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Predictors of Left Ventricular Remodeling after Revascularized Acute Myocardial Infarction

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ABSTRACT

Background: The acute loss of myocardium, following an acute myocardial infarction (AMI) leads to an abrupt increase in the loading conditions that induces a pattern of left ventricular remodeling (LVR). It has been shown that remodeling occurs rapidly and progressively within weeks after the AMI. **Study aim:** The aim of our study was to identify predictors for LVR, and find correlations between them and the cardiovascular (CV) risk factors that lead to remodeling. **Material and methods:** One hundred and five AMI patients who underwent primary PCI were included in the study. A 2-D echocardiography was performed at baseline (day 1 ± 3 post-MI) and at 6 months follow-up. The LV remodeling index (RI), was defined as the difference between the Left Ventricular End-Diastolic diameter (LVEDD) at 6 months and at baseline. The patients were divided into 2 groups, according to the RI: Group 1 – RI >15% with positive remodeling (n = 23); Group 2 – RI ≤15% with no remodeling (n = 82). **Results:** The mean age was 63.26 ± 2.084 years for Group 1 and 59.72 ± 1.267 years for Group 2. The most significant predictor of LVR was the female gender (Group 1 – 52% vs. Group 2 – 18%, p <0.0001). Men younger than 50 years showed a lower rate of LVR (Group1 – 9% vs. Group 2 – 20%, p = 0.0432). In women, age over 65 years was a significant predictor for LVR (Group 1 – 26% vs. Group 2 – 9%, p = 0.0025). The CV risk factors associated with LVR were: smoking (p = 0.0008); obesity (p = 0.013); dyslipidemia (p = 0.1184). The positive remodeling group had a higher rate of LAD stenosis compared to the no-remodeling group (48% vs. 26%, p = 0.002). The presence of multi-vessel disease was shown to be higher in Group 1 (26% vs. 9%, p = 0.0025). The echocardiographic parameters that predicted LVR were: LVEF <45% (p = 0.048), mitral regurgitation (p = 0.022), and interventricular septum hypertrophy (p <0.0001). **Conclusions:** The CV risk factors correlated with LVR were smoking, obesity and dyslipidemia. A >50% stenosis in the LAD and the presence of multi-vessel CAD were found to be significant predictors for LVR. The most powerful predictors of LVR following AMI were: LVEF <45%, mitral regurgitation, and interventricular septum hypertrophy.

Keywords: predictors, left ventricular remodeling, acute myocardial infarction, echocardiography, pPCI

INTRODUCTION

Despite the latest techniques and advances in acute myocardial infarction (AMI) treatment and management, the left ventricular remodeling (LVR) process that leads to congestive heart failure still represents a major problem.

Infarct expansion, regional dilation, thinning of the infarct zone and changes in myocardial contractility occur within the first days after myocardial infarction. Structural alterations in these normal regions have impact on global cardiac function and contribute to a poor clinical outcome in patients with infarct expansion.[1] This process is known as left ventricular remodeling and it usually occurs progressively, in patients after an acute MI, but also in patients with pre-existing chronic cardiovascular diseases, such as chronic high level hypertension, dilated cardiomyopathy or valvulopathies.[2] In the 1st week after a coronary artery occlusion, the infarcted area may show signs of thinning and stretching of the necrotic myocytes.[3,4]

The acute loss of myocardial cells leads to an abrupt increase in the ventricular loading conditions, increased wall stress, general inflammatory response, and induces a pattern of ventricular remodeling. All the previously mentioned processes involve the infarcted area, the border zone and the remote non-infarcted myocardium.[5] It has been shown that ventricular remodeling occurs rapidly and progressively within weeks after the acute MI, and consists in the alteration of the ventricular architecture, within the first hours after myocardial infarction.[6] The changes consist in an increased LV volume, which modifies left ventricular shape, from a physiological elliptical LV chamber shape to a more spherical and dilated configuration in time. On a histological level, the myocytes first undergo a hypertrophy process in the remote myocardium, followed by thinning, cellular apoptosis, myofibroblast proliferation, and by an increased interstitial collagen production that will lead to interstitial fibrosis.[7–9] Studies suggest that the collagen breakdown after myocardial infarction may contribute to the slippage of myocytes.[10]

Recent studies have established that a greater infarct size is associated with a higher risk of LV remodeling over time.[11,12] The development of LVR that takes place post-infarction remains one of the main determinants of long term survival. Over the past decades, scientists and healthcare providers have made substantial efforts to improve the understanding of this process by searching bad or good predictors and also risk factors which can be associated with this process.

