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The impact of a viable myocardium on markers of left ventricular remodeling after an acute myocardial infarction.

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Abstract. Patients with AMI were investigated to determine the impact of a viable myocardial on LV remodeling characteristics. Study participants included 93 patients with an ST-segment elevation in acute coronary syndrome. Stress echocardiograms using dobutamine were performed on each subject.All patients had echocardiography and coronary angiography one and six months following coronary angioplasty. Patients with and without viable areas were split into two groups based on the outcomes of dobutamine stress echocardiography: group I had viable areas, while group II did not. Wall-motion score index, end-systolic volume index (ESVI), end-diastolic volume index (EDVI), and left ventricular ejection fraction (LVEF) were the metrics used to compare the two groups. Therefore, individuals with increasing left ventricular dilatation following reperfusion are distinguished from those with normal left ventricular geometry.
Keywords: viable myocardium, remodeling, acute myocardial infarction, echocardiography.

Aim of the study: to investigate the influence of viable myocardium on left ventricular (LV) remodeling indices after acute myocardial infarction (AMI).

Methods: Two-dimensional echocardiography was done on ninety-three patients diagnosed with acute coronary syndrome with ST-segment elevation (ACS+ST) and successful primary coronary angioplasty within 24 hours of the onset of AMI, and stress echocardiography with low-dose dobutamine was done seven to eight days later. All patients underwent coronary angiography and two-dimensional echocardiography one and six months following coronary angioplasty. Patients were categorized into two groups based on the outcomes of dobutamine stress echocardiography: those with viable periinfarct zones (n = 48; group I) and those without (n = 45; group II).

Results: Between the two groups, there was no difference in the minimum lesion diameter or the lumen of the infarct-responsive artery at months 1 and 6. In comparison to group I patients, group II patients had substantially higher end-diastolic (76±18 vs. 53±14 ml/m2; $p<0.005$) and end-systolic (42±17 vs. 22±11 ml/m2; $p<0.005$) indices at month six following AMI. The best independent predictor of late left ventricular dilatation was the degree of infarct zone viability, which showed a significant inverse correlation with changes in % end-diastolic volume at month 6 ($r = -0.66$; $p < 0.00001$).

Conclusion: The degree of left ventricular dilatation found in AMI after reperfusion is inversely correlated with the amount of myocardial viability that remains in the infarction zone. Therefore, patients who experience progressive left ventricular dilatation following cardiac reperfusion are distinguished from those who retain normal left ventricular geometry by the lack of residual viability in the infarct zone.

List Of Abbreviations
WMSI- wall-motion score index
ESVI - end-systolic volume index
EDVI - end-diastolic volume index
LV - left ventricle
AMI - acute myocardial infarction

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ACS+ST - acute coronary syndrome with ST-segment elevation
LVEF - left ventricular ejection fraction
EchoCG- echocardiography
The definition of left ventricular (LV) remodeling, as stated in the evaluation of the International Forum on Cardiac Remodeling [7], is a change in the LV's size, geometry, and function brought on by an overload or injury. The death of a portion of the functioning left ventricle myocardium that occurs concurrently with an acute myocardial infarction (AMI) is the trigger mechanism for remodeling. Research has demonstrated that one of the key determinants of the type and extent of future remodeling is the degree of cardiac damage brought on by AMI [8]. After an AMI, remodeling is triggered by the necrosis of cardiomyocytes, which raises LV wall stress and lowers ejection percentage. The activation of intracellular biochemical signaling systems in response to a drop in EF results in compensatory cardiac alterations such as dilatation, hypertrophy, and scar formation [5, 15]. Traditionally, renovation following an AMI is separated into early and late remodeling. The necrotic zone's thinning and stretching are the primary features of early remodeling. There is no consistent rise in cardiac enzymes during this phase. Atrial natriuretic peptide production is elevated, the renin-angiotensin-aldosterone system is activated, and the sympathoadrenal system is activated concurrently. When these mechanisms are activated, the intact myocardium contracts more forcefully, the heart rate rises, and hemodynamic compensation is momentarily achieved. Scar development mainly completes the stretching of the infarcted region, usually taking six weeks.

