



# Value of circulating neutrophil receptors in isolated chest trauma patients; can they predict ARDS?

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## Abstract:

**Background:** The dysfunctional immune system is one of the foremost reasons for trauma-related mortality. Severe thoracic injuries are related with serious life-threatening complications such as Acute Respiratory Distress Syndrome (ARDS) in spite of ongoing improvements in mechanical ventilation strategies and supportive care; in addition, the surgical intervention can intensify the condition. ARDS is distinguished by the activation of neutrophils and its recruitment into the alveolar space and interstitium of the lung. The exact pathogenesis of this inflammatory complication which follow chest trauma has varied etiologies and the mechanism is not entirely understood. The purpose of this investigation was to assess the neutrophil cells surface receptors expression in severe chest injury and its contribution to ARDS development. **Methods:** Blood samples were collected from fifty patients with severe isolated chest injury were examined for the neutrophil cell surface receptors expression profile at a varied interval within the initial 24 hrs after injury. Patients were followed for the occurrence of any inflammatory complications during this period. For comparison, 25 healthy subjects were included as a control group. **Results:** Seven patients developed inflammatory complication other than ARDS. Neutrophils showed diminished expression of L-Selectin and C5aR and their levels stayed low until 24 hrs after trauma while both CXCR1 and CXCR2 levels were gradually increased. Furthermore CD11b expression level increased at 3 hrs and then gradually decreased. Serum level of CXCL8/IL-8 and IL-6 were increased and reach maximum levels after 24 hrs. **Conclusion:** Activation of the circulating neutrophil is transient after isolated chest trauma and leads to a systemic inflammatory reaction to a degree not enough and needs another stimulus to cause ARDS.

## Keywords:

Chest trauma; Neutrophil; Surface receptor

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