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Original Article

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Antenatal oligohydramnios of renal origin: long-term outcome

Ilka Klaassen[1], Thomas J. Neuhaus[2], Dirk E. Mueller-Wiefel[1] and Markus J. Kemper[1,2]

[1]Pediatric Nephrology, University Medical Center Hamburg-Eppendorf, Martinistr. 52, D-20246 Hamburg, Germany and [2]Pediatric Nephrology, University Children's Hospital, Steinwiesstr. 75, CH-8032 Zurich, Swizerland

Abstract

Background. Prognosis of fetuses with renal oligohydramnios (ROH) is often still regarded as poor. Neonatal complications and the long-term follow-up of fetuses with ROH in two pediatric centres are described.

Method. 23 fetuses (16 males, 7 females) were included as patients. Primary diseases included congenital anomalies of the kidney and urinary tract ($n=16$), autosomal recessive polycystic kidney disease ($n=4$) and renal tubular dysgenesis ($n=3$). The analysis includes retrospective chart review.

Results. Seven children died (30%), the majority ($n=4$, 17%) within the neonatal period due to pulmonary hypoplasia and renal insufficiency. Fourteen patients (61%) required postnatal mechanical ventilation for a median of 4 (range 1–60) days; 11 infants had an associated pneumothorax. All 16 surviving children have chronic kidney disease (CKD) at a current median age of 5.7 years (range 0.5–14.5), managed conservatively in eight patients [median glomerular filtration rate 51 (range 20–78) ml/min/1.73 m^2]. Eight patients reached end-stage renal disease at a median age of 0.3 years (range 2 days to 8.3 years), including one patient with pre-emptive kidney transplantation. Five of the patients requiring dialysis underwent successful renal transplantation at a median age of 3.5 years (range 2.5–4). Growth was impaired in seven children requiring growth hormone treatment. Cognitive and motor development was normal in 12 (75%) of the 16 patients and showed a delay in four children, including two with associated syndromal features.

Conclusion. ROH is not always associated with a poor prognosis and long-term outcome in survivors is encouraging. The high incidence of neonatal complications and long-term morbidity due to CKD requires a multidisciplinary management of these children.

Correspondence and offprint requests to: Markus J. Kemper, MD, Pediatric Nephrology, University Medical Center, Hamburg-Eppendorf, Martinistr. 52, D-20246 Hamburg, Germany.
Email: kemper@uke.uni-hamburg.de

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Introduction

Congenital anomalies of the kidney and urinary tract (CAKUT) are detected frequently in up to 1% of newborns, and nowadays diagnosis is often established before birth by fetal ultrasound. If CAKUT occurs unilaterally, e.g. hydronephrosis due to ureteropelvic or ureterovesical junction obstruction, or multicystic renal dysplasia, prognosis is generally good [1,2].

Bilateral renal disease with oligohydramnios, however, indicates significant global fetal renal dysfunction and is a risk factor for the development of pulmonary hypoplasia [3,4]. Outcome for fetuses with renal oligohydramnios (ROH) therefore is regarded as poor. In a series from the Mayo Clinic, 18 of 52 (32%) children with CAKUT had oligohydramnios and all children died, including six intrauterine deaths [5]. As fetal and neonatal survival is at risk, termination of pregnancy is frequently practised in this situation [6].

This is in striking contrast to the dramatic progress that has been made in neonatal intensive care including pulmonary management. Also, recent advances in treatment of infants and children with chronic kidney disease (CKD) and end-stage renal disease (ESRD) has improved prognosis also for infants with renal insufficiency considerably [7–10]. Precise information, however, on the clinical situation on short- and long-term outcome data for fetuses presenting with ROH is scarce. In some series of patients with urological disorders, children with ROH are probably included; however, specific details are missing. For instance, in the series by Drozdz *et al.* [11] three of 20 boys with posterior urethral valves required mechanical ventilation; however, no details on the presence of oligohydramnios are given.

