Markers of myocodial fibrosis in acute myocardial infarction

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One of the leading mechanisms in the healing process of acute myocardial infarction (AMI) is damage and loss of extracellular matrix, which plays a leading role in the pathogenesis of LV myocardial remodeling. Tissue inhibitor of matrix metalloprotease-1 is a protein that is a natural inhibitor of metalloproteases, forms complexes with MMP-1 and other metalloproteases (they destroy components of the extracellular matrix of the heart), irreversibly inhibiting their activity. Bayes-Genis A. et al presented histological data on the expression in ruptured and eroded plaques of plasma protein A (PAPP-A), metalloproteinase and insulin-like growth factor-1 detected during pregnancy. Myocardial formation of aldosterone and angiotensin II (as a result of activation of the renin-angiotensin-aldosterone system, including the tissue component) after acute myocardial infarction stimulates the formation of collagen and fibrous tissue in the myocardium with further changes in the structure and geometric characteristics of the left ventricle.

Purpose of the study. To study the markers of interstitial myocardial fibrosis in the acute stage of myocardial infarction and assess the dynamics of these indicators in patients after 2.5-3 weeks of inpatient treatment.

Methods. The study included 31 patients with myocardial infarction with ST segment elevation. The control groups consisted of 20 apparently healthy individuals. During the study, serum parameters of tissue inhibitor of matrix metalloproteinase-1 (TIMMP-1 and aldosterone were measured on days 1-3 after AMI and after 2.5-3 weeks of inpatient treatment.

Results. The level of TIMMP-1 in patients with acute MI was 418.19 ± 103.77 ng / ml and was statistically significantly higher than in practically healthy individuals - 103.44 ± 7.06 ng / ml (p <0.0001). The level of TIMMP-1 in patients with AMI after 3 weeks, against the background of ongoing therapy, was 366.70 ± 93.34 ng / ml, and the decrease in TIMMP-1, compared with the initial value in the acute stage, had a statistical significance (p = 0.046).

The level of aldosterone in acute myocardial infarction was 165.12 ± 32.67 pg / ml and was statistically significantly higher (p <0.0001) than in the control group. In patients with myocardial infarction who are hospitalized, for 2.5-3 weeks, there is a statistically significant (p = 0.003) decrease in the concentration of aldosterone in blood plasma by 15.1% from 165.12 ± 32.67 pg / ml to 140.24 ± 22.78 pg / ml.

Conclusions. In the acute stage of myocardial infarction (1-3 days), there is a significant increase in the concentrations of markers of interstitial myocardial fibrosis, such as tissue inhibitor of matrix metalloproteinase-1 and aldosterone, which reflects the initiation of fibroblast activation processes and, accordingly, stimulation of collagen synthesis. Subsequently, there is a decrease in the concentration of tissue inhibitor of matrix metalloproteinase-1 and aldosterone.