The most widely utilized assessment of such indicators as end-diastolic volume/index, end-systolic volume/index, and EF, the increase in end-diastolic volume/index, is to "measure" the severity of remodeling in echocardiography (Echocardiography). Because LV dilatation increases LV wall stress and deteriorates its pumping capacity, it becomes hemodynamically detrimental and alters the natural shape of the LV cavity, which has a negative impact on prognosis. A multitude of experimental and clinical investigations have demonstrated the adverse effects of an excessive rise in left ventricular diastolic volume/index during the postinfarction period. A wealth of information has been gathered on the correlation between the rise in the index EDVLV during the postinfarction phase and the likelihood of cardiac mortality, myocardial infarction recurrence, congestive heart failure, and embolic stroke [15, 18]. Regarding this, researchers that have examined postinfarction left ventricular remodeling concur that the capacity to forecast left ventricular dilatation following myocardial infarction (MI) is clinically significant. Many research have looked into possible LV dilatation predictions. Age, initial end-diastolic and end-systolic indexes, LV end-diastolic pressure, degree of mitral regurgitation, and AMI size, anterior localization, initial LV EF, stroke index, and TIMI grading of blood flow in the infarct-related artery were among the factors influencing the subsequent dilatation [2].

Purpose of the study: to investigate the influence of viable myocardium on LV remodeling parameters after AMI.

Study Material And Methods

93 patients with ACS and ST-segment elevation were admitted to the Republican Scientific Center of Emergency Medicine's cardiac intensive care unit for the purposes of this study. The patients' average age was 53.9±9.3 years. 8.3±3.7 hours passed between the start of the pain attack and entrance to the clinic. Patients with diabetes mellitus, a history of myocardial infarction, acute cerebral circulation problems, left bundle branch block, atrial fibrillation, LV aneurysm, severe organ failure, and cardiomyopathy were excluded from the study. Within 24 hours of being admitted to the hospital, two-dimensional echocardiography was performed on each patient. Stress-echocardiography with dobutamine, or stress-echoCG, was used on the seventh and eighth days of treatment to assess the viability of the myocardium. Intravenous dobutamine (5 mg/kg body weight per minute) was initiated for three minutes under continuous ECG and two-dimensional echocardiography monitoring. After that, the dosage was raised to 10 mg/kg per minute for an additional three minutes. The development of severe ventricular arrhythmias, angina pectoris, or hypotension were among the criteria for stopping dobutamine therapy. All patients underwent coronary angiography and two-dimensional echocardiography one and six months following primary coronary angioplasty. The American Association of Echocardiography (AAE) guidelines for B- and M-mode echocardiography were followed, with the patient

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positioned on the left side (7,3). A modified Simpson technique was used to measure the left ventricular volume from orthogonal apical long axis projections. Volume was divided by body surface area at each time point to produce volume indices. The formula that yielded the ejection fraction was (end-diastolic volume - end-systolic volume/end-diastolic volume). There were 16 segments that made up the left ventricle (17). Wall motion was graded as follows for each segment: 1 for normal, 2 for hypokinesia, 3 for akinesia, and 4 for dyskienesia. With dobutamine echoCG, the wall-motion score index (WMSI) was computed for every step. The systolic thickening of each segment was also considered in the assessment of regional wall contractility. Plots of the anterior and interior infarct zones were made, and for every stage of dobutamine echocardiography, an index of total and infarct zone wall motion was obtained for each patient (16). The angiography department's X-ray operating room was used for selective coronarography and transluminal balloon angioplasty with coronary artery stenting. Statistical processing of the data obtained in the present study was performed on a personal computer using the EXCEL 7.0 spreadsheet package for Windows.

In the investigation, regression analysis and correlation methods were applied. The arithmetic mean of the variation series ± standard deviation is used to represent all values in the tables. The statistical hypothesis (p=0,05) was an alternative hypothesis with a significance level of at least 95%. Pairing and two-sample Student's t criteria were employed to assess mean equality hypotheses.

Based on the existence or lack of viable cardiac zones, the patients were split into two groups. Contractile reserve was found in 48 patients with dobutamine administration; these patients were classified as having a viable myocardial (group I), while no viable myocardium was found in 45 patients (group II). The features of the patients in the comparative groups are compiled in Table 1. Regarding age, sex, presumed lesion, period from illness beginning to reperfusion, infarct localization, angiographic extent of collaterals, number of arteries damaged, and frequency of coronary risk factors, there were no statistically significant differences between the two groups.

