Lung function

Anti-oxidation and anti-inflammatory benefits of combinated surfactant and N-acetylcysteine treatment of experimental meconium-induced respiratory failure

P. Mikolka[1], *D. Mokra[1], J. Kopincova[1], P. Kosutova[1], A. Calkovska[1]
[1]Jessenius School of Medicine, Comenius University, Biomed and Department of Physiology (Martin, Slovakia)

Aim: In severe meconium aspiration syndrome (MAS), *i.t.* application of exogenous surfactant (S) is used. However, surfactant is inactivated by meconium-induced inflammation and oxidation. To reduce surfactant inactivation and enhance effectiveness of therapy, *i.v.* administration of N-acetylcysteine (NAC) followed delivery of *i.t.* surfactant in experimental model of MAS.

Methods: Anesthetized rabbits with meconium-induced respiratory failure were divided according to the therapy (n=6 each group) to: non-treated (M), treated with monotherapy (M+S, M+NAC) and combined therapy (M+S+NAC), or controls with saline instead of meconium (C), and were ventilated for 5 hours after therapy. After sacrificing the animals, lung edema (wet/dry weight ratio), oxidative damage markers (TBARS, 3-nitrotyrosine, 3NT), and interleukins (IL-2, -6, -13, TNFα) using ELISA and RT-PCR in lung homogenates were determined.

Results: M+S+NAC therapy had superior effect to monotherapies on W/D ratio ($p<0.05$), TBARS ($p<0.01$), 3NT ($p<0.001$), and IL ($p<0.05$) compared to untreated group. Reduction of IL expression was equal to M+S ($p<0.01$ *vs.* M).

Conclusion: N-acetylcysteine combined with exogenous surfactant may reduce lung edema, oxidative damage, levels and expression of cytokines in lung of animal model. This therapy may prevent surfactant inactivation and contribute to better respiratory functions and outcome of patients with MAS.

Supported by: BioMed Martin (ITMS 26220220187), VEGA 1/0291/12, VEGA 1/0305/14, APVV-0435-11.