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Predictive Value of Lung Ultrasonography in Differentiation between Cardiogenic Pulmonary Oedema and Acute Respiratory Distress Syndrome

Mona A. Elhadad [a] , Osama S.Arafa [b], Islam A. Zeater [a], Engy A. Okab [a]

[a] Anesthesia and Intensive Care Department, Faculty of Medicine Benha University, Egypt.

[b] Cardiology Department, Faculty of Medicine Benha University, Egypt.

Corresponding to:
Dr. Islam A. Zeater.
Anesthesia and Intensive Care Department, Faculty of Medicine Benha University, Egypt.
Email:
drislamashraf176@gmail.com

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Abstract:

Background: Acute respiratory distress syndrome (ARDS) is a common clinical syndrome of acute respiratory failure as a result of diffuse lung inflammation and oedema manifested by hypoxemia and stiffness in the lungs. Cardiogenic pulmonary oedema (CPE) occurs when the oedema is secondary to acute cardiac failure. This study aimed to determine the predictive value of chest ultrasonography to differentiate between acute CPE and ARDS and in patients admitted to ICU with acute dyspnea to allow proper management in both cardiac and non-cardiac one. **Methods:** This cross-sectional study included 60 patients divided to two equal groups: Group I: included ARDS patients and group II: included CPE patients. All studied cases underwent clinical examination, laboratory investigations, echocardiography and chest ultrasonography**. Results:** we found 100% sensitivity of abnormal pleural lines and 96.7% of absent lung sliding in prediction of ARDS, also spared lines showed high sensitivity of 93.3% and 100% specificity. The presence of consolidation also showed a high accuracy of 86.6% while that for effusion was 40%. Abnormal pleural lines, absent lung sliding, and spared lines had no role in prediction of CPE, LOW sensitivity and specificity of consolidation, presence of pleural effusion showed a higher accuracy of 60%. **Conclusion:** chest ultrasonography can provide valuable information for differentiating between ARDS and CPE in patients presenting with acute respiratory failure. The presence of abnormal pleural lines, absent lung sliding, spared areas, and consolidations are highly suggestive of ARDS, while their absence combined with the presence of pleural effusion is more indicative of CPE.

Keywords: Predictive; Lung Ultrasonography; Differentiation; Cardiogenic Pulmonary Oedema; Acute Respiratory Distress Syndrome.

Introduction

Acute hypoxemic respiratory failure secondary to pulmonary oedema is a life-threatening condition frequently found in intensive care units. Pulmonary oedema is an abnormal accumulation of extravascular lung water, which may occur when capillary permeability or hydrostatic pressure are increased. Increased capillary permeability is the mechanism underlying non-cardiogenic pulmonary oedema as in adult respiratory distress syndrome (ARDS), whereas the rise in hydrostatic pressure represents the underlying cause of dyspnea in patients with heart failure and cardiogenic pulmonary oedema (CPE) [(1)].

ARDS is a common clinical syndrome of acute respiratory failure as a result of diffuse lung inflammation and oedema manifested by hypoxemia and stiffness in the lungs. ARDS represents a significant proportion of patients with a prolonged hospital stay, especially ICU care, with a longer duration of mechanical ventilation [(2)]. CPE occurs when the oedema is secondary to acute cardiac failure. Although CPE lacks an inflammatory component, its heterogeneous distribution, impaired gas exchange and respiratory mechanics and high mortality rates are shared features with ARDS [(3)].

In current practice and most clinical studies, ARDS is usually differentiated from CPE by the clinical circumstances and by physical findings, but this distinction is often made only by post hoc review after patient discharge or death and is often based on the response to therapy. The ARDS Clinical Trial Network reported that fluid management to decrease cardiogenic fluid retention and the effects of lung permeability and edema will shorten the duration of mechanical ventilation and intensive care without increasing non-pulmonary organ failure [(4)].

