SUMMARY:
Managment of endothelial disfunction at pulmonary endothel induced by iskemia
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Aim: In this study, the effects of L-arginin and N- asetylsystein on demaged pulmonery endothelial cell by hypoxia at the experimentaly hypoxia created human pulmonery endothelial cell medium.

Methods: By using HEPE cell medium the groups acured when it cvered the % 70-80 of the grourd of the wells at flask. By edding L- arginin and N- N-acetylsystein the groups are leaved in % 5 CO_2 that provides normal situation and % 20 CO_2 that provides hypoxic situation to incubate for four hours. At the end of 4 hours the flasks are taken from incubator to laminer cabin. 500 µL $5x10^5$ PMN been prepared from humen blood before added on them and incubated for 30 minutes % 5 and % 20 CO_2 incubator. At the end of the 30 minutes, the flasks are taken from incubator and under the microscope, te apopitosis/normal rate and; adhesion of PMN to endothelial cell which painted with Giemsa are seen. The volues of TNF-α are read at the 450 nm wau lenghet fotometriciy by ELİSA.

Statisticaliy method: Apopitosis and adhesion values within each group and betueen groups ate evaluted by one sideol ANOVA and the effects of L-arginin on two groups are evaluateol by using student test. The results that's p values are below 0,05 are not accepted stastistically meaninful.

Findings: In the study especially in the % 5 CO_2 group there wasn't meaninful at apopitosis when % 20 CO_2 edded, widespred cell death found in the control group and N-acetylsystein group. It has been doserved that L- aginin prevented the cell death in both groups. Alt in he control group in wthich % 5 CO_2 added, L- arginin at 30 µM dose mode the similr adhesion like the control group, at 100 µM dose it has mode a statisticcally not meaninful decrease in adhesion. At 30 µM dose of N- acetylsystein, compared to control group there vas a stastistically important increase in adhesion $p< 0,05$. When % 20 CO_2 edded to control and N- acetylsystein groups, there coldn't make a statisticcally evaluattion becase at widespred cell death. In both doses L- aginin mode an statisticcally not important increase in adhesion. Both in % 5 CO_2 and % 20 CO_2 odded there wasn't a statisticcally important difference betwen TNF-α by the dnegs used.

Results: According to the resulb of this study; when ischemia been mode; it has been fourd thet L- aginin preventth pulmonary endothelial cell deathe and increases adhesion independentty from doses. Immune nutricion can supply support at pulmonary endothelial

cells. In the presens of ischemia; N- acetylsystein con’t show the expected protective functions. Advanced studies are needed in order to show that what may ischemia cause at the pumonary endothelial cell at the presence of infection the effects of it on albumin Leucocyte permeability.

Key words: L-arjinin, N- acetylsystein, iskemia, pulmonar endothelal cell, cell culture, adhesion, apopitosis, TNF-α

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