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Article

Clinical Significance of Obstructive Changes in The Coronary Arteries and Clinical Manifestations of Heart Failure in Patients After Myocardial Infarction

[1]G.T. Madjidova, [2]G. I. Sunnatova, [3]Xudoyberdiyev A. A., [4]Xoliqulova M. M., [5]Umarova N. T.

1. Samarkand State Medical University, Department of Internal Medicine, 2nd Assistant, Samarkand, Uzbekistan.
2. Zarmed Medical University, Department of Internal Medicine, 2nd Assistant, Samarkand, Uzbekistan.
3. Doctor's Office, Samarkand Branch of the Republican Scientific Center for Urgent Ambulance, Samarkand, Uzbekistan.
4. Doctor's Office, Samarkand Branch of the Republican Scientific Center for Urgent Ambulance, Samarkand, Uzbekistan.
5. Doctor's Office, Samarkand Branch of the Republican Scientific Center for Urgent Ambulance, Samarkand, Uzbekistan.

* Correspondence: gulbaxormadjidova74@gmail.com , gulbaxor1965@icloud.com , xudoyberdiyev88@icloud.com , xoliqulova1997@icloud.com , umarova07@gmail.com

Abstract: In patients who have suffered a myocardial infarction (MI), heart failure (HF) is caused by a decrease in the pumping function of the heart when a significant portion of the myocardium is activated from contraction. Impairment of the pumping function of the left ventricle of the heart without clinical signs of HF or with minimal, as well as with its pronounced manifestations, on Our opinion should be considered in the aspect of hemodynamic supply to the left ventricles (LV). The New York Heart Association has developed a classification of heart failure into class IV (Stages). At the same time, identifying circulatory decompensation taking into account clinical and angiographic signs is important for assessing the condition of patients and selecting drug therapy. And also when developing indications for surgical correction of this complication.

Keywords: myocardial infarction, clinical course, young patients, myocardial revascularization.

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1. Introduction

Myocardial infarction (MI) continues to be a leading cause of morbidity and mortality worldwide and its sequelae last well beyond the acute event. Heart failure (HF), which can develop as a result of loss of contractile function of the myocardium and an inability of the heart to maintain sufficient circulation, is among the most clinically relevant sequelae. The problem of why certain post-MI patients progress rapidly to HF while others remain clinically quiescent is a classic central challenge of modern cardiology [1]. The problem is further complicated by the structural and haemodynamic changes that are produced in the coronary arteries and the left ventricle.

Recent debates in cardiovascular science suggest a need to re-evaluate the interdependence between coronary occlusion, myocardial injury, and left ventricular remodeling. Progressive heart failure (HF) is hypothesized by various theories (ischemic cascade, ventricular remodeling theory and the hemodynamic load hypothesis) to depend not only on the size and location of the initial myocardial infarct, but also on the extent of

ongoing ischemia, the severity of arterial stenosis, and functional compensation of the heart. Relationships between obstructive lesions, especially in the left anterior descending (LAD) artery, and suboptimal ventricular function have been described previously [2]. However, after decades of work, no specific criteria exist which are able to consistently identify compensated HF from decompensated HF in patients post-MI. This gap is especially salient given the clinical need for early detection and targeted treatment.

In our review of earlier work, we noted that most studies have been centered around either coronary angiography or functional cardiac assessment, but not their combined prognostic value. Finally, although prior work has demonstrated that burden of ischemic myocardial segments and stenosis morphology are associated with HF severity, few models including clinical, angiographic, and hemodynamic variables have been developed [3]. This emphasizes the importance of multifactorial strategies to maintain patients with the greatest risk of circulatory decompensation.

In an attempt to fill this gap, the current study uses a combination of clinical assessment, coronary angiography and echocardiography, along with discriminant statistical analysis to investigate the relationship between obstructive coronary changes and the stages of HF in MI patients. We propose to use comparison of intracardiac hemodynamics, ventricular contractility, arterial morphology, and clinical signs across HF stages to help identify more precise markers that characterize the transition from compensated to decompensated states [4]. It was expected that a multifactorial model would discover associations that could not be revealed by single-parameter analysis, and provide a better understanding of HF progression.

