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INCIDENCE AND SIGNIFICANCE OF DIASTOLIC CARDIAC FAILURE OF THE LEFT VENTRICLE IN PATIENTS WITH COR PULMONALE CHRONICUM

SUMMARY

The problem of diastolic cardiac failure has been drawing attention in the past 12 years and since then it has been considered a unique clinical entity. It has been proved that the right ventricle (RV) in arterial hypertension (AHT) has impaired diastolic function due to abnormal diastolic function of the left ventricle (LV). Nowadays, diagnosis of diastolic dysfunction is easily obtained by Doppler echocardiography.

The aim of this paper was to evaluate diastolic dysfunction of the left ventricle in patients (pts) with cor pulmonale chronicum (CPC). Fifty patients with cor pulmonale chronicum were involved in group I; fifty-six patients with chronic obstructive lung disease (COLD) without cardiovascular problems were involved in group II-the control group. In all patients, 2D echocardiography and Doppler studies were performed.

Our results suggest that the presence of diastolic dysfunction of the left ventricle in patients with cor pulmonale is a positive proof of aggravation of already present pulmonal failure and pulmonary hypertension and, as a consequence, RV is compromised.

Keywords: **cor pulmonale chronicum, left ventricle, cardiac failure, diastolic failure**

INTRODUCTION

According to definition, cor pulmonale is hypertrophy and dilatation of the right ventricle of the heart, or only dilatation of the right ventricle of the heart, caused by the diseases that obstruct structure and function of the lungs, or function of the lungs only, except in case when changes in the lungs occur as a consequence of the left side heart impairment.

Increased cardiac output (the minute volume – MV) together with myocard hypoxia, including other disorders which occur during cor pulmonale, affect the myocard of the left (LV) and right ventricle (RV), so, to some extent, in cor pulmonale chronicum, the left side heart impairment can occur as well. Autopsy studies of the patients with cor pulmonale (CP) give proof for this. In such patients, hypertrophy and dilatation of both ventricles, significantly higher in RV, were observed.

Due to pulmonary hypertension, hypertrophy of RV causes early diastolic changes of the geometry of the LV, which are presented by lengthening of the isovolumetric relaxation time (IVRT) and by early diastolic filling reduction.

As for dynamic factors, coronary blood flow and myocard perfusion are extremely significant for diastolic function. Myocard hypoxemia and hypoxia development in respiratory failure and CP cause lengthening myocard relaxation time both of LV and RV.

LV diastolic function abnormalities occur when the heart is unable to use preload reserve due to high cardiac filling pressures and when the ventricle is unable to adequately take blood in diastola.

Increased LV filling pressure is shifted to pulmonary veins and capillaries and malfunction in the lungs occur. In patients with CP, along with having pre-capillary pulmonary hypertension, there is also the so-called post-capillary pulmonary hypertension. There is a repercussion of overall hemodynamic situation.

It has been proved that one third of the patients with congestive cardiac failure (CF) have diastolic dysfunction of the LV, while systolic function is preserved. One third of the patients with CF have LV systolic function disorder, and the last third of the patients have both diastolic and systolic CF. Primary diastolic CF of the left ventricle excludes the patients with ventricle systolic function disorder.

Diastolic CF diagnosis is made according to the following criteria (1-4):

• Congestive CF symptoms and signs present (mostly pulmonary congestion manifestation)

• Normal function of the systolic function of the LV present (EF should be about 50%, but not less than 45%), diastolic diameter index LVDI made by ultrasound less than 3,2 cm/m^2, and endidiastolic diameter index EDVI less than 102ml/m^2.

• LV abnormal diastolic function signs present (mostly echocardiogram parameters used), slow isovolumetric relaxation and slow LV filling in early diastola and reduced or only reduced disensibility of LV.

AIMS

The aim of the study was to estimate the presence and some characteristics of diastolic CF of the LV in patients with acute pulmonary failure and cor pulmonale chronicum (CPC).

MATERIAL AND METHODS

This study included 106 patients hospitalized at the Clinic for Cardiovascular Diseases of the Clinical Center in Nis. Fifty patients having CPC were examined. They underwent the analyses twice – on admission to hospital with the diagnosis of CPC and compensated HPS and just before discharge, when chronical respiratory failure compensation was achieved, without any signs of congestive right side heart failure.

The control group consisted of 56 patients with normal respiratory values of arterial blood gases. They were taken to the Clinic by Emergency Health Service, mostly in the afternoon or at night, because of bronchoopstructive lung disease aggravation, having doubts that acute lung oedema is in question. Such patients were kept in hospital only for a short period, and after obligatory examinations and after making proper diagnosis, they were referred to some pulmonary departments. So, the control group consisted of patients without manifested cardiovascular disease.

A. Left ventricle examination

In all patients, heart morphological changes were assessed by imaging of the heart with echocardiography on device Toshiba SSH–140A, according to ASA recommendations. LV mass was counted after Penn convention, using the formula of Devereaux and Reichek.

