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Left ventricular geometry in children with chronic renal failure

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Summary. *The aim of the study was to assess left ventricular (LV) geometry in children with chronic renal insufficiency (CRI), and its relationship with glomerular filtration rate (GFR) and preexisting renal disease. Echocardiography was performed on 56 non-dialysed CRI patients and 56 controls. CRI patients had bigger interventricular septum thickness (0.77±0.17 vs. 0.67±0.12 cm, p=0.002), LV posterior wall thickness (0.79±0.14 vs. 0.71±0.14 cm, p<0.006), LV mass index (LVMI) (40.7±12.2 vs. 31.7±6.3 g/m$^{2.7}$, p<0.0001; 86.4±24.1 vs. 69.1±13.9 g/m^2, p<0.0001), and relative wall thickness (0.38±0.05 vs. 0.34±0.04, p<0.0001) in comparison with controls. Twenty (36%) of CRI patients had LV hypertrophy (LVH). Thirteen patients (23%) had eccentric LVH, 7 (13%) – concentric LVH), and 9 (16%) of patients – concentric LV remodeling. No significant difference was found between LV parameters in patient groups with different GFR. Patients with acquired renal diseases and hereditary nephropathies had significantly higher LVMI than patients with congenital renal abnormalities. Our results indicate that changes of LV geometry are present in children with mild, moderate and predialysis CRI. These findings support the concept of cardiovascular disease risk for patients with different stages of CRI.*

Introduction

Cardiac morbidity has been investigated extensively in adults with chronic kidney disease (CKD) and end-stage renal disease (ESRD) (1, 2). A higher rate of adverse cardiovascular (CV) events is noted among this cohort when compared with those with normal renal function (3, 4). Cardiovascular disease often begins before ESRD, and patients with reduced kidney function are more likely to die of CVD than to develop ESRD (5). Mechanisms as to why renal dysfunction portends increased CV risk are still being elucidated.

Cardiac disease has been studied to a much lesser extent in children and adolescents with ESRD and after renal transplantation (6, 7). There is also evidence that CVD can be a cause of death in the pediatric ESRD population (8, 9). However, only few studies evaluated cardiac changes in children with mild-to-moderate CRI (10).

The purpose of this study was to assess changes of left ventricular (LV) geometry in non-dialyzed children with CRI, and its relationship with glomerular filtration rate (GFR) and preexisting renal disease.

Patients and methods

The study population consisted of 56 patients with CRI treated in Vilnius University Children's Hospital. The inclusion criteria were: age 1 to 18 years; calculated GFR less than 90 ml/min/1.73 m^2; no previous dialysis; absence of congenital, structural, or primary myocardial disease. 56 children chosen as a control group were normotensive, age and sex matched, with no clinical evidence of cardiovascular disease, or no previous or existing renal disease.

Echocardiography was performed in the left lateral supine position using an AV 3 Partner (Esaote Biomedica) or Aloka ProSound SSD 5000 system with 2.5–3.5 Hz or 1.8–3.75 Hz transducer. From 2D directed M-mode echocardiography left ventricular internal end-diastolic diameter (LVIDd), left ventricular internal end-systolic diameter (LVIDs), interventricular septal thickness at end-diastole (IVSd), and left ventricular posterior wall thickness at end-diastole (LVPWd) were measured in the parasternal long and short axis cuts. Measurements were made at or just below the tips of the mitral valve leaflets according to the recommendations of the American Society of Echocar-

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diography (11). Relative wall thickness was measured at the end-diastole as: 2 x LVPWd/LVIDd. The left ventricular mass (LVM) was calculated using the formula of Devereux *et al.* (12). Linear dimensions (LVIDd, LVIDs, LVPWd, IVSd) were compared using direct measurements and measurements indexed to $BSA^{0.5}$ as recommended by Gutgesell and Rembold (13). Volume determinations were calculated by the Teichholz method (14). Left ventricular end-diastolic volume (LVDV) and left ventricular end-systolic volume (LVSV) were indexed to BSA for further comparisons. LVM was indexed to BSA and to height $(Ht)^{2.7}$ according to the allometric regression equation described by de Simone *et al.* (15).