Discriminating ARDS from acute CPE may be challenging in critically ill patients as there could be both overlapping clinical signs and confounders, including past history of respiratory or cardiac diseases. The differentiation between ARDS and CPE is important in order to avoid delaying treatment of fluid retention and avoiding unnecessary testing [(5)]. Since there is no definite "gold standard" for diagnosing ARDS or CPE, there is no technique or known biomarker that can be used to distinguish between the two conditions [(6)].

Chest sonography has emerged as a very promising technique owing to the high sensitivity it has shown in detecting various lung and pleural pathological conditions. Lung ultrasonography (LUS) is widely used to assess lung aeration and extravascular water content. LUS semiotics of interstitial diseases is mainly based on presence, number and distribution of artifacts generated at the level of the pleural line, namely B-lines, reflecting the loss of lung aeration regardless the etiology, on which all the scoring systems are based [(7)].

The purpose of the study was to determine the predictive value of chest ultrasonography to differentiate between acute CPE and ARDS and in patients admitted to ICU with acute dyspnea to allow proper management in both cardiac and non-cardiac one.

Patients and methods

This cross-sectional study included 60 patients and was carried out in Department of Critical Care Medicine, Benha University Hospitals, during the period from August ٢٠٢٣ to August 2024.

An informed written consent was obtained from the patients. Every patient received an explanation of the purpose of the study and had a secret code number. The study was done after being approved by the Research Ethics Committee, Faculty of Medicine, Benha University (**MS 35-5-2023).**

Inclusion criteria were patients of both sexes aged 18- 60 years old who fulfilled the criteria of ARDS according to Berlin's definition as the following [(8)]: Lung injury

of acute, within 1 week of an apparent clinical insult and with progression of respiratory symptoms, bilateral opacities on chest imaging not explained by other lung pathology (e.g. pleural effusion, pneumothorax, or nodules), respiratory failure not explained by heart failure or volume overload, decreased PaO_2/FiO_2 ratio: Mild ARDS: ratio is 201 – 300, moderate ARDS: 101 – 200 Severe ARDS: ≤ 100, and patients who fulfilled the criteria of CPE [9]: The presence of cardiogenic shock, EVLWi > 10 mL/kg, PVPI < 3.0 and echocardiographic signs of increased left atrial pressure, inferred by E/A < 0.75 or > 0.75 or E/A > 1.5 associated with E/E′ > 10, are diagnosed as CPE.

Exclusion criteria were patients aged younger than 18 years or older than 60 years, with a previous history of chronic obstructive pulmonary disease, emphysema, or lung fibrosis, and who refuse to take part in the study.

Grouping: Patients were divided into two groups: Group I: included 30 ARDS patients based on the Berlin ARDS definition of ARDS. Group II: included 30 CPE patients; based on clinical data, ECG, echocardiography, and chest radiography.

All studied cases were subjected to the following: Demographic data collection, including [Age, weight, height, and body mass index]. Complete history taking including: [Duration of symptoms and History of coronary artery disease]. Risk factors including: [Hypertension, diabetes mellitus, hyperlipidemia, previous ischemic stroke or transient ischemic attack, vascular disease, peripheral artery disease, smoking status, family history and chronic heart failure]. Clinical examination: [especially heart rate, systolic and diastolic blood pressure, assessment of body weight, body mass index and waist circumferences]. Laboratory investigations [complete blood count, fasting & random blood glucose level, lipid profile, serum creatinine, sodium and potassium, arterial blood gases and liver function tests].

Echocardiography:

Echocardiography was performed to all patients; the ECG was recorded simultaneously. Digital routine grayscale two-dimensional cine loops from three consecutive heartbeats were obtained at end-expiratory apnea from the standard parasternal long-axis view and three apical views at depths of 12–14 cm and mean frame rates of 67 ± 8 frames/sec. Sector width was optimized to allow for complete myocardial visualization while maximizing the frame rate. Standard LV measurements were obtained in accordance with the current guidelines of the American Society of Echocardiography/European Association of Cardiovascular Imaging [10].

