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Crescent Journal of Medical and Biological Sciences
Vol. 7, No. 1, January 2020, 116–123
eISSN 2148-9696

# Prevalence and Predictors of Left Ventricle Regional Wall Motion Abnormality 6 Weeks After Primary Percutaneous Intervention in Patients With First Acute Anterior Myocardial Infarction

**Sakine Hadi[1], Mehrnoush Toufan Tabrizi[1*], Ahmad Separham[1], Mohammadreza Hoseinalizade[1], Marzie Sadeghi[1], Raziye Parizad[1]**

**Abstract**

**Objectives:** Regional wall motion abnormality (RWMA) occurs after acute myocardial infarction (AMI) or primary percutaneous coronary intervention (PPCI). The current study aimed to assess the prevalence of RWMA after PPCI and to define its baseline clinical, angiographic, and echocardiographic predictors in patients with acute anterior MI.

**Materials and Methods:** Totally, 107 patients (85 males and 22 females with a mean age of 58.21±11.64 years) with first anterior MI treated with PPCI were evaluated, and transthoracic echocardiographic examination was performed at the admission time and after at least 6 weeks. The RWMA was assessed and wall motion score index (WMSI) was calculated by dividing the sum of the wall motion score over the number of visualized segments.

**Results:** The results revealed no statistically significant association between the symptom-onset-balloon time and door-balloon time compared with the WMSI value ($P=0.29$, r=0.105 & $P=0.53$, r=-0.062). Regarding post-PPCI thrombolysis in MI (TIMI) flow grades, patients with a grade II and III TIMI flow had a mean WMSI value of 1.90±0.39 and 1.65±0.31, respectively, that was significantly higher in patients with a grade II TIMI flow ($P=0.002$). During the follow-up echocardiography, 84 (79.2%) patients had a positive RWMA and the follow-up positive RWMA was significantly lower (7.4%) in patients with stable angina ($P=0.01$). Statistically noticeable improvements were reported in the level of WMSI and E/E′ ratio during the follow-up period of echocardiography. In addition, there was a significant relationship between pre- and post-PCI left ventricular end-systolic volume (LVESV) and WMSI compared with post-PCI RWMA ($P=0.03$, $P<0.0001$; $P=0.007$, $P<0.0001$). However, no statistically significant differences were observed in demographic data and MI risk factors considering the incidence of RWMA and the mean value of WMSI.

**Conclusions:** In patients with the first acute MI, higher WMS index and LVESV level were strongly related to RWMA incidence after six weeks which could be applied as the predictor factors of RWMA incidence.

**Keywords:** Myocardial infarction, Primary PCI, RWMA, WMSI

## Introduction

In developing countries, the ST-segment elevation myocardial infarction (STEMI) remains one of the most common causes of mortality and morbidity. However, recent improvements in the management of patients with STEMI including reperfusion therapies such as thrombolysis and primary percutaneous coronary intervention (PPCI) have reduced mortality while increasing the survival rate (1-4). PPCI has also emerged as the most effective reperfusion strategy for patients with STEMI (5). Nonetheless, myocardial remodeling increases the left ventricular volume and wall motion score index (WMSI) and is suggested to be associated with left ventricular remodeling after STEMI (6-8). The extent of infarct size is referred to as one of the factors affecting prognosis in patients suffering from acute myocardial infarction (AMI), therefore, early reperfusion can limit damages to left ventricle (LV) and prevent myocardial dysfunction (2,9,10). In addition, timely reperfusion in patients with STEMI can reduce myocardial cell death, preserve left ventricular function, and thereby eliminating the incidence of heart failure. Therefore, on time PPCI can limit cardiac death in the short and long term. Considering the importance of the reperfusion time, PPCI should be designed to reach "door-balloon time", as well as "total ischemic time" less than 90 and 120 minutes, respectively (11,12). In contrast with the advantages of PPCI in STEMI, reverse LV remodeling occurred in a considerable proportion (37.7%) of STEMI patients who underwent PPCI, manifesting poor prognosis (5,13).