LV diameters are easily measured and are commonly used as a substitute for volumetric analysis to evaluate the LV remodeling caused by ventricular overload or dysfunction.[13] Two-dimensional (2D) echocardiography is a widely available and well-established mean for assessing LV remodeling. The ability of echocardiography to measure cardiac structure and function non-invasively is generally accepted.[14] Also, this technique can be performed in nearly all patients, including those who are critically ill, or on day 1 ± 3 post myocardial infarction, and is not associated with any radiation exposure.[15] The evaluation of left ventricle volumes and ejection fraction is an important aspect of cardiac evaluation in all cardiac disorders. The LV ejection fraction has the advantage of being a simple, numerical parameter that reflects LV function, but it is strongly influenced by loading conditions and does not correlate well with symptom status.[16]

STUDY AIM

The aim of our study was to identify, to count and to find early predictors for LVR, and also to find correlations between them and the risk factors that lead to the progression of ventricular remodeling.

MATERIAL AND METHODS

We conducted a retrospective study, in which we included 105 consecutive patients with acute myocardial infarction (AMI), admitted between September 2014 and March 2015. All patients presented with clinical symptoms of AMI, with ST segment depression greater than 0.1 mV in two or more consecutive leads in the same vascular territory, and also an elevated creatinine-kinase MB (CK-MB) isoenzyme within 24h from the onset of symptoms.

All patients underwent succesful primary percutaneous coronary intervention (pPCI) within 12h from the onset of symptoms. Also, a 2-dimensional echocardiography assessment was performed at baseline (day 1 ± 3 post-MI) and during the 6-month follow-up. The 2-D echocardiographic images were obtained from the parasternal and apical windows.

The echocardiographic measurements of the left ventricle (left ventricular ejection fraction (LVEF); left ventricular diameters) were assessed, both at baseline and after 6 months, as well as the LV remodeling index (RI), defined as the difference between the Left Ventricular End-Diastolic diameter (LVEDD) at 6-month follow-up and baseline.

The study population was divided into 2 groups according to the remodeling index (RI), as follows: Group 1 — RI >15%, classified as the positive remodeling group (n = 23);

