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Left Ventricular Hypertrophy and Cardiac Troponin I in Pediatric Hemodialysis

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Left ventricular hypertrophy (LVH) is associated with cardiovascular morbidity and mortality in hemodialysis patients. Cardiac troponins (cTn) were proposed as markers of cardiac damage, but their value is still debated in hemodialysis patients. Objective: To assess LVH and cTnI in Egyptian pediatric patients on regular hemodialysis and their relationship to dialysis-related hypotension episodes and mortality over a period of one year. Patients and Methods: This study included 30 children on regular hemodialysis. Patients were subjected to 2-D, M-mode Doppler echocardiography with calculation of the left ventricular mass index (LVMI). Serum cTnI was assessed using an immunoassay test. Patients were clinically followed up for one year. Results: LVH (as measured by LVMI) was detected in 66.7 % of patients. Concentric hypertrophy was present in 43.3 % and eccentric hypertrophy in 23.3 % of patients. Although serum cTnI was < 1 ng/ml hypotension episodes occurred in 13.3 % of patients and mortality occurred in 10 % of them. The positive predictive value of LVMI to mortality was 28.6 %; negative predictive value was 95.7 % with 81.5 % specificity and 66.7 % sensitivity. Low fractional shortening (FS) was an excellent predictor of mortality with a positive predictive value of 100 %; the negative predictive value was 96.4 % with 100 % specificity and 66.7 % sensitivity. Conclusion: Hemodialysis pediatric patients had cardiovascular risk factors since LVH was highly prevalent in them. Low FS and increased LVMI remain relatively good predictors of mortality in those patients. Normal cTnI does not preclude cardiovascular risk in hemodialysis pediatric patients and is not a predictor for short-term prognosis (1-year follow-up). ***J Clin Basic Cardiol 2009; 12 (online): 5–10.***

Key words: *hemodialysis, cardiac troponin I, left ventricular hypertrophy, left ventricular mass index.*

Patients on hemodialysis have a high risk for cardiovascular morbidity and mortality [1, 2]. Left ventricular hypertrophy (LVH) and left ventricular systolic dysfunction are highly prevalent and are associated with poor cardiovascular outcomes [3].

Echocardiographic studies have shown that young dialysis patients have abnormalities of both the left ventricular structure and function [4]. LVH in children and adolescents with chronic kidney disease (CKD) is adaptive to improve contractility [5].

The percentage of deaths from a cardiovascular sequel is as common in children with CKD as in adults [3, 6].

Although hemodialysis can reduce injury caused by uremic toxins, it may also increase cardiotoxicity of other factors. The cardio-depressant effects of uremic markers have been described in numerous studies [6–8]. In addition, creation of a hemodialysis arteriovenous fistula is independently associated with further progression of already existing LVH [9].

Increased left ventricular mass index (LVMI) in patients with CKD and on hemodialysis may be the consequence of the coexistence of several abnormalities (fluid overload, hypertension, anemia, etc) [10].

Cardiac troponins (cTn) have been proposed to be markers of cardiac damage, but their value is still debated in hemodialysis patients. They have a complex interrelationship with the disease pathophysiology in patients with renal dysfunction and ESRD [11, 12].

Increased serum cTnT and cTnI were found in adult chronic hemodialysis patients; yet cTnI is elevated less often than cTnT. The basis of these cardiac troponin elevations is unclear. These findings may represent a subclinical myocardial injury, an inflammatory response to CKD or a chronic volume-overloaded state [10, 13, 14].

However, data about the use of cardiac biochemical markers in Egyptian children at risk for cardiovascular damage are lacking.

We aimed to assess LVH and cTnI in Egyptian pediatric patients on regular hemodialysis and their relationship to dialysis-related hypotension episodes and mortality over a period of one year.

