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ECHOCARDIOGRAPHIC ASSESSMENT OF LEFT VENTRICULAR HYPERTROPHY IN PATIENTS WITH END-STAGE RENAL FAILURE

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EHOKARDIOGRAFSKA PROCENA HIPERTROFIJE LEVE KOMORE KOD BOLESNIKA SA ZAVRŠNIM STADIJUMOM HRONIČNE SLABOSTI BUBREGA

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ABSTRACT

Cardiovascular diseases are the most frequent cause of morbidity and mortality in patients with end-stage renal failure (endogenous creatinine clearance < 10 ml/min). Pressure and/or volume overload are the primary causes in development of left ventricular hypertrophy. Left ventricular hypertrophy (concentric, eccentric) leads to the disturbance of diastolic and systolic functions, which precede the development of congestive heart failure. The aim of the paper was to investigate the type and frequency of left ventricular hypertrophy in patients with end-stage renal failure before hemodialysis treatment started. The study included 82 patients (male: female 53:29) with end-stage renal failure, with mean age of 58.17 ± 14.19 years. Investigated parameters of left ventricular hypertrophy evaluation were the following: relative wall thickness-RWT, left ventricular mass index-LVMi, the index of left ventricular end-diastolic volume-iLVEDV. In evaluation of the systolic function we investigated fractional shortening-FSLK and left ventricular ejection fraction-EFLK. Mean RWT was 42.25 ± 8.53%, LVMi was 142.85 ± 38.14 g/m^2, 101.82 ± 40.62 mL/m^2, FSLK 32.88 ± 7.83%, and EFLK 68.51 ± 11.75%. Left ventricular hypertrophy was present in 59 (71.96%) of the patients, left ventricular dilatation in 10 (12.19%), and 13 (15.85%) of the patients had regular echocardiographic findings. The most frequent form of left ventricular hypertrophy (LVH) was eccentric LVH found in 30 (36.59%) patients, while concentric LVH was present in 29 (35.37%) patients. Systolic function disturbance of the left ventricle occurred in 7 (8.54%) patients, while 75 (91.46%) patients had normal left ventricular systolic function. Identification of patients with left ventricular hypertrophy and application of appropriate therapy lead to regression of left ventricular hypertrophy and reduction in cardiovascular morbidity and mortality in patients with end-stage renal failure.

Key words: end-stage renal failure, echocardiography, left ventricular hypertrophy

SAŽETAK

Kardiovaskularne bolesti su najčešći uzrok morbiditeta i mortaliteta kod bolesnika sa završnim stadijumom hronične slabosti bubrega (klirens endogenog kreatinina < 10 ml/min). Opterećenje pritiskom i/ili opterećenje volumenom su primarni uzroci razvoja hipertrofije leve komore. Hipertrofija leve komore (koncentrična, ekscentrična) ima za posledicu poremećaj dijastolne i sistolne funkcije, koji prethode razvoju kongestivne slabosti srca. Rad je imao za cilj da ispita vrstu i učestalost hipertrofije leve komore kod bolesnika sa završnim stadijumom hronične slabosti bubrega pre započinjanja lečenja hemodijalizom. U radu je ispitano 82 bolesnika (M:Ž 53:29) sa završnim stadijumom hronične slabosti bubrega, prosečne starosti 58,17 ± 14,19 godina. Kao parametri za procenu hipertrofije leve komore ispitivani su: relativna debljina zida-RWT, indeks mase leve komore-LVMi, indeks end-dijastolnog volumena leve komore-iEDVLK. Za procenu sistolne funkcije ispitivani su frakciono skraćenje-FSLK i ejekciona frakcija leve komore-EFLK. Prosečna vrednost RWT iznosila je 42,25 ± 8,53%, LVMi 142,85 ± 38,14 g/m^2, iEDVLK 101,82 ± 40,62 mL/m^2, FSLK 32,88 ± 7,83%, a EFLK 68,51 ± 11,75%. Hipertrofija leve komore prisutna je kod 59 bolesnika (71,96%), dilatacija leve komore kod 10 bolesnika (12,19%), a 13 bolesnika (15,85%) je imalo uredan ehokardiografski nalaz. Najčešći oblik hipertrofije leve komore jeste ekscentrična HLK, prisutna je kod 30 bolesnika (36,59%), dok je koncentrična HLK bila prisutna kod 29 bolesnika (35,37%). Poremećaj sistolne funkcije leve komore prisutan je kod 7 bolesnika (8,54%), dok 75 bolesnika (91,46%) ima normalnu sistolnu funkciju LK. Izdvajanje bolesnika sa hipertrofijom leve komore i primena odgovarajuće terapije dovode do regresije hipertrofije leve komore i do smanjenja kardiovaskularnog morbiditeta i mortaliteta bolesnika sa završnim stadijumom hronične slabosti bubrega.

