N-ACETYLCYSTEINE ALLEVIATES THE MECONIUM-INDUCED ACUTE LUNG INJURY

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Meconium aspiration in the newborns causes lung inflammation and injury, which may lead to meconium aspiration syndrome (MAS). In this study, effects of antioxidant N-acetylcysteine on respiratory and inflammatory parameters were studied in an animal model of MAS. Oxygen-ventilated rabbits were intratracheally given 4 mL/kg of meconium suspension (25 mg/mL) or saline. Thirty minutes later, meconium-instilled animals were intravenously administered N-acetylcysteine (10 mg/kg), or were left without treatment. All animals were oxygen-ventilated for additional 5 hours. Ventilatory pressures, oxygenation, right-to-left pulmonary shunts, and white blood cell count were measured in regular intervals. At the end of experiment, trachea and lungs were excised. Left lung was saline-lavaged and total and differential counts of cells in the bronchoalveolar lavage fluid (BAL) were determined. Right lung tissue strips were used for detection of lung edema (expressed as wet/dry lung weight ratio) and peroxidation (expressed by concentration of thiobarbituric acid-reactive substances, TBARS). Lung tissue strips and tracheal strips were used for measurement of airway reactivity to bronchoconstriction mediators. In the final plasma, eosinophil cathionic protein (ECP), TBARS, and total antioxidant status were determined. Meconium instillation induced polymorphonuclear-derived inflammation and oxidative stress. Treatment with N-acetylcysteine significantly improved oxygenation, reduced lung edema formation, decreased number of polymorphonucleras in the BAL fluid, and diminished peroxidation and meconium-induced airway hyperreactivity compared with non-treated animals. Concluding, N-acetylcysteine effectively improved the lung functions in animal model of MAS.

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