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The correlation between myocardial resilience after high-intensity exercise and markers of myocardial injury in swimmers

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Abstract

To investigate how high-intensity exercise influences an athlete's myocardial resilience and the correlation between myocardial resilience and markers of myocardial ischemic injury.

Fifteen swimmers participated in high-intensity exercises. Cardiac ultrasound was performed before and after exercise on each subject. Left ventricular general strain, systolic general strain rate, and the differences (▲general strain and ▲ general strain rate, respectively), before and after exercise were analyzed. Blood was collected at the morning of the exercise day and 6 hours after exercise to measure cardiac enzyme indicators.

The correlation between myocardial resilience and markers of myocardial injury were evaluated. Most cardiac enzymes concentrations increased after exercise ($P < .05$). Cardiac troponin I, creatine kinase MB, and cardiac troponin T were all correlated with the degree of ▲ peak strain (differential value of posterior wall basal segment before and after exercise) and ▲ peak strain rate (differential value before and after exercise) ($P < .05$).

After high-intensity exercise, the concentrations of creatine kinase MB and cardiac troponin T in the blood are positively correlated with two-dimensional ultrasound deformation indices, proving the fact that the seindices can be used as a diagnostic basis for myocardial injury, and are more sensitive than general strain. The two-dimensional strain echocardiogram is non-invasive and easily accepted by the patient. It can make up for the shortage of myocardial enzymes in the injury areas, including weak timeliness and the inability to locate injury.

Abbreviations: 2D = two-dimensional, CK = creatine kinase, CK-MB = creatine kinase MB, cTnI = cardiac troponin I, cTnT = cardiac troponin T, GS = general strain, GSR = general strain rate, S = peak strain, SR = peak strain rate.

Keywords: cardiac troponin T, creatine kinase isoenzyme, cTnT, high-intensity exercise, myocardial enzymes, myocardial injury, myocardial resilience

1. Introduction

Two-dimensional (2D) ultrasound strain technique, also known as the speckle tracking technique, is currently used to accurately detect functional changes in each part of the myocardium.[1] Ultrasound images of 2D strain rate imaging technology is used, and the software will automatically divide left ventricular wall into 18 segmental, record left ventricular total strain, systolic strain rate, and 18 general segmental systolic peak strain (S), peak strain rate (SR).

Indicators such as myocardial general strain (GS) and systolic general strain rate (GSR) are used mainly to measure myocardial resilience. Myocardial resilience refers to the magnitude of deformation of the myocardium during cardiac cycles, which can directly reflect its systolic and diastolic functions.[2] Most studies on 2D strain suggest that myocardial resilience is associated with the physiological and pathological conditions of the myocardium.[3] The myocardium of healthy people can reach the apical segment from the basal segment longitudinally, and myocardial systolic GS varies within the range of 15% to 19%. Comparative evaluation can be conducted through techniques such as magnetic resonance imaging.[3,4] When systolic GSR <–0.80/s is used as the standard, the sensitivity and specificity of detecting abnormal segments are both 85%.[5] Abnormal segments are usually associated with myocardial infarction.[6]

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The study was approved by the General Administration of Sport of China. Informed consent was obtained.

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The datasets generated during and/or analyzed during the current study are publicly available.

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It is a clinically common detection approach to evaluate myocardial physiological status by detecting myocardial enzymes. Cardiac troponin T (cTnT), cardiac troponin I (cTnI), creatine kinase (CK), and creatine kinase MB (CK-MB) are the most commonly used indicators. Several studies have indicated that cTn is the best cardiac injury marker regardless of its specificity or sensitivity to the myocardium. It has gradually replaced CK-MB as the gold standard for the diagnosis of acute myocardial infarction.[7,8]

High-intensity exercise can induce cardiac stress, manifesting in both structure function and physiological status.[9,10] Overtraining or exhaustive exercise can greatly increase the production of cardiomyocyte radicals, increase cardiomyocyte apoptosis, and impact the endocrine function of the heart.[9,10] Therefore, there is some concern that excessive training or long-term exhaustive exercise can damage the normal function of the heart and cause a change in the left ventricle function.

