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Original Research Article

A prospective observational study of prevalence, incidence, and prognostic implications of right-sided heart failure in acute respiratory distress syndrome patients

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ABSTRACT

Background: Acute respiratory distress syndrome (ARDS) is a common reason for respiratory failure in critically unwell patients. Noncardiogenic pulmonary edoema that develops suddenly, acute hypoxemia, and the need for mechanical ventilation are its defining features. This study's major objective was to examine the likelihood of cor-pulmonale in ARDS patients receiving protective ventilation, as well as its prognosis over the long term.
Methods: S. C. B. medical college in Odisha, India served as the setting for this prospective observational study. 100 consecutive patients with moderate to severe ARDS were included in the trial in accordance with the Indian criterion. With an average PEEP of 91 cm H2O and a plateau pressure cap of 31 cm H_2O, these patients were ventilated.
Results: There were 100 patients altogether, 63 males and 37 females, with a mean age of 59-19 years. The interval between the diagnosis of ARDS and TEE was typically 0.75-0.97 days. There were 100 cases of cor pulmonale, which was shown to be common (21.09%). Lung injury occurred more frequently in patients with cor pulmonale compared to other patients when an infectious cause was involved [37 (79.06%) vs. 67 (57.61%)].
Conclusions: The incidence of cor pulmonale in ARDS patients undergoing ventilation with restricted airway pressure is an interesting finding. In our investigation, it showed up as an independent risk factor for 28-day mortality and was linked to sepsis and high driving pressure readings.

Keywords: Right ventricle, Mechanical ventilation, Acute lung injury, Echocardiography

INTRODUCTION

The initial description of acute respiratory distress syndrome (ARDS) was made by Ashbaugh and colleagues in 1967, who described a case series encompassing 12 patients with the condition.[1] Reduced lung compliance, cyanosis that did not improve with oxygen therapy, and diffuse radiographic abnormalities on the chest that suggested infiltrates were all present in these patients. Initially referred to as the adult respiratory distress syndrome, it was later renamed ARDS because it was found to also affect children.[2]

The initial definition lacked precise criteria for systematic patient identification, which led to debates over the syndrome's incidence, natural progression, and associated mortality rates. Pulmonary vascular dysfunction in the context of ARDS can be attributed to various factors. These include hypoxia or the effects of certain mediators like endothelins, thromboxane A2, or leukotrienes, which can lead to pulmonary vasoconstriction.[1] This dysfunction is also exacerbated by intravascular blockage brought on by endothelial enlargement or thromboembolic events and mechanical compression brought on by interstitial edoema. Postmortem studies

have shown that thromboembolic factors are present in about 95% of ARDS cases.[2,3]

A further concern of unhealthy mechanical ventilation is that it might worsen pulmonary vascular dysfunction, which is characterised by high inspiratory pressures and large tidal volumes.[4] It is significant to highlight that, in prior studies carried out before the broad use of lower tidal volume breathing, pulmonary hypertension and right ventricular dysfunction were often observed in ARDS patients (up to 89%) and were connected to worse clinical outcomes.[4,5] This prospective study's major objective was to examine the likelihood of "cor-pulmonale" in "ARDS" patients receiving protective ventilation, as well as its prognosis over the long term.

METHODS

A cross-sectional study was conducted in S. C. B. medical college in Odisha, India comprising of a group of 100 patients from April 2022 to June 2023. The patients who agonized moderate to severe ADRS (respiratory failure or worsening respiratory symptoms or lung collapse were included in the study) underwent transesophageal echocardiography. The exclusion criteria were chronic cor pulmonale, esophageal disease requiring long-term oxygen therapy, significant uncontrolled bleeding, or chronic pulmonary disease requiring home mechanical ventilation. An informed written consent form was provided to the patients and signed by them. The SAPS II score was used to assess the severity of the sickness at admission while evaluating the underlying disorders.[6] The requirement for catecholamine infusion to maintain appropriate arterial pressure was used to define shock. As previously mentioned, sepsis, aspiration, and pneumonia were all identified. The study required at least 28 days of follow-up after hospital discharge.

Echocardiography

TEE was carried out in accordance with standard procedure 1726 by trained operators (competence in advanced critical care echocardiography). We quickly looked at the following echocardiogram pictures: In order to get a short-axis view of the LV, a transgastric method is employed. The interventricular septum's dynamics are then evaluated using this image. Calculate the left ventricular ejection fraction and right ventricle/left ventricle (RV/LV) area ratio using a four-chamber long-axis image. To evaluate the superior vena cava's (SVC) collapsibility, a long-axis M-mode picture of the SVC is displayed. LV systolic dysfunction was determined by a left ventricle ejection fraction of less than 50%.

