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Original Article

Chronic renal disease in children aged 5–18 years: a population-based survey in Turkey, the CREDIT-C study

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Abstract

Background. Data on the epidemiology of chronic kidney disease (CKD), which is a serious health problem and refers to a condition related to irreversible kidney damage that further progress to end-stage renal disease in children, are insufficient and data that are available were based on hospital records. The aim of this nationwide, population-based field study was to determine the prevalence of CKD in children in Turkey and to evaluate the association between CKD and possible risk factors such as obesity and hypertension.

Methods. The study was the paediatric stratum (3622 children aged 5–18 years) of the previously published population-based survey of Chronic REnal Disease In Turkey (CREDIT study). Medical data were collected through home visits and interviews between November 2007 and July 2008; height, weight and blood pressure were also measured. Serum creatinine, total cholesterol, uric acid and complete blood count were determined from 12-h fasting blood samples, and spot urine tests were performed for subjects who gave consent to laboratory evaluation.

Results. Following adjustment according to gender, residence, age groups and geographical regions, the prevalence of children with estimated glomerular filtration rate (eGFR) <75 mL/min/1.73 m^2 was 0.94 [95% confidence interval (CI): 0.63–1.35], and the prevalence of children with CKD Stages 3–5 [National Kidney Foundation—Kidney Disease Outcomes Quality Initiative (K/DOQI)] was 2600 (95% CI 1100–5100) per million age related population. The mean eGFR was found to increase with age; the ratios of children with eGFR <90 and <75 mL/min/1.73 m^2 were higher in younger age groups. The frequencies of overweight and obese children were 9.3 and 8.9%, respectively, and the mean eGFR was lower in patients with higher body mass index. The prevalence of hypertension and hypercholesterolaemia was 6.1 and 5.8%, respectively; the mean eGFR was lower in children with hypercholesterolaemia.

Conclusions. This is the first population-based CKD study performed in children aged 5–18 years. The prevalence of CKD in our study was 25–100 times greater than that found in previous hospital-based studies. Our data suggest that approaches focusing on patients in tertiary centres are likely to lead to patients being missed at early stages of CKD and that a vast majority of these children will never develop symptomatic CKD during childhood.

Keywords: children; chronic kidney disease; epidemiology; hypertension; obesity

Introduction

Chronic kidney disease (CKD) is a serious health problem and refers to a condition related to irreversible kidney damage that further progress to end-stage renal disease (ESRD). Data on the epidemiology of CKD in children are insufficient. The absence of a common definition and a well-defined classification of CKD in children resulted in limited information on the epidemiology of CKD. National Kidney Foundation's Kidney Disease Outcomes Quality Initiative (NKF-K/DOQI) published a classification system for CKD applicable to children in 2003 [1], and papers published afterwards used this classification.

Studies from Europe [France (1975–90), Sweden (1986–94), Italy (1990–2000), Turkey (2005), Belgium (2001–05), Spain (2007–08)] and non-European countries [Chile (1996), Nigeria (1985–2000), Jordan] have reported the incidence of CKD in children to be between 3.0 and 12.1 cases per million; the definition of CKD was not uniform in these studies [ranging from glomerular filtration rate (GFR) <75 mL/min/1.73 m^2 to end-stage kidney disease or creatinine >2 mg/dL], and the data were based on hospital records [2–10].

The prevalence of chronic renal failure in Sweden (between 1986 and 1994, in children aged 6 months–16 years, chronic renal failure was defined as GFR <30 mL/min/1.73 m^2) and Chile (in 1996, in children <18 years of age, chronic renal failure was defined as GFR <30 mL/min/1.73 m^2) were 25 and 42.5 cases per million age-related population (p.m.a.r.p.), respectively [3, 8]. The ItalKid Project, a population-based registry, defined the prevalence of CKD (GFR <75 mL/min/1.73 m^2), by regularly asking all of the paediatric hospitals and adult nephrology units potentially involved in caring for children (age <20 years) with kidney disease, to be 74.7 cases p.m.a.r.p. [4].

