We analysed perinatal parameters taken from the Methods.

Low birth weight has been identified as a risk factor for chronic kidney disease (CKD). Diseases were classified as congenital with onset after 3 months of life (n = 260; 60%), hereditary (n = 82; 19%) and acquired CKD (n = 93; 21%).

Accepted Access publication 31 May 2010
doi: 10.1093/ndt/gfq300
© The Author 2010. Published by Oxford University Press on behalf of ERA-EDTA. All rights reserved. This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (http://creativecommons.org/licenses/by-nc/2.5), which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.
Results. The rates of prematurity and small for gestational age (SGA) were elevated in children with congenital (39.3% and 29.2%), hereditary (24.7% and 22.6%) and acquired CKD (15.5% and 29.3%); these compared to 8% (for both) in the normal population. Newborns with congenital CKD had a significantly lower gestational age [median 38 weeks, interquartile range (IQR) 36–40 weeks] than those with hereditary (39.9 weeks, IQR 37.5–40 weeks) or acquired CKD (40 weeks, IQR 38–40 weeks; P < 0.001). Median birth weight and length were lower in newborns with congenital than in hereditary and acquired diseases [2975 g (IQR 2460–3420 g) versus 3250 g (IQR 2740–3580 g) and 3260 g (IQR 2858–3685 g) (P < 0.01); 49 cm (IQR 47.52) versus 50 cm (IQR 48–52.8) and 51 cm (IQR 49–53) (P < 0.01)]. Head circumference was smaller (P < 0.05), and Apgar scores were lower (P < 0.005) in newborns with congenital diseases than in hereditary and acquired diseases.

Conclusions. Children with congenital CKD had the highest rate of prematurity, a significantly lower birth weight, length, head circumference and Apgar score than newborns with hereditary or acquired CKD. Irrespective of the aetiology of CKD, all of the children had a significantly higher rate of SGA and prematurity than the reference population. We conclude that both SGA and prematurity predispose for advanced renal disease in childhood and that fetal kidney disease impairs fetal growth.

Keywords: chronic kidney disease; fetal programming; low birth weight; prematurity; small for gestational age

Introduction

Intrauterine growth retardation is multifactorial and results from adverse antenatal conditions including maternal health, placental function and fetal health [1,2]. These are known risk factors for increased morbidity and mortality in fetal, neonatal and adult life [3–5]. Intrauterine growth retardation has been associated with a reduced nephron number [6], which can contribute to arterial hypertension and cardiovascular morbidity [7–9]. Recent studies suggest that low birth weight also predisposes to chronic kidney disease (CKD) in later life [3,10]. However, there is a lack of data as to whether, vice versa, fetal renal impairment may lead to intrauterine growth failure and prematurity. Furthermore, the impact of the aetiology of renal disease on intrauterine growth retardation has not been described in the literature. Therefore, we analysed the prevalence of prematurity, small for gestational age (SGA), and anthropometrical and clinical birth data of children with different causes and time of onset of CKD.

Materials and methods

Five hundred and forty-children with CKD stages 3–5 [11] were investigated between 1998 and 2009 in the Department of Paediatric Nephrology of Hannover Medical School and the University Hospital Charité, Berlin, Germany. Of these 540 children, 105 were excluded because of incomplete birth documentation (n = 61), twin or triple pregnancies (n = 13), chromosomal abnormalities (n = 15), unclear diagnosis of renal diseases (n = 13), or lack of referential data with African and native South American ethnicity (n = 3).

Congenital renal diseases were defined as disorders present during fetal life and clinically overt within the first 3 months of life; this includes genetically determined disorders as well as diseases without a known genetic background such as obstructive uropathy. Hereditary renal diseases were defined as genetically determined but were not clinically apparent in the first 3 months of life. Acquired renal diseases had no known genetically determined background and were absent during infancy.

