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THE PROCESS OF COMBINED INFERIOR MYOCARDIAL INFARCTION AND RIGHT VENTRICLE MYOCARDIAL INFARCTION WITH TRANSIENT ARTERIAL HYPOTENSION

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SUMMARY

Aim. To study the arterial hypotension influence to the progress of combined inferior and right ventricle myocardial infarction.

Material and methods. There were studied 66 patients with the primary inferior and right ventricle myocardial infarction. Patients were divided into 2 groups: 1st group – patients with inferior and right ventricle myocardial infarction with stable hemodynamics (n=34); 2nd group – patients with inferior and right ventricle myocardial infarction complicated by arterial hypotension in early period (n=32). Electrocardiography (ECG) and echocardiography (EchoCG) were performed in patients 1, 3, 30 days and 6 months after.

Results. On the 6th month of the disease, despite adequate conservative therapy, there was a significant increase of LV volume index in patients from the 2nd group, thus EDV LV was 164.2±4.2 ml^3, ESV 94.1±2.8 ml^3 (p<0.028). It was positive dynamics in rehabilitationof systolic function of RV in two groups. Thus EDS RV in patients from the 2nd group was 31.4±2.0 sm^3, and in patients from the 1st group 25.1±2.2 cm^3 (p<0.05), ESS RV in 2nd group became 19.8±2.0 cm^3, and in the 1st group 14.4±1.9 cm^3 (p<0.05). To the 6th month of disease FIP LV in the 1st group 40.3±3.2%, in the 2nd group 39.4% (p<0.05 in the group). To the 6th month of disease in the 1st group: ACS repeated episodes were in 6 patients (18%), 4 patients with heart failure FC III (12%), death was in 6% (2 patients). In the 2nd group: ACS repeated episodes were in 10 patients (30%) (OR 2.167; 95% CI 0.674-6.967), 7 patients with heart failure FC III (21%), death was in 10% (3 patients) (OR 2.2; 95% CI 0.190-25.517).

Conclusion. The patients with transitory arterial hypotension at the beginning of disease, have the singes of pathological LV remodeling, which complicate the course of disease, but the RV function becomes better.

Key words: *right ventricle myocardial infarction, echocardiography, left ventricle remodeling, diastolic function*

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INTRODUCTION

Acute myocardial infarction (MI) remains one of the most urgent problems in modern cardiology. MI is associated with significant prevalence and high mortality rates [1-6]. For a long time, the involvement of the right ventricle in the process of acute myocardial infarction was not taken into account. The main interest was represented by the left ventricle, as the main chamber that has a leading pumping role of the heart. Studies of myocardial infarction of the right ventricle (PZ) refer to the 80-ies of the last century. In 1974 Conn first described the unique hemodynamic consequences of myocardial infarction of the right ventricle [7]. Isolated myocardial infarction of the right ventricle is quite rare. According to the pathoanatomical study, it is found in 3-5% of patients with acute myocardial infarction [8]. Much more often myocardial infarction of the right ventricle is detected in combination with transmural myocardial infarction of the left ventricle (LV) of the lower localization [9-14]. The prognosis in patients with right ventricular myocardial infarction is unfavorable, intrahospital lethality is 30-40% [8], while in isolated myocardial infarction of the left ventricle of the posterior inferior localization – only 6% [15]. Clinical manifestations of right ventricular infarction vary widely: from their complete absence to hemodynamically severe hypotension, up to cardiogenic shock [16]. According to the literature, 25 to 50% of the right ventricular infarcts are hemodynamically significant [17, 18]. It is known that the development of the myocardial infarction is accompanied by a violation of synchronicity of contraction of various parts of the left ventricle, cardiac chamber, disadaptive remodeling of the left and right ventricles. Changes in the structure of the ventricular wall, the volume and shape (geometry) of the heart chambers often precede the clinical manifestation of heart failure syndrome, are precursors of cardiac decompensation and adversely affect on the quality of life and survival of patients [19, 20].

The aim of our study was to study left ventricular remodeling, inter- and intraventricular asynchrony in combined myocardial infarction of the lower wall of the left ventricle and right ventricle, with transient arterial hypotension.