In this study, swimmers were selected for high-intensity exercise, blood samples were collected and 2D echocardiogram images were taken before and after exercise. The influence of high-intensity exercise on the athletes' myocardium was analyzed, as well as the correlation between strains in different parts of the myocardium and markers of myocardial injury.

2. Materials and methods

2.1. Participants

Fifteen healthy athletes from the Henan Province Swimming Team were enrolled in the study. After the participants were given all the information on the study protocols, informed consent was obtained from each. An electrocardiogram, echocardiogram, and blood biochemical tests were conducted. Those participants with cardiovascular system diseases were excluded, leaving 15 athletes enrolled in the study. The study was approved by the General Administration of Sport of China. Informed consent was obtained.

2.2. Methods

2.2.1. Exercise and cardiac ultrasound. Approximately 0.5 hour before exercise, a cardiac ultrasound was performed on each athlete. Then the athletes performed a warm up exercise of 2000 meters freestyle swimming in 30 minutes. After warming up, incremental load training of 8×100 meters was conducted, followed by 5×200 meters.

In the 8×100 meters training, the program is that in first 200 meters, after athletes swim each 100 meters, there is a 1 minute rest; after the third 100 meters, there is 5 minutes rest; after the fourth 100 meters, there is 1 minute rest; after the fifth 100 meters, there is 5 minutes rest; in the last 300 meters, there is 5 minutes rest after each 100 meters. Athletes will rest 10 minutes and then start 5×200 meters special training after the 8×100 meters progressive training. The program of 5×200 meters special training is that athletes will rest 5 minutes after swim each 200 meters. When they finish the last 200 meters and go ashore, the collection of cardiac ultrasound image will begin immediately. We set up a simple sickbed by the pool. While athletes finish the training program, they will go ashore immediately and then run to the sickbed. The time is about 45 seconds for athletes from stop swimming to lie down for the ultrasonic testing.

The GE VIVID I portable color ultrasound diagnostic echocardiogram with M3S probe and frame rate >70/s was used for the ultrasound tests on each athlete. The Eco PAC PC ultrasound workstation was used to analyze the images (General Electric).

2.2.2. Blood collection. The venous blood of each athlete was collected immediately after wake up after fasting overnight and 6 hours after exercise. Three milliliters of venous blood were collected from the same-side upper limb of each athlete.

2.3. Test indicators

2.3.1. Electrocardiogram. The athletes were positioned on their left side, chest leads were connected, and the electrocardiogram was performed. 2D grey-scale dynamic ultrasound images of 3 cut surfaces comprising the apical 2-chamber, apical 4-chamber, and apical long-axis views were collected. Each image comprised at least 3 complete cardiac cycles. Analysis software of the 2D-strain rate imaging technique was used to conduct quantitative analysis on each segment of the myocardium. Endocardial clear images of the 3 cut surfaces were selected from 1 side of the valve annulus to the other side of valve annulus through the apical part. The left ventricular endocardial border was manually drawn and the instrument automatically tracked intra-myocardial spots. After tracking, the instrument equally divided the 6 chamber walls of the left ventricle into basal, middle, and apical segments, for a total of 18 myocardial segments. Endocardial clear images of the 3 cut surfaces were analyzed in the same way and the software obtained a bull's eye image of and left ventricle GS data on the 18 segments (Fig. 1). The indicators were GS, GSR, the S of the 18 segments, and the SR of the 18 segments. ▲GS and ▲GSRs represented the difference in GS and GSRs before and after exercise, respectively. ▲ S and ▲ SR represented the difference between peak S and peak SR in each segment.

2.3.2. Blood samples. Blood samples were centrifuged and serum was collected to analyze cTnT, cTnI, CK, and CK-MB levels. The hemoglobin and hematocrit tests were completed within 30 min of blood collection.

2.4. Statistical analysis

All data were represented by the mean ± standard deviation. SPSS17.0 (SPSS Inc., Chicago, IL) and Microsoft Excel 2010 (Microsoft, Redmond, WA) were used for data processing. Normality tests were conducted on all data. For data that followed a normal distribution both before and after exercise, a paired *t* test and bivariate correlation analysis were used, and the Pearson correlation coefficients were calculated. For data that did not follow a normal distribution before or after exercise, the Wilcoxon signed rank test and bivariate correlation analysis, which were paired sample comparison tests, were used, and the Spearman correlation analysis was calculated. A $P < .05$ indicates statistically significant difference.