**Received** 2 January 2018, **Accepted** 17 April 2018, **Available online** 13 May 2018

[1]Department of Cardiology, Shahid Madani Hospital, Tabriz University of Medical Sciences, Tabriz, Iran.
*__Corresponding Author:__ Mehrnoush Toufan Tabrizi, Tel: +989143111284, Email: mtoufan@gmail.com

LV ejection fraction (LVEF) improves in some STEMI patients after effective reperfusion as a consequence of the gradual relief of myocardial stunning while in other patients, irreversible myocardial necrosis may result in chronic LV dysfunction (14). Previous research reported a correlation between regional wall motion abnormality (RWMA) in the follow-up study and pre-PCI ischemic time (6). Further, it was mentioned that reverse LV remodeling happened in about half of the STEMI patients even in successful PPCI area (14,15). Initially, the ischemic myocardium becomes akinetic, with preserved wall thickness. Over a period of 4-6 weeks, an unperfused infracted myocardium results in a definite area of akinesis, along with wall thinning. Similarly, global LV systolic function is moderately depressed after unperfused anterior MI (16). A normal LV filling pattern compatible with the normal cardiac diastolic function was reported in only one-third of patients in the acute phase of MI and an abnormal diastolic function often led to permanent heart failure (17). AMI impairs diastolic and systolic functions. Acutely diastolic relaxation also becomes abnormal with normalization over the subsequent 1 to 2 weeks by successful recanalization (16).

LVEF value is technique-dependent and may not demonstrate the extension of myocardial damage due to regional compensatory effects (17-19). On the other hand, it is estimated that an increase in the WMSI during the first 12-24 hours after STEMI is a predictor of complications during hospital admissions such as malignant arrhythmias, pump failure, and mortality (20-22). In addition, the WMSI can quantitatively measure the LV systolic function following an AMI (23). The WMSI is defined as the measurement of left ventricular wall motility in specific segments with a higher index indicating more severity for abnormal wall movements (19,24). Jurado-Roman revealed that the WMSI has a priority over the LVEF based on its ability for distinguishing the compensatory effect of hyperkinetic segments (25). However, controversies are available over the correlation between an early ventricular wall dysfunction and the long-term prognosis of MI (7,26-28). Moreover, it is impossible to accurately evaluate the true function of hypercontractile segments during the hyper-acute phase. Considering increasing STEMI and successful primary coronary angioplasty, mechanical adverse events demonstrate a significant decline, though RWMA and reverse LV remodeling after MI treated with PPCI remained significant. Therefore, the present study sought to investigate the incidence, as well as the major determinants and predictors of long-term RWMA after PPCI in patients with anterior AMI.

## Materials and Methods

In the present prospective cross-sectional study, 110 patients with the diagnosis of first acute anterior myocardial infarction (MI) were recruited referring to Madani Heart Center and undergoing a PPCI. The exclusion criteria were a need for surgical revascularization, a history of valvular disorders, old MI, and acute inferior MI, as well as the detection of multi-vessel diseases during coronary artery angiogram, complicated anterior MI, and patients with anterior MI receiving thrombolytic therapy.

Therefore, three patients were excluded due to death, cerebrovascular accident, and coronary artery bypass graft, and finally, 107 patients were included in the study. Informed written consent was obtained from the patients before the study and their demographic data, ischemic time, door-balloon duration, and angiogram findings were recorded, followed by careful consideration of the incidence of complications after PPCI.

Following transthoracic echocardiography immediately after admission to the emergency room, the patients were transferred to the catheter laboratory and coronary angiography and PPCI were performed according to the standard artery approach. Furthermore, intracoronary stents were routinely implemented under the discretion of an operator.

