

Usefulness of Non-Invasive Myocardial Work and Systemic Inflammation Assessment in Predicting Left Ventricular Dysfunction in Patients with Acute Coronary Syndrome

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ABSTRACT

Background: Left ventricular (LV) remodeling plays a pivotal role in the pathophysiology of heart failure (HF) following acute coronary syndrome (ACS). Left ventricular ejection fraction (LVEF), left ventricular (LV) volumes and, more recently, speckle tracking echocardiography (STE) are used to describe LV performance. Myocardial work (MW) is a new noninvasive imaging method that integrates loading conditions and can be used to detect early myocardial dysfunction before LVEF decreases.

Aim: This study aims to characterize the relationship between MW, high-sensitivity troponin (hs-cTn I) and C-reactive protein (CRP) as an inflammation biomarker. Their use as predictors for LV dysfunction in the acute phase of ACS is of particular interest but is still under debate.

Methods: Complete clinical examination and two-dimensional echocardiography (2-DE), with speckle-tracking and myocardial work measurements, were performed in the first 24 hours after admission. Locally available biomarkers were assessed in the same timeframe, with special interest in hs-cTn I and CRP, as a marker for inflammation. A follow-up visit, including the same clinical, biological, and echocardiographic measurements, was performed six to eight weeks after the index event.

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Results: We evaluated 56 patients (53 ± 10 years, 45 men) with ACS. Baseline hs-cTn I significantly correlated with baseline global longitudinal strain (GLS) ($r=0.43$, $p=0.001$) and baseline MW parameters (GWI: $r=-0.44$, $p=0.001$; GCW: $r=-0.40$, $p=0.002$). A correlation between hs-cTn I and LVEF was not statistically relevant. C-reactive protein, which was used to assess systemic inflammation, also failed to correlate with LVEF. However, CRP significantly correlated with relevant MW parameters (GWE: $r=-0.53$, $p<0.001$ and GWW: $r=0.48$, $p<0.001$). C-reactive protein levels above 28 mg/L correlated with a decrease in MW performance assessed by GWE, suggesting a possible tendency to adverse remodeling.

Conclusions: C-reactive protein level in the first 24 hours after ACS and its correlation with MW parameters may be a potential indicator of future LV dysfunction and heart failure.

Keywords: cognitive function, bilingual, multilingual, Stroop test.

ABBREVIATIONS

2-DE: two-dimensional echocardiography
 GWI: global work index
 3-DE: three-dimensional echocardiography
 GWW: global wasted work
 ACS: acute coronary syndrome
 LVEF: left ventricular ejection fraction
 AMI: acute myocardial infarction
 LV: left ventricle
 CMR: cardiac magnetic resonance
 MW: myocardial work
 CV: cardiovascular
 PCI: percutaneous coronary intervention
 EF: ejection fraction
 SPSS: statistical package for the social sciences
 GLS: global longitudinal strain
 STE: speckle-tracking echocardiography
 GCW: global constructive work
 STEMI: ST-elevation myocardial infarction
 GWE: global work efficiency
 TTE: transthoracic echocardiography

BACKGROUND

Left ventricular remodeling after acute coronary syndromes (ACS) is a major independent predictor of heart failure. Other predictors used are the patient's history, the dynamics of electric changes, markers of myocyte necrosis and reduced ejection fraction calculated using 2-DE (1). Recently, speckle tracking echocardiography (STE), using global longitudinal strain (GLS), has been used in assessing infarct size, predicting adverse remodeling (2), since it provides a detailed assessment of global and regional myocardial LV deformation, sometimes undetectable with

2-DE LVEF (3). New tools such as noninvasive myocardial work (MW), derived from STE, have been recently used to assess global and regional myocardial performance, considering loading conditions. Until recently, myocardial work has been studied in patients with heart failure, left ventricular dyssynchrony, hypertrophic cardiomyopathy, dilated and hypertensive cardiomyopathy (3). Myocardial work has demonstrated an independent value in predicting LV recovery over GLS in patients with acute coronary syndromes, which makes it a possible, reliable tool in predicting ACS-related HF (3). Based on these results, identifying correlations between MW and widely available biomarkers is essential in order to identify patients at risk for myocardial dysfunction and worse outcomes after an ischemic event.

