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Study the correlation between Echocardiographic assessment of Pulmonary pressure and Outcome of Acute Respiratory Distress syndrome

Gehan S.Morsy[1], Mohamed A.Elassal[2], Tarek S.Essawy[3] and Basem M.Aglan[4]

[1]Critical Care Dept., Faculty of medicine, Benha University

[2]Internal Medicine Dept., Faculty of medicine, Benha University

[3]Chest Disease Dept., Faculty of medicine, Benha University

[4]Cardiothoracic surgery Dept., Faculty of medicine, Benha University

E-Mail: ggsaid63@gmail.com

Abstract

Background: Inflammation, noncardiogenic pulmonary edema, and injury to the alveolar epithelium and the endothelial barrier of the pulmonary arteries lead to sudden respiratory failure in a clinical entity known as respiratory distress syndrome (ARDS). Finding out how well PH evaluated by transthoracic echocardiography predicts 30-day in intensive care unit (ICU) patients with respiratory distress syndrome was the primary goal of study. **Methods:** Hundred individuals diagnosed with respiratory distress syndrome were included in observational research. Group I consisted of patients with ARDS and PH, group II consisted of patients with normal PASP. Each group received an equal number of patients. **Results:** ($r = 0.801$, $P < 0.001$) and LVEF ($r = 0.767$, $P < 0.001$) were positively correlated with PaO2/FiO2 in a substantial way. The results showed clear negative relationships with TR maximal PG ($r = -0.848$, $P < 0.001$), RAP ($r = -0.625$, $P < 0.001$), and PASP ($r = -0.986$, $P < 0.001$). For the purpose of predicting in ARDS patients with PHT, ROC analysis was performed for . A significant under the curve (AUC) of 0.917 was shown, with a 95% confidence range ranging from 0.843 to 0.991 ($P < 0.001$). With a sensitivity of 74.3% and a specificity of 100%, respectively, the optimal cutoff was < 1.5. **Conclusions:** A major predictor of 30-day in patients admitted to the ICU with ARDS is echocardiographic measurement of pulmonary hypertension (PH). Indicators of ventricular dysfunction, pulmonary artery systolic pressure, right atrial pressure, tricuspid regurgitation pressure gradient, and other important echocardiographic measures were substantially linked to increased death rates.

Keywords: Paranasal Sinuses, Anatomical Variations, and CT Scans

Introduction:

Inflammation, noncardiogenic pulmonary edema, alveolar epithelial and endothelial barrier disruption, and abrupt respiratory failure are the hallmarks of respiratory distress syndrome (ARDS) [1].

Respiratory distress syndrome (ARDS) is characterized by chronic pulmonary arterial pressure (sPAP) >30 mmHg or mean pulmonary arterial pressure (mPAP) >25 mmHg. It is possible for critically sick individuals to get PH due to pulmonary thrombosis, ARDS, sepsis, heart failure, or sepsis [2].

Patients with respiratory distress syndrome (ARDS) must undergo mechanical ventilation as part of their therapy. Numerous randomized controlled clinical studies have assessed the safety and effectiveness of different mechanical breathing techniques for ARDS. The only mechanical ventilation therapy that has been shown to increase survival rates so far is low tidal volume ventilation, which should be in all patients with respiratory distress syndrome (ARDS) (≤ 6 mL/kg projected body weight). Rescue treatment for patients with severe hypoxemia may include positive end-expiratory pressure, alveolar recruitment movements, and prone positioning [3].

In order to give an echocardiographic probability level of PH, a thorough echocardiographic examination for suspected pulmonary hypertension (PH) involves measuring the systolic pulmonary arterial pressure (sPAP) and identifying other symptoms that are indicative of PH. Echocardiographic evidence of PH, such as pressure estimation and RV overload/dysfunction indicators [4]. After pulmonary stenosis is excluded and non-invasive estimations of RA pressure (RAP) are considered, the peak tricuspid regurgitation velocity (TRV) and the tricuspid regurgitation pressure gradient (TRPG) are to estimate sPAP. Taking into account the fact that derived variables increase measurement errors and RAP estimates are not always accurate [5].

