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ORIGINAL ARTICLE

Renee F. Robinson · Milap C. Nahata · Elizabeth Sparks · Curt Daniels · Donald L. Batisky · John R. Hayes · John D. Mahan

Abnormal left ventricular mass and aortic distensibility in pediatric dialysis patients

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Abstract There is ample evidence that the same pathophysiological processes that affect cardiovascular function in adults with end-stage renal disease (ESRD) also operate in children with ESRD. In adults undergoing hemodialysis (HD), a good correlation has been established between left ventricular mass (LVM) and aortic distensibility (AD) as markers of cardiovascular disease progression; however, this correlation has not been established in children. Therefore, in this retrospective study we investigated some aspects of cardiovascular damage (i.e., LVM, LVMI, and AD) in children with ESRD undergoing HD (n=9) or peritoneal dialysis (PD, n=9), and analyzed the relationship between AD, LVM, LVMI, pre-dialysis, post-dialysis blood pressure (BP), and demographic factors in children and adolescents with ESRD. Both LVM and AD were significantly greater in the dialysis population than in a control population derived from our institutional files (P=0.015, P=0.001). LVM and LVMI in children undergoing HD (92.9±83.7 g, 80.1±31.1 g/cm) were not statistically different from the values in children on PD (130.0±89.2 g, 89.6±35.9 g/cm), (P=0.3, P=0.5). AD in children on HD (2.2±0.55 cm^2 * $dynes^{-1*10-6}$) was significantly lower than in children on PD (2.7±0.54 cm^2 * $dynes^{-1*10-6}$), (P=0.01). The findings in this study confirm earlier studies that demonstrated that LVMI is greater in children on dialysis. This study also demonstrates that abnormal vascular stiffness, as defined by AD, is present in these children. The degree of vascular stiffness in children receiving HD is greater than in children receiving PD. However, further study is needed to address how control of BP, uremia, and other factors may affect these abnormalities in children with ESRD.

R. F. Robinson (✉) · M. C. Nahata · E. Sparks · C. Daniels · D. L. Batisky · J. R. Hayes · J. D. Mahan
Children's Research Institute,
700 Children's Drive, Columbus, OH 43205, USA
e-mail: Robinsonr@pediatrics.ohio-state.edu
Tel.: +1-614-7224367
Fax: +1-614-7226482

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Introduction

Cardiovascular (CV) disease remains the main cause of death in adults with end-stage renal disease (ESRD) and there is ample evidence that the same pathophysiological processes that affect adults with ESRD also operate in children with ESRD. Recently, Groothoff et al. [1] detailed the impact of CV disease in children with ESRD and noted that CV disease was the most frequent cause of death in this population. In the total population of children and adults undergoing dialysis, the age-adjusted death rate is 3.5 times greater for patients on chronic dialysis than in the general population [2]. CV events, including stroke, are the main cause of death in hemodialysis (HD) patients, accounting for between 41% and 54% of deaths among HD patients in the United States [3]. The pathogenesis for CV changes in patients with ESRD undergoing dialysis is related to the interplay of multiple factors [e.g., poor blood pressure (BP) control, persistent volume overload, oversecretion of parathyroid hormone, uremic toxins, anemia, and elevated cardiac output associated with arteriovenous shunt] [4, 5, 6].

A direct relationship between systolic BP (SBP), diastolic BP (DBP), and increased CV morbidity and mortality in adults is well established [6, 7, 8]. The pathogenesis of hypertension in patients with ESRD is not completely understood but is believed to be related to hypervolemia, altered catecholamine, vasopressin, and endothelin levels and diminished nitrous oxide activity. In dialysis patients, hypertension (HTN) is often not adequately controlled despite antihypertensive medications [9,10]. In the presence of HTN, myocardial and vascular damage occurs, as cell functions are altered and elastic properties of vessels and the heart are changed secondary to diminished arterial relaxation during diastole [4, 6, 11, 12, 13]. The severity and incidence of left ventricular hypertrophy (LVH), arterial stiffness, coronary artery

disease, congestive heart failure, cerebrovascular complications, atherosclerosis, and subsequent morbidity increase with HTN and lead to increased morbidity in patients with ESRD [11,14].

LVH and left ventricular mass index (LVMI) are well accepted measures of CV end organ damage (EOD) [8, 15, 16, 17, 18, 19]. LVH and LVMI are independent risk factors for CV disease and predictive of poor patient outcome in both adults and children with HTN and adults with chronic renal insufficiency [10, 16, 20, 21, 22, 23].

Recently, increased arterial stiffness has been identified as a measurable risk factor for mortality in adults with ESRD [24, 25, 26]. Coronary artery calcifications and reduced arterial wall distensibility have been documented in adolescents and young adults with ESRD [9, 24, 25, 26]. Aortic stiffness can be assessed directly by the measurement of aortic distensibility (AD) [27, 28]. In adults undergoing HD, a good correlation has been established between LVM and AD as markers of CV disease progression [29].

