Nutrition 62 (2019) 20–24

Contents lists available at ScienceDirect

Nutrition

journal homepage: www.nutritionjrnl.com

Applied nutritional investigation

Renal function in prepubertal children born with very low birthweight

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ARTICLE INFO

Article History:
Received 29 July 2018
Received in revised form 3 November 2018
Accepted 21 November 2018

Keywords:
Glomerular filtration rate
Hypertension
Premature newborn
Very low birthweight newborn

ABSTRACT

Objectives: The objective of this study was to evaluate estimated glomerular filtration rates (eGFR) and markers of renal function in very low birthweight (VLBW) children and to relate these parameters to current nutritional status.
Methods: A cross-sectional and controlled study was performed with prepubertal children between ages 5 and 10, including 44 VLBW participants and 30 healthy participants born at full term with an adequate birthweight (control group). The following data were collected: perinatal history; current weight, height and waist circumference; blood pressure (three measures); blood creatinine, urea, uric acid, cystatin-C, and neutrophil gelatinase-associated lipocalin levels; and urine albumin, creatinine, and calcium levels.
Results: Blood pressure, eGFR, albuminuria, concentrations of cystatin-C, neutrophil gelatinase-associated lipocalin, uric acid, urea, creatinine, and fractional calcium excretion did not differ between VLBW and control groups. Regarding the VLBW group, there was no difference in eGFR, albuminuria, and other markers of renal injury in overweight or obese children compared with children with a normal body mass index.
Conclusions: Prepubertal children born with VLBW did not have altered renal function, regardless of their current nutritional status.

Introduction

Preterm infants with very low birthweight (VLBW), newborns with birthweight less than 1500 g, represent a heterogeneous group of children whose survival has increased significantly in recent decades [1]. Despite the current short-term reduction in mortality and negative outcomes, there is growing concern regarding long-term morbidity. A study of a cohort of individuals born prematurely (between 22 and 36 weeks) found higher adulthood mortality related to respiratory, endocrinologic, and cardiovascular diseases [2].

Among those diseases associated with higher cardiovascular risk in preterm infants, high blood pressure and chronic renal disease are notable [3]. Because the development of renal structures is complete around the 34th to 36th wk after conception [4], prematurity may lead to a reduction in glomerulogenesis with a compensatory increase in remaining glomeruli with hyperfiltration, tubular overload, and proteinuria. In the long term, such alterations may cause decreased sodium excretion capacity, greater susceptibility to hypertension, reduced adaptability to injury, and loss of renal function; consequent glomerulosclerosis is a determining factor for the progression of chronic kidney disease [5]. Overall, restricted intrauterine growth and accelerated postnatal weight gain exacerbate these changes [6].

Nonetheless, findings regarding renal disease progression in VLBW newborns are controversial. It is known that the number of nephrons at birth is associated with birthweight, and an increase in 257 426 nephrons per kilogram of birthweight [7] and a reduction in 2.25 mL/min per 1.73 m^3 estimated glomerular filtration rate (eGFR) for each 1 kg less in birthweight [8] is estimated. These changes, combined with different risk factors during neonatal unit hospitalization, such as the use of nephrotoxic drugs, hypoxia, infections, hypotension, and parenteral/enteral nutritional therapy with greater supply of protein and electrolytes [5–7], may be related to renal disease.

Children born prematurely have a high risk for developing other chronic diseases, such as type 2 diabetes, cardiovascular diseases,

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https://doi.org/10.1016/j.nut.2018.11.030
0899-9007/

insulin resistance, and obesity, which in turn are also associated with renal impairment [9].

In view of the gaps in the knowledge about renal function in children born prematurely, this study evaluated eGFR and renal injury markers (tubular and glomerular) for prepubertal children born with very low birthweight (<1500 g) and sought to relate these parameters to current nutritional status.

Methods

Study design

A cross-sectional and controlled study was performed between 2014 and 2015 with prepubertal children between ages 5 and 10 y who were born prematurely and with VLBW (VLBW group, $n = 44$). These children were compared with healthy children of the same sex and age born at term and with adequate weight for their gestational age (control group, $n = 30$).

