Interleukin 8 vs Lung Ultrasound in the diagnosis of ARDS phases

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Abstract

Background: ARDS is an acute inflammatory process of the lungs brought on by direct or indirect damage to the alveolar-capillary membrane

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Introduction: Acute respiratory distress syndrome (ARDS) is clinically identified by bilateral lung infiltrates & severe hypoxemia in the lack of congestive heart failure. Numerous biochemical and cellular processes are associated with lung injury in ARDS, much like they are with other inflammatory processes in the body. **(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-8:** The extensive breakdown of the alveolar epithelium & the oversaturation of the alveolar spaces with proteinaceous exudates containing significant neutrophil concentrations are the hallmark lesions of ARDS. Chemotactic cytokines to a significant part control leukocyte movement. The two main groups of chemokines are α-chemokines and β-chemokines. The former attracts polymorphonuclear neutrophils, while the latter attracts monocytes and lymphocytes. One of the α-chemokines is IL-8 which is the most prevalent cytokine in ARDS studied cases (6)

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

Role of LUS in ARDS:

ARDS symptoms include spared areas, particularly in anterior regions & in the early stages of the disease, multiple B lines, typically with non-homogeneous, gravity-dependent distribution, pleural thickening, subpleural consolidations, decreased or abolished lung sliding, & lung consolidation in dependent lung regions.

The early diagnosis of acute respiratory failure states that the diagnosis of ARDS requires the existence of diffuse ultrasonography interstitial syndrome.

The US is correlated with an "A-line-obliterating" or "B-line" pattern. These A-line & B-line patterns are shown to be easily distinguishable by a bedside clinician utilizing LUS following brief training (8).

The LUS scoring method is currently widely employed in clinical settings. The two lungs were specifically separated into 12 regions, and each region was given a

score between 0 and 3 based on the quantity, distribution, and presence or absence of subpleural consolidation of B-lines. The LUSS is calculated as the sum of the graded results for these 12 locations (9).

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