High-sensitivity troponin has been used to estimate the size of infarcted myocardium, but its accuracy in predicting LV remodeling is modest (4). However, previous studies have shown that CRP significantly correlated with LV remodeling parameters when assessed at two months after PCI. Although resolution of local and systemic inflammation in myocardial infarction is expected after four weeks, a prolonged inflammation phase can also be seen in patients post-ACS. Most studies on inflammatory response in ACS targeted myocardial assessment in the first one up to three months after the index event (5-7).

Based on the recently documented prognostic value of STE and MW (2, 3, 8), our study aimed to assess myocardial work, in addition to conventional parameters of systolic function, in the acute setting of patients with ACS, the relationship with high-sensitivity troponin and systemic inflammation, assessed by CRP. □

METHODS

Patient population and design

This is a single-center prospective study enrolling patients with an ACS (with or without ST-segment elevation). The diagnosis of myocardial infarction was based on the ACS definition according to guidelines: rise and fall in high-sensitivity troponin (hs-cTn I), with at least one value above the 99th percentile upper reference limit; symptoms of myocardial ischemia; new ischemic electrocardiogram (ECG) changes; pathological Q waves or imaging evidence of new loss of viable myocardium (9). The local ethics committee approved the study, and all patients signed the informed consent before being included.

The inclusion criteria were age over 18 years, sinus rhythm and ACS diagnosis following guideline criteria (9). All patients underwent coronary angiography via radial or femoral access and received optimal medical treatment according to current recommendations (10, 11). The exclusion criteria were hemodynamically unstable patients, non-sinus rhythm, severe valvular heart disease, and other pathologies leading to systemic inflammation and decreased life expectancy. Patients who were technically unsuitable for STE analysis were also excluded. Baseline patient characteristics, demographics, laboratory results, 2-DE LVEF, GLS, MW parameters, angiographic findings and medication were recorded.

The first assessment was performed in the first 24 hours after the patient was admitted with an ACS diagnosis. A second visit with the same clinical, biological and echocardiographic assessment was appointed 6-8 weeks after the baseline visit.

Biological assessment included basic laboratory parameters: complete blood count, blood sedimentation rate, coagulation, basal glycemia, glycated hemoglobin, liver enzymes, renal parameters (creatinine, urea, uric acid, ionogram), cholesterol and its fractions, triglycerides and specific parameters like iron and ferritin if needed. Parameters of particular interest in our study were hs-cTn I (Pathfast; LSI Medience) at the exact moment of admission in the emergency department and CRP within 24 hours after hospitalization.

Echocardiography

A comprehensive precordial echocardiography was performed in all patients within 24 hours after admission and at a median follow-up of eight

weeks using a commercially available machine (Vivid E9 or Vivid E95, GE Healthcare) equipped with M5S transducers and analyzed offline on a workstation EchoPAC version 203 (GE Healthcare). All recordings and measurements were performed according to the current systolic and diastolic profile guidelines (12). Left ventricle end-diastolic and end-systolic volumes were measured, and LVEF was calculated using the biplane Simpson method applied to apical four- and two-chamber views for 2-DE. Global longitudinal strain was obtained from two-dimensional grayscale images in apical four-chamber, three-chamber and two-chamber views at a frame rate of 60–70 frames *per second*. Myocardial motion was automatically tracked in the region of interest and adjusted by correcting the endocardial widths or boundaries as necessary.

Myocardial work analysis

Quantifying myocardial work was made using 2D LV longitudinal strain and a noninvasively estimated peak LV pressure, recorded using the patient's brachial blood pressure before each transthoracic echocardiography (TTE). Valvular events were defined using pulsed-wave Doppler acquisitions. This software is based on the theoretical ventricular pressure curve, which is adjusted for each patient based on valvular timing events and peak systolic blood pressure, measured with an arm cuff immediately before the acquisitions. The software provided the following parameters:

- GWI (global work index): the total work of the myocardium within the pressure-strain loops, from mitral closure to mitral opening;
- GCW (global constructive work): systolic work used in shortening of LV during systole and negative work of lengthening of the LV during isovolumic relaxation, meaning the work performed by the myocardium that is actually productive for the LV ejection fraction;
- GWW (global wasted work): the work that was unproductive to LV ejection fraction, a combination of lengthening during systole and shortening during isovolumic relaxation;
- GWE (global work efficiency): the ratio of constructive work to total work (both constructive and wasted work): $GCW / (GCW + GWW)$ (13).