We have two probes; a linear array operates between 6 to 12MHz Curvilinear array operates between 4 to 5 MHz transducer ultrasound machine: at the general ICU of Benha University Hospital was used for lung examination. The examination was performed at the patient's bedside. Lateral or seated positions were used to scan the posterior thorax. In patients in whom the seated position was not possible, a lateral decubitus position was used to examine posterior lung regions. Probes were placed vertically along each intercostal space (the parasternal line, anterior axillary line, and posterior axillary line) on both sides. Data were displayed on a screen. Each hemithorax should be divided into five zones: two anterior zones separated by the third intercostal space, two lateral zones, and one posterior zone.

Chest ultrasonography:

All patients were subjected to LUS examination of the chest upon occurrence of pulmonary oedema. Each hemithorax was divided into six areas: two anterior, two posterior, and two laterals. The anterior chest wall was delineated from the parasternal to the anterior axillary line and was divided into upper and lower halves.

A bedside lung evaluation was done with a convex probe 3.5–5 MHz and a linear probe 7.5–10 MHz (Mindary DP-1100 plus).

The following ultrasonographic signs was investigated to differentiate between ARDS and CPE [11]: alveolo-interstitial syndrome (AIS), pleural line sliding, pleural lines abnormalities, pleural effusion, spared areas, consolidations, lung pulse and subpleural consolidation.

Outcomes:

Primary outcome was determining the predictive value of LUS to differentiate between CPE and ARDS and in patients admitted to ICU with acute dyspnea. Secondary outcomes were in-hospital mortality of enrolled patients and the need for mechanical ventilation of enrolled patients.

Sample size

The sample size was calculated using G power sample size calculator version 3.1.9. The calculated minimal sample size is 60 patients.

Statistical analysis

Statistical analysis was done by SPSS v26 (IBM Inc., Armonk, NY, USA). Quantitative variables were presented as mean and standard deviation (SD) and compared between the two groups utilizing unpaired Student's t- test. Qualitative variables were presented as frequency and percentage (%) and were analyzed utilizing the Chi-square test or Fisher's exact test when appropriate. A two tailed P value < 0.05 was considered statistically significant. Evaluation of diagnostic performance include diagnostic sensitivity, specificity, positive predictive value (PPV), negative predictive value (PPV).

Results

The mean age of studied group I was (48.6±9.35 yrs.) and that of group II was (45.7 ±10.3 yrs. Male patients represent 56.7% and 50% of studied group I and II respectively. Hypertension and DM were the most common comorbidities (40% and 33.3% respectively) among group I cases, 33.3% and 50% respectively among group II cases with no significant difference, while IHD found among 66.7% of group II cases versus 16.7% of group I with significant difference. The vital signs (blood pressure, HR, RR, SPO_2 %, and temperature) of both studied groups has no significant difference among them. Table 1

ABG and investigations done for both studied groups, PO_2 ranged from 50 to 62, PCO_2 ranged from38 to 89 and HCO_3 ranged from19 to 35, K ranged from 2.4 to 6.2. Blood hemoglobin ranged from 8.5 to 16.5 g/dl with a mean of 13.96 g/dl among group I cases and 12.26 g/dl of group II, with no significant difference among both groups, there was a statistically significant decrease in P\F ration among ARDS cases and significant increase of WBC count. Regarding ECHO data, EF was a statistically significantly higher among cases of group I (ARDS) than CPE cases. With significant decrease in LVEDD and LVESD among group I. AIS was present in all of our studied cases (ARDS and CPE), indicating its high sensitivity in the diagnosis. All other ultrasonography signs presented a statistically significant difference in presentation between the two groups Table 2

Table 3 showed a 100% sensitivity of abnormal pleural lines and 96.7% of absent lung sliding in prediction of ARDS, also spared lines showed high sensitivity of 93.3% and 100% specificity. The presence of consolidation also showed a high accuracy of 86.6% while that for effusion was 40%. This table shows that abnormal pleural lines, absent lung sliding, and spared lines had no role in prediction of CPE, LOW sensitivity and specificity of consolidation, while presence of pleural effusion showed a higher accuracy of 60%.