The North American Pediatric Renal Trials and Collaborative Studies (NAPRTCS) have operationally defined the cut-off for inclusion into the CKD registry as a GFR below 75 mL/min/1.73 m^2. European Society for Pediatric Nephrology/European Renal Association-European Dialysis and Transplant Association (ESPN/ERA-EDTA) Registry is a registry on renal replacement therapy patients (CKD Stage 5). ESPN/ERA-EDTA Registry and data from hospital registries are ideal for the incidence and prevalence of renal replacement therapy, but they lack asymptomatic, under-diagnosed, under-reported CKD, especially for less advanced stages of the disease.

There are limited data on the epidemiology of the early stages of CKD in the children as it is often asymptomatic and under-diagnosed. In this population-based field study, we aimed to determine the prevalence of CKD in children in Turkey and to evaluate the association between CKD and possible risk factors such as obesity and hypertension.

Materials and methods

Study population

The present CREDIT-C study, consisting of children aged 5–18 years, was the paediatric stratum of the previously published population-based survey of Chronic REnal Disease In Turkey (CREDIT study) [11]. The data of the children were collected between November 2007 and July 2008 through home visits and interviews by specially trained field study teams. During the interviews, all of the participants were asked to complete the study questionnaire including questions on demographic features, height and weight. For the children who were between 5 and 10 years of age, their parents were asked to complete the questionnaire. Moreover, casual blood pressure (BP) was measured three times, with 10 min intervals. Informed consents were obtained from both parents and children for those aged 11–18 years and from only parents for those aged 5–10 years. The study was approved by the Ethics Committee of the Gazi University Medical Faculty.

Sample size

To the best of our knowledge, there has been no population-based study in the literature calculating the prevalence of CKD in children. Following discussions with paediatric nephrologists from our country, we assumed the prevalence of children with eGFR <90 mL/min/1.73 m^2 to be 2–3%. The study sample was calculated in order to achieve a 2% predicted prevalence of children with GFR <90 mL/min/1.73 m^2; 3865 children were found to be adequate with a cluster effect of 1.2 and a two-sided 95% confidence interval (CI) of ±0.5%. In the present study 3622 children were included. The detailed sampling method of the present study is presented in the Supplementary Data.

Definitions and laboratory assessment

Height percentile and body mass index (BMI) percentile were determined by using CDC growth charts [2–20 years, Centers for Disease Control and Prevention (CDC) growth charts, USA]: growth retardation was defined as height percentile <3rd percentile for age and gender, obesity was defined as BMI ≥95th percentile for age and gender and underweight was defined as BMI <5th percentile for age and gender [12].

The diagnosis and classification of hypertension was based on 'The 4th Report on Diagnosis, Evaluation and Treatment of High Blood Pressure in Children and Adolescents, 2004': Stage 1 hypertension was defined as BP levels that range from the 95th percentile to 5 mmHg above the 99th percentile; Stage 2 hypertension was defined as BP levels that are 5 mmHg above the 99th percentile [13].

For the laboratory assessments, the subjects were visited the day after the interview to collect fasting blood samples. At the same time, morning spot urine samples were obtained from the participants. Additionally, spot urine analysis (by dipsticks) was performed during home visits, and results were recorded on the study questionnaire.

Urine albumin (immunoturbidimetric method) and creatinine (alkaline picrate method), complete blood count, serum creatinine (alkaline picrate method), total cholesterol (cholesterol oxidase method) and uric acid (uricase method) concentrations were determined.

Anaemia was defined according to haemoglobin level for age and gender: haemoglobin level <10.5 g/dL for children below 7 years of age, haemoglobin level <11.0 g/dL for 7–12 years of age, haemoglobin level <12.0 g/dL for girls above 12 years of age and haemoglobin level <14.0 g/dL for boys above 12 years of age [14].