A unique end-systole septal dyskinesia was sought after when slow-motion material was analysed. An aerated modified fluid gelatin solution was injected into the fossa ovalis in order to provide a longitudinal view and assess patent foramen ovale shunting. Two senior researchers with substantial expertise (FB, AMD) used a computer to analyse the echocardiographic pictures offline. In the event of a tie or doubt, a 3rd reading was provided by a blinded expert (AVB) (n=97; overall agreement of 87%).

Whenever feasible, the pulmonary artery systolic pressure (PASP) was measured during transthoracic echocardiography using the tricuspid regurgitation continuous wave "Doppler technique". No values actually observed throughout the trial exceeded the PASP value (20 mmHg), which was attributed to imperceptible tricuspid regurgitation. Cor pulmonale patients did not get rescue therapies or respiratory and hemodynamic therapy in an effort to treat or relieve the disease. Compared to pulmonary hypertension or an isolated RV dilatation, cor pulmonale may demonstrate a more dramatic shift in RV afterload. Three groups were made depending on the severity of pulmonary circulatory system dysfunction to further classify the patients: no dysfunction (PASP B40 mmHg with normal RV size and normal interventricular septum dynamics), moderate dysfunction (PASP), and severe dysfunction (PASP).

Respiratory settings

Volume support and a "target tidal volume (VT)" of 6.0 mL/kg (body weight) were being used to regulate ventilation. At the attending physician's discretion, rescue measures like prone posture and/or inhaled nitric oxide were performed PEEP should be as high as possible while maintaining a maximal inspiratory plateau pressure of 30 cmH_2O in patients with persistent severe hypoxemia (PaO_2/FiO_2 100 mmHg). VT may decrease up till Pplat surpassed the upper bound. Since plateau pressure was less than 30 cmH_2O, the respiratory rate was increased to its highest level without increasing in response to the influence of VT reduction on alveolar ventilation. While inducing intrinsic PEEP, there is no dysfunction (PASP [37.9 mmHg] or a dilated RV without cor pulmonale), and there is modest dysfunction (PASP [37.9 mmHg]).

Statistical analysis

The data were analysed using the statistical programme SPSS Base 13.0 (SPSS Inc, Chicago, IL, USA). The "Student t test" or "Mann-Whitney test" for independent samples was used to compare continuous data, unless otherwise noted, which were provided as mean standard deviation. Categorical variables that were reported as percentages were analysed using the chi-square test or the Fisher exact test. In order to examine survival data and calculate survival probability, standard Kaplan-Meier procedures were employed. Significant univariate risk factors were examined using backward stepwise logistic regression analysis to determine the independent variables affecting cor pulmonale or 28-day mortality. With 100 events recorded, we predicted that a maximum of five variables and nine variables (in the cor pulmonale model) would be needed to prevent overfitting.

RESULTS

Patient characteristics

100 patients in total, 63 males and 37 females, with an average age of 59±19 years, were included. Between the diagnosis of ARDS and TEE, there was an average delay of 0.75-0.97 days. It was determined that 100 people had cor pulmonale, which was common (21.09%; 93.7% CI, 15.1-27.07%). Groups with and without cor pulmonale had similar ages, SAPS II scores, and McCabe and Jackson classes. In patients with cor pulmonale compared to other patients, injury in lung was more frequently caused by an infectious aetiology (non-pulmonary sepsis or pneumonia) [37 (79.60%) vs. 67 (57.60%), p<0.01]. various groups had various respiratory configurations on TEE-day; cor pulmonale group had higher total values and had lower respiratory system compliance scores.

By using multivariate logistic regression analysis, it was discovered that higher driving pressure values of 1791 and viral sources of lung injury were independent variables associated with cor pulmonale (Table 2).

Echocardiographic and hemodynamic findings

Patients with cor pulmonale had lower mean arterial and systolic pressures, quicker heart rates, and higher shock rates at the time of TEE. As expected, the cor pulmonale group had bigger right ventricle dimensions and higher PASP values. Cor pulmonale patients also showed more atrial septal bowing towards the left atrium and more frequent shunting over the patent foramen ovale than other patients. In terms of cardiac index, left ventricle systolic dysfunction, or SVC collapsibility, there was no clear distinction between the two groups.

Table 1: ARDS individuals' clinical features and prognosis, whether they have cor-pulmonale/they don't.