The infarct-responsive artery was either totally or nearly totally blocked in the majority of patients (blood flow 0 or 1, according TIMI). After initial coronary angioplasty, every patient had the best possible angiographic result (residual stenosis <30%, TIMI 3). A stent was inserted into the infarct-responsive artery in 28 patients (17 in group I and 11 in group II; p=0.25). Following coronary angioplasty, the minimum lumen diameter of the diseased vessel rose from 0.10±0.23 to 2.99±0.54 mm in group I and from 0.07±0.29 mm to 2.9±0.56 mm in group II. (Table 2). The angiographic patency rate at one month was 100% in group II and 98% in group I ($p<0.01$). The minimum vessel diameter was 2.76±0.79 in group I and 2.9±0.63 mm in group II ($p<0.01$). After a 6-month period, group I's and group II's angiographic patency rates for the infarct-responsive artery were 98% and 96%, respectively ($p<0.05$). There were no significant differences between the two groups in terms of minimum lumen diameter and restenosis rate (>50%) (Table 2). At first, the wall motion score index (WMSI) was somewhat higher in group I as compared to group II (1.99±0.4 vs. 2.16±0.4; p=0.05), but there was no statistically significant difference between the two groups with respect to LVEF (45±11% vs. 44±10%; $p>0.05$). Group I had a significant improvement in global left ventricular function: LVEF rose from 45±11% at baseline to 56±8% ($p<0.005$) in the first month and to 61±8% after 6 month. Additionally, group II patients' baseline LVEF (44±10% at baseline, 47±15% at one month ($p>0.05$), and 46±13% at six months ($p>0.01$) did not significantly improve. When compared to patients with nonviable infarct zones (group II), patients with infarct zone viability (group I) had notably better global left ventricular function at six months, according to an intergroup comparison. In a similar vein, patients in group I had a significantly higher WMSI than those in group II. WMSI started out at 2.16±0.4 and reduced to 2.02±0.5 ($p>0.05$) after 6 months in group II, while group I experienced a significant decline from 1.99±0.4 to 1.24±0.2 ($p<0.05$). The values of the EDV and ESV indices did not initially differ significantly between the two groups. In group I, the EDV index showed a minor trend to decrease over the course of six months (from 63±18 ml/m2 to 53±14 ml/m2, $p<0.01$). After the 6-month AMI, however, the ESV index in group II patients increased significantly from 64±13 to 74±18 ml/m2 ($p<0.05$) and then to 76±18 ml/m2 ($p<0.01$), which was significantly greater than in group I.Whereas ESVI in group II did not change significantly ($p<0.01$), it did in group I from the beginning to month six. ESVI was considerably higher in group II than in group I during the 6-month period.

The relevance of infarct zone viability in the development of late left ventricular dilatation was evaluated using multiple regression analysis. The parameters that were studied included age, ejection fraction, WMSI, change in WMSI following dobutamine injection, infarct localization, start of reperfusion, collaterals,

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and ACE inhibitor treatment. Factors having p-value<0.01 in univariate analysis were selected for multiple regression analysis. The only significant independent predictor of EDV change over a 6-month period was the change in LV WMSI (assessment of infarct zone viability) caused by dobutamine. The partial correlation coefficient (r=0.48; p<0.01) for the dobutamine-induced alteration in WMSI in the infarct zone was higher at the same moment.

Table 1

Primary clinical and angiographic traits for each group

Age, years	59±11	62±13
Men, %	73 (35)	80 (36)
Diabetes mellitus, %	6 (3)	11 (5)
Arterial hypertension, %	29 (14)	47 (21)
Hyperlipidemia, %	33 (16)	24 (11)
Reperfusion time, min	171±76	167±72
Anterior myocardial infarction, %	56 (27)	53 (24)
Ejection fraction, %	45±11	44±10
LV ESVI (ml/m2)	35±14	36±9
LV EDVI (ml/m2)	63±18	64±13
Infarct-responsive arteries, %		
Right interventricular branch	56 (27)	53 (24)
Right coronary artery	40 (19)	42 (19)
Circumflex artery	4 (2)	5 (2)
Collaterals %	6 (3)	16 (7)
Multivessel lesion	38 (18)	56 (25)

Table 2

Angiographic follow-up of the two study groups

	I group (n=48)	II group (n=45)	Reliability, p
Patency rate, %			
After PCI	100	100	.....