TABLE 1. Summarized results

	Remodeling group (>15%)		No remodeling group (<15%)		p value	RR	95% CI	
	n = 23	%	n = 82	%				
Mean age	63.26 ± 2.084		59.72 ± 1.267		0.1822			
Mean age men	60.45 ± 3.067		58.84 ± 1.414		0.6637			
Mean age women	65.83 ± 2.749		63.67 ± 2.711		0.5839			
Patients >65 years	10.00	0.43	27.00	0.33	0.1897	1.303	0.91	1.87
Patients <50 years	3	0.13	18	0.22	0.1358	0.5909	0.31	1.01
Men	11	0.48	67	0.82	<0.0001	0.5854	0.46	0.72
Men <50 years	2	0.09	16	0.20	0.0432	0.45	0.21	0.91
Women	12	0.52	15	0.18	<0.0001	2.889	1.85	4.6
Women >65 years	6	0.26	7	0.09	0.0025	2.889	1.46	5.81
Smokers	12	0.52	23	0.28	0.0008	1.857	1.3	2.6
Mean cholesterol value	173.9 ± 11.14		193.3 ± 5.334		0.1109			
LAD stenosis >50%	11	0.48	21	0.26	0.002	1.846	1.26	2.74
LAD stenosis >70%	5	0.22	12	0.15	0.2745	1.467	0.81	2.64
LAD occlusion	2	0.09	11	0.13	0.4986	0.69	0.31	1.51
CxA stenosis >70%	1	0.04	8	0.10	0.164	0.4	0.13	1.16
CxA stenosis >50%	5	0.22	14	0.17	0.4756	1.294	0.73	2.27
CxA occlusion	2	0.09	2	0.02	0.0582	4.5	1.13	18.21
2-vessel coronary artery disease	0	0.00	1	0.01				
3-vessel coronary artery disease	6	0.26	7	0.09	0.0025	2.8	1.46	5.81
TIMI 3 flow	11	0.48	56	0.68	0.0063	0.7	0.54	0.89
Left ventricular end-systolic diameter	50.39 ± 1.174		50.24 ± 0.6294					
Mean EF	50.39 ± 1.174		50.24 ± 0.6294		0.912			
EF <45% follow-up	4	0.17	6	0.07	0.0484	2.42	1.08	5.51
Mitral valve regurgitation stage A (primary)	12	0.52	29	0.35	0.0222	1.486	1.07	2.07
Anterior wall hypokinesis	4	0.17	18	0.22	0.4756	0.7727	0.43	1.35
Lateral wall hypokinesis	6	0.26	20	0.24	0.8704	1.083	0.67	1.68
Septal hypokinesis	6	0.26	13	0.16	0.1175	1.625	0.94	2.83
Septal thickness >11 mm	4	0.17	47	0.57	<0.0001	0.2982	0.18	0.46
Anterior MI	8	0.35	39	0.48	0.0848	0.7292	0.51	1.01
Posteroinferior MI	5	0.22	18	0.22	>0.9999			
Lateral MI	4	0.17	17	0.21	0.5891	0.8095	0.45	1.42
Diuretics	9	0.39	50	0.61	0.0029	0.6393	0.47	0.84
ACE inhibitors	16	0.70	73	0.89	0.0014	0.7865	0.67	0.9
HBP st 3	1	0.04	5	0.06	0.7475	0.6667	0.2	2.13
HBP st 2	19	0.83	67	0.82	>0.9999			
Dyslipidemia	11	0.48	49	0.60	0.1184	0.8	0.61	1.03
Diabetes mellitus	5	0.22	15	0.18	0.5963	1.222	0.7	2.12
Obesity	9	0.39	18	0.22	0.0137	1.773	1.14	2.77

Group 2 — RI ≤ 15% classified as the no-remodeling group (n = 82).

We also noted the cardiovascular risk factors (smoking, hypercholesterolemia, hypertension, diabetes mellitus, obesity, dyslipidemia). The study has been carried out in accordance with the code of ethics of the World Medical Association's Declaration of Helsinki. All patients gave written informed consent, and the study protocol was approved by the ethics committee of the Cardio Med Medical Center, where the study was conducted.

Statistical analysis

The statistical analysis was performed using the GraphPad Prism 7 software. Continuous variables were expressed as mean ± standard deviation. Categorical vari-