The same parameters were recorded and measured at the follow-up visit within six to eight weeks after the index hospitalization.

Statistical analysis

All statistical analyses were performed using SPSS version 25.0 (SPSS, Chicago, IL). Continuous variables were expressed as mean $\pm$ SD. Numbers and percentages were used for categorical variables and were compared with the χ^2 test. Nonparametric analysis of overall sensitivity and specificity values and area under the curve (AUC) were applied. Pearson's correlations were used to measure the strength and direction of linear relationship between two continuous variables. R-value > 0.40 was considered suggestive for a good correlation and p-values < 0.05 were considered of statistical significance. $\square$

RESULTS

Characteristics of the population

Fifty-six patients (53 ± 10 years, 45 men) admitted with ACS (89.2% with ST-elevation ACS) were analyzed. Key characteristics, including comorbidities and angiographic profiles, are summarized in Table 1. All patients received optimal medical treatment according to current guidelines, including thrombolysis (in 14 patients) (10, 11). The median time from symptom onset to hospital admission was six hours. The clinical parameters listed in Table 1 are measured prior to TTE. C-reactive protein mean values measured within the first 24 hours after admission were 16.75 mg/dL [range 5.58-29.48 mg/dL], while hs-cTn I values were 3311 μ g/L [range 486.8-10990 μ g/L].

Echocardiographic characteristics and myocardial work indices

Echocardiographic parameters, universally assessed in standard examination and recommended by guidelines, were obtained (12). Speckle tracking echocardiography and myocardial work indices recorded at baseline and at follow-up are shown in Table 2. As expected, baseline parameters measured within 24 hours after hospital admission were significantly lower than the ones recorded at follow-up. All values were below reference values in healthy subjects (14).

Correlation between systemic inflammation and myocardial work

Systemic inflammation assessed by CRP significantly correlated with changes in MW parameters between the two visits, with a negative correlation

TABLE 1. Characteristics of the study population

Clinical characteristics	All patients (N=56)
Age, years	53 $\pm$ 10
Sex, male	45 (80.6)
Hypertension	34 (60.7)
Diabetes	26 (45.6)
Dyslipidemia	52 (92.8)
Smoking	39 (69.6)
Systolic blood pressure	127.5 $\pm$ 17.4
Diastolic blood pressure	77.5 [70-80]
Acute myocardial infarction (AMI) type – ST-segment elevation	50 (89.2)
wwThrombolysis	14 (25)
One-vessel disease	34 (61.8)
Two-vessel disease	17 (30.9)
Three-vessel disease	4 (7.2)
CRP within 24 hours (lab range 1-5 μ g/dL)	16.75 [5.5-29.4]
hs-cTn I at admission (μ g/L)	3311 [486-10990]
Aspirin	56 (100)
Clopidogrel or Ticagrelor	55 (98.2)
Beta-blocker	54 (96.4)
Angiotensin converting enzyme inhibitor/angiotensin receptor blocker	40 (71.4)
Statin	56 (100)
Mineralocorticoid receptor antagonist	17 (30.3)

Data are presented as mean $\pm$ SD, median [interquartile range], or n (%)

TABLE 2. Speckle tracking echocardiography and myocardial work indices

Myocardial work indices	Baseline*	Follow-up*
GWl, mm Hg%	1112 $\pm$ 372.6	1521 $\pm$ 467
GCW, mm Hg%	1248 $\pm$ 408.8	1666 $\pm$ 517
GWW, mm Hg%	117 [77.2-138.8]	91 [59.7-140]
GWE, %	89 [82.7-92]	93 [89.7-95]
GLS	-10 $\pm$ 3.8	-14.2 $\pm$ 4.11

GWl=global work index; GCW=global constructive work; GWW=global wasted work; GWE=global work efficiency; GLS=global longitudinal strain.

Data are presented as mean $\pm$ SD, median [interquartile range].

*Shapiro-Wilk test performed with p > 0.5 for all variables.

for changes in GWE ($r = -0.53$, $p < 0.001$) (Figure 1) and a positive correlation for changes in GWW ($r = 0.48$, $p < 0.001$) (Figure 2).

