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Noninvasive Myocardial Work Indices 3 Months after ST-Segment Elevation Myocardial Infarction: Prevalence and Characteristics of Patients with Postinfarction Cardiac Remodeling

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Background: Assessment of left ventricular (LV) remodeling after ST-segment elevation myocardial infarction (STEMI) is pivotal for patient management. Noninvasive myocardial work indices obtained from echocardiography-derived strain-pressure loops provide a new tool that permits characterization of LV mechanics. We aimed at characterizing myocardial work indices in patients with LV remodeling after STEMI versus patients without remodeling.

Methods: Six-hundred STEMI patients were retrospectively analyzed (456 men, mean age: 61 ± 11 years) and divided according to the presence of LV remodeling 3 months after the index admission (≥20% increase in LV end-diastolic volume). Noninvasive myocardial work indices were measured at 3 months after STEMI.

Results: LV remodeling was observed in 150 patients (25%) who showed more impaired global myocardial work indices compared with their counterparts: work index (1,708 ± 522 mm Hg% vs 1,979 ± 450 mm Hg %; $P < .001$), constructive work (1,941 ± 598 mm Hg% vs 2,272 ± 519 mm Hg%; $P < .001$), and work efficiency (92% [range 88%-96%] vs 95% [range 93%-96%]; $P < .001$). In addition, patients with LV remodeling had significantly increased wasted work (116 mm Hg% [range 73-184 mm Hg%] vs 91 mm Hg% [range 61-132 mm Hg%]; $P < .001$). The frequency of impaired global work index, constructive and work efficiency, and increased wasted work was significantly higher among patients with LV remodeling compared with their counterparts: 21.3%, 34.7%, 34.7%, and 14.0%, respectively, versus 5.3%, 9.6%, 8.9%, and 4.9%, respectively ($P < .001$).

Conclusions: At 3-month follow-up after STEMI, patients with LV remodeling revealed more impaired myocardial work indices compared with patients without LV remodeling. The prevalence of impaired myocardial work indices was higher among patients with LV remodeling compared with patients without. (J Am Soc Echocardiogr 2020;33:1172-9.)

Keywords: ST-segment elevation myocardial infarction, Myocardial work indices, Cardiac remodeling

Left ventricular (LV) cardiac remodeling after ST-segment elevation myocardial infarction (STEMI) is an important risk factor for the development of heart failure and all-cause mortality.[1] Infarct size, microvascular obstruction, and inflammation are important determinants of LV remodeling after STEMI.[2] A recent study including 374 STEMI patients treated with primary percutaneous coronary intervention (PCI) and imaged with cardiovascular magnetic resonance showed small improvement in LV ejection fraction (LVEF) despite small increases in LV volumes at 6 months of follow-up.[3] This suggests that there are compensatory mechanisms that

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Abbreviations
2D = Two-dimensional
GCW = Global constructive work
GWE = Global work efficiency
GWI = Global work index
GWW = Global wasted work
LV = Left ventricular
LVEDV = Left ventricular end-diastolic volume
LVEF = Left ventricular ejection fraction
LVESV = Left ventricular end-systolic volume
LV GLS = Left ventricular global longitudinal strain
PCI = Percutaneous coronary intervention
STEMI = ST-segment elevation myocardial infarction

maintain LV systolic function despite the LV remodeling. Most of the studies evaluating the association between LV remodeling and LV systolic function do not consider loading conditions (such as blood pressure), which could influence that association.

Myocardial energetics and mechanics can be evaluated using two-dimensional (2D) speckle-tracking echocardiography, taking loading conditions into account. This approach may provide further insight into the process of LV remodeling following STEMI and may shed light on how myocardial mechanics and energetics adapt in order to maintain LV systolic function. Noninvasive myocardial work assessment combines the noninvasive measurement of systolic blood pressure (as an estimate of LV pressure [provided there is no LV outflow obstruction]) with global longitudinal strain (GLS) data, which can provide information about myocardial energy efficiency.[4] Global work index (GWI), global constructive work (GCW), global wasted work (GWW), and global work efficiency (GWE) are indices that can be accessed noninvasively with 2D speckle-tracking imaging for myocardial work evaluation.

