THE LEVEL OF CYTOKINES IN THE BLOOD OF PATIENTS WITH ST-ELEVATION ACUTE MYOCARDIAL INFARCTION

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Inflammation plays a key role in the pathogenesis of atherosclerosis, and is involved, on the one hand, in the genesis, development, rupture, and repair of atherosclerotic plaque and, on the other, in post-reperfusion damage, remodeling, and scarring of myocardial tissue. It is also known that an exacerbated inflammatory state plays a role in the development and progression of heart failure.

Tumor necrosis factor alpha (TNF-α) is an inflammatory cytokine synthesized in various blood, endothelial and smooth muscle cells, and in cardiac myocytes. The ubiquity and function of its receptors provide it with the capacity to modulate a diversity of inflammatory processes which are strongly involved in acute coronary syndrome (ACS) and in the development of heart failure due to its negative inotropic action, among others. Several lines of research have indicated that it is an independent predictor of mortality in patients with heart failure and advanced functional class, as well as in the chronic phase of myocardial infarction (MI).

Objective. To study the dynamic of inflammatory mediators – cytokines in ST elevation myocardial infarction (STEMI).

Materials and methods of research. 111 patients with STEMI aged 37 to 88 years were studied. During hospitalization heart failure (HF) was observed in 70 patients (63,1%), according to Killip classification: class I – in 41 patients (36,9%), class II – in 57 (51,4%), class III – in 7 patients (6,3%). HF persisted 21 days after AMI in 63 patients (56,7%). The content of TNF-α, IL-1β, IL-4, IL-6 (pg/ml, M ± m) cytokines was evaluated on day 1, 7 and 21. Experimental group was composed of 20 patients with stable angina pectoris (SAP) and a control group (CG) – of 22 patients. There were no significant differences in gender or age between groups.

Results of research and their discussion. The baseline concentration of TNF-α cytokine in patients with STEMI was 569,6 ± 54,2 pg/ml, that was higher than in patients with SAP and control group ($p < 0,001$). The concentration of TNF-α in group SAP was 57,5 ± 11,3 pg/ml and 40,2 ± 9,6 pg/ml – in control group. It significantly decreased to 66,8 ± 36,5 pg/ml ($p = 0,0001$) and had no significant differences from other groups on day 7. On day 21 TNF-α – content was the same. The baseline concentration of IL-1β was higher in patients with STEMI 550,3 ± 84,2 pg/ml in comparison to group with SAP 216,6 ± 11,3 pg/ml and controls 145,6 ± 12,6 pg/ml ($p < 0,01$). On day 7 the content of IL-1β did not change 609,8 ± 98,6 pg/ml, and it significantly decreased 417,8 ± 71,6 pg/ml ($p = 0,006$) on day 21. The content of IL-1β was significantly higher in patients with STEMI throughout the observation period as compared to other groups ($p < 0,01$). The baseline concentration of IL-4 in STEMI patients was higher 200,0 ± 39,4 pg/ml than in group with SAP 77,7 ± 27,5 pg/ml and CG 68,9 ± 24,4 pg/ml ($p < 0,01$). On day 7 and 21 it remained high and was 129,6 ± 38,3 pg/ml and 179,4 ± 80,1 pg/ml, respectively. The baseline concentration of IL-6 in patients with STEMI was higher 200,0 ± 39,4 pg/ml, than in patients with SAP 59,9 ± 9,6 pg/ml and control group 50,7 ± 8,4 pg/ml ($p < 0,001$). It significantly decreased on day 7 and 21 to 90,1 ± 21,2 pg/ml and 72,1 ± 16,2 pg/ml ($p = 0,001$, $r = 0,02$), respectively, that was significantly higher as compared to other groups during the observation period.

Conclusions. Indicators characterizing inflammation sharply increased on the first day of STEMI and persist in acute and subacute STEMI period, which probably indicates their impact on the progression of CHF.

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