Acute respiratory distress syndrome ARDS

MUHAMMAD RAMZAN
ASSISTANT PROFESSOR

Acute respiratory distress syndrome (ARDS), previously known as **respiratory distress syndrome (RDS)**, **adult respiratory distress syndrome**, or **shock lung**, is a severe, life-threatening medical condition characterized by widespread inflammation in the lungs. While ARDS may be triggered by a trauma or lung infection, it is usually the result of sepsis.

ARDS was recognized as the most severe form of acute lung injury (ALI), a form of diffuse alveolar injury. The AECC (American-European Consensus Conference)defined ARDS as an acute condition characterized by bilateral pulmonary infiltrates and severe hypoxemia in the absence of evidence for cardiogenic pulmonary edema.

Acute respiratory distress syndrome (ARDS) occurs when fluid builds up in the tiny, elastic air sacs (alveoli) in lungs. More fluid in lungs means less oxygen can reach bloodstream. This deprives organs of the oxygen they need to function.
ARDS typically occurs in people who are already critically ill or who have significant injuries. Severe shortness of breath — the main symptom of ARDS — usually develops within a few hours to a few days after the original disease or trauma.

ARDS is a disease of the microscopic air sacs of the lungs (alveoli) that leads to decreased exchange of oxygen and carbon dioxide (gas exchange).
ARDS is associated with several pathologic changes:
the release of inflammatory chemicals, breakdown of the cells lining the lung's blood vessels, surfactant loss leading to increased surface tension in the lung,fluid accumulation in the lung, and excessive fibrous connective tissue formation.
The syndrome has a high mortality between 20 and 50%. The mortality rate with ARDS varies widely based on disease severity, a person's age, and the presence of other medical conditions.

The signs and symptoms of ARDS usually begins within 72 hours of the initial insult or injury to the lung and may include
shortness of breath,
fast breathing,
low oxygen level in the blood
chest x-ray
frequently demonstrates generalized infiltrates or opacities in both lungs, which represent fluid accumulation in the lungs.
Other signs and symptoms that occur in people with ARDS may be associated with the underlying disease process. For example, those with ARDS from sepsis may have low blood pressure and fever, while a person with pneumonia may have a cough

Cause

The predisposing factors of ARDS are numerous and varied. Common causes of ARDS include sepsis, pneumonia, trauma, multiple blood transfusions, babesiosis, lung contusion, aspiration of stomach contents, and drug abuse or overdose.
Other causes of ARDS include burns, pancreatitis, near drowning, or the inhalation of chemical irritants such as smoke, phosgene, or chlorine gas.
Some cases of ARDS are linked to large volumes of fluid used during post-trauma resuscitation.

Diagnosis

ARDS is characterized by the following criteria:
lung injury of acute onset, within 1 week of an apparent clinical insult and with progression of respiratory symptoms ,bilateral opacities on chest imaging not explained by other lung pathology (e.g. pleural effusion, pneumothorax, or nodules)
respiratory failure not explained by heart failure or volume overload
decreased arterial PaO_2/FiO_2 ratio:
- mild ARDS: ratio is 201 - 300 mmHg (≤ 39.9 kPa)
- moderate ARDS: 101 - 200 mmHg (≤ 26.6 kPa)
- severe ARDS: ≤ 100 mmHg (≤ 13.3 kPa)

(a minimum positive end expiratory pressure (PEEP) of 5 cmH2O is required;

it may be delivered noninvasively with CPAP to diagnose mild ARDS). A decreased PaO 2/FiO 2 ratio indicates reduced arterial oxygen content relative to that of the inhaled gas, indicating a failure of the lung to transport oxygen into the blood.
The above characteristics are the "Berlin criteria" of 2012 by the European Society of Intensive Care Medicine, endorsed by the American Thoracic Society and the Society of Critical Care Medicine. They are a modification of the previously used criteria

Positive end-expiratory pressure (**PEEP**) is the pressure in the lungs (alveolar pressure) above atmospheric pressure (the pressure outside of the body) that exists at the end of expiration. The two types of PEEP are extrinsic PEEP (PEEP applied by a ventilator) and intrinsic PEEP (PEEP caused by a non-complete exhalation). Pressure that is applied or increased during an inspiration is termed pressure support.

Pathophysiology
ARDS is associated with diffuse alveolar damage (DAD) and lung capillary endothelial injury. The early phase is described as being exudative, whereas the later phase is fibroproliferative in character.
Early ARDS is characterized by an increase in the permeability of the alveolar-capillary barrier, leading to an influx of fluid into the alveoli. The alveolar-capillary barrier is formed by the microvascular endothelium and the epithelial lining of the alveoli. Hence, a variety of insults resulting in damage either to the vascular endothelium or to the alveolar epithelium could result in ARDS

The main site of injury may be focused on either the vascular endothelium (eg,sepsis) or the alveolar epithelium (eg, aspiration of gastric contents). Injury to the endothelium results in increased capillary permeability and the influx of protein-rich fluid into the alveolar space. Injury to the alveolar lining cells also promotes pulmonary edema formation. Two types of alveolar epithelial cells exist. Type I cells, which make up 90% of the alveolar epithelium, are injured easily. Damage to type I cells allows both increased entry of fluid into the alveoli and decreased clearance of fluid from the alveolar space.

Type II alveolar epithelial cells are relatively more resistant to injury. However, type II cells have several important functions, including the production of surfactant, ion transport, and proliferation and differentiation into type l cells after cellular injury. Damage to type II cells results in decreased production of surfactant with resultant decreased compliance and alveolar collapse. Interference with the normal repair processes in the lung may lead to the development of fibrosis

Neutrophils and some T-lymphocytes quickly migrate into the inflamed lung tissue and contribute in the amplification of the phenomenon. Typical histological presentation involves diffuse alveolar damage and hyaline membrane formation in alveolar walls. Although the triggering mechanisms are not completely understood, recent research has examined the role of inflammation and mechanical stress.

Principles of medical and physical therapy management

Intubation and ventilatory support are implemented if arterial blood gases are severely affected and respiratory distress worsened. An endotracheal tube can be placed through the nose o r mouth or a tracheostomy can be performed. The tidal volume is set at about 10 ml/kg of the patient's body weight. The patient usually establishes the respiratory rate, although it may be rapid. A positive end expiratory pressure (PEEP) of around 12 cm H20 maintains the alveoli open and thereby optimizes gas transfer at end-expiration.

The sitting position optimizes lung capacity. The use of a reclining chair at the bedside perhaps should be considered more often in the management of patients with acute lung injury.

Theoretically, the potential function of all lung fields will be benefited with the lungs in a more upright position. Patients who are not too unstable to tolerate upright positions may respond favorably to the prone position.


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