariate analysis was run to ascertain the effects of male sex, simplified LUS score ≥ 11, quantitative 8- zone LUS score ≥ 4, presence of abnormal pattern, diastolic dysfunction grade and IVCCI ≤ 42 % on the likelihood that septic shock patients will exhibit non-response to fluid therapy. All predictor variables were statistically significant (Table 4).

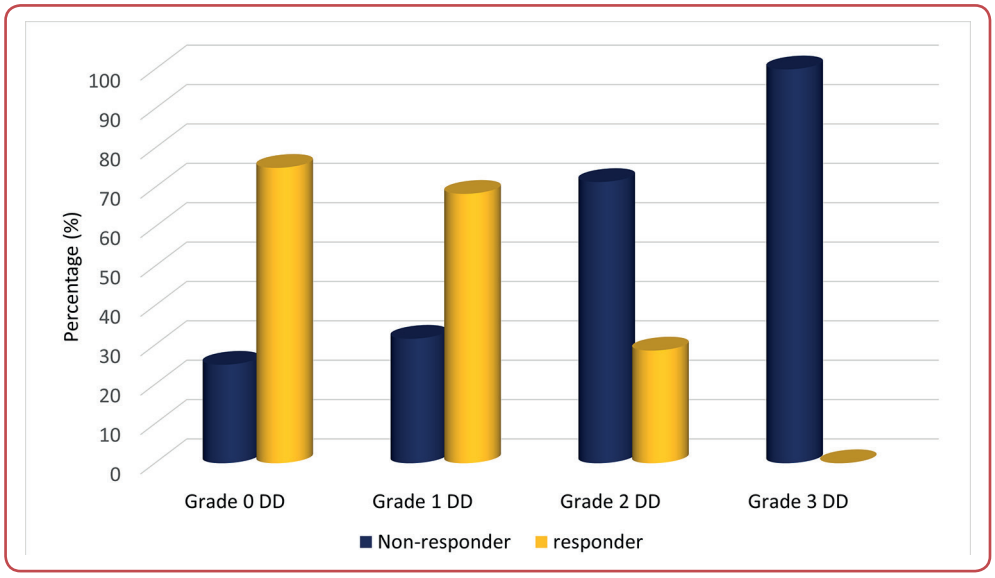

Figure 2: Proportions of fluid responders vs non-responders in each grade of diastolic dysfunction (DD)

Table 2: Correlation between quantitative lung ultrasound (LUS) scores and echocardiographic parameters

Echocardiographic parameter	Simplified LUS score		Quantitative 8-zone LUS score	
	r	p-value	r	p-value
VTI variation	-0.570	< 0.001	-0.463	< 0.001
IVCCI	-0.472	< 0.001	-0.521	< 0.001
Diastolic dysfunction grade	0.489	< 0.001	0.308	0.017

The test of significance was Spearman's correlation. VTI: velocity–time integral; IVCCI: inferior vena cava collapsibility index;

Table 3: Diagnostic performance of lung ultrasound (LUS) scores in predicting fluid unresponsiveness

Diagnostic indicator	Simplified LUS score	Quantitative 8-zone LUS score	Qualitative LUS score (abnormal pattern)
Sensitivity	88.0 %	84.0 %	76.0 %
Specificity	80.0 %	80.0 %	85.5 %
PPV	75.9 %	75.0 %	79.2 %
NPV	90.3 %	87.5 %	83.3 %
Overall accuracy	83.3 %	81.7 %	81.7 %
F1 score	81.5 %	79.2 %	77.6 %
LR +	4.4	4.2	5.32
LR -	0.15	0.2	0.28
MCC	67.1 %	63.2 %	62.1 %

Note: MMC = Matthews' correlation coefficient, PPV = positive predictive value, NPV = negative predictive value, LR+ = positive likelihood ratio, LR- = negative likelihood ratio. Abnormal pattern = 2 or more positive regions in each hemithorax);