For children older than 6 years, left ventricular hypertrophy (LVH) was defined as LVM greater than 95th centile prediction interval for age, sex and height according to Malcolm *et al.* (16). A LVM of 60.1 g (males) and 62.0 g (females) were used to determine LVH in children younger than 6 years. These values are given for 6 years old children on the 5th height centile for the 95th centile for LVM (16). Patients with increased LVM and elevated relative wall thickness (RWT) (=0.41) were considered to have concentric LVH; those with increased LVM and normal RWT (=0.41) had eccentric LVH. Concentric remodeling was defined as elevated RWT, but with normal LVM.

Laboratory data were collected on the day of the echocardiographic evaluation. GFR was calculated from the serum creatinine concentrations, measured by standard laboratory techniques. Patients were divided into 4 groups according to their GFR level: group 1 – GFR <15 ml/min/1.73m², n=11 patients; group 2 – GFR between 15–29 ml/min/1.73m², n=20; group 3 – GFR between 30–59 ml/min/1.73m², n=17; and group 4 – GFR between 60–89ml/min/1.73m², n=8 to compare echocardiographic parametres between these subgroups.

The medical records were reviewed for cause of chronic renal disease, and patients were further divided according to their diagnoses: group 1 – congenital renal abnormalities, n=20 patients; group 2 – acquired renal diseases, n=22; and group 3 – hereditary nephropathies, n=14.

Results were expressed as the mean ± standard deviation. Significance of statistical difference between two samples was evaluated using Student's t test. Statistical significance level was chosen at $p<0.05$.

Results

Patient characteristics

There were 22 boys and 34 girls in the CRI as well as in the control group. Mean CRI patient's age was 10.8±4.2 years (range from 2 to 17 years). The main causes of chronic kidney diseases in the congenital renal abnormalities group were renal hypodysplasias, reflux nephropathies, hydronephrosis; in the acquired renal diseases group – glomerulonephritis, hemolytic-uremic syndrome; and in the hereditary nephropathies – juvenile nephronophthisis, autosomal recessive polycystic kidney disease, cystinosis etc. The mean GFR for children with CRI was 33.6±22.3 ml/min/1.73 m². There was no significant difference in age, weight, height, and BSA among CRI patients and the control group (Table 1).

Cardiac geometry

Echocardiographic measurements of LV structure are presented in Table 1. CRI patients had bigger interventricular septum thickness (IVSd) (0.77±0.17 *vs.* 0.67±0.12 cm, p=0.002), LV posterior wall thickness (LVPWd) (0.79±0.14 *vs.* 0.71±0.14 cm, p<0.006), LV mass index (LVMI) (40.7±12.2 *vs.* 31.7±6.3 g/$m^{2.7}$, p<0.0001; 86.4±24.1 *vs.* 69.1±13.9 g/m^2, p<0.0001), and RWT (0.38±0.05 *vs.* 0.34±0.04, p<0.0001) in comparison with controls. No significant difference was found between LV parameters in patient groups with different GFR (Table 2). Patients with acquired renal diseases and hereditary nephropathies had significantly higher LVMI (p=0.04 and p=0.02, respectively) than patients with congenital renal abnormalities (Table 3).

20 (36%) of CRI patients had LVH (13 (23%) had eccentric LVH, 7 (13%) – concentric LVH), and 9 (16%) of patients – concentric LV remodeling). Abnormalities of LV geometry were more common in the patients with GFR <30 ml/min/1.73m² (Table 2), eccentric LVH being the most common in the group of patients with GFR 15–29 ml/min/1.73m². Concentric LVH and concentric remodeling were equally found in all the groups with different GFR. Abnormal LV geometry was less common in patients with congenital renal abnormalities (Table 3). All types of LV geometry patterns were found in children with acquired renal diseases, while eccentric LVH and concentric remodeling dominated in the group of hereditary nephropathies.

