Comprehensive Assessment of Changes in Left Ventricular Volume and Myocardial Function After Acute Myocardial Infarction in the Elderly: The Role of Real-Time Three-Dimensional Echocardiography and Two-Dimensional Speckle Tracking Imaging

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Research

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Abstract

Background: Conventional echocardiography is not sensitive enough to assess the mild acute myocardial infarction (AMI), especially the non ST elevation AMI (NSTEMI) that the myocardial motion is often normal or mildly reduced. This research attempt to find some new technology to better assess the left ventricular (LV) volume and myocardial function after percutaneous coronary intervention (PCI) in the elderly patients with NSTEMI and ST elevation AMI (STEMI).

Methods: Patients with NSTEMI (n=40) and STEMI (n=40) underwent imaging investigations 1 week and 3 months after PCI. A group of 40 healthy volunteers was used as a control group. The LV end-diastolic volume (EDV), end-systolic volume (ESV), stroke volume (SV), and ejection fraction (EF) were measured using real-time three-dimensional echocardiography (RT-3DE). The longitudinal peak systolic strain (LPSS), radial peak systolic strain (RPSS), circumferential peak systolic strain (CPSS) and rotational peak degree (Rot) in the myocardium supplied by infarct-related artery (Myo-IRA) segments (LPSS_{Ap}, LPSS_{Mid}, LPSS_{Bas}, RPSS_{Ap}, RPSS_{Mid}, RPSS_{Bas}, CPSS_{Ap}, CPSS_{Mid}, CPSS_{Bas}, Rot_{Bas}, Rot_{Ap}) were obtained using two-dimensional speckle tracking imaging (2D-STI). The peak torsion angle (Twist) were calculated.

Results: Compared with control group, in 1 week after PCI, the ESV and EDV in NSTEMI and STEMI patients were significantly increased (P<0.001), the LPSS, RPSS, CPSS in the Myo-IRA segments and the EF, SV, Rot_{Bas}, Rot_{Ap} and Twist in the NSTEMI and STEMI patients were significantly lower (P<0.001), and more so in the STEMI patients. Compared with 1 week after PCI, in 3 months after PCI, the ESV, EDV, SV, EF, RPSS, CPSS, Rot_{Bas} and Twist in NSTEMI and STEMI patients were recovered partially (P<0.01), the LPSS and Rot_{Ap} only in the NSTEMI patients was improved significantly (P<0.01).

Conclusions: The LV volume functions, longitudinal, radial, circumferential, and twist movement in the elderly AMI patients with different degree of transmural damage 1 week and 3 months after PCI can be objectively and sensitively revealed by using RT-3DE and 2D-STI respectively. The LV myocardial multidimensional deformation functions are severely damaged in both NSTEMI and STEMI patients, but the NSTEMI patients have a better prognosis after treatment over a long time.

Background

For better treatment and improved prognosis in patients with acute myocardial infarction (AMI), clinicians have emphasized the division of AMI into non ST elevation acute myocardial infarction (NSTEMI) and ST elevation acute myocardial infarction (STEMI) [1]. In NSTEMI, the myocardial ischemic damage has not yet spread throughout the full thickness, and the electrocardiogram (ECG) shows ST-segment depression or T wave inversion. In STEMI, the ECG shows ST-segment elevation or wide and deep Q wave, corresponding to severe stenosis or occlusion in the coronary arteries, which causes full thickness transmural myocardial damage and can progress to a larger area of myocardial infarction that is indicated by pathological Q waves. Conventional echocardiography is not sensitive enough to assess the...
mild acute myocardial infarction (AMI), especially the non ST elevation AMI (NSTEMI), the myocardial motion is often normal or mildly reduced in such cases, leading to a higher rate of missed diagnosis [2, 3]. It is therefore necessary to explore more sensitive ultrasound techniques to assess such patients. In this study, quantitative assessment of left ventricle (LV) volume and myocardial function was performed after percutaneous coronary intervention (PCI) in the elderly patients with NSTEMI and STEMI by using real-time three-dimensional echocardiography (RT-3DE) and two-dimensional speckle tracking imaging (2D-STI). This study was performed to explore the different transmural extent of AMI lesion, to determine the effects of therapeutic measures, and provide more useful and accurate reference information for the comprehensive analysis of the condition and patient prognosis.