LV mass in grams = 1.04 [(LVID + VST + PWT) ^{3} – (LVID) ^{3}] – 13.6

Mass index LV (Lv mass g/m^2) was counted as a quotient of the LV mass and body surface. We considered referential values of the mass of LV less than 134g/m^2 for men and less than 110g/m^2 for women.

B. Diastolic function examination

LV filling echocardiography was done by use of combined Doppler pulse wave technique (5) along with echocardiography image of the anterior tract of the LV derived from two dimensional echocardiography, using probe apical position. Imaging was done at the end of expirium to avoid respi-ration influence to speed blood flow. The following parameters of LV diastolic function were measured:

• Maximum early phase ventricle filling speed (E)

• Maximum atrium phase filling speed (A)

• Doppler index (E/A)

• Acceleration and deceleration time of E and A waves

All the given parameters used for the left ventricle diastolic function assessment are also used for RV diastolic function assessment. The dynamic of the diastolic filling of the RV is closely related to the filling dynamics of the LV and is independent of the LV mass and of blood pressure. In arterial hypertension, E/A relation in both ventricles is significantly reduced and there is not an important decrease of this relation between the ventricles.

RESULTS

According to morphological parameters of LV, all patients in group II were divided into 5 subgroups, as shown in *Table 1*. Group I (control group) had no cardiovascular diseases and they had normal LV morphology.

The number of patients in group I and group II with five subgroups, as well as their percentage, is

Table 1. Morphological characteristics of LV in patients in group I and group II with subgroups

EXAMINED PATIENTS *LV MORPHLOGICAL CHARACTERISTICS*

Groups	Subgroups	N=106	%	LV morphological characteristics
I	———	56	52.8	No cardiovascular disease
II	I	2	4.0	No left ventricle hypertrophy (LVH)
	II	37	74.0	Left ventricle septum hypertrophy (LVSH)
	III	3	6	Concentric left ventricle hypertrophy (LVCH)
	IV	5	10.0	Eccentric non-dilatation hypertrophy of the left ventricle (LVEH)
	V	3	6.0	Eccentric dilatation hypertrophy of the left ventricle (LVEDH)

shown in the *Table 1*.

LV mass index values were the lowest in control group (I) and the highest in the subgroups IV and V.

As for our patients, we found statistically significant difference in LV mass index between patients in control group (I) and patients in subgroups II, III, IV and V ($p<0.001$).

Mean values of diastolic dysfunction parameters are shown in *Table 2*.

Early diastolic filling values (E) of the control group (I) were significantly higher than the values of the same parameters in subgroups II, III, IV and V ($p< 0.0001$).

Also, there was no statistically significant difference between LV mass and maximum flow speed, nor between the LV mass index and maximum flow speed.

Acceleration time of maximum speed of early filling – E waves, was least in the subgroup V

Table 2. Mean values of the ventricle diastolic function parameters in patients of the control group (I) and subgroups of group II

PARAMETERS	Group I	Subgroups of group II					
	I	I	II	III	IV	V	P
Emax(cm/s)	51.98	44.92	39.89	38.1	38.14	42.93	<0.0001
EAC(ms)	103.1	106.9	101.09	100.98	108.13	91.3	<0.05
EDC(ms)	169	185	196	192.01	204	157	<0.05
Amax(cm/s)	45.01	50.00	51.00	50.35	50.92	48.03	<0.0001
AAC(ms)	82.02	82.0	81.3	85.5	85.06	68.92	<0.0001
ADC(ms)	81.9	78.01	79.1	81.99	81.97	63.9	<0.0001
E/A	1.01	0.89	0.81	0.72	0.71	0.91	<0.001

E max-early diastolic filling; EAC – acceleration time of E;
EDC – deceleration time of E; A max – late diastolic filling;
AAC – acceleration time of A; deceleration time of A;
E/A – ratio between early and late diastolic filling;

and was significantly different from the patients in group I and subgroup I , and it also closely correlated with lower values of partial pressure O_2 in blood (p O_2 < 40 mmhg and p CO^2 > 70 mmHg).

Deceleration time of maximum speed of early filling - E waves, was longest in patients in the subgroup IV and is statistically significantly different from the patients in the control group (I), p<0.001.

The given parameter is also closely related to the lowest values of arterial blood gases (p O^2<30 mmHg).

Mean values of maximum speed of late diastolic filling (Amax) were significantly higher in patients of the II, III, IV and V subgroup than in the control group. There was no correlation between LVM and Amax, nor between LVMI and Amax, but there was a close correlation in patients with serious hypoxemia (partial pressure of oxygen in arterial blood under 40 mmHg).

Doppler index (E/A relation) in the control group was within normal range, while it was progressively decreasing from patients in subgroup I to IV and showed variability from patient to patient, depending on the gas status of arterial blood.

