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Acute respiratory distress syndrome

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Introduction

Medicine, as we know it today is a product of years of research, scientific breakthroughs and technological advancement. With advanced understanding of diseases, their underlying cellular processes, and its clinical manifestations, clinicians are able to effectively identify, treat and in many cases, prevent many disorders which otherwise would result in countless deaths. An example of such disorder is acute respiratory distress syndrome (ARDS), which is a severe form of acute lung injury (ALI), classified by the presence of acute arterial hypoxemia and bilateral pulmonary infiltrates not attributable exclusively to cardiogenic or hydrostatic causes, resulting in impaired oxygenation, and requiring critical care management, which, in many cases includes mechanical ventilation.[1]

First described by Ashbaugh in 1967, ARDS was observed in combat patients who developed respiratory distress, diffuse lung infiltrates, and respiratory failure despite oxygen therapy after occurrence of non-thoracic injuries, severe pancreatitis, massive transfusions, sepsis and various other insults.[2] As compared to decades ago, today, clinicians are armed with advanced knowledge about the pathophysiology, etiology, and clinically proven treatment methods which help to improve mortality rates in cases of ARDS.

There are numerous and varied disorders which are associated with the potential to produce ARDS.[1] Some cause it by direct insult of the lung, while others indirectly cause acute lung injury by initiating an acute systemic inflammatory response.[3] Disorders that can cause acute lung injury and ARDS include, but are not limited to sepsis, shock, drugs, pancreatitis, neurogenic insult, pneumonia, major trauma, pulmonary aspiration or near drowning, burns, toxic gases/fumes inhalation, gat embolism, massive blood transfusion, amniotic fluid embolism, air embolism, eclampsia, poisoning, radiation, and in some cases, over distention caused b by mechanical ventilation.[1,3]

ARDS directly affects lung mechanics, gas exchange and pulmonary vasculature of both lungs,[3] but it is critically important to understand the pathogenesis to fully comprehend the changes that result in ALI and ARDS. A particularly important part of the pathogenesis of ARDS and ALI is the recruitment of inflammatory cells and neutrophils into the lungs, which can be caused by trauma, sepsis, shock or direct lung injury.[3] During an inflammatory crisis, the vascular endothelial cells in the pulmonary vascular systems are activated, which results in expression of the leukocyte adhesion molecule, thus, leading to accumulation of neutrophils within the pulmonary vasculature.[1] These neutrophils and other inflammatory cells are known to release a variety of substances which are destructive to the components of the alveolar wall.[1] In addition, cytokines, proteases, and oxidants released as byproducts of oxidative metabolism by the neutrophils further influence the trafficking of neutrophils into the alveoli to cause further damage.[1] Another important process which leads up to ARDS and ALI is the activation of the coagulation system, which causes a release of procoagulants, resulting in an increase in the activity of proteins that inhibit fibrinolysis, thus, causing an increase in production of thrombin and fibrin, as well as evidence of thrombosis within the pulmonary capillaries.[1]

Despite the number of etiologic factors and various pathogenesis mechanisms that lead to ALI an ARDS, the pathologic findings are relatively similar, regardless of the underlying cause.[1] As described by a pathologist, the pattern of injury seen in ARDS is referred to as diffuse alveolar damage.[1] Injury to type I alveolar epithelial cells and pulmonary capillary cells appear to be the primary factor in pathogenesis.[1] Early in the course of ARDS, often referred to the exudative phase, fluid can be seen in the interstitial space of the alveolar septum and lumen.[1] In some cases, scattered bleeding and regions of collapse may be seen due to inactivation of surfactant and decreased surfactant production from alveolar type II epithelial cells.[1] Microscopic analyses of the lung parenchyma show an influx of inflammatory cells consisting of neutrophils and macrophages in the alveolar lumen and the interstitial space.[1] A characteristic finding among patients with ARDS is the formation of hyaline membrane. Composed of a combination of fibrin, cellular debris, and plasma proteins, hyaline membrane represent protein rich edema fluid that has filled the alveoli and then deposited on the alveolar surface.[1] Proliferative phase then follows exudative phase after 1 to 2weeks where alveolar type II epithelial cells replicate in an attempt to replace the damaged type I epithelial cells, causing development of pulmonary hyperplasia.[1] Another component of the proliferative phase is accumulation of fibroblasts in the pulmonary parenchyma, which in an attempt to repair the damage goes on to develop significant scar tissue, resulting in fibrosis.[1]

The pathophysiologic features of ARDS as a result of the pathology described above cause shunting and V/Q mismatch, deactivates surfactant, increase pulmonary vascular resistance, decrease pulmonary compliance, and decrease the functional residual capacity (FRC).[1] Alveolar flooding, the most striking problem in ARDS, prevents effective ventilation, creating areas of shunt, and causing regions of lung where blood passes without being oxygenated.[1] These areas of shunting and V/Q mismatch in the lungs results from non-uniform distribution of the pathologic process.[1] In addition to complications in oxygenation, interstitial and alveolar fluid also causes surfactant deactivation, resulting in extensive

collapse of alveoli.[1] Resulting hypoxemia from pulmonary edema and V/Q mismatch, along with fluid in the interstitium produces vasoconstriction within the pulmonary arterial system, causing vasoconstriction and increase in resistance of the small pulmonary vessels, which makes it prone to microthrombi, further V/Q mismatch and shunting, as discussed previously.[1] The heterogeneous collapse of the lungs caused by deactivation of surfactant result in a net result of fewer effectively functional alveoli and less volume enters the lung for any given inflation pressure, thus, resulting in lower FRC and decreased pulmonary compliance.[1] This causes the patient to breathe at a much lower overall lung volume than normal, with air entering areas of the lungs which are relatively normal, resulting in rapid but shallow breaths.[1] This type of breathing pattern is inefficient, requiring increased energy demand, and contributes to dyspnea and will eventually result in hypercapnic respiratory failure secondary to ARDS.[1]