The children of the VLBW group were followed up at the outpatient clinic of the Municipal Hospital of São Bernardo do Campo (HMU-SBC). The control group consisted of children recruited voluntarily from the Pediatrics Outpatient Clinic of the Faculdade de Medicina do ABC. Children with chronic diseases, except an overweight status, and those who were using corticosteroids (3 mo before data collection) or hormone replacement were excluded from the study.

HMU-SBC is a baby-friendly hospital that adopts the Kangaroo Method and has a human milk bank. HMU-SBC has the only public maternity ward in the city; it is a reference for high-risk pregnancies, with approximately 350 to 400 deliveries per month performed, representing 80% of the births in São Bernardo do Campo, São Paulo, Brazil. The assistance protocols used are in line with the recommendations of the main national and international scientific societies. The nutritional therapy used in the neonatal care unit for a preterm infant is exclusively mother's milk, preferably raw milk from the newborn's mother or pasteurized human milk when necessary.

To recruit study participants we identified 130 children born prematurely between 2004 and 2009 seen at our outpatient premature clinic; 60 were located and invited to participate. Four of the children were excluded from the study because their parents or guardians did not agree to participate, 4 because they had pubertal development, and 7 because they had chronic non-progressive encephalopathy; 1 was excluded because of the use of growth hormone at the time. The final sample involved 44 children.

To participate in the study, the parents or guardians and the children in both the VLBW group and the control group agreed to and signed informed consent. The study was approved by the Research Ethics Committee of the School of Medicine of ABC (no. 858.358).

Collected data

The medical records of the hospitalization period were evaluated using a standardized form; data on weight, length, head circumference at birth, gestational age, interventions during the neonatal period, and nutritional therapy (parenteral and enteral) were collected. INTERGROWTH-21 was employed to assess adequate birthweight for gestational age [10]. The children were classified as small (SGA), adequate (AGA), and large (LGA) for gestational age when the birthweight for gestational age was less than the 10th percentile, between 10th and 90th percentiles, and greater than the 90th percentiles of INTERGROWTH-21 curve, respectively.

In the VLBW group, the following information was obtained from outpatient clinic follow-up: weight, length, and head circumference at hospital discharge up to 12 mo corrected age. These data were used to evaluate the z scores of body mass index (ZBMI), height for age (ZHA), and head circumference (ZHC) at 12 mo corrected age and the variation of these indicators between discharge and 12 mo corrected age. The total time of breastfeeding and introduction of complementary feeding was also calculated using the chronological age.

After inclusion in the study, clinical evaluation and collection of blood samples for laboratory tests were performed. Weights and heights of the children were obtained according to World Health Organization 1995 standards [11]. These data were used to calculate ZBMI and ZHA according to the recommendation and cutoff points proposed by the World Health Organization in 2007 [12].

The waist circumference measurement was obtained at the midpoint of the iliac crest and last rib, from which the waist-to-height ratio was calculated; the cutoff for inadequate waist/height was greater than 0.5 [13]. The evaluation of pubertal stage was performed by direct evaluation according to the criteria of Marshall and Tanner [14,15]. Blood pressure (BP) was measured using a single measurement on three different occasions, as recommended by the Task Force [16].

Laboratory tests

To perform the analyses, 10 mL of blood was collected after 12 h of fasting. The samples were transported under refrigeration to the Laboratory of Clinical Analysis of the Faculdade de Medicina do ABC, where they were centrifuged and stored at −20°C.

Plasma concentrations of renal function markers were determined: urea, creatinine by enzymatic colorimetric methods; tubular marker neutrophil gelatinase-associated lipocalin by enzyme-linked immunosorbent assay (Bioporto Diagnostic, Hamburg, Germany). Uric acid and cystatin-C (enzyme-linked immunosorbent assay, IBL International, Hamburg, Germany) tubular and glomerular markers were also measured. The plasma creatinine level was used to determine the eGFR according to the Schwartz equation (eGFR [mL/min per 1.73 m^2] = 0.413 × height [cm]/plasma creatinine [mg/dL]) [17].

A single urine sample (20 mL) was also collected for measurements of albumin (Turbidimetry Latex, Spinreact), creatinine, and calcium (colorimetric methods). Albuminuria and fractional calcium excretion were calculated from these samples. Values greater than 30 mg/g were adopted as the cutoff point for the presence of albuminuria [18].

