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MANAGEMENT OF A PATIENT WITH ARDS: A CASE REPORT

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ABSTRACT: Acute Respiratory Distress Syndrome (ARDS) is a permeability pulmonary edema characterized by increased permeability of pulmonary capillary endothelial cells and alveolar epithelial cells, leading to hypoxemia that is refractory to usual oxygen therapy. ARDS is characterized by a brief precipitating event followed by rapidly developing dyspnea. These patients have markedly impaired respiratory system compliance and reduced lung volume. The hypoxemia is refractory to low fraction of oxygen concentration and low positive end expiratory pressure (PEEP). The mortality of ARDS is around 35-40%. Current therapy of ARDS revolves around treatment of underlying cause, lung protective ventilatory strategy and appropriate fluid management. We present a case of ARDS managed in our ICU along with a detailed discussion about the pathophysiology and treatment modalities for the management of a patient with ARDS.

INTRODUCTION: ARDS is a syndrome of acute respiratory failure that presents with arterial hypoxemia, dyspnea and a marked increase in the work of breathing. Most patients require endotracheal intubation and positive pressure ventilation. There are several clinical disorders associated with the development of ARDS including sepsis, pneumonia aspiration of gastric contents and major trauma.¹

Pathology: In the acute phase (the first 1-6 days), there is evidence of interstitial and alveolar edema with accumulation of neutrophils, macrophages and red blood cells in the alveoli. There is also evidence of both endothelial and epithelial injury, often with denuding of the alveolar epithelium. There are prominent hyaline membranes in the alveoli as well. In the sub-acute phase (next 7-14 days) some of the edema has usually been reabsorbed, and there is evidence of attempts at repair with proliferation of alveolar epithelial type II cells. There may also be infiltration of fibroblasts and some evidence of collagen deposition. In the chronic phase (after 14 days) there is resolution of the acute neutrophilic infiltrate with more mononuclear cells and alveolar macrophages in the alveoli, and often more fibrosis with ongoing evidence of alveolar epithelial repair. In many patients, resolution progresses without fibrosis and simply with gradual resolution of the edema and acute inflammation.²

Pulmonary (Primary)	Extrapulmonary (Secondary)
Pneumonia	Sepsis
Aspiration	Pancreatitis
Smoke inhalation	Blood transfusion
Lung Contusion	Fat emboli

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Near drowning	Major burns
Venous air embolism	Poly trauma
	Amniotic fluid embolism
	Neurogenic pulmonary edema
	Cardiopulmonary bypass
	Drug reactions (aspirin, nitrofurantoin)
Pathogenesis: Table 1: Conditions associated with ARDS	

