EVALUATION OF APOPTOSIS IN EXPERIMENTAL MODELS OF RESPIRATORY FAILURE

S. Balentova[1], P. Kosutova[2], H. Pistekova[2], L. Tomcikova[2], M. Adamkov[1], A. Calkovska[2] and D. Mokra[2]

[1]Institute of Histology and Embryology, Jessenius Faculty of Medicine in Martin, Comenius University in Bratislava, 036 01 Martin, Slovakia, balentova@jfmed.uniba.sk

[2]Institute of Physiology, Jessenius Faculty of Medicine in Martin, Comenius University in Bratislava, 036 01 Martin, Slovakia

Objective: Inflammation and oxidative stress are important pathological features of newborns. The aim of our pilot study was to approve if ongoing inflammatory and oxidative changes contribute to apoptosis of lung tissue in two experimental models of respiratory failure: acute respiratory distress syndrome (ARDS) and meconium aspiration syndrome (MAS).

Methods: Model of experimental ARDS was performed with adult rabbits (New Zealand white) by repeated lavage with saline (30 ml/kg), until PaO_2 decreased to <26.7 kPa in FiO_2 1.0 (n=5). In model of MAS, the rabbits were intratracheally administered of meconium suspension (4 ml/kg b.w., 25 mg/ml; n=5) and the third group were represented by healthy controls (n=5). From the moment of full-filled criteria of respiratory failure (induction of ARDS and MAS), animals were ventilated for additional 4 hours. Total and differential white blood cell (WBC) count was determined in blood sample taken at the end of experiment. After sacrifice and excision, left lungs were lavaged by saline (3x10 ml/kg) and in the bronchoalveolar lavage (BAL) fluid was determined total, differential number of cells and their viability. Right lungs were used for estimation of wet/dry weight ratio, apoptosis of lung cells according TUNEL method and other apoptotic markers (caspase-3 etc.).

Results: Both of models of respiratory failure led to increased apoptosis of lung cells and inflammatory markers, however, MAS model displayed more expressive changes. **Conclusions:** Inflammation and oxidative stress in applied models of respiratory failure are responsible for increased apoptosis of lung cells.

Acknowledgement: APVV-0435-11, VEGA 1/0305/14, VEGA 1/0050/11, UK/201/14.