International Journal of
Science Inventions Today
www.ijsit.com
ISSN 2319-5436
Review Article

UTILIZATION OF CARDIAC MRI TECHNIQUE IN VENTRICULAR REMODELING AFTER MYOCARDIAL INFARCTION

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ABSTRACT

Ventricular remodeling after myocardial infarction significantly increases the mortality of heart failure, so the intervention of ventricular remodeling after myocardial infarction becomes a hot spot in the treatment of coronary artery disease. Early detection of ventricular remodeling is important, and in recent years, cardiac MRI, as a tool for assessing the heart, is becoming a common the method of "one-stop" multi-parameter technique can be used to check the macroscopic shape, cardiac function, hemodynamics and the degree of ventricular remodeling is evaluated by the microscopic structure of the order of cardiac fibers.

Keywords: Ventricular remodeling; Magnetic resonance technique; Myocardial infarction

INTRODUCTION

Ventricular remodeling after myocardial infarction refers to the size, quality and geometry of the heart state and the change of cardiac function in the response to myocardial injury. Studies have shown that acute survival rate of myocardial infarction increased significantly [1]. But long-term prognosis after myocardial infarction still poor, ventricular remodeling after myocardial infarction can cause a series of arrhythmia changes in health and cardiac function lead to high death rate and early intervention of myocardial infarction, ventricular remodeling after death is a hot and difficult problem in the treatment of coronary heart disease. Cardiac Magnetic Vibration (CMR) has gradually become a routine examination of heart disease from earlier clinical trials search method [2]

1. Pathological changes of ventricular remodeling

Macroscopic reconstruction: the main morphological change of ventricular remodeling is infarct expansion, myocardial hypertrophy and overall dilatation of the ventricles in non-infarcted areas. A large number of myocardial cells in the infarcted area apoptosis and deletion, under the pressure of the ventricle, the weak scar is easily pulled and lead to ventricular wall thinning, expansion, and even the formation of left ventricular aneurysm. Cause different degrees of heart dysfunction and hemodynamic changes, including myocardial contractility, incoordination, and contractility decrease in cardiac output, heart rate, or arrhythmia. Instead of infarcted areas should be hemodynamic response to overload, maintenance of cardiac function, and myocardial reactive thickening, elongate. Furthermore, increased ventricular pressure during diastole induces an increase in myocardial cell length, causing the heart to increase with the increase of cavity volume, the radius of the heart cavity increases and the stress in the ventricular wall increases left ventricular normal, oval shape loss, and spherical degeneration.Myocardial infarction can lead to changes in heart size, mass, and geometric shape, eventually the decrease of cardiac function is mainly caused by ejection fraction, ventricular volume and peak filling rate, peak systolic rate, stroke volume, cardiac output, and myocardial mass change.

Microscopic reconstruction: the microscopic reconstruction after myocardial infarction mainly manifested as early infarction focal myocardial coagulation necrosis, myocardial interstitial hyperemia, edema, inflammation, infiltration of the cells, followed by microvascular damage and calcification, and proliferation of fibroblasts in the later stage (about 6 weeks), the necrotic myocardial fibers gradually dissolved and absorbed, forming muscle foci and granulation tissue.Rather than myocardial infarction, hypertrophy and apoptosis, fiber row column derangement, vascular regeneration and interstitial collagen hyperplasia are the main mechanisms of late stage remodeling, ultimately, it can lead to heart failure and death. Ventricular remodeling is a mechanism after myocardial infarction, the process of systemic immune repair is accompanied by a series of inflammatory reactions that can lead to myocardial fibrosis in the infarcted and non-infarcted areas.

