Left Ventricular Hypertrophy in Nondiabetics Patients with Predialysis Chronic Renal Disease in the Hospital Center Elbasan

Healthcare

Keywords: Chronic Kidney Disease, Left ventricular hypertrophy; pulse pressure, hypertension, anemia.

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Abstract

Background: Cardiovascular disease (CVD) is still the major cause of death in patients with end stage chronic kidney disease (ES-CKD), with a mortality rate approximately 10 to 30 times greater than that of the general population. Multiple factors are involved in the development of CVD in CKD. Although left ventricular hypertrophy (LVH) is strong predictor of mortality in patients with end-stage renal disease, few studies are available before the start of dialysis treatment in our country. The purpose of this study is to evaluate the prevalence and clinical correlates of LVH in nondiabetic patients with chronic kidney disease (CKD) not yet undergoing renal replacement therapy and to examine the relations between anemia, pulse pressure (PP), hypertension (HTA) with left ventricular hypertrophy (LVH). Materials and methods: We investigated 111 nondiabetic patients with CKD, presented in ambulatory service. Patients excluded from the study were of ischemic heart and valvular heart disease. 26 patients presented second stage of CKD (GFR 60-89,9ml/min). 30 patients presented third stage of CKD (GFR 30–59,9ml/min).32 patients was at 4th stage of CDK (GFR 15-29,9 ml /min) and 23 patients presented 5 th stage of CKD (GFR <15ml/min).Each patient had blood pressure (BP) measured by means of 24-hour ambulatory BP monitoring and left ventricular mass index (LVMi) assessed by means of M-mode echocardiography. Creatinine clearance was estimated by means of the Cockcroft-Gault formula, and hemoglobin were assessed by using routine methods. Results: The prevalence of LVH in nondiabetic predialysis patients with CKD was 81.9%; 22% of whom were women. The prevalence of hypertension was 72,6%. Anemia was present in all patients. In the overall group, prevalences of arterial hypertension,anemia and LVH were high. HTA is associated with LVH in patients with CKD, and the strong relationship between elevated pulse pressure and LVH in those with more advanced CKD suggests that increased arterial stiffness might have a role for LVH well before the start of dialysis therapy. Conclusions: In conclusion, the incidence of LVH was high even among nondiabetics patients under conservative treatment, and, except for age, LVH correlated with reversible factors. The need for strictly diagnosing CKD and preventing LVH in the predialysis phase is emphasized to decrease mortality due to CVD in that population.

Introduction

Cardiovascular complications are the leading cause of death in patients with end-stage renal disease (ESRD), accounting for 43-52% of deaths in these patients. Left Ventricular Hypertrophy (LVH) is a frequent occurrence in patients with CKD and is an important adverse prognostic indicator (1, 2).

Increased systolic blood pressure has been suggested as an independent predictor of left ventricular hypertrophy and its progression over time (2). Anemia is an important determinant of cardiac hypertrophy and a frequent finding in uremic patients (3). Anemia, in the long term, can be associated with progressive LV dilation, new-onset cardiac failure, and death (4). Increased PP is associated with the increase of systolic blood pressure (SBP) and decrease in diastolic blood pressure (DBP). PP reflects stiffness of the large arteries and increases with age (5, 6). PP is recognized as an independent predictor of myocardial infarction, congestive heart failure, and cardiovascular death, even in hypertensive patients who undergo successful antihypertensive drug therapy, especially in older individuals (7). Patients with CKD show higher PP values than control subjects with normal renal function (8). Several studies have shown that PP is a reliable prognostic factor for mortality and cardiovascular disease in predialysis, replacement therapy and renal transplant patients (9).

Purpose of the Study

The purpose of this study is to evaluate the prevalence and clinical correlates of LVH in nondiabetic patients with chronic kidney disease (CKD) not yet undergoing renal replacement therapy and to examine the relations between anemia, pulse pressure (PP), hypertension (HTA) with left ventricular hypertrophy (LVH).

Subjects and methods

We studied 111 nondiabetics patients in the predialysis stages of CKD. The mean age was 42±16,3. This was a cross-sectional study of 111 consecutive chronic kidney disease patients in the nephrology department of our hospital, who had been under conservative treatment for at least three months. The study comprised clinically stable patients with estimated glomerular filtration rate (eGF) ranging from 15 to 60 mL/min, and aged from 18 to 80 years. The exclusion criteria were as follows: acute or chronic infection; autoimmune disease; active treatment with steroids or immunosuppressors; and malignancy. All participants provided written informed consent, which had been approved by the Committees on Ethics in Research of their respective institutions. Demographical and clinical data were collected in the patients' medical records, and were as follows: age; sex; etiology of CKD.

The patients were devised in 4 groups according to the K/DOQI classification based on glomerular filtration (GFR). The first group of patients (26 patients) presented second stage of CKD (GFR 60-89,9ml/min). The second group (30 patients) presented third stage of CKD (GFR 30–59,9ml/min).The third group (32 patients) was at 4th stage of CDK (GFR 15-29,9 ml /min) and the fourth group (23 patients) presented 5 th stage of CKD (GFR <15ml/min). Creatinine clearance was calculated by usingthe Cockcroft and Gault equation.

Hypertension was defined as systolic blood pressure (SBP) > 140mmHg and diastolic blood pressure (DBP) > 90mmHg. All patients were under antihypertensive therapy. Pulse pressure was calculated as a difference between SBP and DBP.

Anemia has been considered as a level of hemoglobin <13mg/dl in the men and <12mg/dl in women.

Echocardiography Left Ventricular Mass index (LVMi) assessed by means of M-mode echocardiography. Left ventricular mass in relation to the patient's height is left ventricular mass index (LVMI) (g/m^2), which was considered normal when lower than 131 g/m^2 in men and lower than 100 g/m^2 in women.(10) Criteria for left ventricular hypertrophy (LVH) were considered LVMI >134 g/m2 for males and >110 g/m2 for female.