The aim of our study was to obtain comprehensive data on short- and long-term outcome of 23 fetuses

with ROH that have been followed in our centres since 1990. This included a detailed analysis of (i) neonatal pulmonary and renal course, (ii) long-term renal outcome as well as (iii) extrarenal morbidity (growth and development).

Patients and methods

A total of 23 infants (16 males, 7 females) with an antenatal diagnosis of ROH that were followed in our centres after birth were included. Data were analysed retrospectively by chart review. Fourteen infants were delivered in our institutions and nine were referred from regional hospitals, all for further treatment of renal disease. Patients were diagnosed between 1990 and 2005. Twelve were followed at the University of Hamburg Children's Hospital (center 1) and 11 at the Children's Hospital of the University of Zurich (center 2). Median gestational age was 37 weeks (range 34–40 weeks). Patient details are presented in Table 1.

Oligohydramnios was defined as the generalized decrease in amniotic fluid. The vertical × horizontal diameter of the largest pocket was less than $2 \times 1\,cm^2$ in ultrasound investigation [12,13]. The oligohydramnios was detected at a median of 30 weeks gestation (range 14–37) in 21 cases: four in the second and 17 in the third trimenon. In two pregnancies, diagnosis of ROH was established immediately prior to birth due to poor maternal compliance with routine antenatal obstetrical visits.

Most children ($n = 16$) had CAKUT: eight infants had posterior urethral valves with associated renal dysplasia, five had bilateral renal dysplasia including one patient with VACTERL association, one patient had bilateral multicystic dysplastic kidneys and two patients had prune-belly syndrome with associated renal anomalies. In addition, four patients had autosomal recessive polycystic kidney disease (ARPKD) and three siblings had renal tubular dysgenesis.

For comparison of parametrical variables the Mann–Whitney U-test was used and for survival analysis the log-rank test (Mantel Cox) were computed using SPSS 13.0. A $P < 0.05$ was considered to be statistically significant.

Results

Mortality

Of the 23 patients seven died (30%; Figure 1 and Table 1). Four patients (17%) died in the neonatal period due to pulmonary hypoplasia and terminal renal insufficiency. None of these infants was dialysed. Three patients died later: one patient with additional multiple cardiac anomalies due to cardiomyopathy at 3.5 months, one patient with associated VACTERL anomalies due to pulmonary hypertension and renal insufficiency at the age of 5 months and one patient with ARPKD due to sepsis at the age of 14.5 months.

Non-survivors had a significantly earlier diagnosis of ROH than survivors (median 25 weeks, range 14–35 *vs* 31, 19–37; $P < 0.05$. Of four fetuses with ROH diagnosed in the second trimenon, two died compared with 5/17 fetuses diagnosed in the third trimenon. Using the diagnostic median of 30 weeks as cutoff, diagnosis of ROH prior to this was associated with a higher overall mortality (6 of 10) than diagnosis after 30 weeks gestation (1 of 11; $P < 0.02$; Figure 2). Also, neonatal mortality was more frequent in fetuses with a diagnosis prior to 30 weeks (4/10 *vs* 0/11, $P < 0.03$).

Neonatal course

Pulmonary outcome. Fourteen neonates (61%), including six of the seven non-survivors required artificial ventilation for a median of 4 (range 1–60) days, including one child with nasal continuous positive airway pressure (CPAP). Four neonates with antenatally suspected pulmonary hypoplasia did not need ventilation. Pneumothorax occurred in 11 infants, including one patient that did not require respiratory support.

Renal outcome. Neonatal renal dysfunction occurred in 20 of 23 children (87%). Most frequently, electrolyte imbalances and metabolic acidosis ($n = 14$) were present. Hypertension was diagnosed in the neonatal period in two infants with ARPKD. Severe renal dysfunction occurred in eight patients, including three of the non-survivors, who were not dialysed (patients 2–4).

Four patients (all survivors) required peritoneal dialysis in the neonatal period; however, only two for the first postnatal weeks. The other two patients started dialysis at day 3 and 4, respectively, and continued renal replacement therapy without major problems until renal transplantation (RT). One surviving patient with severe renal dysfunction suffered from superimposed pre-renal failure, that could be managed conservatively.