The patients were followed at least six weeks after the PPCI and all patients underwent second transthoracic echocardiography with a real-time instrument (General Electric, Vivid 7GE using the linear array probe, USA). Moreover, all examinations were performed by an echocardiography clinical fellow. The LV volume and LVEF were calculated using the apical orthogonal (2ch & 4ch) via the Biplane Simpson method (Figure 1). From the apical 4-chamber view, pulse wave Doppler mitral inflow velocities were derived to assess the diastolic function, followed by measuring the peak early diastolic flow velocity (E), peak late diastolic flow velocity (A), and E/A ratio. Moreover, myocardial peak systolic wave (S’), early diastolic wave (E’), end-diastolic wave (A’) velocities, and E/E’ ratio were recorded by placing the pulsed wave tissue Doppler imaging sample volume at the level of the mitral annulus at the lateral and septal region (Figure 2). Left ventricular systolic and diastolic functions were evaluated according to the integrated data as well. The segment

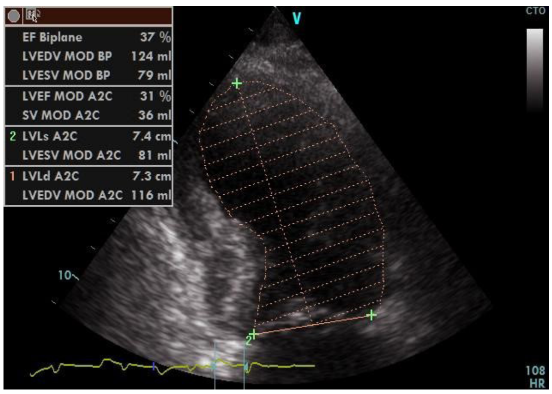


**Figure 1.** Apical Orthogonal Biplane Views Applied for Simpson Method**.**
*Note*. Left ventricle volume and left ventricular ejection fraction were calculated.

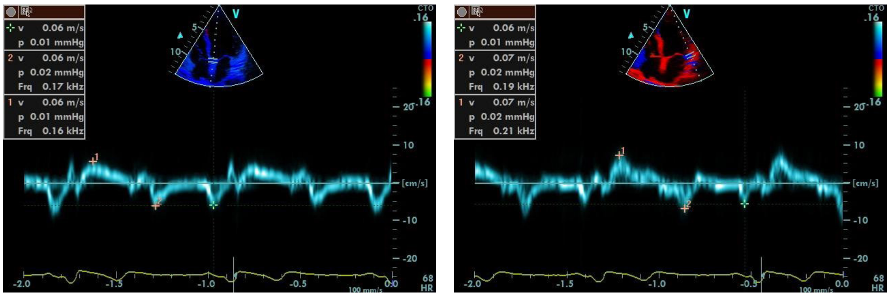

**Figure 2.** Pulsed Wave Tissue Doppler Imaging at the Level of the Septal (A) and Lateral (B) Mitral Annuli
*Note.* Myocardial systolic (Sa, 1), early diastolic (Ea, 2), and slate diastolic velocities (Aa, +) were measured and averaged.

contractility scoring based on segment excursion and wall thickening were defined as normal, hypokinetic, akinetic, dyskinetic, and aneurysm.

According to the method introduced by the American Society of Echocardiography, the WMSI was calculated by dividing the sum of the wall motion score over the number of visualized segments.

All data were analyzed using the SPSS, version 22 (SPSS Inc., Chicago, IL) and mean ± standard deviation was utilized to express the quantitative values. Additionally, the chi-square and McNemar tests were employed to assess the differences between qualitative variables. Similarly, student *t* test or Mann-Whitney U test and ANOVA test were applied to compare the means between the groups. Finally, Pearson correlation coefficient was used to describe the correlation. A *P* value less than 0.05 was considered statistically significant.

## Results

In the present study, 107 patients with STEMI were recruited and underwent a PPCI. In terms of the demographic characteristics of the patients (Table 1), they were mostly males (79.4%) and ninety-one patients (85%) had a positive RWMA. However, no statistically significant differences were observed regarding the demographic data and MI risk factors considering the incidence of RWMA and the mean value of WMSI, the details of which are presented in Tables 1 and 2. In addition, the mean time of the symptom-onset to balloon and door to balloon were 307.27±275.26 and 68.96±81.97 minutes, respectively. The Spearman's test revealed no statistically significant association between symptom-onset-to-balloon and door-balloon time compared with WMSI value ($P$=0.29, r=0.0105; $P$=0.53, r=-0.062). Prior to PPCI, thrombolysis in MI (TIMI) flow grade 0-I, II, and III were detected in 71 (66.4%), 31 (29%), and 4 (3.8%) patients, respectively. Nevertheless, they changed to II and III in 21 (19.6%) and 84 (78.5%) patients after PPCI, respectively. Comparing the WMSI value based on the pre-PPCI, TIMI flow grades showed no statistically significant difference between the groups ($P$=0.12). However, in terms of the post-PPCI TIMI flow grades, the patients with grade II and III

