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Acute respiratory distress syndrome (ARDS) is life threatening condition characterized by the acute onset of pulmonary edema of non-cardiogenic origin, along with bilateral pulmonary infiltrates and reduction in respiratory system compliance. Its definition has been reviewed several times since its first description in the late 1970s and a new definition (the Berlin definition) has recently been proposed. This definition suggested severity-oriented respiratory treatment by introducing three levels of severity according to PaO₂/FiO₂ and positive end expiratory pressure.¹

In spite of several new therapeutic approaches, ARDS mortality still remains high. Therefore, early recognition of ARDS modified risk factors and the avoidance of aggravating factors during the patient's hospital stay can help decrease its development. Even though, there are many extra pulmonary etiologies including sepsis, trauma, massive transfusion, drowning, drug overdose, fat embolism, and pancreatitis; pneumonia, undoubtedly, remains the leading cause of ARDS.² Hence microbiological assessment for any potential pathogens represents the first diagnostic effort. Some authors have suggested the systematic use of thoracic ultrasonography to differentiate it from cardiogenic pulmonary edema, and a pulmonary CT scan for better understanding of the underlying pathophysiology and the evaluation of lung recruitability.^{3,4,5}

Though, quite a few effective therapeutic modalities exist to ameliorate this deadly condition, no drug has proven to be effective in preventing or managing ARDS and the major treatment is supportive care along with adequate nutrition. Lung-protective ventilation, aiming to ensure adequate gas exchange by promoting lung recruitment while minimizing the risk of ventilator-induced lung injury, is still the key for better outcome in ARDS.⁶ Few other efficacious modalities such as short-term use of neuromuscular blockade at initial stage of mechanical ventilation, prone ventilation in severe ARDS and extracorporeal membrane oxygenation in ARDS with influenza pneumonia, and controversial use of inhaled vasodilators, corticosteroids have been documented by some research trials.^{7,8} However, efficient antifibrotic strategies are still lacking for patients with late-stage ARDS and hence, further researches are required to establish new therapies which can address the underlying pathophysiology.

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