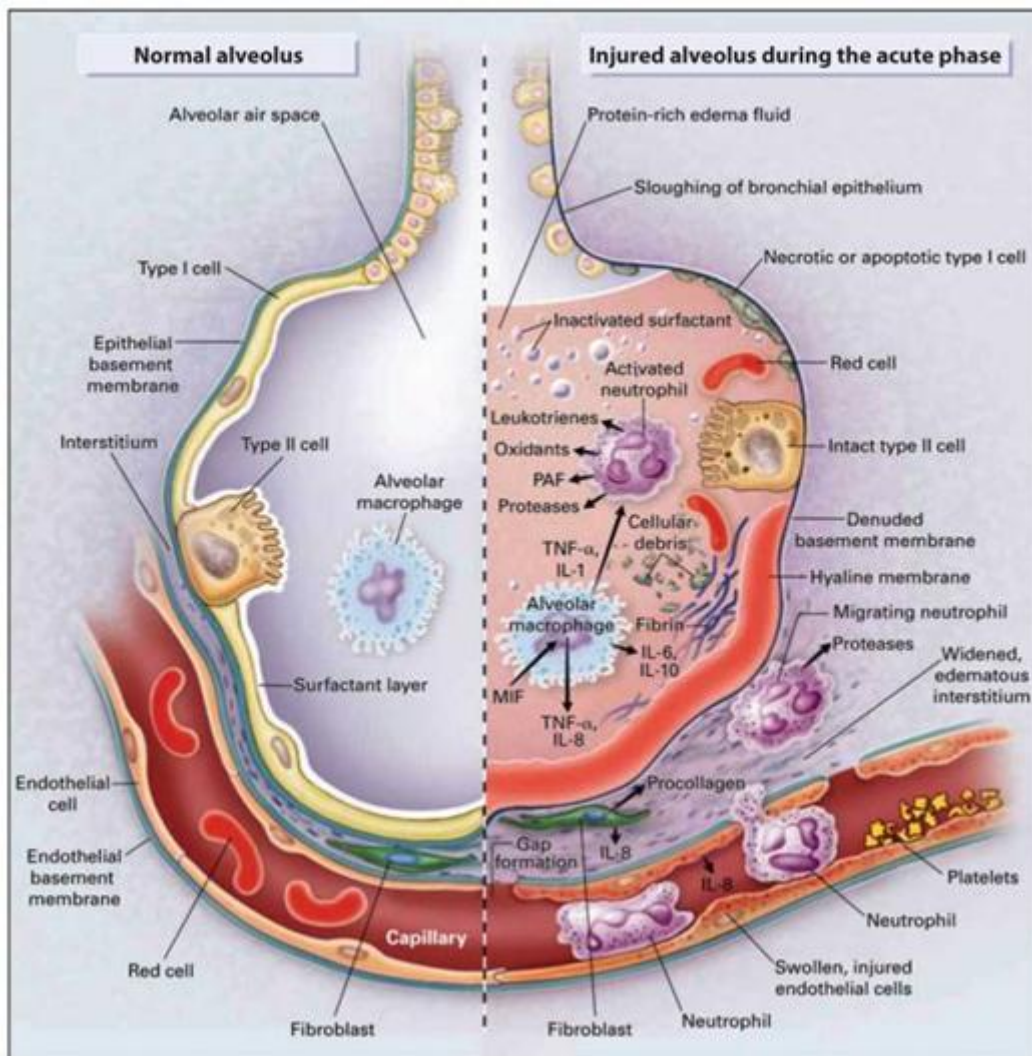


Fig. 1: Pathogenesis of ALI / ARDS

(a) The normal alveolus and (b) the injured alveolus in the acute phase of acute lung injury and the acute respiratory distress syndrome. In the acute phase of the syndrome (b), there is sloughing of both the bronchial and alveolar epithelial cells; protein-rich hyaline membranes form on the denuded basement membrane. Neutrophils adhere to the injured

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capillary endothelium and marginate through the interstitium into the air space, which is filled with protein-rich edema fluid. In the air space, alveolar macrophages secrete cytokines; interleukin (IL)-1, -6, -8, and -10; and tumor necrosis factor α (TNF- α), which act locally to stimulate chemotaxis and activate neutrophils. IL-1 can also stimulate the production of extracellular matrix by fibroblasts. Neutrophils can release oxidants, proteases, leukotrienes, and other pro inflammatory molecules such as platelet-activating factor (PAF). A number of anti-inflammatory mediators also present in the alveolar milieu include IL-1 receptor antagonist, soluble. The influx of protein-rich edema fluid into the alveolus leads to the inactivation of surfactant. Abbreviation: MIF, macrophage-inhibitory factor.

CASE REPORT: A 25 year old female with history of fever, cough and chest discomfort for 5 days presented in the emergency room in an unresponsive and gasping state. On physical examination her pulse rate was 160/min, feeble. Respiratory rate is 8/min. SPO2 - 38% with O2 via face mask at 10 L/min. BP-80/50 mmHg. Respiratory system show decreased bilateral air entry.



Fig. 2: Bilateral fluffy infiltrates suggestive of fulminant ARDS

Patient was immediately intubated, well-sedated and paralyzed and connected to mechanical ventilator on pressure control mode ventilation. Settings were PEEP 18, Respiratory rate 17, FiO2 100%, Inspiratory Pressure 30 cm H2O, Inspiratory time 1.5 sec. Her platelet counts were low (85000). Following this her BP fell further. Our working diagnosis was viral pneumonia progressed to ARDS with Thrombocytopenia.

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Fig. 3: Bilateral pulmonary infiltrates with ET tube; improvement seen after positive pressure ventilation with high PEEP

In the MDICU she was started on Injection Piperacillin + Tazobactam 4.5 gm IV Q6H, Tab Clarithromycin 500mg BD, Tab Oseltamivir 150 BD, Nor-Adrenaline Infusion, Steroids, Morphine-Midazolam Infusion. We kept her well hydrated after confirming the left ventricular function with 2D echo cardiogram. Nor-Adrenaline infusion was titrated to keep the mean arterial pressure more than 70 mmHg. Lab Investigation showed TC 16000, Hb 8.2, Platelet 85000, LFT, RFT and serum electrolytes were within normal limits. Her initial ABG showed severe respiratory acidosis.