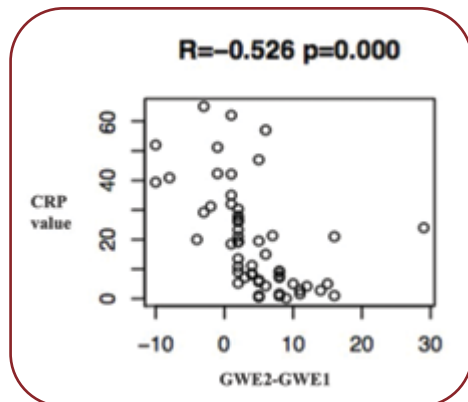


FIGURE 1. Correlation between C-reactive protein (CRP) and global work efficiency (GWE) changes between visits. GWE=global work efficiency; GWE1=GWE at baseline; GWE2=GWE at follow-up.

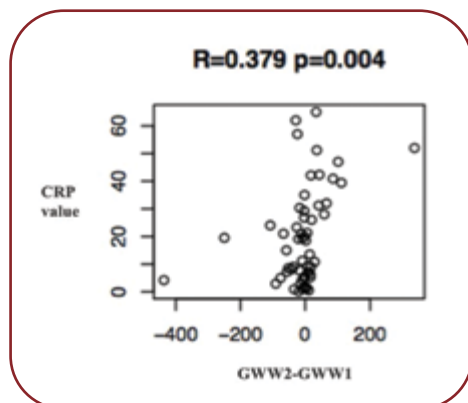


FIGURE 2. Correlation between C-reactive protein (CRP) and global wasted work (GWW) changes between visits. GWW1=GWW at baseline; GWW2=GWW at follow-up.

A CRP level >28 mg/L (laboratory range 1-5 mg/dL) predicted a decrease in global work efficiency from baseline to visit two, which could determine its future use as a possible marker of LV dysfunction, as assessed by myocardial work (Figure 3).

Myocardial work bull's-eye plots in patients with low CRP values are a highly relevant example of MW's role in subclinical LV dysfunction, showing a noticeable improvement in GWE parameters between baseline and follow-up. When comparing MW bull's eye plots obtained in patients with high inflammatory response and, therefore, high CRP values, the images obtained by us suggested a decrease in global efficiency (Figure 4).

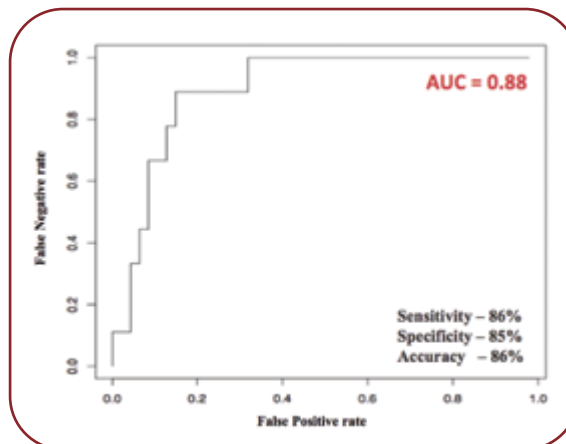


FIGURE 3. A C-reactive protein (CRP) value over 28 mg/L was able to predict a decrease of global work efficiency from baseline to visit 2.