The birth data of the remaining 435 patients, with an estimated glomerular filtration rate <60 mL/min/1.73 m2 using the Schwartz formula [12], differed significantly from the norm (Table 1). Therefore, we made a subgroup analysis of children with congenital renal diseases (260 patients, 60%), hereditary renal diseases (93 patients, 21%) and acquired renal diseases (82 patients, 19%) in order to evaluate differences in perinatal parameters according to the aetiology of the primary renal diseases and the onset of disease manifestation (early onset in fetal life versus onset after 3 months of life).

Children with congenital CKD had renal dysplasia/hypoplasia with or without urinary tract malformations (41%), obstructive uropathy (28%), congenital nephrotic syndrome (12%), autosomal recessive polycystic kidney disease (10%) and other diseases (9%). Hereditary CKD accounted for nephromatosis (46%), cystinosis and oxalosis (29%), factor H-related haemolytic uraemic syndrome (12%), Alport syndrome (8%), and others (5%). Acquired diseases were primary focal and segmental glomerulosclerosis (36%), diarrhoea-associated haemolytic uraemic syndrome (29%), different glomerulopathies (27%), IgA nephropathy, lupus nephritis, membranoproliferative glomerulonephritis, extracapillary proliferative glomerulonephritis and Goodpasture's syndrome), and others (8%).

Patients who died during follow-up were included in the study. All children were Caucasian. Mean age at the last measurement was 12.7 ± 5.5 years (range 0.1–23.3 years). Mean age at start of renal replacement therapy (dialysis or renal transplantation) was 6.8 ± 5.1 years in children with congenital CKD, 10.3 ± 4.0 years in children with hereditary and 8.4 ± 4.4 years in children with acquired CKD.

The Institutional Review Boards of both university hospitals approved this study. Informed patient and/or carer consent was obtained prior to enrolment.

Newborns were classified as SGA if birth weight and/or birth length were below the 10th percentile according to Voigt's National Growth Charts [13]. The local reference data for frequencies of prematurity, SGA and WHO birth weight categories on all pregnancies/year in Lower Saxony (61 025 in 2007) were obtained from the Centre for Quality and Management in Health Care, an institution of the General Medical Council of Lower Saxony, Germany from the years 2000 to 2007 [14].

Birth weight was classified according to WHO categories: appropriate for gestational age (AGA) >2500 g, low birth weight (LBW) <2500 g, very low birth weight (VLBW) <1500 g and extremely low birth weight (ELBW) <1000 g.

Maturity was graduated as ‘over term’ (>42 weeks of gestation), ‘term’ (38–42 weeks of gestation), ‘near term’ (34–37 weeks of gestation), ‘moderate prematurity’ (32–33 weeks of gestation), ‘severe prematurity’ (31–28 weeks of gestation) and ‘extreme prematurity’ (<28 weeks of gestation). Parental height was measured at routine visits.

Birth data (gestational age, Apgar scores at 5 and 10 min, umbilical cord pH, birth weight, length, and head circumference) were taken from the national birth documents, which were taken in the hospital after delivery.

Statistical analysis

Due to the different nature of the variables, parametric and non-parametric tests were applied. Variable normality was tested using the Kolmogorov–Smirnov test and the Shapiro–Wilk test (in cases where the sample size was small to medium). For variables on parametric scales and with a normal distribution for all inferential purposes, the t-test was used. In all other variables, non-parametric statistical tests were used (Mann–Whitney U-test, Kruskal–Wallis one-way analysis of variance and median test).

Group characteristics were presented with the median as the measure of central tendency and the interquartile range (IQR, Q1–Q3) as the measure of variability.

Statistical significance assumption was specified at a level P ≤ 0.05. SPSS version 17.0 was used for data analysis.

For comparison of two proportions expressed only as percentages, MedCalc (MedCalc Software, Belgium) was applied.
Results

Children with congenital CKD had a significantly lower median gestational age (38 weeks, IQR 36–40 weeks) than children with hereditary CKD (median 39.9 weeks, IQR 37.5–40 weeks) and acquired CKD (median 40 weeks, IQR 38–40 weeks) (P < 0.001) (Figure 1).

Birth weight in newborns with congenital CKD was significantly lower (median 2975 g, IQR 2460–3420 g) than in children with hereditary (3250 g, IQR 2740–3580 g) and acquired CKD (3260 g, IQR 2858–3685 g) (P < 0.01) (Figure 1).