Patients and Methods

This study included 30 Egyptian children with CKD on regular hemodialysis at the Center of Pediatric Nephrology and Transplantation, Pediatric Hospital, Cairo University. They were 20 males and 10 females with a mean age of 10.3 ± 3.04 years. Patients were subjected to full medical history taking, family history of renal and cardiac diseases and a thorough clinical examination including weight, height, and blood pressure with calculation of Body Mass Index (BMI). No patient had diabetes, heart failure and none reported smoking or illegal drug use. All children were on regular hemodialysis 3×/week using polysulfone dialyzers, bicarbonate-based dialysate, and controlled ultrafiltration. Dialysis adequacy was assessed by Kt/V, a standard index of dialysis efficiency. The National Kidney Foundation-Dialysis Outcome Quality Initiative Recommendations [15] recommend a Kt/V of at least 1.2. Informed consent was obtained from the parents of the participating children.

Electrocardiography (ECG)

Standard 12-lead ECGs were recorded in all subjects at 25 mm/s and 1-mV/cm calibration. QT interval was calculated and corrected for heart rate using Bazett's formula (QTc=QT/R-R interval).

Echocardiographic Evaluation

Single echocardiographic evaluation was performed before the hemodialysis session. Transthoracic two-dimensionally (2D) guided (M-mode) Doppler echocardiograms were performed with a Hewlett-Packard 5500 SONOS ultrasonic

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machine phased array sector scanner with the 4 and 8 MHz probes according to age. Linear measurements of the LV cavity were obtained. Left ventricle end diastolic diameter (LVEDD), left ventricle end systolic diameter (LVESD), walls (interventricular septum [IVS] and posterior wall [PW]) and calculation of fractional shortening (FS %) as an indicator of LV systolic function were done according to the recommendations of the American Society of Echocardiography. FS value < 28 % was considered lower than normal with impaired LV systolic function [16]. LVM was estimated using the anatomically validated formula of Devereux et al [17]:

$$LVM = 0.80 \times \{1.04 \times [(\text{septal thickness} + \text{LV internal diameter} + \text{posterior wall thickness})^3 - (\text{LV internal diameter})^3]\} + 0.6\ g$$

The LVMI normalizing LVM for patient size was obtained as: LVM/height$^{2.7}$ [18]. LVH was defined as LVMI > 51 g/m$^{2.7}$; a value greater than the pediatric 99th percentile. This percentile is associated with a 4.1-fold risk of cardiovascular morbidity in hypertensive adults [18]. Zoccali et al [19] showed that normalization of LVM for height in m$^{2.7}$ is significantly more predictive of cardiovascular outcome in patients with ESRD than normalization for body surface area, a measure of body size influenced by body weight, which fluctuates in dialysis patients.

The relative wall thickness (RWT) was calculated as 2PW/LVEDD.

Four LV geometric patterns were identified using values of RWT and LVM as follows: normal (no LVH and RWT < 0.44), concentric remodeling (no LVH and RWT > 0.44), concentric hypertrophy (LVH and RWT > 0.44), and eccentric hypertrophy (LVH and RWT < 0.44) [20].

Samples from Patients

Venous blood samples (4 ml) were collected from patients on plain tubes, immediately before the dialysis procedure, centrifuged and serum was separated and stored at –20 °C until analysis.

Laboratory Investigations

Complete blood count, liver and kidney functions, serum calcium, phosphorous, alkaline phosphatase, sodium, potassium, bicarbonate, pH, and ferritin, in addition to cardiac troponin I (cTnI) were done.

Determination of Cardiac Troponin I

Determination of troponin I and its complex troponin I/T/C in serum was done using a one-step serum immunoassay test, Troponin Band™, Cal-Tech Diagnostic Inc. Chino, California, USA. The test employs highly specific antibodies for troponin I and its complex troponin I/T/C in a sandwich immunoassay system and immunochromatographic detection assay [21].

Statistical Analysis

Data were analyzed using the Statistical Package for Social Science (SPSS) version 14 and Microsoft Excel 2003. Descriptive data were expressed as frequency distributions and for numerical data, mean ± SD. Means were compared using the T test and qualitative data using Chi's square test. Pearson's correlations were used for numerical values. P < 0.05 was considered significant.

