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ORIGINAL ARTICLE

Right Ventricular Function and Pulmonary Hypertension in Children with Pneumonia and Respiratory Failure in Pediatric Intensive Care Unit

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ABSTRACT

Background: Understanding the relationships among the right ventricular (RV) function, pulmonary hypertension (PH), pneumonia, and respiratory failure is highly important in the management of these patients in the Pediatric Intensive Care Unit (PICU). We aimed to investigate the associations between RV dysfunction and/or PH in addition to lower respiratory tract infection and respiratory failure.

Methods: We conducted this cross-sectional study on 56 patients who had lower respiratory tract infections as well as respiratory failure and who were admitted to the PICU. Routine laboratory investigations and chest X-rays were performed on all patients. The Pediatric Risk of Mortality (PRISM) score was assessed in addition to conventional echocardiographic evaluation for the assessment of RV systolic function and PH.

Results: An increasing PRISM-III score was significantly independently correlated with an increased risk of PH by 3.459-fold. Significant relationships were revealed between the presence of PH and Fractional Area Change (FAC), Tricuspid Annular Plane Systolic Excursion (TAPSE), Tricuspid Regurgitation (TR) velocity, Peak Atrial A Wave Velocity (PAAT), Tissue Doppler Imaging of the Aortic Valve (TAS), changes in Inferior Vena Cava (IVC) diameter, respiration, and the Early Mitral Inflow Velocity (E) to Late Mitral Inflow Velocity (A) (E/A) ratio. The TAPSE, FAC, PAAT, TAS velocity, and E/A ratio were significantly greater among patients without PH. Pulmonary artery systolic pressure (PASP) with a cut-off point ≥46 could predict mortality with an AUC of 0.956, specificity of 92.3%, sensitivity of 94.1%, positive predictive value of 84.2%, negative predictive value of 97.3%, and overall accuracy of 92.9%. A significant positive association was found between the PASP score and the length of the PICU stay.

Conclusions: High rates of PH leading to respiratory insufficiency among children with lower respiratory tract infections affect a number of clinical outcomes, including the mortality, PICU stay length, and duration of mechanical ventilation.

Keywords: Right ventricular function; Pulmonary hypertension; Pneumonia; Respiratory failure; Pediatric intensive care unit.

INTRODUCTION

Respiratory failure in children occurs when the amount of gas exchanged between the blood and the atmosphere is inadequate to meet the metabolic demands of the body. It is identified when the patient's respiratory system stops delivering enough oxygen to the blood, leading to hypoxemia, or when the patient cannot breathe well enough, causing hypercarbia as well as hypoxemia [1].

Morbidity and mortality in pediatric acute respiratory distress syndrome (PARDS) are mostly associated with the development of a multiple organ dysfunction syndrome (MODS), which is defined by acute hypoxemic respiratory failure [2].

One of the main causes of death in acute respiratory distress syndrome (ARDS) patients is right ventricular dysfunction (RVD), and the severity of RVD is linked to a greater risk of death. Pulmonary ventilation–perfusion mismatches exacerbate oxygenation, which can lead to non-pulmonary organ failure in cases of acute right ventricular (RV) systolic dysfunction as well as pulmonary hypertension (PH) [3].

There has been long-standing evidence in the medical literature of PH in individuals with acute respiratory diseases. Researchers have found evidence of a link between pulmonary arterial hypertension and respiratory illnesses such as pneumonia and acute bronchiolitis. However, these findings are based on very limited research [4].

Within pediatric intensive care units (PICUs), echocardiography has the ability to identify PH, quantify RV systolic function, and elucidate its impact on prognosis and outcomes [5]. Therefore, we aimed to investigate the associations between RVD and/or PH in addition to lower respiratory tract infection and respiratory failure.

METHODS

We carried out this cross-sectional study on 56 patients who had lower respiratory tract infections and respiratory failure and were admitted to the PICU of the Pediatric Department, Zagazig University Hospital, from June 2022 to January 2024. Informed consent was obtained from all parents. Approval for performing the study was obtained from the Institutional Review Board (No. #5384). The research followed the guidelines laid out in the Declaration of Helsinki, which is part of the World Medical Association's Code of Ethics for Research Involving Humans.

Inclusion criteria

All patients who were aged from 1 month to 14 years and had pneumonia with respiratory failure were included.

Exclusion criteria

Patients with pneumonia or respiratory failure due to cardiac causes and chronic lung disease, diagnosed and confirmed by echocardiography and chest X-ray with history assessment, were excluded.

