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Original Paper

Influence of the Method of Definition on the Prevalence of Left-Ventricular Hypertrophy in Children with Chronic Kidney Disease: Data from the Know-Ped CKD Study

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Key Words
Chronic kidney disease • Left-ventricular hypertrophy • Children

Abstract
Background/Aims: Children with chronic kidney disease (CKD) have a high risk of cardiovascular disease. Left-ventricular (LV) hypertrophy (LVH) is an early marker of cardiovascular disease in pediatric CKD, and the prevalence of LVH in pediatric CKD is approximately 20-30% in pre-dialysis CKD patients. However, there is no consensus on the ideal method of defining LVH in pediatric CKD patients. Previous studies have typically used the LV mass index (LVMI), which is calculated as LV mass in grams divided by height in meters to the 2.7th power ≥ 38 g/m$^{2.7}$, to diagnose LVH in children with CKD. Recently, age-specific reference values for LVMI ≥ 95th percentile and LV wall-thickness *z*-score > 1.64 in children were addressed. The aim of this study was to assess the prevalence and contributing factors of LVH in pediatric CKD patients according to each measurement and evaluate the concordance between each measurement.
Methods: We used the baseline data of the KoreaN cohort study for Outcome in patients With Pediatric Chronic Kidney Disease (KNOW-Ped CKD), which is a nationwide, 10-year, prospective,

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observational cohort study of pediatric CKD. A total of 469 patients were enrolled, and 458 patients were included in the final analysis. Univariate and multiple logistic regression analysis were performed to evaluate the association of the variables with LVH. Kappa statistics were used to analyze the concordance. ***Results:*** According to an LVH diagnosis of LVMI ≥ 38 g/m$^{2.7}$, 188 patients (41.0%) were diagnosed with LVH, and the prevalence of LVH was high in younger patients (< 2 years of age). Using the age-specific reference values, 116 patients (25.3%) were diagnosed with LVH, and there was no difference in the prevalence of LVH according to age. Thirty-one patients (6.8%) were diagnosed with LVH using an LV wall-thickness *z*-score > 1.64. There is poor concordance between the diagnosis of LVH using the LV wall-thickness *z*-score and the LVMI method. ***Conclusions:*** The results of this study show that there is poor concordance between the diagnosis of LVH using the wall-thickness *z*-score and the LVMI$^{2.7}$ criteria. Further investigation is needed to estimate the correlation between LVH and cardiac dysfunction and to find a better method for defining LVH in the pediatric CKD cohort and thereby predicting cardiac dysfunction.

Introduction

Children with chronic kidney disease (CKD) are at a high risk of cardiovascular disease, and it was previously reported that cardiovascular disease accounts for 23% of deaths in pediatric and young adult end-stage renal disease patients [1]. The lifespan of children on dialysis was estimated to be shortened by 50 years compared with the control group [2]. The risk factors for cardiovascular disease in CKD include fluid retention, anemia, hypertension, dysregulated metabolism of calcium and phosphorus, and chronic inflammation [2].

Left-ventricular (LV) hypertrophy (LVH) is the most common cardiac alteration and an early marker of cardiovascular disease in pediatric CKD [3]. It has been reported that LVH and diastolic dysfunction are present in early-stage pediatric CKD [4, 5]. The prevalence of LVH is approximately 20-30% in pre-dialysis pediatric CKD patients [2, 3]. In dialysis patients, LVH is usually correlated with hypertension and volume overload, and one report stated that the prevalence is as high as 85% in children on dialysis [3]. Recent reports suggest that effective renal replacement therapy, such as kidney transplantation and peritoneal dialysis, could improve LVH in patients with end-stage renal disease [6, 7].