The aim of this study was to assess myocardial work noninvasively 3 months following the index admission for STEMI and to examine the differences between patients with and those without LV remodeling at that time point.

METHODS

Patient Population

A total of 600 patients admitted with a diagnosis of STEMI treated with primary PCI from September 2012 to June 2017 were included in this retrospective evaluation. Patients with known severe valvular heart disease and particularly any grade of aortic stenosis and previous cardiac surgery before the index event were excluded.

All patients were treated according to the Leiden University Medical Center institutional protocol for patients admitted with STEMI,[5] which is based on contemporary guidelines and provides a clinical framework for optimal guideline-based medical therapy and standardized outpatient follow-up.[6,7] This framework includes the measurement of peak cardiac biomarker levels (troponin T and creatine phosphokinase) and comprehensive baseline 2D echocardiography with estimation of LVEF and biplane LV volumes, within 48 hours of admission. During angiography, the presence of multivessel disease, defined as ≥50% luminal stenosis in addition to the culprit vessel, was recorded. The Killip classification was used to estimate the prevalence of uncomplicated myocardial infarction (Killip I) and symptomatic heart failure (Killip class ≥II) at the time of admission. Clinical, angiographic, and echocardiographic data were retrospectively analyzed in the departmental cardiology information system (EPD-Vision, Leiden, The Netherlands).

At 3 months after index admission for STEMI, patients were referred for echocardiography as part of the institutional protocol, and parameters including heart rate, systolic and diastolic blood pressure, and medical treatment were recorded. From the 2D echocardiographic data, LVEF, biplane LV volumes, LV GLS, and myocardial work indexes were assessed with commercially available software (EchoPAC version 202 software; GE Vingmed Ultrasound, Horten, Norway). Patients were divided according to the presence or absence of LV remodeling, defined as ≥20% increase in LV end-diastolic volume (LVEDV) from baseline.[8]

Echocardiographic Analysis

Patients were scanned in the left lateral decubitus position. Standard 2D grayscale and Doppler images were acquired using a commercially available system (Vivid E95, GE Vingmed Ultrasound) equipped with 3.5 MHz or M5S transducers and analyzed offline using EchoPAC version 202 software (GE Vingmed Ultrasound). All recordings and measurements were made according to current guidelines.[9] Left ventricular end-systolic volume (LVESV), LVEDV, and LVEF were measured from the apical four- and two-chamber views, using the modified Simpson's biplane method.[9] In addition, LV sphericity index was measured on the apical four-chamber view, as described elsewhere.[10] Diastolic function was assessed according to current recommendations.[11] Images from the apical four- and two-chamber and long-axis views were acquired with a frame rate of 56 frames/sec (range 56-60 frames/sec) to assess LV GLS by speckle-tracking echocardiography. Left ventricular GLS was measured using EchoPAC version 202 software (GE Vingmed Ultrasound) and averaged from the peak systolic longitudinal strain of all 17 segments.

Myocardial Work Analysis

Quantification of myocardial work indices was performed using a commercially available software package (EchoPAC version 202 software, GE Medical Systems). As described in previous work,[4,12] myocardial work indices are calculated by integrating LV GLS data and noninvasively estimated LV pressure. Strain was measured using 2D speckle-tracking echocardiography by manually tracing the LV endocardial border in the apical long-axis and two- and four-chamber views. Noninvasively estimated peak LV pressure was measured using the patient's brachial cuff blood pressure recordings with peak systolic LV pressure assumed to be equal to peak arterial pressure. The opening and closing timings of the aortic and mitral valves were identified from the apical three-chamber view or parasternal long-axis views to define the different phases of the cardiac cycle. The LV pressure curve is then constructed using a normalized reference curve adjusted to the different phases of the cardiac cycle. Left ventricular myocardial work was then quantified by calculating the rate of segmental shortening by differentiating the strain curve and multiplying the resulting value by the instantaneous LV pressure. The result is a measure of instantaneous power, which was integrated over time to obtain myocardial work as a function of time.