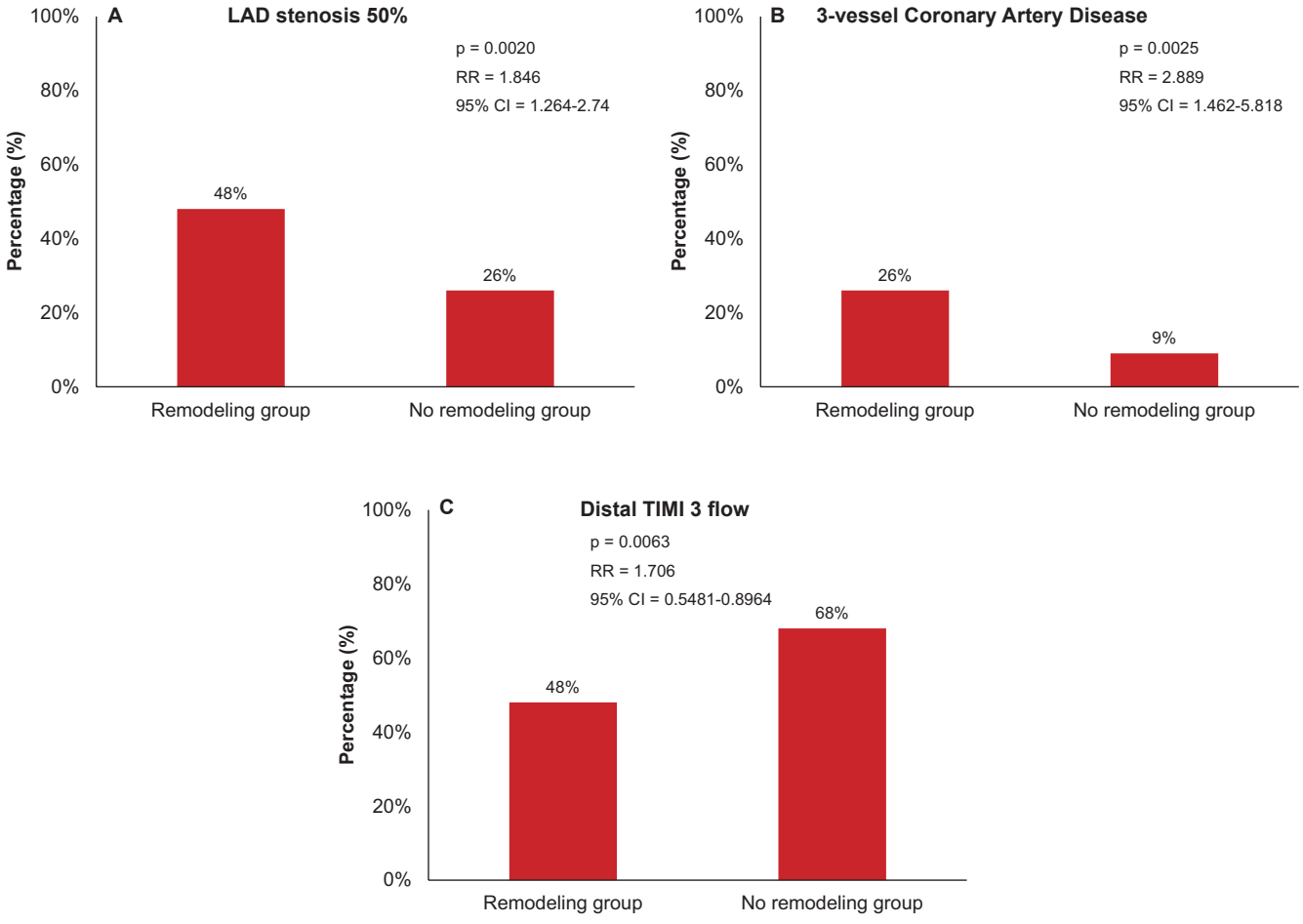

FIGURE 1. Angiographic aspects in the study groups. **A** – Presence of at least one 50% stenosis in the LAD is more frequent in the remodeling group. **B** – 3-vessel disease is more frequent in the remodeling groups. **C** – Presence of a TIMI 3 flow is significantly higher in the non-remodeling group. LAD = left anterior descendent artery

ables were presented as percentages. Categorical data were compared using the Chi-square and Fisher's exact tests. The unpaired t test was used to test for differences between the two groups, regarding the continuous variables.

RESULTS

The study included 105 patients, 78 male and 27 female, aged between 36 and 81 years, divided in two groups, according to the Remodeling index. The patient demographics, echocardiographic measurements and the cardiovascular risk factors are summarized in Table 1.

The mean age of the study population was 63.26 ± 2.084 years in Group 1 (the positive remodeling group) and 59.72 ± 1.267 years in Group 2 (the no remodeling group).

We also compared the mean age between men (60.45 ± 3 years in Group 1 vs. 58.84 ± 1.4 years in Group 2) and women (65.83 ± 2.7 years in Group 1 vs. 63.67 ± 2.7 years in Group 2), the difference was not statistically significant.

The most significant predictor of LVR was the female gender (52% in Group 1 vs. 18% in Group 2, p <0.0001, RR = 2.889, 95% CI = 1.85–4.6), women were more predisposed than men to develop ventricular remodeling after an acute myocardial infarction (48% in Group 1 vs. 82% in Group 2, p <0.0001, RR = 0.58, 95% CI = 0.46–0.72).

