European Respiratory Society
Annual Congress 2013

Abstract Number: 6069

Publication Number: PP150

Abstract Group: 1.1. Clinical Problems

Keywords: no keyword selected

Title: LSC 2013 abstract - Coagulation factor IX deficiency does not afford protection from bleomycin–induced pulmonary fibrosis in mice

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Body: Introduction: Evidence from animal and human studies show the role of the coagulation cascade in acute and chronic lung injury. We hypothesized that the absence of coagulation Factor(F)IX, which is essential for the initiation, amplification and propagation phases of the coagulation cascade would reduce fibrosis development and progression. Methods: We used the murine model of bleomycin-induced pulmonary fibrosis in wild-type (WT; n=14) and FIX deficient mice (FIX ko; n=13). After 14 days, we assessed the markers of tissue fibrosis, inflammatory cell influx in the bronchoalveolar lavage fluid (BALF), and cytokines levels in the BALF, blood and lung homogenate of the animals. Results: Mortality during the experiment was higher in the FIX ko compared to WT mice (23% versus 7%). The remaining FIX ko (n=10) developed pulmonary fibrosis to a degree similar to WT mice (n=13). There was no significant difference in the Ashcroft score between WT and FIX ko mice (4.011±0.4 versus 4.2±0.4), in the alpha-actin score (0.94±0.09 versus 0.70±0.07) and in the inflammatory cell number. In contrast, we observed a higher pulmonary hemorrhage score in the FIX ko mice, as well as significant elevations in levels of cytokines IL-10, IFNγ, 12 and IL-6 in their plasma. Conclusions: our results suggest that FIX deficiency is detrimental in pulmonary fibrosis. In line with the results of Noth et al, our results suggest to proceed with caution when using anticoagulation as a treatment for IPF. Noth et al Am J Respir Crit Care Med. 2012 Jul 1;186(1):88-95.