HIGHLIGHTS

- Impaired myocardial work indices are frequent in patients with LV remodeling.
- Persistent anaerobic myocardial metabolism could explain impaired myocardial work.
- Interestingly, patients without LV remodeling may have impaired myocardial work.

The following parameters were calculated:

GWI: defined as total work within the area of the LV pressure-strain loops, calculated from mitral valve closure to mitral valve opening;
GCW: defined as work performed during shortening in systole, adding negative work during lengthening in isovolumic relaxation;
GWW: defined as negative work performed during lengthening in systole, adding work performed during shortening in isovolumetric relaxation;
GWE: calculated as the sum of constructive work in all LV segments, divided by the sum of constructive and wasted work in all LV segments, expressed as a percentage.

In addition, regional LV constructive and wasted work was analyzed and compared between patients presenting with versus without LV remodeling at 3 months of follow-up.

Statistical Analysis

Continuous data were presented as mean ± SD if normally distributed and as median and interquartile range if nonnormally distributed. Categorical data were presented as frequencies and percentages and were compared with the χ^2 test. Continuous data were compared using the Student's *t* test if normally distributed or the Mann-Whitney *U* test if nonnormally distributed. Comparisons between patients with LV remodeling versus patients without LV remodeling were analyzed by the Student's *t* test or Mann-Whitney *U* test.

All statistical analyses were performed with SPSS software version 23.0 (IBM SPSS Statistics for Windows, Armonk, NY). A *P* value < .05 was considered statistically significant.

RESULTS

Study Population

Of the 600 STEMI patients (456 men [76%], mean age, 61 ± 11 years), 150 (25%) showed LV remodeling at 3 months after the index event. The clinical characteristics for each subgroup are summarized in Table 1. Patients showing LV remodeling had significantly higher values of creatine phosphokinase (2,059 U/L [range, 1,107-3,868 U/L] vs 965 U/L [range, 429-1,926 U/L]; $P < .001$) and troponin T (5.34 μg/L [range, 2.53-9.86 μg/L] vs 2.49 μg/L [range, 1.07-5.45 μg/L]; $P < .001$) at baseline in comparison with patients without LV remodeling.

Table 1 Clinical characteristics

Clinical characteristics	All patients (*N* = 600)	No LV remodeling (*n* = 450)	LV remodeling (*n* = 150)	*P* value
Age, years	61 ± 11	61 ± 11	59 ± 11	.105
Sex, male	456 (76)	332 (73.8)	124 (82.7)	.027
Killip classification I	562 (93.7)	425 (94.4)	137 (91.3)	.175
Heart rate, bpm	66 [59-76]	66 [60-76]	66 [58-74]	.387
Systolic blood pressure, mm Hg	135 [124-149]	135 [124-150]	135 [123-146]	.279
Diastolic blood pressure, mm Hg	80 ± 12	80 ± 12	80 ± 12	.654
Maximum creatine phosphokinase value at baseline, U/L	1,151 [511-2,416]	965 [429-1,926]	2,059 [1,107-3,868]	<.001
Maximum troponin T at baseline, μg/L	2.98 [1.27-6.5]	2.49 [1.07-5.45]	5.34 [2.53-9.86]	<.001
Hypertension	222 (37)	166 (36.9)	56 (37.3)	.066
Diabetes	43 (7.2)	31 (6.9)	12 (8.0)	.199
Dyslipidemia	122 (20.3)	90 (20)	32 (21.3)	.833
Smoking	248 (41.3)	181 (40.2)	67 (44.7)	.742
Positive family history	262 (43.7)	201 (44.7)	61 (40.7)	.511
Left anterior descending coronary artery	284 (47.3)	206 (45.8)	78 (52)	.272
Right coronary artery	229 (38.2)	180 (40)	49 (32.7)	.272
Left circumflex coronary artery	87 (14.5)	64 (14.2)	23 (15.3)	.272
Aspirin	582 (97)	442 (98.2)	140 (93.3)	.002
Prasugrel or Clopidogrel	599 (99.8)	449 (99.8)	150 (100)	.563
Beta-blocker	560 (93.3)	423 (94)	137 (91.3)	.257
Statin	587 (97.8)	440 (97.8)	147 (98)	.871
Angiotensin converting enzyme inhibitor/angiotensin receptor blocker	574 (95.7)	427 (94.9)	147 (98)	.105
Mineralocorticoid receptor antagonist	11 (1.8)	7 (1.6)	4 (2.7)	.380