Materials And Methods

Study population

Eighty consecutive elderly patients diagnosed with AMI were prospectively enrolled in our hospital. All patients had chest pain and increased levels of myocardial injury markers. Selective coronary arteriography (SCA) showed that at least one coronary artery had subtotal or total occlusion. All patients were suitable candidates for PCI. Based on ECG findings before PCI, we divided the patients into the NSTEMI group (n=40, 27 men, 13 women, mean age 70.45 ± 4.08 years, median age 70 years, age range 65-79 years) and the STEMI group (n=40, 28 men, 12 women, mean age 71.07 ± 4.15 years, median age 71 years, age range 65-79 years). The exclusion criteria were: patients with old myocardial infarction, valvular heart disease, congenital heart disease, cardiomyopathy, arrhythmia, and multiple organ complications caused by severe hypertension and diabetes. In addition, a control group was added, composed of randomly selected healthy volunteers (n= 40, 25 men, 15 women, mean age 70.53 ± 4.11, median age 70 years, age range 65-79 years).

Echocardiographic imaging and analysis

Echocardiography was performed by using a Vivid 7 Dimension ultrasound scanner (GE, USA) with 3V volume phased array transducer (frequency range of 1.5-3.2 MHz) and M3S volume phased array transducer (frequency range of 1.7-3.4MHz); Echo PAC workstation, equipped with 4D LV Volume TomTec and 2D strain analysis software. Patients were placed in the left lateral decubitus position for transthoracic echocardiography 1 week and 3 months after PCI, and the images were acquired with a simultaneous ECG signal after the patients were instructed to maintain steady breathing.

RT-3DE: In the two-dimensional conditions, a 3V probe was placed at the apical four-chamber view of the LV until the ideal image appeared, after which the 4D function key was selected and the full-volume condition was started. RT-3DE data sets were acquired when the patient held their breath, by using a 60°×60° wide-angle acquisition mode in which 4 wedge-shape sub volumes were obtained from 4 consecutive cardiac cycles with the acquisition triggered to the R wave of the ECG. The images were then transferred to the Echo PAC workstation. Using 4D-LV analysis software (4D LV volume Tom-Tec, GE Medical System), the sagittal and coronal sections of the middle LV were located, along with the cross-
section located papillary muscle level, and the endocardial end-diastolic and end-systolic tracings. Based on this data, the software automatically calculated the left ventricular end-diastolic volume (EDV), end-systolic volume (ESV), stroke volume (SV), and ejection fraction (EF). The values of three cardiac cycles were measured and the average of the three readings was used.

2D-STI: Using the M3S probe, two-dimensional images were recorded at high frame rates (50-80 frames/s) from the short-axis mitral, papillary muscle, and apical level of the LV over 3-5 consecutive cardiac cycles. Images were also recorded from the apical long-axis, four- and two-chamber views of the LV during the end-expiratory breath hold. These images were then transferred to the Echo PAC workstation. 2D strain analysis software was used to obtain the tracking myocardial segments throughout the cardiac cycle trajectory, especially the strain-time curves and the rotation-time curve. The myocardial segments that were indicated by the SCA as having lesions of coronary blood supply 1 week after PCI showed a significantly lower longitudinal peak strain (the same levels were significantly lower than the average longitudinal peak strain of 1 to 3 segments) were selected as myocardium supplied by the infarct-related artery (Myo\textsubscript{IRA}) segments (basal, middle, apical). In NSTEAMI: the base had 77 Myo\textsubscript{IRA} segments, the middle had 75, and the apex had 59. In STEAMI: the base had 65 Myo\textsubscript{IRA} segments, the middle had 67, and the apex had 74. The longitudinal peak systolic strain (LPSS), radial peak systolic strain (RPSS), and the circumferential peak systolic strain (CPSS) in all the above Myo\textsubscript{IRA} segments were measured at 1 week and 3 months after PCI. Using software to analyze the groups, the LV rotational peak degree in basal segments (Rot\textsubscript{Bas}), rotational peak degree of apical segments (Rot\textsubscript{Api}), and LV peak torsion angle (Twist) were calculated (LV Twist = Rot\textsubscript{Api} - Rot\textsubscript{Bas}). The values were measured over three cardiac cycles and the average was calculated.