2. Evaluation of ventricular remodeling after myocardial infarction using common techniques of cardiac magnetic resonance imaging

MRI: is a valuable diagnostic tool for its use continuous measurement of 3D-FIESTA sequence can provide new insights into the progress of ventricular remodeling solution. For the evaluation of the overall cardiac function, the SSFP sequence of the light blood film is currently available the techniques used can clearly show ventricular wall motion, ventricular shape, and other messages the quantitative parameters of ventricular function can be obtained by software post-processing, such as ventricular volume, myocardial mass, ejection fraction, cardiac output, and so on, reflect the heart completely functional status. Compared with ultrasound, MRI has high temporal and spatial resolution the overlap algorithm is used to delineate the volume of ventricle directly without geometric hypothesis, the reliability of the ventricular volume measurement is assured, while the echocardiogram will be applied to the left ventricle assuming geometric ellipsoid is calculated in the myocardial infarction, left ventricular aneurysm and heart when the muscle hypertrophy, the ventricles are irregular shape, there is a big error, and the level of choice the subjective influence of the operator is greater. Of ventricular dysfunction and ventricular remodeling cardiac MRI is of greater diagnostic value [3].

Changes in cardiac function parameters and diffusion tensor imaging based on magnetic resonance measurements (DTI) imaging techniques show the evolution of the arrangement of the cardiac fiber bundle and the ventricular weight the basis of the pathological changes is consistent and can be used as ventricular weight after myocardial infarction predictive means of structure. During the ventricular remodeling, the functional indexes decreased frequently. Cardiac function parameter number is an important index to evaluate ventricular remodeling, filling rate peak and end systolic volume and increased end diastolic volume, in which ventricular diastolic volume increased by more than 20%, considered left ventricular remodeling was evaluated with peak filling rate. Protti and so on [4] think left ventricular end systolic volume > 32 ml is a good predictive indicator of poor remodeling combined with infarct size > 36%, better reconstruction of late stage adverse reconstruction is possible. The left ventricular mass index increased significantly at 1 weeks after MI, 6 months, and left the ventricular mass index decreased but increased after 1 years, suggesting a late compensatory left ventricular hypertrophy also indirectly reflects the dynamic changes of ventricular remodeling.

MRI can also evaluate ventricular remodeling from local wall changes trellis labeling of anterior magnetic resonance imaging is a reference standard for evaluating regional myocardial function. By gridding the myocardium, we can make a three-dimensional analysis of the rotation, strain, displacement and deformation of the local myocardium. Special software has been used to empty the grid dynamic analysis of changes in the myocardium allows measurements of regional myocardial motion strain and strain rates are considered. The study considers left ventricular remodeling and ventricular myocardium after myocardial infarction the change in wall stress is relevant.

Myocardial perfusion:

Magnetic resonance myocardial perfusion was performed using a T1 weighted sequence to display gadolinium contrast agent's secondary myocardial changes are mainly used to detect ischemic myocardium, ischemia, or the infarcted myocardium shows signs of reduction. Myocardial perfusion imaging has become the focus of clinical application is not limited to the qualitative diagnosis of the naked eye after the post-processing, the perfusion curve is obtained, and the image is semi quantitatively evaluated myocardial activity can only be assessed, and microvascular obstruction can also be assessed.

Microvascular obstruction can predict poor left ventricular remodeling and acute myocardial infarction posterior cardiac events [5]. Both infarcted and peripheral myocardium are present after acute myocardial infarction the diastolic dysfunction of coronary arteries is different, and the non-infarcted area is capillary compensatory vasodilation, and the remodeling of these microvasculature may contribute to the decline of cardiac function exhausted development. It is well known that the extent of stenosis determined by coronary angiography is comparable with that of coronary angiography incomplete perfusion of myocardial perfusion. Watkins and other [6] studies showed that the magnetic resonance load heart sensitivity and specificity of muscle perfusion in the diagnosis of functional coronary artery disease 91% and 94%, respectively, for the evaluation of revascularization, treatment and prognosis it has important clinical value. Several studies have shown that microvascular changes occur after myocardial infarction and infarction, the distance may be poor ventricular remodeling index [7, 8]. Wrong the infarct zone presents temporary vascular dilatation dysfunction at 1~2 weeks after MI, late myocardial infarction also leads to ventricular remodeling in [9]. if there is microvascular obstruction plug prompt patient prognosis is poor, Wu et al [10] believe that even after infarct size is controlled, microvascular status remains a powerful prognostic marker.

