

SOME ASPECTS OF LEFT VENTRICULAR REMODELING IN PATIENTS WITH CHRONIC RENOCARDIAC SYNDROME

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ABSTRACT

Features of left ventricle (LV) remodeling of patients with renal arterial hypertension with chronic renocardiac syndrome were studied. The study involved 107 patients with chronic kidney disease (CKD). Highest frequency of ventricular myocardium remodeling in patients with chronic renal failure are concentric hypertrophy and eccentric hypertrophy of LV observed in 76.6% of patients and there by the beginning of the development of renal failure. Concentric hypertrophy with the most common in patients with CKD III-IV, and eccentric hypertrophy frequency increases with the progression of CKD. At a certain stage in the development of chronic renal failure hypertrophy of LV has adaptive value by increasing the wall thickness of LV. Increased of LV myocardial mass in renal hypertensive patients with renocardiac syndrome most achieved in the initial stage of CKD, and then progressively decreases with the growth of the severity of CKD. Similar changes occur with Interventricular septum thickness, although the thickness of the back wall continues to grow. This demonstrates the progression of left ventricular myocardial muscle changes as strengthening LV dilation and increased frequency of eccentric hypertrophy of myocardium.

Key words: left ventricle remodeling, left ventricle hypertrophy, renocardiac syndrome.

INTRODUCTION

Chronic overload of the left ventricle in hypertension leads to structural and morphological reorganization of the myocardium, which combines the concept of "remodeling", which is characterized by the presence of hypertrophy, dilatation and changes in the geometry of the heart cavities and myocardium as a whole, and ultrastructure of myocardium, which is ultimately remodeling of the myocardium and the integral substrate that determine the occurrence and progression of heart failure [1; 2; 3].

Today, special importance is given to the evaluation of the pathogenetic pathways of heart failure [4; 5; 6]. Violation of LV systolic function, the primacy of which there is no doubt in focal destructive processes in the myocardium occurs in patients with hypertension, usually with long-standing pathological process. However, clinical signs of heart failure in hypertension observed in the early stages of its development. This is due process violation diastolic ventricular relaxation, the appearance of diastolic dysfunction [7; 8; 9; 10; 11]. This requires a differentiated assessment

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of the contribution of the structural and morphological changes of the myocardium inherent remodeling in hypertension in the development of clinical manifestations of heart failure.

AIM

study features of left ventricle remodeling of patients with renal arterial hypertension with chronic renocardiac syndrome.

METHODOLOGY

The study involved 107 patients with chronic kidney disease (CKD), the cause of which chronic glomerulonephritis (75 men and 32 women). Preserved renal function, CKD stage I, was found in 42 patients with CKD stage II - 20, III CKD stages - in 27 and stage IV CKD - in 18 patients. Clinical characteristics of patients are presented in Table 1.

RESULTS AND DISCUSSION

Increased blood pressure was observed in all patients. Average SBP and DBP was greatest in patients with CKD stage IV and was $(188,6 \pm 30,2)$ and $(108,4 \pm 8,8)$ mm Hg accordingly. Hb level was reduced, starting with CKD III. In single portions microalbuminuria (30 to 300 mg / l) was detected in 17 (15.9%) patients, proteinuria 1 g / l - in 58 (54.2% patients), 1 to 3 g / l - in 19 patients

(17.8%) and more than 3.0 g / l - the remaining 6 (12.1%) patients.

frequency of the EH remained almost at the same level, accounting for 33.8%. Significantly reduced

Table 1. Clinical characteristics of patients with chronic renocardiac syndrome by clinical and laboratory parameters

Index	CKD			
	CKD I	CKD II	CKD III	CKD IV
Total, n, %	42 (39,3 %)	20 (18,7 %)	27 (25,2 %)	18 (16,8 %)
M / F	30/12	12/8	23/4	10/8
Sistolic AP	167,0±14,3	187,4±18,2	188,6±30,2	196,0±21,8
Diastolic AP	93,7±8,5	107,1±6,3	108,4±8,8	110,0±8,8
Creatinin	0,062±0,011	0,114±0,029	0,360±0,17	1,08±0,031
Hb, g/l	120,4±12,4	114,3±19,5	100,6±13,7	94,8±18,4
Glomerular filtration velocity, ml/min	102,0±16,5	58,8±8,6	31,2±7,2	18,6±9,7
Reabsorption %	99,1±12,2	84,6±2,6	75,1±1,9	59,4±3,8

The examination is set normal geometry (NG) in 9 (8.4%), concentric remodeling (CR) in 16 (15.0%), concentric hypertrophy (CH) in 50 (46.7%) and eccentric hypertrophy (EH) in 33 (29.9%) patients. Moreover, NG was observed only in patients with CKD and where it was 21.4%. In this same group of patients with CR was observed in 9 (21.4%), CH - in 15 (35.7%) and EH - 10 (26.8%) patients. Among patients with the presence of chronic renal failure, CKD stages II-IV, found the CR in 7 (10.8%), CH in 35 (53.8%), EH in 23 (35.4%) patients, and NG was absent (Table 2).