The echocardiographic likelihood of PH should be assigned based on the peak TRV, not the predicted sPAP, according to the guidelines. Although a peak TRV greater than 2.8 m/s might indicate PH, TRV is not a reliable indicator of PH existence or absence.88 There is insufficient evidence to lower the TRV threshold in light of the updated hemodynamic definition of PH (Supplementary Data, Section [6]).

Patients with severe tricuspid regurgitation (TR) or those with carbon monoxide levels due to liver disease or sickle cell disease (SCD), an incorrect assessment of the TR jet's tricuspid valve closure artefact, or an incorrect assignment of a peak TRV due to maximum velocity boundary artefacts can cause the TR velocity to underestimate or overestimate pressure gradients. A low, moderate,

or echocardiographic likelihood of PH is therefore defined by additional characteristics associated with RV shape and function [7].

The tricuspid annular plane systolic excursion (), RV fractional change (RV-FAC), RV free-wall strain, and tricuspid annulus velocity (S′ wave) are some of the echocardiographic measures of RV function that are derived from tissue Doppler imaging. Additionally, 3D echocardiography may lead to the determination of RV ejection fraction (RVEF). In addition, PH may be better diagnosed using the /sPAP ratio, which is a non-invasive assessment of RV-PA coupling [8].

study aimed to evaluate the prognostic value of transthoracic echocardiography-assessed pulmonary hypertension for 30-day in intensive care unit (ICU) patients with respiratory distress syndrome.

Patients and Methods:

One hundred individuals suffering from respiratory distress syndrome (ARDS) were the subjects of observational research. From March 2020 through March 2024, the research was carried out in the ICUs at Banha University Hospital and Quesna Central Hospital.

The Benha University Hospital Research Ethics Committee gave its permission. Before any patient or first-degree family could participate, their written informed permission had to be acquired.

Our study included patients between the ages of 18 and 70 who met the following criteria: ARDS diagnosed within one week of a known clinical insult; bilateral opacities on chest X-rays that could not be explained by effusion, collapse, or nodules; respiratory failure that was not caby fluid overload or heart failure; and transthoracic echocardiography done within 48 hours of admission.

Patients with congestive heart failure, congenital cardiac disease, severe mitral stenosis, aortic stenosis, or insufficiency were not eligible to participate.

Two equal groups of patients were formed: Here we have two groups: one for instances of respiratory distress syndrome (ARDS) linked to pulmonary hypertension, and another for situations when the systolic pressure of the pulmonary arteries is normal.

Methods:

All the studied cases were subjected to: In order to diagnose ARDS, collect data on clinical management, take a patient's history, conduct a physical examination, and conduct laboratory tests (complete blood count, serum electrolytes (sodium and potassium), renal function tests (urea and creatinine), arterial blood gases, lactate levels, and inflammation markers). At the time of ARDS diagnosis, the following symptoms must be present in the patient: Starts suddenly and occurs within a week after a recognized trauma. Nodules, effusion, or collapse may account for the bilateral opacities seen on chest x-rays. Are not experiencing respiratory failure as a result of fluid overload or cardiac failure.

We shall classify the severity of ARDS as follows: Mild respiratory distress syndrome (ARDS): - A Pao2/Fio2 ratio of 200-300 with a positive end expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) of 5 cm H2O or more. In a moderate case of respiratory distress syndrome (ARDS), the blood oxygen saturation to partial pressure ratio (Pao2/Fio2) must be between 100 and 200 the continuous positive airway pressure (CPAP) must be at least 5 cmH2O. Severe respiratory distress syndrome (ARDS): - Pao2/Fio2 = 100 ≦ with positive end expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) > 5 cmH2o.

Experienced cardiologists who were not privy to the study's goals conducted standardized echocardiographic assessments within one day after intensive care unit admission. Important metrics comprised:

Information on the right ventricle, including the maximum velocity of tricuspid regurgitation (TR), the right atrial pressure, and the tricuspid annulus systolic plane excursion () in centimeters. Having a measurement less than 1.6 cm will be to identify RV dysfunction.

Information on ventricular systolic function: ventricular ejection fraction (%) and ventricular systolic dysfunction (yes/no). EF < 50% will be to identify ventricular systolic dysfunction.

