Acute respiratory distress syndrome: Pulmonary and extrapulmonary not so similar

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Abstract

Acute respiratory distress syndrome (ARDS) is characterized by acute onset respiratory failure with bilateral pulmonary infiltrates and hypoxemia. Current evidence suggests different respiratory mechanics in pulmonary ARDS (ARDSp) and extrapulmonary ARDS (ARDSexp) with disproportionate decrease in lung compliance in the former and chest wall compliance in the latter. Herein, we report two patients of ARDS, one each with ARDSp and ARDSexp that were managed using real-time esophageal pressure monitoring using the AVEA ventilator to tailor the ventilatory strategy.

Keywords: Acute lung injury, acute respiratory distress syndrome, esophageal pressure, lung compliance, transpulmonary pressure

Introduction

Acute respiratory distress syndrome (ARDS) is a clinical syndrome characterized by acute onset respiratory failure resulting from various direct or indirect injuries to pulmonary parenchyma or vasculature. It has been postulated that ARDS during the initial phase may have different phenotypes depending on the type of insult involving either the lung parenchyma or the vasculature.[1,2] The former has been defined as pulmonary ARDS (ARDSp) and the latter as extrapulmonary ARDS (ARDSexp).[1] In ARDSp, alveolar epithelium is the principal site of injury,[3] whereas in ARDSexp, the primary site of insult is the capillary endothelium.[4,5] Although ARDSp is associated with more severe lung insult, current literature does not suggest the association of the type of ARDS with mortality.[6-8] Herein, we report two patients of ARDS, one each with ARDSp and ARDSexp who were managed using real-time esophageal pressure monitoring using the AVEA™ ventilator to tailor the ventilatory strategy.

Case Reports

Case 1

A 42-year-old female, a patient of dermatomyositis on oral immunosuppressive agents (60 mg/day of oral prednisolone and 100 mg/day of azathioprine), presented to the emergency department with a history of fever, dry cough, and breathlessness of 3-day duration. On examination, she had tachypnea and hypotension. Arterial blood gas analysis revealed hypoxemia [Table 1]. Imaging of the thorax (radiograph and computed tomography) revealed features of left mid zone consolidation with diffuse ground glass opacification [Figure 1]. A diagnosis of interstitial lung disease with severe community-acquired pneumonia with ARDS was considered. She was intubated and ventilated according to the ARDSnet protocol[9] (low-tidal volume strategy) using the AVEA™ ventilator (CareFusion, Germany). After fluid resuscitation (target

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central venous pressure of 12 cm of saline and inferior vena cava collapsibility index <15–20%), vasopressors (noradrenaline and vasopressin) were added to maintain a mean arterial blood pressure of 65–70 mmHg. She was initiated on antibiotics, stress ulcer prophylaxis, and deep venous thrombosis prophylaxis. To facilitate ventilation, vecuronium was given as continuous infusion along with midazolam and fentanyl.

The arterial saturation, however, did not show any improvement despite 12 h of ventilation. Due to hemodynamic instability, prone position ventilation was not attempted. A special nasogastric tube with an esophageal catheter (mounted with a 10 cm long esophageal balloon to measure esophageal pressure) was inserted through the nasal route to a depth of 60 cm from the incisors and then withdrawn to a depth of 40 cm to record the esophageal pressures. Once in the stomach (determined by a transient increase in the pressure during a gentle compression of the abdomen) the balloon was withdrawn slowly into the esophagus (determined by increased cardiac artifacts). The position was also confirmed by the changes in the transpulmonary pressure during tidal ventilation. The transpulmonary and esophageal pressures were recorded by giving a 5 s hold at the end-inspiration and end-expiration, respectively, which revealed that lung compliance was disproportionately lower than the chest wall compliance suggesting ARDSp [Table 2]. The ventilator settings were then adjusted to maintain an end-expiratory transpulmonary pressure between 0 and 10 cm of H_2O and an end-inspiratory transpulmonary pressure of <25 cm of H_2O.[10] However, her clinical condition did not show any improvement and she finally succumbed to her illness after 4 days of hospitalization due to refractory hypoxemia and shock.