The outcome of studied patients, 30% of them died in ICU and 70% discharged, with mean hospital stay of 9.5 ± 3.1 days. Table 4

Table 1: Basic characteristics and vital signs of the studied group

	Group I N=30	Group II N=30	P
Age (years)			
Mean ± SD	48.6 ± 9.35	45.7 ± 10.3	0.322
	32-60	35-60	NS
Gender	N (%)	N (%)	
Male	17 (56.7%)	15 (50%)	0.123
Female	13 (43.3%)	15 (50%)	NS
Comorbidities			
No	5 (16.7%)	7 (23.3%)	0.363 NS
HTN	12 (40%)	10 (33.3%)	0.432 NS
DM	10 (33.3%)	15 (50%)	0.213 NS
CKD	6 (20.0%)	5 (16.7%)	0.876 NS
IHD	5 (16.7%)	20 (66.7%)	**0.002 S**
Smoking			
No	6 (20%)	5 (16.7%)	0.765
Ex-smoker	11 (36.7%)	10 (33.3%)	NS
Current smoker	13 (43.3%)	15 (50%)	
Vital signs			
Systolic blood pressure (mm\Hg)	124.3 ± 11.7	123.3 ± 12.5	0.903
Range	90-150	90-160	NS
Diastolic blood pressure (mm\Hg)	80.9 ± 13.9	84.9 ± 10.4	0.324
Range	60-100	65-95	NS
HR	107.2 ± 11.5	110.2 ± 10.8	0.123
Range	88-120	85-120	NS
RR	22.7 ± 4.37	24.4 ± 3.23	0.763
Range	14-38	15-36	NS
SPO_2 (%)	89.5 ± 2.13	87.3 ± 4.22	0.211
Range	83-92	83-92	NS
Temperature	37.5 ± 0.39	37.8 ± 0.42	0.205
Range	37-38.5	37-38.5	NS

NS: P-value>0.05 is not significant

Table 2: Validity data of U/S findings as predictors in detection of ARDS and CPE

	Sensitivity	Specificity	PPV	NPV	Accuracy
	ARDS Detection				
Abnormal pleural Lines	100%	93.3%	93.8%	100%	96.7%
Absent lung Sliding	96.7%	100%	100%	96.8%	98.3%
Spared lines	93.3%	100%	93.8%	100%	96.7%
Consolidation	76.7%	96.7%	95.8%	80.6%	86.7%
Pleural effusion	46.7%	33.3%	41.2%	38.5%	40%
	CPE Detection				
Abnormal pleural lines	6.7%	0%	6.3%	0%	3.3%
Absent lung sliding	0%	3.3%	0%	3.2%	1.7%
Spared lines	0%	6.7%	0%	6.3%	3.3%
Consolidation	3.3%	23.3%	4.2%	19.4%	13.3%
Pleural effusion	66.7%	53.3%	58.8%	61.5%	60%

Table 3: ABG and investigations, ECHO data and chest U/S findings of the studied group