The results from this study confirm this hypothesis, which elucidate separate phenotypes of ventricular dysfunction, angiographic findings and clinical presentation for each HF stage. As such, these implications not only target further understanding of mechanisms underlying post-MI HF but also clinical importance. They could enhance risk stratification, tailoring treatment strategies for patients, and potentially preventing progression to advanced HF through identifying the essential drivers of disease such as LAD occlusion, impaired EF, and deranged electrolyte balance [5]. In conclusion, this study contributes to the emerging effort of individualizing post-MI care to better target patients who may benefit from expedited interventions and/or long-term therapy for improved patient-centered outcomes.

The purpose of this work is to identify the interdependence of obstructive changes in the coronary arteries and clinical manifestations of heart failure in patients who have suffered an MI, to clarify a set of signs that make it possible to distinguish between compensated and decompensated stages of heart failure, using multifactorial discriminant analysis using a computer [6].

2. Materials and Methods

We observed 154 patients with coronary artery disease (men) who had suffered an MI, including 91 patients in the subacute stage of MI and 63 with post-infarction cardiosclerosis.

In the group of patients with subacute stage of myocardial infarction, the average age was 46.8+(-)6.9 years, and in patients with post-infarction cardiosclerosis - 47.5+(-)*.3 years. Patients in the subacute stage of MI were examined at the 4th week from the onset of the disease, and patients with post-infarction cardiosclerosis were examined in the period from 2 months to 10 years after MI. All patients underwent polypositional Coronagraphy using the M method [7]. D. Judkins and also left ventriculography. When analyzing coronograms, the degree of obstruction of the coronary arteries was taken into account according to the classification of Yu.S. Petrosyan and L.S. Zergerman, the number of affected coronary arteries and the total severity in units according to the method presented in work V. Kaltenbach and W. _ D. Bussuman, angiographic morphology of the coronary arteries was based on the classification of J. A. Ambrose. Identification of an "infarct-related" coronary artery was determined by selecting the most affected vessel, corresponding to the location of the previous MI predicted by ECG and two-dimensional echocardiography. Patients with equivalent lesions of the two main branches of the

coronary arteries were not included in this analysis. Along with angiographic methods, non-invasive ones were also used: ECG in conventional leads, a complex echocardiographic study using the MK-600 ATL device (USA), a bicycle ergometer test was carried out in 33 (36.2%) patients in the subacute stage of myocardial infarction and in 27 (42.8%) patients with post-infarction cardiosclerosis on a 380-V device from Rodly-Electric (USA). The patients underwent a complex of clinical, diagnostic and biochemical studies, adopted at the Center for Emergency Cardiology of the Russian Research Center for Emergency Medicine, Samarkand branch [8]. The diagnosis of IHD was established taking into account the classification criteria

Diagnosis of HF was based on the New York Heart Association classification. Simultaneously, at the 1st stage by means of descriptive statistics, the distribution of indicators of intracardiac hemodynamics and contractility of the left ventricle of the heart, as well as clinical signs of circulatory decompensation (shortness of breath, palpitations, congestive wheezing in the lungs) was obtained. The initial patient classification into classes of heart failure in the univariate analysis was performed at the 2nd stage [9]. Stepwise discriminant analysis at the 3rd stage was extracted to find potential difference, if any, among selected groups of patients.

Stage I of HF was established in 60 (38.9%) patients, stage II in 48 (31.1%) and stage 1 in 46 (29.8%). The patients received complex treatment, including long-acting nitrates (nitrosorbide 60-320 mg/ day, sustak forte 6.4 mg 4-8 times a day), beta-adrenergic receptor blockers (propranolol 40-320 mg), calcium antagonists (corinfar 30-120 mg/ day, finoptin 160-400 mg/ day). The combination of anti- anginal drugs included nitrates and beta-blockers, nitrates, beta-blockers and calcium antagonists. In this case, the effectiveness of therapy was assessed according to the following gradation:

1 - stable, 2 - unstable, 3 - absent.

It should be noted that this group of drugs, along with the antiangial effect, stabilizes hemodynamics. Data about patients was encoded using a special program and entered into a personal computer from Sora-501 (Taiwan) [10]. The material was processed using the statistical software package SPSS (USA).