Ključne reči: hronična slabost bubrega, ehokardiografija, hipertrofija leve komore

INTRODUCTION

Left ventricular hypertrophy is the leading risk factor for development of cardiovascular morbidity and mortality in patients with end-stage renal failure (endogenous creatinine clearance < 10 ml/min) (1, 2). Other risk factors for left ventricular hypertrophy in those patients are anemia, hypertension, extracellular fluid volume increase, blood flow through arterial-venous fistula, internal uraemic milieu (oxidative stress, microinflammation, hyperhomocysteinemia) and disturbed calcium and phosphate homeostasis (2–6).

Left ventricle pressure load (hypertension, aortic stenosis and blood vessel atherosclerosis) leads to the increased stress of left ventricular wall in systole, which initiates the process of its remodeling: parallel alignment of new sarcomeres, thickening of left ventricle wall without increase in the ventricular diameter (increased ratio between wall thickness and left ventricle diameter - h/r > 0.45 – concentric hypertrophy of the left ventricle) (7–10).

Volume overloads the left ventricle due to increased water and salt uptake, anemia and increased blood flow through arterial-venous fistula (Q_{AV} ≥ 1000 ml/min), that accompanies alignment of new sarcomeres first in line and then parallel. The consequence is an increase in wall thickness and left ventricle diameter (h/r < 0,45 - eccentric hypertrophy of the left ventricle) (7–10).

Left ventricular hypertrophy goes through two stages (Figure 1). In adaptation stage, left ventricular hypertrophy shows protective action, and occurs in response to the increased tension stress of left ventricular wall. When volume and pressure overload the left ventricle chronically and without control, adaptive hypertrophy turns into maladaptive hypertrophy of the left ventricle where myocytes are lost, systolic function is disturbed,

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and heart insufficiency and lethal outcome are developed (7–11).

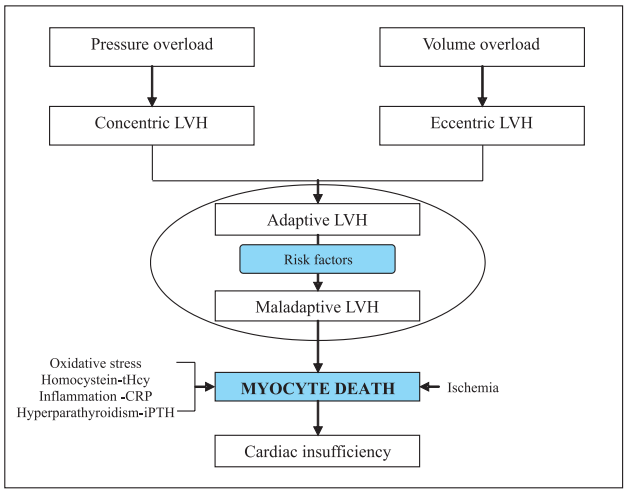

Figure 1. Pathogenesis of left ventricular hypertrophy in patients with end-stage renal failure.

Disturbances of morphology and left ventricular function in hemodialysis patients are manifested as systolic dysfunction, concentric left ventricular hypertrophy, eccentric left ventricular hypertrophy, or left ventricular dilatation.

Left ventricular systolic function is disturbed if echocardiographic examination shows left ventricular fraction shortening - FSLK ≤ 25% and left ventricular ejection fraction - EFLK ≤ 50% (12–15). Concentric left ventricular hypertrophy is defined as thickness of myocardial interventricular septum of the left ventricle in diastole > 11 mm, thickness of the posterior left ventricle wall in diastole > 11 mm, internal diameter of the left ventricle in diastole < 4.7 cm, increased left ventricular mass index, with normal fraction shortening and relative wall thickness of the left ventricle - RWT > 45% (12–15). Eccentric left ventricular hypertrophy is characterized by thickening of the left ventricle wall, increased internal diameter of the left ventricle (internal LV diameter at the end of diastole > 57 mm), normal left ventricular fraction shortening and relative wall thickness of the left ventricle - RWT ≤ 45% (12–15). In dilatation of the left ventricle, internal diameter of LV at the end of diastole is > 57 mm, LV volume > 90 mL/m^2, and the systolic function and left ventricular mass index are normal (12–15).

Identification of patients with an increased risk of development of left ventricular hypertrophy and application of appropriate therapy to achieve target values of risk factors, lead to regression of left ventricular hypertrophy, reduced rates of cardiovascular morbidity and mortality, and improvement in quality of life in patients with end-stage renal failure that start treatment with regular hemodialyses (16–20).