3. Results

3.1. Basic characteristics

The baseline characteristics of the participants were as follows, 9 males, 6 females; average age was 16.33 ± 1.95 years; average exercise years were 7.07 ± 2.12; exercise levels were 5 master grade, 8 level 1, and 2 level 2.

3.2. Changes in myocardial injury markers before and after exercise

3.2.1. Hemoglobin and hematocrit. A paired sample *t* test was conducted on hemoglobin and hematocrit results (Table 1). The

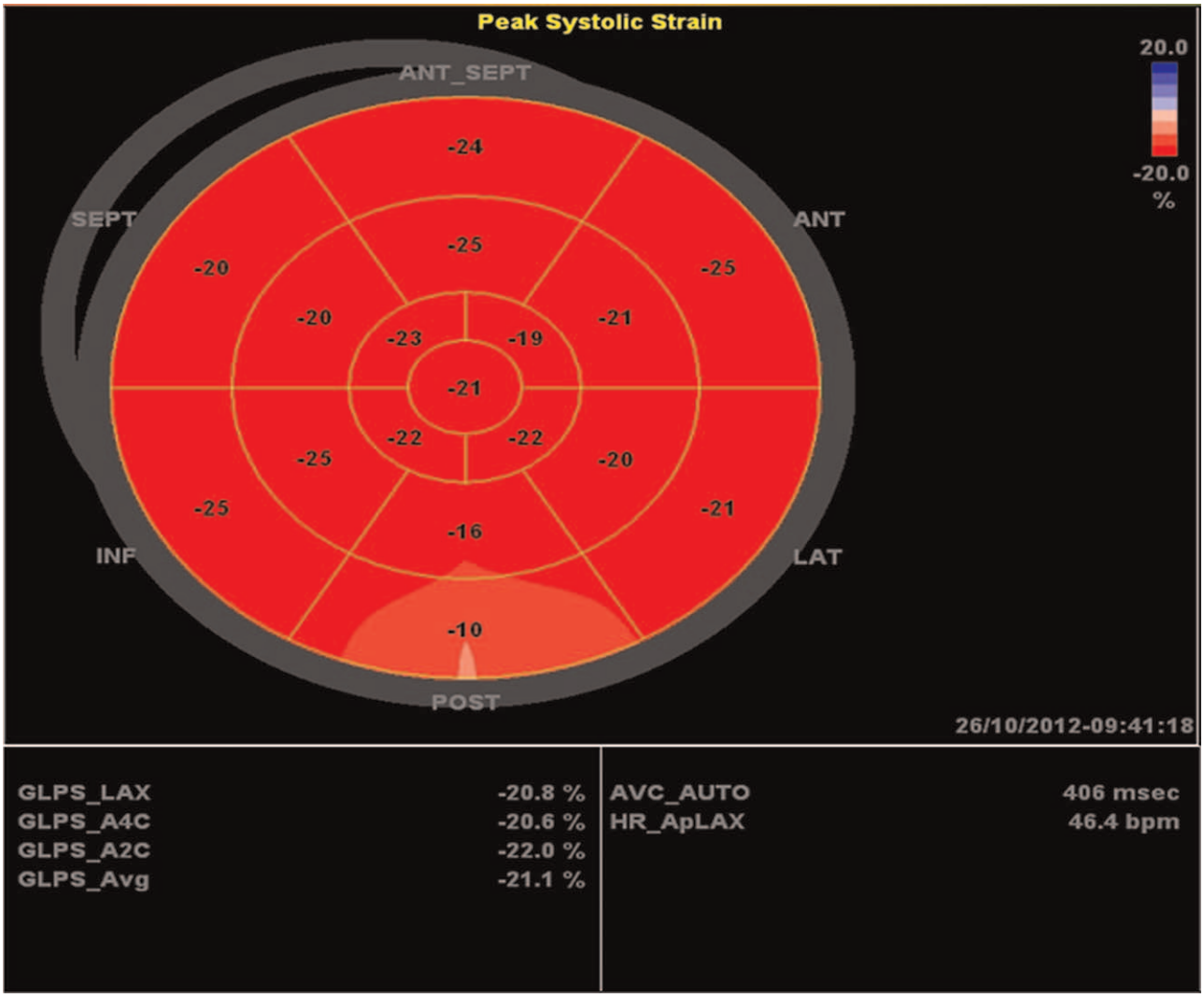

Figure 1. Bull's eye figure of the 18 myocardial segments.