		Studied group I N=30		Studied group II N=30		P*
		Mean ± SD Range		**Mean ± SD**		
ABG and investigations						
PH		7.31 ± 0.093		7.32 ± 0.11		0.221 HS
Range		7.18-7.53		7.21-7.53		
$PaCo_2$ (mmol/L)		60.9 ± 15.4		63.5 ± 12.6		0.321 NS
Range		38-89		38-85		
PaO_2 (mmHg)		55.7 ± 3.52		53.7 ± 4.72		0.225 NS
Range		50-61		50-62		
HCo_3		26.7 ± 3.41		25.5 ± 4.12		0.763 NS
Range		19 ± 35		20 ± 35		
P\F ratio		165.7 ± 55.6		215.4 ± 61.2		**0.002 S**
Range		135-221		165-257		
K (mEq/L)		4.69 ± 0.93		3.89 ± 1.13		0.433 NS
Range		2.4-6.2		2.4-6.2		
SGOT (U/L)		39.6 ± 3.84		40.6 ± 5.24		0.776 NS
Range		16-45		16-46		
SGPT (U/L)		38.6 ± 7.45		35.6 ± 3.14		0.231 NS
Range		21-40		23-42		
Creatinine (mg/dl)		1.06 ± 0.38		0.96 ± 0.18		0.305 NS
Range		0.8-1.3		0.7-1.2		
Haemoglobin (g\dl)		13.96 ± 3.45		12.26 ± 3.55		0.109 NS
Range		8.5-16.5		8.5-16.5		
Platelets ($x10^3$)		363.5 ± 64.4		367.8 ± 54.4		0.219 NS
Range		256-556		250-546		
WBCs		11.4 ± 2.32		9.53 ± 3.22		**0.01 S**
Range		5.5-12.6		4.5-11.5		
Troponin						0.282 NS
Median (range)		0.5 (0 – 0.7)		1 (0.2 – 1)		
ECHO data						
EF Simpson (%)						<0.001 HS
Mean ±SD		53.6 ± 8.14		41.5 ± 3.15		
LVEDD						<0.001 HS
Mean ±SD		45.2 ± 4.48		35.1 ± 5.44		
LVESD						<0.001 HS
Mean ±SD		28.5 ± 3.14		21.3 ± 2.79		
US findings						
AIS	Yes	30	100	30	100	NS
Abnormal pleural lines	Yes	30	100	2	6.7	<0.001 HS
Absent or reduced lung sliding	Yes	29	96.7	0	0.0	<0.001 HS
Spared lines	Yes	28	93.3	0	0.0	<0.001 HS
Consolidation	Yes	23	76.7	1	3.3	<0.001 HS
Evidence of pleural effusion	Yes	14	46.7	20	66.7	0.007 S

*Independent sample t-test, NS: P-value>0.05 is not significant, HS: P-value<0.001 is high significant S: P<0.05 is significant

Table 4: Outcome and length of hospital stay among the studied group

		Group I N=30		Group II N=30		P
		N	%	N	%	
Outcome	Died	3	10.0	4	13.3	0.332
	Discharged	27	90.0	26	86.7	NS
Hospital stay (days)	Mean ± SD	9.5 ± 3.1		8.2 ± 2.12		0.063
	Range	5-18		4-12		NS
Mechanical ventilation		11	36.7	3	10	**0.03 S**

Discussion

In our study, the mean age was similar between the groups (48.6 years for ARDS and 45.7 years for CPE).

Male patients represent 56.7% and 50% of studied group I and II respectively with no significant difference in gender distribution. This is in line with a previous study conducted by Sanjan et al [(12)], who found patients were predominantly males, with an average age of around 50 years.

This is unlike Heffernan et al [(13)], who found that females have more risk for ARDS, which was explained by the proinflammatory role of oestrogen.

In our study, comorbidities were prevalent in both groups, with hypertension and diabetes mellitus being the most common. Interestingly, ischemic heart disease (IHD) was significantly more prevalent in the CPE group (66.7%) compared to the ARDS group (16.7%). This finding aligns with the known association between cardiac dysfunction and CPE [(14)].

In our study, there were no significant differences in vital signs between the two groups, which is not unexpected given that both conditions can present with similar clinical manifestations of respiratory distress. This underscores the challenge in differentiating ARDS from CPE based on clinical examination alone. Arterial blood gas analysis showed no significant differences in pH, $PaCO_2$, PaO_2, or HCO_3 between the groups. However, the P/F ratio was significantly lower in the ARDS group (165.7 ± 55.6) compared to the CPE group (215.4 ± 61.2).

This finding is consistent with the Berlin definition of ARDS, which uses the P/F ratio as a key criterion for diagnosis and severity classification [(15)].

In our study, the white blood cell count was significantly higher in the ARDS group, which may reflect the inflammatory nature of ARDS compared to CPE.

This finding is supported by Hutabarat et al [(16)] who reported elevated inflammatory markers in ARDS patients compared with the non-ARDS group**.**

In our study, echocardiography revealed significant differences between the two groups. The ARDS group had a higher ejection fraction (EF) and larger left ventricular end-diastolic and end-systolic diameters (LVEDD and LVESD) compared to the CPE group.

These findings are consistent with the pathophysiology of the two conditions, where CPE is often associated with left ventricular dysfunction, while cardiac function is typically preserved in ARDS [(17)].