Discussion

Important observation of this study is that not only children with advanced CRI, but also children with mild or moderate CRI had abnormalities of LV geometry. LVH and geometry pattern in patients with CRI are associated with important clinical and prognostic implications, probably due to several biological and pathophysiological differences in hemodynamic profiles and left ventricular systolic and diastolic dysfunction

Table 1. **Demographics and echocardiographic data in patients with CRI and healthy controls (mean value ± standard deviation)**

	CRI patients, n=56	Controls, n=56	p value
Age (years)	11.1±3.9	10.8±4.2	n.s.
Gender (boys/girls)	22 / 34	22 / 34	n.s.
Weigh (kg)	36.4±15.7	39.6±16.0	n.s.
Height (cm)	140.9±22.5	145.3±24.6	n.s.
Body surface area (m^2)	1.19±0.35	1.27±0.4	n.s.
LVIDd (cm) LVIDdI (cm/m)	4.21±0.59 3.92±0.38	4.23±0.56 3.82±0.24	n.s. n.s.
LVIDs (cm) LVIDsI (cm/m)	2.73±0.45 2.45±0.57	2.74±0.44 2.47±0.22	n.s. n.s.
IVSd (cm) IVSdI (cm/m)	0.77±0.17 0.71±0.11	0.67±0.12 0.6±0.07	0.0015 <0.0001
LVPWd (cm) LVPWdI (cm/m)	0.79±0.14 0.74±0.1	0.71±0.14 0.64±0.08	0.0057 <0.0001
LVDV (cc) LVDVI (cc/m^2)	81.2±25.3 69.0±14.6	82.1±24.9 65.2±7.9	n.s. 0.05
LVSV (cc) LVSVI (cc/m^2)	28.0±12.2 23.6±8.0	29.4±11.4 22.9±4.8	n.s. n.s.
RWT	0.38±0.05	0.34±0.04	<0.0001
LVM (g) LVMI ($g/m^{2.7}$) LVMI (g/m^2)	104.5±44.5 40.7±12.2 86.4±24.1	90.0±37.5 31.7±6.3 69.1±13.9	n.s. <0.0001 <0.0001

n.s. – not significant.

(17, 18). LVH has been identified as a strong independent predictor of cardiovascular morbidity and mortality in the adult population (19).

LVH as an adaptive response to chronic pressure or volume overload is a beneficial response initially, because it maintains systolic function, allows an increase in working capacity, and reduces wall stress and energy consumption (1). Persistent LVH, however, may become detrimental, mainly because it reduces the coronary perfusion reserve and alters diastolic left ventricular function.

The contribution of LVH to children morbidity is unknown. The risk of premature CVD or death in the otherwise normal child is small, and it is not surprising that LVH, as a CV risk factor has not been studied in children.

The increasing use of echocardiography in clinical cardiology has greatly facilitated the detection of cardiac abnormalities and evaluation of LVM and dimensions in young patients.

Increased LVM or LVH has been described in children with CRI and ESRD and after renal transplantation (20). In the only large study performed by European Dialysis and Transplant Association in children up to the age of 15 years, 51% of patients on hemodialysis, 29% on peritoneal dialysis, and 22% after renal transplantation were reported to have LVH by echocardiographic examination. However, only 60% of the study population was available for assessment ant methodological details were not collected (21, 22). Other echocardiographic studies usually comprised only small groups of school children and adolescents analyzed by variable methods and rarely longitudinally. Only few studies evaluated LVH in children with mild-to-moderate CRI. Mitsnefes *et al.* have shown that children with mild and moderate CRI and on chronic dialysis had increased LVM. 21% of children with CRI, and 41% of pediatric dialysis patients had LVH (10).

Our study was probably the first attempt to evaluate the left ventricular geometry in the relatively large young non-dialyzed patients group with different stages of CRI. Comparison of our results and results of dif-