History and clinical evaluation

All patients were subjected to detailed history taking, including personal history (age, sex, socioeconomic details), past medical history, and history of the present illness, including its duration. Symptoms suggestive of PH were searched for, including cough, dyspnea, recurrent chest infection, hemoptysis, and chest wheezes.

Anthropometric measurements, such as weight, height, and head circumference, were measured. The body mass index (BMI) was calculated as the ratio of weight (kg) to squared height (m^2).

Chest examination was performed, and the breathing difficulty was graded as follows: grade 1 (tachypnea more than 60 breaths per minute and explosive nasal flaring), grade 2 (retraction of the subcostal and intercostal spaces), grade 3 (grunting), and grade 4 (cyanosis). Also, a full cardiac evaluation was done.

Laboratory investigations

Laboratory investigations included complete blood count (CBC), C-reactive protein (CRP), liver function tests, kidney function tests, arterial blood gas (ABG), and electrolytes.

Radiological evaluation

Chest X-ray was routinely performed on admission to detect the cause of illness and to detect any chest disease, lung opacity, hyperinflation, or pericardial effusion.

Assessment of the Pediatric Risk of Mortality (PRISM) score

We utilized the Pediatric Risk of Mortality (PRISM) score, which includes 14 variables (medical and physiological data), with the maximum severity value from the initial 24 hours applied to each. It is a prognostic score utilized in many PICUs to determine the severity of disease, and it has outstanding discriminatory performance and prediction [6].

Conventional echocardiographic evaluation

Sedation with an aqueous solution of chloral hydrate (50 mg per kilogram) was administered to children who became agitated 15 minutes before the research began. The entire research was ECG gated, and standard 2D echocardiography was performed using a GE Vivid E95 machine. An experienced pediatric cardiologist performed an echocardiogram with the patient lying flat, following the standards established by the American Society of Echocardiography. The echo showed the heart valves in all of their normal configurations with M-mode, 2D, TDI, pulsed, and continuous Doppler flow [7]. The following measurements were focused on and recorded:

Assessment of left ventricular systolic function in M mode: Using the M-Mode modality on the left ventricle (LV) in a short-axis view at the level of the papillary muscle, we measured the following parameters: left ventricular end-diastolic diameter (EDD), left ventricular end-systolic diameter (ESD), left ventricular ejection fraction (EF), and left

ventricular functional shortening (FS).

Measurements of the left ventricle's end-diastolic and end-systolic volumes were used to determine the EF as follows:

$$EF = \frac{EDV - ESV}{EDV}$$

Assessment of RV systolic function: To measure the RV systolic function, the tricuspid annular plane systolic excursion (TAPSE), tricuspid annular systolic velocity, fractional area change (FAC), and qualitative assessment (normal, mildly diminished, substantially diminished, or severely diminished) were performed. The TAPSE is an abnormal TAPSE that is more than two standard deviations lower than the stated normal value for age in pediatric patients [8].

To determine the RV fractional area change (RV-FAC), a four-chamber view was used to follow the endocardium of the RV during the relaxation and contraction phases, beginning at the annulus and terminating at the annulus again. The FAC was determined by dividing the difference between the end-diastolic and end-systolic areas by 100 and then dividing that result by the end-diastolic area. A FAC level less than 35% was considered abnormal [9].

Assessment of PH: The PH was assessed from the tricuspid regurgitant velocity, pulmonary artery acceleration time (PAAT), and ventricular septum position. We used a qualitative method to evaluate RV dilation. One way to assess TAPSE is by taking photos of the displacement of the tricuspid valve from the top four chambers and then using M-mode interrogation to measure it [10].

Pulmonary artery systolic pressure (PASP) was deemed high and defined as PH when it was more than 35 mm Hg; a mild case was defined as a PASP less than 45 mm Hg; a moderate case was defined as a value between 45 and 70 mm Hg; and a severe case was defined as a value greater than 70 mm Hg [11].

Patients were categorized into two groups according to the presence of PH, and their clinical and laboratory characteristics, primary outcomes (needs for mechanical ventilation and duration, length of hospital stay), and secondary outcomes (mortality) were compared.

Statistical analysis

Data were analyzed using SPSS version 18 (*USA*). Data were presented as mean ± standard deviation (SD), median, interquartile range (IQR), and range for quantitative variables or numbers and percentages (%) for categorical variables. The following statistical tests were used: the Chi-square test and Fisher's exact test for categorical variables, the Student's t-test for normally distributed numerical variables, and the Mann–Whitney U test for non-normally distributed variables. A p-value less than 0.05 was considered statistically significant, and a p-value less than 0.001 was considered statistically highly significant.