In general, a diagnosis of LVH is made using echocardiography on the basis of the LV mass index (LVMI) to normalize LV mass to body size, and there are a variety of methods to define LVMI that have been used in children and adults. In adults, LVMI is usually calculated by dividing LV mass (in grams) by body surface area [8]. LV mass increases in a cubic exponential relationship with body height in healthy children, and the recommended calculation for LVMI in children is usually acquired by dividing LV mass by height in meters raised to the allometric power of 2.7 [9]. However, there is no consensus on the ideal method of defining LVH in pediatric CKD patients. Previous studies in CKD have typically used LVMI ≥ 38.6 g/m$^{2.7}$ to diagnose LVH because the cut-off of 38.6 g/m$^{2.7}$ represents the 95th percentile of the healthy pediatric population [10, 11]. One report recommended that an LVMI of 51 g/m$^{2.7}$ should be used for the diagnosis of LVH because this cut-off is associated with cardiovascular morbidity in patients with hypertension [12]. Recently, age-specific reference values for LVMI ≥ 95th percentile were addressed, and it was recommended that LV mass/height$^{2.7}$ should be compared with percentile curves for patients aged < 9 years [13]. However, there is a question as to whether LVMI (g/m$^{2.7}$) accurately represents the relationship between LV mass and body size throughout a child's growth. A novel method of expressing LV mass relative to body size in children was reported in which LV mass-for-height centile curves were proposed to be superior to LVMI (g/m$^{2.7}$) as a method of normalizing LV mass to body size in children [14]. According to this novel method, those with an LV mass above the 95th percentile for height (i.e., LV wall-thickness *z*-score > 1.64) were classified as

having LVH [14]. Recently, a report analyzed the concordance of the LVMI ($g/m^{2.7}$) and the LV mass *z*-score in the diagnosis of LVH in children with hypertension [15]. However, there was little data analyzing the concordance of the methods to define LVH in children with CKD.

The aim of this study was to assess the prevalence and contributing factors of LVH in pediatric CKD patients according to each measurement and evaluate the concordance between each measurement in the diagnosis of LVH in a pediatric CKD cohort.

Materials and Methods

Study design and population

We used the baseline data of the KoreaN cohort study for Outcome in patients With Pediatric Chronic Kidney Disease (KNOW-Ped CKD), a nationwide, 10-year, prospective, observational cohort study of pediatric CKD, including CKD stage 1-5 pediatric patients, from April 2011 to February 2016. Seven major pediatric nephrology centers in Korea have participated in the study and enrolled 469 children with CKD for the comprehensive assessment of the clinical findings. The detailed design and methods of the KNOW-Ped CKD were published [16]. Detailed information was also previously described elsewhere (NCT02165878 at http://www.clinicaltrials.gov). This study protocol was approved by the institutional review board at each participating clinical center in 2011. All of the participants provided written informed consent encompassing the study's purpose, duration, procedures, alternatives, risks, benefits, and subject's rights to withdraw from the study at any time. Among the 469 patients initially included, 11 children were excluded because of the violation of the inclusion criteria, withdrawal of consent or a transfer to another center not participating in the study; a total of 458 patients were included in the final analysis.

Ethical statement

All of the data were obtained in accordance with the ethical principles for medical research involving human subjects established in the Helsinki Declaration of 1975 and revised in 2000.

Clinical and laboratory measurements

Data on medical history, medication use, socio-economic status, and quality of life were collected through a self-administered questionnaire. The definition of co-morbidity included heart failure, arrhythmia, urinary tract infection, diabetes, retinopathy, sensorineural hearing loss, developmental delay, and metabolic syndrome. The biochemical values were measured at the hospital laboratory of each participating center, and serum samples were sent to the central laboratory (Lab Genomics, Korea) for measurement of creatinine, intact parathyroid hormone (iPTH), and 25-OH vitamin D. CKD was defined and staged according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria [17]. The estimated glomerular filtration rate (eGFR) was calculated using the bedside CKiD formula [18].

Blood pressure (BP) was measured using a mercury sphygmomanometer after the subject had rested for 5 min in a sitting position. All BP measurements were taken on the right arm three times with a cuff appropriate for arm circumference. The average value of the second and third measurements of systolic BP and diastolic BP were used for subsequent analyses and based on a decision for hypertension. The definition of hypertension was when the average value of the systolic and/or diastolic BP measurements were ≥ 95th percentile for age, gender, and height [19].