Table 2. **Echocardiographic data in CRI patients groups with different GFR**

	Group 1 n=11	Group 2 n=20	Group 3 n=17	Group 4 n=8
GFR (ml/min/1.73 m^2)	11.8±2.4	19.9±4.4	44.3±8.3	74.6±7.8
Age (years)	11.5±3.8	12.1±3.7	9.1±4.4	10.75±4.6
Gender (boys/girls)	6/5	7/13	6/11	4/4
Weigh (kg)	36.6±15.6	35.2±13.0	34.1±20.1	37.4±16.0
Height (cm)	139.8±19.5	141.9±21.7	133.9±27.7	145±26.0
Body surface area (m^2)	1.18±0.32	1.19±0.31	1.11±0.43	1.23±0.37
LVIDd (cm) LVIDdI (cm/m)	4.1±0.57 3.8±0.4	4.37±0.59 4.07±0.39	3.92±0.58 3.83±0.39	4.3±0.59 3.95±0.32
LVIDs (cm) LVIDsI (cm/m)	2.63±0.47 2.19±0.77	2.82±0.44 2.5±0.72	2.54±0.44 2.48±0.29	2.83±0.48 2.59±0.3
IVSd (cm) IVSdI (cm/m)	0.78±0.09 0.73±0.09	0.8±0.18 0.74±0.11	0.66±0.14 0.64±0.08	0.78±0.18 0.71±0.13
LVPWd (cm) LVPWdI (cm/m)	0.8±0.11 0.75±0.1	0.83±0.14 0.7±0.09	0.71±0.12 0.7±0.09	0.8±0.19 0.73±0.11
LVDV (cc) LVDVI (cc/m^2)	76.2±22.8 65.0±15,7	88.6±26.4 75.4±15.5	68.7±22.6 64.0±13.2	85.7±27.1 70.4±11.6
LVSV (cc) LVSVI (cc/m^2)	24.1±13.5 19.6±8.6	29.5±13.3 25.5±10.5	24.4±9.9 22.3±5.6	31.5±13.2 25.6±7.2
RWT	0.4±0.05	0.38±0.05	0.36±0.05	0.37±0.05
LVM (g) LVMI (g/m$^{2.7}$)	99.4±30.2 40.7±13.5	117.2±46.8 44.6±11.2	78.1±32.9 35.4±10.8	111.1±54.7 39.7±12.9
LV geometry, n (%): Normal Concentric LVH Eccentric LVH Concentric remodeling	 5 (45) 2 (18) 1 (9) 3 (27)	 6 (30) 3 (15) 9 (45) 2 (10)	 11 (65) 0 2 (12) 4 (24)	 5 (63) 2 (25) 1 (13) 0

ferent studies measuring LVMI in pediatric patients with CRI by echocardiography is presented in Table 4. There is no universal agreement as to the manner in which data should be presented. For comparison of our data with the data of other studies we indexed LVM to BSA and height$^{2.7}$.

Nowadays, four distinctive patterns of LVH and geometry are generally accepted, based on echocardiographic LVMI and RWT: normal geometry, concentric remodeling, concentric hypertrophy and eccentric hypertrophy. In principle, two forms of LVH may be distinguished: pressure overload which causes a disproportionate growth of cardiomyocytes leading to concentric (symmetric) LVH with thickening of both septum and posterior wall, but with normal cavity dimensions; and volume overload resulting on dilatation of the chamber and increased wall thickness sufficient to counterbalance the dilatation. This eccentric (asymmetric) form of LVH is characterized by a predominant thickening of the septum and a lower LVM to volume ratio. The type of LVH and the consequence of LVH depend on several factors, e.g. its initial cause, the age at onset, the rapidity of onset, genetic factors, neurohumoral activation, effects of growth factors. All this conditions contribute to the heterogeneity of the LVH response. Individuals with concentric hypertrophy are at the greater risk of cardiovascular events than patients with eccentric hypertrophy since the deleterious effects of cardiac alterations are more pronounced in pressure-overload than in volume-overload hypertrophy (23).

A high incidence of LVH, mainly attributable to asymmetric septal hypertrophy, was found in children treated by dialysis (24). Some reports suggested that

Table 3. **Echocardiographic data in CRI patients with different causes of chronic renal disease**