We noticed subcutaneous emphysematous changes over her chest and neck region. She maintained a good urine output. Despite mechanical ventilation with high positive pressure, a saturation reading above 80% could not be attained in the first day of admission. On the 2nd day her saturation showed improvement. The PEEP was gradually reduced, as she had recurrent episodes of Hypotension. By 3rd day her ABG showed improvement but her TC continued to increase. She was started on Inj. Meropenem, Inj. Netilmycin, Inj. Moxifloxacin. Chest physiotherapy was started on day 4. FiO₂ and PEEP were gradually decreased over the next few days to maintain her saturation and ABG within normal limits. Her TC decreased and inotropic supports were stopped. By day 5 she was maintaining spontaneous ventilation on CPAP and was extubated the next day. She was ambulated on 7th day and was maintaining saturation of 99% in room air by day 9. She was shifted to ward by day 11.

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Fig. 4: Clear lung fields with minimal infiltrates

DISCUSSION: Acute Respiratory Distress Syndrome (ARDS) is a permeability pulmonary edema characterized by increased permeability of pulmonary capillary endothelial cells and alveolar epithelial cells, leading to hypoxemia that is refractory to usual oxygen therapy. The first definition of ARDS dates back to Asbaugh and colleagues in 1976.³ Followed by the American-European consensus Conference definition in 1994. Recently, a new consensus definition of ARDS, the Berlin definition has been published. ARDS is characterized by a brief precipitating event followed by rapidly developing dyspnea. These patients have markedly impaired respiratory system compliance and reduced aerated lung volume. The hypoxemia is refractory to low fraction of oxygen concentration and low positive end expiratory pressure (PEEP). The mortality of ARDS is around 35-40%. Current therapy of ARDS revolves around treatment of underlying cause, lung protective ventilatory strategy and appropriate fluid management.

ARDS used to be diagnosed when patient fulfilled the following criteria: Acute onset, Presence of predisposing conditions (Table), bilateral infiltrates on chest X ray, PaO₂/FiO₂ less than 200 for ARDS and less than 300 for ALI. Pulmonary arterial occlusion pressure less than 18 mmHg or no clinical evidence of left sided heart failure.

The new definition of ARDS is called Berlin ARDS definition.

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Berlin definition for acute respiratory distress syndrome.	
Acute respiratory distress syndrome	
Time	Within one week of a known clinical event, or new event, or worsening of respiratory symptoms.
Chest X-ray ^a	Bilateral opacities— not completely explained by pleural effusion, lobar or pulmonary collapse, or nodules.
Origin of edema	Respiratory failure, not fully explained by heart failure or volume overload. Need for objective evaluation (echocardiography) to rule out hydrostatic edema when there is no risk factor.
Oxygenation ^b	
Mild	200 mmHg < PaO ₂ /FiO ₂ ≤ 300 mmHg with PEEP or CPAP ≥ 5 cm H ₂ O ^c
Moderate	100 mmHg < PaO ₂ /FiO ₂ ≤ 200 mmHg with PEEP ≥ 5 cm H ₂ O
Severe	PaO ₂ /FiO ₂ ≤ 100 mmHg with PEEP ≥ 5 cm H ₂ O

CPAP, continuous positive airway pressure; FiO₂, fraction of inspired oxygen; PaO₂, partial pressure of oxygen; PEEP, Positive end-expiratory pressure.

^a Chest X-ray or tomography.

^b If the altitude is higher than 1,000 meters above sea level, a correction factor must be applied as follows: [PaO₂/FiO₂ × (barometric pressure/760)].

^c Can be released noninvasively in cases of mild ARDS.