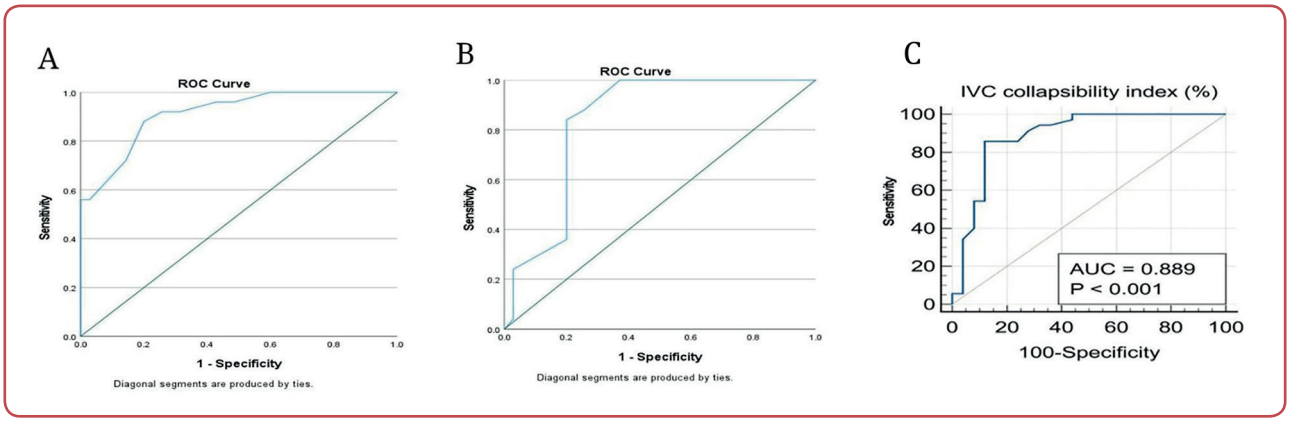

Figure 3: Receiver operating characteristic (ROC) curve of simplified lung ultrasound (LUS) scores
(A) ROC curve of simplified LUS score in discriminating non-responders from responders (AUC [95 % CI] = 0.913 [0.844 – 0.983], p < 0.001). (B) ROC curve for quantitative 8-zone LUS score in discriminating non-responders from responders (AUC [95 % CI] = 0.834 [0.732 – 0.942], p < 0.001). (C) ROC curve for inferior vena cava collapsibility index (IVCCI) for discriminating non-responders (AUC [95 % CI] = 0.889 [0.781 – 0.955], p < 0.001).

Table 4: Univariate analysis for prediction of fluid unresponsiveness

Predictor	COR	95 % CI of COR	p-value
Male sex	3.4	1.141 - 10.302	0.028
Simplified LUS score ≥ 11	29.3	6.790 - 126.718	< 0.001
Quantitative 8-zone LUS score ≥ 4	21.0	5.430 - 81.209	< 0.001
Abnormal pattern (presence of 2 or more positive zones in each hemithorax)	19.0	5.083 - 71.027	< 0.001
Diastolic dysfunction	8.4	2.279 - 30.924	0.001
IVC collapsibility index ≤ 42 %	44.0	9.495 - 203.900	< 0.001

Note: COR = crude odds ratio, CI: confidence interval; LUS: lung ultrasound; IVC: inferior vena cava;

Table 5: Multivariate regression analysis for prediction of fluid unresponsiveness

Predictor	p-value	AOR	95 % CI of AOR
Model (1)			
Sex	0.752	1.384	0.184 - 10.392
Simplified LUS score ≥ 11	0.005	37.487	2.999 - 468.527
Diastolic dysfunction	0.821	1.393	0.078 - 24.742
IVC collapsibility index ≤ 42 %	0.001	58.006	5.148 - 653.600
Model (2)			
Sex	0.591	0.582	0.081 – 4.188
Quantitative 8-zones LUS score ≥ 4	0.005	30.205	2.851 – 320.042
Diastolic dysfunction	0.476	2.448	0.209 - 28.670
IVC collapsibility index ≤ 42 %	0.001	60.140	5.723 - 631.983
Model (3)			
Sex	0.768	1.330	0.200 - 8.841
Presence of abnormal pattern	0.006	25.324	2.514 - 255.054
Diastolic dysfunction	0.407	2.638	0.267 - 26.087
IVC collapsibility index ≤ 42%	0.001	55.970	5.518 - 567.670