Surprisingly, men younger than 50 years showed a significantly lower rate of positive LVR, compared to older male subjects (9% in Group 1 vs. 20% in Group 2, p = 0.0432, RR = 0.45, 95% CI = 0.21–0.91). In women, age over 65 years was a significant predictor for developing LVR (26% in Group 1 vs. 9% in Group 2, p = 0.0025, RR = 2.889, 95% CI = 1.46–5.81).

Several cardiovascular risk factors were identified in a higher extent in the group with positive LVR compared

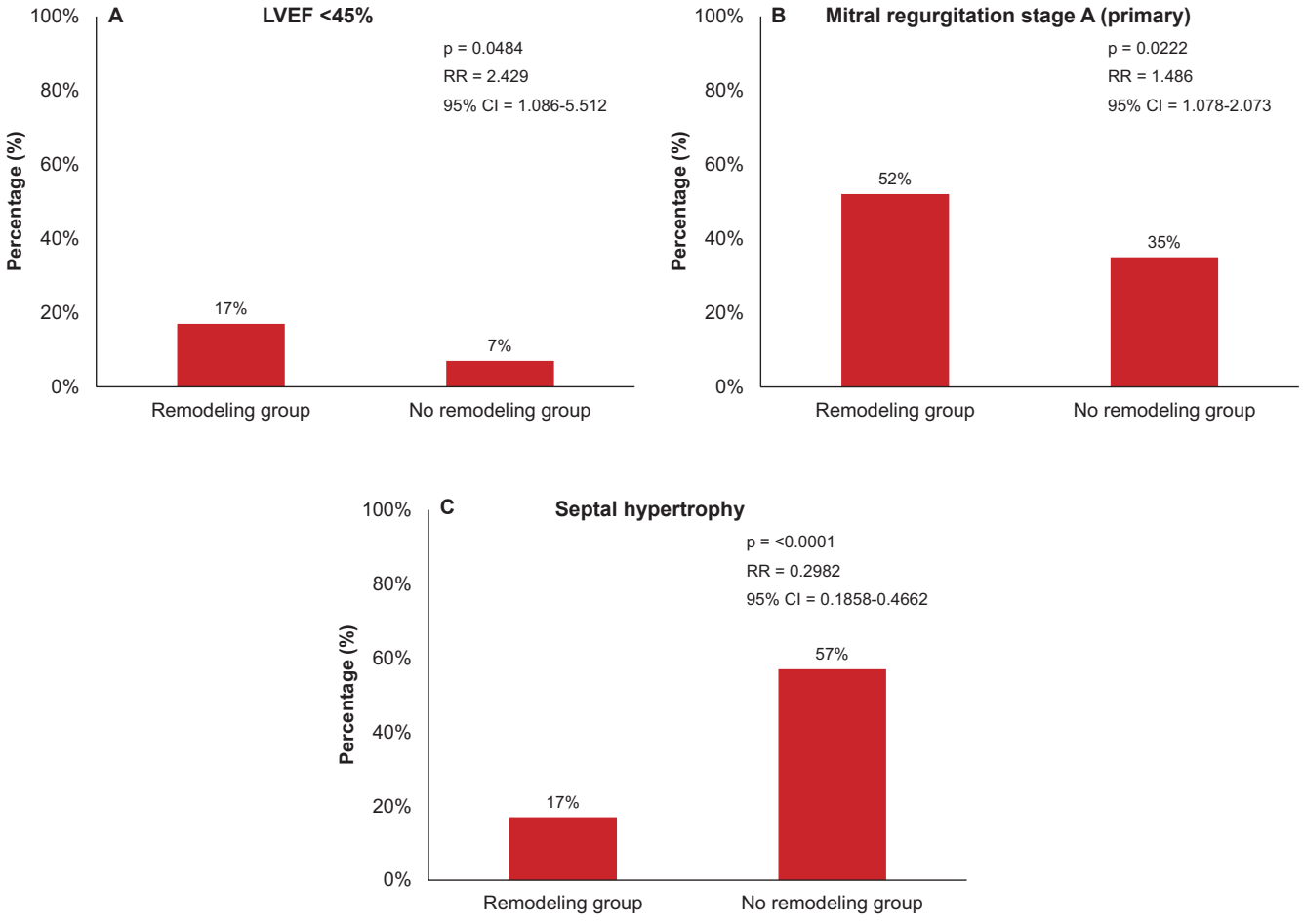

FIGURE 2. Echocardiographic aspects in the study groups. **A** – Left ventricular ejection fraction is significantly lower in the non-remodeling group. **B** – Mitral regurgitation is present in a significantly higher extent in the remodeling group. **C** – Septal hypertrophy is present in a significantly higher extent in the non-remodeling group. LVEF = Left ventricular ejection fraction