A chest X-ray and twelve-lead electrocardiogram were performed. Cardiomegaly is present when the cardiothoracic ratio is more than 55% on the chest X-ray, and the patient with cardiomegaly was regarded as LVH according to the chest X-ray. The electrocardiogram was interpreted at each center on the basis of published normal electrocardiogram standards for infants and children by Davignon et al [20].

Two-dimensional echocardiography was performed by pediatric cardiologists in each center to measure cardiac parameters according to the American Society of Echocardiography (ASE) pediatric guidelines [21]. LV mass was calculated from the M-mode measurements of the interventricular septum (IVS), LV inner dimension (LVID), and LV posterior wall thickness (LVPW) using the Devereux formula: $0.8 \times \{1.04[(IVS+LVID+LVPW)^3-(LVID)^3]\}+0.6$ g [22, 23]. The relative wall thickness (RWT) was calculated to assess the LV geometry using the following formula: (IVS +LVPW)/(LVID). The cut-off value for RWT

was 0.42 [24]. Based on LVMI and RWT, the LV geometry was categorized as either concentric hypertrophy (LVMI ≥ 38 $m^{2.7}$ and RWT > 0.42), eccentric hypertrophy (LVMI ≥ 38 $m^{2.7}$ and RWT ≤ 0.42), concentric remodeling (LVMI < 38 $m^{2.7}$ and RWT > 0.42), or normal (LVMI < 38 $m^{2.7}$ and RWT ≤ 0.42).

Baseline demographics and clinical and laboratory values were extracted from the web-based data management system, PhactaX (version 1.0), with the assistance of the Division of Data Management of the Seoul National University Medical Research Collaborating Center.

Statistical analysis

Continuous variables are expressed as the mean ± standard deviation or median (interquartile range). Continuous variables are compared between groups using the independent *t*-test, Kruskal-Wallis test and the Wilcoxon rank-sum test. Categorical variables are expressed as percentages and compared between groups with the Chi-square test and Fisher's exact test. The logistic regression analysis was used to determine the odds ratio (OR) and confidence interval (CI) for the presence of LVH associated with each variable. Kappa statistics were used to analyze the concordance. Concordance was graded using 0 to indicate no concordance and 1 to represent complete concordance. Statistical analyses were performed using SPSS software, version 19.0 (SPSS Inc.).

Table 1. Demographic and clinical data in the entire patient cohort

Variables	Total patient cohort (n = 458)
Age (years)	9.9±5.5
Sex (male/female)	310/148
Causes of chronic kidney disease	
Primary glomerulopathy	66 (14.4)
Secondary glomerulopathy	64 (14.0)
Reflux nephropathy	70 (15.3)
Renal dysplasia/hypoplasia	187 (40.8)
Others	71 (15.5)
Chronic kidney disease stage	
I	71 (15.5)
II	123 (26.9)
III	146 (31.9)
IV	89 (19.4)
V	29 (6.3)
Duration of primary disease (years)	5.8±4.8
Body mass index *z*-score	-0.4±1.4
Hemoglobin (g/dL)	12.2±2.0
Serum Ca (mg/dL)	9.3±0.7
Serum P (mg/dL)	4.8±1.0
Intact parathyroid hormone (pg/mL)	101.2±157.5
25 (OH) vitamin D (ng/mL)	21.5±10.8
Left ventricular mass index (g/$m^{2.7}$)	39.2±17.7
Ejection fraction (%)	66.9±7.3
Fractional shortening (%)	36.8±5.0
Patients receiving antihypertensive medications	148 (32.5)

Data are presented as the mean ± standard deviation given in the parentheses or as the number of patients with the percentage given in the parentheses

Results

Baseline characteristics of subjects

Demographic and clinical data for the 458 patients are listed in Table 1. The most common cause of CKD was congenital anomalies of the kidney and urinary tract (40.8%), and 66 children (14.5%) had primary glomerulonephritis.