results showed that both hemoglobin concentration and hematocrit percentage decreased after exercise, but the changes were not significant different for these indicators. Therefore, it was concluded that the increase in myocardial injury markers did not result from a decrease in blood volume.

3.2.2. Myocardial injury markers before and after exercise. A paired sample *t* test was conducted for CK-MB levels before and after exercise and a Paris sample comparison and Wilcoxon signed rank test were conducted on cTnT, cTnI, and CK before and after exercise (Table 2). The results showed that the concentrations of cTnT, CK, and CK-MB after exercise were significantly higher than those before exercise ($P < .01$). However, the concentration of cTnI showed only an increased trend after exercise, but not significant difference ($P > .05$).Thus, the results of this study suggested that exercise promoted the release of cTnT, CK, and CK-MB. Therefore, cTnT, CK, and CK-MB might be used as sensitive markers in further study.

Table 1

Comparison between hemoglobin and hematocrit before and after exercise.

	Before exercise (n = 15)	After exercise (n = 15)
Hemoglobin (g/L)	145.33 ± 11.73	141.07 ± 14.22
Hematocrit (%)	42.87 ± 3.31	40.63 ± 5.31

Table 2

Comparison of myocardial injury markers before and after exercise.

Myocardial injury markers	Before exercise (n = 15)	After exercise (n = 15)
cTnT (ng/mL)	0.00473 ± 0.000884	0.00687 ± 0.004103**
cTnI (ng/mL)	0.0145 ± 0.032555	0.013 ± 0.022221
CK (U/L)	143.80 ± 73.08	191.93 ± 126.40**
CK-MB (U/L)	9.80 ± 2.81	12.07 ± 3.22**

CK = creatine kinase, CK-MB = creatine kinase MB, cTnI = cardiac troponin I, cTnT = cardiac troponin T.

* As compared with that before exercise, $P < .05$

** As compared with that before exercise, $P < .01$.

Table 3

Analysis of the correlation among ▲ GS, ▲ GSRs, and myocardial injury markers.

Relative coefficient R	▲ cTnT (n=15)	▲ cTnI (n=15)	▲ CK (n=15)	▲ CK-MB (n=15)
▲ GS (%)	0.432	–0.183	0.048	0.649**
▲ GSR (%)	0.553*	0.477	0.273	0.589*

CK = creatine kinase, CK-MB = creatine kinase MB, cTnI = cardiac troponin I, cTnT = cardiac troponin T, GS = general strain, GSR = general strain rate.
* Represents $P<.05$
** Represents $P<.01$.

3.2.3. Correlation between myocardial injury markers and mechanical work index

3.2.3.1. Correlation between myocardial GS and myocardial injury markers. Bivariate correlation analysis was conducted between ▲ cTnT, ▲ cTnI or ▲ CK, and ▲ GS or ▲ GSRs, and Spearman coefficients were calculated (Table 3).

The results indicated that the ▲ cTnT and ▲ GSR were positively correlated (r=0.553, $P<.05$). There was no linear correlation between ▲CK and ▲GS or ▲CK and ▲GSRs, while ▲ CK-MB was positively correlated with both ▲ GS (r=0.649, $P<.01$), and ▲ GSRs (r=0.589, $P<.05$). ▲ cTnT concentration was positively correlated with myocardial GSR, and the extent of the ▲ CK-MB was consistent with that of myocardial ▲ GS.