	Group 1 n=20	Group 2 n=22	Group 3 n=14
GFR (ml/min/1.73 m^2)	29.6±21.1	38.7±22.6	30.7±20.7
Age (years)	10.9±4.0	10.5±4.1	11.1±5.1
Gender (boys/girls)	7/13	9/13	7/7
Weigh (kg)	33.0±14.7	35.7±15.2	39.1±19.6
Height (cm)	138.4±21.6	140.1±23.5	138.3±30.0
Body surface area (m^2)	1.13±0.33	1.18±0.34	1.21±0.44
LVIDd (cm) LVIDdI (cm/m)	4.0±0.63 3.82±0.35	4.27±0.47 4.02±0.42	4.28±0.71 4.01±0.42
LVIDs (cm) LVIDsI (cm/m)	2.6±0.49 2.47±0.29	2.78±0.34 2.39±0.84	2.78±0.54 2.58±0.26
IVSd (cm) IVSdI (cm/m)	0.73±0.13 0.69±0.08	0.77±0.19 0.73±0.16	0.80±0.19 0.74±0.09
LVPWd (cm) LVPWdI (cm/m)	0.76±0.13 0.72±0.1	0.82±0.13 0.77±0.12	0.79±0.17 0.73±0.07
LVDV (cc) LVDVI (cc/m^2)	72.6±25.9 64.4±13.9	83.2±21.4 72.9±15.8	85.2±30.9 72.1±14.7
LVSV (cc) LVSVI (cc/m^2)	25.9±11.2 22.6±6.6	27.0±12.5 24.0±11.0	30.6±13.5 25.1±5.4
RWT	0.38±0.06	0.38±0.05	0.37±0.05
LVM (g) LVMI (g/m$^{2.7}$)	89.4±36.9 36.0±8.9*$^{\#}$	108.5±43.6 45.1±18.4$^{\#}$	111.9±55.4 44.8±11.1*
LV geometry, n (%): Normal Concentric LVH Eccentric LVH Concentric remodeling	 12 (60) 2 (10) 2 (10) 4 (20)	 9 (41) 5 (23) 6 (27) 2 (9)	 6 (43) 0 5 (36) 3 (21)

* statistically significant difference ($p<0.05$) between groups 1 and 3.
$^{\#}$ statistically significant difference ($p<0.05$) between groups 1 and 2.

Table 4. **LV mass index in pediatric patients with CRI compared with controls (results of different studies)**

Authors	CRI	Controls
Johnstone et al. 1996 (7)	80.6±29.3 g/m^2 n=32	66.9±17.6 g/m^2 n=60
Litwin et al. 1997 (21)	65.4±35 g/m^2 n=12	51.3±15 g/m^2 n=7
Mitsnefes et al. 2004 (22)	35.4±10.6 g/m$^{2.7}$ n=33	31.7±5.4 g/m$^{2.7}$ n=33
Own results 2005	86.4±24.1 g/m^2 40.7±12.2 g/m$^{2.7}$ n=56	69.1±13.9 g/m^2 31.7±6.3 g/m$^{2.7}$ n=56

eccentric hypertrophy is the predominant form of LVH in dialysis patients (25). Johnstone *et al.* found more concentric ventricular hypertrophy (7). According to recent views, LVH in ESRD combines the features of concentric and eccentric hypertrophy (23). According to data of Mitsnefes *et al.*, concentric LVH was more common in children with CRI, while eccentric LVH was the most common abnormal geometric pattern in dialysis patients (26). Concentric hypertrophy is thought to result in a stiffer left ventricle. On the other hand, eccentric hypertrophy is more likely to result from increased circulatory blood volume. This volume overload may lead to increased LV dimensions. It is possible that pressure overload may be important in the patients with CRI, while volume overload is more important in patients treated by dialysis. Similarly, in our study concentric LVH and concentric remodeling were equally found in all the groups with different GFR, eccentric LVH being the most common in the group of patients with lower GFR.

In our study, no significant difference could be found between LV parameters in patient groups with different GFR. Similarly, no influence of GFR on echocardiographic parameters was established in acute glomerular disorders (27). There are a lot of hemodynamic, vascular, humoral, metabolic and toxic factors influencing cardiac function and structure in chronic renal disease (20). The results of our study could be influenced by the lack of taking into account anemia, blood pressure, duration of the disease and other factors. How these factors effect LV geometry requires further study.

According to our data, patients with acquired renal diseases and hereditary nephropathies had significantly higher LVMI than patients with congenital renal abnormalities. Abnormal LV geometry was less common in patients with congenital renal abnormalities as well. On the other hand, eccentric LVH and concentric remodeling dominated in the group of hereditary nephropathies. These data suggest that the cause of CRI may play some role in LV geometry pattern, possibly due to different factors influencing cardiac structure during different preexisting disease.

In conclusion, our results indicate that changes of left ventricular geometry are present in children with mild, moderate and predialysis CRI. These findings support the concept of cardiovascular disease risk for patients with different stages of CRI. Cardiac complications in children with chronic renal failure deserve regular clinical and echocardiographical monitoring in order to minimize later cardiovascular morbidity by appropriate treatment. Further studies of left ventricular hypertrophy in children with chronic renal diseases are needed.

