ISCHEMIC AND REPERFUSIVE RELEASE OF THE ENDOGENOUS PURINES AND ITS INFLUENCE ON THE MYOCARDIAL VIABILITY DURING -ADRENERGIC BLOCKADE

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The aim of this study was to estimate ischemic and reperfusive release of myocardial adenosine degradation products (MADP) during -adrenergic blockade and its relation to infarct size (IS) and viable myocardium size (VM).

In a group of 24 shepherd-mongrel dogs, randomly assigned to a metoprolol (M-) and placebo-group (P-group), occlusion of the left anterior descending coronary artery (LAD) followed by reperfusion with recombinant tissue plasminogen activator was performed. Regional myocardial blood flow (MBF) was measured by the radiolabelled microsphere technique. Blood samples from aorta and great cardiac vein were collected to evaluate the concentrations of MADP. The triphenyltetrazolium chloride perfusion and fixation technique was used for infarct size measurement. MBF in the area at risk decreased in both groups during ischemia, but it was significantly higher (p = 0.013) in M-group. Recanalization of LAD was associated with an increase in flow in postischemic vascular bed. MBF was significantly higher (p = 0.024) in M-group during late reperfusion. In M-group IS was smaller (p = 0.007) and VM was bigger (p = 0.007). The correlation between arterial adenosine concentration during early reperfusion and IS (p = 0.44, r = -0.588) or VM (p = 0.036, r = 0.607) in M-group was noted. Values of net MADP balances significantly increased during early reperfusion. The correlation between reperfusive net MADP balance and IS (p = 0.00005, r = 0.906) or VM (p = 0.016, r = -0.675) in M-group was observed.

The amount of MADP released during reperfusion correlates with the IS and is inversely proportional to the area of VM. The endogenously released adenosine may have additional cardioprotective effect during -adrenergic blockade.

Key words: myocardial infarction, viable myocardium, reperfusion, ischemia, preconditioning, -adrenergic blockade, adenosine, inosine