**Table 1.** Comparing Pre-PPCI RWMA and WMSI Considering Age, Gender, and Risk Factors

| Variable | | RWMA | | *P* Value | WMSI | *P* Value |
|---|---|---|---|---|---|---|
| | | Negative | Positive | | Mean ± SD | |
| Gender | Male | 12 (11.2%) | 73 (68.2%) | 0.74* | 1.66±0.33 | 0.06*** |
| | Female | 4 (3.7%) | 18 (16.9%) | | 1.82±0.36 | |
| Age | | 57.56±13.07 | 58.21±11.64 | 0.83*** | - | - |
| Diabetes mellitus | Present | 2 (1.9%) | 16 (14.9%) | 1* | 1.73±0.39 | 0.57*** |
| | Non-present | 14 (13%) | 75 (70%) | | 1.68±0.33 | |
| Hypertension | Present | 8 (7.4%) | 39 (36.4%) | 0.59** | 1.69±0.37 | 0.97*** |
| | Non-present | 8 (7.4%) | 52 (48.8%) | | 1.69±0.31 | |
| Hyperlipidemia | Present | 5 (4.6%) | 16 (14.9%) | 0.3* | 1.73±0.39 | 0.72**** |
| | Non-present | 11 (10.5%) | 75 (70%) | | 1.68±0.32 | |
| Cigarette smoking | Present | 8 (7.4%) | 40 (37.3%) | 0.65** | 1.66±0.32 | 0.36*** |
| | Non-present | 8 (7.4%) | 51 (47.9%) | | 1.72±0.35 | |
| Stable angina | Present | 1 (1%) | 14 (13%) | 0.46* | 1.71±0.28 | 0.8*** |
| | Non-present | 15 (14%) | 77 (72%) | | 1.69±0.35 | |
| Family history | Present | 2 (1.9%) | 20 (18.6%) | 0.52* | 1.72±0.32 | 0.7*** |
| | Non-present | 14 (13%) | 71 (66.3%) | | 1.69±0.34 | |

Note. PPCI: Primary percutaneous coronary intervention; RWMA: Regional wall motion abnormality; WMSI: Wall motion score index; SD: Standard deviation; *Fisher's exact test; **Chi-square test; ***Student's t-test; ****Mann-Whitney U test.

**Table 2.** Comparing Post Follow-up RWMA and WMSI Considering Age, Gender, and Risk Factors

| | | RWMA | | *P* Value | WMSI | *P* Value |
|---|---|---|---|---|---|---|
| | | Negative | Positive | | Mean ± SD | |
| Gender | Male | 18 (16.8%) | 68 (63.7%) | 1.00* | 1.56±0.47 | 0.85**** |
| | Female | 4 (3.7%) | 17 (15.8%) | | 1.59±0.49 | |
| Age | | 56.81±13.35 | 58.20±11.25 | 0.62*** | - | - |
| Diabetes mellitus | Present | 1 (1%) | 17 (15.8%) | 0.11* | 1.56±0.48 | 0.05**** |
| | Non-present | 21 (19.5%) | 68 (63.7%) | | 1.72±0.37 | |
| Hypertension | Present | 11 (10.5%) | 35 (32.7%) | 0.48** | 1.59±0.45 | 0.45**** |
| | Non-present | 11 (10.5%) | 50 (46.3%) | | 1.54±0.49 | |
| Hyperlipidemia | Present | 4 (3.7%) | 17 (15.8%) | 1.00* | 1.56±0.48 | 0.59**** |
| | Non-present | 18 (16.8%) | 68 (63.7%) | | 1.60±0.44 | |
| Cigarette smoking | Present | 11 (10.5%) | 37 (34.5%) | 0.62** | 1.58±0.50 | 0.88**** |
| | Non-present | 11 (10.5%) | 48 (44.5%) | | 1.54±0.43 | |
| Stable angina | Present | 7 (6.5%) | 8 (7.4%) | 0.01* | 1.52±0.53 | 0.87**** |
| | Non-present | 15 (14%) | 77 (72.1%) | | 1.58±0.47 | |
| Family history | Present | 7 (6.5%) | 15 (14%) | 0.23* | 1.51±0.47 | 0.32**** |
| | Non-present | 15 (14%) | 70 (65.5%) | | 1.58±0.47 | |

