Acute Respiratory Distress Syndrome

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The Berlin definition of ARDS

1. Timing—patients with ARDS should be identified within 72 hours of a recognized risk factor and nearly all patients should be identified within 7 days.

2. Chest imaging—bilateral opacities consistent with pulmonary edema are seen on chest radiograph or chest computed tomography (CT) scan.

3. Origin of pulmonary edema—ARDS may be present in the setting of coexisting cardiogenic edema or volume overload if in the opinion of the treating physicians the respiratory failure is not fully explained by these conditions and there is a recognized risk factor for ARDS. In the absence of a recognized risk factor for ARDS, there should be some objective assessment of cardiac function such as echocardiography or pulmonary capillary wedge pressure measurement.

4. Oxygenation—because PEEP can affect the Pa_{O_2}/FI_{O_2} ratio there should be a minimum of 5 cm H_2O PEEP and 10 cmH_2O in those patients with severe ARDS ($Pa_{O_2}/FI_{O_2} < 100$).2

The Berlin definition for ARDS eliminates the use of the term "acute lung injury" and classifies the severity as

mild ARDS ($PaO_2/FIO_2 \leq 300$ but >200),

moderate ARDS ($PaO_2/FIO_2 \leq 200$, but >100),

severe ARDS ($PaO_2/FIO_2 \leq 100$), with associated morality rates of 27%, 32%, and 45%, respectively.2

Risk Factors

• BOX 36.4 Common Clinical Risk Factors for ARDS

Sepsis and the systemic inflammatory response syndrome (SIRS)
Prolonged hypotension/shock
Trauma (long bone fractures, lung contusion, fat embolism)
Acid aspiration
Near-drowning
Multiple emergency blood product transfusions
Pancreatitis
Disseminated intravascular coagulation
After cardiopulmonary bypass
Burn injury

• BOX 36.5 Common Pulmonary and Extrapulmonary Causes of ARDS

Direct Pulmonary Causes

Pneumonia
Acid aspiration
Inhalational lung injury
Lung contusion
Chest trauma
Near-drowning

Extrapulmonary Causes

Sepsis—systemic inflammatory response syndrome
Shock—hypotension
Pancreatitis
Trauma (fat embolism)
After cardiopulmonary bypass
Massive transfusion therapy
Burns
Elevated intracranial pressure (traumatic brain injury, intracerebral hemorrhage)

Incidence and Prevalence

The exact incidence and prevalence of ARDS has been variableMand influenced by the definition used (Box 36.6).
Early reports suggested that there were 150,000 patients with ARDS each year in the United States. This statistic led to an estimated incidence of 75 cases per 100,000 population.

Proposed estimates have suggested that the incidence of ARDS ranges from 1.5 to 64 cases per 100,000 population.

The authors noted that increasing age was associated with an increased incidence and mortality rate in ARDS.

A recent Spanish prospective observational study (the ALIEN) using the AECC definition and treatment with lung protective ventilatory support strategies reported an ARDS incidence of 7.2 per 100,000 population per year with an ICU and hospital mortality rate of 42.7% and 47.8%, respectively.

Clinical Manifestations

When ARDS becomes clinically apparent, the patient is usually noted to be in significant respiratory distress manifesting dyspnea, tachypnea, accessory muscle use, and increased work of breathing.

ARDS typically presents as an acute catastrophic complication in a patient who has one or more of the clinical risk factors.

The precipitating injury need not directly involve the pulmonary system.8,20 Past definitions have emphasized the need to exclude patients with previous or known chronic pulmonary or cardiovascular diseases.

The AECC definition excluded patients with elevated left-sided heart filling pressures and chronic infiltrative lung disease as the cause of the radiographic or physiologic alterations, but the Berlin definition allows for the concomitant presence

The physiologic hallmark of ARDS is the presence of hypoxemia despite high concentrations of inspired oxygen, evidence of an increased shunt fraction, decreased pulmonary compliance, and increased VD ventilation.

The chest radiographic manifestation of ARDS is the presence of bilateral pulmonary infiltrates with a normal cardiac silhouette.