Based on the heart to lung ratio from the chest X-ray, 86 patients (18.8%) were diagnosed with LVH, which was associated with a younger age (< 2 years of age, $P < 0.0001$), low body weight ($P = 0.0002$), low gestational age ($P < 0.0001$), and low birth weight ($P = 0.0014$) by univariate logistic regression. Although 17 patients (53.1%) were diagnosed with LVH in the patients below 2 years, 36 patients (11.1%) were compatible with LVH according to chest the X-ray in those above 2 years. An electrocardiogram revealed that 38 patients (8.3%) had LVH, which was associated with systolic hypertension ($P = 0.0334$) and short stature ($P = 0.0374$) by univariate logistic regression. However, there was no concordance between the diagnosis of LVH by chest X-ray and electrocardiogram. The Kappa coefficient showed a relatively low concordance value of 0.2351 between the chest X-ray and the electrocardiogram.

A total of 148 patients (32.5%) received antihypertensive medications, and there was a significant difference in the proportion of patients with the antihypertensive medications according to age (Table 2). The levels of LVMI were higher in younger patients (especially, < 2 years of age) compared to older children (Table 2). Eccentric hypertrophy was found in

Table 2. Hypertension and left ventricular mass according to age

	Total (n = 458)	0-2 years (n = 43)	2-6 years (n = 77)	6-12 years (n = 125)	12-20 years (n = 213)	*P*-value
Number of patients receiving antihypertensive medications (%)	148 (32.5)	8 (18.6)	13 (16.9)	40 (32.0)	87 (41.2)	< 0.001[a]
LVMI (g/m$^{2.7}$)	39.2±9.3	60.3±0.32	46.3±9.3	37.7±8.4	33.5±7.4	< 0.001[b]

LVMI, Left ventricular mass index, Data are presented as the mean ± standard deviation in the parentheses or as the number of patients with the percentage given in parentheses, [a]There was statistical significance by the Chi square test, [b]There was statistical significance by the Kruskal-Wallis test

Table 3. Associated factors of left ventricular hypertrophy with the definition of LVMI ≥ 38 g/m$^{2.7}$

	Univariate logistic regression		Multivariable logistic regression	
	Unadjusted OR(95% CI)	*P*-value	Adjusted OR(95% CI)	*P*-value
Age	0.863 (0.830, 0.897)	< 0.0001	0.880 (0.829, 0.935)	< 0.0001
BMI *z*-score	1.393 (1.177, 1.648)	0.0001	1.893 (1.432, 2.502)	< 0.0001
iPTH	1.002 (1.001, 1.004)	0.0028	1.004 (1.002, 1.006)	0.0005
Duration of primary disease	0.871 (0.831, 0.913)	< 0.0001	0.918 (0.862, 0.977)	0.0072

LVMI, Left ventricular mass index; OR, Odds ratio; CI, Confidence interval; BMI, Body mass index; iPTH, intact parathyroid hormone