Hyperuricaemia was defined as uric acid level >6.6 mg/dL for 5–11 years of age, >7.7 mg/dL for boys above 11 years of age and >5.7 mg/dL for girls above 11 years of age [15].

Hypercholesterolaemia was defined as high total cholesterol ≥95th percentile for age and sex [16].

Hypercalciuria was defined as urinary calcium/creatinine ratio above 0.20 (mg/mg). The estimated GFR (eGFR) was calculated by using original Schwartz [$k \times$ length (cm)/creatinine (mg/dL)]: k was 0.70 if boy ≥13 years, and 0.55 otherwise [17, 18]. According to the K/DOQI classification system, CKD is defined by a presence of kidney damage for ≥3 months [as defined by structural or functional abnormalities of the kidney, with or without decreased GFR, manifested by one or more of the following features: (i) abnormalities in the composition of the blood or urine, (ii) abnormalities in imaging tests or (iii) abnormalities on kidney biopsy] or a GFR < 60 mL/min/1.73 m^2 for ≥3 months [1]. As we were not able to perform imaging studies in such a population-based field study, it was not possible to identify patients in CKD Stages 1 and 2 which correspond to kidney damage with normal or increased GFR >90 mL/min/1.73 m^2 and kidney damage with mild decrease in GFR 60–89 mL/min/1.73 m^2, respectively. As a result, this study presents data based on eGFR only; we presented the prevalence of children with eGFR below 90 mL/min/1.73 m^2 and with CKD Stages 3, 4 and 5 which correspond to GFR limits of 30–59, 15–29 and <15 mL/min/1.73 m^2, respectively. The cut-off point of 75 mL/min/1.73 m^2 was to compare our data with certain registries (NAPRTCS, Italian registry) where CKD is defined by a GFR below 75 mL/min/1.73 m^2 [4, 19].

Macroalbuminuria was defined as an albumin-to-creatinine ratio of 300 mg/g or higher [1].

Statistical analysis

Analyses were performed according to the age groups, gender, geographical area and residence. Student's t-tests and the Mann–Whitney U-tests or Kruskal–Wallis tests were used for comparisons of continuous variables, which showed normal and non-normal distribution, respectively. χ^2 tests were used to compare proportions between the groups. In order to eliminate the influence of children without data on eGFR, the adjustment was performed according to gender, residence, age groups and geographical regions. Statistical significance was defined as $P < 0.05$.

Results

A total of 3622 children aged 5–18 years were enrolled. Demographic features of the study group are presented in Table 1. The distribution of the study population was parallel with the general Turkish population according to gender, residence, age groups and geographical regions.

Table 1. Demographic features of the study population

	General Turkish population		Study	
	n	%	*n*	%
Gender				
Female	8 868 377	49.9	1796	49.6
Residence				
Urban	12 530 140	70.4	2469	68.2
Age, years				
Total	—	—	11.88 ± 3.40	
Female	—	—	11.97 ± 3.40	
Male	—	—	11.80 ± 3.40	
Age groups, years				
5–12	10 283 822	58.0	2027	56.0
13–18	7 490 290	42.0	1595	44.0
Geographical regions				
Central Anatolia	2 626 736	14.8	613	16.9
Mediterranean	2 243 288	12.6	384	10.6
Aegean	2 342 248	13.2	407	11.2
Marmara	5 552 172	31.2	1327	36.6
Black Sea	1 754 574	9.9	321	8.9
East Anatolia	1 453 607	8.2	266	7.3
South-eastern Anatolia	1 806 143	10.2	304	8.4

Table 2. Distribution of BMI groups, hypertension and certain laboratory features

