

EARLY OXIDATIVE AND INFLAMMATORY CHANGES OF HEART AND LIVER ASSOCIATED WITH PRIMARY ACUTE LUNG INJURY

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Background and aim: Acute lung injury (ALI) is characterized by diffuse lung damage, neutrophil-mediated inflammation, edema, and surfactant dysfunction leading to hypoxemia. Severe ALI accelerates progression of injury to other organs (heart, liver, kidneys etc.) what can worsen the patient's status. Whereas an indirect effect of ALI on extra-pulmonary organs is poorly known, this study evaluated oxidative and inflammatory modifications of heart and liver in two experimental models of ALI compared to healthy non-ventilated controls (C-NoVent) and healthy controls ventilated with oxygen (C-Vent).

Methods: ALI was induced in young rabbits by 1) intratracheal instillation of neonatal meconium (25 mg/ml, 4 ml/kg; Mec-ALI model), or by 2) repetitive saline lung lavage (30 ml/kg, 6-12 times; Lav-ALI model). Animals with both models of ALI and ventilated controls (C-Vent) were oxygen-ventilated for additional 5 h. Total and differential counts of blood leukocytes, concentrations of markers of inflammation (TNF α ; IL-6), oxidative damage to lipids (thiobarbituric acid reactive substances, TBARS) and proteins (3-nitrotyrosine, 3NT), and endothelial damage (receptor for advanced glycation end products, RAGE) in heart and liver homogenates were determined.

Results: In both ALI models, increased total leukocyte and neutrophil counts in the blood and elevated markers of inflammation, oxidative and vascular damage in the heart and liver were detected compared to controls.

Conclusion: In both models of ALI, early inflammatory, oxidative and endothelial changes were found, indicating deleterious indirect effect of ALI on extra-pulmonary organs.

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