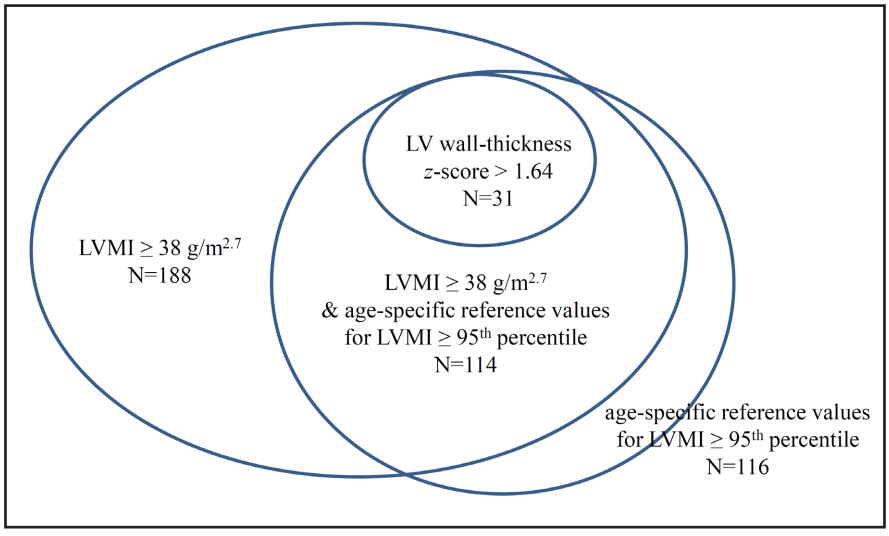

Fig. 1. Concordance of the measures of left-ventricular hypertrophy in children with chronic kidney disease. In the patients with LVH diagnosed by LVMI ≥ 38 g/m$^{2.7}$, 114 patients (60.6%) fulfill the criteria of LVH by age-specific reference ≥ 95 percentile. Among the 114 patients with LVMI ≥ 38 g/m$^{2.7}$ and age-specific reference ≥ 95 percentile, 31 patients (27.2%) were compatible with LVH by the LV wall thickness *z*-score > 1.64.

28.6% of the children, concentric hypertrophy in 12.2%, and concentric remodeling in 7.2%. Concentric hypertrophy was associated with systolic hypertension.

Prevalence and contributing factors of LVH

Based on an LVH diagnosis using LVMI ≥ 38 g/m$^{2.7}$, 188 patients (41.0%) were diagnosed with LVH, and the prevalence of LVH was higher in younger patients (especially < 2 years of age) compared to older children (P < 0.001). When LVH was diagnosed with LVMI ≥ 38 g/m$^{2.7}$, LVH was associated with younger age, a high level of the body mass index (BMI) *z*-score, high levels of iPTH, and the duration of primary disease by multivariable logistic regression (Table 3). Using the age-specific reference values for LVMI ≥ 95th percentile, 116 patients (25.3%) were diagnosed with LVH, and there was no difference in the prevalence of LVH according to age. Among the patients with LVMI ≥ 95th percentile, 114 patients (98%) were also compatible with LVH diagnosis using LVMI ≥ 38 g/m$^{2.7}$ (Figure 1). When LVH

Table 4. Associated factors of left ventricular hypertrophy with the definition of the age-specific reference values for LVMI ≥ 95th percentile

	Univariate logistic regression		Multivariable logistic regression	
	Unadjusted OR(95% CI)	*P*-value	Adjusted OR(95% CI)	*P*-value
Systolic hypertension	3.318 (1.872, 5.881)	< 0.0001	2.805 (1.368, 5.751)	0.0049
Anemia	1.939 (1.266, 2.970)	0.0023	2.178 (1.216, 3.902)	0.0089
BMI *z*-score	1.359 (1.127, 1.638)	0.0013	1.339 (1.082, 1.656)	0.0073
iPTH	1.002 (1.001, 1.004)	0.0007	1.003 (1.001, 1.005)	0.0034
Duration of primary disease	0.899 (0.853, 0.948)	< 0.0001	0.906 (0.849, 0.967)	0.0029

LVMI, Left ventricular mass index; OR, Odds ratio; CI, Confidence interval; BMI, Body mass index; iPTH, intact parathyroid hormone

Table 5. Associated factors of left ventricular hypertrophy with the definition of LV wall-thickness *z*-score > 1.64

	Univariate logistic regression		Multivariable logistic regression	
	Unadjusted OR(95% CI)	*P*-value	Adjusted OR(95% CI)	*P*-value
Co-morbidity	0.256 (0.112, 0.585)	0.0012	0.035 (0.004, 0.277)	0.0015
Weight *z* score	1.592 (1.212, 2.090)	0.0008	3.602 (1.948, 6.660)	< 0.0001
Hemoglobin	0.639 (0.524, 0.778)	< 0.0001	0.447 (0.292, 0.686)	0.0002
Duration of primary disease	0.908 (0.826, 0.997)	0.0441	1.268 (1.063, 1.512)	0.0082
LVMI (g/m$^{2.7}$)	1.119 (1.084, 1.155)	< 0.0001	1.271 (1.156, 1.398)	< 0.0001

