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Acute myocardial infarction: biological role of postinfarction tissue

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Abstract

This study aimed to analyze morphological changes of postinfarction tissue during the restorative period after acute myocardial infarction (AMI), which, in our opinion, can change the perception of AMI in terms of its biological significance.

Methods. Objects of the study were cardiac biopsy specimens from 27 AMI patients who died in the hospital within 7 to 34 days after the onset of AMI, as well as heart biopsy specimens from 8 people aged 45 to 61 years old who deceased due to an unnatural cause (car accident) and whose autopsies revealed large foci of postinfarction tissue. The deceased patients had history of coronary heart disease (CHD) and arterial hypertension (AH).

Results. During the autopsy we diagnosed extensive intramural myocardial infarctions localized in the anterior-lateral walls of the left ventricle with the infarction zone spreading to the apex and the anterior part of the interventricular septum. All deceased patients had a severe atherosclerotic lesion in left anterior descending artery; thrombotic masses were revealed in the upper third of coronary artery in 19 deceased patients (11 men and 8 women). Postinfarction fibrous tissue was detected at the infarction site on average by 29 to 30 days after the onset of AMI. The special feature was that its fibers were oriented parallel to the preserved muscle fibers, and the remains of the preserved muscle tissue fibers, breaking on the border with the infarction site, were continued by the fibers of newly formed postinfarction connective tissue. There was a small amount of glycogen and oxidation-reduction enzymes present in the postinfarction tissue, which were also present in the preserved cardiomyocytes.

Conclusion. Thus, according to the results obtained, AMI is a compensatory-adaptive process aimed at correcting the anatomical-physiological mismatch between

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hypertrophied myocardium and the state of coronary vessels affected by atherosclerotic process in postnatal (often late) ontogenesis.

Key words: myocardial infarction, regeneration, compensatory-adaptive process, vascular atherosclerosis.

Conflict of interest: none declared.

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Introduction

Myocardial infarction (MI) is one of the clinical forms of coronary heart disease, and is characterised by an ischemic necrosis of myocardial area due to atherosclerotic vascular lesions [1, 2]. Due to prolonged ischemia of myocardial area, a focus of necrosis with leukocyte shaft formation occurs [3]. The pathogenesis of acute myocardial infarction (AMI) is based on the following mechanisms: destruction of atherosclerotic plaque, due to sudden increase of sympathetic nervous system activity (as a result of elevated blood pressure (BP), sudden increase of HR and intensification of coronary circulation) [1, 2]. The thrombus formation in the area of ruptured atherosclerotic plaque [3, 4] and vasoconstriction are also key components. Analyzing AMI morbidity and mortality [5, 6], the authors have concluded that after acute stage of the disease and in rehabilitation period — the greatest importance is given to the state of preserved myocardial tissue, and the role and state of postinfarction area is often neglected.

The aim is to analyze morphological changes occurring in postinfarction tissue during the restorative period after AMI, which, in our opinion, can change the perception of AMI in terms of its biological significance.

Methods

Objects of the study were cardiac biopsy specimens from 27 AMI patients who died in the hospital within 7 to 34 days after the onset of AMI (14 males, 13 females), as well as heart biopsy specimens from 8 people aged 45 to 61 years old (5 men, 3 females) who died due to an unnatural cause (car accident) and whose autopsies revealed large foci of postinfarction tissue (without confirmed AMI history). The deceased patients had history of coronary heart disease (CHD) and arterial hypertension (AH).

For histopathology, 10 slices were cut from the infarct areas and at the border with the preserved muscle tissue. The prepared paraffin sections (6 μm) were stained with standard hematoxylin and eosin, van Gieson and Heidenhain's iron hematoxylin. Part of the material was examined for redox enzymes: tetrazolium method to detect succinate dehydrogenase, lactate dehydrogenase and NAD-diaphorase [1].

Significance relative to control: Mann-Whitney U criterion ($p < 0.001$).