Statistical analysis

Statistical analysis was performed using SPSS 24.0 (IBM Corp., Armonk, NY, USA). Categorical variables are presented as the absolute number and percentage and were compared using the χ^2 test. Continuous variables were evaluated for normality with Kolmogorov-Smirnov test. Those following a normal distribution are presented as the mean ± standard deviation; those not following a normal distribution are described as the median and interquartile range. Student's t test and the Mann-Whitney test, respectively, were used for comparison. Spearman's correlation was used to measure the association between albuminuria and eGFR. A level of significance of 5% was adopted for rejection of the null hypothesis.

The sample size was calculated for the main study by adopting $\alpha = 0.05$ (bidirectional), $\beta = 0.10$, and an addition of 20% to cover for possible losses. At least 32 children in each group were necessary to detect differences in eGFR between the VLBW and control groups [19].

Results

In VLBW group, 19 of the participants (43.2%) were male, and the birthweight and gestational age means were 1157 ± 242 g and 30.0 ± 2.3 wk, respectively. There was no statistically significant difference for clinical and laboratory variables with respect to gender (Table 1).

Table 1
General characteristics of mothers in very low birthweight (VLBW) and control groups

Variable	Units	VLBW group ($n = 44$)	Control group ($n = 30$)	P
Maternal age	Years	32.9 ± 8.2	34.4 ± 6.1	0.932
Schooling	Years	4.0 (0.0; 5.0)	4.0 (3.0; 5.0)	0.566[†]
Smoking	Yes	6 (13.6%)	1 (3.3%)	0.232[†]
Alcohol ingestion	Yes	4 (9.1%)	0 (0.0%)	0.142[†]
Pregnancies	Number	1.0 (1.0;3.0)	1.0 (1.0; 2.0)	0.289[†]
Type of delivery	Caesarian section	21 (47.7%)	11 (36.7%)	0.474[‡]
Gestational age	weeks	30.5 ± 2.3	39.0 ± 1.4	< 0.001

VLBW, very low birthweight.
Student's t test level of significance.
[†]Mann-Whitney test significance level.
[‡]χ^2 test significance level.

Table 2
Comparison of age, sex, nutritional status, waist-to-height ratio, and blood pressure among very low birthweight (VLBW) and control groups

Variable	Units	VLBW group ($n = 44$)	Control group ($n = 30$)	P
Age	Years	6.9 ± 1.5	7.1 ± 1.3	0.606*
Sex	Male	19 (43.2%)	13 (43.3%)	0.589†
Weight at birth	Grams	1157 ± 242	3196 ± 348	0.019*
Height/age	z Score	−0.43 ± 1.11	0.17 ± 0.95	0.017*
Short stature	ZHA < −2	4 (9.1%)	0 (0.0%)	0.142†
Body mass index for age	z Score	0.11 ± 1.34	-0.02 ± 0.96	0.317*
Nutritional status	Obesity	5 (11.3%)	0 (0.0%)	0.221†
	Overweight	4 (9.0%)	5 (16.6%)	
	Normal weight	35 (79.5%)	24 (80%)	
	Underweight	0 (0.0%)	1 (3.3%)	
Waist/height	cm/cm	0.47 ± 0.05	0.44 ± 0.03	0.868*
	>0.5	8 (18.2%)	1 (3.3%)	0.074†
Systolic blood pressure	mm Hg	91.4 ± 10.9	93.4 ± 8.8	0.377*
	>90th percentile	1 (2.3%)	1 (3.3%)	0.650†
Diastolic blood pressure	mm Hg	43.7 ± 16.1	42.5 ± 13.2	0.667†
	>90th percentile	0 (0.0%)	0 (0.0%)	NC†

NC, not calculated; ZHA, z score height for age; VLBW, very low birthweight.
*Student's t test level of significance.
†χ^2 Test significance level.

A total of 12 (27.3%) and 32 (72.7%) children were born with a weight less than and greater than 1000 g, respectively. Regarding adequation for gestational age, 15 (34.1%) were SGA and 29 (65.9%) AGA. Renal function markers and eGFR did not differ between the SGA children and those who were AGA (data not shown).