Note: AOR = adjusted odds ratio, CI: confidence interval; LUS: lung ultrasound; IVC: inferior vena cava;

Multivariate logistic regression analysis demonstrated that all three models were statistically significant ($p < 0.001$). Model 1 demonstrated that only simplified LUS score ≥ 11 and IVC collapsibility index ≤ 42 %. were statistically significant independent predictors of fluid unresponsiveness. Model 2 demonstrated that only quantitative eight zones LUS score ≥ 4 and IVC collapsibility index ≤ 42 % were statistically significant independent predictors of fluid unresponsiveness. Model 3 demonstrated that only presence of abnormal pattern and IVC collapsibility index ≤ 42 % were statistically significant independent predictors of fluid unresponsiveness (Table 5).

Discussion

Fluid resuscitation is essential for the initial treatment of cases with septic shock or sepsis and is thought to enhance clinical results. The lack of proof in terms of the amount of fluid required to resuscitate septic cases suitably results in significant variation in practice, with cases being either over-resuscitated, under-resuscitated.[17]

The present study demonstrated significantly higher baseline VTI in non-responders than responders which is in agreement with preceding studies.[18, 19] However, recent studies reported that baseline stroke volume, cardiac index, VTI and COP didn't vary between fluid responders and non-responders.[20] This incongruity may be due to the variance in study population.

Furthermore, a higher prevalence and higher grades of diastolic dysfunction were observed among non-responders. The higher the grade of diastolic dysfunction, the greater the proportion of non-responders, establishing it as a key predictor of unresponsiveness in septic shock cases.

Diastolic dysfunction is prevalent in ICU cases without history of cardiovascular diseases and normal LVEF and is related to increased death.[11] It is characterised by impaired relaxation or elevated filling pressures which limits preload accommodation making patients fluid-unresponsive despite hypovolemia. Consequently, increases in intravascular volume predominantly translate into elevated filling pressures rather than effective enhancement of COP.[21]

In the current study, non-responders showed significantly lower IVCCI compared to responders with strong discriminative ability for identifying fluid unresponsiveness. This finding aligns closely with previous critical care ultrasound studies emphasising the superiority of dynamic IVC indices over static measures and strongly support IVCCI as a reliable, non-invasive bedside predictor of fluid unresponsiveness, preload intolerance and venous congestion in septic shock.[22] The capability of IVCCI for predicting fluid responsiveness varies among studies. This variability is attributed to heterogeneity among the examined

subjects, the lack of a conclusive best approach for the measurement of fluid responsiveness and variability in operator method.[23]

In the current study, both simplified LUS score and quantitative 8 region LUS score showed significant negative correlations with IVCCI and VTI variation and significant positive correlation with the grade of diastolic dysfunction. These results reinforce the relationship between elevated LUS scores, increased filling pressures and venous congestion. Simplified LUS score was significantly elevated in non-responders than responders with a cutoff ≥ 11 showed excellent diagnostic performance for predicting fluid unresponsiveness, reinforcing the utility of lung ultrasound as a safe bedside instrument for guiding fluid therapy endpoints.

These findings are similar to previous studies that identified similar LUS cut off value with comparable diagnostic accuracy in detecting endpoint of fluid resuscitation and accurate prediction of increased EVLW.[24] Others reported that simplified protocol illustrated excellent correlation with EVLW index measured by transpulmonary thermodilution over a wide range of lung hydration grades although different cut off values were observed possiply due to absolute lung water quantification invasively rather than dynamic fluid responsiveness.[25] Recent study reported significant negative correlation between simplified LUSS and IVCCI reinforcing the association between lung ultrasound findings and intravascular congestion but showed a higher cut off with moderate accuracy for prediction of hypervolemia.[26]