Information on the diastolic function of the ventricle: LV end-diastolic volume (E/E'), prevalence of ventricle dysfunction (Yes/No). E/E' > 13 individuals will be considered to have ventricular diastolic dysfunction. Based on the results of the transthoracic echocardiography (TTE), patients were divided into two groups: those with PHTN and those without.

Statistical analysis

Version 28 of SPSS (IBM, Armonk, New York, USA) was for statistical analysis. We the independent t-test to compare the groups under study based on the means and standard deviations of the quantitative data. The Chi-square test was to compare numerical and percentage-based categorical data. In order to forecast in ARDS patients with PHTN, ROC analysis were performed on several echo parameters. The Spearman's correlation was to conduct the correlation analyses. Echocardiographic characteristics were in multivariate logistic regression studies to forecast death in PHTN patients with ARDS. There were no one-sided statistical tests. Significance was determined by p-values lower than 0.05.

Results:

With a p-value of less than 0.001 and 0.004 respectively, Group II had a substantially higher mean arterial pressure (PH) than Group I. Group II

had a significantly lower heart rate than Group I (P-value <0.001). The two groups were not significantly different in terms of age, gender, temperature, respiration rate, salt levels, creatinine levels, hematocrit percentages, or white blood cell (WBC) counts. Compared to Group II, Group I had a considerably higher mean potassium level (P-value <0.001). Listing 1

Table (1) Summary of the research groups' demographics and laboratory results

		Group I (n = 50)	Group II (n = 50)	P-value
Age (years)		58 ±12	60 ±8	0.205
Gender	Males	28 (56)	25 (50)	0.548
	Females	22 (44)	25 (50)	
Mean arterial pressure (mmHg)		58 ±14	70 ±17	<0.001*
Temp (Celsius)		38.1 ±0.4	38.2 ±0.6	0.473
PH		7.1 ±0.21	7.21 ±0.16	0.004*
Heart rate (b/pm)		124 ±14	113 ±15	<0.001*
Respiratory rate (cycles/min)		22 ±3	23 ±5	0.436
Sodium (meq/L)		134 ±4	135 ±6	0.311
Potassium (meq/L)		5.4 ±0.9	4.7 ±0.9	<0.001*
Creatinine (mg/dl)		2.9 (1.1 - 5.6)	2 (0.9 - 7)	0.3
Hematocrit (%)		32 ±3	34 ±7	0.248
WBC count		16 ±3	17 ±5	0.589

*Denotes a major p-value; SD stands for standard deviation. Pulmonary artery, millimeters of mercury, percent, potential of hydrogen, and beats per minute are all abbreviations in medical terminology. units of measurement include milliequivalents per liter (meq/L), milligrams per deciliter (mg/dl), percent, and white blood cell (WBC).

The Group I had a substantially greater incidence of renal damage, vasopressor use, percentage of patients ventilated during echo, TR maximal PG, RAP, PASP, LV E/E', and LV diastolic dysfunction compared to Group II (P<0.05). Scores on the Glasgow Coma Scale and the PaO2/FiO2, were much lower in Group I than in Group II (P = 0.011 and 0.001 to correspond, respectively). Group I had a higher frequency of using percentages of inspired oxygen (FiO2 > 50%) compared to Group II (P-value <0.001). Half of the patients in Group I had RV (right ventricular) dysfunction, but only 30% of those in Group II did (P = 0.041). The median number of ventilator-free days in Group II was 30 (ranging from 0 to 30 days), with a P-value of less than 0.001, compared to a substantially lower number of 1 in Group I (ranging from 0 to 30 days). Second Table

Outcome, clinical findings, and echocardiographic data for the groups that were evaluated are shown in Table (2)