with the group with no LVR. Smoking status proved to be a significant risk factor in the ventricular remodeling process and the long-term prognosis (52% vs. 28%, p = 0.0008, RR = 1.857, 95% CI = 1.3–2.6).

Obese patients were significantly more exposed to develop LVR than the normal-weight subjects (39% in Group 1 vs. 22% in Group 2, p = 0.013, RR = 1.773, 95% CI = 1.14–2.77). We observed that arterial hypertension did not differ significantly between the positive remodeling and the no-remodeling group (83% vs. 82%, p >0.999).

Also, the rate of dyslipidemia was not significantly different between the 2 groups (48% in Group 1 vs. 60% in Group 2, p = 0.1184). Twenty patients were diabetic in our study, but the number did not differ between the two groups, thus, diabetes was found not to be a predictor for positive LVR (22% in Group 1 vs. 18% in Group 2, p = 0.5963).

Angiographic results

A significant coronary lesion was defined as a stenosis greater than 50% on the left main and the left anterior descendant artery (LAD). We found that the positive remodeling group had a higher rate of LAD stenosis compared to the no-remodeling group, the difference being statistically significant (48% vs. 26%, p = 0.002, RR = 1.847, 95% CI: 1.26–2.74), demonstrating that LAD stenosis greater than 50% plays an important role in the remodeling development (Figure 1, Diagram A). Furthermore, the presence of multi-vessel disease was shown to be higher in the remodeling group (26% vs. 9%, p = 0.0025, RR = 2.8, 95% CI: 1.46–5.81), hence proving that the three-vessel CAD was a significant predictor for LVR (Figure 1, Diagram B).

An optimal blood flow to the infarcted region has beneficial effects in attenuating ventricular enlargement. The distal TIMI flow was measured after pPCI, and we found

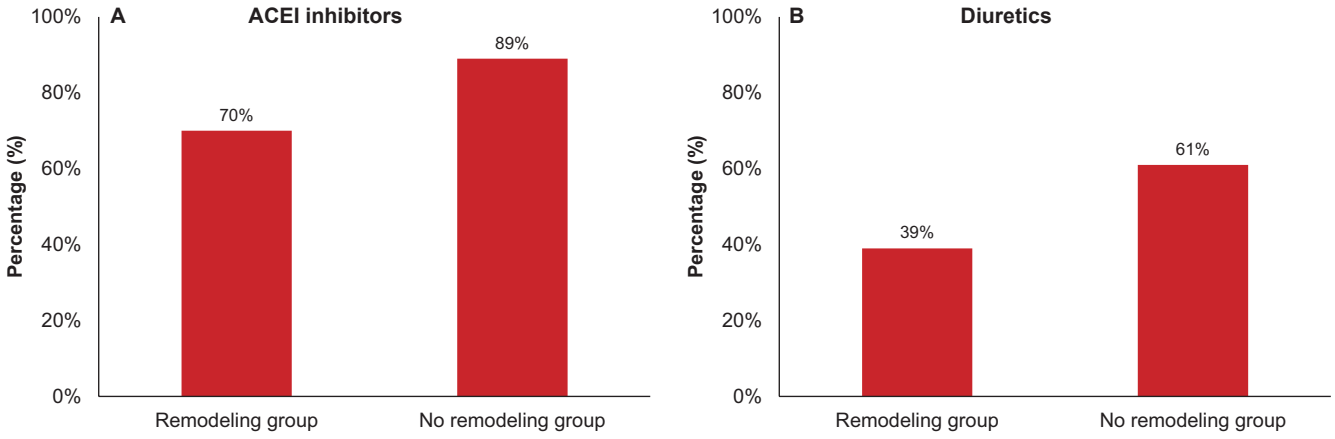

FIGURE 3. Bar graphs illustrating the treatment. **A** – The ACEI therapy after myocardial infarction is a significant protective factor against the progression of LVR. **B** – The therapy including diuretics after myocardial infarction protects againgst the rapid progression of LVR.

that the no-remodeling group had a significantly higher rate of optimal TIMI flow following revascularization (48% vs. 68%, p = 0.0063, RR = 0.7, 95% CI: 0.54–0.89) (Figure 1, Diagram C).