Note. PPCI: Primary percutaneous coronary intervention; RWMA: Regional wall motion abnormality; WMSI: Wall motion score index; SD: Standard deviation; *Fisher's exact test; **Chi-square test; ***Student's t-test; ****Mann-Whitney U test.

TIMI flow had the mean WMSI values of 1.90±0.39 and 1.65±0.31, respectively, which was significantly higher in patients with the grade II TIMI flow (*P*=0.002). Further, the culprit segment of the left anterior descending (LAD) artery differentiated during angiography as the proximal and non-proximal segment of LAD in 56 (52.3%) and 51 (47.7%) patients, respectively. However, no statistically significant difference was observed between the mean value of WMSI and the culprit segment for LAD (*P*=0.95). 5 (4.8%) patients underwent plain old balloon angioplasty. Meanwhile the coronary stent was implemented in 98 (93.3%) patients and 2 (1.9%) patients received both techniques as well. Furthermore, no heart failure, cardiogenic shock, death, cardiopulmonary arrest, or ventricular tachycardia, and flutter were reported during hospital admission.

During follow-up echocardiography, 84 (79.2%) patients had a positive RWMA (Table 2) and thus had significantly a lower incidence of chronic stable angina (*P*=0.008). Using a cumulative odds of ordinal logistic regression with a proportional odds of the effect of gender, diabetes mellitus, hypertension, hyperlipidemia, cigarette smoking, and familial history on the incidence of RWMA at least 6 weeks after the first AMI, no statistically significant correlation was found between these variables and the incidence of RWMA (Table 2).

Echocardiographic variables before PPCI and after the follow-up period are listed in Table 3, including left ventricular end-systolic volume (LVESV), left ventricular end-diastolic volume, left atrial volume index (LAVI), WMSI, mitral valve early diastolic velocity (MVEV), mitral valve late diastolic velocity (MVAV), mitral valve E/A ratio, and E/E' ratio. Statistically significant improvements were found in the level of WMSI and E/E' ratio during the follow-up period of echocardiography. The patients were compared in terms of the WMSI rate considering the LVESV alteration including a decrease or an increase in the level of LVESV. The patients, who had an increase in the LVESV during the follow-up period, had a mean WMSI rate of 1.77±0.44, which was significantly higher (*P*<0.0001) compared to the WMSI rate in patients with a decreased LVESV (1.26±0.31). Moreover, there was a statistically significant difference in the mean WMSI rate between the patients with increased and decreased LV end-diastolic volume (LVEDV) after 6 weeks of follow up (1.65±0.46 and 1.28±0.37, respectively, *P*<0.0001). Similarly, the comparison of the mean WMSI between the patients with increased and decreased LAVI level showed

**Table 3.** Comparing Pre-PPCI and Post Follow-up Echocardiographic Parameters

| | Echocardiography | | *P* Value |
|---|---|---|---|
| | Before PPCI | After 6 Weeks Follow up | |
| LVESV | 65.75±23.10 | 70.13±29.44 | 0.07* |
| LVEDV | 111.44±24.55 | 123.89±32.25 | <0.0001* |
| LAVI | 31.56±7.92 | 35.98±11.73 | <0.0001** |
| WMSI | 1.69±0.34 | 1.57±0.47 | 0.003** |
| MVEV | 60.43±17.99 | 55.85±16.33 | 0.030.07** |
| MVAV | 60.23±14.82 | 61.75±16.85 | 0.39* |
| MV E/A | 1.06±0.45 | 0.99±0.45 | 0.270.39** |
| E/E' | 11.33±4.06 | 8.92±3.33 | <0.0001** |