Variables	Cor pulmonale, (n=77)	Non-cor pulmonale, (n=23)	P value
Sex			
Male	48	15	0.47
Female	29	8	0.91
Age (In years)	59.0±19.0	61.0±17.0	0.43
Cause of lung injury, (n, %)			
Non-pulmonary sepsis	21 (26.58)	11(52.38)	
Pneumonia	5 (6.32)	3 (14.28)	
Aspiration	17 (21.51)	2 (9.52)	
Other causes	36 (45.56)	5 (23.80)	
Arterial blood gases on TEE-day			
PaO_2 (mmHg)	27±0.3	7±3	0.33
PaO_2/FiO_2 ratio (mmHg)	13±7	9±0.1	0.57
$PaCO_2$ (mmHg)	29±3	5±1.3	<0.01
pH	7.13±0.9	7.21±0.13	<0.01
Respiratory settings at the time of TEE			
Tidal volume (ml/kg)	6.11±1.0	6.09±1.3	0.047
PEEP (cmH_2O)	9±1	7±5	<0.01
Respiratory rate (bpm)	27±3	21±1	0.03
Plateau pressure (cmH_2O)	31±15	27±11	0.02

Table 2: Risk factors for cor pulmonale in ARDS patients.

Variables	Univariate logistic regression	Multivariate regression
Infection as cause of lung injury	3.78 (1.33-5.09), p<0.01	2.39 (1.07- 5.21), p=0.03
Respiratory settings at the time of TEE		
Tidal volume (mL/kg)	0.73 (0.53- 1.00), p=0.05	NI
PEEP (cmH_2O)	1.13 (1.21-1.25), p=0.01	NI
Respiratory rate (bpm)	0.91 (0.97-1.13), p=0.03	NI
Plateau pressure (cmH_2O)	0.93 (0.87-0.95), p=0.04	NI
Arterial blood gases on TEE-day		
pH (0.1 per unit)	0.73 (0.51-0.93), p=0.01	I/NR
$PaCO_2$ (mmHg)	1.01 (01.09-01.03), p=0.03	NI

DISCUSSION

Our study's key finding was that 79% of the 100 ARDS patients who were treated along with airway pressure limitation had cor-pulmonale. Higher driving pressure values and viral sources of lung damage were linked to the development of cor pulmonale. It was a separate risk factor for 29-day death and was connected to hemodynamic impairment.

Prevalence of cor pulmonale

Patients with ARDS may experience increased RV following load due to pulmonary vasoconstriction, vaso-occlusion, and/or endothelial failure.[7] During ARDS, harmful mechanical ventilation may exacerbate pulmonary vascular dysfunction. The occurrence of cor pulmonale has reduced as protective ventilatory techniques for ARDS are used more frequently in clinical settings.[8] However, our data shows that 79% of ARDS patients continue to develop cor pulmonale even in the modern era of protected mechanical ventilation. Despite the fact that the tidal volume in that trial was slightly higher (0.9 mL/kg), these results are consistent with another study that discovered a comparable prevalence (21%) of cor pulmonale in ARDS patients ventilated using a strategy supposed to minimise airway pressures.

Risk factors for cor pulmonale

Cor pulmonale and viral causes of lung damage are related. In 1984, Parker et al published the first report of reversible myocardial depression in sepsis patients.[9] They did this by using radionuclide cineangiography to identify LV systolic failure and dilatation.[9] Studies using echocardiography or pulmonary artery catheterization further confirmed these findings, and it was also found that more than 26.58% of septic patients commonly exhibited an RV systolic dysfunction. The cause of septic cardiomyopathy is strongly suggested to be impaired intrinsic contractility brought on by circulating cytokines.[10,11] One hypothesis is that an RV with sepsis-induced lower intrinsic contractility may be more vulnerable to ARDS-induced increased afterload and pulmonary vascular dysfunction.

Reduced respiratory system compliance was observed in cor pulmonale patients in our investigation, which most likely indicated a more severe degree of lung injury in this population. Because the main goal of our ventilation strategy was to reduce pressure plateau, these patients' severely compromised lung mechanics were ventilated at lower VT and PEEP levels. Multivariate study revealed an independent relationship between cor pulmonale and higher driving pressure levels.[12] Driving pressure may more accurately reflect tidal distending pressure, which is thought to represent the mechanical forces acting on the capillaries and alveoli during insufflations.

Limitation

The limitations of this study include a small sample population who were included in this study. The findings of this study cannot be generalized for a larger sample population.

CONCLUSION

In a sizable group of patients ventilated with airway pressure limiting (ADRS), cor-pulmonale was seen in 22% of cases. Greater driving pressures and sepsis were connected to a higher incidence of cor-pulmonale. Multivariate study revealed an association between it and greater mortality. Future studies on ARDS patients with cor pulmonale should focus on specific medications and interventions meant to treat RV and pulmonary vascular dysfunction.

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