Echocardiographic results

When analyzing the echocardiographic parameters measured during the 6 months of follow-up, we found that 17% of patients from Group 1 (positive LVR) had a LVEF <45%, whereas only 7% of patients in the other group had a decreased ejection fraction (p = 0.0484) (Figure 2, Diagram A). Therefore, a LVEF <45% was proved to have a significant predictive capacity for LVR following an acute MI (RR = 2.42, 95% CI: 1.08–5.51).

The presence of mitral regurgitation (MR) influences the left ventricle by increasing the loading conditions, depending on the magnitude of the regurgitant leak. In our study population, we found that 52% of patients who presented with MR showed to have postitive LV remodeling after an acute MI, compared to only 35% of patients with MR that had no signs of LVR (p = 0.0222, RR = 1.486, 95% CI: 1.07–2.07) (Figure 2, Diagram B). The presence of mitral regurgitation resulted to be a significant predictor for LVR after an acute myocardial infarction.

When measuring the septal thickess upon echocardiographic examination, we found that the no-remodeling group had a higher frequency of interventricular septal hypertrophy (>11 mm thick) compared to the other group, therefore, the thickness of the septum is an important predictive factor for the LVR process (17% vs. 57%, p <0.0001, RR = 0.2982, 95% CI: 0.18–0.46) (Figure 2, Diagram C).

The left ventricular end-systolic diameter (LVESD) measured during the 6-month follow-up was significantly correlated with the LVEF for the no-remodeling group (p <0.0001, $r^2 = 0.1905$) and also with the positive remodeling group (p = 0.01505, $r^2 = 0.273$).

Treatment regimens

It is well known that angiotensin-converting enzyme inhibitors (ACEI) slow down the left ventricular remodeling process following an acute MI. In our study, 89% of patients in the no-remodeling group had received ACEI, compared to the other group, where only 70% of patients were treated with ACEI, the difference being highly statistically significant (p = 0.0014, RR = 0.78, 95% CI: 0.67–0.9) (Figure 3, Diagram A).

Myocardial infarction is one of the most common causes of systolic left ventricular failure. The therapy also includes associations between ACEI, digitals and diuretics. Diuretics alone should not be used as a long-term therapy in the post infarction period. In our study cohort, patients who had also received diuretics as a part of their treatment, showed to be less exposed to developing left ventricular remodeling (39% in Group 1 vs. 61% in Group 2, p = 0.0029, RR = 0.6393, 95% CI: 0.47–0.84) (Figure 3, Diagram B).

DISCUSSIONS

For both women and men, coronary heart disease (CHD) is the largest contributor to cardiovascular disease (CVD) morbidity and mortality.[17] However, gender has a profound impact, with important differences in cardiac re-

modeling between females and males.[18] Several studies have researched and demonstrated differences between males and females in cardiovascular disease. Concerning left ventricular remodeling after an acute myocardial infarction, systematic gender studies are still lacking. Recent studies regarding ventricular remodeling, performed on a laboratory rat population, did not find a significant difference between genders.[19,20] Moreover, studies have tried to find correlations between the level of estrogen and testosterone, related with age, in women that showed ventricular remodeling. After menopause, the cardiovascular risk becomes balanced between genders, and once affected by ischemic heart disease, females may have a worse prognosis than males.[21] In our study, the most significant predictor of LVR was the female gender; women were more predisposed than men to develop ventricular remodeling after an acute myocardial infarction.

Cigarette smoke exposure increases the risk of ventricular remodeling following acute myocardial infarction.[22] Similarly, our study showed that smokers were at higher risk for developing left ventricular remodeling following an acute MI.