DISCUSSION

Diastolic relaxation and filling abnormalities were examined in patients with coronary disease (myocardial ischemia). After cardiological procedures – Percutaneous Transluminal Coronary Angioplasty (PTCA) and aortal coronary bypass – there have been some improvements in these patients. Myocardial ischemia and hypoxia lead to diastolic dysfunction at cell level through calcium ion unity abnormalities. After cell contraction, reestablishment of the low concentration of calcium in the cytosol depends on calcium sequestration in sarkoplasmatic reticulum, on sarcoplasmatic reticulum Ca-ATPase, an energy demanding process. This process is impaired by myocard hypoxia and occurs even before inhibition of the energy demanding processes responsible for contraction. It has been suggested that hypoxemia, having myocardial hypoxia as a consequence, probably damages normal ventricles relaxation by abnormal calcium transportation.

Despite numerous investigations so far, there have not been enough clinical studies dealing properly with the LV diastolic heart failure problem in patients with right side heart failure syndrome within CPC entity. Therefore, dysfunction of the RV in acute state of chronic pulmonary failure, chronic pulmonary hypertension and CP still requires further investigations.

In arterial hypertension, the tension rise in the right half of the heart, systolic function disorder and RV hypertrophy have been proved. The RV in hypertension also has diastolic function disorder, and these abnormalities are closely related to the LV function disorder (6,7). Diastolic dysfunction of the RV in arterial hypertension can be explained by the following mechanisms:

- Joint role of intraventricular septum
- Cardiac muscle tissue distribution and construction common to both ventricles
- Growth factors that stimulate myocites of the LV to hypertrophy and stimulate the RV myocites as well, so RV hypertrophy occurs, which explains RV diastolic dysfunction.

In similar way, LV diastolic heart failure in patients with CPC with pulmonary hypertension as pathogenesis can be explained.

In our study, there were no positive correlations between LV mass and diastolic parameters. The authors of the Framingham study got the similar results. However, Ren et al. found that mass index ILV reversely correlates with maximum diastolic filling (r = 0, 89).

Among numerous causes of chronic respiratory failure LV weakness can be one of them. LV failure causes anatomic and functional abnormalities of the lungs which obstruct gases exchange and lead to aggravation of respiratory gases in arterial blood. The most significant example is acute cardiogen pulmonary edema, when acute respiration failure occurs, as well as death threat due to severe hypoxemia. The less LV dysfunctions present, the less blood gases changes occur, but added to already existing ones in patients with chronic respiratory failure, they can cause a condition, according to arterial blood findings, usually qualified as respiration decompensata, or acute aggravation of the chronic respiration failure. The purpose of this information was to point out to some mechanisms and possible therapeutic treatment of this, still unsatisfactorily explained condition.

CONCLUSION

Diastolic dysfunction of the LV myocardium in patients with CPC is a primary weakness of the left ventricle, which is clinically asymptomatic and unrecognized for a long time. Because of that, it is necessary to do minucious cardiac and echocardiograph examination of all the patients with acute chronic respiratory failure and decompensated CPC, to find out LV diastolic dysfunction presence, which can be detected by ultrasound heart examination, so proper therapies can be applied to prevent aggravation of already aggravated pulmonary failure by primary pulmonary or non- pulmonary disease.

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ZASTUPLJENOST I ZNAČAJ DIJASTOLNE SRČANE INSUFICIJENCIJE LEVE KOMORE KOD BOLESNIKA SA HRONIČNIM PLUĆNIM SRCEM

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SAŽETAK

Problem dijastolne srčane insuficijencije privlači pažnju u poslednjih 12 godina i od tada je izdvojen kao poseban klinički entitet. Dokazano je da desna komora u arterijskoj hipertenziji ima poremećenu dijastolnu funkciju, što je u uskoj vezi sa abnormalnom dijastolnom funkcijom leve komore. Danas se dijagnoza dijastolne disfunkcije može lako postaviti zahvaljujući mogućnostima koje pruža Doppler ehokardiografija.

Cilj rada bio je da se prouči pojava dijastolne disfunkcije leve komore kod naših pacijenata sa hroničnim plućnim srcem. Prospektivnom analizom je obuhvaćeno 50 bolesnika sa hroničnim plućnim srcem – grupa II - i 56 bolesnika sa hroničnom plućnom opstruktivnom bolešću – dakle, bolesnici bez postojanja manifestnog kardiovaskularnog oboljenja - grupa I – kontrolna grupa. Kod svih bolesnika urađen je 2D i Doppler ehokardiografski pregled.

Naši rezultati ukazuju da je nalaz postojanja dijastolne disfunkcije kod bolesnika sa hroničnim plućnim srcem siguran dokaz pogoršanja već postojeće plućne insuficijencije i plućne hipertenzije, koji posledično kompromituju desnu komoru i sigurno deklanširaju njenu funkciju.

***Ključne reči*: hronično plućno srce, leva komora, srčana insuficijencija, dijastolna srčana insuficijencija leve komore**