RESULTS

The median age of the patients was 1 month, with a range from 1 month to 13 years. 58.9% were females, and 51.8% needed mechanical ventilation (MV). Approximately 33.9% had no PH, while 28.6%, 35.7%, and 1.8% had PH grades 1, 2, and 3, respectively (Table 1).

A significantly greater platelet count was found among patients with PH (P=0.002), whereas potassium was significantly lower (P=0.022). Additionally, the TAPSE, FAC, PAAT, TAS velocity, and E/A ratio were significantly greater among patients without PH (P<0.05), while the TR velocity was significantly greater among patients with PH (P<0.0001). Also, there was a statistically significant difference between patients with PH and those without PH regarding the change in IVC diameter with respiration (P=0.002). Moreover, the PRISM III score was significantly greater in patients with PH (P<0.001) (Table 2).

A significant relationship was revealed between the presence of PH and mortality (P<0.001). Additionally, the duration of MV was significantly greater in those with PH (P<0.001) (Table 3).

Increasing the PRISM-III score significantly independently correlated the risk of PH by 3.459-fold (Table 4).

A significant relationship was found between the presence of PH and overall survival (P=0.014). No patients without PH died (Table 5).

PASP with a cut-off value ≥46 could predict mortality with an area under the curve (AUC) of 0.956, sensitivity of 94.1%, specificity of 92.3%, positive predictive value (PPV) of 84.2%, negative predictive value (NPV) of 97.3%, and overall accuracy of 92.9% (P <0.001). RV-FAC with a cut-off point ≤21 could predict mortality with an AUC of 0.763, sensitivity of 82.4%, specificity of 76.9%, PPV of 60.9%, NPV of 90.9%, and overall accuracy of 78.6% (P=0.002). TAPSE with a cut-off point ≤14.5 can predict mortality with an AUC of 0.649, sensitivity of 52.9%, specificity of 69.2%, PPV of 42.9%, NPV of 77.1%, and overall accuracy of 64.3% (P=0.079) (Figure 1).

Pulmonary hypertension significantly increased the risk of mortality (P<0.001), and moderate and

severe PH increased the risk of mortality by 48-fold. Also, a PASP value ≥46 mmHg significantly increased the risk of mortality by 192-fold (P<0.001). Moreover, moderate and severe PH significantly increased the risk of needing MV by 18-fold (P<0.001), and a PASP value ≥46 mmHg significantly increased the risk of needing MV by 17.71-fold (P<0.001) (Table 6). Overall survival was significantly greater in patients with PASP <46 mmHg (P=0.004) (Figure 1).

The PASP was significantly positively correlated with both the length of PICU stay and MV duration (P=0.011 and <0.001, respectively). The TAPSE was significantly negatively correlated with the MV duration (P=0.007), as were the RV-FAC and MV duration (P=0.032) (Supplementary Table 1).

Table (1): Demographic data, outcomes, and echocardiographic measurements of the studied patients

Variables	**Total patients (N=56)**	
Age (in years) • **Median (IQR)** • **Range**	 1 month (0.69 – 6 years) 1 month – 13 years	
Gender • **Male** • **Female**	 23 (41.1%) 33 (58.9%)	
PRISM III • **Median (IQR)** • **Range**	 6.45 (6 – 14) 5 – 21	
Need for MV • **Yes** • **No**	 29 (51.8%) 27 (48.2%)	
MV duration (in days) • **Median (IQR)** • **Range**	 7 (5 – 8) 3 – 18	
LOS in the PICU (in days) • **Median (IQR)** • **Range**	 10 (7 – 12) 4 – 25	
Mortality • **No** • **Yes**	 39 (69.6%) 17 (30.4%)	
Echocardiographic measurements	**Mean ± SD**	**Range**
LVEDD (mm)	29.95 ± 9.08	18 – 50
LVESD (mm)	17.27 ± 4.66	10 – 31
EF (%)	68.46 ± 6.0	46 – 79
FS (%)	38.3 ± 4.9	22 – 48
TAPSE (mm)	15.14 ± 3.38	9 – 20
FAC (%)	27.39 ± 9.38	15 – 40
TAS velocity (cm/s)	11.96 ± 2.63	7 – 16
PAAT (cm/s)	103.64 ± 15.81	60 – 130
TR Velocity (cm/s)	2.76 ± 0.54	2.8 – 3.9
IVC Diameter (mm)	10.7 ± 2.7	6 – 16
IVC Change with respiration	**n**	**%**
• **<50%**	13	23.2%
• **>50%**	43	76.8%
RV Diameter (mm)	18.73 ± 3.87	10 – 26
PASP (mmHg)	41.1 ± 12.8	20 – 70