**Statistical analysis**

Data analysis was performed by using the standard software (SPSS, Version 20.0). Continuous variables were presented as mean ± standard deviation (SD). The normality test was used to compare continuous variables, and one-way analysis of variance (ANOVA) was used to compare echocardiographic values of AMI patients in the different times (1 week and 3 months after PCI) with those of the controls. Continuous variables from the different groups were compared by using Fisher's least significant test. All $P$ values were two-sided, and $P$ values less than 0.05 were considered statistically significant.

**Results**

**LV volume function**

One week after PCI, compared with the control group, the ESV and EDV in the NSTEAMI and STEAMI patients were significantly increased ($P<0.001$), the EF and SV were significantly lower in both groups ($P<0.001$). Three months after PCI, the ESV and EDV were decreased in the NSTEAMI and STEAMI patients compared with 1 week after PCI ($P<0.01$), the reduction were particularly evident in the NSTEAMI group, and no statistically significant differences were seen when compared with the control group ($P>
0.05). The EF and SV were significantly improved in both groups, and showed a significant difference compared with 1 week after PCI ($P<0.01$), however, the EF in both groups were still significantly lower when compared with the control group ($P<0.001$). (Table 1).

**LV LPSS**

In the control group, the LPSS of the apical myocardial segments was higher than that of the base and middle segments. In NSTEMI and STEMI patients, due to the longitudinal strain-time curve distribution disorder, the apical, middle, and basal strain gradient differences were not obvious. A Bull's-eye strain map showed that the LPSS in the Myo$_{IRA}$ segment were significantly lower, as indicated by the red area becoming light pink or even blue. One week after PCI, the LPSS of the Myo$_{IRA}$ segment in the NSTEMI and STEMI patients were significantly lower compared with the control group ($P<0.001$). Three months after PCI, the LPSS of the Myo$_{IRA}$ segments in the NSTEMI patients, although significantly improved compared with 1 week after PCI ($P<0.01$), were still significantly lower when compared with the control group ($P<0.001$). In STEMI patients, the LPSS of the Myo$_{IRA}$ segments showed no significant improvement compared with 1 week after PCI ($P>0.05$). (Table 2, Fig.1).

**LV RPSS**

In the control group, the RPSS of the middle myocardial segments was higher than that of the base and apical segments. In the NSTEMI and STEMI patients with radial strain-time curve distribution disorder, the middle, basal, and apical strain gradient differences were not obvious. One week after PCI, the RPSS of the Myo$_{IRA}$ segment were significantly lower in the NSTEMI and STEMI patients when compared with the control group ($P<0.001$), more so in the STEMI patients. Three months after PCI, the RPSS of the Myo$_{IRA}$ segments RPSS in NSTEMI and STEMI patients were significantly improved compared with 1 week after PCI ($P<0.01$). The changes were more significant in the NSTEMI patients, but the middle Myo$_{IRA}$ segments showed significant differences when compared with the control group ($P<0.001$). (Table 3).

**LV CPSS**

In the control group, the CPSS of the apical myocardial segments was higher than that of the base and middle segments. In NSTEMI and STEMI patients with circumferential strain-time curve distribution disorder, the apical, middle, and basal strain gradient differences were not obvious. One week after PCI, the CPSS of the Myo$_{IRA}$ segment in the NSTEMI and STEMI patients were significantly lower when compared with the control group ($P<0.001$), more so in the STEMI patients. Three months after PCI, the CPSS in the Myo$_{IRA}$ segments in the NSTEMI and STEMI patients were significantly improved compared with 1 week after PCI ($P<0.01$), more so in the NSTEMI patients, but significant differences when compared with the control group ($P<0.001$). (Table 4).