Vaikų, sergančių lėtiniu inkstų funkcijos nepakankamumu, kairiojo skilvelio geometrija

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Raktažodžiai: vaikai, echokardiografija, lėtinis inkstų funkcijos nepakankamumas, širdis.

Santrauka. *Darbo tikslas.* Įvertinti vaikų, sergančių lėtiniu inkstų funkcijos nepakankamumu kairiojo skilvelio geometriją ir jos ryšį su glomerulų filtracijos greičiu bei pirmine inkstų liga. Širdies echokardiografija iki pradedant hemodializę atlikta 56 ligoniams, sergantiems lėtiniu inkstų funkcijos nepakankamumu, ir 56 vaikams, kurie sudarė kontrolinę grupę. Ligoniams, sergantiems lėtiniu inkstų funkcijos nepakankamumu, rasta storesnė tarpskilvelinė pertvara (0,77±0,17 ir 0,67±0,12 cm, p=0,002), kairiojo skilvelio užpakalinės sienos storis (0,79±0,14 ir 0,71±0,14 cm, p<0,006), kairiojo skilvelio masės indeksas (40,7±12,2 ir 31,7±6,3 g/m^{2,7}, p<0,0001; 86,4±24,1 ir 69,1±13,9 g/m^2, p<0,0001) ir santykinis sienos storis (0,38±0,05 ir 0,34±0,04, p<0,0001) lyginant su kontroline grupe. 20 (36 proc.) ligonių, sergančių lėtiniu inkstų funkcijos nepakankamumu, rasta kairiojo skilvelio hipertrofija. 13 (23 proc.) nustatyta ekscentrinė kairiojo skilvelio hipertrofija, 7 (13 proc.) – koncentrinė kairiojo skilvelio hipertrofija ir 9 (16 proc.) ligoniams – koncentrinis kairiojo skilvelio remodeliavimasis. Kairiojo skilvelio parametrai statistiškai nesiskyrė tarp ligonių, kurių skirtingas glomerulų filtracijos greitis. Reikšmingai didesnis kairiojo skilvelio masės indeksas buvo rastas ligoniams, sergantiems įgytomis inkstų ligomis ir paveldimomis nefropatijomis nei sergantiems įgimtomis inkstų anomalijomis. Šio tyrimo duomenimis, kairiojo skilvelio geometrijos pokyčiai jau randami vaikams nedidelio, vidutinio ir preterminalinio lėtinio inkstų funkcijos nepakankamumo stadijose. Šie radiniai patvirtina teiginį, kad pacientai, kuriems nustatytos įvairios lėtinio inkstų funkcijos nepakankamumo stadijos, turi didesnę riziką sirgti širdies ir kraujagyslių ligomis.