	N	%	95% CI
BMI groups			
Underweight	243/3571	6.8	6.0–7.7
Normal	2679/3571	75.0	73.6–76.4
Overweight	331/3571	9.3	8.3–10.3
Obese	318/3571	8.9	8.0–9.9
Growth retardation	867/3575	24.3	22.9–25.7
Hypertension			
>95th percentile	216/3549	6.1	5.3–6.9
Stage I	183/3549	5.2	4.5–5.9
Stage II	33/3549	0.9	0.6–1.3
Macroalbuminuria	52/3031	1.7	1.3–2.2
Hypercalciuria	208/3026	6.9	5.99–7.83
Leucocyturia	54/3056	1.8	1.33–2.30
Haematuria	34/3063	1.1	0.77–1.55
Anaemia	656/3016	21.8	20.3–23.3
Hyperuricaemia	28/3174	0.9	0.6–1.3
Hypercholesterolaemia	183/3171	5.8	5.0–6.6

Distribution of BMI groups and the prevalence of growth retardation, hypertension, macroalbuminuria, hypercalciuria, leucocyturia, haematuria, anaemia, hyperuricaemia and hypercholesterolaemia are shown in Table 2. The prevalence of obesity was higher in younger age group: 13.5% for 5–7 years, 10.5% for 8–10 years, 8.6% for 11–13 years and 6.4% for 14–18 years.

eGFR was calculated in 3079 children. The mean eGFR was slightly higher among males (132.58 ± 25.48 mL/min/1.73 m^2), compared with girls (125.03 ± 19.71 mL/min/1.73 m^2; $P < 0.001$). Ninety-four children had an eGFR below <90 mL/min/1.73 m^2 [94/3079; 3.05% (95% CI: 2.47–3.72)], and 24 children had an eGFR below <75 mL/min/1.73 m^2 [24/3079; 0.78% (95% CI: 0.50–1.16)]. Following adjustment according to gender, residence, age groups and geographical regions, the prevalence of children with eGFR below <90 mL/min/1.73 m^2 was 3.47% [107/3082 (95% CI: 2.85–4.18)], and the prevalence of children with an eGFR below <75 mL/min/1.73 m^2 was 0.94% [29/3082 (95% CI: 0.63–1.35)]. The prevalence of children with CKD Stages 3–5 (K/DOQI) are shown in Table 3; the prevalence of children with CKD Stages 3–5 (K/DOQI) was 1300 (95% CI: 350–3300) p.m.a.r.p. for unadjusted data, and 2600 (95% CI: 1100–5100) p.m.a.r.p. for adjusted data, respectively. The ratios of children with eGFR <90 mL/min/1.73 m^2 (3.5% among males versus 2.6% among girls; P: 0.138) or <75 mL/min/1.73 m^2 were comparable (1.0% among males versus 0.6% among girls; P: 0.216) between genders.

Table 3. Prevalence of children with K/DOQI Stages 3–5

K/DOQI Stages	*n*	% (95% CI)
Stage 3–5 (GFR <60 mL/min/1.73 m^2)	4/3079	0.13 (0.035–0.33)
Stage 3 (GFR 30–59 mL/min/1.73 m^2)	2/3079	0.07 (0.008–0.23)
Stage 4 (GFR 15–29 mL/min/1.73 m^2)	1/3079	0.03 (0.0008–0.18)
Stage 5 (GFR <15 mL/min/1.73 m^2)	1/3079	0.03 (0.0008–0.18)

The mean eGFR increased with age; the ratios of children with eGFR <90 and <75 mL/min/1.73 m^2 were higher in younger age groups (Table 4).

The mean eGFR was lower in patients with higher BMI; obese children had the lowest eGFR ($P < 0.001$). The ratios of children with eGFR <90 and <75 mL/min/1.73 m^2 were higher in obese patients, but did not reach statistical significance (Table 4).

The mean eGFR was lower in children with hypercholesterolaemia; the ratios of children with eGFR <90 and <75 mL/min/1.73 m^2 were higher in children with hypercholesterolaemia (Table 4).

The mean eGFR did not differ significantly according to the presence of hypertension or proteinuria.

