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INTERLEUKIN 6 IN DIAGNOSIS OF ARDS PHASES

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ABSTRACT:

Background: ARDS is an acute inflammatory process of the lungs with overall mortality ranging from 35% to 50%

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Introduction: Acute respiratory distress syndrome (ARDS) is a clinical condition that manifests suddenly leading to diffuse lung injury and extremely severe hypoxemia. Diffuse pulmonary edema, which cannot be attributed to cardiac failure, renal failure, or excessive hydration, is the pathophysiology of ARDS **(1).**

The Berlin definition of ARDS (2)*:*

• Acute onset (within 7 days of new or worsening respiratory symptoms mainly dyspnea)

• Bilateral radiographical opacities that are not fully explained by effusion, atelectasis, or masses

• Arterial hypoxemia defined by thresholds:

- ***Mild:*** 200 < PaO2/FiO2 ratio ≤300 mm Hg, on CPAP or PEEP≥5 cm H2O
- ***Moderate:*** 100< PaO2/FiO2 ratio ≤200 mm Hg, on PEEP ≥5 cm H2O
- ***Severe:*** PaO2/FiO2 ratio ≤100 mm Hg, on PEEP ≥5 cm H2O

• Identified risk factor for ARDS (if no clear risk factor, exclude heart failure as a cause)

*** Indicators to predict the prognosis of ARDS:**

- Clinical factors like oxygenation index and ventilator settings.
- Physiologic factors like pulmonary function.
- Radiologic factors like chest CT.
- Pathologic factors like lung biopsies and biomarkers, principally protein from biomaterials like blood, urine, sputum, and bronchoalveolar lavage fluid.
- Biomarker **(3).**

1- A hypercoagulation protein called plasminogen activator inhibitor-1 (PAI-1) blocks the fibrinolytic mechanism. A reduction in protein C causes hypercoagulation because it is an anticoagulant factor that is made in the liver. Patients with ARDS had greater plasma levels of PAI-1 and decreased plasma levels of protein C **(4).**

2- IL-1, IL-6, and IL-8 levels in serum or plasma were considerably greater in non-survivors than in survivors at the time of the beginning of ARDS **(5).**

IL-6: The generation of TNF and IL-6 is currently considered to be another significant inflammatory mediator in the development of SIRS. SIRS's onset, progression, and uncontrolled reactions are all significantly influenced by IL-6, which is also directly related to the severity and prognosis of the condition **(6).**

Activated macrophages release IL-6, which promotes acute-phase reactions in the liver. It has been suggested that IL-6 integrates signals created early in the inflammatory response since it is activated in part by TNF-α and IL-1β. ARDS, severe pneumonia, or both were discriminated from cardiogenic pulmonary edema by elevated IL-8 and IL-6 levels.

There are 5 main factors to consider while researching and locating ARDS biomarkers: (7)

(I) to foretell the onset of ARDS in high-risk patients;

(II) to classify the severity of the disease into more precise phenotypes or categories;

(III) to offer a fresh understanding of its pathogenesis in order to develop novel therapeutics;

(IV) to track treatment response, and

(V) to aid in outcome prediction

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