Data are presented as mean ± SD, median [interquartile range], or *n* (%).

Table 2 Echocardiographic characteristics of the study population 3 months post-STEMI

Echocardiographic characteristics	No LV remodeling (*n* = 450)	LV remodeling (*n* = 150)	*P* value
LV ESV, mL	32 [24-43]	56 [40-76]	<.001
LV EDV, mL	84 [68-101]	118 [97-148]	<.001
Sphericity index	2.19 ± 0.32	1.88 ± 0.27	<.001
E wave, cm/sec	64 ± 18	67 ± 21	.115
A wave, cm/sec	74 ± 21	69 ± 18	.013
E/A ratio	0.82 [0.67-1.11]	0.93 [0.72-1.30]	.004
Deceleration time, msec	248 ± 84	230 ± 72	.025
LVEF, %	60 [55-66]	53 [45-61]	<.001
LV GLS, %	–18 [–20 to –16]	–16 [–18 to –13]	<.001

Data are presented as mean ± SD or median [interquartile range].

Table 3 Global LV myocardial work indices and postinfarction LV remodeling at 3 months

Myocardial work indices	No LV remodeling (*n* = 450)	LV remodeling (*n* = 150)	*P* value
GWI, mm Hg%	1,979 ± 450	1,708 ± 522	<.001
GCW, mm Hg%	2,272 ± 519	1,941 ± 598	<.001
GWW, mm Hg%	91 [61-132]	116 [73-184]	<.001
GWE, %	95 [93-96]	92 [88-96]	<.001

Data are presented as mean ± SD or median [interquartile range].

The echocardiographic characteristics of the study population at 3 months follow-up are shown in Table 2. Patients with LV remodeling had significantly larger LVEDV and LVESV, significantly lower LVEF, and more impaired LV GLS in comparison with patients without LV remodeling.

Myocardial Work Indices

Myocardial work indices of the study population at 3-month follow-up are shown in Table 3. Differences in GWI, GCW, GWW, and GWE were statistically significant between the two groups. In comparison with patients without LV remodeling, patients with LV remodeling presented significantly more reduced GWI, GCW, and GWE and more increased GWW.

Table 4 summarizes the regional values of LV myocardial constructive and wasted work in patients presenting with versus without LV remodeling at 3-month follow-up. Among patients with LV remodeling, the regional values of constructive work were significantly lower compared with those in patients without LV remodeling, particularly apical and midventricular segments and the segments supplied by the left anterior descending coronary artery. The regional GWW followed a similar pattern.

Based on previous normal reference values,[13] patients with LV remodeling had more frequently impaired values of GWI, GCW, and GWE and increased GWW compared with patients without LV remodeling (21.3%, 34.7%, 34.7%, and 14.0%, respectively, vs 5.3%, 9.6%, 8.9%, and 4.9%, respectively; $P < .001$; Table 5 and Figure 1). Figure 2 presents examples of the myocardial work indices in a patient with LV remodeling post-STEMI and a patient without LV remodeling.