Significant differences (P < 0.05) between rates (%) of perinatal parameters in the different groups are marked with A, B, C, D and E.

Table 1. Rates of prematurity, SGA and different categories of low birth weight in 435 newborns with congenital, hereditary and acquired chronic kidney disease compared with the reference population

<table>
<thead>
<tr>
<th></th>
<th>NB with CKD</th>
<th>NB with CKD</th>
<th>NB with CKD</th>
<th>NB with CKD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All (n = 435)</td>
<td>Congenital (n = 260)</td>
<td>Hereditary (n = 93)</td>
<td>Acquired (n = 82)</td>
</tr>
<tr>
<td>Maturity (gestational age in weeks)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Over term (&gt;42)</td>
<td>0.5</td>
<td>0.8</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Term (38–42)</td>
<td>67.7</td>
<td>59.9</td>
<td>75.3</td>
<td>84.5</td>
</tr>
<tr>
<td>Near term (34–37)</td>
<td>24.4</td>
<td>30.5</td>
<td>18.3</td>
<td>13.1</td>
</tr>
<tr>
<td>Moderate prematurity (32–33)</td>
<td>3.8</td>
<td>4.2</td>
<td>4.3</td>
<td>1.2</td>
</tr>
<tr>
<td>Severe prematurity (31–28)</td>
<td>2.9</td>
<td>3.8</td>
<td>2.1</td>
<td>0</td>
</tr>
<tr>
<td>Extreme prematurity (&lt;28)</td>
<td>0.7</td>
<td>0.8</td>
<td>0</td>
<td>1.2</td>
</tr>
<tr>
<td>Missing data</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total prematurity (537)</td>
<td>31.8 DE</td>
<td>39.3 CDE</td>
<td>24.7 BE</td>
<td>15.5 ABE</td>
</tr>
<tr>
<td>WHO birth weight categories</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AGA (&gt;2500 g)</td>
<td>76.6 ED</td>
<td>71.9 ED</td>
<td>78.5 E</td>
<td>90.2 AB</td>
</tr>
<tr>
<td>LBW (&lt;2500 g)</td>
<td>22.9 ED</td>
<td>28 ED</td>
<td>19.8 E</td>
<td>9.8 AB</td>
</tr>
<tr>
<td>VLBW (&lt;1500 g)</td>
<td>3.2 E</td>
<td>4.2 E</td>
<td>2.2</td>
<td>0</td>
</tr>
<tr>
<td>ELBW (&lt;1000 g)</td>
<td>0.9</td>
<td>1.2</td>
<td>0</td>
<td>1.2</td>
</tr>
<tr>
<td>SGA &lt;10th percentile</td>
<td>27.8 E</td>
<td>29.2 E</td>
<td>22.6 E</td>
<td>29.3 E</td>
</tr>
</tbody>
</table>

NB, newborns; AGA, appropriate for gestational age; LBW, low birth weight; VLBW, very low birth weight; ELBW, extremely low birth weight; SGA, small for gestational age.
No statistically significant differences were found between children with hereditary and acquired CKD in birth weight, length, head circumference, Apgar after 5 and 10 min, and gestational age.

Birth parameters of children with CKD stage 3–5 differed from the normal population (Table 1, Figure 4). In all 435 children with CKD, the percentage of premature births (<37 weeks of gestation) was 31.8% and thus higher (P < 0.0001) than in the reference population (~8%). This proportion was 39.3% in children with congenital CKD compared with 24.7% in children with hereditary and 15.5% in children with acquired CKD (Table 1). The frequencies of near term deliveries, moderate, severe, and extreme prematurity and birth weight categories according to the WHO for all children with CKD, children with congenital, hereditary and acquired CKD, and the local reference group are listed in Table 1.

The rate of newborns meeting the criteria for SGA with a birth weight and/or length <10th percentile was between 8.1% and 8.7% during 2000 and 2007 in the reference population, and compared with 29.2% in newborns with congenital CKD (P < 0.0001), 22.6% in hereditary (P < 0.0001) and 29.3% in newborns with acquired CKD (P < 0.0001) (Figure 4).