RESEARCH MATERIAL

Prospectively, within 6 months, 66 patients with primary myocardial infarction of the lower wall of the left ventricle and right ventricle were examined. All patients included in the study were male and comparable in age and risk factors for coronary heart disease (table 1).

Table 1. Characteristics of patients included in the study

Indicators	1st group (n=34)	2nd group (n=32)
Age, years	55,32±6,69	55,29±2,21
Systolic blood pressure, mmHg	105,16±8,2	90,35±7,52*
Diastolic blood pressure, mmHg	67,26±8,03	58,71±6,7*
Height, cm	170,84±4,02	172,14±6,28
Body weight, kg	77,79±5,23	79,43±6,39
Total cholesterol, mmol/l	4,82±0,42	4,80±0,49

*Note: * - p <0.001 reliability of differences between compared groups, blood pressure*

Acute myocardial infarction of the lower wall of the left ventricle and right ventricle was diagnosed in accordance with generally accepted criteria of the European Society of Cardiology (EOK). The presence of arterial hypotension was diagnosed with a decrease in systolic blood pressure lasting up to 30 minutes within 80-90 mm Hg, in the absence of clinical signs of hypoperfusion.

Criteria for inclusion in the study:

- Acute myocardial infarction of the lower wall of the left ventricle and right ventricle;
- Informed consent to participate in the study

Criteria for exclusion from the study:

- Cardiogenic shock;
- Patients with postinfarction cardiosclerosis, ischemic cardiopathy;
- Patients with previous chronic heart failure;
- Hypertrophy of the left atrium, left ventricle, right atrium, right ventricle;
- Blockade of the left leg of the bundle of Gies
- Blockade of the right leg of the bundle of Gies
- Accompanying severe diseases of the liver, kidneys, diabetes mellitus
- Congenital or acquired heart defects
- Atrioventricular blockades of I, II, III degrees, chronic.

Depending on the presence of arterial hypotension patients were divided into two groups. Patients with myocardial infarction of the left ventricular and right ventricular base without arterial hypotension (n=32) were included in group 1, and patients with myocardial infarction of the left ventricular and right ventricular of lower walls, complicated by arterial hypotension, were included in the 2nd group (n=34). Groups of patients by age and accompanying pathology were comparable.

Treatment. All patients received thrombolytic therapy (streptokinase 1.5 million IV drip for 30 minutes at the prehospital stage), acetylsalicylic acid (250 mg once in the prehospital stage, then 100 mg per day), clopidogrel (the first dose is 300 mg, then 75 mg in day), heparin (7.5 thousand units 2 times a day – 5 days), bisoprolol (initial dose 1.25 mg/day, then up to 2.5 mg/day), statins (atorvastatin 80 mg/day), enalapril (up to 20 mg/day). Patients of the second group, if necessary, received sympathomimetics (dopamine 2-20 μg/kg/min intravenously), after stabilization of hemodynamics, they also got beta-blockers and inhibitor angiotensin converting enzyme, under the control of hemodynamics.

METHODS OF RESEARCH

ECG was recorded in 12 standard leads, as well as in RV3-RV4.

Echocardiography. The following parameters were analyzed: anteroposterior size of the left atrium (LA, cm) in diastole; end-diastolic size of the left ventricle (LVEDD, cm); the final systolic size of the left ventricle (LVESD, cm); end-diastolic volume of the left ventricle (LVEDV, ml^3); end-systolic volume of the left ventricle (LVESV, ml^3); ejection fraction of the left ventricle (LVEF, %) (according to Simpson using bi-plane mode); end-diastolic area of the right ventricle (RVEDA, cm^2); the end-systolic area of the right ventricle (RVESA, cm^2); area change fraction of the right ventricular area (RVFAC, %), calculated by the formula: RVEDA – RVESA / RVEDA x 100%; TAPSE (excursion of the tricuspid ring, mm). Patients of both groups had hypokinesis of the basal lower subordinate, basal lower, middle lower subpermoral, middle lower, apical lower segments of the left ventricle; anterior, lateral and lower free walls of the right ventricle (RV).