Table 2: Berlin definition of ARDS

Treatment of ARDS: A large number of pharmacological therapies have been evaluated for the treatment of ALI/ARDS. These treatments include glucocorticoids, surfactants, inhaled nitric oxide, antioxidants, protease inhibitors and a variety of other anti inflammatory treatments. Unfortunately, to date none of these pharmacological treatments has proven to be effective.⁴

However, despite the lack of a specific pharmacological treatment, lung protective ventilation has reduced mortality of ALI from 40% in 2000 to 25% in 2006.⁵ Further the use of a fluid conservative strategy after patients with ARDS are no longer in shock has reduced the duration of mechanical ventilation.⁶ Ventilation in ARDS: NIV/CPAP has a very limited role in a patient developing ARDS. Most patients show very little or transient improvement with NIV and instead proceed to tracheal intubation before a major deterioration occurs. In the following situations of ARDS, mechanical ventilation should be initiated electively- Persistent hypoxemia (SpO₂ < 90% on face mask oxygen or NIV.), excessive work of breathing and high minute ventilation, hemodynamic instability.

In ARDS, mechanical ventilation is primarily used to reverse hypoxemia and decrease the work of breathing. Positive pressure ventilation is unphysiological and adverse effects of this must be prevented or rapidly reversed. Initially there may be a significant hemodynamic deterioration which requires adequate monitoring and reversal with fluids and appropriate ionotrope and vasopressor agents. High volumes, high airway pressures and repeated opening and closing of collapsed alveoli may further damage the lung, worsen the ARDS and contribute to systemic inflammation. These patients are prone to ventilator associated pneumonia due to prolonged ventilation required and occasionally due to use of corticosteroid. Oxygenation goal should be PaO₂ of 55-80 mmHg or SpO₂ of 88-95%. pH goal should be between 7.30 and 7.45.

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Rescue strategies may be needed for life threatening hypoxemia. The following strategies are used:

Recruitment maneuver: Application of high level of sustained airway pressure to open up the collapsed alveoli and then high PEEP to prevent re collapse. Complications include hypotension, desaturation, cardiac arrhythmia and barotrauma.

Inverse ratio Ventilation: Prolongation of inspiratory time results in increase in mean airway pressures often improving oxygenation.

Prone Position: It can be considered in severe hypoxemia when $PaO_2/FiO_2 < 140$ mmHg, or $PaO_2 < 55$ with $FiO_2 > 0.7$. Contraindicated in severe shock, raised ICP, spinal instability, recent thoracoabdominal surgery, massive hemoptysis and open wounds on ventral surface.

High frequency Ventilation: Mechanical ventilation with Respiratory rate more than 100 breaths per minute. Higher mean airway pressure is attained so there is more alveolar recruitment.

Hemodynamics is maintained with fluids, vasopressors and dobutamine for a low cardiac index. Monitoring fluid status with central line is required. Conservative fluid management strategy should be adopted but not at risk of organ perfusion. Present guidelines recommend to initiate enteral immunonutrition with formulation containing anti-inflammatory lipid profile, that is eicisapentenoic acid, gamalinoleic acid (GLA) (Omega 3 fish oil) and anti-oxidants.

CONCLUSION: ALI/ARDS occurs in 200, 000 people per year in the US and has an estimated mortality rate of 40%. There are several clinical disorders associated with the development of ARDS, but the pathogenesis involves inflammatory injury to the lung endothelium and epithelium, which causes a marked increase in lung vascular and epithelial permeability and the passage of protein rich edema fluid into the air spaces. The initial lung injury can be compounded by ventilator associated lung injury, particularly when high tidal volumes and inflation pressures were used. Clinical recovery from ARDS depends on the use of lung protective ventilation with lower tidal volumes and the reduction of airway pressures, which facilitate the resolution of inflammation, repair of the injured epithelium and removal of edema fluid from the air spaces.

Currently in spite of the remarkable advancements in the understanding of its pathogenesis, the only effective therapeutic measure to decrease mortality is low tidal volume mechanical ventilation and prone ventilation for severe ARDS cases. In extreme cases, ECMO seems to serve as a bridge to recovery and enables lung protective ventilation. Most ARDS patients die of multi organ failure rather than irreversible respiratory failure indicating that ARDS, is closely associated with other organs by neurological, biochemical, metabolic and inflammatory reactions. Moreover the lungs may play an important role in the development of non-pulmonary organ failure in ARDS. Thus early recognition of ARDS modified risk factors and the avoidance of aggravating factors (eg. Non protective mechanical ventilation, multiple blood product transfusion, positive fluid balance, ventilator associated pneumonia and gastric aspiration) can help decrease its incidence.

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