LV, Left ventricle; OR, Odds ratio; CI, Confidence interval; LVMI, Left ventricular mass index

Table 6. Concordance of the measures of left ventricular hypertrophy in children with chronic kidney disease

	Kappa (95% C.I)	*P*-value
LVMI ≥ 38 g/m$^{2.7}$ vs. age-specific reference ≥ 95 percentile	0.636(0.566, 0.706)	<0.0001
LVMI ≥ 38 g/m$^{2.7}$ vs. LV wall thickness *z*-score > 1.64	0.189(0.129, 0.249)	<0.0001
Age-specific reference ≥ 95 percentile vs. LV wall thickness *z*-score > 1.64	0.353(0.258, 0.447)	<0.0001

CI, Confidence interval; LVMI, Left ventricular mass index; LV, Left ventricle

was diagnosed with the age-specific reference values for LVMI ≥ 95th percentile, LVH was associated with systolic hypertension, anemia, high levels of BMI *z*-score, high levels of iPTH, and the duration of primary disease by multivariable logistic regression (Table 4). Thirty-one patients (6.8%) were diagnosed with LVH using the LV wall-thickness *z*-score > 1.64. The patients with LVH using a *z*-score > 1.64 were compatible with LVMI ≥ 38 g/m$^{2.7}$ and age-specific reference values for LVMI ≥ 95th percentile. When using a *z*-score > 1.64, LVH was associated with the presence of co-morbidity, high levels of the body weight *z*-score, low levels of hemoglobin, and the duration of primary disease (Table 5). Using the age-specific reference values for LVMI ≥ 95th percentile, the Kappa coefficient was 0.636 and 0.353 with the LVMI ≥ 38 g/m$^{2.7}$ method and the wall thickness *z*-score method, respectively. However, the Kappa coefficient showed a relatively low concordance value of 0.189 between the LVMI ≥ 38 g/m$^{2.7}$ method and the wall thickness *z*-score method. This finding suggests that there is a poor concordance between the diagnosis of LVH using the LV wall-thickness *z*-score and the LVMI$^{2.7}$ method (Table 6).

Discussion

The results of this study show that there is poor concordance between the diagnosis of LVH using the wall-thickness *z*-score and the LVMI$^{2.7}$ criteria. Recently, pediatric data for the prevalence of LVH and risk factors in CKD have been reported, and the definition

of LVH in children with CKD varies across different studies. Although a study reported that the method for indexing LV mass by height^3 is useful for determining the presence of LVH in children and adolescents, LV mass/height$^{2.7}$ is usually regarded as an appropriate method for indexing LV mass for body size among the indexes based on height in children [8, 9, 14]. In the general population, LVH is commonly defined as LVMI above 38 g/m$^{2.7}$ and corresponds to the 95th percentile of the pediatric population. However, a recent report suggested that LV mass/height$^{2.7}$ should be compared with percentile curves in younger children because LVMI varies with age [13]. According to that study, the 95th percentile of LVMI ranged from 80 g/m$^{2.7}$ for newborns to 40 g/m$^{2.7}$ for 11-year-old children [13]. In our study, the prevalence of LVH was 41.0% and especially high in younger children below the age of 2 years when using the definition of LVMI ≥ 38 g/m$^{2.7}$, and these findings suggest that there is a risk of misdiagnosing younger healthy children as LVH if the cut-off of LVMI ≥ 38 g/m$^{2.7}$ is used for all patients from neonates to adolescents. When we define the LVH as above the 95th percentile, the prevalence of LVH was 25.3% and there was no difference in the prevalence of LVH according to age. This result was compatible with previous studies in pediatric CKD and defining LVH using percentile values could be a better method in the study of pediatric CKD patients. However, there remains some controversy as to the optimal method for indexing LV mass by body size and the cut-off value of LVH in children.