The investigation purpose was to determine the type and frequency of disturbances in morphology and systolic function of the left ventricle in patients with end-stage renal failure before initiation of hemodialysis treatment.

MATERIAL AND METHODS

The study included 82 patients from the Department of Nephrology, Clinic of Urology and Nephrology, Clinical Center „Kragujevac“, Kragujevac, respecting the Helsinki Declaration on medical research, and after the patients had given their consent for participation.

The study included patients with end-stage renal failure (endogenous creatinine clearance < 10 ml/min) before initiation of hemodialysis treatment, with various primary renal diseases.

Basic observation characteristics were relative wall thickness of the left ventricle, left ventricular mass index, the index of left ventricular end-diastolic volume, left ventricular fraction shortening, and left ventricular ejection fraction.

All patients had echocardiographic examination on SHIMADZU 2200 machine, with 2.5 MHz probe, before hemodialysis treatment was initiated.

Left ventricular hypertrophy was determined by measurement of left ventricular mass index - LVMi:

$$\text{iEDV} = \frac{(EDDLK)^3 x 0,001047}{TP} \quad \text{g/m}^2,$$

where: IVSd - thickness of interventricular septum in diastole (mm), ZZLKd - thickness of the posterior wall in diastole (mm), TP - body surface area (m^2). Normal left ventricular mass index is ≤ 131 g/m^2 in men and ≤ 100 g/m^2 in women.

Left ventricular volume is calculated using the following equation:

$$\text{iEDV} = \frac{(EDDLK)^3 x 0,001047}{TP} \quad \text{ml/m}^2,$$

where: EDDLK - end-diameter of the left ventricle (mm), a TP – body surface area (m^2). Normal index of left ventricular end-diastolic volume is ≤ 90 ml/m2.

Relative wall thickness - RWT was determined using the equation:

RWT = [(2 x ZZLKd)/EDDLK] x 100%,

where: ZZLKd represents the posterior wall thickness in diastole (mm), and EDDLK is end-diastolic diameter of the left ventricle (mm).

Left ventricular fraction shortening (FS %), as an indicator of the systolic function, was calculated as:

$$\text{FS} = \frac{(EDDLK - ESDLK)}{EDDLK} x100 \ \%,$$

where: EDDLK is left ventricular end-diastolic diameter (mm), ESDLK is end-systolic diameter of the left ventricle (mm). Normal fraction shortening of the left ventricle is 42 ± 8%.

Ejection fraction of the left ventricle (EF %), as an indicator of the systolic function, was calculated using the formula:

$$EF=\frac{(EDVLK-ESVLK)}{EDVLK}x100\ \%,$$

where: EDVLK is left ventricular end-diastolic volume (ml), ESVLK - end-systolic volume of the left ventricle (ml). Normal ejection fraction of the left ventricle is 67 ± 9%.

RESULTS

The prospective study, whose results are shown in figure 2 and 3, was performed at the Clinic of Urology and Nephrology, CC „Kragujevac", in Kragujevac.

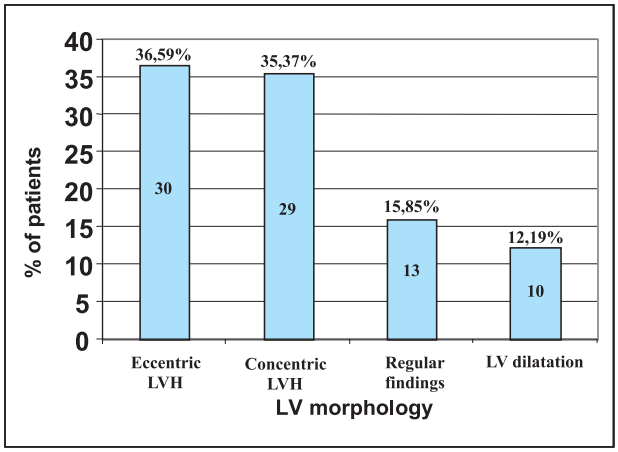

Figure 2. Distribution of patients according to echocardiographic findings.