DISCUSSION

This retrospective study demonstrated that patients who develop LV remodeling after STEMI show more impaired indices of myocardial work compared with patients without LV remodeling. In addition, the percentages of reduced GWI, GCW, and GWE and the percentage of increased GWW were higher among patients with LV remodeling compared with patients without LV remodeling. Interestingly, patients without LV remodeling also showed some degree of myocardial work impairment, which, while unproved, could be related to further adverse remodeling at longer-term follow-up.

Association between Conventional Echocardiographic Measures of LV Systolic Function and LV Remodeling

One of the main determinants of LV remodeling after STEMI is the infarct size. The first approach to estimate the infarct size is the measurement of biomarker release (troponin and creatine phosphokinase).[14,15] However, the kinetics of that release depend on the timing and method of coronary reperfusion as well as the LV myocardial mass.[16] It is well known that patients with LV hypertrophy may show a more pronounced peak of troponin and creatine phosphokinase release compared with patients with normal LV mass.[17,18] Therefore, the accuracy of biomarkers to estimate the infarct size and predict subsequent LV remodeling is modest. When using imaging modalities, echocardiography is usually the first method to estimate the infarct size since this method is widely available and can be performed at the bedside. The LVEF and wall motion score index are established surrogates of infarct size and predictors of LV remodeling.[19,20] However, assessment of LVEF by 2D echocardiography is influenced by LV geometry, and inter- and intraobserver variability are both relatively high. While the use of an echocardiographic enhancement agent may help to delineate the endocardial border and facilitate assessment of wall motion, contrast echocardiography remains underutilized. The new gold standard to assess infarct size is late-gadolinium contrast-enhanced cardiovascular magnetic resonance.[21,22] However, this technique is not widely available and may not be feasible in patients who are hemodynamically compromised. Strain imaging techniques are not influenced by geometrical assumptions, and when performed on echocardiographic data, the analysis is more reproducible than LVEF assessment.[23] Left ventricular GLS measured after STEMI has been associated with LV

Table 4 Regional LV constructive and wasted myocardial work and postinfarction LV remodeling at 3 months

LV segments	Regional constructive work, mm Hg%			Regional wasted work, mm Hg%		
	No LV remodeling (*n* = 450)	LV remodeling (*n* = 150)	*P* value	No LV remodeling (*n* = 450)	LV remodeling (*n* = 150)	*P* value
Basal inferior	2,039 ± 679	1,990 ± 743	.456	82 [29-182]	71 [20-170]	.241
Basal posterior	2,007 ± 725	1,947 ± 685	.376	115 [45-259]	104 [28-240]	.115
Basal lateral	2,059 ± 661	1,988 ± 591	.221	92 [31-184]	55 [21-126]	.001
Basal anterior	1,771 ± 604	1,553 ± 597	<.001	74 [27-151]	84 [30-164]	.205
Basal anteroseptal	1,855 ± 615	1,645 ± 683	<.001	98 [33-190]	112 [43-228]	.041
Basal septal	1,626 ± 504	1,572 ± 533	.271	81 [31-178]	73 [27-170]	.377
Mid inferior	2,161 ± 597	1,924 ± 652	<.001	31 [8-77]	52 [14-121]	.003
Mid posterior	2,011 ± 626	1,843 ± 642	.005	45 [17-107]	48 [14-98]	.622
Mid lateral	1,928 ± 620	1,773 ± 632	.009	37 [10-89]	28 [8-82]	.203
Mid anterior	2,055 ± 674	1,746 ± 723	<.001	28 [6-80]	47 [14-101]	.007
Mid anteroseptal	2,379 ± 754	1,945 ± 930	<.001	44 [12-120]	83 [24-194]	<.001
Mid septal	2,211 ± 585	1,860 ± 698	<.001	52 [16-115]	79 [28-149]	.002
Apical inferior	3,002 ± 1,035	2,352 ± 1,188	<.001	46 [13-131]	112 [23-293]	<.001
Apical posterior	2,562 ± 872	2,107 ± 1,027	<.001	53 [17-128]	103 [20-271]	<.001
Apical lateral	2,584 ± 1,068	2,028 ± 1,216	<.001	51 [12-133]	91 [27-221]	<.001
Apical anterior	2,812 ± 1,146	2,171 ± 1,294	<.001	57 [11-147]	108 [28-278]	<.001
Apical anteroseptal	2,867 ± 1,065	2,209 ± 1,328	<.001	62 [23-158]	116 [22-313]	.001
Apical septal	2,963 ± 1,086	2,288 ± 1,350	<.001	60 [15-186]	134 [23-320]	<.001