Delayed enhancement of myocardial magnetic resonance:

Myocardial magnetic resonance delayed enhancement (DE-MRI) is used to detect myocardial tissue in patients with coronary heart disease the important method of activity has become a parameter to evaluate the formation of myocardial scar after myocardial infarction standard. At present, patients with acute and chronic myocardial infarction have a large range of myocardial infarction according to ECG and echocardiogram. However, ECG DE-MRI can only be rough it was speculated that the transmural and infarct size could not be displayed in the infarct site, and there was no Q the rate of myocardial infarction is also high, which cannot be clearly diagnosed by ECG based on the response of the myocardial wall movement, the infarct is not clearly indicated the extent of the myocardium is limited by ventricular function. However, large magnetic resonance imaging, any angle, good spatial resolution, and high resolution of soft tissue identify any site and different degrees of myocardial infarction. Animal experimental study the size and morphology of the delayed myocardial infarction were demonstrated and histologically examined very close. The size, shape, volume, and histology of the delayed enhancement zone of the myocardium infarct size was highly correlated.

DE-MRI parameters such as transmural thickness, segment number and location, infarct size and microvascular obstruction are closely related to ventricular remodeling, increasing the left ventricle reliability of reconstructed prediction [11].

The transmural extent of delayed enhancement is related to the degree of coronary stenosis, which is directly related to prognosis, can predict functional recovery after revascularization status and myocardial reserve [12]. Studies have shown that if MRI is on the heart the muscle fiber scar involves the wall thickness more than 50%, then carries on the blood vessel reconstruction to contract the possibility of functional recovery is small, [13]. Tarantini and other [14] studies also found transmural walls the amount of muscle necrosis is a major determinant of left ventricular remodeling and function. And heart the location of myocardial infarction is related to ventricular remodeling, and the ventricular structure changes in patients with inferior myocardial infarction the change was significant and the systolic function was significantly reduced by [15].

In addition, infarct size can also be used to assess ventricular remodeling independently. Kim and other [16] studies suggest that sensitivity and specificity are 95% when assessing ventricular remodeling by dividing the left ventricular area by 23%. In the study of Tarantini and [14], the sensitivity, specificity and coincidence rate were 92%, 93% and 93%, respectively, when the cut-off value was set to 24%. Moreover, the chance of reconstruction increases by 2.8 times with an increase of 10% of the infarct size. However, infarct size is affected by the risk of myocardial infarction product range (AAR) effect, and use T2 and delayed enhancement magnetic resonance AAR and myocardial infarction area were measured by AAR. The degree of correction of myocardial infarction area that myocardial salvage index (MSI), is considered to be one of the main factors, independent adverse left ventricular remodeling (OR=0.64; 95% confidence interval: 0.49~0.84 P, =0.001 [17]).

Myocardial diffusion tensor imaging:

Magnetic resonance diffusion imaging (MRI) is a noninvasive imaging method for evaluating microscopic diffusion motion of water molecules in living tissues. The change of myocardial fiber direction and diffusion index can be obtained.The myocardial fibers generally go for sub endocardial myocardial longitudinal arrangement, medial oblique arrangement and epicardial circular arrangement. Myocardial fiber tractography provides a new perspective and imaging method for studying myocardial remodeling after myocardial infarction. Rubenstein [18] by SE-DTI and FSE-DTI two methods showed that the myocardial fibers running, and histological observation of the coincidence rate were 95% and 86%, direction DTI can even observe the myocardial fiber tissue substitute. In the process of ventricular remodeling, the arrangement of myocardial fibers will change greatly, and the arrangement of intact myocardial tissue in infarcted zone, adjacent region and distal part is different. It shows that the arrangement of myocardial fibers is disorder, thickening and even fracture, and the degree of disorder of fiber arrangement is closely related to the prediction of survival and risk factors.