The most striking differences between ARDS and CPE were observed in the chest ultrasonography findings were:

1) AIS that was present in all patients from both groups, indicating its high sensitivity but low specificity in differentiating ARDS from CPE. In accordance, Refaat et al [(7)] reported that, both groups cardiogenic and non-cardiogenic were characterized by AIS (alveolar interstitial syndrome) which is characterized by multiple diffuse vertical

artifacts (B-lines), and correlates with extravascular lung water by 100% .

2) Abnormal pleural lines were present in 100% of ARDS patients but only 6.7% of CPE patients. This finding showed excellent sensitivity (100%) and specificity (93.3%) for ARDS, with an accuracy of 96.7%. In harmony, Refaat et al. [7] reported that pleural line abnormalities were detected in 100%of non-cardiogenic group and 0%of cardiogenic group.

3) Absent or reduced lung sliding was observed in 96.7% of ARDS patients but none of the CPE patients. This sign demonstrated high sensitivity (96.7%) and specificity (100%) for ARDS, with an accuracy of 98.3%. This corroborates the findings of Copetti et al [18], who reported that reduced lung sliding was more common in ARDS than in acute pulmonary oedema (APE).

4) Spared areas were present in 93.3% of ARDS patients but absent in all CPE patients. This finding showed high sensitivity (93.3%) and specificity (100%) for ARDS, with an accuracy of 96.7%. In accordance, Kasem et al [19] reported that, spared area was absent in patients with CPE and present in all patients with ARDS.

5) Consolidation was more prevalent in ARDS (76.7%) compared to CPE (3.3%). This sign showed good sensitivity (76.7%) and excellent specificity (96.7%) for ARDS, with an accuracy of 86.7%. In Agreement with Sanjan et al [12] on 73 respiratory distressed patients found that consolidation was present in moderate (100%) and sever (92.3) % ARDS.

6) Pleural effusion was more common in CPE (66.7%) than in ARDS (46.7%). While this finding had moderate sensitivity and specificity for CPE, it was the only ultrasonographic sign that showed better predictive value for CPE than for ARDS. Similar results were reported by Refaat et al [7] who reported that, pleural effusion represents 40.5%of non-cardiogenic group &100%of cardiogenic group, pleural effusions were more frequently seen in APE than in ARDS. Absence of Pleural effusion has Sensitivity of59.5%& Specificity 100%

In our study, there was no significant difference in mortality or length of hospital stay between the two groups. However, the need for mechanical ventilation was significantly higher in the ARDS group (36.7%) compared to the CPE group (10%).

Kasem et al [19] found that most patients with ARDS required invasive mechanical ventilation, and CPAP was required in only 17.1% of patients with ARDS. However, the majority of patients with CPE required CPAP, and much less commonly, they required invasive mechanical ventilation and a simple oxygen mask. Regarding the outcome, in-hospital mortality was higher in the ARDS group than CPE group. This can be explained by numerous factors, including patient-related factors such as age and the severity of underlying disease.

Conclusion

Our study demonstrated that chest ultrasonography can provide valuable information for differentiating between ARDS and CPE in patients presenting with acute respiratory failure. The presence of abnormal pleural lines, absent lung sliding, spared areas, and consolidations are highly suggestive of ARDS, while their absence combined with the presence of pleural effusion is more indicative of CPE.

References

1. Swenson KE, Swenson ER. Pathophysiology of Acute Respiratory Distress Syndrome and COVID-19 Lung Injury. Crit Care Clin. 2021;37:749-76.
2. Matthay MA, Zemans RL, Zimmerman GA, Arabi YM, Beitler JR, Mercat A, et al. Acute respiratory distress syndrome. Nat Rev Dis Primers. 2019;5:18.
3. Amado-Rodríguez L, Rodríguez-Garcia R, Bellani G, Pham T, Fan E, Madotto F, et al. Mechanical ventilation in patients with cardiogenic pulmonary edema: a sub-analysis of