Left ventricular remodeling was evaluated by calculating geometric parameters: systolic sphericity index (SSI), calculated

according to the formula: SSI = LVESD / Hs, where Hs is the height of the left ventricle in systole; diastolic sphericity index (DSI), calculated according to the formula: DSI = LVEDD / Hd, where Hd is the height of the left ventricle in diastole; myocardial stress diastolic (MSD, units), calculated according to the formula: MSD = 0.334 x arterial pressure diastolic x LVEDD / thickness of posterior wall of left ventricle in diastole (LVPWTd) x (1+(LVPWTd / LVEDD)); the relative wall thickness of the interventricular septum (RWT IVS, cm), calculated by the formula: RWT IVS = 2 x WT IVSD / LVEDD; relative thickness of the left ventricular posterior wall (RTPWLV, cm), calculated by the formula: RTPWLV = 2 x LVPWTd / LVEDD; the relative thickness of the walls of the left ventricle (2H/D) according to A. Canauetal (1992): 2H/D = (LVPWTd + WT IVSD) / LVEDD.

In order to differentiate the normal and pseudonormal types of transmittal blood flow, a Valsalva test was used. As a result, there was a decrease in venous return to the heart, which translated the pseudo-normal type of transmittal blood flow into a type of delayed relaxation (the ratio E/A >1 before the sample is transformed at the height of the Valsalva test in E/A <1).

Definition of global intraventricular asynchrony (GVA) and interventricular asynchrony (IVA) (Fabian Knebel, Rona Katharine Reibis et al., 2004). Global intraventricular asynchrony (GVA) was assessed in the M-modal mode as the time difference between the maximal contraction of interventricular septum и posterior wall of left ventricle in diastole. Interventricular asynchrony was calculated in the Doppler mode as the difference between the pre-injection intervals between the aortic flow (from Q on the ECG to the onset of the aortic flow) and pulmonary flow (from Q on the ECG to the onset of the pulmonary flow).

The test with 6-minute walking was conducted with the aim of objectifying the functional status of the patient with chronic heart failure, and also for assessing the physical tolerance and effectiveness of the therapy.

Statistical analysis of the results was carried out using the SPSS program. Intergroup variables were analyzed using the Independent-Samples T-test (Kolmogorov-Smirnov test) to determine the reliability of the differences. Intragroup variables were exposed to the Paired-Samples T-test to determine the reliability of the differences. The data are presented in the form of a normal distribution of the arithmetic mean and standard deviation. The level of statistical significance was assumed to be p <0.05.

RESULTS AND DISCUSSION

Analysis of the results of ECHO on the 3rd day of the disease showed that patients with combined myocardial infarction with unstable hemodynamics showed more pronounced signs of systolic dysfunction of both left ventricle and right ventricle, the data are presented in table 2. Thus, end-diastolic volume of left ventricle (LVEDV) end-systolic volume of left ventricle (LVESV) were more in patients of 2nd group than in patients of 1st group. (p<0,0001).The size of the right ventricle in patients of the 2nd group significantly exceeded the parameters of 1st group (p<0,05), which was accompanied by a lower level of fraction area change of right ventricle in patients of the 2nd group (p<0,05), this fact was confirmed by TAPSE indicator in patients of the 2nd group (p<0.05).

Table 2. Indices of systolic function of LV and RV in patients with MI on day 3 of the disease