In this study we investigated the extent of CV damage (i.e., LVM, LVMI, and AD) in children with ESRD undergoing HD or peritoneal dialysis (PD), and analyzed the relationship between AD, LVM, LVMI, pre-dialysis and post-dialysis BP, and demographic factors in children and adolescents with ESRD.

Materials and methods

All patients with ESRD who were on dialysis (n=18, mean age 57±72 months, range 3–211 months) in June 2000 at the Children's Hospital, Columbus, Ohio were included in this retrospective, cohort study, which was approved by the independent investigational review board at the Children's Research Institute. All clinical, laboratory, and echocardiographic reports prior to June 2000 were obtained from patient medical records. Demographic data included age, gender, race, height, weight, and body surface area (BSA). Underlying primary diagnosis, date and age at diagnosis of ESRD, medications received, mode of dialysis (HD or PD), and duration of dialysis prior to echocardiogram were obtained. Standard laboratory parameters and BP measurements were obtained. SBP and DBP were measured with the appropriate-size cuff, from the right arm of the seated patient, by the dialysis nurse with the Dinamap 8100 during routine visits for PD patients and prior to and after dialysis in HD patients.

Doppler, two-dimensional and M-mode echocardiograms were performed on clinically stable, supine patients during routine clinic visits (on non-dialysis days for HD patients, while off dialysis for PD patients). Border detection allowed visualization of myocardial and valvar motion as well as anatomical substrates of flow abnormalities. M-mode echocardiograms were obtained using standard techniques with appropriate transducers for body size to obtain LVM. Recordings were reviewed by the study cardiologist to evaluate LVM, LVMI, and AD parameters. Measurements of interventricular septal thickness, posterior wall thickness, and the left ventricular internal dimension in end diastole were used to calculate LVM according to the American Society of Echocardiography and indexed to BSA [30, 31, 32]. LVMI was calculated as LVM divided by patient height to account for body size.

M-mode measurements of the ascending and descending aorta were used to determine AD according to the formula aortic distensibility=2(systolic diameter-diastolic diameter)/(pulse pressure)(diastolic diameter) and the stiffness index according to the formula stiffness index=natural log (SBP)/(DBP) (systolic diameter-diastolic diameter)/(diastolic diameter). For the purposes of this study, LVH and AD were compared with age-, gender-, and BSA-matched control data from normal children studied in our institution.

Descriptive statistics are presented using the chi-squared test. Frequency distribution and data are expressed as mean±SD. A two-sample t -test was used to compare means and standard deviations of continuous variables on type of dialysis (HD versus PD) and categorical variables were compared using the chi-squared test. Pearson correlation and coefficient of determination (r^2) were utilized to assess strength and direction of the relationship between the three continuous variables (LVM, LVMI, and AD) of the HD and PD patients and to determine if the extent of variability in LVM and LVMI was explained by AD. A one-sample t-test was utilized to compare the sample LVM, LVMI, and AD for PD and HD patients with normal pediatric values from our institution.

Average monthly pre-dialysis and post-dialysis casual BP measurements and mean arterial pressures (MAP) were compiled for the duration of dialysis for up to 3 years prior to the echocardiogram in children on long-term dialysis. Actual values and standard BP categories were used to establish the proportion of BP measurements considered to be elevated [33]. The relationship between BP measurements (i.e., average pre-dialysis SBP, average pre-dialysis DBP, average post-dialysis SBP, average post-DBP, pre-dialysis MAP, post-dialysis MAP, and the proportion of elevated pre- and post-dialysis SBP and DBP measurements) and subsequent LVM, LVMI, and AD were assessed by Spearman's rho. The relationships of the independent risk factors (i.e., HTN, age, duration of ESRD, and biochemical markers of disease) and the dependent variables (i.e., AD, LVM, and LVMI) were examined by step-wise regression.

Table 1 Underlying diagnosis leading to end-stage renal disease (ESRD) in children undergoing dialysis

Diagnosis	Number
Renal dysplasia	5
Obstructive uropathy	3
Chronic nephritis	2
Nephrotic syndrome	1
Otobrachiorenal syndrome	1
IgA nephropathy	1
Reflux nephropathy	1
Henoch-Schonlein purpura	1
Diffuse mesangial sclerosis	1
Wegener granulomatosis	1
Prune-belly syndrome	1

Results

Our patient population was diverse in age (12.7± 5.8 years), age at diagnosis of ESRD (range 1–17 years), and underlying diagnosis (Table 1). Patient gender (10 males, 8 females), mode of dialysis (PD=9, HD=9), and BSA (1.2±0.5 m^2) did not differ between PD (1.26± 0.6 m^2) and HD (1.14±0.5 m^2) patients (P=0.7).