Data are presented as mean ± SD or median [interquartile range].

Table 5 Number (%) of patients with impaired myocardial work indices and postinfarction LV remodeling at 3 months based on normal reference range

Percentage of patients	No LV remodeling (*n* = 450)	LV remodeling (*n* = 150)	*P* value
GWI			
Preserved	426 (94.7)	118 (78.7)	<.001
Reduced	24 (5.3)	32 (21.3)	<.001
GCW			
Preserved	407 (90.4)	98 (65.3)	<.001
Reduced	43 (9.6)	52 (34.7)	<.001
GWW			
Preserved	428 (95.1)	129 (86.0)	<.001
Increased	22 (4.9)	21 (14.0)	<.001
GWE			
Preserved	410 (91.1)	98 (65.3)	<.001
Reduced	40 (8.9)	52 (34.7)	<.001

remodeling.[24-26] Joyce *et al.*[24] showed that patients with reduced baseline LV GLS (absolute value less than 15.0%) exhibited greater LV dilatation at 3 and 6 months compared with patients with more preserved LV GLS (15.0% or greater).

Furthermore, LV GLS had incremental value over troponin levels and wall motion abnormalities to predict further LV dilation (increase in LVEDV). Still, conventional 2D echocardiographic measurements of LV volumes and systolic function using LVEF and 2D LV GLS do not take into consideration the load dependency and do not truly represent LV contractility.[27]

Noninvasive Myocardial Work Indices in Postinfarction LV Remodeling

Although patients with LV remodeling at 3-month follow-up had significantly more impaired noninvasive measures of myocardial work compared with patients who did not show remodeling, the majority of non-LV remodeling patients had preserved GWI, GCW, and GWE and normal GWW. Conversely, and of interest, a small proportion of patients without LV remodeling had impaired values of noninvasive myocardial work indices. These findings may be explained by the association between myocardial work indices and myocardial energetics. Altered energy metabolism has been reported in LV remodeling with a switch from aerobic (free fatty acid) to anaerobic (glucose) metabolism, with a lower ATP yield, thereby reducing myocardial contraction.[28] Russell *et al.*[4] showed that noninvasive myocardial work indices have a strong correlation with regional myocardial glucose (anaerobic) metabolism as assessed by positron emission tomography using F18 fluorodeoxyglucose. Accordingly, impaired noninvasive myocardial work indices may be observed more frequently in patients with LV remodeling because of persistent anaerobic myocardial metabolism. Among patients who do not show LV remodeling after STEMI but have impaired noninvasive myocardial work indices, there still may be impaired (persistent anaerobic) myocardial metabolism that may lead to LV remodeling at a later stage. Although the majority of patients with STEMI present with LV remodeling in the first 3-6 months of follow-up, there remains a proportion of patients who may reveal LV remodeling after 6 months of follow-up.[29]

Alterations in myocardial mechanics and LV remodeling have been reported in a previous study that demonstrated that strain and wall stress are able to predict LV remodeling.[30] Patients with ischemia present changes in contractility with more dyssynchronous contraction and more wasted work. In the normal heart, the LV contraction is