Results

Table 1 summarizes clinical, dialysis, laboratory data as well as ECG and echocardiographic data of our group of children on regular hemodialysis.

Table 1. Data of chronic hemodialysis patients

Clinical, dialysis and laboratory data		Echocardiographic and electrocardiographic data	
Items	**Patients (n = 30)**	**Items**	**Patients (n = 30)**
Age (years)	10.3 ± 3.04	HR (beats/minute)	110.78 ± 18.42
Weight (kg)	23.65 ± 7.21	Ao (cm)	2.18 ± 0.37
Height (cm)	121.72 ± 15.20	LA (cm)	2.60 ± 0.41
Body Mass Index (kg/m^2)	15.99 ± 4.02	PA (cm)	2.0 ± 0.34
Dialysis duration (years)	1.47 ± 1.03	IVS (cm)	1.05 ± 0.25
Dialysis session duration (hours)	3 ± 0.26	LVPW (cm)	0.93 ± 0.20
Dialysis flow (ml/m^2 surface area)	158.46 ± 20.33	LVEDD (cm)	3.87 ± 0.74
*Kt/V	1.41 ± 0.28	LVESD (cm)	2.47 ± 0.59
MBP (mmHg)	97.24 ± 11.54	FS (%)	36.45 ± 7.28
MBP post-dialysis (mmHg)	80.69 ± 7.64	LVM (gm)	124.5 ± 54.3
Blood urea nitrogen (mg/dl)	73.17 ±17.52	LVMI (g/m$^{2.7}$)	76.28 ± 38.09
Creatinine (mg/dl)	7.34 ± 1.67	RWT	0.50 ± 0.15
Calcium (mg/dl)	9.38 ± 1.15	PR interval (sec)	0.14 ± 0.02
Phosphorus (mg/dl)	6.72 ± 2.05	QT interval (sec)	0.29 ± 0.04
Calcium-phosphorus product	62.93 ± 20.28	QTc	0.39 ± 0.04
Alkaline phosphatase (U/l)	377.52 ± 229.47	Prolonged QTc (n & %)	3 (10 %)
Sodium (mmol/l)	134.88 ± 4.69	RAD (n & %)	3 (10 %)
Potassium (mmol/l)	5.3 ± 1.40	LAD (n & %)	1 (3.3 %)
pH	7.26 ± 0.08	LVH (n & %)	1 (3.3 %)
Bicarbonate (mmol/l)	18.06 ± 2.92	LV strain (n & %)	2 (6.7 %)
Albumin (gm/dl)	4.27 ± 0.42	RBBB (n & %)	3 (10 %)
HB (gm/dl)	10.08 ± 1.78	Depressed ST (n & %)	1 (3.3 %)
Cardiac troponin I (ng/ml)	< 1 ng/ml	Elevated ST (n & %)	1 (3.3 %)

Data expressed as mean ± standard deviation, except for numbers between parentheses
MBP: mean blood pressure; Kt/V: standard index of dialysis efficiency; Ao: aorta diameter; HR: heart rate; LA: left atrial diameter; PA: pulmonary artery diameter; IVS: interventricular septum thickness in diastole; LVPW: left ventricular posterior wall thickness in diastole; LVEDD: left ventriclar end diastolic diameter; LVESD: left ventricular end systolic diameter; FS: fractional shortening; LVM: left ventricular mass; LVMI: left ventricular mass index; RWT: relative wall thickness; QTc: corrected QT interval; RAD: right atrial dilatation; LAD: left atrial dilatation; LVH: left ventricular hypertrophy; RBBB: right bundle branch block

The etiology of CKD in the studied patients was urinary tract disorders in 14 patients (46.7 %), congenital anomalies in 4 (13.3 %), glomerulonephritis in 2 (6.7 %) and non-identifiable cause in 10 patients (33.3 %).