**LV Twist**
When viewed from the apex to the bottom of the heart, the apical group showed a counter clockwise rotation were positive number; The basal group showed a clockwise rotation movement, were negative number. Overall, the LV predominantly showed a counterclockwise twisting motion. The Rot$_{Api}$ of the control group was greater than the Rot$_{Bas}$, and showed a gradient decreasing trend, but in the NSTEAMI and STEAMI groups, this was not obvious 1 week after PCI. Only in the NSTEAMI group did this become relatively obvious at 3 months after PCI. One week after PCI, the LV Rot$_{Bas}$, Rot$_{Api}$, and Twist were significantly lower in the NSTEAMI and STEAMI patients when compared with control group ($P<0.001$); the reduction was particularly evident in the STEAMI group. Three months after PCI, the Rot$_{Api}$ in the NSTEAMI patients and Rot$_{Bas}$ in both groups were significantly improved compared with 1 week after PCI ($P<0.01$); the Rot$_{Api}$ in the NSTEAMI patients improved most significantly, and the difference was not statistically significant compared with the control group ($P>0.05$). The Rot$_{Api}$ in the STEAMI group were improved, but the difference was not statistically significant when compared with 1 week after PCI ($P>0.05$). The Twist in both groups improved significantly compared with 1 week after PCI ($P<0.01$), but the difference remained statistically significant compared with the control group ($P<0.001$). (Table 5, Fig. 2).

**Discussion**

**Assessment of changes in LV volume and myocardial function by using RT-3DE**

RT-3DE can fully display the three-dimensional shape of the heart in different phases. It can portray selected subendocardial myocardium, can measure the heart chamber volume function without relying on assumption of the geometry of the heart chamber, and its accuracy is not affected by the deformation of the heart chamber, segmental wall motion abnormalities, or other pathological states [4]. Research has shown that the results of using RT-3DE and cardiac magnetic resonance imaging to measure EDV, ESV, and SV had a high degree of similarity [5, 6]. RT-3DE provided accurate and reproducible quantitative characteristics for quick and accurate evaluation of cardiac function.

As myocardial infarction is associated with infarction location, size, collateral circulation and other factors, the systolic function decreases in a certain degree; necrotic myocardium cell elongation as well as the destruction of muscle fiber, and connective tissue structure results in thinning and stretching of the regional wall; this results in an increase in LV internal diameter and overall capacity at end-systole; the increase in LV volume increases the systolic wall shear stress, leading to myocardial necrosis bundle fracture, further expansion of the infarct area, wall thinning, and ultimately functional decline [7, 8]. The use of PCI clinical treatment for AMI is widespread; recently, minimally invasive vascular recanalization surgery has increased in popularity. This procedure uses the opening of the IRA to reduce the myocardial necrosis area, limit the infarct size and transmural extent, effectively terminate the vicious cycle of ischemic damage and systolic dysfunction, and finally provided a firm foundation for LV functional recovery [9].
In this study, although the opening of the IRA was early, one week after PCI, the ESV and EDV in NSTEAMI and STEAMI patients were still significantly increased; the EF and SV was significantly lower in both groups when compared with the control group. Three months after PCI, the EDV, ESV, EF, and SV in both groups had been recovered partially. This study showed that early reperfusion therapy could inhibit LV remodeling and improve LV function, mainly through the reduction of myocardial necrosis, limiting infarct size and the extent of transmural damage. However, in myocardial necrosis, even if improvement in the blood supply could not restore myocardial function, as the viable myocardium included hibernating myocardium, stunned myocardium, and injured myocardium, the systolic function improvement or recovery took a long time [10, 11].

