



ACUTE CORONARY SYNDROME IS A CLINICAL PICTURE OF CORONARY HEART DISEASE

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Annotatsiya: *As a result of the study, the possibility of predicting the outcomes of acute coronary syndrome is presented, since the determination of protein growth factors (CRP, C3 and VEGF) in dynamics makes it possible to prevent the development of acute myocardial infarction.*

Keywords: *acute coronary syndrome, myocardial infarction, growth factors, cytokines*

Relevance: coronary artery disease is a consequence of morph functional instability of atherosclerotic plaque, causing occlusive thrombosis or thromboembolism of the coronary artery with the formation of necrosis of the corresponding myocardial vascularization basin.

In this regard, it is extremely important to have a reliable personalized prognosis of clinical outcomes of ACS even at the hospital stage, since the choice of patient management protocols, as well as the optimization of methods and means of treatment depends on it.

The purpose of the study: to study cytokines and growth factors in ACS.

Material and methods of the study: The reliability of the differences in the mean values was evaluated on the basis of the Student's criterion (t) with the calculation of the probability of error (P) when checking the normality of the distribution and the equality of the general variances (F – Fisher criterion).

The results of the study and their discussion.

Acute ST segment elevation in MI, both anterior and diaphragmatic, is most common in men aged 54-59 years.

In the studied group of patients with ACS, mortality was 11.2%, while in the group of patients with MI with a Q wave; it was 10%, with a significant frequency of anterior myocardial lesion and blockage of the Gas bundle leg in men.

The research found an increase in the level of IL-1 to $83,8 \pm 1,3$ PG/ml in patients with ACS transition to aim, to $90.5 \pm 1,71$ PG/ml in patients with ACS transformation of the PS against the indicators of control- $72,8 \pm 0,76$ PG/ml ($P < 0.01$).

Concentrations of IL-10-like anti-inflammatory cytokine showed a decrease in the transformation of ACS in aim to $15.6 \pm 0,5$ PG/ml ($P < 0.001$), and the transition AUX in substation has its rise to $44.3 \pm 0,59$ PG/ml ($P < 0.001$). Vascular Endothelial Growth Factor (VEGF)-activator of angiogenesis, responsible for restoring oxygen supply to tissues in a situation where blood circulation is insufficient. In physiological concentrations, endothelia (ET) acts on endothelial receptors, causing the release of relaxation factors, in higher concentrations it activates receptors on smooth muscle cells, stimulating persistent vasoconstriction primarily at the level of microcirculation.



The constrictor activity of endothelin-1 can

to be a factor of vasospasm enhancement, closing the vicious circle of path biochemical reactions and aggravating cerebral ischemia.

In our studies, the study of VEGF concentration showed a sharp increase, regardless of the clinical form of ACS transformation, to 147.92 ± 1.5 and 90.2 ± 1.0 relative to the control- 57.50 ± 1.0 in patients in critical condition with AMI and PS, respectively.

The established phenomenon indicates vascular inflammation during the first 3 days in patients of the observed group. At the same time, a higher concentration of VEGF, as well as its increase in dynamics, indicates the state of vasoconstriction and the risk of developing AMI.

C-reactive protein (CRP) is an acute phase protein, a sensitive indicator of tissue damage in inflammation, necrosis and trauma. It is a marker in acute phases of various inflammatory processes. It is absent in the blood serum of a healthy person.

Protein is synthesized in the liver. IL-6, IL-8 and TNF- α regulate the formation process. The concentration of CRP in the blood has a high correlation with the activity of the disease, the stage of the process.

In the absence of obvious causes, a slight increase in CRP indicates chronic subclinical inflammation of the vascular wall. This can be a factor in cardiovascular catastrophes: atherosclerosis, heart attack, thromboembolism and stroke. The informative value of the CRP index determined by highly sensitive methods is higher in this regard than the determination of low-density lipoprotein cholesterol. The risk of cardiovascular complications in patients with increased CRP increases with parallel increased other risk factors: cholesterol, fibrinogen, homocysteine, etc. CRP reflects the severity of the immune-inflammatory process, correlating with markers of T-cell activation, and can serve as an indicator of the activity of the disease, the severity of coetaneous and pulmonary fibrosis.

In patients with ACS, a statistically significant increase in its level was found to 5.7 ± 0.06 mg/l with the development of AMI ($P < 0.001$) and to 8.90 ± 0.19 mg/l with the development of PS ($P < 0.001$) versus the control - 1.78 ± 0.09 mg/l.

The established significant increase in the level of CRP in ACS indicates chronic subclinical inflammation of the vascular wall. At the same time, an increase in CRP was noted depending on the clinical form of ACS transformation: a progressive increase in its level from 3.2 times in AMI to 5.0 times in PS.

The complement system is a cascade system of proteolysis enzymes for humeral support of the body and maintenance of homeostasis. The complement consists of 11 serum proteins, which make up 9 components of the complement (C1-C9). C3 and C5 are involved in the regulation of the inflammatory reaction.

In the study, patients with ACS showed an increase in the level of C3 by 1.63 times (55.5 ± 0.9 ng/ml, $P < 0.01$) in AMI and by 1.84 times (62.8 ± 1.19 ng/ml, $P < 0.01$) in PS, against the control - 34.2 ± 1.02 ng/ml, which indicates the activation of the inflammatory process in the body.

Conclusions: The study of growth factors and immunity parameters in ACS in the first hours allows predicting the outcome. At the same time, IL-10 acts as an indicator for predicting the transformation of ACS into AMI. By studying protein growth factors (CRP, C3 and VEGF) in dynamics, it is possible to prevent the development of AMI.