Discussion

There is a limited number of studies on the epidemiology of CKD in children, and the prevalence of CKD in the general population of children and adolescents has not been extensively studied. The published studies mainly concentrate on the late and severe stages of CKD and there are no population-based studies targeting the earlier stages of CKD or under-diagnosed CKD in children. This study provides the first population-based prevalence study of CKD in children aged 5–18 years.

Difficulties of scintigraphic GFR measurement led clinicians to practical, bed-side methods. The NKF K/DOQI Clinical Practice Guidelines, published in 2003, recommend that the formulas by Schwartz *et al.* [17, 18] or Counahan *et al.* [20] be used to estimate GFR [1]. We also used 'original' Schwartz formula which is still the preferred, practical and reliable method for GFR estimation in such a large study group.

For the correct interpretation of GFR values in children and adolescents there needs to be a clear understanding that the normal level of GFR varies according to age, gender and body size. The mean GFR in children aged

Table 4. Mean eGFR and ratios of children with eGFR <90 or <75 mL/min/1.73 m^2 according to age groups, BMI groups, hypercholesterolaemia, hypertension and proteinuria

Feature	Mean eGFR mL/min/1.73 m^2	P-value	eGFR <90 mL/min/1.73 m^2	P-value	eGFR <75 mL/min/1.73 m^2	P-value
Age groups						
5–7 years (*n*: 342)	119.18 ± 20.18$^{\$,@}$	<0.001$^{\Omega}$	6.4%$^{@}$	<0.001$^{©}$	2.3%$^{@}$	0.001$^{©}$
8–10 years (*n*: 676)	122.62 ± 19.24$^{\$,@}$		4.0%$^{@}$		0.9%	
11–13 years (*n*: 1003)	128.78 ± 23.11*$^{,\#,@}$		3.2%*$^{,@}$		0.8%	
≥14 years (*n*: 1058)	135.87 ± 23.92*$^{,\#,\$}$		1.2%*$^{,\#,\$}$		0.2%*	
BMI groups						
Underweight (*n*: 203)	133.30 ± 24.45$^{\#,\$,@}$	<0.001$^{\Omega}$	3.0%	0.318$^{©}$	1.0%	0.715$^{€}$
Normal (*n*: 2342)	129.19 ± 22.95*$^{,@}$		2.8%		0.6%	
Overweight (*n*: 285)	128.18 ± 22.79*$^{,@}$		3.2%		0.7%	
Obese (*n*: 244)	122.71 ± 21.64*$^{,\#,\$}$		4.9%		1.2%	
Hypercholesterolaemia						
Present (*n*: 176)	120.53 ± 27.33	<0.001$^{£}$	13.1%	<0.001$^{©}$	4.0%	<0.001$^{¥}$
Absent (*n*: 2900)	129.29 ± 22.71		2.4%		0.7%	
Hypertension						
Present (*n*: 171)	128.17 ± 20.76	0.517$^{£}$	1.2%	0.238$^{¥}$	0.6%	1.000$^{¥}$
Absent (*n*: 2882)	129.01 ± 22.96		3.0%		0.7%	
Proteinuria						
Present (*n*: 44)	131.47 ± 19.60	0.301$^{£}$	0.0%	0.642$^{¥}$	0.0%	1.000$^{¥}$
Absent (*n*: 2649)	128.49 ± 22.88		3.1%		0.8%	

*Significant compared with 5–7 years or with underweight, where appropriate, at $P < 0.05$.
$^{\#}$Significant compared with 8–10 years or with normal weight, where appropriate, at $P < 0.05$.
$^{\$}$Significant compared with 11–13 years or with overweight, where appropriate, at $P < 0.05$.
$^{@}$Significant compared with ≥14 years or with obese, where appropriate, at $P < 0.05$.
$^{\Omega}$Kruskal–Wallis.
$^{£}$Mann–Whitney *U*.
$^{©}$Pearson χ^2.
$^{€}$Pearson χ^2 (Monte Carlo).
$^{¥}$Fisher's exact test.