*Note*. PPCI: Primary percutaneous coronary intervention; FU:xxx; LVESV: Left ventricular end-systolic volume; LVEDV: LV end-diastolic volume; LAVI: Left atrial volume index; WMSI: Wall motion score index; MVEV: mitral valve early diastolic velocity, MVAV: Mitral Valve late diastolic velocity.
*Paired samples t test; **Wilcoxon signed-rand test.

no statistically significant difference between the patients (1.67±0.30 and 1.73±0.39, respectively, *P*=0.016). Pre-PCI and post-PCI echocardiographic parameters considering final RWMA were separately assessed and the results represented a statistically significant relationship between pre- and post-PCI LVESV and WMSI compared with post-PCI RWMA (Table 4).

## Discussion

The remodeling of the LV in patients with MI is considered as a strong predisposing factor of heart failure and mortality (29,30). The prevalence of post-MI left ventricular systolic dysfunction was reported to be between 27% and 60% (26, 31). However, the recovery of the damaged ventricular wall in the affected segments takes several weeks and depends on several factors such as the collateral coronary flow and pain onset to reperfusion time (21,32). The benefits of PPCI over thrombolysis was described in improving survival (33). In an animal study, the evaluation of RWMA after a coronary artery ligation revealed that regional wall damage developed during the first 10 minutes of ischemia and no significant alteration occurred in the next hours indicating the importance of the early PPCI (34). However, some studies suggested that pre-MI systolic function and the presence of WMA were related to post MI ejection fraction value (6). Additionally, transthoracic echocardiography provides an applicable modality for evaluating left ventricular systolic function following an infarction. However, common echocardiographic parameters are incapable of predicting the long-term outcomes in patients with MI, which prevents the assessment of long-term prognosis. Nonetheless, the WMSI represents left ventricular systolic function after an AMI, which provides a quantitative alternation for ejection fraction.

**Table 4.** The comparison of Post-PCI RWMA With Pre-PCI and Post-PCI Echocardiographic Parameters

| | | RWMA (Post-PCI) | | *P* Value |
|---|---|---|---|---|
| | | Positive | Negative | |
| Pre-PCI | LVESV | 68.19±23.76 | 56.54±18.08 | 0.03* |
| | LVEDV | 112.50±25.33 | 107.45±21.47 | 0.39* |
| | LAVI | 31.49±8.05 | 31.86±7.57 | 0.77** |
| | WMSI | 1.74±0.33 | 1.52±0.31 | 0.007* |
| | MVEV | 60.47±18.55 | 59.45±16.13 | 0.81* |
| | MVAV | 59.32±15.38 | 62.95±11.76 | 0.28** |
| | MVE/A | 1.09±0.48 | 0.96±0.34 | 0.24* |
| | E/E' | 11.22±4.26 | 11.64±3.22 | 0.33** |
| Post-PCI | LVESV | 76.32±29.26 | 51.9±22.20 | <0.0001* |
| | LVEDV | 127.86±32.72 | 112.41±32.05 | 0.50* |
| | LAVI | 36.21±11.58 | 35.9±12.56 | 0.50** |
| | WMSI | 1.67±0.46 | 1.20±0.32 | <0.0001** |
| | MVEV | 55.21±16.98 | 57.18±14.39 | 0.73** |
| | MVAV | 61.30±16.68 | 63.45±17.44 | 0.57** |
| | MVE/A | 0.99±0.47 | 1.00±0.42 | 0.60** |
| | E/E' | 8.84±3.47 | 9.09±2.83 | 0.50** |

*Note*. PPCI: Primary percutaneous coronary intervention; LVESV: Left ventricular end-systolic volume; LVEDV: LV end-diastolic volume; LAVI: Left atrial volume index; WMSI: Wall motion score index; MVEV: mitral valve early diastolic velocity, MVAV: Mitral Valve late diastolic velocity.
*Independent samples *t* test; **Man-Whitney U test.