Parameter	1 group (n=34)	2 group (n=32)			
LA, cm	3,22±0,18	3,23±0,11	2H/D, units	0,36±0,02	0,31±0,02
LVEDD, cm	5,1±0,29	5,9±0,23*	MSD, units	187,2±12,5	210,3±14,2*
LVESD, cm	3,5±0,16	4,1±0,19*	RWT IVS, cm	0,29±0,02	0,33±0,02**
LVEDV, ml^3	134,8±3,0	152,5±2,27***	RTPWLV, cm	0,29±0,02	0,32±0,01***
LVESV, ml^3	68,3±4,3	79,73±4,02***	EDP, mm Hg	14,12±0,45	18,25±0,5***
LVEF, %	49,3±2,6	47,7±2,24	IVA, ms	37,4±4,2	46,1±3.7***
RVEDA, cm^2	29,21±1,6	33,44±1,32*	GVA, ms	98,55±12,4	142,6±12,4*
RVESA, cm^2	18,6±1,2	23,1±1,6*	Elv, cm/s	52,6±2,4	44,5±2,8*
RVFAC, %	36,3±2,1	30,8±1,6*	Alv, cm/s	61,3±2,2	68,2±2,4*
TAPSE (ETR), mm	15,2±1,6	10,1±1,7*	E/Alv, units	0,85±0,03	0,65±0,03***
IVS, cm	0,97±0,03	0,97±0,03	DTlv, ms	215,4±9,2	223,86±7,6
LVPWD, cm	0,88±0,03	0,89±0,03	Erv, cm/s	41,2±3,2	37,79±3,1
DSI, units	0,51±0,02	0,59±0,02**	Arv, cm/s	60,1±3,4	62,9±4,01
SSI, units	0,50±0,02	0,51±0,03	E/A, units	0,68±0,04	0,60±0,04
			ETrv, ms	284,3±3,1	299,5±3,2*

*Note: *p <0,05; ** p <0,001; *** p <0,0001 compared with the same value in the opposite group.*

LA – left atrium; LVEDD – end-diastolic size of the left ventricle; LVESD – end systolic size of the left ventricle; LVEDV – end-diastolic volume of the left ventricle; LVESV – end-systolic volume of the left ventricle; LVEF – left ventricular ejection fraction; RVEDA – end-diastolic area of the right ventricle; RVESA – end-systolic area of the right ventricle; RVFAC – fraction area change of the right ventricle; TAPSE (ETR) – excursion of the tricuspid ring; IVS – interventricular septum; LVPWD – Left ventricular posterior wall diastole; DSI – diastolic sphericity index; SSI – systolic sphericity index; 2H/D – the relative wall thickness of the left ventricle; MSD – myocardial stress diastolic; RWT IVS – the relative thickness of the interventricular septum; RTPWLV – the relative thickness of the posterior wall of the left ventricle; EDP – end-diastolic pressure; IVA – interventricular asynchrony; GVA – global intraventricular asynchron; Elv – the maximum rate of early filling of the left ventricle; Alv – maximum late filling rate of the left ventricle; E/Alv – ratio of maximum rates of early and late filling of the left ventricle; DTlv – the deceleration time of early left ventricular filling; Erv – maximal speed of early filling of the right ventricle; Arv – maximum rate of late filling of the right ventricle; E/A – ratio of maximum rates of early and late filling of the right ventricle; ETrv – ejection time from the right ventricle to the pulmonary artery

In the dynamics, on the 30th day from the onset of the disease, a significant increase in the left ventricular end-systolic volume in patients of the 2nd group was found and was 86.5±3.8 ml3, versus 76.4±4.2 ml3, ($p<0.035$). End-diastolic LV volume (LVEDV) in patients of the 2nd group did not actually change. There was a marked decrease in LVEF in patients of both groups: LVEF in patients of the 1st group was 46,4±2,7%, in patients of the 2nd group – 44,6±2,64% ($p<0,0001$ in the dynamics within the group). By the 6th month of the disease, despite adequate conservative therapy, there was a significant increase in the volume of left ventricular parameters in patients of 2nd group, so LVEDV became 164.2±4.2 ml3, LVESV – 94.1±2.8 ml3 ($p<0.028$). The positive dynamics in restoration of RV systolic function in patients of both groups was traced. For example, RVEDA in the patients of the 2nd group was 31.4±2.0 cm^2, while in the patients of the 1st group it was 25.1±2.2 cm^2 ($p<0.05$), RVESA in the patients of the 2nd group – 19.8±2,0 cm^2, in patients of the 1st group – 14,4±1,9 cm^2 ($p<0,05$). By the 6th month of the disease, fractional area change of the right ventricle (RVFAC) in patients of the 1st group was 40.3±3.2%, in patients of the 2nd group – 39.4% ($p<0.05$ within the group).