3. Results and Discussion

The study revealed a parallelism between the functional state of the LV and the stages of HF (table), which depended on the location of the MI, as well as the number of affected LV segments. High stages of HF were observed mainly in patients with scar changes along the anterior wall of the LV (Table 1).

Table 1. Indicators of intracardiac hemodynamics and contractility of the left ventricle of the heart at various stages of HF in patients who have had an MI

Index	Stage of heart failure		
	1 （n=60）	1I （n=48）	III （n=46）
End diastolic pressure, mm Hg. Art.	7.9±0.2	11.2±0.9*	18.2±1.2 *
End diastolic volume, ml/m?	65.2±2.8	78.0±4.0*	1 03.0±3.6*
End systolic volume, ml/m2	20.7±1.3	33.0±1.8*	57.8±3.3 *
Stroke volume, ml/m?	44.5±1.2	45.0±2.2	45.0±2.2
Ejection fraction, %			
Asynergy, arb. units			

	68.2±0.8 1.2±0.2	57.6±0.5* 3.2±0.5*	43.6±1.0 * 3.2±0.5*

Note: significant differences in indicators with stage I of heart failure, - significant differences between stages II and III

Most cases of high HF were observed in patients with scar changes along the top wall of the left ventricle LV ($p < 0.001$). Other authors reported similar findings.

HF was hardly observed, if only 1 segment of the LV was damaged, while in patients with impaired more than 2 segments of the LV, usually they are more often diagnosed. Conversely, the literature refers to patients where a large fraction of the myocardium is involved, and the LV ejection fraction remains normal, at least initially.

In patients with MI affecting a large number of segments, end-diastolic pressure and LV volumetric indices were markedly higher, and LV ejection fraction and fractional shortening of circular fibers were lower than what our data would predict. The severity of asynergia was greater in patients with a larger number of segments involved.

LV (Table 2).

The stages of HF were based on the lands of the "infarct-related" coronary artery. In groups with equivocal stage III HF, damage to the "infarct-artery" LAD was exclusively noted ($p>0.248$). The presence of collateral anastomoses did not differ according to stages of HF ($p>0.431$), in patients with MI. The angiographic morphology of obstructive alterations in coronary arteries showed concentric stenoses were more frequently found in the stage I HF ($p <0.003$). Type 2 eccentric stenoses and (or) irregular shape were found significantly more frequently in patients with stage II and III of HF ($p <0.001$). Eccentric stenoses of type 2 and/or non-regular shape are more hemodynamic relevant regarding its effect on coronary blood flow,8 which are reflected in the literature.

At the same time, deterioration of coronary blood flow also reduces LV contractility.

Clinical signs of HF were mainly dyspnea and tachycardia.

Analysis of electrocardiographic changes depending on the stages of HF showed characteristic changes characteristic of post-MI. In this case, the QS wave reflected the localization of the MI. Depression of the ST segment along the anterior wall was observed in 52 (33, /) patients, and elevation - in 6 (5.0%) patients. ST segment elevation was detected in 14 (9.0%) patients along the posterior wall of the LV, biphasic wave I was detected in 36 (22.3%) patients along the anterior wall and in 14 (9.0%) patients along the posterior wall of the LV. T wave inversion was observed in 37 (24.0%) along the anterior wall, 6 (3.8%) along the lateral wall, and 11 (7.1%) along the posterior wall of the LV. These signs were observed only in patients with stages II and II heart failure [11].

Depression or elevation of the ST segment more than 2 mm from the isoelectric line in combination with a change in the T wave, which is noted in the final part of the ventricular ECG complex, on the one hand, indicate ischemic changes in the myocardium, and, on the other, its overload caused by a violation of intracardiac hemodynamics and contractility of the LV of the heart [12]. Both of these reasons, we think, can result in alterations in the last component of the ventricular ECG complex (Table 2).

