Intramedullary Nailing Alters Pulmonary Neutrophil Deposition and Cell Surface Receptor Expression in Experimental Orthopaedic Trauma in Rats

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Purpose: Polymorphonuclear neutrophils (PMNs) are the most highly abundant immune cells in circulation. Excessive PMN deposition and activation in vital organs is associated with inflammatory complications such as acute respiratory distress syndrome (ARDS). Intramedullary nailing (IMN) for long bone fractures has been identified as a risk factor for the development of inflammatory complications. However, the impact of IMN on pulmonary neutrophil deposition and activation is unclear. We hypothesized that IMN is associated with temporary increased pulmonary neutrophil presence and activation.

Methods: A rat model including IMN and a femur fracture was utilized. Groups were terminated after 3, 7, and 14 days. Lung parenchymal and broncho-alveolar lavage fluid (BALF) PMNs were collected and analyzed by flow cytometry. Cell counts as well as membrane expression levels of CD11b, CD62L, and CD11a were compared between groups.

Results: Pulmonary neutrophil numbers were increased 3 days after insult. Additionally, cell surface expression levels of CD11b ($P < 0.01$) and CD62L ($P < 0.01$) on parenchymal neutrophils were found to be increased after 3 days of observation as well. A gradual restoration of neutrophil activation and numbers was observed thereafter. Activation status of the broncho-alveolar neutrophil pool differed significantly from their parenchymal counterparts.

Conclusion: We demonstrated that our standardized rat model is feasible to study long-term innate immune responses in the tissue compartment to trauma. This study further shows that IMN is associated with temporary increased pulmonary neutrophil deposition. Moreover, concurrent increased integrin and selectin receptor expression on lung neutrophils was found. This phenomenon suggests that integrin and selectin receptor dynamics modulate PMN pulmonary tissue homing. Upcoming studies should focus on the modulation of integrin and/or selectin signaling on neutrophils and the potential to interfere in lung neutrophil homing processes.

See the meeting app for complete listing of authors' disclosure information.