**Assessment of LV systolic Multi-dimensional deformation by using 2D-STI**

The ventricular myocardial band originates from the pulmonary roots, encircles the right ventricle up to the LV apex, then terminates at the aortic root as a double helix; in this, the muscle fibers are in a clockwise direction from the outside into the interior and in a counterclockwise direction from the inside to the outside [12, 13]. The unique arrangement of myocardial fibers causes not only longitudinal, circumferential, and radial movements of the heart during the cardiac cycle, but also contortion of the myocardium, causing the long axis of the heart to shorten or elongate, thickening or thinning of the ventricular wall, and ventricular twisting. 2D-STI enables clinicians to identify ultrasound speckle signal within the myocardium frame by frame and obtain rapid quantitative values for each strain of myocardial segments, reflecting the deformation of myocardial segments. As it is less affected by the surrounding tissue, it enables an objective, accurate, and rapid assessment of myocardial motion [2, 14, 15].

Longitudinal strain is myocardial from the base to the apex in the long axis direction of the deformation ability. It is mainly affected by subendocardial helical fibers, the decrease in strain value and stenosis domination of the wall segment enables accurate assessment of the extent of myocardial injury [16]. Radial strain is the centrality movement of myocardial along the short axis direction. It depends on the subendocardium (58%), the mid layer annular (25%), and the subepicardium fibers (17%) contraction, reflecting the systolic wall thickening and enables evaluation of transmural injury extent [17]. Circumferential strain is the centrality movement of myocardial along the short axis direction, which is mainly caused by the contraction of the mid layer annular fibers, and it mainly indicates the extent of the transmural injury [16]. Myocardial ischemia first affects the subendocardial myocardium, along with the aggravation of ischemia, gradually affecting the mid layer and epicardial myocardium. Therefore, myocardial ischemia first affects myocardial longitudinal movement, while progression of the illness gradually affects the radial movement and circumferential movement [16, 17].

In this study, in the control group comparison of each level, multi strain has certain rules. For LPSS and CPSS: the apex is greater than the base and middle, there may be a relationship between the apex and the first contraction of the cardiac cycle of the site, most easily influenced by preload. For RPSS: the middle is greater than the base and apex, possibly due to the presence of papillary muscle and lead to middle radial strain dominance. One week after PCI, normal multi strain gradient was not obvious in the
NSTEAMI and STEAMI groups. Segmental abnormality was noted, and two Myo-IRA segment LPSS, RPSS, and CPSS were significantly lower, more so in the STEAMI group. The possible reasons are: First, coronary artery stenosis or occlusion during AMI, myocardial metabolism is in short supply, coupled with the calcium overload, acidosis, and other factors, resulting in myocardial cell edema, necrosis, rupture, and intercellular narrowing, which reduced the strain capacity significantly [18]. Although the PCI patency of the infarct related coronary artery was intact, the effect may be more likely to limit the scope and transmural extent of infarction. Second, There may be some degree of reperfusion microcirculation disturbance after AMI, such as production of oxygen free radicals, inflammatory reaction, endothelial cell injury, and so on [19, 20]. Third, the improvement or recovery of the contractile function of the hibernating myocardium, stunned myocardium, and injured myocardium would take a long time [10, 11], especially in elderly patients. Finally, NSTEAMI mainly causes platelet thrombosis, while coronary subtotal occlusion causes more subendocardial myocardial necrosis, rarely involving the entire myocardium [21, 22]. STEAMI mainly causes fibrin thrombosis with more severe stenosis or occlusion of the coronary artery leading to myocardial transmural necrosis [23], therefore, in the STEAMI group, multi-strain reduction is particularly evident.

