LIPID PEROXIDATION AND ANTIOXIDANT STATUS IN EXPERIMENTAL ACUTE LUNG INJURY

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Disrupted oxidant–antioxidant balance has a major role in the genesis of respiratory diseases (1), such as acute lung injury (ALI) or acute respiratory distress syndrome. In the present study we investigated changes of lipid peroxidation and antioxidant status in blood serum and in lung homogenates in the dynamics of experimental acute lung injury. Male adult rats weighting 200-230 gwere used for the experiments. For modeling of acid aspiration-induced ALI, anesthetized rats underwent tracheostomy and insertion of a fine-bore cannula into the anterior segment of the left lung. This was followed by the instillation of either 1 ml/kg HCI, pH 1.2 (n=6) or 1 ml/kg saline in control rats (n=6) (2). All animals were studied 6 h after acid instillation. Lung tissue homogenates and blood serum were analyzed for thiobarbituric acid-reactive species (TBARS) and superoxide dismutase (SOD) levels using spectrophotometry. We found that TBARS values were significantly higher in the acid-instillation group than those in the control group. The respective values were 4.8±0.1 *vs.* 3.3±0.2 mmol/l ($p<0.01$) in the serum and 7.8±0.5 *vs.* 4.4±0.3 mmol/kg ($p<0.01$) in the lung tissue homogenates. There were also increases in the SOD levels in the acid-instillation group compared with the control group; 51.4±1.2 *vs*. 49.6±1.0% ($p>0.05$) in the serum and 49.5±1.0 *vs.* 35.3±2.3% ($p<0.001$) in the lung homogenates, respectively. The appearance of products of lipid peroxidation in the serum and lungs suggests that TBARS are useful markers of oxygen-radical-mediated tissue injury. Activation of antioxidant protection in the first hours after injury has a compensatory nature and it is realized by increasing SODty activ.

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