The diffusion index includes anisotropic fraction (FA), average apparent dispersion coefficient (ADC), axial diffusion coefficient [lambda (parallel)], radial diffusion (lambda (vertical)) and fiber helix angle. The DTI parameter changes dynamically with time after infarction. The apparent diffusion coefficient in the infarct zone was lower than that in the non-infarct zone and increased with time. The anisotropy fraction was higher in the infarct zone and was greatest at day 28, which was attributed to the development of collagen fibrils in the structure. The percentage of left hand spiral fibers in the infarct area is closely related to infarct size and predicts ejection fraction. This technique may help us understand the structural relevance of functional remodeling after myocardial infarction.

Cardiac magnetic resonance spectroscopy:

Cardiac magnetic resonance spectroscopy (MRI) is a noninvasive imaging method for assessing myocardial metabolism. Imaging with self MRI signals can obtain basic information about myocardial metabolism. The study of cardiac magnetic resonance spectroscopy mainly for 1H and 31P nuclear spectrum determination, the 31P spectra of the majority, mainly for the early decrease of PCr and iP increased, CPr/APT ratio decreased. A large number of experimental studies have shown that myocardial PCr levels are reduced in patients with chronic heart failure, and that the ratio of CPr/APT in the myocardium is significantly decreased whether or not it is caused by ischemia.Moreover, coronary artery stenosis in 70% patients during exercise PCr/ATP ratio decreased significantly (average decline of 35%) [19], Lombardo [20] found in myocardial infarction and peripheral non-infarcted metabolism can be detected in reducing the degree of myocardial infarction determines not around the infarcted myocardium can reduce the degree of metabolism, assessment of myocardium post infarction ventricular remodeling. Moreover, the abnormal myocardial energy metabolism is related to the severity of the left ventricular dysfunction, which indirectly reflects the severity of myocardial infarction.

T1 mapping and extracellular volume measurement:

T1 is the inherent property of an organization, and the T1 map can directly quantify the T1 value of the organization [21]. At present, T1 mapping uses MOLLI sequence [22], which is divided into contrast agents, pre-injection and post T1 mapping according to whether or not to use contrast agents. Extracellular matrix volume fraction is the percentage of extracellular matrix volume in the whole myocardial tissue volume. It is a relatively stable parameter index based on T1 mapping technique [21]. Quantitative imaging of extracellular volume fraction of the cells can detect subtle abnormalities in these extracellular volumes (ECV) of the delicate myocardium, with age variations and non-infarct areas diffusely fibrosis related [23].

Myocardial T1 mapping and ECV are considered as markers of early ventricular remodeling. The T1 mapping allows direct quantification of tissue T1 values to assess the severity of acute myocardial infarction injury without contrast agents [24]. A contrast agent case detection of myocardial fibrosis with magnetic resonance enhanced early after myocardial infarction and edema, necrosis and collagen deposition in the

extracellular matrix volume fraction increased, the infarct area, edge area and peripheral area to enhance the T1 value before and after quantitative calculation, extracellular matrix volume fraction, the reconstruction of extracellular matrix interstitial volume fraction can be assessed in different areas of myocardial extracellular volume changes, predict the degree of ventricular remodeling. The study found that in the early stage (<72 h) has not yet appeared their overload factors lead to myocardial fibrosis in the infarct zone, there have been ECV expansion, leading to ventricular remodeling, in the later stage, the degradation of the extracellular matrix may cause harmful inflammation, can also cause adverse ventricular remodeling and heart failure [25].

Prospect:

Currently, magnetic resonance imaging can be used to evaluate ventricular remodeling, including T2WI and T2*WI, myocardial marker imaging, and ultrashort echo magnetic resonance imaging. T2 weighted magnetic resonance imaging can easily detect myocardial hemorrhage, and myocardial hemorrhage after reperfusion is also an independent predictor of adverse remodeling in the left ventricle. Myocardial marker imaging can noninvasively assess myocardial deformation, that is, ventricular remodeling, by means of the myocardial marker imaging cannot be synchronized with myocardial index, i.e., circumferential ratio. Ultrashort echo magnetic resonance imaging can be performed without the use of exogenous contrast the collagen fibrils in the dead band of the stem were detected directly to reflect the remodeling of the cardiac muscle fiber. However, these technologies are still in the stage of research and development and have not yet been popularized.

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