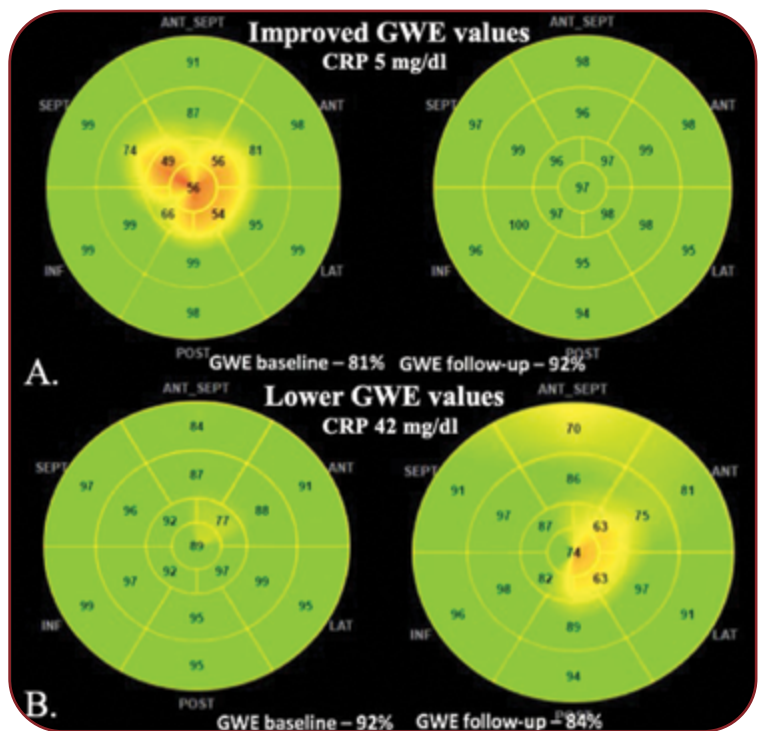


FIGURE 4. Changes in global work efficiency (GWE) parameters between visits: improved values in patients with lower C-reactive protein (CRP) values (A) and lower GWE in patients with high CRP values (B)

Correlation between high-sensitivity troponin, LVEF by 2-DE, GLS and MW

In our study, myocardial necrosis, which was assessed by hs-cTn I at baseline, did not correlate with LVEF, but it significantly correlated with baseline GLS ($r=0.43$, $p=0.001$) and two baseline MW parameters (GWI: $r=-0.43$, $p=0.001$; and GCW: $r=-0.40$, $p=0.002$) (Figure 5). □

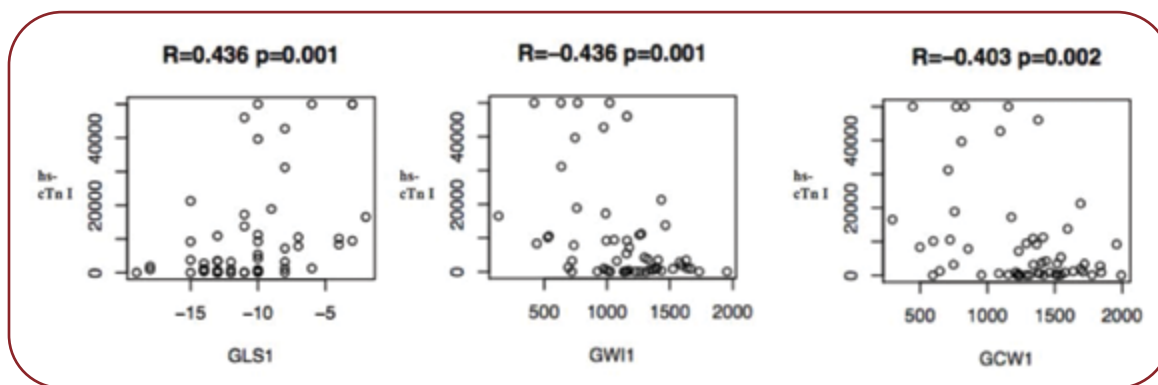


FIGURE 5. Correlation between high-sensitivity troponin (hs-cTn I) and global longitudinal strain (GLS), global work index (GWI) and global constructive work (GCW) at baseline. GLS1=GLS at baseline; GWI1=GWI at baseline; GCW1=GCW at baseline.

DISCUSSION

Our prospective study of patients with ACS (with or without ST-segment elevation) has shown that myocardial work analysis is a useful tool in assessing LV performance. Although biomarkers that could predict LV remodeling are still under debate, the present analysis shows a statistically significant correlation between MW parameters and systemic inflammatory response assessed by CRP.

C-reactive protein and noninvasive myocardial work in patients with ACS

In our study, CRP values correlated with LV dysfunction, which were assessed by changes in GWE measured in the first 24 hours after admission and two months after the acute event. Patients with lower CRP values had improved MW parameters expressed by a higher GWE value at follow-up than at baseline. In contrast, GWW assessed after two months was increased in patients with higher baseline CRP values, indicating an inefficient myocardial contraction. Although data on MW in patients with ACS is still scarce, it has been previously demonstrated that those who developed LV remodeling after ST-segment elevation myocardial infarction showed more impaired myocardial work compared with patients without LV remodeling (15). Impaired MW indices may be observed more frequently in patients with LV remodeling because of anaerobic myocardial metabolism, as proven by the correlation with anaerobic metabolism assessed by positron emission tomography using F18 fluorodeoxyglucose (16). Beyond