Variables	Total patients (N=56)	Variables
Grades of pulmonary hypertension	**n**	**%**
• **Absent**	19	33.9%
• **Mild**	16	28.6%
• **Moderate**	20	35.7%
• **Severe**	1	1.8%

cm/s, centimeters per second; EF, Ejection fraction; FAC, Fractional area change; FS, Fractional shortening; IQR, Interquartile range; IVC, Inferior vena cava; LOS in the PICU, length of stay in the pediatric intensive care unit; LVEDD, Left Ventricular End-Diastolic Diameter; LVESD, Left Ventricular End-Systolic Diameter; mm, millimeters; MV, Mechanical ventilation; PAAT, Peak atrial A wave velocity; PASP, Pulmonary artery systolic pressure; PRISM III, Pediatric risk of mortality; RV, Right ventricle; TAPSE, Tricuspid annular plane systolic excursion; TAS, Tissue Doppler Imaging of the Aortic Valve; TR, Tricuspid regurgitation

Table (2): The relationship of pulmonary hypertension with laboratory data, echocardiographic measurements, and PRISM III scores

Variables	No pulmonary hypertension (n=19)	Pulmonary hypertension (n=37)	t	*P*-value
	Mean ± SD	**Mean ± SD**		
• **Laboratory data**				
Glucose (mg/dL)	100.79 ± 9.46	105.35 ± 9.46	-1.571	0.122
Potassium (mEq/L)	4.34 ± 0.6	4.09 ± 0.47	2.353	0.022*
Creatinine (mg/dL)	1.15 ± 0.34	1.1 ± 0.32	0.481	0.633
Urea (mg/dL)	42.0 ± 7.39	39.73 ± 8.7	0.971	0.336
WBCs (cells/mm3)	14.768± 3.75	13.78 ± 3.53	0.982	0.331
Platelets (cells/mm3)	178.84 ± 28.84	208.32 ± 34.72	-3.178	0.002*
PT (seconds)	12.38 ± 0.72	12.22 ± 0.69	0.822	0.415
PTT (seconds)	29.89 ± 3.35	29.84 ± 2.52	0.065	0.948
• **Echocardiographic measurements**				
LVEDD (mm)	28.84 ± 8.53	30.51 ± 9.41	-0.649	0.519
LVESD (mm)	16.11 ± 3.91	17.86 ± 4.94	-1.349	0.183
EF (%)	70.26 ± 5.65	67.54 ± 4.23	1.631	0.109
FS (%)	40.0 ± 4.23	37.854± 5.07	1.812	0.076
TAPSE (mm)	17.95 ± 2.48	13.7 ± 2.84	5.521	<0.001**
FAC (%)	34.89 ± 7.01	23.54 ± 8.05	5.212	<0.001**
TAS velocity (cm/s)	14.37 ± 1.34	10.73 ± 2.24	6.48	<0.001**
PAAT (cm/s)	118.58 ± 10.17	95.97 ± 12.33	6.872	<0.001**
TR Velocity (cm/s)	2.14 ± 0.2	3.07 ± 0.36	-12.404	<0.0001**
IVC diameter (mm)	10.89 ± 2.75	10.73 ± 2.74	0.213	0.832
IVC Change with respiration ▪ **<50%** ▪ **>50%**	 0 (0%) 19 (100%)	 13 (35.1%) 24 (64.9%)	 F	 0.002*
E/A ratio	1.31 ± 0.15	1.18 ± 0.23	2.134	0.037*
RV Diameter (mm)	17.68 ± 3.59	19.27 ± 3.95	-1.466	0.148
• **PRISM III**				
Median (IQR)	6 (5.2 – 6.3)	7 (6.1 – 18.2)	Z= -4.03	<0.001**

t: Student's t-test; F: Fisher's exact test; Z: Mann-Whitney U test
** Statistically significant: P-value < 0.05*
*** Statistically highly significant: P-value < 0.001*
cm/s, centimeters per second; E/A ratio, Ratio of Early Mitral Inflow Velocity (E) to Late Mitral Inflow Velocity (A); EF, Ejection fraction; FAC, Fractional area change; FS, Fractional shortening; IQR, Interquartile range; IVC, Inferior vena cava; LVEDD, Left Ventricular End-Diastolic Diameter; LVESD, Left Ventricular End-Systolic Diameter; mm, millimeters; PAAT, Peak atrial A wave velocity; PRISM III, Pediatric risk of mortality; PT, Prothrombin time; PTT, Partial thromboplastin time; RV, Right ventricle; TAPSE, Tricuspid annular plane systolic excursion; TAS, Tissue Doppler Imaging of the Aortic Valve; TR, Tricuspid regurgitation; WBCs, White blood cell count

Table (3): The relationship between the presence of pulmonary hypertension and in-hospital outcomes