The median hospital length of stay of the children in the VLBW group was 51.0 d (41.1; 73.3), with median oxygen use of 18.1 d (3.5; 28.5). No child required dialysis during the neonatal period. Regarding nutritional therapy, the onset of parenteral and enteral nutrition occurred in 2.0 d (2.0, 3.7) and 3.0 d (1.0, 3.0), respectively.

Through chronological age, the total time of breastfeeding and the introduction of complementary feeding in VLBW group were 9.7 ± 8.3 mo and 6.9 ± 1.5 mo, respectively.

In VLBW group, variations in ZBMI, ZHA, and ZHC for hospital discharge up to 12 mo corrected age were 0.85 ± 0.22, 3.33 ± 0.27, and 1.85 ± 0.22, respectively. At 12 mo corrected age, an underweight status (ZBMI < −2) was identified in 6 participants (14.3%), overweight/obesity (ZBMI > +1) was identified in 4 (9.5%), and short stature (ZHA < −2) was identified in 23 (54.8%).

With respect to current nutritional evaluation, 5 (11.3%), 4 (9,1%), and 4 (9.1%) of the children in the VLBW group were obese, overweight, and had a short stature, respectively (Table 2). However, no statistically significant difference was identified in relation to ZBMI, waist-to-height ratio, and BP (systolic and diastolic) between the groups (Table 2). In turn, the mean ZHA in the VLBW group was lower than that in the control group (−0.43 ± 1.11 versus 0.17 ± 0.95, $P = 0.017$).

None of the children evaluated exhibited arterial hypertension. A first systolic BP measurement greater than the 90th percentile was found for only 1 participant in the VLBW group and another in the control group, although the other BP measurements were normal.

Seven (15.9%) and 1 (3.3%) of the VLBW and control group participants, respectively, had an eGFR <90 mL/min per 1.73 m^2 ($P = 0.132$). Albuminuria >30 mg/g was found in 9 (20.5%) and 8 (26.7%) children in the VLBW and control groups, respectively ($P = 0.581$). Mean values of eGFR; concentrations of cystatin-C, neutrophil gelatinase-associated lipocalin, uric acid, urea, and creatinine; and fractional calcium excretion did not differ between the groups (Table 3).

Table 4 shows the eGFR and renal injury markers in the VLBW children with and without excess weight. Overall, current nutritional status did not influence renal function.

Discussion

The present study found no change in eGFR or markers of renal function in prepubertal children born with VLBW. There was also no impairment in renal function in those who were overweight or obese at the time of evaluation compared with those with a normal body mass index.

Similar studies found a reduction in the eGFR, impairment in tubular function [19], higher concentrations of cystatin-C, and

Table 3
Comparison of the glomerular filtration rate and markers of renal injury in very low birthweight (VLBW) and control groups

Variable Renal injury function and markers	Units	VLBW group ($n = 44$)	Control group ($n = 30$)	P
Glomerular filtration rate	mL/min per 1.73 m^2	116.9 ± 23.8	118.3 ±2 1.5	0.804*
Albuminuria	mg/g	11.0 (8.2; 21.8)	11.6 (5.5; 33.6)	0.095†
Uric acid	mg/dL	3.02 ± 0.75	2.83 ± 0.86	0.130*
Urea	mg/dL	29.5 ± 7.6	25.1 ± 6.9	0.393*
Creatinine	mg/dL	0.43 ± 0.08	0.44 ± 0.07	0.177*
Cystatin C	ng/mL	5.3 ± 1.7	5.6 ± 1.2	0.121*
NGAL	ng/mL	0.23 ± 0.19	0.22 ± 0.22	0.203*
Fractional calcium excretion	mg/g	1.0 (0.0;11.6)	1.1 (0.0;11.5)	0.190†

NGAL, neutrophil gelatinase-associated lipocalin; VLBW, very low birthweight.
*Student's t test level of significance.
†Mann-Whitney test significance level.