Based on logistic regression analysis with LVH as the dependent variable, routine laboratory parameters, such as serum creatinine, blood urea nitrogen, albumin, calcium, and phosphorus, were not predictive of LVH. Moreover, the measurements did not differ between children on HD or PD (Table 2).

The difference in average SBP and DBP was not statistically significant between children receiving HD or PD (129±13.2 mmHg, 81±21.2 mmHg, 128±12.4 mmHg,

Table 2 Comparison of laboratory parameters in children undergoing peritoneal (n =9) and hemodialysis (n =9)

	Hemodialysis		Peritoneal dialysis		P
	Mean	SD	Mean	SD	
Blood urea nitrogen (mg/dl)	61.4	25.8	51.2	27.1	0.43
Serum creatinine (mg/dl)	7.7	2.2	7.7	2.5	1
Calcium (mg/dl)	9.8	1.9	9.7	2	0.89
Phosphorus (mg/dl)	5.9	1.9	5.9	2.5	1
Albumin (g/dl)	3.9	1.5	3.4	0.7	0.35
Protein (g/dl)	6.5	1.1	6.4	0.9	0.83

83±14.5 mmHg) (P=0.84, P=0.87, respectively). The percentage of patients in the two dialysis populations receiving antihypertensive therapy (HD=89%, PD=77.8%) was also not statistically significant (Pp=0.52). In these patients angiotensin converting enzyme inhibitor (n=7, 39%), calcium channel blockers (n=6, 33%), diuretics (n=3, 17%), and other antihypertensive drug therapies (n=2, 11%) were used. Of those patients receiving antihypertensive therapy, 28% received more than one antihypertensive medication.

Average LVM (133.3±95.7 g), LVMI (137.1±97.8 g/cm), and AD (2.7±1.8 cm^2 * $dynes^{-1*10-6}$) were compared with the parameters in normal children at our institution. Both LVM and AD were significantly greater in the dialysis population than in the normal population (P=0.015, P=0.001). LVM and LVMI in children undergoing HD (92.9±83.7 g, 80.1±31.1 g/cm) were not statistically different from the values in children undergoing PD (130.0±89.2 g, 89.6±35.9 g/cm) (P=0.3, P =0.5). AD in children undergoing HD (2.2±0.55 cm^2 * $dynes^{-1*10-6}$) was significantly lower than in children undergoing PD (2.7±0.54 cm^2 * $dynes^{-1*10-6}$) (P=0.01). Neither LVM nor LVMI correlated with duration of ESRD diagnosis (P=0.611, P=0.716, respectively) or AD (P=1.90). LVMI also correlated with patient age (P=0.042). In logistic regression analysis with LVM as a dependent variable, AD predicted only 18% of the increase in LVM in children on PD and 8% of the increase in children on HD. Examination of the relationship between AD and LVMI did not reveal a trend in either the PD or HD population (r^2=0.18, P=0.19, r^2=0.08, P=0.30).

Average pre- and post-dialysis SBP and DBP measurements and pre- and post-dialysis MAP are provided in Table 3. Average SBP and DBP did not correlate with the presence of increased or decreased LVM or AD.

LVM did not correlate with pre-dialysis or post-dialysis SBP, DBP, or MAP. Decreased AD correlated with the diagnosis of HTN (P=0.012) and with elevated pre-dialysis BP measurements, but not with average SBP and DBP. The mode of dialysis was not correlated with pre-dialysis SBP, DBP, and MAP or post-dialysis SBP, DBP, and MAP readings (P=0.382, P=0.593, P=0.500, P=0.944, P=0.493, P=0.920, respectively). In an additional logistic regression analysis, the dependent variables LVM and AD were not significantly influenced by SBP (P=0.42, and P=0.16 respectively).

Table 3 Blood pressure parameters in the study population (*SBP* systolic blood pressure, *DBP* diastolic blood pressure, *MAP* mean arterial pressure)

	Hemodialysis	Peritoneal dialysis
	Mean±SD	Mean±SD
Pre-dialysis SBP	125±19	116±18
Pre-dialysis DBP	83±17	79±17
Pre-dialysis MAP	97±17	91±17
Post-dialysis MAP	87±29	89±7
Post-dialysis SBP	116±20	116±10
Post-dialysis DBP	82±26	75±6

Discussion

Echocardiography is a standard method of monitoring disease progression and EOD in patients with HTN. LVM and LVMI are two well-established echocardiographic measures of vascular and myocardial EOD. These measures are predictors of long-term outcome and LVM is an independent risk factor for patient survival in children, adolescents, and adults with HTN or ESRD [16, 17, 18, 22, 34, 35, 36]. AD is a measurement of vascular elasticity, is a marker for the development or regression of LVH, and is a strong independent predictor of CV mortality in adults with essential HTN and those with ESRD undergoing dialysis [24, 29, 37,38].