The study was conducted in accordance with the standards of Good Clinical Practice and the principles of the Declaration of Helsinki.

Results

Autopsy diagnosed the extensive intramural myocardial infarctions localized in the anterior-lateral walls of the left ventricle with infarction zone spreading to the apex and the anterior part of the interventricular septum. All deceased patients had a severe atherosclerotic lesion in left anterior descending artery; thrombotic masses were found in the upper third of coronary artery in 19 deceased (11 men and 8 women).

The healing process of AMI went according to the classical course described in the available literature. The formation of postinfarction tissue drew our attention.

During the muscle detritus resorption, many different cellular elements gradually accumulated in the necrotic focus. These included macrophages and lymphocytes as well as a significant number of spindle cells containing oval or oval elongated large nuclei with soft clumps of chromatin (unlike coarse, clumpy chromatin of fibroblast nuclei) evenly distributed along the nucleus. The cytoplasm of such cells was stained pale pink. According to morphological parameters, such cells corresponded to myoblasts. Specific large nuclei with serrated chromatin

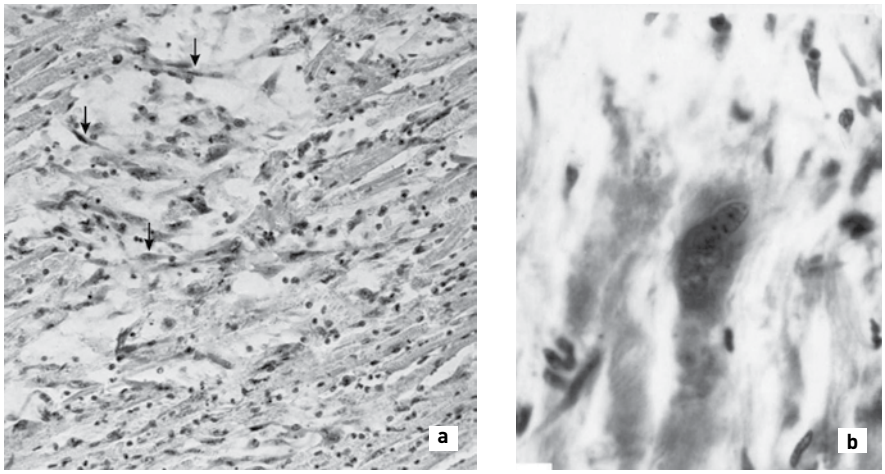

Fig. 2. The pattern of mutual arrangement of muscle fibers and postinfarction tissue. Autopsy data: continuation of the fibers of the newly formed tissue by the stumps of muscle fibers and their parallel course to the preserved muscle fibers. Van Gieson staining, x 400

arrangement were detected, which corresponded to the criterion of cardiomyocyte mitosis. A small ring of cytoplasm, which was stained pale pink with eosin, was often detected around such nuclei, which gave them similarity to myoblastic-like structures.

Postinfarction fibrous tissue was detected at the infarction site on average by 29 to 30 days after the onset of AMI. The special feature was that its fibers were oriented parallel to the preserved muscle fibers, and the remains of the preserved muscle tissue fibers, breaking on the border with the infarction site, were continued by the fibers of newly formed postinfarction connective tissue.

Due to the significant cardiomyocyte hypertrophy in the myocardium, the formation of large foci of tissue arisen in the place of the dead myocardium was observed. When analyzing the newly formed tissue, low-differentiated myogenic elements — myoblasts and muscle buds along with the connective tissue elements participated in its formation (Fig. 1). The structure of the mutual compound of the preserved muscle and fibers of the newly formed postinfarction tissue was noteworthy — the stumps of the muscle fibers at the border with the postinfarction tissue were cut off and continued by the fibers of the newly formed postinfarction tissue. At the same time, its fibers continued their course, parallel to the preserved muscle fibers (Fig. 2). The postinfarction tissue showed a small amount of glycogen and redox enzymes, which were also present in the preserved cardiomyocytes.