Long-term course

Long-term renal outcome and morbidity (Figure 3). The surviving 16 children have a current median age of 5.7 (range 0.5–14.5) years. All developed CKD, which was managed conservatively in eight patients (50%), with a median glomerular filtration rate (GFR) of $51\,ml/min/1.73\,m^2$ (range 20–78).

Eight of 16 patients (50%) required renal replacement therapy at a median age of 0.3 years (range 2 days to 8.3 years); seven were started on peritoneal dialysis; however, two later switched to haemodialysis. One patient underwent pre-emptive kidney transplantation (KT) at age 8 years. Five of the children requiring dialysis received a successful renal graft at a median age of 3.5 years (range 2.7–4). Early diagnosis of ROH did not predict the risk of ESRD.

Table 1. Individual patient data

Patient	Sex	Renal and extrarenal diagnosis	First diagnosis of ROH (GW)	Gestation (weeks)	Mechanical ventilation (days)	Pneumo-thorax	Neonatal pulmonary and renal problems and outcome	Renal follow-up	Development and growth; duration of follow-up
1	m	PUV	18	39	No	No	Respiratory failure, died in delivery room		
2	m	PUV, bilateral renal dysplasia	28	38	Yes (28)	Yes	ESRD (no dialysis). Died after 4 weeks (HMD)		
3	f	Tubular dysgenesis, sister of 16	25	35	Yes (4)	Yes	ESRD (no dialysis) and respiratory failure, died after 4 days (HMD)		
4	f	Tubular dysgenesis, sister of 16	26	34	Yes (2)	Yes	ESRD (no dialysis) and respiratory failure, died after 2 days		
5	f	ARPKD	29	35	Yes (7)	Yes	Ventilation, no renal problems	Hypertension, cardiomyopathy, CKD, died after 14.5 months (pseudomonas sepsis)	
6	m	ARPKD, Multiple cardiac anomalies	35	39	No	No	CKD	Died after 3.5 months due to CKD and cardiomyopathy	
7	m	Vacterl-association with bilateral renal dysplasia	14	34	No	No	Colostomy, pulmonary hypertension	Died after 5 months of CKD and pulmonary hypertension	
8	m	Bilateral renal dysplasia	33 (amniotic infusion)	40	No	No	CKD, Acidosis	ESRD, PD started age 8.3, switched to HD age 8.7	GH since age 3.9, Height SDS −1.59, 11.8 years
9	m	ARPKD	32	37	Nasal CPAP (1)	No	CKD, Hypertension	GFR 20 ml/min/1.73 m^2	Height SDS −0.59, 7.5 years
10	f	Bilateral renal dysplasia	28	38	Yes (22)	No	ESRD, PD since day 4	PD started day 4 to age 2.9 until KT	GH age 1.5–2.9, Height SDS −2.4, 6.1 years
11	m	PUV, bilateral renal dysplasia	20	37	Yes (5)	Yes	acute pre-renal failure, feeding problems	PD age 1.2, KT age 3.5	Developmental delay, GH age 1.0 to KT, Height SDS −2.65, 6.1 years
12	f	Bilateral renal dysplasia, Syndromal features	36	40	No	No	CKD	CKD, GFR 52 ml/min/1.73 m^2	Development delay, Height SDS −2.6, 5 years