Variables	No pulmonary hypertension (N=19)	Pulmonary hypertension (N=37)	χ2	*P*-value
	N (%)	N (%)		
Need for MV • **No** • **Yes**	 12 (63.2%) 7 (36.8%)	 15 (40.5%) 22 (59.5%)	2.572	0.109
Mortality • **Non-survivors** • **Survivors**	 0 (0%) 19 (100%)	 17 (45.9%) 20 (54.1%)	12.535	<0.001*
	Median (IQR)	**Median (IQR)**	**Z**	***P*-value**
MV duration (in days)	4 (3 – 5)	7 (6 – 9.25)	-3.213	<0.001*
ICU duration (in days)	9 (5 – 12)	11 (7 – 12.5)	1.113	0.266

χ2: Chi-square test; Z: Mann-Whitney U test
** Statistically highly significant: P-value < 0.001*
MV, Mechanical ventilation; ICU, Intensive care unit; IQR, Interquartile range

Table (4): Multivariate regression analysis of factors associated with pulmonary hypertension

	β	*P*-value	AOR	95% CI	
				Lower	Upper
PRISM-III score	1.241	0.035*	3.459	1.093	10.951

PRISM III, Pediatric Risk of Mortality; AOR, adjusted odds ratio; CI, Confidence interval
** Statistically significant: P-value < 0.05*

Table (5): Survival analysis of patients according to the presence and severity of pulmonary hypertension

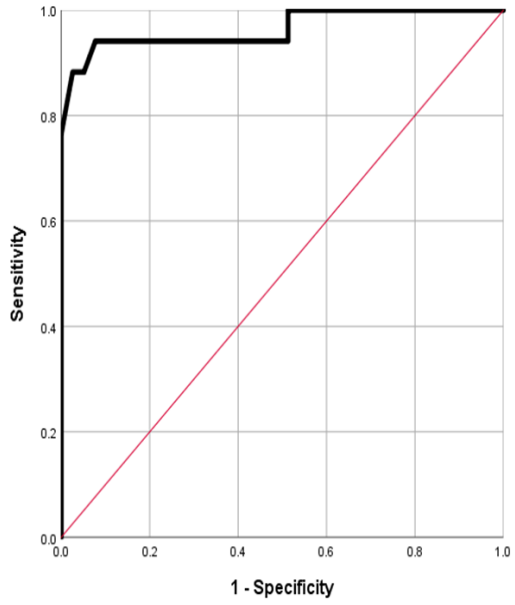
			Total	No. of Events	*P*-value
No pulmonary hypertension			19	0 (0%)	0.014*
Pulmonary hypertension			37	17 (45.9%)	
Overall			56	17 (30.4%)	
Grades of PH	**Total**	**No. of Events**	**Mean ± SE**	**95% CI**	**P-value**
Mild	16	1 (6.2%)	14.0 ± 0	14 – 14	0.288
Moderate & severe	21	16 (76.2%)	14.35 ± 1.21	11.97 – 16.72	
Overall	37	17 (45.9%)	14.69 ± 1.12	12.49 – 16.9	

** Statistically significant: P-value < 0.05*
PH, Pulmonary hypertension

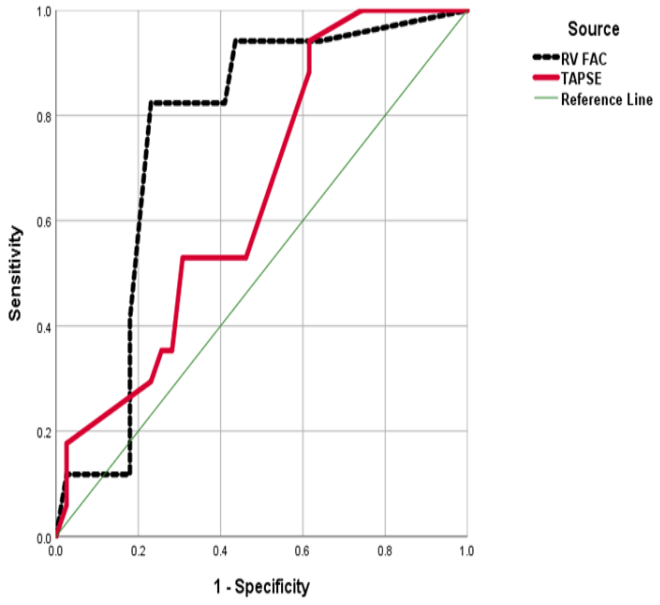
Table (6): The relationship between mortality and presence, grades of pulmonary hypertension, and suggested cut-off value of PASP