A recent report stated that LVMI (g/m$^{2.7}$) showed a strong correlation with height in children < 140 cm tall, which suggests that LVMI does not work reasonably well in those cases [14]. However, there was no significant relationship between LVMI and height in children ≥140 cm tall [14]. According to this finding, Foster et al. suggested a novel method using LV mass-for-height centile curves that describe the distribution of LV mass relative to height among healthy, non-obese children, and, using this method, patients with an LV mass above the 95th percentile for height (*z*-score > 1.64) were classified as having LVH [14]. Using this novel method, the prevalence of LVH in this study was 6.8%, and there was no concordance between LVMI and the LV mass-for-height centile method. Considering that the cohort included a high proportion of patients < 140 cm tall, there is a possibility that the centile method could reflect the prevalence and associated factors of LVH in pediatric CKD. Additionally, LVH defined by the novel method of LV mass above the 95th percentile for height was associated with co-morbidity, and there is a possibility that this novel method could predict morbidity in pediatric CKD patients.

In previous studies that assessed LV geometry in children with CKD, concentric LVH appeared more frequently in pre-dialysis patients. Mitsnefes et al. reported that eccentric LVH was more common in children on dialysis [4]. In our study, eccentric LVH was more common in pre-dialysis children, which could be due to a few reasons. Usually, eccentric remodeling is thought to be caused by an adaptation to preloading, such as fluid retention, and anemia. There is a possibility that more effective interventions for volume overload and renal anemia are needed in our patients. Another explanation is that the proportion of LV geometry may be associated with the cut-off used to define LVH. In our study, the cut-off of LVMI ≥ 38 g/m$^{2.7}$ was used, and the other definitions of LVH can lead to a different proportion of LVH geometry.

In our study, the common risk factors for LVH included the BMI *z*-score, levels of iPTH, anemia, and the duration of primary disease, and these findings are compatible with previous studies. The ESCAPE Trial Group reported that the prevalence of LVH in children with CKD is related to male gender, anemia, and high BMI [10]. Usually, hypertension has been a minor contributor to LVH in early CKD, and both our study and the ESCAPE Trial are suggestive of this finding. However, there was a few studies that hypertension may be a major contributor [25, 26]. Mitsnefes et al. reported that LVH was more common in children with either confirmed or masked hypertension compared with those with normal BP, and casual BP measurements alone are insufficient to predict LVH [25]. In our study, the lack of data for masked hypertension could influence the results, and the future study of the prevalence of masked hypertension based on ambulatory BP monitoring and its association

with LVH is necessary. Additionally, a study by Kupferman et al. suggested that BP control could cause LVH regression in children with CKD [26]. In our study, renal anemia and the long duration of the disease could especially play a role in the high proportion of eccentric LVH. A previous report stated that increased cardiac output might predispose pediatric CKD patients to LVH, and no association with PTH or anemia was observed [27]. A longitudinal study, including the association between BP control and LVH change, and the risk factors and morbidity associated with LVH based on the data from the KNOW-Ped CKD should be conducted in the future.

This study had several limitations. First, there is lack of ambulatory blood pressure monitoring, and the data for masked hypertension might be insufficient. Second, two-dimensional echocardiography was performed by pediatric cardiologists in each center and there was no central reading of the echocardiography. The lack of assessment by a single investigator blinded to medical therapy could influence the validity of the echocardiography data.

There has been little study of the concordance of the different LVH definition in children with CKD [28-30]. Simpson et al. reported that 5 different methods of LVM indexation influenced the categorization of pediatric CKD patients with respect to LVH [28]. Ruebner et al. suggested that indexing LVM to estimated lean body mass could be an alternative to height indexing in pediatric CKD patients because indexing LVM to height might cause a sex difference in the proportion of LVH without the increased prevalence of cardiovascular disease risk factors [29]. However, there is no data as to which method could predict cardiovascular events in pediatric CKD patients. It is important to accurately diagnose LVH in pediatric CKD patients because LVH is an early marker of cardiac dysfunction, and appropriate intervention can prevent cardiac deterioration.

Conclusion

Further study is necessary to estimate the correlation between LVH and cardiac dysfunction and to find a better method of defining LVH in the pediatric CKD cohort, thereby predict cardiac dysfunction.

Disclosure Statement

None of the authors have conflicts of interest to disclose.

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