Risk factors for cardiovascular disease included hypertension which was present in 23 (76.7 %) patients, including 13 (43.3 %) with uncontrolled hypertension and 10 (33.3 %) whose blood pressure was controlled using antihypertensive drugs. Out of the 30 patients 7 (23.3 %) were using one antihypertensive drug with a dose ≤ 1.5 mg/kg/day, 11 (36.7 %) were using higher doses or 2 antihypertensive drugs while 5 (16.7 %) were using more than 2 antihypertensive drugs.

Serum cTnI levels of the children included in the study were within normal boundaries (< 1 ng/ml).

Patients were classified into 2 groups according to LVMI: the first with LVH (LVMI > 51 g/m$^{2.7}$) and the second with normal LVM. Comparisons between both groups are illustrated in Tables 2 and 3.

Twenty patients were found to have LVH (increased LVMI), representing 66.7 % of the studied population. Concentric hypertrophy was present in 13 patients and eccentric hypertrophy was detected in 7 patients. Six cases had concentric remodeling. RWT was increased in 19 patients.

Additional echocardiographic findings were detected including pericardial effusion in one patient, mitral regurgitation (MR) in 5 patients (3 with trivial MR, 2 with mild MR), one patient had mild tricuspid regurgitation and another one tricuspid regurgitation and pulmonary hypertension. All these patients in addition to those who had prolonged QTc (3 patients), LV strain (3 patients) and depressed ST segment (one patient) were of the second group (LVH).

There was a statistically significant difference between both groups regarding pH and HCO_3 (p = 0.02 and p = 0.01, respectively) (Table 2). Also, a significant difference was found between both groups concerning LVPW (p = 0.02) (Table 3). A significantly larger proportion of patients with LVH were receiving more than 2 antihypertensives (p = 0.008). A positive correlation was found between fractional shortening (FS) and BMI (r = 0.35, p = 0.05).

A one-year clinical follow-up of the studied patients revealed that repeated hypotensive episodes during hemodialysis occurred in 4 cases (13.3 %), with no significant differences of LVMI, RWT, and FS between them and those without hypotensive episodes (p > 0.05). In contrast, patients who developed hypotensive episodes had a significantly longer mean QTc (p = 0.016). During the period of follow-up, 3 patients died (10 % of the studied group).

At a cut-off of 97 g/m$^{2.7}$, the positive predictive value of LVMI to mortality was 28.6 %; the negative predictive value was 95.7 % with 81.5 % specificity and 66.7 % sensitivity. Low FS was an excellent predictor of mortality in the studied group with a positive predictive value of 100 % and a negative predictive value of 96.4 % with 100 % specificity and 66.7 % sensitivity (Table 4).

Table 2. Comparison of clinical and laboratory data between patients with normal left ventricular mass and those with left ventricular hypertrophy

Items	Normal LVM (n = 10)	LVH (n = 20)	p
Age (years)	11.61 ± 3.77	9.7 ± 2.54	0.12
Body Mass Index (kg/m^2)	14.10 ± 2.68	16.94 ± 4.30	0.07
Duration of dialysis (years)	1.59 ± 1.07	1.42 ± 1.04	0.68
MBP (mmHg)	102.22 ± 7.95	95 ± 12.35	0.12
MBP post-dialysis (mmHg)	83.89 ± 8.21	79.25 ± 7.12	0.13
Dialysis flow (ml/m^2 surface area)	155.50 ± 21.24	159.86 ± 20.33	0.61
Kt/V	1.35 ± 0.13	1.45 ± 0.33	0.40
Blood urea nitrogen (mg/dl)	76.67 ± 22.08	71.6 ± 15.46	0.48
Creatinine (mg/dl)	7.67 ± 1.75	7.2 ± 1.66	0.50
Calcium (mg/dl)	9.55 ± 0.84	9.31 ± 1.28	0.61
Phosphorus (mg/dl)	7.1 ± 3.03	6.55 ± 1.49	0.51
Calcium-phosphorus product	67.45 ± 29.12	60.89 ± 15.34	0.43
Alkaline phosphatase (U/l)	345.22 ± 183.98	392.05 ± 250.26	0.62
Sodium (mmol/l)	135.45 ± 0.58	133.75 ± 10.25	0.72
Potassium (mmol/l)	5.19 ± 1.31	5.2 ± 1.37	0.92
pH	7.32 ± 0.07	7.23 ± 0.06	0.02*
Bicarbonate (mmol/l)	21.13 ± 3.58	16.84 ± 1.49	0.01*
Albumin (gm/dl)	4.22 ± 0.49	4.30 ± 0.39	0.65
HB (gm/dl)	9.89 ± 2.08	10.17 ± 1.68	0.71