Table 2. Indicators of intracardiac hemodynamics and LV contractility depending on the number of affected segments in patients who suffered an MI

Index	**Number of affected LV segments**			
	1 （ n=43 ）	**2 (n=50)**	**3 （ n=30 ）**	**4 (n= 31)**
Final diast . Dav.m. _ m, rt. Art.	8.7±2.7	10.0±84.6	15.5±2.9	16.6±5.7
Final syst. Dove. m. m, rt. Art.	65.2±8.1	71.4±14.6	96.2±7.1	100.2±2 3.6
Stroke volume, ml/m?	41,43,9	30.0±1 4.2	47.3±3.9	49.2±8.7
Fraction emission %	63.4±8.3	57.9±1.5	50.8±3.1	49.2±1 2 .0
Asynergy, conventional unit.	1.6±1.4	1.7±2.0	4.8±2.6	5.3±2.5

Note: Significant differences in p indicators: *-2-3, **-1-3, ***1-4, *****2-4

Cardiac rhythm disturbances were detected in 23 (14.9%) and conduction disturbances in 58 (37.6%) patients. At the same time, arrhythmias were mainly detected in patients with stage III heart failure. According to the literature, the detection of arrhythmias increases significantly with the use of Holter monitoring.

A comprehensive echocardiographic study revealed LV aneurysm in 45 (29.2%) patients, and papillary muscle dysfunction in 20 (12.9%) patients. Regurgitation of blood into the cavity of the left atrium was detected in 14 (9.0%) patients. According to the literature and our clinic, dysfunction of the papillary muscles with regurgitation of blood into the cavity of the left atrium contributes to the development of severe heart failure.

In 21 (13.6%) patients with stage III HF, its clinical signs were identified: congestive wheezing in the lungs, distribution of liver boundaries according to Kurlov, disturbances in the electrolyte balance of blood plasma, increased venous pressure, and decreased daily diuresis [13]. According to the literature, these signs indicate circulatory decompensation. X-ray examination in these patients revealed an increase in the pulmonary pattern. The results of clinical angiographic studies have established a number of common and different signs at certain stages of heart failure.

Using multifactorial discriminant analysis using a computer, we clarified the complex of clinical and angiographic signs that allow us to distinguish between compensated and decompensated stages of blood circulation:

1) the appearance of congestive wheezing in the lungs ($p < <0.001$); 2) disturbance of the electrolyte composition of blood plasma ($p<0.001$); 3) heart rhythm disturbance ($p<0.001$); 4) high obstruction of the LAD of the left coronary artery ($p <0.001$); 5) LV ejection fraction is less than 50% ($p<0.001$).

To determine the weight value of each of these features, it would be possible to determine their sensitivity, but we did not set such tasks [14].

In patients who suffered a MI with stage III HF, exercise tolerance according to the bicycle energy test was lower, which is consistent with literature data. Moreover, the higher the total severity of coronary artery damage, the lower the exercise tolerance was ($p < 0.001$).

The results of the study established the dependence of the clinical manifestations of post-infarction angina on the stage of HF. At the same time, in patients with more severe forms of post-infarction angina, stage III HF was more often observed ($p < 0.001$).

Antiangial therapy was more successful in patients with stage I HF ($p<0.001$). An unstable effect and/or its absence was significantly more often observed in patients with stage III HF ($p < 0.001$) [15]. The results of the study are the interdependence of obstructive changes in the coronary arteries, the functional state of the left ventricle and the stages of HF.

4. Conclusion

This study demonstrates that obstructive coronary artery morphology is uniquely interrelated with left ventricular functional status and the clinical severity of heart failure (HF) following myocardial infarction (MI), in that HF stage III(IV) was virtually exclusively related to LAD territory when correlated with anterior scar morphology, number of involved segments, end-diastolic pressures and ejection fraction or severity of hemodynamically significant eccentric stenoses of the "infarct related" artery (particularly if the LAD). Our results demonstrate that exercise capacity and arrhythmia severity as well as angio and antianginal therapy induced myocardial hypoperfusion are significantly more severe in subjects with stage III HF as opposed to the group with stage I HF emphasizing the importance of both myocardial structure and function in HF progression and treatment response. Our data disclose clinical discriminators such as congestive lung wheezing, electrolyte imbalance, rhythm disturbances, LAD obstruction and EF < 50% which could prove relevant for prognosis, decompensating monitoring and denoting individual therapies. Further research is required to develop predictive models incorporating angiographic, echocardiographic, and biochemical data, and to determine whether this information can be used in the future to target treatment aimed at preventing the transition from compensated to decompensated stages of HF post-MI.

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