機械通氣引發的肺傷害會增加大白鼠肺臟的血管收縮素

Ventilation-induced lung injury increases lung angiotensin II in rats

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摘要

我們假設機械通氣導致的肺傷害與肺臟的血管收縮素有關。成年公Sprague-Dawley老鼠隨機分爲二組，接受機械通氣兩小時：(1)高潮氣容積(40mL/kg)、零吐氣末陽壓組；(2)低潮氣容積(8mL/kg)、正吐氣末陽壓組(5cmH2O)。沒有接受機械通氣的老鼠當控制組。高潮氣容積、零吐氣末陽壓組的氣管肺泡灌洗液的蛋白含量及肺傷害指數顯著地比控制組和低潮氣容積、正吐氣末陽壓組高。接受機械通氣兩組的氣管肺泡灌洗液的巨噬細胞發炎蛋白質－2和肺臟血管收縮素顯著地高於控制組。所有研究老鼠的肺臟血管收縮素與氣管肺泡灌洗液的巨噬細胞發炎蛋白質－2數值呈正相關。這些結果表示局部血管緊縮素系統與機械通氣導致肺傷害的病理生理機轉有關，並且建議阻斷血管收縮素可能有治療機械通氣導致肺傷害的潛在功效。

Abstract

We hypothesized that lung injury and inflammation in ventilation-induced lung injury (VILI) are related to angiotensin (Ang) Ⅱ. Adult male Sprague-Dawley rats were randomized to receive two ventilation strategies for 2 hours: 1) the high-volume zero PEEP group (HVZP) was ventilated with a high tidal volume (40mL/kg) and zero positive end expiratory pressure (PEEP); 2) the low-volume with PEEP group (LVP) was ventilated with a low tidal volume (8mL/kg) and PEEP (5cmH2O). Another group which did not receive ventilation served as the control. Total protein in bronchoalveolar lavage fluid (BALF) was significantly higher in HVZP group than in the control and LVP groups. Rats treated with HVZP ventilation had a significantly higher lung injury score than did the control and LVP groups. BALF macrophage inflammatory protein-2 (MIP-2) and lung Ang Ⅱ were significantly higher in HVZP and LVP groups when compared with the control group. Lung Ang Ⅱ correlated positively with MIP-2 in BALF in all study rats. These results indicate that local angiotensin system is involved in the pathogenesis of VILI

and suggest that blockade of Ang Ⅱ might have potential therapeutic implications in alleviating VILI.