Three months after PCI, the LPSS in the Myo-IRA segments was not obvious improvement in the STEAMI group and there was obvious improvement in the NSTEAMI group, but it was still significantly lower than that seen in the control group. This is mainly because the longitudinal movement of the left ventricular myocardium mainly affected the subendocardial helical muscle bundles, the coronary artery supplies the myocardium with blood from the outside to the inside. As the diameter of the subendocardial myocardial blood vessels is small, the peripheral resistance is relatively large; this results in the subendocardial muscle fibers being damaged firstly and seriously in case of myocardial blood supply disorders [24, 25]. The blood supply is difficult to restore even with prolonged treatment. The lower incidence of damage in the NSTEAMI group could be due to the fact that the entire myocardial layer was not damaged. Regarding the CPSS and CPSS in the Myo-IRA segments: the NSTEAMI and STEAMI groups both showed improvements. The changes in the NSTEAMI group were statistically significant, but differences in some segments of the NSTEAMI group were not statistically significant compared with the control group. The possible reasons for this are: First, PCI can effectively improve perfusion of the myocardial cells, especially in the outer layer, the middle layer, and the myocardial infarction edge of myocardial ischemic area, but recovery of the contractile function of the hibernating myocardium, stunned myocardium, and injured myocardium need a long time. Second, in the elderly AMI patients, ischemia-reperfusion injury can improve after a period of treatment. Finally, although NSTEAMI mainly involves subendocardial infarction, but in the short term, the middle layer myocardium and subepicardial myocardium, which are also partly involved, need a long time to recover. The above study results also suggested that in the NSTEAMI patients with PCI treatment after 3 months, the prognosis was better than that seen in the STEAMI patients.

Viewed from the apex to the bottom of the heart, when the heart contracts, the basal part of the LV rotates clockwise and the apical part rotates counterclockwise, the interaction of these two counter-rotating
forces produces torsional motion. In this study, the control group Rot_{Api} was greater than the Rot_{Bas}, showing a decreasing trend. The cardiac cycle in the LV performance for the twisting motion was in a counterclockwise motion, this is possibly because the myocardial fibers from the inside to the outside of the cross wall show a gradual decline in stress gradient [26], and from the apex to the base, the stress gradient also progressively decreases. Therefore, the apical difference between the maximum stress in the left ventricular twist contribution is greater than that of the basal. In the pathological state of regional myocardial fibers disorder and inconsistent contraction, the core of this twisting motion will change. One week after PCI, in the NSTEMI and STEMI groups, the Rot_{Bas}, Rot_{Api}, and Twist were significantly lower, the apical to basal Rot gradient was not obvious, the apical rotational movement that plays a decisive role in the LV twisting motion was still significantly impaired, which may be related to the fact that the apical level is the thinnest part in the LV and more sensitive to ischemic injury [27]. In addition, some studies showed that when systolic dysfunction was limited to the endocardial level, counterclockwise rotation of the subepicardium relative to the clockwise rotation of subendocardium was more dominant, which could lead to enhanced counter-clockwise rotation of the LV myocardium and increase the Twist angle [28].

However, the results of this study showed that in both NSTEMI and STEMI groups, regardless of transmural, the rotating and twisting movements were severely damaged. In the NSTEMI group, although mainly the subendocardial AMI was damaged, the outer layer myocardial also showed rotation and torsion movement disorders, which was impaired more obviously in the more severe transmural extent of the STEMI group. Three months after PCI, in the NSTEMI and STEMI groups, the Rot_{Bas}, Rot_{Api}, and Twist were improved, suggesting that PCI therapy can effectively save the state at a low perfusion in the infarct-adjacent myocardium. After the opening of IRA, the rotating and twisting movement was improved, which may be related to the establishment of the collateral circulation and the recovery of myocardium function. In addition, in the NSTEMI group, the Rot_{Api} improved especially compared with 1 week after PCI. This may be related to fact that the apical subepicardial myocardial fibers with respect to the subendocardial myocardial fiber rotation advantage gradually recovered. In the STEMI group, the Rot_{Api} improved to some extent, but the difference was not statistically significant compared with 1 week after PCI, suggesting severe transmural myocardial necrosis, the advantage of the outer myocardium relative to the inner movement no longer exist. In particular, apical rotating movement, which plays a leading role in the LV twisting movement, was significantly weakened.