Statistical Analysis

Data are expressed as the mean ±SD. Spearman correlation was used to assess the relationship between LVMI and the variables (SBP, DBP, hemoglobin, pulse pressure). *P* value of < 0,05 was considered to be statistically significant. Statistical analysis were performed using the computer software SPSS 8.0

Results

The population studied was 54% male and 46% female. The prevalence of LVH was 81.9%, 22% of whom were women. The prevalence of hypertension was 72,6%. Anemia was present in all patients. The specific data for the studied parameters are presented in tab.1.

Table 1. Clinical, laboratory and echocardiography parameters by renal function. Hb- Hemoglobin, SBP –systolic blood pressure; DBP – diastolic blood pressure, LVMI–left ventricular mass index

Parameters	**GFR 60-89,9 ml/min (n=26)**	**GFR 30–59,9 ml/min (n=30)**	**GFR 15-29,9ml/min (n=32)**	**GFR <15 ml/min (n=23)**	**Total (n=111)**
Hb, mg/dL	8,6±1,2	8,2± 1,8	7,8±1,2	7,3 ± 1,5	7,9±1,4
SBP, mm Hg	160.3±16	162,4±18	148,0±21	160,±912	157,9±17
DBP, mm Hg	93.2±7	92,.4±10	91.8±12	84,1±9,6	90,4±12,15
Pulse pressure, mmHg	67,1±6	69,±78	56,8±12	76,9±0,8	65,1±6,7
LVMI, g/m2	135.40±55	145.±18	160.±32,75	190.±56	157,5444.

The correlations between left ventricular mass index and hemoglobin, systolic blood pressure, diastolic blood pressure and pulse pressure for each group and for all patients are presented in tab.2

Table 2.Correlations values of left ventricular mass index and Hb, SBP, DBP and PP Hb - Hemoglobin, SBP– systolic blood pressure; DBP – diastolic blood pressure, PP- Pulse pressure

	Group I		**Group II**		**Group III**		**Group IV**		**Total**	
Parameters	r	**P**	**r**	**P**	**r**	**P**	**r**	**P**	**r**	**P**
Hb	0.07	NS	-05	NS	-0,07	NS	-0,16	NS	-0,2	0.01
SBP	-0,17	NS	0,09	NS	-0,02	NS	0,58	0.01	0,2	0,04
DBP	-0,03	NS	0.05	NS	0,43	0,01	0,33	NS	0.017	NS
PP	-0,2	NS	0,01	NS	-0,24	NS	0,44	0,03	0,6	0,01

Discussion

Cardiovascular disease is the major cause of death in patients with CKD prior to dialysis.(12-13) Left ventricular hypertrophy is present in 81.9% of the patients starting renal replacement therapy.(11) Most of the patients assessed in this study were in stage 4 of CKD. Most patients with LVH were males; women did not show the same LVH prevalence, especially in stage 3. This high LVH prevalence helps to keep mortality high and impairs the quality of life of that population. Most of the studies show prevalence 40-80% of this cardiac geometric anomaly in the pre-dialysis patients.

The prevalence of hypertension was also high (72.6 %) and anemia was present in all patients, independently from the stage of CKD.

Strict control of blood pressure (BP) is known to be one of the best practices to prevent LVH. (14) Of all patients, 49% showed high systolic BP levels, and 34% showed high diastolic BP levels. Of those with LVH, 54% and 41% had increased systolic and diastolic BP levels, respectively. In addition, diastolic BP was an independent determinant of LVH. This is a worrying result, because BP control should no be a difficult target to be reached, at least for most patients, considering the existing therapeutic armamentarium. We insist in the importance of strict BP control as an efficient measure for preventing both LVH and CKD progression.[24]

The prevalence of all parameters studied grows with the progression of CKD.Of the variables tested, hemoglobin, SBP and PP predicted independently the occurrence of LVH. In the first and second group there is no significant correlation between LVMI and various parameters (Hb, SBP, and PP), although the prevalence of LVH was high in both groups. This data may be explained by the relatively small number of patients presented in relatively early stages of CKD. The third group shows correlation between LVMI and DBP, otherwise the 4th group shows a strong correlation with SBP and PP. When analyzed separately none of the groups demonstrates any correlation between lower levels of Hb and LVMI. In contrast, when all the patients were analyzed, it resulted a strong inverse correlation between levels of Hb and LVMI. By the other hand, it resulted also a strong positive correlation between SBP and PP with LVMI.We thought that this controversially data are result of the small number of patients for each group, and when we analyzed a significant number of patients the correlations emerged clearly. Anemia contributes to volume overload.Our patients presented a high prevalence of hypertension; therefore peripheral resistance may have playeda significant role in LVH observedin our patients. The analysis of dates shows that PP and SBP were predictors of LVH.

Conclusion

In conclusion, the incidence of LVH was high even among nondiabetics patients under conservative treatment, and, except for age, LVH correlated with reversible factors. The need for strictly diagnosing CKD and preventing LVH in the predialysis phase is emphasized to decrease mortality due to CVD in that population.

This study has shown a strong association between CKD and LVH in predialysis patients. The patients were anemic and presented high prevalence of hypertension. In our study pulse pressure, SBP and anemia are important predictor factors for development of left ventricular hypertrophy.

The results have shown that some determinants are reversible factors. Different studies have shown that control of hypertension and anemia lead to a decrease of LVH prevalence. The effect of PP reduction on LVH in CKD remains to be determined. Therefore, more evidences are necessary to evaluate the role of PP reduction as a therapeutic target in the treatment of patients with CKD.

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