Category 1 :**ARDS**
Category 2 :**Sepsis - other**

A103 - ***Genetic variants of intron region of aquaporin aqp5 gene and development of pulmonary edema in lung infection complicated by septic shock***

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Introduction:

Product of AQP5 gene belongs to a family of aquaporins (AQPs), membrane proteins, responsible for the selective transmembrane transport of water. However, the value of polymorphic variants AQP5 in the development and progression of pul monary edema in severe lung infection was studied so far. The aim of the investigation was to determine the value of genetic variants of a single nucleotide polymorphic site rs3736309 of intron 3 of aquaporin 5 (AQP5) gene in the course of critical illness in patients with documented pulmonary infection.

Methods:

Patients with critical illness admitted to the intensive care units were examined during the course of treatment (n=86, age 27 to 82 years, mean age 53.20±14.34 years). Main diagnosis included malignancies (15%), peritonitis (16%) and necrotizing pancreatitis (37%). Patients developed nosocomi al pneumonia (55%), acute respiratory distress syndrome (ARDS) (54%), septic shock (48%), ARDS combined with septic shock (33%). DNA genotyping was carried out using tetra primer polymerase chain reaction (PCR). Statistical processing was performed using GraphPad InStat program (GraphPad, USA).

Results:

The distribution of frequencies of genotypes AA, GA and GG (AQP5, rs3736309) in cohort of patients corresponded to Hardy Weinberg equilibrium (P=0.923) and was similar to frequencies of the alleles determined in healthy Caucasian individuals (literature data) (P>0.05). In a subgroup of patients with septic shock and AQP5 AA (rs3736309) genotype the lower EVLWI values were found compared to patients with geno types GG and GA with septic shock in spite of the same approach to treatment. Genetic variant AQP5 G+ (rs3736309) contributed to the development of pulmonary edema resistant to treatment (odds ratio, OR = 6,75; P=0.032). Only the subgroup of patients with septic shock and geno type G + (but not all patients or the subgroup of patients without septic shock of the same genotype) were char acterized by significantly elevated levels of surfactant protein SP D in plasma compared to patients of genotype AQP5 AA with septic shock (P<0.05).

Conclusions:

In septic shock, the presence of homozygous variant allele A (AA) of AQP5 rs3736309 is a favor able factor for patients developing the pulmonary edema. The presence of allele AQP5 G (rs3736309) is a risk fac tor for developing severe pulmonary edema and unfavorable prognosis in spite of treatment.