2–21 years ranges from 126 to 140 mL/min/1.73 m^2 [21, 22]. Consistent with previous studies, the mean eGFR in our study population was 132.58 ± 25.48 mL/min/1.73 m^2 in boys and 125.03 ± 19.71 mL/min/1.73 m^2 in girls.

In our study, the mean eGFR was found to be significantly lower in younger age groups. Increased rate of obesity in younger age groups had probably resulted in lower mean eGFR in younger age groups. In addition, percentages of children with eGFR <90 and <75 mL/min/1.73 m^2 were consistently highest in the 5–7 years age group which draws our attention to the younger age groups that involve the risk of CKD underlying congenital abnormalities of kidney and urinary tract (CAKUT) (Table 4).

In relatively recent hospital-based studies, the prevalence of CKD was 74.7 p.m.a.r.p. in Italy (0–19 years; GFR < 75 mL/min/1.73 m^2), 56 p.m.a.r.p. in Belgium (0–19 years; CKD 3–5) and 71.1 p.m.a.r.p. in Spain (0–17 years; CKD 2–5), that is <0.01% [4, 6, 7]. The tertiary paediatric nephrology centre in Kuwait provided data on children aged 0–15 years with a GFR <50 mL/min/1.73 m^2 [23]. The mean incidence was found to be as high as 38 p.m.a.r.p., whereas the prevalence increased from 188 in 1996 to a rate as high as 329 p.m.a.r.p. in 2003.

Our data revealed a prevalence of 0.78% (95% CI: 0.50–1.16) for eGFR < 75 mL/min/1.73 m^2, and 0.13% (95% CI: 0.035–0.33) for eGFR <60 mL/min/1.73 m^2 (CKD Stages 3–5). Even if paediatric CKD is an underdiagnosed problem, the prevalence of CKD in our study is 25–100 times greater than that found in previous hospital-based studies. This suggests that a vast majority of these children will never develop symptomatic CKD, at least during childhood. These children may develop CKD when they are young adults.

A possible explanation is the view that nephrologists probably under-diagnose CAKUT in young adult patients, and this diagnosis can account for many of the 30% that currently have no specified primary renal disease in young adults with CKD [24]. It is known that while paediatric registries have reduced the number of children with 'no specific diagnosis' from 39% in 1976 to fewer than 5%, the adult registries still report rates of 20–27%, which rise to 28–36% when all unspecified groups, predominantly 'glomerulonephritis (GN) (not examined histologically)', are considered together [24]. According to the United States Renal Data System data, CAKUT falls from 31% for ages 0–19 years to only 5% for ages 20–30 years [25]. Generally, eGFR declines relatively slowly in the CAKUT groups, at 2–3 mL/min/year [26–28], suggesting that patients with less severe CAKUT will not reach end-stage until adulthood.

Another important result of our study was that no association was found between eGFR and hypertension or proteinuria. Although it was reported that hypertension and proteinuria are the most important independent risk factors for progression of renal disease in children as well as in adults [29–31], there are no evidence-based data for early stages of CKD, except diabetes mellitus, in children. In adults, hypertension and proteinuria (usually related with diabetes) are two important

causes of CKD; this pattern was also observed in the adult stratum of the CREDIT study [11]. In children, however, these findings are usually results of glomerular diseases. As Hogg [32] pointed out, although mass screening programmes are now well established in Japan, Taiwan and Korea, there is a movement away from mass screening to detect CKD in children and adolescents in North America and Europe. Our data also support the view that screening programmes for early stages of CKD in children that recommend using dipstick proteinuria and hypertension should be reconsidered.

Hypertension and obesity in children and adolescents have increased over the past few decades and many reports have highlighted the close relationship of this with kidney function. That is why examining GFR and CKD in the general paediatric population is worthwhile. In our study, the frequencies of overweight and obese children were 9.3 and 8.9%, respectively; increasing BMI was associated with a lower eGFR. The ratio of children with GFR <90 and <75 mL/min/1.73 m^2 was higher in obese patients, but it did not reach statistical significance (Table 4).

