Hyperbaric oxygen attenuation of lipopolysaccharide-induced acute lung injury involves heme oxygenase-1

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摘要

Abstract

Background: Hyperbaric oxygen (HBO) attenuates lipopolysaccharide (LPS)-induced acute lung injury. This beneficial effect of HBO involves inhibition of inducible nitric oxide synthase (iNOS) expression and subsequent nitric oxide (NO) biosynthesis. We sought to investigate the role of heme oxygenase-1 (HO-1) on this HBO inhibition of iNOS induction and acute lung injury in septic rat lungs. Methods: Before the experiment, 72 rats were randomly allocated to receive HBO or air treatment. With or without HBO pre-treatment, the rats were further divided into the following subgroups (n = 6): (i) LPS injection, (ii) normal saline (N/S) injection, (iii) hemin (a HO-1 inducer) plus LPS, (iv) hemin alone, (v) tin protoporphyrin (SnPP; a HO-1 inhibitor) plus LPS, and (vi) SnPP alone. All rats were maintained for 6 h and then sacrificed with a high-dose pentobarbital injection. Lung injuries and relevant enzymes expression were thus assayed. Results: Histological analysis, PMNs/alveoli ratio, and wet/ dry weight ratio measurements demonstrated that LPS caused significant lung injury and HBO and/or hemin significantly attenuated this LPS-induced lung injury. Increased pulmonary iNOS expression and NO production were associated with lung injury. Induction of HO-1, by HBO and/or hemin, significantly attenuated this LPS-induced iNOS expression and acute lung injury. SnPP, on the contrary, offset the effects of HBO and worsened the LPS-induced lung injury. Conclusions: HBO may act through inhibiting pulmonary iNOS expression to attenuate LPS-induced acute lung injury in septic rats. Furthermore, this HBO attenuation of iNOS expression involves HO-1 induction.