3.2.3.2. Correlation between peak strain of each myocardial segment and myocardial injury markers. Bivariate correlation analysis was conducted among ▲ cTnT, ▲ cTnI, ▲ CK, and the change in the S of the 18 segments, and Spearman correlation coefficients were calculated. Bivariate correlation analysis was also conducted between ▲ CK-MB and the anterior wall basal segment (basAntSept ▲S), lateral wall apical segment (apLat ▲S), and inferior wall middle segment (midInf ▲ S), and Spearman correlation coefficients were calculated. Analysis was conducted between ▲ CK-MB and the posterior wall basal segment (basPost ▲ S), posterior wall middle segment (midPost ▲ S), posterior wall apical segment (apPost ▲ S), anterior interval apical segment (apAntSept ▲ S), anterior interval middle segment (midAntSept ▲ S), posterior interval basal segment (basSept ▲ S), posterior interval middle segment (midSept ▲ S), posterior interval apical segment (apSept ▲ S), lateral wall middle segment (midLat ▲ S), lateral wall basal segment (basLat ▲ S), inferior wall basal segment (basInf ▲ S), inferior wall apical segment (apInf ▲ S), anterior wall apical segment (apAnt ▲ S), anterior wall middle segment (midAnt ▲ S), and basAnt ▲ S, and Pearson correlation coefficients were calculated (Table 4).

The results showed that ▲CTnT was positively correlated with apLat ▲ S (r=0.531, $P<.05$). ▲ CTnI was positively correlated with basPost ▲ S (r=0.638, $P<.005$), midPost ▲ S (r=0.743, $P<.001$), midAnt ▲ S (r=0.667, $P<.05$), and basAnt ▲ S (r=0.647, $P<.05$). ▲ CK was negatively correlated with basPos ▲ S (r=0.581, $P<0.05$).

3.2.3.3. Correlation between SR of each myocardial segment and myocardial injury markers. Bivariate analysis was conducted between ▲CTnT, ▲CTnI, and ▲CTnI and changes in myocardial peak SR of the 18 left ventricular segments. Spearman coefficients were calculated. Bivariate analysis was conducted between ▲CK-MB and basInf ▲ SR, midInf ▲ SR, apInf ▲ SR, and basAnt ▲ SR and Spearman coefficients were calculated. Analysis was also conducted between ▲ CK-MB and basPost ▲ SR, midPost ▲ SR,

Table 4

Correlation between peak ▲ S and changes in myocardial injury markers.

Relative coefficient r	▲ cTnT (n=15)	▲ cTnI (n=15)	▲ CK (n=15)	▲ CK-MB (n=15)
Posterior wall basal segment ▲ S	0.103	0.638*	0.581*	–0.352
Posterior wall middle segment ▲ S	0.183	0.743**	–0.247	0.102
Posterior wall apical segment ▲ S	–0.377	0.243	0.418	0.789**
Anterior interval apical segment ▲ S	0.513	–0.261	0.25	0.483
Anterior interval middle segment ▲ S	–0.275	0.667*	0.311	0.196
Anterior interval basal segment ▲ S	–0.04	0.647*	0.327	0.259
Posterior interval basal segment ▲ S	–0.07	–0.495	–0.284	0.117
Posterior interval middle segment ▲ S	–0.062	–0.372	–0.222	0.271
Posterior interval apical segment ▲ S	–0.227	0.229	0.002	0.345
Lateral wall apical segment ▲ S	0.531*	0.248	0.063	0.318
Lateral wall middle segment ▲ S	–0.37	–0.161	–0.154	0.159
Lateral wall basal segment ▲ S	0.026	–0.055	–0.324	–0.243
Inferior wall basal segment ▲ S	–0.073	0.193	0.265	–0.06
Inferior wall middle segment ▲ S	–0.099	0.422	0.195	–0.079
Inferior wall apical segment ▲ S	–0.07	0.298	–0.03	0.419
Anterior wall apical segment ▲ S	–0.377	–0.05	–0.113	0.504
Anterior wall middle segment ▲ S	–0.282	–0.335	0.166	0.558*
Anterior wall basal segment ▲ S	0.062	–0.28	0.057	0.297

CK = creatine kinase, CK-MB = creatine kinase MB, cTnI = cardiac troponin I, cTnT = cardiac troponin T, S = peak strain.
* Represented $P<.05$.
** Represented $P<.01$.

Table 5

Analysis of the correlation between ▲ SR and changes in myocardial injury markers.