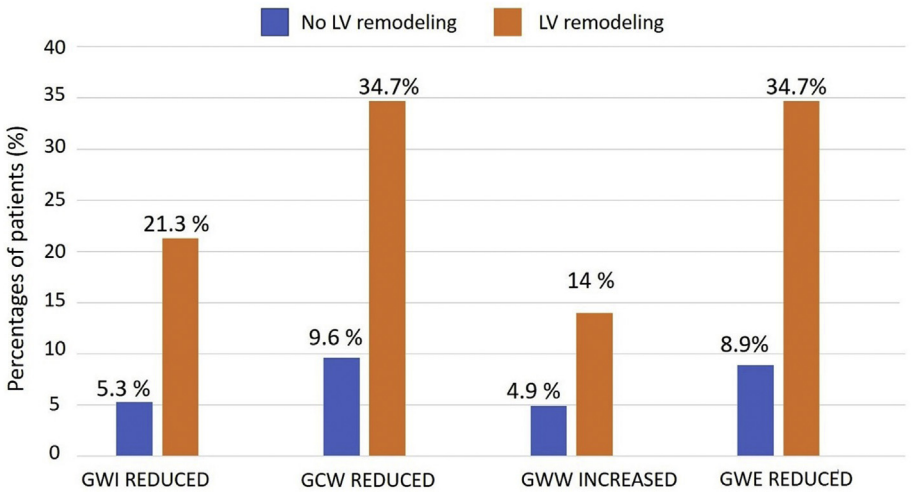

Figure 1 Percentages of LV remodeling in each category of myocardial work based on normal reference ranges[13]: GWI reduced: men <1,270 mm Hg% and women <1,310 mm Hg%; GCW reduced: men <1,650 mm Hg% and women <1,544 mm Hg%; GWW increased: men >238 mm Hg% and women >239 mm Hg%; GWE reduced: men <90% and women <91%.