On day 3 of the disease, a significant increase in diastolic spherical index was registered in patients of the 22nd group ($p<0.007$). In patients with MI with unstable hemodynamics on day 30 of the disease, an increase in the diastolic and SSI was noted. So the DSI in patients of the 2nd group was 0.61±0.02 compared to 0.57±0.02 in patients of the 1st group ($p<0.05$). At 6 months from the onset of the disease in patients of the 2nd group, signs of disadaptive remodeling with an increase DSI and SSI were observed: DSI – 0.68±0.03 units ($p<0.038$), SSI – 0.59±0.02 units ($p<0.038$). The differences between the groups for the thickness of IVS and LVPW were not observed. The LVEDP is significantly higher in patients of 2nd group on day 3 of the onset of MI ($p<0.0001$).

It is known that acute myocardial infarction is the most severe manifestation of coronary heart disease. The defeat of a large area of the heart muscle and the "expansion" of the scar leads to a regional mechanical heterogeneity. As a result, asynchronous movement between the damaged and intact parts of the left ventricular wall arises and increases. Recently, great importance in the development and progression of heart failure has been attributed to cardiac asynchronism, which is an independent predictor of the development of heart failure. It is shown that in 40% of patients with myocardial infarction, especially in case of conduction disorders, coordination of the reduction of the walls of the left ventricle is disturbed already in the first days. Asynchronous reduction is observed, cardiac efficiency as a pump worsens, heart failure develops and / or progresses [21-29].

As the results showed, a significant increase in both global intraventricular asynchrony (GVA) and interventricular asynchrony (IVA) was recorded in 2nd group in patients on day 3. So IVA in patients of the 1 group 37.4±4.2 ms versus 46.1±3.7 ms in

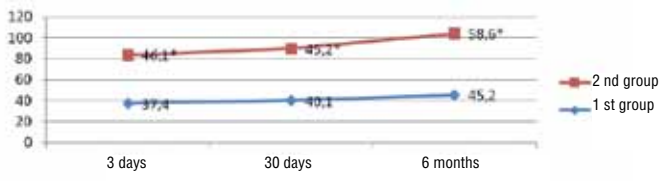

Figure 1. Dynamics of the severity of IVA, ms for 6 months in patients with MI, * p <0.05

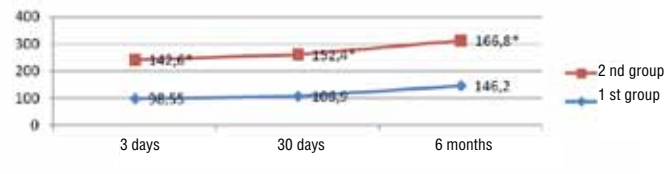

Figure 2. Dynamics of the severity of GVA, ms for 6 months in patients with MI, * p <0.05

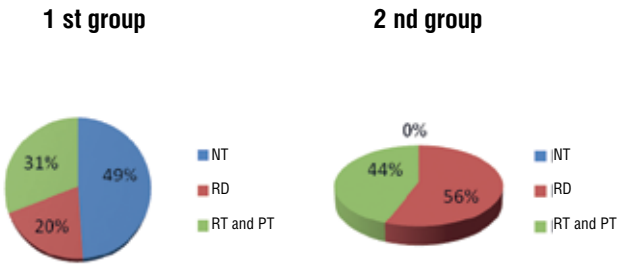

Figure 3. Diastolic function of the LV in patients with MI at 6th months of the disease

Note: NT is a normal type of diastolic function, RD is a relaxation disorder, RT is a restrictive type of diastolic function, PT is a pseudonormal type of diastolic function.

patients of the 2nd group ($p<0.0001$), GVA in patients of the 1st group 98.55±12.4 ms versus 142, 6±12,4ms ($p<0,015$), the data is in Figure 1. By 30 days from the onset of the disease, an increase in GVA was recorded in patients of both groups, but to a greater extent in patients of the 2nd group ($p<0.05$). By the 6-month period of the disease there was an increase in IVA and GVA in patients of the 2nd group, which is reflected in Figures 1 and 2.We analyzed the types of transmittal Doppler flow and their occurrence in patients with MI. Patients in 1st group in 6 months of the disease had a more pronounced diastolic LV dysfunction in 31% of cases in the form of a restrictive and pseudo normal type of diastolic function, with diastolic left ventricle normalization in 20%, and in 49% of patients – relaxation disturbances persisted. In patients in the 2nd group in the 6th month of the disease, an unfavorable type of left ventricular diastolic dysfunction (restrictive and pseudonormal) was recorded in 44% of cases, and in the remaining 56% of the patients there were signs of disturbance of the relaxation (Fig. 3).

