Angiotensin-converting enzyme inhibitor captopril attenuates ventilator-induced lung injury in rats

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摘要.

Abstract

We hypothesized that lung inflammation and parenchymal apoptosis in ventilator-induced lung injury (VILI) are related to ANG II and assessed the ability of the angiotensin-converting enzyme inhibitor captopril to attenuate VILI in rats. Adult male Sprague-Dawley rats were randomized to receive two ventilation strategies for 2 h: 1) tidal volume of 40 ml/kg, respiratory rate of 25 breaths/min, and inspiratory O2 fraction of 0.21 [high-volume, 0 positive end-expiratory pressure (HVZP) group] and 2) injection of captopril (100 mg/kg ip) 30 min before HVZP ventilation (HVZP + CAP group). Another group, which did not receive ventilation, served as the control. Mean arterial pressure was significantly lower in the HVZP + CAP group than in the HVZP group at 2 h of ventilation. Total protein levels were significantly higher in bronchoalveolar lavage fluid (BALF) recovered from HVZP-ventilated rats than from controls. BALF macrophage inflammatory protein-2 and lung ANG II were significantly higher in the HVZP group than in the control and HVZP + CAP groups. Lung ANG II levels correlated positively with BALF protein and macrophage inflammatory protein-2. The number of apoptotic airway and alveolar wall cells was significantly higher in the HVZP and HVZP + CAP groups than in the control group and significantly lower in the HVZP + CAP group than in the HVZP group. These results suggest that the efficiency of captopril to attenuate VILI is related to reduction of inflammatory cytokines and inhibition of apoptosis and indicate that VILI is partly mediated by the local angiotensin system.