Data expressed as mean ± standard deviation. *p significant if < 0.05. LVM: left ventricular mass; LVH: left ventricular hypertrophy; MBP: mean blood pressure; Kt/V: standard index of dialysis efficiency

Discussion

Morbidity and mortality rates in hemodialysis patients remain a problem. They are associated with an increase of cardiovascular risk factors [10]. Identifying modifiable risk factors in those patients may lead to clinical strategies to reduce morbidity and mortality.

In the current study, LVH (assessed by increased LVMI > 51 g/m$^{2.7}$) was detected in 66.7 % of children on chronic hemodialysis. This coincided with the findings of other investigators who stated that the increase in LVMI was prevalent in chronic hemodialysis patients [4, 22, 23]. Many studies reported that LVH was present in > 70 % of patients on hemodialysis [2, 4]. Also, it was found that LVH is the most frequent cardiac abnormality in patients with ESRD [24].

We found no significant correlation between LVMI and any of the clinical or laboratory data of the patients, which was in agreement with other investigators [5] who found that children undergoing chronic hemodialysis have increased LVMI with no relationship between LVMI, creatinine con-

Table 3. Comparison of electrocardiographic and echocardiographic data between patients with normal left ventricular mass and those with left ventricular hypertrophy

Items	Normal LVM (n = 10)	LVH (n= 20)	p
Age (years)	11.61 ± 3.77	9.7 ± 2.54	0.12
Aorta diameter (cm)	2.27 ± 0.44	2.14 ± 0.33	0.37
Left atrial diameter (cm)	2.69 ± 0.41	2.56 ± 0.41	0.42
Pulmonary artery diameter (cm)	1.96 ± 0.32	2.03 ± 0.36	0.60
Interventricular septum thickness in diastole (cm)	1.17 ± 0.29	0.99 ± 0.22	0.07
LVPW (cm)	1.04 ± 0.19	0.87 ± 0.18	0.02*
LVEDD (cm)	3.96 ± 0.92	3.83 ± 0.66	0.66
LVESD (cm)	2.62 ± 0.82	2.39 ± 0.44	0.32
Fractional shortening %	34.07 ± 8.01	37.64 ± 6.78	0.21
Relative wall thickness	0.56 ± 0.18	0.47 ± 0.13	0.14
PR interval (sec)	0.15 ± 0.02	0.14 ± 0.02	0.45
Corrected QT interval	0.40 ± 0.04	0.39 ± 0.03	0.54

Data expressed as mean ± standard deviation; *p significant if < 0.05; LVM: left ventricular mass; LVH: left ventricular hypertrophy; LVPW: left ventricular posterior wall thickness in diastole; LVEDD: left ventriclar end diastolic diameter; LVESD: left ventriclar end systolic diameter; LVMI: left ventricular mass index

Table 4. Mortality predictors in chronic hemodialysis patients

Items	Cut-off	Sensitivity (%)	Specificity (%)	Positive predictive value (%)	Negative predictive value (%	Accuracy (%)
LVMI	> 51	100	37	15	100	43.3
LVMI	> 97	66.7	81.5	28.6	95.7	80
FS	< 26	66.7	100	100	96.4	96.7

LVMI: left ventricular mass index; FS: fractional shortening.

centration, glomerular filtration rate, and blood pressure. It was recommended to control blood pressure, anemia, and hypervolemia which may be important in the prevention of or in improving LVH [4].