	Group I (n = 50)	Group II (n = 50)	P-value
kidney injury	40 (80)	25 (50)	0.002*
Glasgow Coma Scale	11 (3 - 15)	15 (6 - 15)	0.011*
FiO2			
>50%	45 (90)	30 (60)	<0.001*
< 50%	5 (10)	20 (40)	
Fluids received 6 hrs before echo (ml)	1500 ±226	1500 ±452	1.0
Receipt of vasopressors during echo	40 (80)	30 (60)	0.029*
Norepinephrine dose (ug/kg/min)	1.9 (0.8 - 3.5)	2 (0.2 - 3.5)	0.766
Ventilated during echo	35 (70)	15 (30)	<0.001*
PaO2/FiO2 ratio	100 (50 - 260)	185 (65 - 280)	0.001*
Echocardiographic findings			
TR maximum PG (mmHg)	55 ±12	22 ±3	<0.001*
RAP (mmHg)	10 (5 - 15)	5 (0 - 10)	<0.001*
PASP (mmHg)	66 ±14	28 ±3	<0.001*
(cm)	1.61 ±0.23	1.85 ±0.27	<0.001*
RV dysfunction	25 (50)	15 (30)	0.041*
LVEF (%)	48 ±10	52 ±9	0.061
LV systolic dysfunction	25 (50)	15 (30)	0.041*
LV E/E'	11.2 ±1.4	9.8 ±1.8	<0.001*
LV diastolic dysfunction	50 (100)	40 (80)	<0.001*
Outcome			
30-d	35 (70)	20 (40)	0.003*
Ventilator-free days (days)	1 (0 - 30)	30 (0 - 30)	<0.001*

The data is as the mean ± standard deviation, median (range), or frequency (%) with a noticeable p-value. numbers; percentages; The partial pressure of oxygen to fraction of inspired oxygen ratio is reby PaO2/FiO2.

Milliliters (ml) and micrograms per kilogram per minute (ug/kg/min) are the units of measurement. PASP is for pulmonary artery systolic pressure; TR stands for tricuspid regurgitation; RAP stands for right atrial pressure; and PG stands for pressure gradient. RV stands for the right ventricle, LVEF for the ventricle, and for the tricuspid annular plane systolic excursion. The ratio of the diastolic velocity of the mitral annulus to the inflow velocity of the mitral valve is E/E'.

Age, When comparing the group to the non-group, there was a significant increase in heart rate, respiration rate, potassium levels, creatinine levels, and white blood cell count (P-value <0.05). When comparing the group to the non-group, there was a significant decrease in MAP, PH level, sodium levels, and hemocrit percentages (P 0.05). Those who perished and those who made it through the time frame under consideration did not vary considerably with respect to gender or temperature. Third Table

Table (3) Demographics and laboratory results broken down by death rate

	Mortality		
	Yes (n = 65)	**No (15)**	**P-value**
Age (years)	60 ±13	52 ±4	0.002*
Gender			
Males	20 (57.1)	8 (53.3)	0.804
Females	15 (42.9)	7 (46.7)	
Mean arterial pressure (mmHg)	51 ±10	72 ±9	<0.001*
Temp (Celsius)	38.1 ±0.4	38.1 ±0.2	0.623
PH	7.01 ±0.19	7.3 ±0.07	<0.001*
Heart rate (bpm)	131 ±11	110 ±7	<0.001*
Respiratory rate (cycles/min)	23 ±2	20 ±3	<0.001*
Laboratory findings			
Sodium (meq/L)	133 ±3	138 ±1	<0.001*
Potassium (meq/L)	5.8 ±0.6	4.5 ±0.8	<0.001*
Creatinine (mg/dl)	3.3 (2.2 - 5.6)	1.1 (1.1 - 2.1)	<0.001*
Hematocrit (%)	31 ±3	36 ±2	<0.001*
WBC count	18 ±3	13 ±2	<0.001*

*Minimum statistically significant value; mmHg, Celsius, PH, bpm, milliequivalents per liter, milligrams per deciliter, %, and white blood cell are all abbreviations for units of measurement.

In the group, renal damage was evident in 100% of cases, but in the non-group, it was much less common (P < 0.001). The scores of patients who passed away on the Glasgow Coma Scale were significantly lower than the complete scores of survivors (P < 0.001). The group had a significantly higher prevalence of patients needing amounts of oxygen (FiO2 > 50%) compared to the non-group (P = 0.001). The group received larger dosages of norepinephrine than the non-group (P < 0.001). Among patients who did not survive, mechanical breathing was far more common (85.7%) than among those who did survive (33.3%, P < 0.001). The group had inferior oxygenation compared to the non-group, as shown by the considerable differences in the PaO2/FiO2 ratio (P < 0.001). The group had nno days without a ventilator, in contrast to the non-group which had almost the whole duration without a ventilator (P < 0.001). Patients who did not survive had a considerably larger TR maximal pressure gradient (PG) than those who did (P-value <0.001). In comparison to the non-group, the group had a significantly higher RAP (P-value of <0.001). The cohort had a considerably higher PASP compared to the living group (P < 0.001). When comparing the group to the non-group, there was a significant decrease in and LVEF (P < 0.001). The group had a considerably higher prevalence of RV dysfunction, LV systolic dysfunction, and LV E/E' ratio (P < 0.001). Section 4