13	m	PUV, prune-belly syndrome	19 (amniotic infusion)	35	Yes (5)	No	CKD	ESRD, PD age 0.2, switched to HD age 2.3, KT age 4.0	GH age 1.0 to KT, Height SDS −1.2, 5.2 years
14	m	PUV, Trisomy 21 syndrome	unknown	36	No	No	CKD, recurrent pyelonephritis	CKD, GFR 78 ml/min/1,73m^2	Developmental delay, Height SDS −2.7, 3.3 years
15	m	Prune-belly syndrome with renal dysplasia, megacystis	34	35	No	Yes	CKD	PD age 0.5, KT age 3.5	GH age 1.5 to KT, Height SDS −2.9, 4.6 years
16	m	PUV, bilateral renal dysplasia	33	37	Yes (7)	Yes	CKD	PD started age 0.25	Height SDS −0.5, 1.7 years
17	f	Multicystic renal dysplasia	unknown	40	Yes (4)	No	ESRD PD started day 3	PD until KT at age 2.5 years	GH age 1.0 to KT, Height SDS −2.5, 3.4 years
18	m	PUV, VUR	32	34	Yes (60)	Yes	Acute renal failure, PD for 21 day, small-bowel-perforation, myocardial infarction, sepsis	GFR 45 ml/min/1.73 m^2	Mild developmental delay, Height SDS −1.58, 6 months
19	m	PUV, bilateral renal dysplasia	37	38	Yes (3)	Yes	CKD	GFR 69 ml/min/1.73 m^2, Enuresis	Height SDS 0.1, 7.7 years
20	m	Bilateral renal dysplasia, vesico-ureteral reflux	36	40	No	No	CKD	CKD, GFR 50 ml/min/1.73 m^2	Height SDS −1.9, 7.9 years
21	m	ARPKD	30	36	Yes (1)	Yes	CKD, Hypertension	CKD until preemptive KT at age 8.0	GH age 4.0–7.5 Height SDS 0.0, 14.5 years
22	f	Tubular dysgenesis	28	38	Yes (26)	Yes	Acute renal failure, PD for 4 weeks	CKD, GFR 47 ml/min/1.73 m^2, hyperparathyroidism	Height SDS −1.59, 14.3 years
23	m	PUV, bilateral renal dysplasia	32	34	No	No	CKD, maximum. serum creatinine 3.1 mg/dl	GFR 60 ml/min/1,73 m^2	Height SDS 0.18, normal development, 9 months

ARPKD, autosomal recessive polycystic kidney disease; CKD, chronic kidney disease; ESRD, end-stage renal disease; GH, growth hormone; HMD, hyaline membrane disease; KT, kidney transplantation; ROH, oligohydramnios of renal origin; PD, peritoneal dialysis; PUV, posterior urethral valves; VUR, vesico ureteric reflux; GFR, glomerular filtration rate.

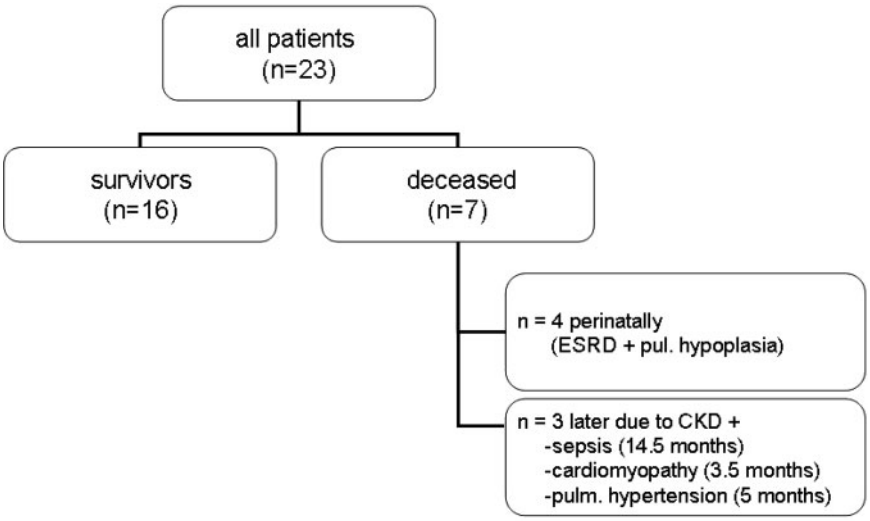

Fig. 1. Schematic representation of survival in patients with ROH.