Table 4
Comparison of the glomerular filtration rate and markers of renal damage in overweight/obese and normal body mass index very low birthweight children

Variable	Units	Overweight/obesity ($n = 9$)	Normal BMI ($n = 35$)	P
Glomerular filtration rate	mL/min per 1.73 m^2	120.8 ± 26.3	115.9 ± 23.5	0.582*
Albuminuria	mg/g	8.8 (7.0;19.9)	11.2 (8.2;27.4)	0.567†
Uric acid	mg/dL	3.3 ± 0.8	2.9 ± 0.7	0.288*
Cystatin C	ng/mL	5.0 ± 1.3	5.4 ± 1.8	0.550*
NGAL	ng/mL	0.24 ± 0.14	0.23 ± 0.20	0.893*
Fractional calcium excretion	mg/g	1.12 (0.4; 3.6)	1.03 (0.4;3.2)	0.456†

BMI, body mass index; NGAL, neutrophil gelatinase-associated lipocalin.
*Student's *t* test significance level.
†Mann-Whitney test significance level.

smaller renal size [20] in children and adolescents who were born preterm. However, differences among the studies may explain divergent results, such as inclusion in the studies only children with extremely low birthweight (birthweight <1000 g) [19,20], older age groups, no description of pubertal stage [19], and the use of only birthweight without considering gestational age for classification of newborns.

Regarding our study, the included children presented several factors known to confer a risk of kidney disease progression. Some of these factors are related to prematurity (low gestational age, birthweight, and high percentage of SGA), and others result from hospitalization (prolonged hospitalization time and use of oxygen therapy). Nevertheless, no alteration in function or early markers of renal injury was identified.

Although there are limitations to our conclusions owing to the model used in the study, we can suggest that at least two factors present early in life may have been related to this protective effect. The first is the exclusive use of human milk throughout the hospital stay. Human milk has a lower quantity but a better quality of protein compared with that in infant formula. It also contains lower concentrations of electrolytes such as sodium and better bioavailability of many micronutrients and other factors, such as long-chain polyunsaturated fatty acids, which may contribute to a reduction in inflammation and protect kidney development [21].

A clinical trial published in 2011 found that formula-fed infants' kidney size was bigger than that of breastfed infants at 6 mo old. The authors suggested this finding could be related to greater protein content in infant formula [22]. A prospective cohort study in the Netherlands reported that kidney volume and eGFR were lower in breastfed children compared with nobreastfed children at school age [23]. These results suggest that breastfeeding in term infants is associated with subclinical changes in kidney outcomes in childhood.

The second factor is the pattern of nutritional recovery in the first year of life. During this period, the catch-up growth of height and head circumference was greater than that of the BMI. Although there are no data available in the literature on the ideal recovery for VLBW, a balanced catchup has been suggested to play a protective role for the development of chronic non-communicable diseases, including hypertension and chronic kidney disease [24,25], in this population.

These two early factors combined with a long-term healthy lifestyle may protect renal function until adulthood [25]. Future studies with a follow-up in adolescence and adulthood may confirm this hypothesis.

The percentage of overweight and obesity in the VLBW group was similar to that identified in the general population of the same age group in our country [26]. An overweight status is associated with the progression of renal disease through glucose intolerance and hypertension. In our study, all children had normal blood pressure, and no individuals with glucose intolerance were included; thus we can consider this absence of an association between current excess weight and impairment in renal function to be pertinent. However, we cannot rule out the possibility that with increased age and pubertal development, along with other risk factors, this association will not arise.

Among the strengths of this study are the description of the gestational age of preterm infants, the inclusion of a single center with uniformity in treatment protocol, and the description of the evolution of growth during the first year of life in VLBW group. In turn, we can mention the following as weaknesses: the lack of detailed information on prenatal care, maternal diseases, and nutritional status; the absence of a comparison group with VLBW that had received infant formula; lack of information on early growth and feeding practices in the control group; and finally, the evaluation of albuminuria through a single collection of an isolated sample.

Prepubertal children born with VLBW did not have alteration in renal function, blood pressure, or the evaluated markers of renal function. The presence of overweight and obesity was also not associated with higher blood pressure or impairment of renal function.

Acknowledgments

The authors thank the children and their families, the nursing team of the Municipal University Hospital of São Bernardo do Campo, the team of the Laboratory of Clinical Analysis of the School of Medicine of ABC, the Research and Extension Nucleus in Health Center (Núcleo de Pesquisa e Extensão em Saúde), and American Journal Experts.

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