Hypertension is not included in the definition of CKD, but it should be noted that high BP is a common consequence and may be a presenting sign of CKD in children and adolescents. In the adult stratum of the study (CREDIT study), hypertension was found to be associated with lower levels of mean GFR and higher rate of CKD 3–5 [11]. In contrary, such an association was not present for children and adolescents in our study. However, the overall prevalence of hypertension was 6.1% which is another threatening factor for the progressive kidney and cardiovascular damage.

We are aware of the limitations of our study: eGFR was based on a single creatinine measurement and no imaging studies have been performed which might have resulted in over- and under-diagnosis, respectively. Despite its limitations, this is the first population-based field study focusing to detect CKD in children and it supported the idea that CKD is an under-reported and/or under-diagnosed public health problem.

Although there are an increasing amount of studies on CKD in children in recent years, there are still limited data, especially on the earlier stages of CKD. The present study revealed that approaches focusing on patients in tertiary centres are likely to lead to patients being missed at early stages of CKD. A better knowledge of the epidemiology of CKD in children is essential in order to make early diagnosis and develop preventive and therapeutic measures, not only in children but also in young adults.

Supplementary data

Supplementary data are available online at http://ndt.oxfordjournals.org.

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References

1. Hogg RJ, Furth S, Lemley KV *et al*. National Kidney Foundation's Kidney Disease Outcomes Quality Initiative clinical practice guidelines for chronic kidney disease in children and adolescents: evaluation, classification, and stratification. *Pediatrics* 2003; 111: 1416–1421.
2. Deleau J, Andre JL, Briancon S *et al*. Chronic renal failure in children: an epidemiological survey in Lorraine (France) 1975–1990. *Pediatr Nephrol* 1994; 8: 472–476.
3. Esbjörner E, Berg U, Hansson S. Epidemiology of chronic renal failure in children: a report from Sweden 1986–1994. Swedish Pediatric Nephrology Association. *Pediatr Nephrol* 1997; 11: 438–442.
4. Ardissino G, Daccò V, Testa S *et al*. Epidemiology of chronic renal failure in children: data from the ItalKid project. *Pediatrics* 2003; 111: e382–e387.
5. Bek K, Akman S, Bilge I *et al*. Chronic kidney disease in children in Turkey. *Pediatr Nephrol* 2009; 24: 797–806.
6. Mong Hiep TT, Ismaili K, Collart F *et al*. Clinical characteristics and outcomes of children with stage 3–5 chronic kidney disease. *Pediatr Nephrol* 2010; 25: 935–940.
7. Areses Trapote R, Sanahuja Ibáñez MJ, Navarro M, Investigadores Centros Participantes en el REPIR II. Epidemiology of chronic kidney disease in Spanish pediatric population. REPIR II Project. *Nefrologia* 2010; 30: 508–517.
8. Lagomarsimo E, Valenzuel A, Cavagnaro F *et al*. Chronic renal failure in pediatrics 1996 Chilean survey. *Pediatr Nephrol* 1999; 13: 288–291.
9. Anochie I, Eke F. Chronic renal failure in children: a report from Port Harcourt, Nigeria (1985–2000). *Pediatr Nephrol* 2003; 18: 692–695.
10. Hamed RM. The spectrum of chronic renal failure among Jordanian children. *J Nephrol* 2002; 15: 130–135.
11. Suleymanlar G, Utas C, Arinsoy T *et al*. A population-based survey of Chronic REnal Disease In Turkey—the CREDIT study. *Nephrol Dial Transplant* 2011; 262: 1862–1871.
12. Centers for Disease Control and Prevention/National Center for Health Statistics (CDC/NCHS). CDC growth charts: United States, 2000. http://www.cdc.gov/growthcharts
13. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics* 2004; 114: 555–576.
14. Bertil Glader. Chapter 447: the anemias. In: Kliegman RM, Behrman RE, Jenson HB *et al*. (eds). *Nelson Textbook of Pediatrics*, 18th edn. Philadelphia, PA: Saunders Elsevier, 2007: 2003.
15. Meites S (ed). *Pediatric Clinical Chemistry, Reference (Normal) Values*, 3rd edn. Washington, DC: American Association for Clinical Chemistry, 1989.
16. Neal WA. Disorders of lipoprotein metabolism and transport. In: Kliegman RM, Behrman RE, Jenson HB *et al*. (eds). *Nelson Textbook of Pediatrics*, 18th edn. Philadelphia, PA: Saunders Elsevier, 2007, p. 589.
17. Schwartz GJ, Haycock GB, Edelmann CM, Jr *et al*. A simple estimate of glomerular filtration rate in children derived from body length and plasma creatinine. *Pediatrics* 1976; 58: 259–263.
18. Schwartz GJ, Gauthier B. A simple estimate of glomerular filtration rate in adolescent boys. *J Pediatr* 1985; 106: 522–526.