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After one month	98 (1)	100	0.51
After 6 months	98 (1)	96 (2)	0.48
Restenosis (≥50%),%			
After one month	4 (2)	2 (1)	0,53
After 6 months	23 (11)	22 (10)	0,57
Minimum coronary artery lumen diameter, mm			
Initial	0.10±0.23	0.07±0.29	0.58
After PCI	2.99±0.54	2.90±0.56	0.43
One month later	2,76±0,79	2,90±0,63	0,35
After 6 months	2,38±0,92	2,22±0,99	0,42

Percutaneous coronary intervention (PCI). Values in parentheses are the number of patients.

DISCUSSION

This research shows that even after primary coronary angioplasty, when the lumen of the infarct-responsive artery is restored, and when there is no appreciable residual stenosis, individuals with acute myocardial infarction still exhibit left ventricular dilatation. Simultaneously, myocardial vitality in the infarction zone is correlated with the extent of left ventricular dilatation. Accordingly, our findings imply that patients who experience progressive left ventricular dilatation following myocardial infarction are distinguished from those whose left ventricular geometry remains normal by the lack of residual viability of the infarct zone. A significant contributing factor to the onset of chronic heart failure may be left ventricular dilatation (1, 13). Cavity dilatation that is out of proportion to the alteration in LV geometry is a hallmark of left ventricular dilatation, which is caused by persistent modifications to the left ventricle's remodeling (13). The two main parameters that are hypothesized to determine LV remodeling in postinfarction patients are the perfusion status of the infarct-responsive coronary artery and the final infarct size (3,4), among other factors that influence LV dilatation (6). Evaluation of the infarct size alone is insufficient to predict left ventricular dilatation, even though large-focal myocardial infarction invariably results in LV remodeling. In fact, the thickness of viable myocardium inside the infarct zone is inversely correlated with the degree of dilatation (9). According to new experimental research on late reperfusion, islets of viable subepicardial myocytes that are saved by antegrade blood flow might stop left ventricular dilatation (11). The experimental findings of cardiac viability remaining in the infarct zone, as this work demonstrates, establish an important and independent predictor of later alterations in the geometry and function of the left ventricle. According to our data, patients without any remaining myocardial viability in the infarct zone had a considerably higher degree of asynergy when measuring the extent of the infarct. It is evident that variations in left ventricular volume can account for this, at least in part. Nevertheless, after adjusting for infarct size, infarct zone viability was the most powerful independent predictor of left ventricular dilatation. The correlation between the change in left ventricular end-diastolic volume and the index of wall motion in the infarct zone was less than that between the index of end-diastolic volume and infarct zone viability. The existence of maintained blood flow in the infarcted area (3,12) and the absence of residual stenosis (<1.5 mm) in the vessel (17) mitigate the impact of infarct size on later left ventricular remodeling. We only included patients with open lumen of the infarct-related arteries and without considerable residual stenosis to prevent confounding of these two variables on subsequent changes in left ventricular dimensions.

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Furthermore, the rates of restenosis and subsequent lumen opening were similar in both patient groups. The degree of residual viability is directly correlated with a decrease in LV dilatation, indicating that blood flow preservation within the infarct zone is insufficient to prevent remodeling when the infarct zone is no longer viable. These findings further support and broaden the finding of Ito et al. (13) that capillary integrity in the infarct zone is a sensitive indicator of myocardial vitality that shields patients receiving reperfusion from left ventricular remodeling. The idea is supported by the agreement between our results and those of Ito et al., who used different approaches to investigate different aspects of myocardial viability. (2008). Growing over time, LV dilatation is a significant aspect of remodeling that is linked to both a rise in end-systolic volume and a decline in cardiac performance (13).

The current investigation shows that individuals with nonviable infarct zones showed gradual diastolic dilatation, which was followed by a concordant, albeit not equal, increase in end-systolic volume. This suggests that the left ventricular ejection fraction was preserved over time. This is unsurprising, given early-phase left ventricular dilatation seems to be the primary compensation mechanism for the myocardium's loss of contractility and restores the initially decreased ejection volume (14). Consequently, the ejection fraction does not alter during the early stages of left ventricular remodeling. Only 1-3 years following infarction, patients with increasing left ventricular dilatation experience a considerable drop in ejection fraction, as demonstrated by Gaudron et al. (10).

Conclusions.

1. Independent of the size of the infarct and the condition of the infarct-related artery lumen, the presence of a relatively large volume of viable myocardium after reperfusion in the myocardial infarction area significantly contributes to the preservation of the left ventricle's shape and size, preventing postinfarction remodeling.
2. It is possible to identify patients who are at a high risk of progressive dilatation of the left ventricle by using echocardiography in the early stages of the disease and using dobutamine.

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