GLS, MW is independently correlated with LV recovery (3). Butcher S. and his colleagues have recently shown that higher MW parameters in patients with an LVEF $\leq 40\%$ following ST-elevation segment ACS were associated with greater chances of LVEF improvement and even normalization after six months from the index event (17). A recent study conducted by Lustosa et al showed that reduced GWE ($<86\%$) measured by TTE within 48 hours of admission in ST-segment elevation myocardial infarction patients was associated with worse long-term survival. The same analysis reported that GWE had a more significant incremental benefit than LVEF and higher GWE values correlated with improved outcomes, regardless of other clinical factors like age, LVEF or GRACE score (18). In a more recent study on patients with ACS (with and without ST-segment elevation), in which major events (ME) like cardiovascular death, HF and unplanned coronary revascularization were observed, GWE was the only transthoracic parameter that has been independently associated with the long-term occurrence of ME (19). Considering this, finding biomarkers that could correlate with MW indices like GWE might be of particular interest in easily identifying patients at risk of worse outcomes. Although myocardial wasted work has not been thoroughly analyzed in patients with ACSs or ischemic heart disease, available data suggest that higher GWW correlate with the extent of coronary lesions and subclinical ischemia (20).

Since systemic inflammation is one of the key chains of AMI pathophysiology, we assessed CRP values in the acute setting. In our study, the

cut-off CRP value for progression of LV dysfunction was 28 mg/dL (laboratory range 0-5 mg/dL). Based on these measurements, a five- to six-fold increase in CRP should indicate patients who could be at risk for LV dysfunction or adverse remodeling after ACS. These findings align with previously reported data. According to a study conducted by Orn *et al*, C-reactive protein two days after PCI in patients with STEMI was significantly correlated with the infarct size and parameters of LV remodeling (21). When assessing remodeling determinants, besides echocardiography parameters, inflammation has been proven to be an important element leading to HF in ACS patients, especially in ST-segment elevation myocardial infarction (22).

High sensitivity troponin and speckle tracking parameters in patients with acute coronary syndrome

Our data showed no correlation between hs-cTn I at admission and LVEF and suggested a correlation only with baseline GLS and baseline MW parameters like GCW and GWI. The levels of Hs-cTn I did not correlate with changes in these measurements between admission and follow-up. This finding questions the prognostic value of hs-cTn I in assessing a potential LV remodeling following ACS, which aligns with current data. Although high levels of cardiac troponins can predict adverse outcomes after ACS (23), a strong correlation with cardiac dysfunction has not been consistent in recent studies (24). Since it has been previously shown that impaired longitudinal function assessed by GLS was significantly and independently related to in-hospital HF patients with MI, including those with preserved LVEF (25), the relationship between hs-cTn I and baseline GLS could identify patients at risk for adverse events. Until now, the value of GWI has been proven in predicting mid-term cardiovascular events after ACS, while the role of GCW is still under debate; therefore, our study did not support the use of hs-cTn I in assessing the progression of LV dysfunction (26).

Changes in GLS did not correlate with CRP values at admission, which may suggest a higher sensitivity of MW in detecting changes in LV performance.

Study limitations

One of our study's limitations is the small number of patients with heterogeneous angiographic profiles that must be considered when assessing myocardial damage and viability. Another limitation is the relatively early follow-up, without a third visit, which could confirm remodeling after extended timeframes. Most patients with ST-elevation myocardial infarction present with LV remodeling in the first three to six months; some patients develop LV remodeling after more than six months (27). However, the patients are still under evaluation, with perspectives of new data on late outcomes. Although high-sensitivity CRP is recommended in inflammation assessment, due to its limited availability in many centers, our study shows that non-high-sensitivity CRP is also a reliable marker of acute myocardial injury. □

CONCLUSION

This prospective study of patients with ACS indicates that CRP levels measured within the first 24 hours of hospital admission show a significant correlation with changes in myocardial work parameters like global work efficiency and global wasted work, when reassessed two months after ACS. These findings suggest that early CRP assessment may serve as a prognostic biomarker for identifying patients at risk of developing myocardial dysfunction. These findings may have two key clinical implications: firstly, the necessity for a more comprehensive assessment of patients at risk for adverse cardiac remodeling, potentially using myocardial work parameters and early measurement of CRP; and secondly, the future perspectives of anti-inflammatory therapeutic strategies aimed at mitigating myocardial injury. □

Conflicts of interest: none declared.

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