

Original Article

Pulse Pressure is Independent Risk Factor of Left Ventricular Hypertrophy in Chronic Kidney Disease

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Abstract

Background. The aim of the study was to evaluate the relations between pulse pressure (PP), hypertension and anemia with left ventricular hypertrophy (LVH).

Methods. The risk factors and prevalence of LHV were evaluated in 111 patients with CRF.

Results. LVH was diagnosed in 81,9 % of all patients. The prevalence of hypertension was 72,6%. Anemia was present in all patients.

Of the variables tested lower levels of hemoglobin, systolic blood pressure (SBP) and PP predicted the occurrence of LVH.

Conclusions. This study has shown a strong association between chronic kidney disease (CKD) and LVH in pre dialysis patients.

Pulse pressure, SBP and anemia play an important role in the development of left ventricular hypertrophy in CKD patients.

Keywords: anemia, hypertension, left ventricular hypertrophy, pulse pressure

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

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 is 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 [12].

Increased PP is associated with the increase of systolic blood pressure (SBP) and decrease in diastolic blood pressure (DBP) [4] PP reflects stiffness of the large arteries and increases with age [5,6]. Increasingly, 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 [5]. Several studies have shown that PP is a reliable prognostic factor for mortality and cardiovascular disease in

predialysis, replacement therapy and renal transplant patients [8].

The aim of our study was to evaluate the relations between pulse pressure, hypertension and anemia with left ventricular hypertrophy.

Patients and methods

The study is trans-sectional. We studied 111 in the predialysis stages of CDK. The mean age was 42±16,3. The patients were divided 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 60-89,9 ml/min). The second group (30 patients) presented third stage of CKD (GFR 30–59,9 ml/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 5th stage of CKD (GFR <15 ml/min). Creatinine clearance was calculated by using the Cockcroft and Gault equation.

Hypertension was defined as systolic blood pressure (SBP) > 140 mmHg and diastolic blood pressure (DBP) > 90 mmHg. 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 <13 mg/dl in the men and <12 mg/dl in women.

Echocardiography was performed using the Vingmed System Five echocardiographic system equipped with 2.5 MHz transducers. M-Mode and 2D measurements were done in accordance with methods recommended by the American Society of Echocardiography. Criteria for left ventricular hypertrophy (LVH) were considered LVMI >134 g/m² for males and >110 g/m² for females. Cardiac mass was calculated using Reichek and Devereux formula.

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 was performed using the computer software SPSS 8.0.

Results

LVH was diagnosed in 81,9 % of all patients. The prevalence of hypertension was 72,6%. Anemia was present in all patients. The group with second stage of CKD had an

average of Hb 8.6 ± 1.2 mg/dl, SBP 160.3 ± 16 mmHg, DBP 93.2 ± 7 mmHg, Pulse pressure 67.1 ± 6 mmHg and LVMI 135.40 ± 55 g/m². The group with third stage of CKD presented with Hb 8.2 ± 1.8 mg/dl, SBP 162.4 ± 18 mmHg, DBP 92.4 ± 10 mmHg, Pulse pressure 69.7 ± 8 mmHg and LVMI 145 ± 18 g/m². The group with fourth stage of CKD presented with Hb 7.8 ± 1.2 mg/dl, SBP 148.0 ± 21 mmHg, DBP 91.8 ± 12 mmHg, Pulse pressure 56.8 ± 12 mmHg and

LVMI 160 ± 32.75 g/m². The group with fifth stage of CKD presented with Hb 7.3 ± 1.5 mg/dl, SBP 160.9 ± 12 mmHg, DBP 84.1 ± 9.6 mmHg, Pulse pressure 76.9 ± 0.8 mmHg and LVMI 190 ± 56 g/m². The average data for all groups were Hb 7.9 ± 1.4 mg/dl, SBP 157.9 ± 17 mmHg, DBP 90.4 ± 12.15 mmHg, Pulse pressure 65.1 ± 6.7 mmHg and LVMI 157.5 ± 44 g/m².

The data for the studied parameters are presented in Table 1.

Table 1. Clinical, laboratory and echocardiography parameters by renal function

Parameters	GFR 60-89,9 ml/min (n=26)	GFR 30-59,9 ml/min (n=30)	GFR 15-29,9 ml/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	160,9±12	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	69,7±8	56,8±12	76,9±0,8	65,1±6,7
LVMI, g/m ²	135.40±55	145±18	160±32.75	190±56	157,5±44.

GFR - Glomerular filtration; Hb - Hemoglobin; SBP - systolic blood pressure; DBP - diastolic blood pressure; LVMI - left ventricular mass index

We found a significant correlation between LVMI and DBP in the patients presenting IV stage of CKD, also correlations between LVMI with SBP and PP in patients presenting V stage of CKD. When we studied the data for all series there were a strong correlations between LVMI with Hb, SBP and PP.

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

Table 2. Correlations values of left ventricular mass index and Hb, SBP, DBP and PP

Parameters	Group I		Group II		Group III		Group IV		Total	
	r	P	r	P	r	P	r	P	r	P
Hb	0,07	NS	-0,05	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

Hb - Hemoglobin; SBP - systolic blood pressure; DBP - diastolic blood pressure; PP - Pulse pressure; r - Correlation coefficient

Discussion

Cross-sectional studies have shown that left ventricular hypertrophy (LVH) is the most frequent cardiac alteration in CKD and is an independent risk factor for survival.

The data of the prevalence of LVH in chronic renal failure are controversial. It was shown in 25% to 87% of predialysis patients [1] and in 50% to 97% of dialysis patients [2]. In the absence of intrinsic heart disease, LV enlargement is most probably attributable to chronic volume flow overload associated with anemia, the presence of arteriovenous shunts, and sodium and water retention [3]. Anemia is an important determinant of cardiac hypertrophy, which is a frequent finding in uraemic patients [9]. Wide pulse pressure (PP) has been associated with increased cardiovascular disease among hypertensive subjects [13]. 10 mmHg increases in PP was found to increase the risk of major cardiovascular complications and mortality by nearly 20% [6]. Elevated pulse pressure in CKD has been shown to be associated with left ventricular hypertrophy [8]. Arteriosclerosis simultaneously tends to increase SBP and lower DBP, resulting in a widened PP, which paves the way

for CVD morbidity, because elevated SBP is associated with a greater left ventricular workload, enhancing myocardial wall stress and oxygen demand. In addition, the decreased DBP may result in a reduced coronary perfusion pressure, resulting in a decreased myocardial oxygen supply and a greater risk for myocardial ischemia and infarction [5].

This study was undertaken to determine the prevalence of LVH and some factors associated with it. The prevalence of LVH is high (72,6%) in our patients. The prevalence of hypertension was also high (81,9 %) and anemia was present in all patients, independently from the stage of CKD. 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 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 with strong inverse correlation between levels of Hb and LVMI. On the other hand, it resulted also with a strong positive correlation between SBP and PP with LVMI.

We assumed 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 in our study resulted as an independent predictor of LVH. The presence of anemia contributes to volume overload, which may be the basic player in installing LVH. The analysis of data shows also that PP and SBP were predictors of LVH. Our patients presented a high prevalence of SBP; that is also accompanied with widening of PP, therefore peripheral resistance may have played a significant role in LVH observed in our patients.

To consider the relations of these variables with LVH, especially in early stages of CKD, studies with larger number of patients are required.

Conclusion

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

Conflict of interest statement. None declared.

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