

**SUMMARY:**

Management of endothelial dysfunction at pulmonary endothel induced by ischemia  
Metin ÖZTÜRK. MD.

**Aim:** In this study, the effects of L-arginin and N- acetylsystein on damaged pulmonary endothelial cell by hypoxia at the experimentaly hypoxia created human pulmonary endothelial cell medium.

**Methods:** By using HEPE cell medium the groups acured when it cvered the % 70-80 of the ground of the wells at flask. By edding L- arginin and N- N-acetylsystein the groups are leaved in % 5 CO<sub>2</sub> that provides normal situation and % 20 CO<sub>2</sub> that provides hypoxic situation to incubate for four hours. At the end of 4 hours the flasks are taken from incubator to laminar cabin. 500 µL 5x10<sup>5</sup> PMN been prepared from humen blood before added on them and incubated for 30 minutes % 5 and % 20 CO<sub>2</sub> 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 fotometriciycy by ELİSA.

**Statisticaliy method:** Apopitosis and adhesion values within each group and between 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 meaniful.

**Findings:** In the study especially in the % 5 CO<sub>2</sub> group there wasn't meaniful at apopitosis when % 20 CO<sub>2</sub> 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<sub>2</sub> 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 meaniful 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<sub>2</sub> edded to control and N- acetylsystein groups, there coldn't make a statisticcally evaluattion becace at widespred cell death. In both doses L- aginin mode an statisticcally not important increase in adhesion. Both in % 5 CO<sub>2</sub> and % 20 CO<sub>2</sub> odDED there wasn't a statisticcally important difference between TNF-α by the dnegs used.

**Results:** According to the resulb of this study; when ischemia been mode; it has been found that L- aginin preventth pulmonary endothelial cell deathe and increases adhesion independtly 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- $\alpha$

**Correspondence address:** Metin ÖZTÜRK. MD.

ADÜ Faculty of Medicine Department of Anesthesiology and Reanimation, AYDIN