19. Fivush BA, Jabs K, Neu AM *et al*. Chronic renal insufficiency in children and adolescents: the 1996 annual report of NAPRTCS. North American Pediatric Renal Transplant Cooperative Study. *Pediatr Nephrol* 1998; 12: 328–337.
20. Counahan R, Chantler C, Ghazali S *et al*. Estimation of glomerular filtration rate from plasma creatinine concentration in children. *Arch Dis Child* 1976; 51: 875–878.
21. Atiyeh BA, Dabbagh SS, Gruskin AB. Evaluation of renal function during childhood. *Pediatr Rev* 2001; 17: 175–179.
22. Heilbron DC, Holliday MA, Al-Dahwi A *et al*. Expressing glomerular filtration rate in children. *Pediatr Nephrol* 1991; 5: 5–11.
23. Al-Eisa A, Naseef M, Al-Hamad N *et al*. Chronic renal failure in Kuwaiti children: an eight-year experience. *Pediatr Nephrol* 2005; 20: 1781–1785.
24. Neild GH. What do we know about chronic renal failure in young adults? I. Primary renal disease. *Pediatr Nephrol* 2009; 24: 1913–1919.
25. U.S. renal data system, USRDS 2005. *Annual Data report: Atlas of End-Stage Renal Disease in the United States, National Institutes of Health*. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, 2005.
26. Neild GH, Thomson G, Nitsch D *et al*. Renal outcome in adults with renal insufficiency and irregular asymmetric kidneys. *BMC Nephrol* 2004; 5: 12.
27. Gonzalez Celedon C, Bitsori M, Tullus K. Progression of chronic renal failure in children with dysplastic kidneys. *Pediatr Nephrol* 2007; 22: 1014–1020.
28. Wingen AM, Fabian-Bach C, Schaefer F *et al*. Randomised multicentre study of a low-protein diet on the progression of chronic renal failure in children. European Study Group of Nutritional Treatment of Chronic Renal Failure in Childhood. *Lancet* 1997; 349: 1117–1123.
29. Harambat J, van Stralen KJ, Kim JJ *et al*. Epidemiology of chronic kidney disease in children. *Pediatr Nephrol* 2012; 27: 363–373.
30. Jafar TH, Stark PC, Schmid CH *et al*., AIPRD Study Group. Progression of chronic kidney disease: the role of blood pressure control, proteinuria, and angiotensin-converting enzyme inhibition: a patient-level meta-analysis. *Ann Intern Med* 2003; 139: 244–252.
31. Ardissino G, Testa S, Daccò V *et al*., Ital Kid Project. Proteinuria as a predictor of disease progression in children with hypodysplastic nephropathy. Data from the Ital Kid Project. *Pediatr Nephrol* 2004; 19: 172–177.
32. Hogg RJ. Screening for CKD in children: a global controversy. *Clin J Am Soc Nephrol* 2009; 4: 509–515.

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