In order to investigate whether the results in children with congenital, hereditary and acquired CKD were reproducible in a subsample analysis, we analysed the rates of SGA and prematurity in four groups of children with the most frequent single diagnoses: (i) hypoplasia/dysplasia as a congenital disease, (ii) nephropathies as hereditary, (iii) diarrhoea-associated haemolytic uraemic syndrome, and (iv) idiopathic focal and segmental glomerulosclerosis or other glomerulonephritides as acquired diseases. The rate of SGA was 33.3% in newborns with hypoplasia/dysplasia, 20.7% in nephropathies, 21.7% in diarrhoea-associated haemolytic uraemic syndrome and 24.4% in glomerulopathies. The rates of prematurity (<38 weeks of gestation) were 34.3%, 17.1%, 4.3% and 17.8%, respectively.
stage 3 and reduced adaptation to neonatal stress. Intrauterine growth, an increased rate of premature birth most from adverse prenatal conditions, leading to impaired ease. Therefore, children with congenital CKD suffered between children with hereditary and acquired renal disease. No significant differences were found in these parameters newborns with hereditary or acquired CKD. In contrast, significantly lower gestational age, birth weight, length, head circumference, and Apgar score, after 5 and 10 min, than newborns with hereditary or acquired CKD. In contrast, no significant differences were found in these parameters between children with congenital and acquired renal disease. Therefore, children with congenital CKD suffered most from adverse prenatal conditions, leading to impaired intrauterine growth, an increased rate of premature birth and reduced adaptation to neonatal stress.

In our study, the prevalence of SGA in children with CKD stage 3–5 was three times higher than in the reference population (between 8.1% and 8.7% during 2000 and 2007) [14], irrespective of congenital, hereditary or acquired causes of CKD.

As diarrhoea-associated haemolytic uraemic syndrome, idiopathic focal glomerulosclerosis and different forms of glomerulonephritis were the predominant causes of acquired kidney diseases, it may be speculated that children with SGA are more susceptible to developing high-grade CKD in the course of the disease compared with children with appropriate weight/length for gestational age and acquired kidney diseases.

It is unclear if children with low-grade CKD 1 and 2 are more frequently SGA newborns, but SGA has been shown to negatively influence the course of renal diseases such as idiopathic nephrotic syndrome [15], IgA nephropathy [16] and diabetic nephropathy [17]. SGA may therefore be a risk factor for progression of CKD in children.

This study evidences that one-third of children with CKD were born pre-term. Prematurity was significantly more frequent in children with congenital CKD (39.3%) than in children with hereditary (24.7%) and acquired CKD (15.5%). However, children with hereditary and acquired CKD also presented with a 3- and 2-fold elevated rate of pre-term delivery compared to ∼8% in the reference population [14]. These findings suggest that not only SGA but also prematurity is a factor for chronic kidney disease later in childhood. We conclude from our data that nephrologists taking the medical history of their patients should ask for gestational age and other birth parameters.

Compared with the reference population, the rate of low birth weight was significantly elevated only in children with congenital and hereditary CKD. This can be explained by the fact that the majority of premature newborns with acquired CKD were near-term premature and therefore did not meet the criteria of a birth weight <2500 g. Additionally, term newborns meeting the 10th percentile SGA criteria may also present with a birth weight of >2500 g. Therefore, using only low birth weight as a parameter for intrauterine growth retardation is likely to underestimate the whole at-risk population.

Our study shows a >2-fold increased rate of low birth weight in children with congenital and hereditary CKD, and therefore supports the findings of Vikse et al. [3] of an increased risk in newborns with low birth weight for the development of end-stage renal disease during the first 14 years of life. Furthermore, our data extend the findings of Vikse et al. [3] by showing that both the prevalence of SGA and prematurity was significantly elevated in children with CKD 3–5.