Patients with MI were followed up for 6 months. We evaluated the frequency of development of lethal outcomes, repeated episodes of acute coronary syndrome, repeated hospitalization, heart failure. On the 30th day of the disease one death was observed in patients of the 2 nd group, which was 3.7%; repeated cases of acute coronary syndrome in patients of the 1st group – one case (3.4%), in patients of the 2nd group – six (22.2%) ($p<0.02$) (OR 8, 95% CI 0.894-71.598). Clinical signs of heart failure were noted in nine patients of the 2 nd group (33.3%), in two patients of the 1st group (6.8%) (OR 6.75, 95% CI 1.304-34.943) ($p<0.01$).

By the 6th month of the disease in patients of the 1st group: repeated episodes of acute coronary syndrome – In 6 patients (18%), cardiac insufficiency of FC III in 4 people (12%), lethal outcomes – 2 people (6%). In patients of the 2nd group: repeated episodes of ACS – 10 people (30%) (OR 2.167, 95% CI 0.674-6.967), cardiac insufficiency of the FC III – 7 people (21%), lethal outcomes – 3 people 10%) (OR 2.2, 95% CI 0.190-25.517).

DISCUSSION

In our study, in an acute period of combined MI, violations of systolic and diastolic functions of both ventricles were revealed, more pronounced in patients with arterial hypotension. In the dynamics, there was an improvement in the systolic and diastolic functions of the right ventricle in both groups of patients studied on the background of aggravation of left ventricular dysfunction, more pronounced in group 2. Patients with transient arterial hypotension undergo pathological remodeling of the left ventricle. Clinically, the group with arterial hypotension also has a complicated course of the disease. The data obtained by us correspond to the studies of Klein and Garg and others [30-32], reporting a severe clinical course of MI of the LV in combination with MI of the RV. Clinical manifestations of RVI vary widely: from their complete absence to hemodynamically severe hypotension, up to cardiogenic shock [16]. Thus, arterial hypotension in patients with MI of the lower wall of the LV and RV is a manifestation not only of RV dysfunction, but also of the LV. In addition, in dynamics, the function of the right ventricle improves both in patients with arterial hypotension, and without it. This fact is also reflected in the study of Bowers and others, reporting restoration of right ventricular function after reaching reperfusion [7]. Based on the results of other studies, the majority of patients with acute right ventricular myocardial infarction experience an early improvement in hemodynamics, and then a recovery of the right ventricle, even in the absence of reperfusion of the infarction-related artery [32-34]. Further prognosis depends on left ventricular ischemia [35-37]. Thus, the complicated clinical course of the disease is associated with increasing left ventricular dysfunction in patients with combined myocardial infarction with arterial hypotension.

CONCLUSIONS

1. Patients with combined MI with arterial hypotension in the onset of the disease were characterized by more pronounced signs of pathological remodeling, diastolic LV dysfunction, inter- and intraventricular asynchrony, with the above changes progressing over time (by the 6th month of the disease).

2. By the 6th month of the disease, improvement of the RV functions occurred not only in patients of 1st group, but also in patients of 2nd group (with arterial hypotension).

3. Within 6 months there was a more severe clinical course in patients with unstable hemodynamics at the onset of the disease. Thus, in patients of the 1st group: repeated episodes of acute coronary syndrome – in 6 patients (18%), cardiac insufficiency of the FC III – in 4 people (12%), lethal outcomes – 2 people (6%). In patients of the 2nd group: repeated episodes of acute coronary syndrome – 10 people (30%) (OR 2.167, 95% CI 0.674-6.967), heart failure of FC III – 7 people (21%), deaths – 3 people 10%) (OR 2.2, 95% CI 0.190-25.517).

