

A 10 year audit of antenatal ultrasound detection of renal disease

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Abstract. During a 10 year period, a renal tract anomaly was suspected on antenatal ultrasound in 125 fetuses, an incidence of five per 1000 births. 107 live births resulted. Three of six babies with renal failure were stented antenatally. A chromosome anomaly was present in three fetuses (2.4%). No live birth resulted in 14 pregnancies associated with oligohydramnios and no visible fetal bladder. Of those with renal agenesis or bilateral cystic dysplasia, one had a chromosome defect and a further four had extrarenal anomalies. Nine fetuses demonstrated isolated parenchymal hyperechogenicity and trisomy occurred in two of four with bilateral change in enlarged kidneys. All 14 babies with unilateral renal cysts had normal renal function postnatally, but only three of the cystic kidneys showed function. Antenatally, typical multicystic change was seen in 10, and smaller unevenly distributed cysts in four kidneys. Contralateral parenchymal echogenicity and/or pelvicalyceal distension indicating dysplasia was identified in four fetuses. Of 78 fetuses with isolated pelvic with or without calyceal distension the outcome was completely normal in 59% with unilateral, and in 48% with bilateral changes. One baby with unilateral and three with bilateral changes required dialysis or renal transplantation. One of four babies with antenatal ureteric distension had renal failure. Only one of six fetuses with bladder distension is alive in renal failure after *in utero* stenting.

The antenatal diagnosis of fetal abnormality has improved, largely due to the current availability of high resolution ultrasound equipment. The proportion of renal abnormalities diagnosed by ultrasound has risen from 15% in 1984 [1] to 80% or more in recent studies [2, 3]. Ultrasound is able to demonstrate the fetal bladder shortly after the 11th week of gestation when the permanent kidney first produces urine, and it is possible to follow the changes of nephrogenesis which continue until 38 weeks gestation [4]. However, the antenatal appearances of renal tract anomalies may still give rise to confusion in diagnosis and result in inappropriate management.

Renal agenesis is probably the result of a growth disturbance before the 5th week of gestation which prevents the ureteral bud from joining the metanephric blastema, while cystic dysplasia arises from abnormal developmental events just following this stage. There is considerable variation in the presentation of cystic dysplasia; ultrasound appearances are not just confined to the subtypes described by Potter [5] as IIA, known as multicystic kidneys, and IIB in which kidneys show fewer and smaller cysts. Early ureteral or urethral obstruction can

result in cystic change with or without hydronephrosis.

Fetal renal echogenicity is associated with cystic and other dysplasia, autosomal recessive infantile polycystic kidney disease (IPKD), autosomal dominant adult polycystic kidney disease (APKD) and normal kidneys. The echogenicity is probably due to interstitial or vascular infiltration, or to microscopic cortical cysts in IPKD [6, 7].

Methods

This audit was conducted over the 10 year period 1986–1995 in a District General Hospital with approximately 2600 deliveries a year and a total of 25 382 deliveries during this time.

All pregnancies received a routine second trimester scan between 17 and 20 weeks' gestation. Fetal renal appearances were monitored by further scans in pregnancy and post-natal investigation was continued, when necessary, up to 1 year of age. Diagnosis was confirmed by post-mortem, operation, or one or more of the following post-natal investigations: renal biopsy, ultrasound, intravenous urography or micturating cystography.

This audit was facilitated by the Northern Region Congenital Abnormality Survey (NorCAS) which provides a comprehensive central register for anomalies detected or suspected antenatally and their post-natal outcome [8].

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Antenatally identified renal abnormalities were grouped as follows:

- (1) Oligohydramnios and no visible fetal bladder.
- (2) Isolated unilateral or bilateral parenchymal hyperechogenicity.
- (3) Unilateral cysts (other than those in Group 1).
- (4) Collecting system distension:
 - (i) of renal pelvis (\pm calyces); anteroposterior measurement of 5 mm or more at 16–20 weeks' gestation or 8 mm or more after 28 weeks' gestation.
 - (ii) above including visible ureter.
 - (iii) above including urinary bladder distension.