The present study revealed that 43.3 % of our patients had concentric hypertrophy, which was consistent with other investigators who found that the majority of patients with LVH showed concentric hypertrophy [25]. This could put our patients at a higher cardiovascular risk since previous investigators found that concentric LVH characterizes a more severe impairment of the cardiovascular system with more prevalence in non-survivors [26].

We also found that eccentric hypertrophy was present in 23 % of our patients. Previous researchers [27] reported that eccentric LVH was associated with a greater incidence of adverse cardiovascular events compared with concentric LVH which is the opposite of the previous opinion.

In the present study, there was no statistically significant correlation between blood pressure (BP) and LVMI, RWT or any of the clinical or laboratory data of the studied group. It is to be noted, however, that 76.7 % of patients in our study were receiving antihypertensive drugs, and that such treatment as well as the need for more than one drug were associated with a higher prevalence of LVH in our patients. Other investigators [28] found that BP was highly predictive of the LVMI in adult men for all BMI categories, and for overweight and obese females. However, other studies reported that normotensive hemodialysis patients, without significant pressure and volume overloads, still had increased LVM that was partially explained by the persistent flow overload and subclinical LV dysfunction [29].

We found that dialysis-related hypotension occurred in 13.3 % of the patients with no relation to LVH or RWT. Other investigators found that dialysis-related hypotension responded rapidly to emergency resuscitation but with diminished left ventricular function [30].

Slow dialysis sessions may be needed to prevent hypotension from occurring especially in the presence of concentric LV geometry [31].

We found that QTc was prolonged in our pediatric patients who had hemodialysis-related hypotensive episodes. This coincided with the results reported by other investigators [32] who mentioned that hemodialysis increases the QTc interval in ESRD patients which is mainly related to rapid changes in plasma electrolyte concentrations.

Cardiac troponins (cTn) have been proposed to be markers of cardiac damage, but their value is still debated in hemodialysis patients [12].

In the present study, we measured pre-dialysis cTnI in chronic hemodialysis children and found it < 1 ng/ml. Marjani et al [33] found that levels of cTnI were < 0.1 microg/l at predialysis compared with post-dialysis, whereas levels of cTnI significantly increased in 54.5 % of hemodialyzed patients at post-dialysis.

Previous investigators found that serum cTnI is not affected in patients with renal failure in pre/post-hemodialysis, and further added that its diagnostic utility in these patients is unreliable [34].

It was reported that only about 3–10 % of cTnI is released as free particles, and that cTnI is hydrophobic; therefore, it may bind with serum proteins or with the surface area of the dialyser. Also, several biochemical modifications of cTnI take place in the blood stream, including phosphorylation, oxidation, and proteolysis, potentially affecting its detection by immunoenzymatic methods [10]. This concept was confirmed by other investigators, who reported increased serum concentration of the cTnI-I complex in 39.5 % of patients treated with hemodialysis and the absence of free cTnI in all subjects [35].

Katerinis et al [36] reported that low-grade cTnI elevation only occurs in a very small percentage of adult hemodialysis patients having long-standing severe cardiac disease (> 70 years of age with heart failure, history of severe coronary artery disease, previous myocardial infarction, coronary stenting, and/or bypass).

We observed that, although cTnI was < 1 ng/ml in our patients, hypotension episodes and mortality occurred in some patients. Accordingly, we do agree with what was mentioned by other investigators [13] that normal cardiac troponin levels do not preclude cardiovascular risk and mortality, at least within a year.

It was reported that intradialytic hypotension was associated with elevated cTnI 44 for hours following hemodialysis and these cases were more likely to experience cardiovascular events or death [37]. Unfortunately, we did not measure cTnI post-dialysis.