A conflict of interests. All the authors declare no potential conflict of interest requiring disclosure in this article.

BIBLIOGRAPHY

1. *Bystrov V.V., Makarenko A.S., Dyachok S.V., Rakov A.L. Features of the electrocardiographic pattern in isolated myocardial infarction of the right ventricle. Klin.medicina 2001;6:61-63 [in Russian].*
2. *Netyazhenko V.Z., Plienova O.M., Stroganova N.P. et al. Clinical aspects of thrombolytic therapy. Clinicalpharmacology, physiology, biochemistry 1997;1:71-80 [in Ukrainian].*
3. *Ostroumov E.N., Karmer A.Ya., Ermolenko A.E. Emission fraction of the right ventricle as an indicator of the effectiveness of revascularization in patients with ischemic heart disease with congestive circulatory insufficiency. Cardiology 1996;4:57-61. [in Russian].*
4. *Antman E., Hand M., Armstrong P. et al. Focused Update 2007 of the ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction (STEMI): A Report of the ACC/AHA Task Force on Practice Guidelines. Circulation 2008;117:296-329.*
5. *Oguzhan A., Abaci A., Eryol N.K. Evaluation of right ventricular function by colour Doppler tissue imaging can help to identify patients with right ventricular infarction. Eur. Heart J. 2000;21:110-119.*
6. *Serrano Junior C.V., Ramires J.A., Cesar L.A. Prognostic significance of right ventricular dysfunction in patients with acute inferior myocardial infarction and right ventricle involvement. Clin. Cardiology 1995;18:1210-1218.*
7. *Bowers T.R., O'Neill W.W., Grines C. et al. Effect of reperfusion on biventricular function and survival after right ventricular infarction. N Engl J Med. 1998; 338:933–40.*
8. *Sagawa K. The End-systolic pressure-volume relation of the ventricle: definition, modification and clinical use. Circulation 1981;63:1225-1227.*
9. *Braat S.H., Brugada P., de Zwaan C. et al. Value of electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction. Brit. Heart J. 1983;49:368-372*
10. *Cohn J.N., Guiha N.H., Broder M.I., Limas C.J. Right ventricular infarction: Clinical and hemodynamic features. Amer. J. Cardiology 1974; 33:209-214.*
11. *Klein H.O., TordjmanT., Ninio R. et al. The early recognition of right ventricular infarction: diagnostic accuracy of the electrocardiographic V4R lead. Circulation 1983;67:558-565.*
13. *Lopez-Sendon J., Coma-Canella I., Gamallo C. Sencitivity and specificity of hemodynamic criteria in the diagnosis of acute rigft ventricular infarction. Circulation 1981; 64:515-525.*
14. *Stoian I., Ginghina C., Coman I. The relation between right ventricular ejection fraction and left ventricular wall motion score. Eur. Heart J 2001; 22:580-586.*
15. *Orlov M.V., Grachev S.P., Nesvetov V.N. Complex ultrasound in the diagnosis and evaluation of heart function with myocardial infarction of the right ventricle. Cardiology 1989; 3:41-45 [in Russian].*
16. *Isner J.M., Roberts W.C. Right ventricular infarction complicating left ventricular infarction secondary to coronary heart disease: Frequency, location, associated findings and significance from analysis of 236 necropsy patients with acute or healed myocardial infarction. Am J Cardiol. 1978;42:885–94.*
17. *Shah P.K., Maddahi J., Berman D.S. Scintigraphically detected predominant right ventricular dysfunction in acute myocardial infarction: Clinical and hemodynamic correlates and implications for therapy and prognosis.J Am CollCardiol. 1985;6:1264–72.*
18. *Horan L.G., Flowers N.C. Right ventricular infarction: Specific requirements of management. Am Fam Physician. 1999;60:1727–34.*
19. *Lamas G.A., Pfeffer M.A. Left ventricular remodeling after acute myocardial infarction: clinical course and beneficial effects of angiotensin; converting enzyme inhibition. Am. Heart. J 1991; 121 (4), part. 1:1194–1202.*