Clinical and echocardiographic results categorized by rate (Table 4)

	Mortality		
	Yes (n = 65)	**No (15)**	**P-value**
kidney injury	35 (100)	5 (33.3)	<0.001*
Glasgow Coma Scale	3 (3 - 15)	15 (15 - 15)	<0.001*
FiO2			
> 50%	35 (100)	10 (66.7)	0.001*
< 50%	0 (0)	5 (33.3)	
Fluids received 6 hrs before echo (ml)	1571 ±178	1333 ±244	0.003*
Receipt of vasopressors during echo	35 (100)	5 (33.3)	<0.001*
Norepinephrine dose (ug/kg/min)	2 (1.1 - 3.5)	0.8 (0.8 - 0.8)	<0.001*
Ventilated during echo	30 (85.7)	5 (33.3)	<0.001*
PaO2/FiO2 ratio	85 (50 - 140)	180 (170 - 260)	<0.001*

Ventilator-free days (days)	0 (0 - 3)	30 (27 - 30)	<0.001*
Echocardiographic findings			
TR maximum PG (mmHg)	59 ±11	46 ±7	<0.001*
RAP (mmHg)	15 (10 - 15)	5 (5 - 15)	<0.001*
PASP (mmHg)	73 ±9	49 ±9	<0.001*
(cm)	1.51 ±0.17	1.85 ±0.16	<0.001*
RV dysfunction	25 (71.4)	0 (0)	<0.001*
LVEF (%)	45 ±9	58 ±4	<0.001*
LV systolic dysfunction	25 (71.4)	0 (0)	<0.001*
LV E/E'	11.7 ±1.3	10.1 ±1.1	<0.001*
LV diastolic dysfunction	35 (100)	15 (100)	-

*Highly significant P-value; n: quantile; %: percentage The partial pressure of oxygen to fraction of inspired oxygen ratio is reby PaO2/FiO2. Milliliters (ml) and micrograms per kilogram per minute (ug/kg/min) are the units of measurement. PASP is for pulmonary artery systolic pressure; TR stands for tricuspid regurgitation; RAP stands for right atrial pressure; and PG stands for pressure gradient. Right ventricle systolic excursion () is the acronym for "tricuspid annular plane." An abbreviation for "ventricle" and "ventricular ejection fraction" diastolic velocity of the mitral annulus divided by inflow velocity of the mitral valve

PaO2/FiO2 showed that there were strong positive relationships with (r = 0.801, P < 0.001) and LVEF (r = 0.767, P < 0.001). The results showed that there were strong negative relationships with TR maximal PG, RAP, PASP, and LV E/E'. Echocardiographic data were in multivariate logistic regression studies to predict in ARDS patients with PHTN, adjusting for gender and age. An increased risk of death of about 14% was related with a one unit rise in TR maximal PG (95% CI = 1.047 - 1.239, P = 0.003). Two studies found that there was an increased risk of death for every one unit rise in RAP (95% CI = 1.435 0 3.953, P = 0.001). The risk of death rose by 31% for every unit rise in PASP (95% CI = 1.105 - 1.553, P = 0.002). There was a 77.5% decrease in risk for every one unit rise in (95% CI = 0.1 - 0.648, P = 0.004). The odds ratio (OR) was 0.628 (95% CI: 0.429–0.918, P = 0.016), meaning that for every one unit increase in LVEF, the risk of death was 37.2% lower. The odds ratio (OR) was 10.93 (95% CI = 2.117 - 56.424, P = 0.004), meaning that the risk of death was ten times higher for every unit rise in LV E/E'. The fifth table

Table (5) Echocardiographic results and the PaO2/FiO2 ratio as well as multivariate logistic regression analysis of echocardiographic parameters for prediction