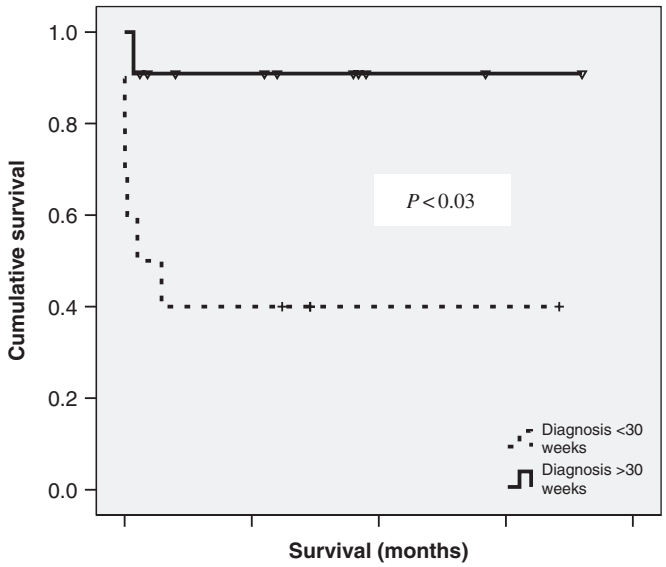

Fig. 2. Cumulative survival is significantly worse ($P < 0.03$) in infants with a diagnosis of ROH before the 30th gestational week.

Extrarenal morbidity

Growth. Current median height of the 16 children is −1.59 standard deviation (SDS) (range −2.9 to +0.9); (Figure 4). Seven patients required treatment with recombinant growth hormone (GH) for a median period of 3.5 years (range 1.5–7.2), which was discontinued at the time of renal transplantation. One patient on haemodialysis still receives GH. Two patients with SDS <−2 currently show catch-up growth after RT.

Development. Gross cognitive and motor development is normal in 12 of 16 patients (75%). They have normal motor function and attend normal schools (primary school: $n = 3$, secondary school: $n = 3$, kindergarten; $n = 4$, excluding two patients younger than 3 years).

Developmental delay is present in four children. One patient with concomitant birth asphyxia (patient 11) developed spastic quadriplegia (legs more than arms) with minor cognitive deficits, but attended kindergarten. One girl (patient 12) with associated congenital muscular hypotonia, clubfeet and dysmorphic facial features has moderately delayed motor and speech development but attends kindergarten. One patient (patient 14) has Down's syndrome, with associated developmental delay and muscular hypotonia. One patient after a protracted neonatal course and long-term ventilation and multiple complications (patient 18) has muscular hypotonia and feeding problems.

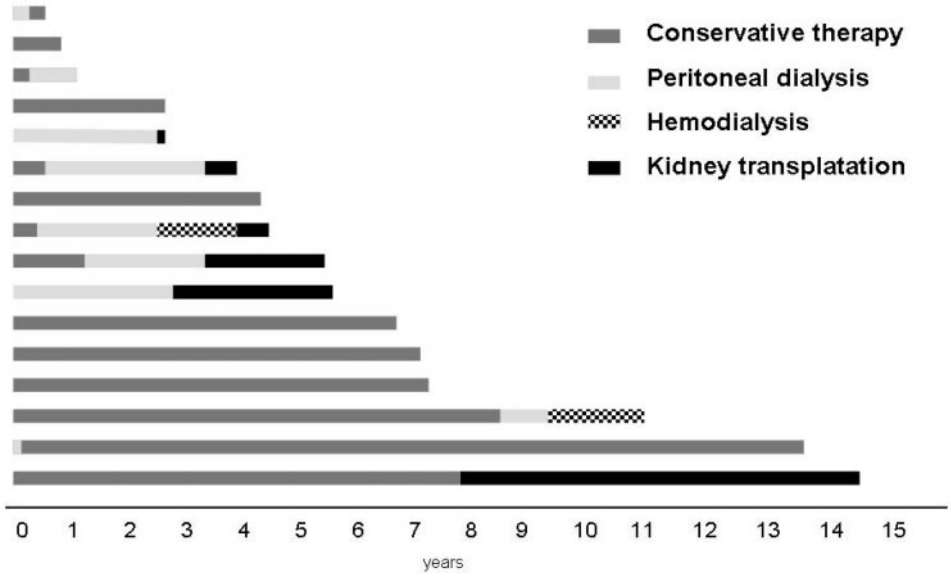

Fig. 3. Schematic representation of individual clinical course in survivors (dark grey: conservative treatment, light grey: peritoneal dialysis, chequered pattern: haemodialysis, and black: kidney transplantation).