This variation mostly due to protocol heterogeneity, patient populations and different reference standards. Collectively, these findings highlight that while absolute LUS cutoffs may vary, elevated LUS scores consistently identify pulmonary congestion and fluid intolerance across critical care settings. The current study demonstrated significantly higher quantitative eight zone LUS score in non-responders compared to responders with cut off ≥ 4 was optimal for predicting fluid unresponsiveness with good diagnostic performance. In comparison, a previous study evaluated multiple LUS scoring protocols against EVLWi evaluated by trans pulmonary thermodilution in cases with moderate-to-severe COVID-19 reported that eight regions anterior-lateral score correlated significantly with EVLW index and performed comparably to the more extensive global LUS protocol in detecting increased lung water with cutoff ≥ 8/24 showed high sensitivity but low specificity.[27] The lower cut off and higher specificity showed in the current study may be explained by variation in sample size, methodology and patient populations, suggesting the ability of current methodology to detect interstitial overload at earlier stages.

In the current research, qualitative assessment of eight zone LUS protocol showed that abnormal pattern, defined as two or more positive zones (≥ 3 B-lines) in each hemithorax, was significantly more frequent among non-responders than responders. This qualitative B-line based criteria demonstrated good diagnostic performance for predicting fluid unresponsiveness, reflecting its association with more advanced pulmonary congestion, indicating that qualitative ultrasound findings may confirm, rather than screen for, fluid unresponsiveness. A previous study evaluated the diagnostic accuracy of the qualitative eight zone LUS protocol against the transpulmonary thermodilution-derived EVLW index and demonstrated that the presence of ≥ 3 B-lines in any two regions on each side (abnormal pattern) exhibited low sensitivity but high specificity in predicting increased EVLWI, suggesting that the qualitative eight zone protocols may be more appropriate to include fluid overload instead of excluding it.[14] A substantially higher sensitivity in current study while maintaining comparable specificity, likely reflecting differences in the physiological endpoint assessed.

It was previously reported that presence of more than three positive chest quadrants illustrated good performance for predicting increased EVLW index however, detection of more than four positive chest quadrants at LUS examination illustrated higher specificity and lower sensitivity for expecting increased EVLW index.[28]

These findings highlight the importance of the extent and distribution of B-lines rather than their just presence. In this cohort, the requirement for bilateral involvement across multiple regions likely explains the balanced diagnostic performance observed and reinforces the clinical utility of qualitative LUS in guiding fluid management decisions.

Multivariate analysis revealed that LUS score, whether assessed using simplified protocol,

quantitative eight zone scoring, or qualitative B-line–based patterns, along with IVCCI were independent predictors of fluid unresponsiveness in septic shock. This result consistent with previous studies which reinforce that LUS can be safely used to decide early fluid restriction or de-resuscitation and highlighting the ability of LUS to accurately reflect pulmonary congestion and EVLW, thereby identifying patients at risk of preload intolerance and fluid overload.[24, 27]

Limitations of this study is that it is a single centre study with relatively small sample size, observational design which limits assessment of the impact of ultrasound-guided decisions on patient-centred outcomes and absence of comparison with transpulmonary thermodilution derived EVLWI or other invasive gold standards, which may affect lung ultrasound thresholds.

Conclusion

In conclusion, LUS may serve as a non-invasive bedside tool for predicting fluid unresponsiveness and detecting the endpoint of fluid therapy in septic shock, particularly the simplified protocol. Integrating LUS with echocardiography enables multimodal assessment, promoting individualised approach to fluid therapy that reduce overload risks in non-responders.

Ethics

This research was approved by the Institutional Research Board of Faculty of Medicine, Mansoura University (Approval No MD.24.03.840, dated 1 May 2024). Written informed consent has been obtained from all participants or their legal surrogates after ensuring confidentiality. The research has been conducted in line with the Declaration of Helsinki and its amendments.

Acknowledgement

The authors thank the staff of the Critical Care Unit, Faculty of Medicine, Mansoura University, for their cooperation and support in conducting this study.

Conflicts of interest

The authors declare that there is no conflict of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data access

The data that support the findings of this study are available from the corresponding author upon reasonable individual request.

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