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References

1. Raine AEG, Schawrz U, Ritz E. Hypertension and cardiac problems. In: Oxford textbook of clinical nephrology. 2nd ed. Oxford: Oxford University Press; 1998. p. 1885-918.
2. Levey AS, Eknoyan G. Cardiovascular disease in chronic renal disease. Nephrol Dial Transplant 1999;14:828-34.
3. Sarnak MJ, Levey AS, Schoolwerth AC, Coresh J, Culleton B, Hamm LL, et al. Kidney disease as a risk factor for development of cardiovascular disease: A statement from the American Heart Association Councils on kidney in cardiovascular disease, high blood pressure research, clinical cardiology, and epidemiology and prevention. Hypertension 2003;42:1050-65.
4. McCullough PA. Why is chronic kidney disease the "spoiler" for cardiovascular outcomes? J Am Coll Cardiol 2003;41:725-8.
5. Garg AX, Clark WF, Haynes RB, House AA. Moderate renal insufficiency and the risk of cardiovascular mortality: Results from the NHANES I. Kidney Int 2002;61:1486-94.
6. Schärer K, Ulmer HE. Cardiovascular complications of renal failure. In: Holliday MA, Barratt TM, Vernier RL, editors. Pediatric nephrology. 2nd ed. Baltimore: Williams and Wilkins; 1987. p. 887-96.
7. Johnstone LM, Jones CL, Grigg LE, Wilkinson JL, Walker RG, Powell HR. Left ventricular abnormalities in children, adolescents and young adults with renal disease. Kidney Int 1996;50:998-1006.
8. Brunner FP, Broyer M, Brynger H. Demography of dialysis and transplantation in children in Europe, 1985. Report from the European Dialysis and transplant Association Registry. Nephrol Dial Transplant 1988;3:235-43.
9. Litwin M, Grenda R, Prokurat S, Abuauba M, Latoszynska J, Jobs K, et al. Patient survival and causes of death on hemodialysis and peritoneal dialysis – single-center study. Pediatr Nephrol 2001;16:996-1001.
10. Mitsnefes MM, Kimball TR, Witt SA, Glascock BJ, Khoury PR, Daniels SR. Left ventricular mass and systolic performance in pediatric patients with chronic renal failure. Circulation 2003;107:864-8.
11. Sahn DJ, DeMaria A, Kisslo J, Weyman A. Recommendations regarding quantitation in M-mode echocardiography results: of a survey of echocardiographic measurements. Circulation 1978;58:1072-83.
12. Devereux RB, Alonso DR, Lutas EM, Gottlieb GJ, Campo E, Sachs I, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol 1986;57:450-8.
13. Gutgesell HP, Rembold CM. Growth of the human heart relative to body surface area. Am J Cardiol 1990;65:662-8.
14. Teichholz LE, Kreulen T, Herman MV, Gorlin R. Problems in echocardiographic volume determinations: echocardiographic-angiographic correlations in the presence of absence of asynergy. Am J Cardiol 1976;37:7-11.
15. de Simone G, Daniels SR, Devereux RB, Meyer RA, Roman MJ, de Divitiis O, et al. Left ventricular mass and body size in normotensive children and adults: assessment of allometric relations and impact of overweight. J Am Coll Cardiol 1992; 20:1251-60.
16. Malcolm DD, Burns TL, Mahoney LT, Lauer RM. Factors affecting left ventricular mass in childhood: The Muscatine Study. Pediatrics 1993;92:703-9.
17. Jaroch J, Loboz-Grudzien K, Kowalska A. Haemodynamic profiles in different patterns of left ventricular hypertrophy and geometry in patients with hypertension. Kardiol Pol 2001; 55:273-85.
18. McCullough PA. Cardiorenal risk: An important clinical intersection. Rev Cardiovasc Med 2002;3:71-6.
19. Levy D, Anderson KM, Savage DD, Kannel WB, Christiansen JC, Castelli WP. Echocardiographycally detected left ventricular hypertrophy: Prevalence and risk factors. The Framingham Heart Study. Ann Intern Med 1988;108:7-13.
20. Schärer K, Schmidt KG, Soergel M. Cardiac function and structure in patients with chronic renal failure. Nephrol 1999;13: 951-65.
21. Loirat C, Ehrich IMH, Geerlings W, Jones EHP, Landais P, Mallick NP, et al. Report on management of renal failure in children in Europe XXII, 1992. Nephrol Dial Transplant 1994; 12:1557-60.
22. Litwin M, Kawalec W, Grenda R. Left ventricular changes in the course of chronic renal failure and during dialysis therapy. In: Timio M, Wizemann V, Venanzy S, editors. VI European Meeting on Cardionephrology. Editoriale Bios Cosenza; 1997. p. 93-6.
23. London GM. Heterogeneity of left ventricular hypertrophy – does it have clinical implications? Nephrol Dial Transplant 1998;13:17-9.
24. Morris KP, Skinner JR, Wren C, Hunter S, Coulthard MG. Cardiac abnormalities in end-stage renal failure and anaemia. Arch Dis Child 1993;68:637-43.
25. Hüting J, Kramer W, Schütterle G, Wizemann V. Analysis of left ventricular changes associated with chronic hemodialysis. Nephron 1988;49:284-90.
26. Mitsnefes MM, Kimball TR, Border WL, Witt SA, Glascock BJ, Khoury PR, et al. Impaired left ventricular diastolic function in children with chronic renal failure. Kidney Int 2004;65:1461-6.
27. Jankauskiene A, Jakutovic M, Cerniauskiene V, Malikenas A. Echocardiographic findings in children ill with acute postinfectious glomerulonephritis. Eur J Pediatr 2003;162:500-5.

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