Expression of bax protein and morphological changes in the myocardium

February 27 2017

The expression of Bax protein, marker of intracellular pathway of apoptosis initiation, in viable left ventricular cardiomyocytes and morphological changes in the myocardium in acute pressure overload of the left ventricle were studied in experiment on male rabbits. The content of Bax protein in the cardiomyocyte cytoplasm decreased, this indicating that the mitochondrial pathway was not involved in the realization of the apoptotic program. This decrease was associated with manifest destructive changes in the left ventricular myocardium.

The study was carried out on male Chinchilla rab- bits (3-3.5 kg) distributed into 4 groups, 4 per group: control (intact animals) and 3 experimental (rabbits with acute LV overload after 1, 3, and 5 days).

The animals were kept and handled in accordance with the Order No. 755 of the Ministry of Health of the USSR (August 12, 1977), and the European Convention for protection of Vertebrates Used in Experiments or with Other Research Purposes (Strasbourg, 1986). Pressure overload of LV was created surgically by narrowing grid. The proportion of equally distant points occupied by positively stained cardiomyocyte cytoplasm to the total count of points occupied by the cytoplasm was calculated.

Morphometric analysis of the myocardium was performed on histological sections stained with he- matoxylin and eosin. The content (vol%) of muscular fiber, cardiomyocyte nuclei, foci of destruction and infiltration, and volume of extracellular space were measured under a

microscope at ×400 in 30 visual fields using Avtandilov grid. The nucleus/cytoplasm ratio, i.e. the ratio of cardiomyocyte nuclei to muscular fiber was calculated (in %).

Our previous study of LV cardiomyocyte apop- tosis on an identical experimental model showed an increase in activities of effector caspase-3 and initia- tory caspase-8 mediating realization of apoptotic cell death by the external pathway. The decrease in Bax protein expression detected in the present study indicated that initiation of apoptotic processes in LV cardiomyocytes in acute LV overload did not depend on the mitochondrial pathway and was caused mainly by the receptor-mediated signal mechanisms.

Hence, acute LV pressure overload is associated with lesser Bax expression protein in LV cardiomyo- cytes combined with manifest morphological changes in the myocardium most pronounced on day 5 of the process

Provided by RUDN University