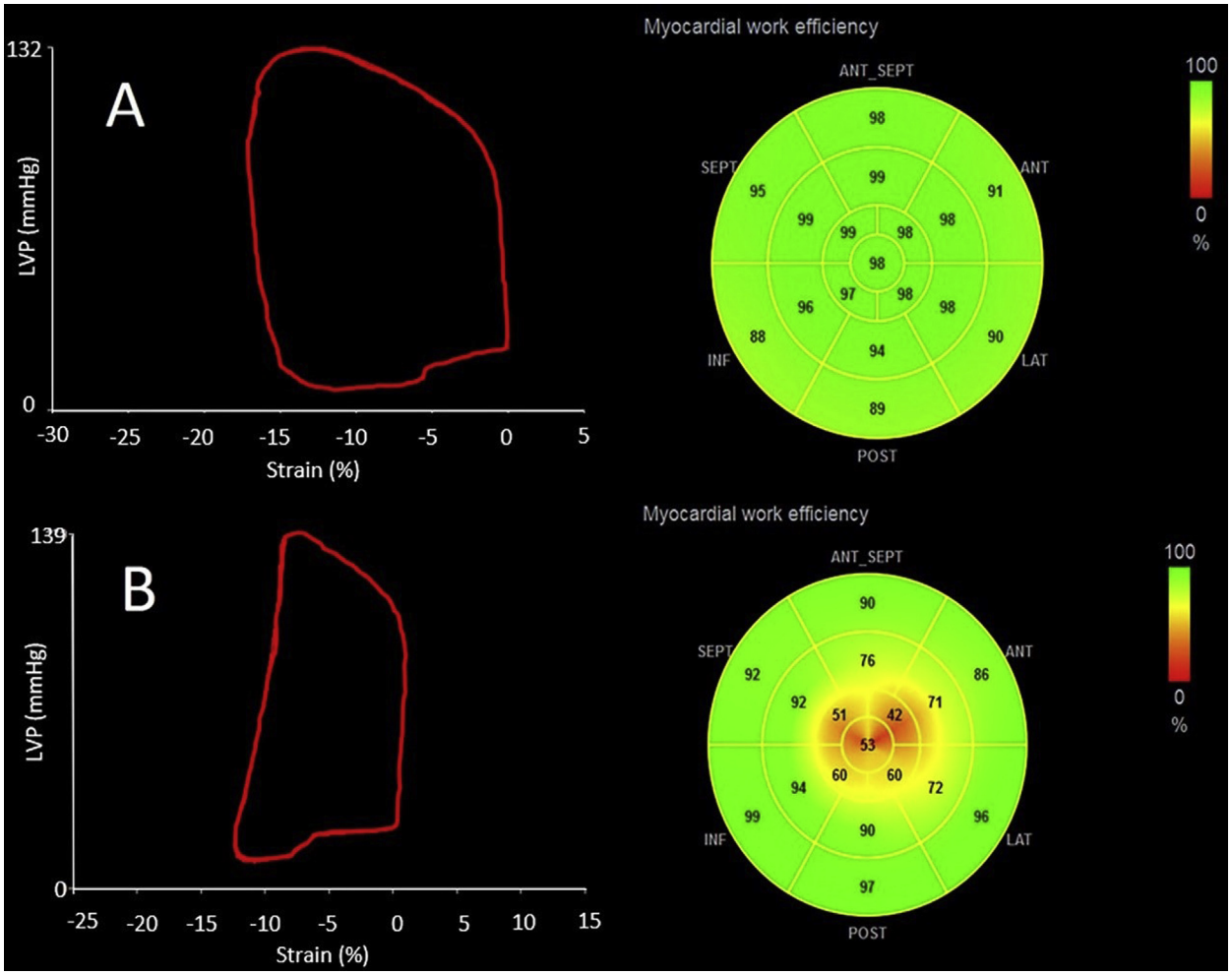

Figure 2 Pressure-strain loops curve and GWE bull's-eye plots showing segmental GWE of patients without LV remodeling **(A)** and with LV remodeling **(B)**. As conventionally accepted, normal GWE are presented in *green* and reduced GWE are presented in *yellow*. The bull's-eye of the no-remodeling patient with preserved LVEF (>50%) shows homogeneous normal GWE. In the remodeling patient with reduced LVEF (<40%), reduced GWE is observed in the LV apical segment consistent with the region of infarction and the culprit coronary artery (mid-left anterior descending coronary artery).

homogeneous and the work performed by the LV segments contributes to systolic function. In contrast, after STEMI, the infarcted segments are stretched by the remote noninfarcted segments. This results in hypertrophy and myocyte elongation in the noninfarcted zone, resulting in increased wall mass, larger LV volumes, and more wasted work. In the present study, patients with LV remodeling at 3 months of follow-up presented with higher values of GWW compared with patients without LV remodeling, which can be explained by these mechanisms.[31] In addition, we observed that values of myocardial constructive and wasted work were more impaired in the segments supplied by the left anterior descending coronary artery among patients with LV remodeling. However, the distribution of the infarct-related coronary artery was not different between patients with and without LV remodeling, suggesting that regional constructive and wasted work affecting the segments supplied by the left anterior descending coronary artery may be important contributors to the remodeling process regardless of the infarct-related coronary artery.

Study Limitations

One of the limitations of the study is its retrospective design. Furthermore, data on systolic and diastolic blood pressure when the echocardiogram was performed during the index admission were not systematically available. Therefore, the study cannot analyze predictors of LV remodeling in this population. The incremental value of myocardial work indices analyzed at baseline over other well-known predictors of remodeling such as troponin levels cannot be assessed. Furthermore, late gadolinium contrast cardiovascular magnetic resonance data to correlate with myocardial work indices at 3 months after STEMI were not available.

CONCLUSION

In the present study, patients with STEMI who developed LV remodeling at 3-month follow-up showed a higher prevalence of impaired myocardial work indices compared with patients who did not have LV remodeling. Of interest, this phenomenon was also encountered in (a minority of) patients without remodeling at 3 months. The clinical implications of impaired myocardial work indices need further study with long-term follow-up of patients.

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