	PaO2/FiO2 ratio	
	r	**P**
TR maximum PG (mmHg)	-.848	<0.001*
RAP (mmHg)	-.625	<0.001*
PASP (mmHg)	-.986	<0.001*
(cm)	.801	<0.001*
LVEF (%)	.767	<0.001*
LV E/E'	-.850	<0.001*
	OR (95% CI)	**P-value**
TR maximum PG (mmHg)	1.139 (1.047 - 1.239)	0.003*
RAP (mmHg)	2.382 (1.435 - 3.953)	0.001*
PASP (mmHg)	1.31 (1.105 - 1.553)	0.002*
	0.255 (0.1 - 0.648)	0.004*
LVEF (%)	0.628 (0.429 - 0.918)	0.016*
LV E/E'	10.93 (2.117 - 56.424)	0.004*

Notable p-value; r: correlation coefficient; P: p-value PG stands for pressure gradient, TR for tricuspid regurgitation. LVEF is for ventricular ejection fraction; RAP stands for right atrial pressure; PASP stands for pulmonary artery systolic pressure; LV E/E' is the ratio of mitral inflow velocity to mitral annular diastolic velocity. The acronym "OR" stands for odds ratio, whereas "CI" stands for confidence interval.

ROC research was conducted on TR max PG to foretell death in PHTN patients with ARDS. With a 95% confidence range of 0.728 - 0.948 (P < 0.001), it showed a noteworthy AUC of 0.838. With a sensitivity of 71.4% and a specificity of 100%, the optimal cutoff was > 53. For the purpose of predicting in ARDS patients with PHT, ROC analysis was performed for . A significant under the curve (AUC) of 0.917 was shown, with a 95% confidence range ranging from 0.843 to 0.991 (P < 0.001). With a sensitivity of 74.3% and a specificity of 100%, respectively, the optimal cutoff was < 1.5. Picture 1

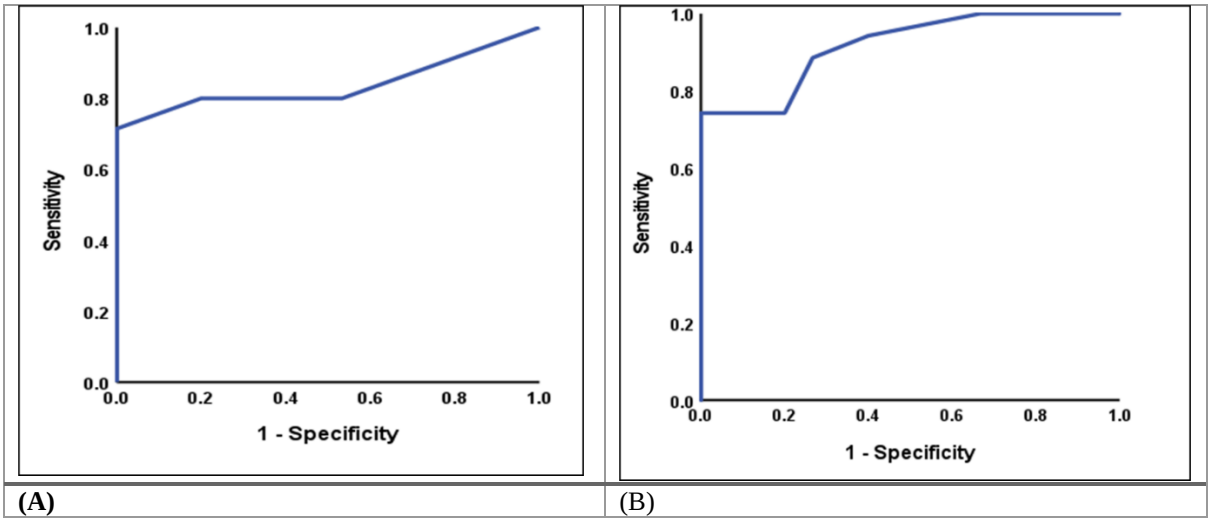

Fig. (1) ROC analysis for predicted in ARDS patients with PHTN using (A) TR max PG and (B)

Discussion

Inflammation and noncardiogenic pulmonary edema cause abrupt respiratory failure in respiratory distress syndrome (ARDS) [9]. Damage to alveoli and endothelial cells contributes to condition.