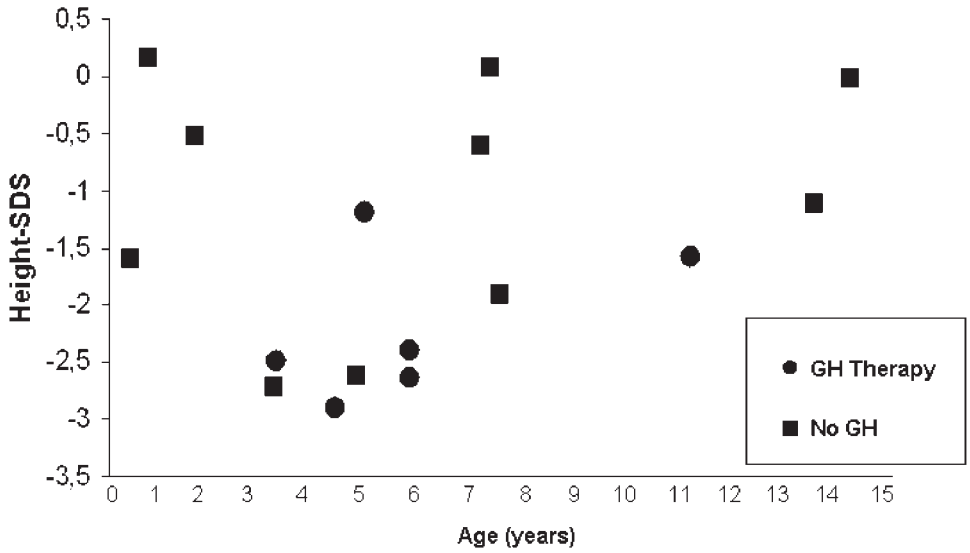

Fig. 4. Present height SDS of the survivors (filled circle with GH-treatment, filled square without GH-treatment).

Discussion

This report indicates that not all fetuses with ROH have a poor outcome. Although neonatal mortality and morbidity are significant, multidisciplinary efforts can result in an encouraging long-term prognosis. Although all surviving patients have CKD, sometimes requiring renal replacement therapy from the first days of life, the majority of patients can be managed conservatively.

Very few data on early neonatal outcome of ROH are available. One reason for this might be the fact that termination of pregnancy is still frequently practised for fetuses with ROH [6]. In contrast, one recent study documented a survival of 91% for male patients with ROH, lower urinary tract obstruction and vesicoamniotic shunting [14]. No precise data, however, on neonatal and long-term morbidity were stated in this publication. In a study from Zaccara *et al.* [15], 12 of 18 patients with an intrauterine diagnosis of bladder outlet obstruction and ROH could be followed-up after birth; of these, three died due to pulmonary hypoplasia compared with none in a group without ROH.

Our study for the first time presents detailed data on the neonatal clinical course after ROH, extending a previous limited experience in 10 patients [16]. Pulmonary complications due to pulmonary

hypoplasia and pneumothorax were frequent, and most children required assisted ventilation. Initial management in a neonatal intensive care setting is therefore mandatory, and affected fetuses should ideally be referred to tertiary perinatal centers. This need is further underlined by the fact that respiratory failure may also increase the risk of perinatal asphyxia impairing long-term neurological outcome. Antenatal sonographic suspicion of pulmonary hypoplasia, however, was not a specific prognostic marker, as some of these neonates had an uncomplicated pulmonary course without need for oxygenation or ventilation. Even sophisticated technology such as magnetic resonance-calculation of lung volumes in fetuses with ROH failed to predict the postnatal pulmonary course [17].