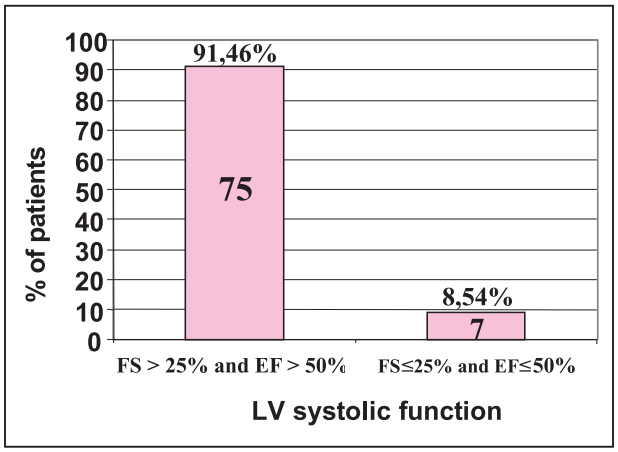

Figure 3. Distribution of patients according to systolic function of left ventricle.

The study included 82 patients (male: female 53: 29) with end-stage renal failure, who started hemodialysis treatment in 2003, 2004 and 2005, and whose mean age was 58.17 ± 14.19 years. Mean value for thickness of the interventricular septum in diastole- IVSd was 11,31 ± 1,84 mm, the posterior wall thickness in diastole-ZZLKd 11,34 ± 1,70 mm, relative wall thickness of the left ventricle-RWT was 42,25 ± 8,53%, left ventricular mass index-LVMi 142,85 ± 38,14 g/m^2, the index of end-diastolic volume-iEDVLK 101,82 ± 40,62 mL/m^2, left ventricular fraction shortening-FSLK 32,88 ± 7,83%, and left ventricular ejection fraction-EFLK 68,51 ± 11,75%.

Left ventricular hypertrophy was found in 59 (71.96%) patients, dilatation of the left ventricle in 10 (12.19%) patients, and 13 (15.85%) patients had regular echocardiographic findings (Figure 2).

The most frequent form of left ventricular hypertrophy was eccentric LVH, which was found in 30 (36.59%) patients, while concentric LVH was identified in 29 (35.37%) patients.

Disturbance of the left ventricular systolic function occurred in 7 (8.54%) patients, while 75 (91.46%) patients had a normal systolic function of the left ventricle (Figure 3).

DISCUSSION

Cardiovascular diseases are the most frequent cause of morbidity and mortality in patients with end-stage renal failure (1). In patients with end-stage renal failure, among all cardiovascular complications, the greatest prevalence was found for left ventricular hypertrophy, ischemic heart disease and congestive heart failure (21, 22). Concentric left ventricular hypertrophy primarily depends on left ventricular pressure overload and atherosclerosis. Volume overload (increased water and salt uptake), anemia and increased blood flow through A-V fistula (Q_{AV}> 1000 ml/min) lead to development of eccentric left ventricular hypertrophy (23).

In patients with chronic renal insufficiency, the prevalence of left ventricular hypertrophy is approximately 40% and increases to 75% in patients with end-stage renal failure (1, 2). Among our patients with end-stage renal failure the prevalence of left ventricular hypertrophy was 71.96%, which is in keeping with published results of other trials. Concentric left ventricular hypertrophy was diagnosed in 35.37% and eccentric left ventricular hypertrophy in 36.50% of the investigated patients. Only 15.85% of the patients with end-stage renal failure, that were about to start hemodialysis treatment, had normal echocardiographic findings.

Left ventricular hypertrophy is an independent risk factor for development of arrhythmias, sudden heart death, heart failure and ischemic heart disease (14). Concentric left ventricular hypertrophy leads to disturbances of heart diastolic function, whose consequences could be lung edema and development of hypotension during hemodialysis (14, 24). Disturbance of the systolic function is frequently associated with dilatative cardiomyopathy (small ejection fraction + increased internal diameter of the left ventricle in diastole), accompanied by development of congestive heart failure (25).

The main risk factors for development of left ventricular hypertrophy in patients with end-stage renal failure are susceptible to changes, and achievement and maintenance of desired values of risk factors on long-term basis lead to regression of left ventricular hypertrophy, reduced cardiovascular morbidity, and improvement in quality of life for these patients (19, 20).

In conclusion, left ventricular hypertrophy was found in 59 (71.96%) patients, left ventricular dilatation in 10 (12.19%), and 13 patients (15.85%) had regular

echocardiographic findings. The most frequent form of left ventricular hypertrophy was eccentric LVH, which was found in 30 (36.59%) patients, while 29 (35.37%) patients had concentric LVH. Disturbance in the systolic function of the left ventricle was present in 7 (8.54%) patients, and 75 (91.46%) patients had a normal systolic function of the left ventricle.

High prevalence of left ventricular hypertrophy among patients with end-stage renal failure that initiate hemodialysis treatment demands on early detection and identification of patients with left ventricular hypertrophy, along with timely application of appropriate therapy in order to reduce cardiovascular morbidity and mortality in these patients.

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