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Review article

Myocardial remodeling as an important mechanism in the development of Chronic Heart Failure

Yuan Cao[1], Pradeep Depark[1], Deepark Sharma[1] and Yanmin Xu[1,2]*

[1]*Tianjin Key Laboratory of Ionic-Molecular Function of Cardiovascular disease, Department of Cardiology, Tianjin Institute of Cardiology, Second Hospital of Tianjin Medical University, People's Republic of China.*

[2]*Institute of cardiovascular Tianjin PR China, No 23 ping jiang road of HE XI district Tianjin, China.*

***Corresponding author:** Yanmin Xu, Tianjin Key Laboratory of Ionic-Molecular Function of Cardiovascular disease, Department of Cardiology, Tianjin Institute of Cardiology, Second Hospital of Tianjin Medical University, People's Republic of China.

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Introduction

Heart failure (HF) is a leading cause of late morbidity and mortality after acute myocardial infarction worldwide. Nearly half of the patients with heart failure are associated with coronary heart disease. The treatment of chronic heart failure has changed from emphasis on improving symptoms and quality of life to focus on prevention and delay of myocardial remodeling, reducing mortality and readmission rate. Myocardial remodeling is an important mechanism in the development of chronic heart failure. Development of the HF phenotype in these patients arise from a complex, progressive, molecular and cellular transformation called "ventricular remodeling" first described by Tenant and Wiggers. Ventricular remodeling includes dilation of the ventricle, the formation of scar and geometrical charges in the overall left ventricle (LV) shape (i e, ellipsoid to more spherical). In this paper, according to the treatment guidelines and large-scale research results, the research progress of myocardial remodeling is summarized as follows.

Early Cellular Changes in myocardial remodeling

Remodeling depends on infarct size, remodeling begins with an acute infarct, leading to myocardial injury and death. But involves a progressive group of changes that occur in both infarcted and non-infarcted myocardia. Early changes can be seen within hours to days of an acute myocardial injury. Myocardial necrosis result in an influx of inflammatory cells, including macrophages and other antigen-presenting cells. These processes occur early, about 3-4 day, in the development of an acute MI. the influx of these inflammatory cells leads to be the destruction of the collagen scaffolding that helps to maintain ventricular shape. Leading to regional thinning and dilation of the myocardium in the infarcted areas. During this period, fibroblasts are also directed to the site of myocardial injury and begin to deposit new a collagen matrix that contributes to scar formation in the immediate post-infarct period.

Late Cellular Changes in myocardial remodeling

Over the following weeks to months, the viable myocardium undergoes a series of changes. Principally, given increased load on the non-infarcted myocardium, myocytes undergo eccentric hypertrophy, further leading to LV cavity dilation. these processes are initially compensatory and aimed at preserving cardiac output in response to infarcted myocardium and the resulting non-compliant scar formation. Over time, these changes increase LV size, which causes increasing wall stress and further dilation. these process lead to increase in LV end-systolic and end-diastolic volumes, increase preload –dependent myocardial oxygen demand, and may ultimately promote increased areas at risk for ischemia. Progressive dilation leads to further hemodynamic consequence, including the formation of possibly both ischemic and functional mitral regurgitation, which have been previously reviewed, as LV preload increases without the subsequently ability to generate sufficient myocardial contractility due to the thinning of the myocardial wall, end-systolic volumes rise and result in a depression of LV ejection fraction (EF). These processes are central to the development of ischemia-driven dilated cardiomyopathy.

Ventricular remodeling predicts of heart failure

Ventricular remodeling is a predictor of heart failure, an increase of at least 20% of left ventricular end-diastolic ventricular volume (LVEDV) from the first post infarction imaging. as the