Kanwar et al [38] reported that cardiac troponins (I and T) did not further predict the risk for death in adult chronic hemodialysis patients without a coronary disease equivalent. Also, other investigators found no significant predictable effect of cTnI on cardiac events after a 6-month follow-up in chronic hemodialysis adult patients [39]. Moreover, Helleskov Madsen et al [40] reported that the prognostic value of cTnT appears superior to cTnI in patients with ESRD.

Regarding prolonged studies in adult hemodialysis patients, Brunet et al [41] found that cTnI is not predictive of long-term mortality over 2.5 years while cTnT was predictive. They reported that the 2.5-year mortality was associated with increased levels of cTnT but not with increased levels of cTnI.

Furthermore, previous studies found no statistically significant correlations between cTnI and variables such as glomerular filtration rate, LVH, LVMI, inflammation, anemia or hyperphosphatemia in patients with CKD with or without hemodialysis [10].

Consequently, a normal cTnI does not exclude cardiovascular morbidity and mortality in pediatric patients on chronic hemodialysis, at least within a one-year follow-up.

The discrepancy of the results of cTnI concentrations observed in various studies of hemodialysis patients may be due to different categories of patients or the use of different diagnostic tests. Currently, there are many available tests to measure cTnI concentration, which vary in terms of the type of reagents and monoclonal antibodies used, various epitopes of cTnI recognized and cut-off values for the diagnosis [42]. Also, recently Lippi et al [43] reported that sampling time

and ultrafiltration coefficient of the hemodialysis membrane should be regarded as potential sources of variability in the clinical interpretation of troponin measurement in hemodialysis patients [43].

We found that although the mean value of Kt/V was within normal range in our group of hemodialysis patients a large percentage of them had developed LVH. This suggests that a Kt/V of 1.2 may not be sufficient to avoid myocardial injury or to serve as a predictor of cardiovascular morbidity and mortality for younger patients. Chazot et al [44] reported that although Kt/V targets are fulfilled in an increasing number of patients, observational studies show that individuals on hemodialysis continue to experience a high rate of complications, including LVH and death [44].

In the present study, we found that LVH was a sensitive but poorly specific short-term mortality predictor. Using a higher LVMI (97 g/m$^{2.7}$) as a cut-off value for specificity and overall accuracy increased with a modest compromise in sensitivity. Given the duration of our study (one year), specificity of LVH as a long-term mortality predictor may be much higher than we found. It was found that LVM is an independent predictor of cardiovascular events in patients undergoing dialysis [19]. Other follow-up studies revealed that an increase of LVMI was associated with a 62 % increase in the incident risk of fatal and non-fatal cardiovascular events [23].

We also found that FS < 28 % was an excellent predictor of mortality in our studied pediatric patients, which coincided with the findings of other investigators [13, 45]. It was suggested that nocturnal hemodialysis could improve FS in patients with impaired LV systolic function as well as LVM and selected measures of quality of life [46, 47].

This could be a point for further study as it identifies subclinical cardiac damage that could be treated to hopefully reduce cardiovascular morbidity and mortality in this high-risk population.

Conclusion

Hemodialysis is associated with accumulation of cardiovascular risk factors since LVH was highly prevalent in pediatric patients on hemodialysis. Low FS and increased LVMI remain relatively good indicators and predictors of mortality in chronic hemodialysis pediatric patients. LVMI > 51 g/m$^{2.7}$ is highly sensitive while > 97 g/m$^{2.7}$ is highly specific as a short-term mortality predictor.

Normal cTnI does not exclude cardiovascular risk in chronic pediatric hemodialysis patients.

We recommend early detection and treatment of the etiological causes of CKD with identification of patients at an increased risk for development of LVH and application of appropriate therapy which might be important in preventing LVH in those patients, or in regression of LVH and its deleterious consequences in hemodialysis children to improve survival and quality of life.

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