Category :**Sepsis: basic mechanisms**

A93 - ***Effects of xenon on proinflammatory activation and apoptosis of human neutrophils (ex vivo study)***

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Introduction:

The aim of the study was to evaluate the effect of xenon on the activation of human neutrophils in ex vivo conditions

Methods:

After receiving informed consent, 10 healthy volunteers had their blood drawn twice: before xenon inhalation (xenon - 30 vol.%, oxygen - no more than 40 vol.%, the rest - nitrogen) and immediately after. The duration of inhalation in all patients was 60 minutes. After neutrophil isolation from the patient's serum, lipopolysaccharide (LPS) was added to the 4 million/ml cell concentrate at a dose of 200 ng/ml. The effect of xenon on the severity of inflammatory activation of neutrophils was assessed by the level of expression of CD11b and CD66b adhesion molecules on their surface and phosphorylation of pro-inflammatory kinases: ERK1/2 and kinase-p38

Results:

The addition of lipopolysaccharide to the neutrophil incubation medium caused their activation, significantly increasing the phosphorylation of the key pro – inflammatory kinases of neutrophils: ERK1/2 and kinase-p38. After xenon anesthesia, there was a decrease in the expression of CD11b and CD66b adhesion molecules on the neutrophil surface and reducing the phosphorylation (activation) of pro-inflammatory kinases: ERK1/2 and MAP-kinase p38, which demonstrated its anti-inflammatory effect. The addition of LPS to the neutrophil incubation medium reduced their ability to spontaneous apoptosis 22 hours after isolation, which was 22,6%, which was 60% less than in the control group-56,3% ($p<0,05$). Xenon inhalation significantly increased to 41,35%, ($p<0,05$). increased the ability of neutrophils to spontaneous apoptosis after incubation with LPS

Conclusion:

Inhalation of xenon 30 vol% for 60 minutes has a pronounced anti-inflammatory effect on neutrophils, reducing their activation by inhibiting pro-inflammatory kinases: ERK1/2 and MAP-kinase p38, reducing the expression of activation markers CD11b and CD66b on the surface of neutrophils and increasing their ability to spontaneous apoptosis