Correlation coefficient r	▲ cTnT (n=15)	▲ cTnI (n=15)	▲ CK (n=15)	▲ CK-MB (n=15)
Posterior wall basal segment ▲ SR	0.095	0.725*	–0.293	–0.093
Posterior wall middle segment ▲ SR	–0.066	0.593	–0.333	–0.013
Posterior wall apical segment ▲ SR	–0.498	0.651*	0.181	0.552*
Anterior interval apical segment ▲ SR	–0.348	0.145	0.54*	0.646**
Anterior interval middle segment ▲ SR	–0.458	–0.05	0.211	0.125
Anterior interval basal segment ▲ SR	0.088	–0.009	0.084	0.012
Posterior interval basal segment ▲ SR	0.154	–0.119	0.007	–0.272
Posterior interval middle segment ▲ SR	–0.275	0.202	0.352	0.286
Posterior interval segment ▲ SR	–0.304	0.541	0.177	0.085
Lateral wall apical segment ▲ SR	–0.44	0.633*	0.2	0.336
Lateral wall middle segment ▲ SR	–0.293	0.211	–0.172	–0.093
Lateral wall basal segment ▲ SR	0.018	0.234	–0.249	–0.108
Inferior wall basal segment ▲ SR	0.235	0.381	0.197	–0.054
Inferior wall middle segment ▲ SR	0.026	0.367	0.359	0.052
Inferior wall apical segment ▲ SR	0.033	0.28	0.234	0.212
Anterior wall apical segment ▲ SR	–0.238	–0.142	0.363	0.615*
Anterior wall middle segment ▲ SR	0.088	0.289	0.315	0.412
Anterior wall basal segment ▲ SR	0.077	0.17	0.431	0.171

CK = creatine kinase, CK-MB = creatine kinase MB, cTnI = cardiac troponin I, cTnT = cardiac troponin T, SR = peak strain rate.
* Represented $P<.05$.
** Represented $P<.01$.

apPost ▲ SR, apAntSept ▲ SR, midAntSept ▲ SR, basAntSept ▲ SR, basSept ▲ SR, midSept ▲ SR, apSept ▲ SR, apLat ▲ SR, midLat ▲ SR, basLat ▲ SR, apAnt ▲ SR, and midAnt ▲ SR. Pearson coefficients were calculated (Table 5).

The results showed that there was no linear correlation between ▲ cTnT and the changes in peak SR in the 18 left ventricular segments. ▲ cTnT was positively correlated with baPost ▲ SR ($r=0.725$, $P<.05$), apPost ▲ SR ($r=0.651$, $P<.05$), and apLat ▲ SR ($r=0.633$, $P<.05$). ▲ CK-MB was positively correlated with apPost ▲ SR ($r=0.552$, $P<.05$), apAnt ▲ SR ($r=0.615$, $P<.05$), and apIntSept ▲ SR ($r=0.646$, $P<.01$).

4. Discussion

Our current study showed that the concentrations of CK-MB and cTnT in the blood were positively correlated with 2D ultrasound deformation indices after high-intensity exercise, while the change in myocardial SR of the apical segment is positively correlated with cTnT, CK, and CK-MB.

Myocardial enzyme markers are commonly used in clinical settings to represent physiological status. Previous studies have suggested that high-intensity exercise can cause micro damage to the myocardium.[11] This study has compared 2 indicators, hemoglobin concentration and hematocrit percentage-that reflect body plasma volume. The results of the study found that although the indicators decrease as a result of exercise, the difference is not statistically significant. The concentrations of cTnT, CK, and CK-MB were also compared before and after exercise and all values were greater than those before exercise. Given that there was no statistical difference in indicators that reflected plasma volume, the results of this study infer that exercise promotes the release of cTnT, CK, and CK-MB in the cells but has little influence on cTnI concentration.

The concentrations of myocardial injury markers are significantly different before and after exercise, which indicates myocardial injury. GS after exercise is also distinctly different after exercise. D'Andrea et al[8] evaluated the local and overall myocardial functions in normal athletes using speckle tracking techniques. By applying GS and GSR imaging techniques, it was found that professional soccer players had GSRs of the interventricular septum and left ventricular lateral walls that were higher than those in non-athletes.[12] They suggested that GSR could be used as an effective indicator by which to evaluate left ventricular systolic function and physiological status.