In the present cross-sectional study, a significant improvement was detected in the echocardiographic parameters during a 6-week follow-up period. Similarly, Faustino et al reported significant improvements in echocardiographic parameters such as LVEF and WMSI and the comparison of admission echocardiography time to the follow up was similar to this study (35). The authors believe that improved echocardiographic finding during the follow-up period may demonstrate well-timed and successful revascularization that can restrict further myocardial remodeling. In addition, considering the early PPCI effect on the development of reversed remodeling, an early PPCI not only prevents myocardial remodeling chain but also stimulates the reversed remodeling process that enhances the LV dysfunction recovery in patients with the first AMI. Contrarily, Rácz et al demonstrated that the symptoms-onset-balloon time was not correlated with the severity of MI culprit vessel thrombosis and RWMA, but echocardiographic variables had statistically significant correlations with the symptom-onset-balloon time 3 months after the PCI. However, the early PCI led to an improvement in echocardiography parameters (36). One could expect a low prevalence of RWMA following mechanical reperfusion. Our data showed that despite PPCI, RWMA occurred in 79% of the patients who were successfully treated with PPCI, which was significantly higher compared with previous studies. In the current study, the mean total ischemic time was 307 minutes, which represented that the majority of patients were the latecomers. Similarly, no significant relationship was found between the symptom-onset-balloon time and the RWMA incidence. In a study by Neuman et al, only 31% of the patients had segmental wall motion abnormalities following the first AMI (20). Such a discrepancy in the findings may be related to patients' referral time and total ischemic time, which was greater in this study compared to previous studies. Likewise, Choi et al revealed that wall motion abnormalities, left atrium (LA) and LV dilatation were the predictors of poor prognosis and increased mortality whereas constant LA and LV size resulted in a reduction in remodeling and improved the prognosis (15). Despite the findings of previous studies, no significant correlation was reported between the total ischemic time and the WMSI rate in this study. Therefore, the prolonged duration of the symptom onset to PPCI in all patients might cause a bias in the evaluation of treatment time impact on the WMSI rate. Another study regarding evaluating LV remodeling after anterior MI, Savoye et al found a decrease in WMSI while an increase in LVEF that resulted in the recovery of left ventricular systolic function. They further reported a significant increase in LVEDV and found that recent improvements in AMI management fail to abolish LV remodeling, which remains a relatively frequent event after an initial anterior wall AMI (37). In addition, Świątkiewicz et al. revealed that despite successful primary PCI, patients with a first STEMI suffered from moderate left ventricular systolic dysfunction at early and long-term follow up. In a 12-month follow-up, the patients demonstrated an improvement in global and regional left ventricular systolic function (38). Our findings revealed an increased LVESV and LVEDV levels during the follow-up period in patients who had a higher WMSI rate after PPCI. In addition, a significant relationship was reported between the echocardiographic parameters of the admission time, including the LVESV and WMSI and the incidence of RWMA, at the end of the follow-up period.

The extent of microvasculature dysfunction and thrombosis was proved to be an independent predisposing factor regarding developing LV dysfunction and wall motion abnormalities (7). Therefore, severe microvasculature occlusions result in a vast extension of the MI region, an increase in RWMA, and finally, a reduction in LVEF (39, 40). Yoon et al studied the prioritization of the WMSI in comparison to the ejection fraction of LV and concluded that a reduction in LVEF and an increase in the WMSI were considered as the independent predictors of ventricle remodeling after an AMI (40). Xiang et al.also confirmed that WMS and global registry of acute coronary event (GRACE) score were the independent predictors of major adverse cardiac events in patients with AMI in 12-month follow-up, and the combined application of WMS and GRACE score could significantly improve the predictive value (41). On the other hand, another study described the prioritization of combined LVEF and the WMS index to LVEF alone in the prediction of cardiovascular events and the long-term prognosis of patients with AMI (19). However, the findings of the present study indicated a significant relationship between the WMSI rate prior to PPCI and the prevalence of RWMA after the follow-up. Overall, the symptom-balloon time had no significant effect on admission time WMSI, the RWMA rate, long-term LV ejection fraction, and systolic function. In other words, the WMSI rate was higher in patients who had lower TIMI scores after PPCI. Therefore, partial reperfusion and failure to complete reperfusion (as a consequence of microvasculature occlusions) led to LV systolic dysfunction and subsequent increase in the WMSI rate with a higher incidence of LV remodeling. On the other hand, no statistically significant correlation was found between proximal LAD thrombosis and the WMSI rate.