first imaging study with cardiac magnetic resonance is usually performed a few days after myocardial infarction, early ventricular remodeling, which is the phase of remodeling that occurs in the first hours after myocardial infarction, could not be recognized, leading to an underestimation of the final ventricular dilatation. Also, a kind of compensatory adaptive response of the body, which changes the original shape and function of the heart due to various damage factors. After myocardial injury, the ventricular load increased, at this time, the heart function is still within the physiological range, or there is an imperceptible reduction, and the clinical manifestations and activities are not obvious. This stage is in the subclinical stage of cardiac insufficiency. With the continuous deposition of intercellular glia, the wall of the ventricles is becoming thicker, and the function of the heart is decreasing. The ventricles can't shoot out the normal amount of blood to meet the normal needs of the body, and the clinical manifestations of cardiac dysfunction are gradually emerging. The patients showed decreased exercise tolerance, panting after exercise and even limited lying down, sitting upright and breathing. In the process of myocardial remodeling, the ventricular structure and function gradually changed, the ventricular ejection capacity decreased, and the ventricular ejection capacity further decreased after the relative closure of valves caused by the enlargement of cardiac cavity. Especially in the case of mitral valve closure, when the whole heart is enlarged, the mitral valve is seriously and relatively incomplete, and the serious regurgitation of the mitral valve further reduces the ejection efficiency, which cannot meet the normal perfusion of the body. The symptoms of decreased peripheral perfusion were more obvious in patients with end-stage heart failure.

The character of left ventricular remodeling

Left ventricular remodeling is characterized by a progressive increase in both end-diastolic (LVEDV) and end-systolic volumes (LVESV). The increase in LVESV can precede the increase in LVEDV, as a consequence of an impaired systolic function that causes a reduction in stroke volume. The imaging modalities used to noninvasively assess ventricular volumes and function are echocardiography, radionuclide ventriculography, and cardiac magnetic resonance (CMR). A reduction in left ventricular ejection fraction (LVEF) is often observed during post infarct remodeling, predicting heart failure and increased mortality. Ventricular remodeling accompanies different heart diseases, such as dilatative non ischemic cardiomyopathy and cardiac hypertrophy in chronic hypertension and implies a change in myocardial anatomical structure. Post infarct remodeling is a specific type of left ventricular remodeling that is a consequence of an increase in both preload and afterload causing an enlargement of ventricular chamber and a hypertrophy of normal myocardium. The increase in preload is sustained by the phenomenon of infarct expansion, which is an enlargement of infarct scar. This causes a regional increase in the ventricular volume subtended by the expanded infarcted myocardial wall. In infarcted myocardium, ventricular contraction is not symmetrical, because the necrotic segments have lost their contractility, as a result, the force generated by the normal remote myocardium during contraction is not counterbalanced by an equal and opposite force, and the infarcted ventricular wall is thus stretched by an increased wall tension that is not homogeneously distributed in the left ventricle. Infarcted wall usually has longer contraction times than the healthy remote myocardium. This wall motion defect has been recognized as a risk factor for the development of remodeling, and it can be assessed with echocardiography or cine CMR. To maintain a normal, stoke volume with a reduced number of normally working myocardial segments, the healthy myocardium has to produce a greater pressure. The increase in workload (afterload) on healthy myocardium causes a hypertrophy of cardiomyocytes. In post infarct ventricular remodeling, hypertrophic cardiomyocytes are longer than normal cardiac cells. Post infarct ventricular remodeling was characterized by a lengthening of cardiomyocytes, especially in the area surrounding the infarct scar, but also in remote myocardium. This type of ventricular hypertrophy has been termed eccentric and contributes to the worsening of ventricular dilatation during remodeling.

Cardiac hypertrophy that occurs during post infarct remodeling is accompanied by an increase in extracellular matrix, which is mainly constituted by collagen. This phenomenon is due to an increased activity of cardiac fibroblasts in response to different soluble fibrogenic mediators, such as transforming growth factor-α (TGF-α) and systemic and local activation of renin-angiotensin aldosterone system (RAAS). The mediators of the RAAS that promote ventricular remodeling are angiotensin II and aldosterone. Remodeling is a pathologic process that involves the entire ventricle, leading to a change in its global structure. There are two types of causes of remodeling: mechanical and biochemical. While mechanical causes, as previously described, are an increase in both preload and afterload, biochemical causes are linked to the production of soluble mediators capable of promoting ventricular remodeling. This causes a regional increase in the ventricular volume subtended by the expanded infarcted myocardial wall. In infarcted myocardium, ventricular contraction is not symmetrical, because the necrotic segments have lost their contractility, as a result, the force generated by the normal remote myocardium during contraction is not counterbalanced by an equal and opposite force, and the infarcted ventricular wall is thus stretched by an increased wall tension that is not homogeneously distributed in the left ventricle. Cardiac hypertrophy that occurs during post infarct remodeling is accompanied by an increase in extracellular matrix, which is mainly constituted by collagen.