**Limitations**

Several limitations of this study must be considered. First, RT-3DE frame rate is low, 60°×60°-Spatial Imaging, some patients with multivessel disease cannot fully display excessive expansion of the LV. The imaging effect needs to be supplemented by a water bag. Second, 2D-STI is based on the two-dimensional gray-scale image of the speckle tracking imaging, which cannot reflect the stereo motion of the heart and the tracking accuracy is lower than that seen in real time 3D speckle tracking technology. Finally, if the STI technical frame rate is too low, it may render the image tracking unstable, and if the
frame rate is too high, it will reduce the image resolution, causing “lost track” imaging, resulting in the side-wall radial, circumferential strain reducing the accuracy of the data. Repeatability of the measurements is also reduced.

Conclusions

In this study, although the opening of the IRA was early, one week after PCI, the LV volume and myocardial multi-dimensional deformation functions are severely damaged in the NSTEAMI and STEAMI patients, especially in the STEAMI patients. Three months after PCI, the functions in both groups had been recovered partially, but the LPSS and Rot-API only in the NSTEAMI patients were improved significantly, and the NSTEAMI patients have a better prognosis than do STEAMI patients after treatment over a long time. The LV volume functions, longitudinal, radial, circumferential, and twist movement in the elderly AMI patients with different degree of transmural damage 1 week and 3 months after PCI can be objectively and sensitively revealed by using RT-3DE and 2D-STI respectively. It can provide more useful and accurate reference information for comprehensively analyzing the patient's condition, predicting the progress of the disease and guiding the therapy in clinical practice.

Abbreviations

2D-STI: two-dimensional speckle tracking imaging; AMI: acute myocardial infarction; CPSS: circumferential peak systolic strain; CPSS\textsubscript{Api}: circumferential peak systolic strain in the apical segments; CPSS\textsubscript{Mid}: circumferential peak systolic strain in the middle segments; CPSS\textsubscript{Bas}: circumferential peak systolic strain in the basal segments; ECG: electrocardiogram; EDV: end-diastolic volume; EF: ejection fraction; ESV: end-systolic volume; IRA: infarct-related artery; LPSS: longitudinal peak systolic strain; LPSS\textsubscript{Api}: longitudinal peak systolic strain in the apical segments; LPSS\textsubscript{Mid}: longitudinal peak systolic strain in the middle segments; LPSS\textsubscript{Bas}: longitudinal peak systolic strain in the basal segments; LV: left ventricular; Myo-IRA: myocardium supplied by infarct-related artery; NSTEAMI: non ST elevation acute myocardial infarction; PCI: percutaneous coronary intervention; Rot: rotational peak degree; Rot\textsubscript{Api}: rotational peak degree in the apical segments; Rot\textsubscript{Bas}: rotational peak degree in the basal segments; RPSS: radial peak systolic strain; RPSS\textsubscript{Api}: radial peak systolic strain in the apical segments; RPSS\textsubscript{Mid}: radial peak systolic strain in the middle segments; RPSS\textsubscript{Bas}: radial peak systolic strain in the basal segments; RT-3DE: real-time three-dimensional echocardiography; SCA: Selective coronary arteriography; STEAMI: ST elevation acute myocardial infarction; SV: stroke volume; Twist: peak torsion angle.

Declarations

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**Availability of data and materials**

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

**Authors’ contributions**

Yujiao Deng and Xin Huang as the co-first author, Yukun Luo and Jian Cao as the corresponding authors, they are designed this study and analyzed and interpreted the patient data, drafting the manuscript, control and guarantee that all aspects of the work were investigated and resolved. Lu Liu, Shasha Sun, Jiakun Luo, Fan Tian, Yufei Ma, Bo Guan, Yan Yu acquisition of data, analysis and interpretation of data, revising the manuscript, control and guarantee that all aspects of the work were investigated and resolved. All authors read and approved the final manuscript.

**Ethics approval and consent to participate**

This study has been approved by the Chinese PLA General Hospital Medical Ethics Committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. All persons gave their informed consent prior to their inclusion in the study.

**Consent for publication**

Not applicable.

**Competing interests**

The authors declare that they have no conflict of interests.

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Tables

Due to technical limitations, table 1,2,3,4,5 is only available as a download in the Supplemental Files section.