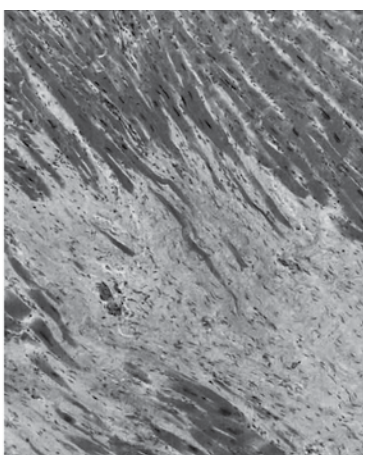

Fig. 1. Low-differentiated muscle elements in the forming postinfarction tissue: a) muscle bud at the border with postinfarction cardiosclerosis, x Immersion; b) myoblasts in the zone of postinfarction tissue formation. Hematoxylin and eosin staining, x 400

Discussion

It is known from the literature that in the majority of cases, people who have suffered an AMI gradually improve their state of health [1-5], haemodynamics stabilise and electrophysiological parameters normalise. The majority of people, especially young and middle-aged people, return to work. The explanation for this is the restoration of functional activity of the heart due to regenerative hypertrophy (intracellular regeneration). In our opinion, taking into account the results of the present work, the restoration of cardiac activity is associated with the development of a specific tissue at the infarct site.

There are reports in the literature that connective tissue is involved in the contractile activity of a number of muscular organs: skeletal muscle [7, 8], in the heart during diastole [5]. Data from an experimental study showed the contraction of tissue formed in place of dead myocardium as a result of ligation of the descending coronary artery.

The ability of the connective tissue to contract was explained by the fact that it contains myofibroblasts — cells containing smooth muscle myosin with a contractile apparatus. Especially noteworthy is the discovery of a specific protein in fibroblasts called caldesmon, which is similar in structure to the tro-

pomyosin complex and is involved in the interaction between actin and myosin in heart and skeletal muscle [7].

In the studied heart biopsy specimens, a peculiar tissue appeared in the place of the dead heart muscle, with the main characteristics of a fibrous component, but with the participation of the heart muscle elements in its formation. A kind of myofibrous tissue with the ability of contractile activity was formed. When studying histological preparations of the deceased heart, there was an evidence that the fibres of the newly formed tissue, being a continuation of the stumps of muscle fibres, continued their parallel course, participating in the contractile activity of the heart. Indirect evidence of this is the presence in the newly formed tissue of all the components present in cardiomyocytes (glycogen, redox enzymes).

During the contractile activity of the normal heart, the interstitial connective tissue contracts together with the muscle fibres. On this basis, it is difficult to imagine that post-infarction tissue remains intact outside the contractile rhythm. If this is the case, the role of myocardial infarction as a pathological process is questionable.

Several factors contribute to the aetiology of AMI: atherosclerotic lesions of the coronary arteries and hypertrophic changes in the left ventricular myocardium. In our opinion, the occurrence of AMI in these conditions can be regarded as a compensatory adaptive process aimed at eliminating the discrepancy between hypertrophic myocardial mass and altered vessels incapable of providing adequate blood supply to the myocardium. After AMI, the dead tissue is replaced by undemanding tissue that is resistant to hypoxia and, at the same time, participates to some extent in the contractile activity of the heart. Thus, the formation of post-infarction myofibrous tissue in the heart leads to an optimisation of the ratio between pathologically altered vessels and left ventricular muscle mass.

Conclusion

On the basis of the evidence presented here, indicating the potential involvement of post-infarct tissue in cardiac contractility, AMI is a compensatory and adaptive process designed to correct the anatomical and physiological mismatch between the hypertrophied myocardium and the state of the coronary arteries affected by the postnatal atherosclerosis process.

Conflict of interest: none declared.

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