Our study showed that releasing cTnT into the blood was correlated with the overall deformation rate of left ventricle in the systolic phase and the apLat ▲ S. The function that exercise promoted the release of cTnI into the blood was correlated with basPost ▲ S, basAnt ▲ S, midAnt ▲ S, basPost ▲ S, apPost ▲ S, and apLat ▲ S. The function that exercise promotes the release of CK into the blood was correlated with basPos ▲ S and apAntSept ▲ S. The function that exercise could promote the release of CK-MB into the blood was correlated with deformation degree of the overall left ventricle, midAnt ▲ S and apPost ▲ S as well as with the deformation rate of posterior wall, anterior wall, and anterior intervals. At the same time, the study found, by summarizing the myocardial parts that were associated with myocardial injury, myocardial injury easily occurs on the posterior wall and anterior interval. When there was clinical myocardial injury, the changes in the myocardial enzymes had specific timeliness and negative situations, which might be associated with the areas of injury. Myocardial enzymes were sensitive to injury in some areas but not in others. However, the release of myocardial enzymes is diffusive, thus this cannot be used as an indicator of injury area. 2D ultrasound not only can qualitatively detect myocardial injury, but can locate the injury relatively correctly, which mitigates the shortage of myocardial enzymes in specific areas of injury.

A recent meta-analysis showed that exercise intensity and age were the most powerful determinants of cTn release. Diastolic function was influenced by exercise HR and cTn release, which implied that exercise bouts at high intensities were enough to elicit cTn release and reduced LV diastolic function.[13] The results were consistent with our study. Another study investigat-

ing the cardiac structure and function in long-term elite master endurance athletes with special focus on the right ventricle by contrast enhanced cardiovascular magnetic resonance imaging, showed that a chronic right ventricular function damage in elite endurance master athletes with lifelong high training volumes seems to be unlikely.[14] The contrary results might be caused by the use of a cross sectional study design which might have led to a recruitment bias.

During the process of applying the 2D strain ultrasound technique to evaluate the influence of exercise on the heart, we found that the 2D strain ultrasound also had certain disadvantages. First, the respiration rate of the athletes after exercise was relatively acute and more gas enters the lungs, in which case the track of speckles and the accuracy in evaluating myocardial function would be affected. Second, although 2D strain had no angular dependence, there was controversy in that ultrasonic motion in a perpendicular direction which was more susceptible to a greater incidence of errors. Third, the heart was a 3D structure, while 2D strain was only an estimation on a 2D level, indicating that it could reflect only myocardial strain laterally and could not fully and truly reflect the entire strain. Finally, the sample size for this study was relatively small.

In conclusion, after high-intensity exercise, the concentrations of CK-MB and cTnT in the blood are positively correlated with 2D ultrasound deformation indices, proving the fact that the seindices can be used as a diagnostic basis for myocardial injury, and are more sensitive than GS. 2D ultrasound deformation indices are correlated with myocardial injury to some degree. The change in myocardial SR of the apical segment is positively correlated with cTnT, CK, and CK-MB, proving that the apical segment has greater sensitivity to motor stimulation and thus 2D strain ultrasound technique can be used at early stages of cardiac injury more easily. The 2D strain echocardiogram is non-invasive and easily accepted by the patient. It can make up for the shortage of myocardial enzymes in the injury areas, including weak timeliness and the inability to locate injury.

Author contributions

CG is responsible for the guarantor of integrity of the entire study, study concepts & design, definition of intellectual content, clinical studies, experimental studies, data acquisition & analysis, statistical analysis, manuscript preparation; CL is responsible for the guarantor of integrity of the entire study, study concepts & design, definition of intellectual content, clinical studies, experimental studies, data acquisition & data analysis, statistical analysis, manuscript preparation; JHZ is responsible for the literature research, manuscript editing; YM is responsible for the clinical studies; XXM is responsible for the data acquisition, data analysis; MHX is responsible for the guarantor of integrity of the entire study, definition of intellectual content, clinical studies, experimental studies, data acquisition, statistical analysis, manuscript review. All authors read and approved the final manuscript.
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Methodology: Minhao Xie.
Resources: Can Gao, Chen Liang.
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