Whether antepartum amnioinfusion is a way to prevent or ameliorate pulmonary hypoplasia in neonates is a matter of debate. Experimental evidence is in favour and supported by recent studies; however, prospective data for ROH are lacking [9,18–20]. Two patients in our series received amnioinfusion treatment, but the series is too small to allow conclusions. Also, the value of antenatal surgery in ROH due to obstructive uropathies to improve pulmonary (and renal) function is under debate and is currently viewed with caution [21].

In the neonatal period, only a minority of patients in our series actually needed dialysis, comparing data of infants with other causes of neonatal renal failure [22]. Neonatal renal dysfunction, electrolyte disorders or hypertension, however, were present in almost all patients. Thus, nephrological expertise is necessary for optimal treatment. In addition, some patients required urological interventions, again underlying the multidisciplinary approach, including diagnostic imaging facilities and genetic expertise. The latter is demonstrated by the heterogeneous postnatal course in a family with three fetuses suffering from renal tubular dysgenesis [23]. Whether more aggressive treatment of renal insufficiency (e.g. dialysis in all patients with severe renal dysfunction), further improves outcome, cannot be concluded from our data.

The long-term outcome is mainly influenced by the degree of CKD. Although care of infants with CKD is a challenge, recent reports demonstrate a good prognosis so that treatment is recommended [24]; this notion is also supported by our results. A large proportion of these patients can be managed conservatively for a long time, despite antenatal evidence of severe renal disease [7]. Unfortunately, efforts to objectively assess fetal renal function non-invasively and accurately remains unsatisfactory [25].

Extrarenal morbidity includes growth retardation, which can be managed successfully by optimized nutrition, growth hormone and erythropoietin treatment and RT. Neurological outcome, though not evaluated prospectively and in precise detail, is appropriate for age in most patients. Two children with developmental delay had associated congenital anomalies. Future efforts should also focus on elimination of additional risk factors for developmental delay, such as perinatal asphyxia present in one of our patients. However, our data clearly indicate that renal dysfunction in a patient without asphyxia is not a risk factor for adverse neurological outcome, although neonates with asphyxia often have renal dysfunction [3].

Of the potential risk factors discussed, early diagnosis of ROH, i.e. occurrence in the first and second trimester, has been regarded an indicator of poor outcome [26] but not unanimously [13]. In some series, patients with cardiac malformations and termination of pregnancies were included. In our series, patients with poor overall survival had a significantly earlier diagnosis; however, this has to be viewed with caution, since early diagnosis is not able to differentiate between poor and good outcome. Individual patients with a very early diagnosis did well and vice versa some patients with late (or delayed) diagnosis died or had poor renal outcome. Mortality after the neonatal period, e.g. due to sepsis and heart disease is not always and necessarily related to ROH. Furthermore, interindividual fetal diagnostic accuracy as well as patient compliance with antenatal routine visits will remain a problem and results may not always be reliable. Nevertheless, early presentation of ROH must be viewed critically and future studies should prospectively address the predictive value of antenatal sonographical tools, e.g. time of diagnosis and other indices of severity.

The present observation is limited by the selected patient group, limited sample size and the retrospective analysis. Only fetuses followed in our tertiary pediatric nephrology centres were included, thus excluding fetuses not surviving intrauterine life due to intrauterine death or termination of pregnancy. Nevertheless, we believe that our conclusions are valid, since all infants with postnatal care were included, indicating that an overall negative prognosis for fetuses with ROH is not justified. This has important consequences for antenatal and genetic counseling. Our data confirm the obligation to implement a clinic for antenatal counseling for fetuses with CAKUT and renal disorders. Especially if ROH is present, the multidisciplinary approach has to be established before birth, including a neonatologist and pediatric nephrologists. Counseling of affected families should aim at neutral, balanced information to parents with individualized, independent decisions according to the family situation.

In summary and conclusion, our data indicate that the majority of fetuses with ROH have an encouraging long-term prognosis if a multidisciplinary approach is available [27]. Future efforts should include improved antenatal detection of risk factors and interdisciplinary antenatal counseling. If a decision is made to deliver the fetus, referral to a tertiary neonatal and pediatric nephrology centre seems mandatory.

Conflict of interest statement. None declared.

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