	Non-survivors (n=17)	Survivors (n=39)	*P*-value	COR (95% CI)
	N (%)	N (%)		
PH				
• **Absent**	0 (0%)	19 (48.7%)	<0.001**	∞
• **Present**	17 (100%)	20 (51.3%)		
Grades of PH	**(n=17)**	**(n=20)**		
• **Mild**	1 (5.9%)	15 (75%)	<0.001**	48 (5.01 – 459.84)*
• **Moderate/severe**	16 (94.1%)	5 (25%)		
PASP (mmHg)				
• **<46 mmHg**	1 (5.9%)	36 (92.3%)	<0.001**	192 (18.52 – 990.36)*
• **≥46 mmHg**	16 (94.1%)	3 (7.7%)		
	Need MV (n=29)	**No need (n=27)**		
PH				
• **Absent**	7 (24.1%)	12 (44.4%)	0.109	2.51 (0.8 – 7.86)
• **Present**	22 (75.9%)	15 (55.6%)		
Grades of PH	**(n=22)**	**(n=15)**		
• **Mild**	4 (18.2%)	12 (80%)	<0.001**	18 (3.4 – 95.21)*
• **Moderate/severe**	18 (81.8%)	3 (20%)		
PASP (mmHg)				
• **<46 mmHg**	12 (41.4%)	25 (92.6%)	<0.001**	17.71 (3.51 – 89.38)*
• **≥46 mmHg**	17 (58.6%)	2 (7.4%)		

P: for Chi-square test; COR: crude odds ratio; CI: confidence interval
*** Statistically highly significant: P-value < 0.001*
MN, Mechanical ventilation; PASP, Pulmonary Artery Systolic Pressure; PH, Pulmonary hypertension



(A)



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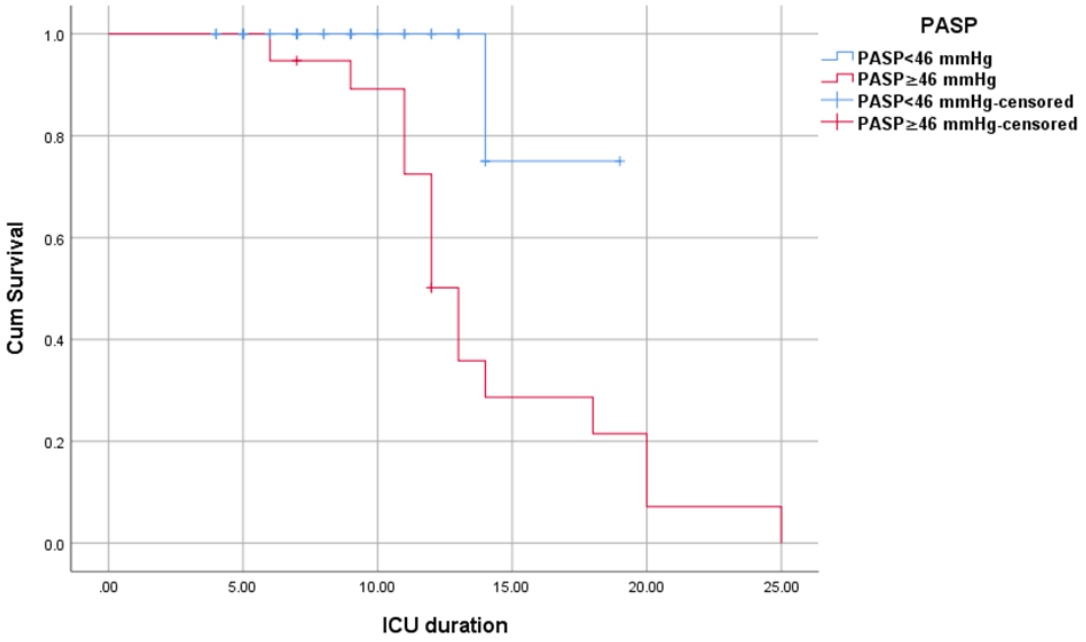

(C)

Figure 1: (A) ROC curve showing the performance of the PASP in the prediction of mortality. (B) ROC curve showing the performance of the RV-FAC and TAPSE in the prediction of mortality. (C) Kaplan–Meier plot showing the relationship between the cut-off of PASP and overall survival. *PASP (mmHg), Pulmonary Artery Systolic Pressure; RV-FAC, Right Ventricular Fractional Area Change; TAPSE, Tricuspid Annular Plane Systolic Excursion.*

DISCUSSION

Critically ill children face significant challenges for PICU staff when dealing with pneumonia and respiratory failure. When breathing becomes inadequate, a condition known as respiratory failure occurs, and artificial ventilation may be needed. In contrast, pneumonia is a prevalent respiratory illness that causes lung parenchyma inflammation and consolidation [12].