Case 2

A 24-year-old male presented with fever, diarrhea, breathlessness, and decreased urine output of 5-day duration. Physical examination revealed hepatomegaly and bilateral basal crackles. A diagnosis of malaria was made based on a positive rapid malaria antigen
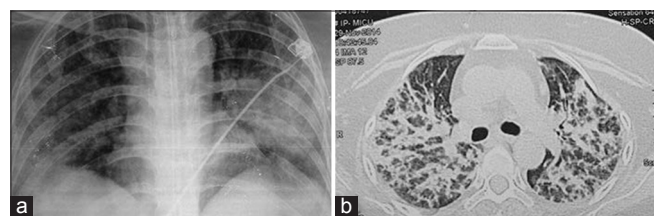

Figure 1: Chest radiograph (a) revealing bilateral reticular opacities with left mid-zone consolidation; high-resolution computed tomography (b) of chest revealing bilateral septal thickening with ground-glass opacification

test. He was given intravenous artesunate and oral doxycycline. Two days after admission, he complained of dyspnea and distension of abdomen. On examination, he was tachypneic and his saturation was 89% (at FiO_2 of 0.4). Abdominal examination revealed increase in the abdominal girth with findings suggestive of free fluid (dullness in flanks and positive shifting dullness). The abdominal pressure was 22 cm of saline. Abdominocentesis revealed hemorrhagic fluid. A contrast-enhanced computed tomography (CT) of abdomen revealed hemoperitoneum with intact intestines and abdominal vasculature [Figure 2].

Table 1: Baseline parameters of the index cases

Baseline parameters	Case 1	Case 2
Hemoglobin (g/dL)	8	11.9
Total leukocyte count (/mm³)	22,000	31,900
Platelet count (lakh/mm³)	2.2	0.57
Blood urea (mg/dL)	182	202
Serum creatinine (mg/dL)	1.8	5
Serum albumin (g/dL)	2.5	2.7
Serum bilirubin (mg/dL)	0.2	0.59
Aspartate transaminase (U/L)	81.8	82.2
Alanine transaminase (U/L)	36.3	237.7
Alkaline phosphatase (U/L)	125	106
Prothrombin index (%)	80	56
Procalcitonin (mg/mL)	10.3	8.6
Blood sugar (mg/dL)	178	128
PaO_2/FiO_2	70.4	146.4
SOFA score	15	14
Height (cm)	162	167
Ideal body weight (kg)	55	64

SOFA: Sequential organ failure assessment

Table 2: Ventilator and physiological parameters at baseline and 48 h

Parameters	Case 1		Case 2	
	Baseline	48 h	Baseline	48 h
Mode of ventilation	Volume control	Volume control	Volume control	Volume control
Tidal volume (mL)	240	260	360	360
PEEP (cm H_2O)	13	13	16	8
FiO_2	1	1	0.6	0.24
I: E ratio	1:1	1:1	1:1	1:2
Respiratory rate (/min)	35	35	30	22
Peak pressure (cm H_2O)	36	38	30	16
Plateau pressure (cm H_2O)	30	32	26	10
Mean airway pressure (cm H_2O)	22	24	22	14
Transpulmonary pressure (end inspiratory; Ptp plat), in cm H_2O	16	18	10	5
Transpulmonary pressure (end expiratory; Ptp PEEP), in cm H_2O	3	4	3	1
Esophageal pressure (end inspiratory), in cm H_2O	14	11	24.5	10.6
Esophageal pressure (end expiratory), in cm H_2O	12	9	22	2.5
Chest wall compliance, in mL	108	108	70	189
Lung compliance, in mL	17	14	80	100
Respiratory system compliance, in mL	13	13	32	65
Abdominal pressure (cm saline)	12	13	22	16

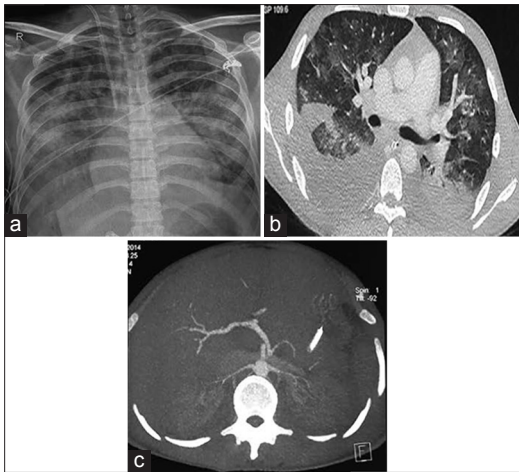PEEP: Positive end-expiratory pressure

Figure 2: Chest radiograph (a) revealing bilateral perihilar opacities; high-resolution computed tomography (b) of thorax showing bilateral ground-glass opacification and pleural effusion; contrast-enhanced computed tomography of abdomen (c) revealing hemoperitoneum with intact intestine and abdominal vasculature