The resulting pathophysiology of ARDS presents clinicians with a clinical picture reflecting presence of non-cardiogenic pulmonary edema along with presence of underlying disease in the lungs. Clinical symptoms of ARDS and ALI ensue within several hours to a day following the initial insult leading to pulmonary injury.[1] Generally, the patient exhibits the symptoms of dyspnea and tachypnea, although a chest x-ray early in the course of ARDS may not reveal any significant findings.[1] An arterial blood gas during initial stages of ARDS may reveal normal ventilation, but an oxygenation disturbance, with an increase in alveolar-arterial difference on partial pressure of oxygen ($A\text{-}aDO_2$).[1] As fluid and protein continue to leak into the pulmonary vasculature into the interstitium and alveolus, clinical findings become more significant, resulting in extreme dyspnea and tachypnea, and rales/coarse crackles may be heard upon chest auscultation.[1] Chest radiograph at this point would reveal abnormal findings of extensive interstitial and alveolar edema, and further deteriorating oxygenation and $AaDO_2$ in the arterial blood gas.[1]

The official diagnosis of ARDS is generally based on a combination of clinical and radiologic findings. The 2012 Berlin definition of ARDS describes the full criteria for establishing the diagnosis of ARDS, and describe in detail each of these criteria and associated pathology and pathophysiology.[1] In the criteria of timing, the new or worsening symptoms of respiratory symptoms is within 1 week of a known clinical insult. In the criteria for chest imaging, there is presence of bilateral opacities which is not fully explained by effusions, lobar/lung collapse, or nodules.[1] In the criteria for origin of edema, there is presence respiratory failure secondary to pulmonary edema not fully explained by cardiac failure of fluid overload.[1] This finding requires objective assessment (electrocardiography, PCWP, PAP, and etc) to exclude hydrostatic edema if no risk factor for ARDS is present.[1] In the criteria of oxygenation, the PaO_2/FiO_2 (P/F ratio) is used to describe the severity of the ARDS.[1] Mild ARDS is suggested by a P/F ratio <300 but >200 with PEEP or CPAP>5.[1] Moderate ARDS is suggested by a P/F ratio <200 but >100 with PEEP or CPAP >5.[1] Severe ARDS is suggested by a P/F ratio of <100 with PEEP or CPAP of >5.[1]

Management of ARDS requires critical care, and centers of 3 main issues of treatment of underlying causes, interruption of the pathogenetic sequences of events involved in development of capillary leak, and support of gas exchange and until the pulmonary process improves.[1] In numerous cases, the treatment of underlying disease process is not possible or successful, thus supportive case is administered using mechanical ventilation along with management of other treatable abnormalities.[1] In cases where the underlying cause of ARDS is sepsis and infection, treatment with appropriate antibiotics and draining/ debriding (if possible) of infected site is crucial to allowing the pulmonary vasculature of resolve the acute ARDS process.[1] In patients who are mechanically ventilated for respiratory failure, the most effective strategy involves applying lower tidal volumes (4-6mL/kg) than the traditional tidal volumes (8-12mL/kg) used in prior practice before the ARDS net study and many other research studies which changed the clinical practice in management of ARDS patients.[1] Using lower tidal volumes helps to prevent barotrauma caused by excessive volumes which over distend the alveoli, causing further microscopic damage to the alveoli.[1] In addition, the application of optimal PEEP during mechanical ventilation helps to enhance the opening of the collapsed alveoli, which improves oxygenation by increasing FRC and decreasing shunt, while providing alveolar stability and preventing atelectrauma, which causes further microscopic alveolar damage and occurs with repeated opening and closure of unstable alveoli resulting from surfactant deactivation.[1]

The ARDS net study, which shapes the current strategy to manage mechanical ventilation in patients with ARDS and ALI was a multicenter, parallel group, randomized control research trial funded by the National Heart, Lung and Blood Institute of the National Institute of Health between March 1996 and 1999.[4] The research trial studied and compared the effect of using lower tidal volumes in opposition to high tidal volumes in patients with ALI and ARDS.[4] The study was performed across 10 university hospitals affiliated with the ARDS net group, and consisted of 861 patients.[4] The research study concluded that treatment with a ventilation approach of low tidal volume of 4-6mL/kg, designed to protect the lungs from excessive stretch resulted in improvements in clinical outcomes of patients with acute lung injury and acute respiratory distress syndrome.[4]

ARDS does not discriminate against any particular race, gender or age group, but can affect everyone equally. Much progress has already been made in the understanding of the pathophysiologic mechanisms of ARDS, yet, it still poses a challenge to the Physicians and medical staff. Supportive therapy using lower tidal volumes aimed at preventing further alveolar damage, and maintaining adequate gas exchange and oxygenation is critical in the management of ARDS. It is important for clinicians to understand the pathophysiologic mechanisms which cause and lead to ARDS, because early identification, and early implementation of effective treatment strategies in ARDS could prevent further physiologic damage in patients, and could possibly improve mortality rates.

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None.

Conflict of interest

The author declares no conflict of interest.

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