Pneumonia accounts for 19% of deaths in children less than five years old in Egypt, a country where children under the age of five make up almost 13.4% of the overall population. According to estimates, there are between 0.11 and 0.20 cases of pneumonia per 1,000 children in Egypt each year [13].

While the primary focus in these cases is often on the pulmonary pathology itself, it is crucial to recognize the potential impact of these conditions on right ventricular (RV) function and the development of pulmonary hypertension (PH). The right ventricle plays a vital role in pumping blood into the pulmonary circulation, and any impairment in its function can have severe consequences on overall hemodynamics [14].

One of the most popular and helpful non-invasive imaging methods for diagnosing PH is echocardiography. Particularly relevant to PH are the cardiac structures and functions described in great detail, particularly the right ventricle and pulmonary arteries [15].

Understanding the relationships among RV function, PH, pneumonia, and respiratory failure is highly important in the management of patients in the PICU. Early recognition of RV dysfunction and PH can aid in risk stratification, guiding therapeutic interventions, and improving outcomes [9].

In the present study, approximately 66.1% of patients had PH; 28.6%, 35.7%, and 1.8% had grades 1, 2, and 3, respectively. A significant relationship was revealed between the presence of PH and mortality (P<0.001).

These results are compatible with those of Guzmán et al. [16], who reported that overall mortality was greater among patients with moderate to severe PH who were admitted to the PICU than among individuals without PH. Approximately 82% of juvenile acute respiratory distress syndrome (ARDS) patients have PH, which is related to worse outcomes on its own.

This was in accordance with the findings of Maria et al. [17], who reported that PH was present in 89.9% of cases. A total of 7.5% of the population died. An optimum threshold value of 54 mm Hg was determined via the receiver operating characteristic (ROC) curve evaluation for differentiating between patients with and without mortality on the basis of pulmonary pressure. Among patients whose pulmonary pressure was greater than 54 mmHg, the mortality rate was 30.0%, whereas it was only 0.7% for patients whose pressure was lower than this threshold. Pulmonary vascular insufficiency was found to be an independent risk factor for greater 60-day mortality among patients who had ARDS, according to Bull et al. [18].

The current study did not find a statistically significant correlation between PH and PO2, or PCO2. Patients with PH had a greater mean PaCo2 value than did those without PH, as shown by the unlikely correlation between the two variables identified by Guzmán et al. [16] and pulmonary pressure. Since hypercapnia during mechanical ventilation might be caused by vascular collapse or excess alveolar distension, it is generally thought to be harmless or even advantageous.

Our findings clearly revealed a significant relationship between the presence of PH and platelet count (significantly greater in those with PH, with a p value of 0.002) and potassium (significantly lower in those with PH, with a p value of 0.022).

Mondéjar-Parreño et al. [19] reported that hypokalaemia (lower potassium levels) in children with PH may involve various underlying mechanisms. It is possible that potassium levels are influenced by the hemodynamic changes and alterations in renal function associated with PH. However, the specific reasons for lower potassium levels in this context require further investigation. Importantly, electrolyte imbalances can have significant clinical implications and should be closely monitored and managed in children with PH.

In agreement with our findings, Vrigkou et al. [20] reported that an elevated platelet count (thrombocytosis) in children with PH could indicate a potential association between platelet activation and the pathophysiology of PH. Platelets are involved in thrombosis, vascular remodelling, and inflammation, all of which can amplify the impact of PH. Further examination and diagnostic tests, including echocardiography, may be needed when the PLT alone is insufficient to identify or predict the presence of PH.

The current study revealed that the PRISM III score was significantly higher among patients who had PH ($p<0.001$). Increasing the PRISM-III score significantly independently correlated with the risk of PH by 3.459-fold. In agreement with our findings, Guzmán et al. [16] revealed a statistically significant difference between their two studied groups regarding PRISM since it was greater in the PH group.

The present study revealed significant relationships between the presence of PH and FAC, TAPSE, TR velocity, PAAT, and TAS velocity; IVC diameter; respiration; and the E/A ratio. The TAPSE, FAC, PAAT, TAS velocity, and E/A ratio were significantly greater among patients without PH ($p<0.001$). TR velocity was significantly greater among patients who had PH ($p<0.0001$). Additionally, there was a non-significant relationship between the presence of PH and the LVEDD, LVESD, EF, FS, IVC diameter, or RV diameter.

