Cardiopulmonary, Histologic, and Inflammatory Effects of Intravenous Na2S After Blunt Chest Trauma-Induced Lung Contusion in Mice

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Résumé en anglais	Background: When used as a pretreatment, hydrogen sulfide (H2S) either attenuated or aggravated lung injury. Therefore, we tested the hypothesis whether posttreatment intravenous Na2S (sulfide) may attenuate lung injury.Methods: After blast wave blunt chest trauma or sham procedure, anesthetized and instrumented mice received continuous intravenous sulfide or vehicle while being kept at 37°C or 32°C core temperature. After 4 hours of pressure-controlled, thoracopulmonary compliance-titrated, lung-protective mechanical ventilation, blood and tissue were harvested for cytokine concentrations, heme oxygenase-1, IκBα, Bcl-Xl, and pBad expression (western blotting), nuclear factor-κB activation (electrophoretic mobility shift assay), and activated caspase-3, cystathionine-β synthase and cystathionine-γ lyase (immunohistochemistry). Results: Hypothermia caused marked bradycardia and metabolic acidosis unaltered by sulfide. Chest trauma impaired thoracopulmonary compliance and arterial Po2, again without sulfide effect. Cytokine levels showed inconsistent response. Sulfide increased nuclear factor-κB activation during normothermia, but this effect was blunted during hypothermia. While histologic lung injury was variable, both sulfide and hypothermia attenuated the trauma-related increase in heme oxygenase-1 expression and activated caspase-3 staining, which coincided with increased Bad phosphorylation and Bcl-Xl expression. Sulfide and hypothermia also attenuated the trauma-induced cystathionine-β synthase and cystathionine-γ lyase expression. Conclusions: Posttreatment sulfide infusion after blunt chest trauma did not affect the impaired lung mechanics and gas exchange but attenuated stress protein expression and apoptotic cell death. This protective effect was amplified by moderate hypothermia. The simultaneous reduction in cystathionine-β synthase and cystathionine-γ lyase expression supports the role of H2S-generating enzymes as an adaptive response during stress states.
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