The stratification of our study may be criticized because of a possible overlap between congenital, hereditary and acquired causes of CKD. Hypoplasia, dysplasia, obstructive uropathy and autosomal recessive polycystic kidney disease are known to have their origin in fetal life due to genetic or non-genetic causes, and these cases were classified as congenital CKD. In a subgroup analysis of only newborns with hypoplasia/dysplasia, we received the same elevated rates of SGA and prematurity as well as in the nephronophthisis patients as a clear-cut hereditary subgroup. Not all children underwent genetic testing, and therefore, our data on acquired CKD may be influenced by unidentified hereditary cases. For example, ∼20% of all children with idiopathic glomerulonephritis and steroid-resistant nephrotic syndrome may suffer from genetic defects [18]. We found an elevated rate of prematurity in the whole group of patients with acquired diseases but not in the subgroup analysis of children with diarrhoea-associated haemolytic uraemic syndrome. In contrast, children with idiopathic focal and segmental glomerulosclerosis and other acquired forms of glomerulonephritis had an elevated SGA rate of 24.4%, and the prematurity rate was 17.8%. We cannot rule out that some children with genetically determined glomerulonephritis and focal and segmental glomerulosclerosis may have been under-diagnosed in our subgroup of acquired CKD.

Irrespective of the three aetiological groups, increased rates of SGA and prematurity in children with CKD 3–5 could clearly be demonstrated, suggesting that SGA and prematurity predispose to high-grade CKD in childhood. Intrauterine growth retardation and prematurity were shown to impair fetal renal development leading to proteinuria, arterial hypertension and reduced glomerular filtration rate in

Fig. 4. Rates of prematurity and SGA in newborns with congenital, hereditary and acquired chronic kidney disease and in the reference population [14].

Discussion

This study demonstrates that children with CKD stages 3–5 differed significantly in their birth parameters from the normal population. The aetiological subgroup analysis showed that children with congenital CKD had a significantly lower gestational age, birth weight, length, head circumference, and Apgar score, after 5 and 10 min, than newborns with hereditary or acquired CKD. In contrast, no significant differences were found in these parameters between children with congenital and acquired renal disease. Therefore, children with congenital CKD suffered most from adverse prenatal conditions, leading to impaired intrauterine growth, an increased rate of premature birth and reduced adaptation to neonatal stress.

In our study, the prevalence of SGA in children with CKD stage 3–5 was three times higher than in the reference population (between 8.1% and 8.7% during 2000 and 2007) [14], irrespective of congenital, hereditary or acquired causes of CKD.

As diarrhoea-associated haemolytic uraemic syndrome, idiopathic focal glomerulosclerosis and different forms of glomerulonephritis were the predominant causes of acquired kidney diseases, it may be speculated that children with SGA are more susceptible to developing high-grade CKD in the course of the disease compared with children with appropriate weight/length for gestational age and acquired kidney diseases.

It is unclear if children with low-grade CKD 1 and 2 are more frequently SGA newborns, but SGA has been shown to negatively influence the course of renal diseases such as idiopathic nephrotic syndrome [15], IgA nephropathy [16] and diabetic nephropathy [17]. SGA may therefore be a risk factor for progression of CKD in children.

This study evidences that one-third of children with CKD were born pre-term. Prematurity was significantly more frequent in children with congenital CKD (39.3%) than in children with hereditary (24.7%) and acquired CKD (15.5%). However, children with hereditary and acquired CKD also presented with a 3- and 2-fold elevated rate of pre-term delivery compared to ∼8% in the reference population [14]. These findings suggest that not only SGA but also prematurity is a factor for chronic kidney disease later in childhood. We conclude from our data that nephrologists taking the medical history of their patients should ask for gestational age and other birth parameters.

Compared with the reference population, the rate of low birth weight was significantly elevated only in children with congenital and hereditary CKD. This can be explained by the fact that the majority of premature newborns with acquired CKD were near-term premature and therefore did not meet the criteria of a birth weight <2500 g. Additionally, term newborns meeting the 10th percentile SGA criteria may also present with a birth weight of >2500 g. Therefore, using only low birth weight as a parameter for intrauterine growth retardation is likely to underestimate the whole at-risk population.