the LUNG SAFE study. J Intensive Care. 2022;10:55.
4. Tsuchiya N, Griffin L, Yabuuchi H, Kawanami S, Shinzato J, Murayama S. Imaging findings of pulmonary edema: Part 1. Cardiogenic pulmonary edema and acute respiratory distress syndrome. Acta Radiol. 2020;61:184-94.
5. Brusasco C, Santori G, Tavazzi G, Via G, Robba C, Gargani L, et al. Second-order grey-scale texture analysis of pleural ultrasound images to differentiate acute respiratory distress syndrome and cardiogenic pulmonary edema. J Clin Monit Comput. 2022;36:131-40.
6. Omar MM, Kamel MH, Wahdan SME-N, Adel El-Fallah A, AboYoussof SM. The Role of Chest Ultrasound Combined with Plasma Brain Natriuretic Peptide in The Differentiation between Cardiogenic and Noncardiogenic Pulmonary Edema. The Egyptian Journal of Hospital Medicine. 2023;90:1019-25.
7. Refaat MM, Elassal MA, Essawy TS, Hasaneen AB. Role of chest ultrasound to differentiate between acute cardiogenic pulmonary edema and non-cardiogenic pulmonary edema (acute respiratory distress syndrome). Benha Journal of Applied Sciences. 2022;7:15-20.
8. Matthay MA, Arabi Y, Arroliga AC, Bernard G, Bersten AD, Brochard LJ, et al. A New Global Definition of Acute Respiratory Distress Syndrome. Am J Respir Crit Care Med. 2024;209:37-47.
9. Zanza C, Saglietti F, Tesauro M, Longhitano Y, Savioli G, Balzanelli MG, et al. Cardiogenic Pulmonary Edema in Emergency Medicine. Adv Respir Med. 2023;91:445-63.
10. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28:1-39.e14.
11. El-Naggar T, Sharkawy SH, Abdel-Hamid HM, El-Din Mohamad HS, Mohamed RMA. Role of chest ultrasonography in differentiating between acute cardiogenic pulmonary edema and acute respiratory distress syndrome. Egypt J Bronchol 2016;10:319-23.
12. Sanjan A, Krishnan SV, Abraham SV, Palatty BU. Utility of Point-of-Care Lung Ultrasound for Initial Assessment of Acute Respiratory Distress Syndrome Patients in the Emergency Department. J Emerg Trauma Shock. 2019;12:248-53.
١٣. Heffernan DS, Dossett LA, Lightfoot MA, Fremont RD, Ware LB, Sawyer RG, et al. Gender and acute respiratory distress syndrome in critically injured adults: a prospective study. *J Trauma*. 2011;71:878-83; discussion 83-5.
١٤. Suling F, Agustian Z, Soenarta AA. Cardiogenic pulmonary edema et causa hypertension entering the emergency unit UKI General Hospital from January - November 2018. *J Hypertens*. 2019;37:23-9.
15. Ranieri VM, Rubenfeld GD, Thompson BT, Ferguson ND, Caldwell E, Fan E, et al. Acute respiratory distress syndrome: the Berlin Definition. Jama. 2012;307:2526-33.
16. Hutabarat E, Syarani F, Bihar S, Eyanoer P. Inflammatory Markers of ARDS Events among Patients with Severe and Critical COVID-19 Infection at Adam Malik General Hospital, Medan, North Sumatera. Respiratory Science. 2023;3:170-6.
17. Vignon P, Colreavy F, Slama M. Pulmonary Edema: Which Role for Echocardiography in the Diagnostic Workup? 2011. p. 177-94.
18. Copetti R, Soldati G, Copetti P. Chest sonography: a useful tool to differentiate acute cardiogenic pulmonary edema from acute respiratory distress syndrome. Cardiovasc Ultrasound. 2008;6:16.
19. Kasem SM, Soliman MM, Sadek SH, Thabet AA, Tawfeek SE-H, Elhefny NEAM. Predictive value of chest ultrasound in differentiation between cardiogenic and noncardiogenic pulmonary edema: a cross-sectional study. Research and Opinion in Anesthesia & Intensive Care. 2022;9.

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