Results

A renal abnormality was antenatally suspected in 125 fetuses during the 10 year period studied. A further five were lost to follow-up and are excluded from the study. A significant renal tract abnormality was not identified antenatally in eight babies. Of 107 live births, six babies have renal failure, three of whom were stented antenatally.

Group 1. Oligohydramnios with no fetal bladder identified

This was identified in 14 pregnancies and there were no live births. Normal renal tissue was identified at post-mortem examination in three after spontaneous abortion at 20, 24 and 26 weeks' gestation. Bilateral cystic dysplasia was recognized in six fetuses of which two were terminated, two spontaneously aborted, and two died shortly after birth. Two fetuses with bilateral cystic dysplasia had other structural abnormalities, congenital diaphragmatic hernia (1) and congenital heart defect (1), and another fetus had a balanced inversion of chromosome 2. Renal agenesis was diagnosed in five fetuses of which four were terminated and one spontaneously aborted at 23 weeks' gestation. Spina bifida was detected in one fetus and a congenital heart defect in another in this subgroup.

Group 2. Isolated unilateral or bilateral parenchymal echogenicity

Eight live births resulted from the fetuses in this group. Bilateral, large echogenic kidneys were identified in four pregnancies, one was terminated when trisomy-13 was diagnosed. Another baby was born with Down's syndrome and an atrioventricular septal defect. In a further two babies, post-natal scans showed small cortical cysts and renal function was normal. Bilateral increased

echogenicity in normal sized kidneys resulted from adult polycystic disease in two related fetuses. Unilateral echogenicity in a normal sized kidney resulted post-natally in megaureter and reflux (1), a small dysplastic kidney (1) and a normal kidney (1).

Group 3. Unilateral cystic change (other than in Group 1)

Unilateral renal cysts were identified in 14 fetuses, all of whom were liveborn. In four, the cysts co-existed with, or were preceded by, ipsilateral pelvicalyceal distension (PCD). No renal tissue was found post-natally in two of the 10 fetuses with a typical multicystic kidney antenatally. In the four fetuses with smaller, randomly distributed cysts antenatally, no renal tissue was found in one and small cystic dysplastic kidneys demonstrating function in three. The contralateral kidney exhibited pelvicalyceal distension and/or parenchymal echogenicity in four fetuses but all babies had normal renal function and the postnatal outcomes are given in Table 1.

Group 4. Distension of the renal collecting system

Unilateral distension of a renal pelvis with or without calyces

This was identified in 32 fetuses, all liveborn but one baby had renal failure and the post-natal findings are given in Table 2.

Table 1. Post-natal outcome of those fetuses with unilateral renal cysts and abnormality of contralateral kidney

Cystic kidney	Contralateral kidney
Small cysts + PUJ obstruction	Normal kidney
Multicystic kidney	Hydroureter and ureteric reflux
Small cyst + PCD	PUJ obstruction
Small cystic dysplastic + PCD	Normal kidney

PUJ, pelviureteric junction; PCD, pelvicalyceal distension.

Table 2. Post-natal findings in fetuses with unilateral distension of a renal pelvis with or without calyces

Normal kidneys/full or extrarenal pelvis	19
Unilateral reflux	1
Bilateral reflux	2
Unilateral megaureter	5
Bilateral megaureter	1 ^a
Unilateral PUJ obstruction	4

^a One baby had renal failure. PUJ, pelviureteric junction.

Table 3. Post-natal findings in fetuses with bilateral distension of the renal pelves with or without calyces

Normal kidneys/full or extrarenal pelves	22
Unilateral reflux	3
Bilateral reflux	3
Primary unilateral megaureter	5
Primary bilateral megaureter	3 ^a
Unilateral PUJ obstruction	6
Bilateral PUJ obstruction	4

^a Three babies required dialysis.
PUJ, pelviureteric junction.

Bilateral distension of the renal pelves with or without calyces

This was seen in 46 fetuses. Three required dialysis, two following antenatal stent placement and the post-natal findings are listed in Table 3.