Our study shows a >2-fold increased rate of low birth weight in children with congenital and hereditary CKD, and therefore supports the findings of Vikse et al. [3] of an increased risk in newborns with low birth weight for the development of end-stage renal disease during the first 14 years of life. Furthermore, our data extend the findings of Vikse et al. [3] by showing that both the prevalence of SGA and prematurity was significantly elevated in children with CKD 3–5.

The stratification of our study may be criticized because of a possible overlap between congenital, hereditary and acquired causes of CKD. Hypoplasia, dysplasia, obstructive uropathy and autosomal recessive polycystic kidney disease are known to have their origin in fetal life due to genetic or non-genetic causes, and these cases were classified as congenital CKD. In a subgroup analysis of only newborns with hypoplasia/dysplasia, we received the same elevated rates of SGA and prematurity as well as in the nephronophthisis patients as a clear-cut hereditary subgroup. Not all children underwent genetic testing, and therefore, our data on acquired CKD may be influenced by unidentified hereditary cases. For example, ∼20% of all children with idiopathic glomerulonephritis and steroid-resistant nephrotic syndrome may suffer from genetic defects [18]. We found an elevated rate of prematurity in the whole group of patients with acquired diseases but not in the subgroup analysis of children with diarrhoea-associated haemolytic uraemic syndrome. In contrast, children with idiopathic focal and segmental glomerulosclerosis and other acquired forms of glomerulonephritis had an elevated SGA rate of 24.4%, and the prematurity rate was 17.8%. We cannot rule out that some children with genetically determined glomerulonephritis and focal and segmental glomerulosclerosis may have been under-diagnosed in our subgroup of acquired CKD.

Irrespective of the three aetiological groups, increased rates of SGA and prematurity in children with CKD 3–5 could clearly be demonstrated, suggesting that SGA and prematurity predispose to high-grade CKD in childhood. Intrauterine growth retardation and prematurity were shown to impair fetal renal development leading to proteinuria, arterial hypertension and reduced glomerular filtration rate in

Fig. 4. Rates of prematurity and SGA in newborns with congenital, hereditary and acquired chronic kidney disease and in the reference population [14].
Perinatal parameters and kidney disease in childhood

The underlying mechanisms leading to advanced kidney failure are likely associated with a reduction of kidney volume [21], nephron number [6,8,9] and an increase of glomerular volume [22,23] in neonates with low birth weight.

We ascertained that congenital CKD was associated with disturbed fetal growth, and it may be asked: ‘What came first, the chicken or the egg?’ The mechanisms affecting antenatal growth in a fetus with renal disease are poorly understood. It is unclear if lower birth weight, length, head circumference and Apgar scores in newborns with congenital CKD were only attributable to prematurity. We suggest that there may be additional, as yet unknown, mechanisms in children with congenital CKD influencing birth parameters and postnatal growth and development.

Parental height was not found to play a major role in influencing birth parameters in our three study groups. The severity of growth retardation at birth may be a predictor for postnatal growth failure in children with CKD 3–5. Especially in children with congenital CKD, antenatal growth impairment aggravates the growth deficit in childhood, which is usually most severe in the first year of life [24]. Therefore, adequate medical treatment including nutrition, treatment of metabolic acidosis, renal bone disease, anaemia and early implementation of growth hormone therapy should be ensured in those infants with congenital kidney diseases and reduced birth parameters. However, forced catch-up growth in the first years of life may be a major determinant of renal damage [25,26]. Therefore, we analysed data on height and weight at the age of 2 years which was available in a subsample of 135 children with CKD. Children with congenital CKD (n = 96) had a mean height of 82.9 ± 4.0 cm and weight of 11.1 ± 1.7 kg at 2 years of age and compared with 84.4 ± 5.9 cm and 11.7 ± 2.2 kg in children with hereditary CKD (n = 24) and 85.4 ± 2.5 cm and 11.5 ± 1.2 kg in children with acquired CKD (n = 15). We conclude from this data that weight catch-up occurred in infants with congenital CKD, whereas the height difference between the three groups persisted despite early growth hormone therapy in 17% of infants with congenital CKD. Further prospective studies on antenatal and postnatal growth in healthy children and in children with CKD should be initiated in order to elucidate the relation between early catch-up growth and postnatal renal dysfunction.