The increased incidence of arterial abnormalities, CV complications, and death in adults with ESRD has been widely established [35, 36, 39, 40]. In contrast, the onset and rate of progression of CV sequelae in children and adolescents with ESRD is uncertain [1]. CV changes in children and adolescents with ESRD who require HD or PD have been documented; however, the inherent resilience of the CV system in children and the greater ability to adjust to sustained BP changes may account for the lower CV morbidity seen in children and adolescents. Recent observations define a clear increase in CV mortality and morbidity in children and adolescents with ESRD [1,9]. Predictors of cardiovascular EOD in children and adolescents with ESRD have not been well established. In our study LVM and AD in children on PD and HD were abnormal compared with normal subjects in our institution. Furthermore, these absolute values for the CV parameters were worse in the subjects undergoing HD (e.g., higher LVM, lower AD) than those on PD. Although the difference in LVM was not statistically significant based on mode of dialysis, AD was significantly lower (and therefore worse) in children undergoing HD.

This early indication of functional abnormalities in the large arteries (decreased AD) may be related to poor control of BP, particularly between HD sessions, in this group of subjects. The impact of these findings on the choice of dialysis modalities in children and the ability to control BP between dialysis sessions needs to be further evaluated. In the adult literature, long-term continuous ambulatory PD may be associated with more severe LVH and BP fluctuations, and subsequently poorer outcomes than in patients on HD [41]. Persistent volume and pressure load, hypoalbuminemia, and more consistent uremia in PD patients may contribute to the more rapid progression of LVH over time seen in adults [41].

In our study, the mode of dialysis did not correlate with casual SBP and DBP readings. The full impact of casual BP on markers of EOD in children with ESRD is uncertain [42]. It has been proposed that aggressive antihypertensive treatment with agents that lower pulse pressure, such as nitrates and angiotensin-converting enzyme inhibitors, which have been shown to improve the functional abnormalities of the large arteries and prevent structural changes in the aortic wall, are warranted in this population [43, 44, 45]. In our study, the influence of BP control on LVM and AD was not significant, but this finding may have been affected by the cross-sectional nature of this study, small sample size, and the large variability of BP control due to medical therapy and compliance. Prospective studies involving greater numbers of patients are required to determine the full relationship between casual and ambulatory BP and EOD in children and adolescents undergoing PD or HD and to determine whether a specific dialysis modality is associated with more rapid progression of LVH and worse AD in children.

A good correlation between LVMI and AD has been noted in adults on HD [29]. Matsumoto et al. [29] reported that changes in LVM are positively correlated with AD in adults undergoing HD. In our study LVM and the LVMI did not correlate with AD. This may be explained by the small overall sample size and the fact that half of the children were on PD. Further study is necessary to determine if a relationship exists between LVM and vascular distensibility in children and if the mode of dialysis has an impact on the progression of LVH and decreased AD over time in these children. If AD does not correlate with LVH in larger studies of children with ESRD, this may indicate that AD is influenced by different factors than those that lead to LVH in this population.

Biochemical parameters have been shown to independently contribute to CV remodeling in adults on HD [24]. Abnormalities in calcium and phosphorus metabolism have been shown to independently contribute to impaired left ventricular diastolic function and subsequent LVH in these individuals [46]. In our study, laboratory parameters were not predictive of LVH and did not differ between children on HD or PD. A strong association between hypoalbuminemia, an indicator of patient nutritional status, and cardiac morbidity and mortality in adults with ESRD undergoing HD or PD has been established [40,47]. However, in our small sample size serum albumin did not correlate with either LVM or AD. In a number of studies serum albumin is correlated with worse outcome in children on PD and HD [41,47]. However, the lack of correlation in our study may be due to the diversity of the dialysis population, the small sample size, and the variability in duration of HTN seen in our population.

In conclusion, young adults with ESRD undergoing HD or PD are at increased risk of developing LVH and decreased AD. LVH and decreased vascular distensibility increase mortality risk in adults and may predispose children with ESRD to increased mortality as they grow older. The findings in this study confirm earlier studies that demonstrated that LVMI is greater in children on dialysis. This study also demonstrates that abnormal vascular stiffness, as defined by AD, is present in these children. AD did not simply correlate with LVM and may be influenced by different factors than those that lead to LVH in this population. The degree of vascular stiffness in children undergoing HD is greater than that in children on PD. Further study is needed to address how control of BP, uremia, and other factors may affect these abnormalities. Such efforts may decrease progression of CV remodeling and prevent EOD and subsequent mortality in children with ESRD.

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