Group I had a greater risk of 30-day at 70% compared to 40% in Group II, which is an outcome measure. The number of days each group went without using a ventilator was significantly different. In comparison to Group II, Group I had a median of one day without a ventilator.

Also, a blinded cardiologist reevaluated , Tei index, RV-fractional change (RV-FAC), PASP, and septal shift in a retrospective single-center cohort pilot investigation by Shah et al. (JK). Included were 38 patients. The 30-day survival rate was to categorize the patients. Twenty patients made it through the first 30 days after TTE, whereas eighteen perished (i.e., 47% rate; 18 out of 38 patients died within time frame) [10].

In terms of demographics related to death, the group was noticeably older than the non-group. The group had a lower MAP than the non-group. Compared to the non-group, the group had a much lower PH level. In comparison to the non-group, the group had much greater heart and respiratory rates. Those who perished and those who made it through the time frame under consideration did not vary considerably with respect to gender or temperature.

The sodium levels in the group were noticeably lower than those in the non-group, according to the laboratory data that were analyzed by mortality. In comparison to the non-group, the potassium levels in the group were significantly greater. In comparison to the non-group, the group had a higher median creatinine level.

The group had lower hemocrit percentages than the non-group. In comparison to the non-group, the WBC count was noticeably greater in the group.

The average age of the group was significantly greater than that of the survivors in our analysis of patients with ARDS, indicating that age is a strong predictor of death in population. It has long been believed that older patients have less physiological reserves and are more likely to have negative outcomes in critical situations because of more comorbidities and less organ function capability [11]. finding is in line with that theory.

In comparison to those who did not survive, survivors had greater pH, PaO2■FiO2 ratios, and . Scores on the APACHE II, SAPS II, and SOFA were all lower among survivors compared to those who did not survive. In contrast to the moderate ARDS group, the severe ARDS group had a substantial reduction in and a rise in Tei index [10].

When looking at clinical data based on mortality, it was shown that kidney damage was present in 100% of the group, but much lower in the non-group (33.3%) ($P < 0.001$). Scores for patients who passed away were much lower than complete scores for survivors on the Glasgow Coma Scale. Patients in the group were more likely to need amounts of oxygen ($FiO2 > 50\%$) than those in the non-group (66.7% vs. 100%; $p = 0.001$). The fluid management was also substantially different across the groups; those who died received more fluids than those who did not ($P = 0.003$). It was shown that vasopressors were necessary in the group (100%) but only in a small percentage in the non-group (33.3%; $P < 0.001$).

was further shown by the norepinephrine dosage, which was greater in the group than in the non-group. Patients who did not survive were far more likely to have had mechanical breathing. Indicative of worse oxygenation in the group compared to the non-group, the PaO2/FiO2 ratio showed substantial differences. Finally, there was a striking difference in the number of days without a ventilator between the groups; those who died had practically no such

days, those who did not died had almost the whole duration.

If we look at the echocardiographic results by rate, we see that the TR maximum PG was much greater in the dead than in the living. Similarly, compared to the non-group, the group had a much higher median RAP. The cohort had a much higher PASP compared to the living cohort. The was significantly lower in the group that experienced death when contrasted with the group that did not ($P < 0.001$).

The group had a substantially higher prevalence of RV dysfunction and LV systolic dysfunction ($P < 0.00$). LVEF was likewise significantly lower in the group compared to the survival group. Those who did not make it had a larger LV E/E' ratio than those who did.

Direct compression of the pulmonary arteries, dysfunction of the pulmonary endothelial cells, remodeling of the pulmonary vascular system, micro thrombi, mechanical ventilation strategies (PEEP and transpulmonary pressures), and metabolic disturbances (hypercapnia, acidosis, etc.) are the main causes of RV dysfunction in patients with ARDS who have normal baseline cardiac function. (12, 13, 14).

($r = 0.801$, $P < 0.001$) and LVEF ($r = 0.767$, $P <0.001$) were positively correlated with PaO2/FiO2 in a substantial way. The results showed that there were strong negative relationships with TR maximal PG, RAP, PASP, and LV E/E'.