Obesity has been increasing in epidemic proportions in the last decades and it has been demonstrated to have many adverse effects on hemodynamics and cardiovascular (CV) structure and function.[23,24] Overweight and obese individuals are often more exposed to developing left ventricular (LV) chamber dilation.[25] Ventricular remodeling, LV diastolic dysfunction, and elevated filling pressures may contribute to the development of heart failure in obese patients.[26] Obesity is most often associated with concentric LV remodeling.[27] In our analysis, obese patients were significantly more exposed to developing LVR than the normal weight subjects, thus concuring with the literature data. The association between obesity, heart failure and ventricular remodeling could result from a direct adverse effect caused by obesity on the cardiac function and structure. Indirectly, obesity promotes worse outcomes due to the coexisting disorders, such as coronary artery disease, high blood pressure or diabetes. Moreover, obesity is often considered to produce a state of chronic "volume overload".[28]

Recent studies have shown that approximately 50% of patients with STEMI have multivessel disease.[29,30] Our data showed that a LAD stenosis greater than 50% plays an important role in the remodeling development. Furthermore, the presence of multi-vessel disease was shown to be higher in the remodeling group, hence proving that the three-vessel CAD was a significant predictor for LVR.

Several studies have shown that a long treatment regimen with angiotensin-converting enzyme inhibitors, even if administered a few weeks after the initial myocardial infarction, can reduce the degree of postinfarction left ventricular dilation. Long-term therapy with an angiotensin converting enzyme inhibitor can favorably influence the loading conditions on the left ventricle and reduce progressive ventricular enlargement.[31–36] Also, studies have shown that the ACE inhibitors treatment leads to a significant reduction in the left ventricular end-systolic volume, an increased stroke volume and ejection fraction.[37] A large number of studies have demonstrated an increased survival in patients with MI, who had received ACE inhibitors in their therapy.[38,39] Correspondingly, our research has shown that patients who did not develop LVR were more likely to have received ACEI in their treatment plan, compared to those who actually did undergo left ventricular remodeling after the MI.

Study limitations

Our study had several limitations with reference to the small number of patients that were included, the patients represent only the subset of patients that had survived after myocardial infarction, the echocardiographic assessment of global left ventricular systolic function is usually performed subjectively. Two-dimensional echocardiography does not offer very precise data about the ventricular volumes, the contractility or the infarct size. LV diameter can be used to assess ventricular size and to diagnose ventricular enlargement, but because of the large range of error, this method is not suitable for precise assessment and follow-up of LV size in enlarged ventricles, for which volumetric analysis should be preferred.[36]

We also did not assess regional contractility or other parameters, all of these being easier and better assessed with a three dimensional echocardiography and cardiac magnetic resonance.

Despite its limitations, 2D echocardiography remains the most widely used non-invasive technique for clinical assessment of LV systolic function, and is likely to remain so because it is non-invasive, inexpensive, and widely available. The problems with accuracy and reproducibility of volumes and ejection fraction, pertained largely to the geometric challenges of 2D imaging, are likely to be solved by a 3D approach.

The study group was small, however the results were statistically significant compared with other studies.

CONCLUSIONS

Several variables have been shown to predict an increase in the left ventricular volume after myocardial infarction.

Women were more predisposed than men to develop LVR after AMI. Positive left ventricular remodeling was higher in men aged over 50 and women aged over 65 years.

The cardiovascular risk factors that were correlated with a positive left ventricular remodeling were smoking, obesity, as well as the presence of dyslipidemia.

The presence of a left anterior descendant artery stenosis of more than 50% and the presence of multi-vessel coronary artery disease were found to be significant predictors for LVR.

The echocardiographic parameters that had a predictive capacity for the progression of LVR after an MI were: a LVEF <50%, the presence of mitral regurgitation, and the interventricular septum hypertrophy.

Regarding the treatment regimens, patients who had received angiotensin-converting enzyme inhibitors were less likely to undergo the LVR process. Moreover, patients who had also received diuretics as a part of their treament plan, were less exposed to developing a positive remodeling of the left ventricle.

CONFLICT OF INTEREST

None declared.

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