incidence of CR to 7.4% (p <0.01) compared with patients with CH and EH. In patients with CKD IV - CH was observed in 55.6%, which was lower than the frequency of unreliable CH with CKD III. The increased frequency of EH to 44.4%, which also did not differ significantly from both the CH in patients with CKD IV, and EH in patients with CKD III. Thus, the highest frequency of left ventricular myocardial remodeling in patients with chronic renal insufficiency up CH and EH - LV observed in 76.6% of patients and there by the beginning of kidney failure. CH with the most common in

Table 2 : The frequency and nature of remodeling depending on the stage of CKD

LV Remodeling	CKD I	CKD II	CKD III	CKD IV	Total
n	42	20	27	18	107
NG	9 (21,4 %)	-	-	-	9 (8,4 %)
CR	9 (21,4 %)	5 (25,0 %)	2 (7,4 %)	-	16(15 %)
CH	15 (35,7 %)	19 (45,0 %)	16 (59,4 %)	10	50 (46,7 %)
EH	10 (26,8 %)	6 (30,0 %)	9 (33,8 %)	55,6 % 8	33 (29,9 %)
LV Hypertrophy	8 (19,1) 34 (80,9)	4 (20 %) 16 (80 %)	1 (3,7 %) 26/96,3%	18 100 %	13(12,1%) 94(87,9%)
Total	42 100 %	20 100 %	27 100 %	18 100 %	107 100 %

In CKD II in 5 (25%) patients found the CR and pronounced myocardial hypertrophy was observed in 16 patients (80%). The frequency of CH and EH ventricular myocardium was observed in 45.0% and 30.0%, respectively.

Progression of CKD to stage III was characterized by an increase in the number of patients with CH to 59.4% (p <0.05), while the

patients with CKD III-IV, and the frequency of EH increases with the progression of CKD.

Changing of structural and functional parameters of LV myocardium depending on the stage of CKD manifested an increase in end-systolic volume (ESV) and end-diastolic volume (EDV) of LV (Table 3).

Table 3 : Structural and functional parameters of LV myocardium of patients with chronic renocardiac syndrome

Index	CKD				
	CKD I	CKD II	CKD III	CKD IV	Контроль
N	42	20	27	18	24
Left Atrium, sm	3,0 ± 0,64	3,1 ± 0,64	3,5 ± 0,35	3,5 ± 0,36	3,0 ± 0,80
Right Ventricle, sm	2,65 ± 0,87	2,1 ± 0,64	2,1 ± 0,45	2,9 ± 0,58	1,5 ± 0,54
EDV, sm ³	124,8 ± 20,4	160,8 ± 25,6*	143,1 ± 20,8*	134,3 ± 22,6*	124,2 ± 28,1
ESV, sm ³	50,2 ± 10,7	74,0 ± 14,6**	64,8 ± 16,8**	64,9 ± 14,3**	48,4 ± 9,61
Interventricular septum thickness, sm	1,2 ± 0,38	1,4 ± 0,47*	1,4 ± 0,45*	1,3 ± 0,18**	0,9 ± 0,04
Thickness of the LV posterior wall, sm	1,04 ± 0,12	1,2 ± 0,23**	1,4 ± 0,26*	1,3 ± 0,18*	1,02 ± 0,03
LV relative wall thickness, sm	0,38 ± 0,13	0,38 ± 0,61**	0,49 ± 0,61*	0,42 ± 0,06*	0,35 ± 0,04
Index of LV myocardial mass, g/m ²	102,4 ± 24,8*	149,8 ± 21,6*	140,8 ± 32,4*	120,8 ± 20,6*	84,8 ± 19,6

*p < 0,01, **p < 0,05 – reliability values of differences compared with the control group

Compared to the performance of the control group in the absence of chronic renal failure was observed only slight, at 3.7%, an increase of ESV. In patients with CKD, ESV was increased compared to controls by 34.6% (p < 0.01) and EDV by 22.8% (p < 0.05). CKD stage III was characterized by an increase in ESV compared to the control by 25.3% (p < 0.01) and EDV by 13.2% (p < 0.05). In CKD stage IV increase compared to control ESV was 25.4% (p < 0.01), and an increase in EDV - only 7.5% (p > 0.05).

When comparing changes in ESV and EDV regardless of the presence of chronic renal failure the most significant increase in these parameters was observed in patients with CKD stage II. With the progression of CKD decreasing as ESV and EDV.

Changes in left ventricular myocardium, namely interventricular septum thickness, thickness of the LV posterior wall and LV relative wall thickness, depending on the presence of chronic renal failure manifested by progressive thickening of the LV posterior wall and some decrease thickness of the LV posterior wall in patients with CKD stage IV. thickness of the LV posterior wall in patients with CKD and was the same with the following indicators control group, whereas in CKD II it was increased by 15.0% (p < 0.05), with CKD III - 27.1% (p < 0.01) in CKD IV by 21.5% (p < 0.01).