In line with our findings, Himebauch et al. [9] reported that in the first week after the start of pediatric ARDS, patients have a worse chance of extubation and a higher risk of death in the PICU if they experience new or chronic right ventricular systolic dysfunction. In particular, RV global longitudinal strain and FAC remained mostly unaltered in non-survivors after pediatric ARDS began but showed a linear improvement in survivors over the first week after the disease started.

Similar findings were obtained by Himebauch et al. [21], who reported that children afflicted with pediatric ARDS presented detectable, common, and severity-independent PH and early ventricular failure. There was a correlation between right ventricular dysfunction and PICU mortality, as well as a correlation between right ventricular dysfunction and PH and a decreased likelihood of extubation.

Mekontso Dessap et al. [22] reported that there was a stronger correlation between RV systolic overload and an RV/LV end diastolic area ratio greater than 0.6 in more than 60% of patients with PH and low PEEP, rather than the PEEP level. This discovery suggests that the presence of PH does not necessarily affect ventilator settings. High vascular resistance is often associated with a collapsing lung volume.

Our findings clearly revealed a significant relationship between the presence of PH and overall

survival (p=0.014). No patients without PH died. There was a statistically non-significant relationship between the grade of PH and overall survival.

In the present study, we found that PASP with a cut-off point ≥46 can predict mortality (AUC: 0.956) with a sensitivity of 94.1%, specificity of 92.3%, positive predictive value of 84.2%, negative predictive value of 97.3%, and overall accuracy of 92.9% (p < 0.001). A PASP ≥46 mmHg significantly increased the risk of mortality by 192-fold (p <0.001).

These results were compatible with those of Guzmán et al. [16], who reported that the best pulmonary pressure threshold value for differentiating patients with and without death was 54 mm Hg, according to ROC curve analysis. The ROC curve-based bivariate analysis revealed an increased death rate when the PASP cut-off was 54 mm Hg. In patients who made it through the ordeal, the PASP decreased by an average of 14 mm Hg, but in those who did not, it did not rise or fall.

In the present study, we found a significant relationship between the presence of PH and mechanical ventilation duration, which was greater in those with PH. Moderate and severe PH increased the risk of needing mechanical ventilation by 18-fold. There was a statistically significant relationship between the need for mechanical ventilation and the suggested cut-off of the PASP for PH. A PASP≥46 mmHg increased the risk of needing mechanical ventilation by 17.71-fold.

Similar findings were obtained by Guzmán et al. [16], who reported that, compared with children without PH, those with PH-associated ARDS (PASP values greater than 54 mm Hg) required mechanical ventilation for a considerably longer period of time. The bivariate study revealed that mechanical ventilation was needed for a longer period when the PASP cut-off value was 54 mm Hg on the basis of the ROC curve. According to Bull et al. [18], pulmonary vascular insufficiency increases the demand for cardiovascular support and decreases the number of ventilation-free days in patients with ARDS.

Our findings clearly revealed that there was a significant positive correlation between the PASP score and the length of ICU stay. There was no significant correlation between the TAPSE and ICU duration. There was a significant correlation between the RV-FAC and the duration of the ICU stay. There was a significant relationship between the presence of PH and the duration in the ICU.

This finding was comparable with that of Guzmán et al. [16], who reported that the length of time a patient spent in the PICU was significantly greater for those with moderate to severe PH. Using a cut-off value of 54 mm Hg for the PASP in the bivariate analysis based on the ROC curve, a lengthier PICU stay was observed. Patients with ARDS are more likely to require cardiovascular support and have a longer duration of stay in the ICU if they have pulmonary vascular failure, according to Bull et al. [18].

Limitations of the study

While the study included a reasonable number of PICU patients (56), a larger sample size would provide greater statistical power and precision in the association between right ventricular dysfunction and/or PH with lower respiratory tract infection and respiratory failure. The duration of follow-up may have been insufficient to capture long-term outcomes. There is also a risk of interobserver variability, as multiple doctors have conducted echocardiographic assessments. The accuracy of echocardiography in the PICU can be influenced by patient condition, with factors such as apnea, bradycardia, arterial hypotension, or changes in skin color potentially affecting hemodynamic measurements. This study's single-center design minimizes the risk of random variation and interobserver variability.

CONCLUSION

High rates of pulmonary hypertension leading to respiratory insufficiency among children with lower respiratory tract infections affect a number of clinical outcomes, including mortality, PICU stay length, and duration of mechanical ventilation. When dealing with children in the PICU who have pneumonia and respiratory failure due to pulmonary hypertension, echocardiography evaluation of right ventricular function is essential. By monitoring and optimizing right ventricular function, healthcare providers can improve outcomes and provide targeted interventions to mitigate the impact of pulmonary hypertension in this vulnerable patient population.

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