A high-resolution CT of the thorax demonstrated diffuse ground-glass opacities with bilateral pleural effusion and basal consolidation [Figure 2]. He was intubated due to worsening respiratory failure (hypoxemia and tachypnea) and drowsiness. He was ventilated as per ARDS net protocol (low-tidal volume strategy) using the AVEA™ ventilator. An esophageal balloon was inserted as described above and the transpulmonary and esophageal pressures were recorded by giving a 5 s hold at end-inspiration and end-expiration, respectively. The measured parameters showed a proportionate reduction of chest wall and lung compliance suggestive of ARDSexp [Table 2]. The ventilator settings were then adjusted to maintain an end-expiratory transpulmonary pressure between 0 and 10 cm of H_2O and an end-inspiratory transpulmonary pressure of <25 cm of H_2O.[10] His respiratory failure resolved over the next 48 h of hospitalization and he was successfully extubated after 3 days of invasive mechanical ventilation. He was discharged 2 weeks after hospitalization and is currently doing well on follow-up.

Discussion

The two index cases emphasize the contrasting respiratory mechanics in ARDSp and ARDSexp. Both the cases of ARDS were managed by using similar ventilator strategy (ARDSnet protocol), but had different clinical profile, radiology and pulmonary mechanics, and clinical outcome. To the best of our knowledge, this is the first report from the Indian subcontinent utilizing the AVEA™ ventilator for using pleural pressure for partitioning the lung mechanics.

Although direct measurement of pleural pressures is the gold standard, due to its invasive nature, estimation by determining changes in esophageal pressures is the current method of choice for calculating pleural pressures.[1,11] The mechanical variations of the respiratory system that are observed in ARDS have been attributed primarily to lung because chest wall elastance was considered to be near normal.[11] However, in a study of 21 patients with ARDS (ARDSp and ARDSexp), a higher chest wall elastance was seen in ARDSexp whereas higher lung elastance was seen in ARDSp, suggesting stiffer lungs in ARDSp and stiffer chest wall in ARDSexp.[1] During mechanical ventilation, transmission of alveolar pressure to thoracic cavity depends on the lung and thoracic cage elastance ($P_{pl} = P_{aw} \times E_{cw}/E_{tot}$ and $P_{tp} = P_{aw} \times E_{lung}/E_{tot}$).[1,10] In patients with lower chest wall compliance (ARDSexp), higher pressures are transmitted to the pleura whereas in patients with poor lung compliance (ARDSp), lower pressures are transmitted to the pleura. Higher pleural pressures are associated with hemodynamic effects whereas higher transpulmonary pressures can cause lung over-distension.[1,7,12] Thus, compartmentalization of the respiratory system may enable in choosing patients with ARDS who are likely to benefit from the application of PEEP.

In case 1 (ARDSp), application of PEEP did not result in the improvement of respiratory system compliance despite maintaining an end-expiratory transpulmonary pressure above zero. On the other hand, in case 2 (ARDSexp), application of PEEP resulted in the improvement in respiratory system compliance (ARDSexp). Thus, the most important implication of differentiating ARDSp from ARDSexp is that for a given applied airway pressure, the transpulmonary pressures (distending pressure of the lungs) are higher in ARDSp as compared with ARDSexp (case 1 vs. case 2; 16 vs. 10). Although application of PEEP leads to increased end-expiratory lung volumes in both ARDSp and ARDSexp, it results in recruitment only in ARDSexp and causes alveolar overstretching in ARDSp, thereby increasing the risk of volutrauma.[1] This suggests that recruitment maneuvers will cause alveolar recruitment in ARDSexp and not in ARDSp.[13] This was also observed in case 2 where the application of PEEP led to improvement in respiratory mechanics, alveolar recruitment, and oxygen status. Further, in a recent study, mechanical ventilation guided by esophageal pressures was associated with a significant improvement in oxygenation and compliance in comparison to standard care.[10]

Conclusion

Although ARDS represents a clinical syndrome leading to respiratory failure, compartmentalization of respiratory system into ARDSp and ARDSexp may lead to better understanding of respiratory mechanics and adoption of different ventilator strategies.

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Conflicts of interest

There are no conflicts of interest.

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