Reaction of Myocardial Mast Cells during Neoangiogenesis Induced by YAG:Nd Laser

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Time course of morphofunctional changes in myocardial mast cells of Wistar rats irradiated with a YAG:Nd laser is studied. Mast cells were detected on serial sections by toluidine blue staining. A drastic increase in mast cell degranulation around the laser channel was observed on days 1-20 of experiment. Mast cell degranulation may be the first stage in neoangiogenesis in the myocardium in response to laser injury.

Key Words: *mast cells; neoangiogenesis; transmyocardial revascularization of the heart*

Laser transmyocardial revascularization of the heart (TMR) used in distal coronary insufficiency is a prospective surgical method for treating coronary disease. This short intervention rapidly improves blood supply to ischemic myocardium due to formation of new capillaries in it. Clinical effectiveness of TMR is well established; however, the mechanism of neoangiogenesis is unknown. The recovery of myocardial blood supply after TMR may be associated with additional blood supply to ischemic myocardium: blood ejected from left-ventricular cavity is delivered into the channels produced by a laser [1,3,5,12,13]. The probability of neoangiogenesis in response to laser radiation has been neglected [2,8].

We studied the process of stimulated neoangiogenesis in response to TMR and tried to explain this phenomenon from the viewpoint of cell-to-cell interactions in surgical damage to the myocardium.

MATERIALS AND METHODS

Experiment was carried out on 50 adult Wistar rats weighing 150-180 g. The myocardium was irradiated with a YAG:Nd laser (λ=1.06 μm, 10 Wt, light guide diameter 0.3 mm, duration 1-2 sec). The rats were anesthetized with calypsol. After skin incision and preparation of the chest, 1 or maximally 2 channels through the entire thickness of left-ventricular myocardium were formed with laser light guide oriented to the apical pulse.

For morphological analysis of the myocardium and mast cells (MC), the animals were sacrificed 1 h, 1, 3, 5, 7, 10, and 20 days after the operation.

Heart tissue was fixed in 10% neutral formalin infused through the abdominal aorta. Serial myocardial sections were stained with hematoxylin and eosin to visualize the injury and characterize the processes in the laser channel and in adjacent myocardium. Mast cells were stained with toluidine blue at pH 2.0. The number of MC and the ratio of intact to degranulated MC were determined along the entire new channel and in the adjacent intact zones of the myocardium.

RESULTS

Morphological study of transmyocardial channel and adjacent zones of injured and intact myocardium showed that the channel was immediately filled with blood. Then fibrin threads formed and neutrophilic leukocytes accumulated in the adjacent zones of the myocardium. After 2-3 weeks the new channel was obliterated and replaced with granulation tissue. The bulk of the cells were fibroblasts of different shape. In general, the reaction of the myocardium to high-

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performance YAG:Nd laser can be regarded as an inflammatory process with slightly expressed exudative and pronounced proliferative phase.

Examination of the myocardium at different periods revealed formation of capillaries by days 5-7. By day 30, new small arteries of muscular type were seen in the damaged zone.

Comparison of morphological changes in the myocardium and morphofunctional characteristics of mast cells it showed degranulation of MC adjacent to the new channel, starting from the first hours after TMR. On days 1-21 after TMR, the number of degranulated MC decreased at sites distant from the focus of injury.

Formation of new blood vessels in laser channel is an element of adaptive reaction improving blood supply to the ischemic zone.

Mast cells were located along capillaries in the thickness of the myocardium and subepiendocardially. Laser light destroys MC and activates their degranulation. This creates the prerequisites for stimulation of local neoangiogenesis: production of proteases, arachidonic acid derivatives active toward vascular endothelium, heparin and heparan sulfate [4,6,7], and stem factors (basic fibroblast stem factor and platelet stem factor) from MC granules [10,11]. Stimulation of MC degranulation was observed in zones adjacent to the channel formed by a laser. We think that this phenomenon develops and is maintained due to local thermal effect created by a laser beam. This is confirmed by the presence of numerous degranulating MC in the area adjacent to the transmyocardial channel. A similar effect was observed in rat mesentery. Mast cell degranulation occurred in a 3-5 mm laser and mechanical wound of the root of the tongue in rats [9]. Laser produced the most pronounced effect. Cooling of the rat tongue surface before laser irradiation inhibited degranulation of MC. This fact demonstrates the significance of thermal factor in laser-induced MC degranulation. It should be stressed that MC degranulation lasts 20-21 days after TMR. This phenomenon is long-lasting, because local laser exposure triggers an inflammatory process with strong proliferative phase. We believe that the inflammatory process induces local production of interleukin-1 that induces MC and causes their degranulation [10]. Long duration of MC degranulation can be explained by slowly fading local inflammation, maintaining degranulation of these cells.

Therefore, activation of MC degranulation by local laser exposure is a factor stimulating neoangiogenesis in the myocardium in TMR. Degranulation of MC and subsequent stimulation of neoangiogenesis can be regarded as a component of adaptive reaction of the myocardium to laser-induced injury.

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