LV diastolic dysfunction was reported in most of the patients with AMI successfully treated by PCI. It was explained that STEMI patients treated by early reperfusion did not show better diastolic function in comparison to late reperfused patients (17). Tissue Doppler imaging was presented as a more objective assessment of myocardial contractility, as well as myocardial diastolic function (42). According to our results, a significant improvement was found in the E/E’ ratio during the follow-up echocardiographic evaluation, which was compatible with the recovery of diastolic function. Contrarily, Chen et al (17) observed no statistical difference between early and late reperfused STEMI patients respecting LAVI. In addition, our results showed no statistically significant correlation between LAVI and WMSI in spite of significantly increased LAVI in post-PCI echocardiography.

Based on the results, patients with a higher thrombosis grade and severe occlusion were more susceptible to the increase in the WMSI, which contradicts the results of previous studies (26,37).

It seemed that shorter symptom-balloon time may not be a significant predictor of LVEF improvement. It is supposed that symptom-balloon time correlates with other factors influencing the improvement of LVEF, including the restoration of TIMI-3 flow and lower peak creatine kinase myocardial band (14). Therefore, it can be estimated that complications during revascularization and latent reperfusion due to severe thrombosis can lead to a reduction in the distal coronary flow, myocardial damage, a higher WMSI, and severe LV systolic dysfunction.

The first limitation of the present study was the assessment of echocardiographic findings by a single echocardiologist rather than employing a second one. Further, laboratory tests and their value effects on long-term cardiac outcomes, as well as the mortality of the patients with STEMI with the first AMI were not considered in this study. The overall delayed referral and intervention time restricted the assessment of the influence of timely primary coronary angioplasty on echocardiographic parameters in our AMI patients. Another limitation was the relatively short observation period while the RWMA process may occur over successive weeks to months.

## Conclusions

An early PPCI in patients with the first AMI was associated with a lower WMSI contributing to a significant improvement of left ventricular function and ejection fraction. Therefore, early reperfusion and revascularization not only prevents the development of RWMA but also can improve the long-term prognosis of the patients. The findings of the present study showed a considerable proportion (79%) of ST-segment elevation MI patients who were treated by PPCI presented with RWMA. Moreover, higher pre- and post-PCI WMSI and LVESV level were strongly related to the RWMA incidence after six weeks in patients with first AMI. Therefore, it is supposed that higher pre- and post-PCI WMSI and LVESV level could be presented as the predicting factors of RWMA incidence at the follow-up examination. It was further suggested that delayed coronary angioplasty and partial reperfusion play a role in the deterioration of RWMA and WMSI. However, the expected beneficial effects of PPCI on the echocardiographic parameters and clinical outcomes remained unknown. Accordingly, further prospective studies with on time PPCI and longer follow-up are needed in this respect.

## Conflict of Interests

The authors declare that they have no conflicts of interest.

## Ethical Issues

The study proposal was approved by the Ethics Committee affiliated with Tabriz University of Medical Sciences, that conformed with the Helsinki Declaration (1995) which was revised in 2001. The ethical approval was obtained (the ethical code of IR.TBZMED.REC.1395.1314) from the Research Ethics Committee in the Faculty of Medicine at Tabriz University of Medical Sciences.

## Financial Support

This study was funded by Tabriz University of Medical Science.

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