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IMMUNOLOGICAL STATE OF ORGANISM WITH ACUTE ISHEMIC HEART DISEASE

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Development of myocardial infarction(MI) evolves sensitization of lymphocytes towards myocardial antigen and formation of antibodies against tropomyosin and actin. MI induces infiltration of immune cells even in remote non-infarcted myocardium. The autoimmune mechanism plays an important role in ventricular remodeling after MI.

We aimed to compare immunocompetent cell activity in patients with MI, unstable angina(UA) and chronic coronary disease(CCD). Macrophage – lymphocyte interaction plays an essential role both in cell-mediated and antibody-mediated immune reactions.

We investigated activity of immunocompetent cells according to macrophage-lymphocyte rosette (MLRos) formation in patient's blood leukocyte cultures. Our previous studies showed that amount of MLRos increases in cases of allergy and autoimmune diseases revealing the presence of any type of immune conflict in organism and decreases in cases of acute viral hepatitis and sepsis reflecting a low immune reactivity of organism (Shvelidze T., Saralidze T., Saralidze N.). We studied 15 patients with ST-elevation MI who did not undergo reperfusion therapy because of different reasons twice: after 24 hours and after 10 days of MI onset, 15 patients with unstable angina (UA), 10 with past STEMI, 8 patients with stable angina of exertion(SA).

MLRos in vitro was increased in patients with STEMI. In the acute stage, after 24 hours it was 46,3±1,4% and after 10 days it reached 59,1±3,9% (as in immune-sensitized patients), while in healthy donors it is 37,4±2,2%. In cases of UA the amount of MLRos was in the normal range 33,2±11,2%. It also increased in patients with past STEMI (even 1-3 years after MI) - 58,0±6,5%. In patients with SA it remained in normal range. Increase of MLRos after STEMI, especially after 10 days underlines increased activity of monocytes and lymphocytes, and therefore immune reactivity of organism which is caused by contact of cardiac antigens with immunocompetent cells and immune sensitization, that remains during years after MI. On the contrary, the normal range of MLRos in patients with UA and SA points that immune sensitization to cardiac antigens didn't occur.

Results of our study shows that immune sensitization to myocardial antigen is characteristic of myocardial infarction rather than ischemia without necrosis. Persistent immune activation after STEMI may contribute to post-infarction ventricular remodeling and heart failure. Therefore regulation of immune function can be considered as a new target in the treatment of MI. It's reasonable to use the method of MLRos formation in vitro for estimation of organism's immune reactivity in patients with IM in a dynamic way to improve long-term outcomes.

Keywords: myocardial infarction, angina, macrophage-lymphocyte rosettes