Recent reports have cautioned that even among trained experts there is often disagreement concerning the interpretation of the chest radiograph. Chest CT has also demonstrated that the radiographic injury is not homogeneous and has a predominance in the dependent portions of the lung.

It is important to remember that ARDS is a clinical syndrome and the diagnosis is made clinically, not on the basis of a single radiograph, ABG analysis, or laboratory test.

Pathologic Manifestations

Type 1 alveolar epithelial cells compose the major gas exchange surface of the alveolus and are integral to the maintenance of the permeability barrier function of the alveolar membrane.

Type 2 pneumocytes are the progenitors of type 1 cells and are responsible for surfactant production and maintaining homeostasis.

During ARDS there is damage to both capillary endothelial and alveolar epithelial cells.

Cellular injury and alteration of the normal barrier function results in a permeability defect that gives way to flooding of the alveoli with protein-rich fluid and inflammatory cells. This results in the alteration of pulmonary mechanics, physiology, and gas exchange.

There is alteration of surfactant that results from damage to the type 2 pneumocyte and from the inactivation and dilution of alveolar surfactant from the protein and fluid that have entered into the alveolar space, respectively. Surfactant dysfunction can lead to atelectasis and a further reduction in pulmonary compliance.

In addition, dysfunction of the alveolar epithelial cells can impair the resorption of fluid from the alveolar space, which augments the parenchymal injury process and gas exchange abnormalities.

The observed pathologic findings in ARDS depend on the timing of the tissue sampling.

Histologic features of the injury include microthrombi composed of platelets and white blood cells within the capillary lumen, denudation of the alveolar epithelial lining cells, swelling of the capillary endothelial cells, interstitial and alveolar infiltration by polymorphonuclear leukocytes (PMNLs), and hyaline membrane formation within the alveoli.

An intense inflammatory reaction involving PMNLs, activated monocytes, macrophages, and endothelial cells is present in the fibroproliferative phase of lung injury.

Proinflammatory and antiinflammatory molecules produced by these activated cells may be found in the circulating blood and bronchoalveolar lavage fluid.

This phase may be followed by fibrosis, but this fibrosis does not appear to have the same permanence as typical fibrosis would have and may actually resolve over time in some survivors of the injury

Pathophysiology

ARDS may develop as a result of epithelial or endothelial cell injury.

Both sites of injury and cells are important for maintenance of normal barrier function and are capable of initiating an inflammatory response.

In the majority of clinical settings the initial site of the ARDS involves the capillary endothelial cell, which may be the initial manifestation of a “panendothelial cell injury” resulting from SIRS.

Endothelial cell injury compromises the integrity of the vascular barrier and results in transudation of fluid and inflammatory mediators into the interstitial tissues and ultimately into the alveoli.8

The frequent occurrence and early involvement of lung dysfunction as a component of multiple organ dysfunction/failure lends support to the hypothesis of a panendothelial cell injury as one of the target injuries in the setting of sepsis or SIRS.81

• BOX 36.7 Basic Management Strategies for Patients With ARDS

Identify and treat underlying/predisposing cause of ARDS
Ventilatory support
- Lung-protective ventilatory support strategy
- Application of PEEP (per ARDS Network protocol)

Restore and maintain hemodynamic function
- Conservative fluid replacement strategy using goal-oriented approach
- Vasopressor and inotropic support as needed to meet goals

Prevent complications of critical illness
- Stress ulcer (stress-related mucosal disease) prophylaxis
- Preventive strategies for pulmonary embolism and deep venous thrombosis
- Prevent infections such as ventilator-associated pneumonia
- Control glucose and metabolic function

Prevent development of multiple-organ dysfunction/failure
Ensure adequate nutrition
Avoid oversedation and medication errors
Use of weaning protocol with spontaneous breathing trials when ready to wean
Cautious use of steroids for fibroproliferative phase (avoid if patient has received neuromuscular blocking drugs)

ARDS, *Acute respiratory distress syndrome;* PEEP, *positive end-expiratory pressure.*