Interventricular septum thickness in patients with CKD stages II and III exceed the performance of the control group by 35.7% (p < 0.01) in CKD IV - 30.8% (p < 0.05). Index of thickness of the LV posterior wall was slightly increased in patients with CKD III and IV.

These changes in the thickness of the left ventricular myocardium reflected in the progressive increase LV relative wall thickness (increased only by 7.9% (p < 0.05) in I and II stages CKD). A significant increase in performance LV relative wall thickness observed only in patients with CKD stages III and IV in 28.6% and 16.7% (p < 0.01), respectively.

The most significant changes in the structural and morphological parameters related to myocardial interventricular septum thickness that were significantly increased in all patients with hypertension, as the presence of chronic renal failure and those without. Interventricular septum thickness exceed the performance of the control group in patients with CKD and 17.2% in CKD II - 43.4%, with CKD III - 39.8% in CKD IV - 29.8% (p < 0, 01).

Thus in patients with CKD stage and Interventricular septum thickness moderately correlated with the level of systolic blood pressure (BP) (r = 0.38; p < 0.05).

Moderate positive correlation was observed with systolic BP and diastolic BP in patients with CKD II ($r = 0.38$ and 0.36 , respectively, $p < 0.05$), III CKD ($r = 0.39$ and 0.35 , $p < 0.05$) and CKD IV ($r = 0.48$ and 0.35 , respectively, $p < 0.05$). In addition, since the stage III CKD, there was a positive correlation between LV relative wall thickness and systolic BP and diastolic BP ($r = 0.32$ and 0.34 , respectively).

Also found a strong negative correlation between Index of LV myocardial mass and LV relative wall thickness on the one hand, and the level of Hb, on the other hand, in patients with CKD III ($r = -0.77$ and $r = -0.72$; $p < 0.05$) and in CKD IV ($r = -0.38$ and $r = -0.35$; $p < 0.05$, respectively).

Analyzing changes in general Index of LV myocardial mass in CKD patients regardless of the presence of chronic renal failure, it should be noted that the maximum rate was Index of LV myocardial mass in CKD patients II, where it exceeded not only the level of the control group by 43.4% ($p < 0.01$) but levels in patients with CKD and 31.6% ($p < 0.01$), CKD III by 6.0% ($p > 0.05$) and CKD IV by 19.4% ($p < 0.05$). Thus, the increase in LV myocardial mass (MM) most accomplished in the initial stage of CKD, and then progressively decreases with the growth of the severity of CKD. Similar changes occur with the thickness of the IBE, although the thickness of the posterior wall continues to grow. This demonstrates the progression of left ventricular myocardial muscle changes as strengthening LV dilation and increased frequency of EH.

Dysfunction of nephrons in renal disease, decreased sodium filtration and increasing its reabsorption are one of the highlights of hypertension in CKD as a major factor in myocardial remodeling. Increased sensitivity of smooth muscle cells of the vascular wall to the pressor effects of humoral factors and reduce the impact of substance vasodilatory and sodium retention, leading to the development of hypervolemia. In hypertensive chronic heart overload pressure and volume results in the development of structural changes in it, one of which is ventricular myocardial hypertrophy of the heart. At a certain stage in the development of chronic renal failure LVH has adaptive value by increasing left ventricular wall thickness and maintaining adequate opportunities to develop intraventricular pressure in systole.

The progressive increase in left ventricular mass, reduced coronary reserve as a result of non-compliance with the blood supply to myocardial

ventricular overload and chronic hypoxia alter the electrophysiological properties of the myocardium and influencing mechanism of contraction / relaxation of cardiomyocytes. This increases the amount of collagen matrix in myocardium, enhanced process of apoptosis. This leads to a further stage of LV remodeling, in which a set of changes in its shape and functioning occur in response to hemodynamic conditions and pathological processes in the myocardium. Morphological substrate of these changes is the LV cavity dilatation, and remodeling hypertrophic myocardial cells increases the number of lysosomal structures. High lysosomal and phagocytic activity of leukocytes observed in the myocardium, indicating the start of the process of cytoplasmic degeneration running programmed autolysis. Chronic self-destructive proteolysis in affected cardiocytes and apoptotic morphological changes in nuclei and cytoplasm may lead to the death of a controlled cardiocytes of hypertrophied myocardium.

CONCLUSION

1. Highest frequency of ventricular myocardium remodeling in patients with chronic renal failure are concentric hypertrophy and eccentric hypertrophy of LV observed in 76.6% of patients and there by the beginning of the development of renal failure. Concentric hypertrophy with the most common in patients with CKD III-IV, and eccentric hypertrophy frequency increases with the progression of CKD. At a certain stage in the development of chronic renal failure hypertrophy of LV has adaptive value by increasing the wall thickness of LV.
2. Increased of LV myocardial mass in renal hypertensive patients with renocardiac syndrome most achieved in the initial stage of CKD, and then progressively decreases with the growth of the severity of CKD. Similar changes occur with Interventricular septum thickness, although the thickness of the back wall continues to grow. This demonstrates the progression of left ventricular myocardial muscle changes as strengthening LV dilation and increased frequency of eccentric hypertrophy of myocardium.

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