Improved right and ventricular functions are linked to enhanced oxygenation status, as shown by the positive relationships with and LVEF. LVEF, which indicates ventricular systolic function, and , which measures right ventricular function (which reflects the ventricle's longitudinal contraction and evaluates the RV's base-to-apex shortening), both reflect the heart's ability to adapt to the pulmonary vascular changes in ARDS. provides additional evidence that improved oxygenation is associated with intact or mildly compromised cardiac performance, which may be attributable to less pulmonary vascular resistance and more efficient pulmonary gas exchange.

In a similar vein, Shah et al. discovered that was significantly associated with an elevated risk of death within 30 days after TTE ($P = 0.004$). Further, the Tei index strongly correlated negatively ($r = −0.46$, $P = 0.018$) with PaO2:FiO2 ratios, whereas showed a positive connection ($r = 0.52$, $P = 0.003$) with these ratios [10].

In addition, the study by Tamborini et al. highlighted a positive correlation between (20.2+/-5 versus 24.7+/-4 mm, $p<0.01$) and ventricular ejection fraction, as well as a negative correlation with pulmonary pressure [15].

As a predictor of mortality in patients with respiratory distress syndrome (ARDS) and pulmonary hypertension (PHTN), the receiver operating characteristic (ROC) analysis of the tricuspid regurgitation maximum pressure gradient (TR max PG) had a strong predictive value of 0.838. A statistical significance was demonstrated with a p-value less than 0.001, and the reliability of the marker was confirmed with a confidence interval spanning from 0.728 to 0.948. To accurately identify persons without the result, the optimal cutoff value for TR max PG was determined to be more than 53 mmHg, which had a sensitivity of 71.4% and a specificity of 100%. Patients who did not pass away throughout the study period were correctly classified as at risk, indicating that a TR max PG > 53 mmHg is a very precise determinant of mortality in this population.

With an under the curve (AUC) of 0.917, the ROC analysis for Tricuspid Annular Plane Systolic Excursion () showed a strong predictive accuracy for among ARDS patients with pulmonary hypertension (PHT). number, backed by a 95% confidence range of 0.843 to 0.991 and a p-value of less than 0.001, shows that is a strong predictor of death. There was a perfect match between the 1.5 cm or smaller cutoff value and a sensitivity of 74.3 percent (meaning it accurately identified around 74.3 percent of the patients who passed away) and a specificity of 100 percent (meaning it accurately identified every patient who made it). Based on these findings, it seems that an of 1.5 cm or less is a very precise indicator of a higher risk of in group of patients, successfully detecting all instances that do not end in death avoiding false positives. Echocardiographic data were in multivariate logistic regression studies to predict in ARDS patients with PHTN, adjusting for gender and age. An increased risk of death of about 14% was related with a one unit rise in TR maximal PG (95% CI = 1.047 - 1.239, $P = 0.003$). Two studies found that there was an increased risk of death for every one unit rise in RAP (95% CI = 1.435 0 3.953, $P = 0.001$). The risk of death rose by 31% for every unit rise in PASP (95% CI = 1.105 - 1.553, $P = 0.002$). There was a 77.5% decrease in risk for every one unit rise in (95% CI = 0.1 - 0.648, $P = 0.004$). The risk of death was 37.2% lower for every one unit increase in LVEF (OR = 0.628, 95% COI = 0.429 - 0.918, $P = 0.016$), suggesting that LVEF is a strong protective factor. On the other hand, there was a striking difference in the Ventricular E/E' ratio (LV E/E'). The risk of death was ten times higher for every unit increase in ratio (OR = 10.93, 95% CI = 2.117 - 56.424, $P = 0.004$), suggesting that it might be a powerful predictor of a bad prognosis.

The results of Shah et al. corroborate our own; when they multivariate regression models to further examine the connection between and mortality, they found that independently maintained statistically significant association [10].

Conclusions:
The research shown that in critically ill patients hospitalized to the intensive care unit (ICU) due to respiratory distress syndrome (ARDS), echocardiographic evaluation of pulmonary hypertension (PH) is an essential determinant of 30-day mortality. Indicators of ventricular dysfunction, pulmonary artery systolic pressure, right atrial pressure, tricuspid regurgitation pressure gradient, and other important echocardiographic measures were substantially linked to increased death rates. Echocardiography is a vital tool for directing clinical therapy and improving patient prognostication in the ICU environment, and discovery is in line with the study's goal of evaluating the prognostic importance of pulmonary hypertension in ARDS patients.

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