20. Sabbah H.N., Goldstein S. Ventricular remodelling: consequences and therapy. Eur. Heart. J 1993;14:24–29.
21. Bordachar P., Garrigue S., Lafitte S. et al. Interventricular and intra-left ventricular electromechanical delays in right ventricular paced patients with heart failure: implications for upgrading to biventricular stimulation. Heart 2003; 89:1401-1405.
22. Cardim N., Castela S., Cordeiro R. et al. Tissue Doppler imaging assessment of long axis left ventricular function in hypertrophic cardiomyopathy. Rev Port Cardiol 2002 Sep; 21(9): 953-85
23. D'Andrea A., Ducceschi V., Caso P. et al. Usefulness of Doppler tissue imaging for the assessment of right and left ventricular myocardial function in patients with dual-chamber pacing. Int J Cardiol. 2001 Nov; 81(1): 75-83
24. Kass D.A., Chen C.H., Curry C. et al. Improved Left Ventricular Mechanics From Acute VDD Pacing in Patients With Dilated Cardiomyopathy and Ventricular Conduction Delay. Circulation. 1999; 99: 1567-1573
25. Yu C.M., Lin H., Yang H. et al. Progression of systolic abnormalities in patients with "isolated" diastolic heart failure and diastolic dysfunction. Cirulation 2002; 105: 1195–201
26. Wildgruber M., Bielicki I., Aichler M. et al. Assessment of myocardial infarction and postinfarction scar remodeling with an elastin-specific magnetic resonance agent. Circ Cardiovasc Imaging. 2014; 7(2): 321-329.
27. Reis Filho J.R., Cardoso J.N., Cardoso C.M. et al. Reverse Cardiac Remodeling: A Marker of Better Prognosis in Heart Failure. Arq Bras Card. 2015; 104(6): 502-506.
28. Grothoff M., Elpert C., Hoffmann J. et al. Right ventricular injury in ST-elevation myocardial infarction: risk stratification by visualization of wall motion, edema, and delayed-enhancement cardiac magnetic resonance. Circ Cardiovasc Imaging. 2012; 5(1): 60-68.
29. Ondrus T., Kanovsky J., Novotny T. et al. Right ventricular myocardial: From pathophysiology to prognosis. Exp Clin Cardiol. 2013; 18(1): 27-30.
30. Yasuda T., Okada R.D., Leinbach R.C. et al. Serial evaluation of right ventricular dysfunction associated with acute inferior myocardial infarction. Am Heart J. 1990;119:816–22. [PubMed]
31. Chanal A.A., Kim M.Y., Borg A.N. et al. Primary angioplasty for infarction due to isolated right ventricular artery occlusion. World J Cardiol. 2014; 6(11): 1223-1226.
32. O'Rourke RA, Dell'Italia LJ. Diagnosis and management of right ventricular myocardial infarction. CurrProblCardiol. 2004;29:6–47. [PubMed]
33. Lim St., Marcovitz P., O'Neill W.A., Goldstein J.A. Right ventricular performance is preserved during stress in patients with chronic proximal right coronary occlusion. J Am CollCardiol 2001;37:2 Suppl A:347A.
34. Yasuda T., Okada R.D., Leinbach R.C.,et al. Serial evaluation of right ventricular dysfunction associated with acute inferior myocardial infarction. Am Heart J .1990; 119:816–822.
35. Sheehan F.H., Braunwald E., Canner P. et al. The effect of intravenous thrombolytic therapy on left ventricular function: a report on tissue-type plasminogen activator and streptokinase from the Thrombolysis in Myocardial Infarction (TIMI Phase I) Trial. Circulation 1987; 75:817–829.
36. Bates E.R., Califf R.M., Stack R.S. et al. Thrombolysis and Angioplasty in Myocardial Infarction (TAMI-1) trial: influence of infarct location on arterial patency, left ventricular function and mortality. J Am CollCardiol 1989; 1:12–18.
37. Owens C.G., McClelland A.J., Walsh S.J. et al. In-hospital percutaneous coronary intervention improves in-hospital survival in patients with acute inferior myocardial infarction particularly with right ventricular involvement. J InvasiveCardiol 2009; 21: 40–44.