Newborns with congenital CKD had a significantly lower birth weight, length, head circumference and Apgar score. Thus, we conclude that fetal kidney dysfunction may cause fetal growth impairment. Children with acquired CKD also had a significantly higher rate of prematurity and SGA than the reference population, therefore raising the question of whether both prematurity and SGA predispose to renal disease later in life. Further investigations on the underlying mechanisms of individual predisposition and possible genetic or environmental determinants should be initiated, as well as analysis of the long-term effect of antenatal growth impairment on postnatal growth and development in these children. Using only low birth weight as a parameter of intrauterine growth retardation will underestimate the at-risk population.

Routine postnatal screening programmes should rule out kidney disease and arterial hypertension in children born preterm, SGA and/or with low birth weight. In addition, nephrologists should routinely ask for birth data in adolescent and adult patient medical history. Our results suggest that SGA and prematurity-induced susceptibility to renal diseases begins in childhood. Additionally, SGA, prematurity and low birth weight are likely to be important risk factors for postnatal growth failure.

Acknowledgements. This project was supported by a grant from the German Federal Ministry of Education and Research (BMBF), reference number 01EO0802. The content of this article is the sole responsibility of the authors.

Conflict of interest statement. None declared.

References

Association of risk factors for cardiovascular disease and glomerular filtration rate: a community-based study of 4925 adults in Beijing

Fan Wang, Ping Ye, Leiming Luo, Wenkai Xiao and Hongmei Wu

Department of Geriatric Cardiology, Chinese PLA General Hospital, Beijing, 100853, China

Correspondence and offprint requests to: Ping Ye; E-mail: yeping@sina.com

Abstract

Background. Several large prospective studies have reported that a low estimated glomerular filtration rate (eGFR) or chronic kidney disease (CKD) is independently associated with cardiovascular disease (CVD) events and all-cause mortality in high-risk populations. However, findings from community-based population studies are scarce and inconsistent. We investigated the level of eGFR and the relationship between CVD risk factors and eGFR or CKD in the population of Beijing, China.

Methods. This is a community-based observational survey in residents from three communities in Beijing for a routine health status checkup. Out of 5100 individuals who were eligible for inclusion, 4925 (96.57%) had complete data and were investigated the level of eGFR and the associated factors of reduced renal function. 2085 individuals with albuminuria values were included in the analyses on the associated factors of CKD. A questionnaire was used for risk factors of CVD. Anthropometry and blood pressure were measured. Serum creatinine, total cholesterol, triglyceride (TG), low-density lipoprotein cholesterol, high-density lipoprotein cholesterol and serum glucose were detected. The urine albumin–creatinine ratio (ACR) was used as an expression for albumin excretion. The oral glucose tolerance test was performed for the participants with no history of diabetes to diagnose diabetes. eGFR was evaluated by the Chinese modified Modification of Diet in Renal Disease equation. Reduced renal function was defined as normal renal function: eGFR ≥90 mL/min/1.73 m²; mild reduced renal function: eGFR 60–89 mL/min/1.73 m²; moderate to severe reduced renal function: eGFR <60 mL/min/1.73 m². CKD was diagnosed as eGFR <60 mL/min/1.73 m² or albuminuria was present.

Results. The prevalence of mild reduced renal function (eGFR 89–60 mL/min/1.73 m²), moderate to severe reduced renal function (eGFR <60 mL/min/1.73 m²) and CKD was 31.50% (1557/4925), 13.27% (652/4925) and 18.90% (394/2085) in the present study, respectively. The proportion of risk factors was higher in the low level of eGFR. Risk factors that exposed to reduced renal function were slightly different between male and female. The results of multivariate logistic regression analysis showed older age [increased by 10 years; odds ratios (OR)=1.22], male gender (OR=1.38), diabetes (OR=1.67), hyperten-