題名:N-acetylcysteine attenuates acute lung injury induced by fat embolism

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摘要:OBJECTIVES: Fat embolism syndrome is a clinical issue in subjects with long-bone fracture. It may lead to acute lung injury. The mechanisms and therapeutic regimen remain unclear. The present study was designed to investigate the pathologic and biochemical changes after fat embolization in isolated rat lungs, and to test the effects of posttreatment with N-acetylcysteine (NAC). DESIGN: Prospective, randomized, controlled animal study. SETTING: University research laboratory. SUBJECTS: A total of 36 perfused lungs isolated from Sprague-Dawley rats. INTERVENTIONS : The isolated lungs were randomly assigned to receive physiologic saline solution (vehicle group), fat embolism (FE group), or FE with NAC posttreatment (FE + NAC group). There were 12 isolated lungs in each group. FE was produced by introduction of corn oil micelles. NAC at a dose 150 mg/kg was given 10 mins after FE. MEASUREMENTS AND MAIN RESULTS: The extent of acute lung injury was evaluated by lung weight change, protein concentration in bronchoalveolar lavage, and exhaled nitric oxide. We also measured the pulmonary arterial pressure and capillary filtration coefficient and determined the nitrate/nitrite, methylguanidine, tumor necrosis factor-alpha, and interleukin-1beta in lung perfusate. Histopathologic changes of the lung were examined and quantified. The levels of neutrophil elastase and myeloperoxidase were determined. The expression of inducible nitric oxide synthase was detected. FE caused acute lung injury as evidenced by the lung weight changes, increases in exhaled nitric oxide and protein

concentration in bronchoalveolar lavage, pulmonary hypertension, increased capillary filtration coefficient, and lung pathology. The insult also increased nitrate/nitrite, methylguanidine, tumor necrosis factor-alpha, and interleukin-1beta in lung perfusate, increased neutrophil elastase and myeloperoxidase levels, and upregulated inducible nitric oxide synthase expression. Posttreatment with NAC abrogated these changes induced by FE. CONCLUSION : FE caused acute lung injury and associated biochemical changes. Posttreatment with NAC was effective to alleviate the pathologic and biochemical changes caused by FE.