Ureteric distension with a normal bladder volume

This was found in four fetuses. All were liveborn but one baby had renal failure as a result of bilateral non-obstructed megaureters associated with reflux. The post-natal findings of unilateral ureteric distension were a ureterocele (2) and primary megaureter without reflux (1).

Bladder distension

This was identified in six fetuses and resulted in one livebirth in renal failure. Bladder distension was identified before 16 weeks' gestation in association with urethral valves (1) and prune belly syndrome (1); both pregnancies were terminated. Bladder distension was identified by the routine scan in four fetuses associated with bilateral hydronephrosis, all due to bladder outflow obstruction. Oligohydramnios was present in three and these pregnancies were terminated. In the fourth, insertion of bilateral ureteric stents at 24 weeks' gestation, resulted in a live baby requiring dialysis.

The eight renal abnormalities unidentified antenatally were bladder extrophy (1), ectopic ureterocele (1), posterior urethral valves (1), and bilateral primary megaureter with reflux (5).

Discussion

Despite the improvements in antenatal detection of fetal renal abnormality, there continue to be problems with diagnosis, interpretation and management. In addition, the identification of features which may represent physiological variation results in investigation which can be costly, time consuming and give rise to unnecessary parental anxiety.

Termination can be offered if a renal cause for oligohydramnios and absent bladder are diagnosed before 24 weeks' gestation. Although first trimester

diagnosis with vaginal sonography is possible [9], oligohydramnios frequently prevents confident diagnosis of renal agenesis or cystic dysplasia in small kidneys [10]. Although severe oligohydramnios from other causes such as growth retardation, idiopathic or ruptured membranes results in very few live births [11, 12], a more aggressive attempt at diagnosis and identification of associated abnormalities by amnio-infusion [13, 14] may be appropriate.

Isolated renal parenchymal echogenicity is a non-specific finding and difficult to evaluate; but it was in this group, and not in those with pelvicalyceal distension, that two fetuses with trisomy occurred. Hence, this audit suggests that karyotyping is indicated when both kidneys are large and echogenic. The antenatal appearance of large bright kidneys in IPKD is well documented [15, 16], although is rare [17] and was not diagnosed during this study. Also, the gestation when changes of IPKD can be identified, if at all, depends on the degree of gene expression and the severity of the disease [18]. Adult polycystic kidney disease may present similarly [19, 20], although we first suggested this diagnosis in the third trimester when echogenic changes in normal sized kidneys were identified. Renal echogenicity may also be due to small cortical cysts or result in normal kidneys [21].

The antenatal appearances of cystic dysplasia vary enormously [22], but the typical multicystic kidney does not usually present a diagnostic problem. Post-natally almost 80% of cystic kidneys had no functioning renal tissue and 40% demonstrated contralateral dysplasia, so further scans in pregnancy and after birth are necessary. Perhaps surprisingly, no baby in this group was born with abnormal renal function.

The threshold of fetal renal pyelectasis which results in significant post-natal disease remains controversial. Grignon et al [23] consider an anteroposterior (PA) pelvic measurement of under 10 mm to be physiological and NorCAS have similarly revised their notification criteria. This study uses the original NorCAS 5 mm limit, which, although causing unfounded parental anxiety, identified babies with clinically significant ureteric reflux, obstruction of the pelviureteric junction and ureterovesical junction. Three babies with renal failure had pelvic dimensions under 10 mm when scanned initially.

Antenatal ureteric distension more reliably indicates a significant, although possibly unilateral, abnormality and a distended fetal bladder has a poor prognosis. Unlike other reports [24, 25], no baby in this group was found to have a chromosome abnormality.

It is therefore in those fetuses with apparently isolated distension of one or both renal pelves,

that errors of interpretation, and hence mismanagement are most likely to occur. Although 59% of fetuses with unilateral and 48% with bilateral hydronephrosis had normal kidneys post-natally, failure to identify parenchymal echogenicity, small cysts, or ureteric distension antenatally, may change what is thought to be a low risk unilateral problem into a bilateral abnormality resulting in renal failure.

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