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Fetal hydronephrosis: is there hope for consensus?

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Abstract This review article aims at summarizing the data regarding fetal and neonatal hydronephrosis, at correlating controversial data with the differences in the practice of obstetrical sonography from one country to another, and finally, at presenting our own criteria for fetal renal collecting system dilatation along with our own guidelines of postnatal investigation.

Keywords Urinary tract · Fetal diagnosis · Hydronephrosis

Introduction

Urinary tract abnormalities represent 20% of all congenital malformations [1]. Severe urinary tract abnormalities are present in 10% of all terminations for lethal abnormalities [2]. In most cases, ultrasound (US) is able to differentiate between a normal and abnormal fetal urinary tract. In complex uropathies or with impaired sonographic visibility, fetal magnetic resonance (MR) imaging can be a valuable additional method [3].

Hydronephrosis refers to renal collecting system dilatation, regardless of its aetiology. In pyelectasis (pelviectasis) the renal pelvis is dilated; in caliectasis dilatation of renal calyces is seen. The early demonstration of hydronephrosis has several benefits: the preservation of the renal function, the prevention of urinary infections and the treatment of urinary tract

obstructions. For example, the clinical picture of life-threatening septicaemia of boys with posterior urethral valve (PUV), a common finding in pre-US era, is extremely rare nowadays since about 80% of cases are detected prenatally, providing an opportunity to start antibiotic prophylaxis and to decompress the obstructed system immediately after birth [4].

According to the American Institute of Ultrasound Medicine [5] and the American College of Radiology guidelines, the kidneys and the bladder are always included in second and third trimester fetal sonography.

Is anything more promising and inspiring than assessing fetal anatomy, selecting in utero potential patients at risk for postnatal investigation and accordingly preventing further damage? In the last 20 years, hundreds of articles have addressed the issue enthusiastically. Where do we stand now?

In this review we will try to summarize the often confusing conclusions regarding fetal and neonatal hydronephrosis, to analyse controversial data in the light of the various lines of practice, and to present our guidelines in Sainte Justine Hospital.

The fetal urinary tract

Embryology

The development of the urinary tract arises from the intermediate mesoderm. The pronephros, a non-functional tissue, forms in the 3rd–5th week of gestation. It is followed by formation of the mesonephros, which produces small amounts of urine between 5 and 11 weeks of gestation. After the regression of the mesonephros, the ureteric bud, which appears during the 4th week of gestation, induces the formation of the metanephros, the definitive kidney. The collecting ducts, calyces, renal pelvis and ureter arise from the ureteric bud of mesonephric origin, whilst the nephrons and tubules arise from the metanephric blastema.

The first sign of renal tubular function appears in the human metanephric kidney between 9 and 12 weeks of gestation. By the 14th week of gestation, the loop of Henle becomes functional and tubular reabsorption occurs. New nephrons are formed up to the 36th week of gestation in the human fetus. Nephrogenesis is complete at birth in full-term infants, but nephron formation continues after birth in preterm infants [6, 7].

Sonography

The kidneys can be identified both transvaginally and transvesically from 13 to 14 weeks of gestation as oval echogenic structures lateral to the upper lumbar spine [8]. Some authors [9] have outlined the value of transvaginal sonography in displaying the fetal kidneys during the first and early second trimester.

The size of kidneys increases progressively [10], but the ratio between renal and abdominal circumferences is relatively stable (0.27–0.30) during normal pregnancy. The kidneys are hyperechoic compared to the liver and pancreas up to 17–18 weeks of gestation. As the hyperechogenicity decreases, the cortico-medullary differentiation gradually appears, starting from 20 weeks G.A. (w.G.A.) and it is more conspicuous in the third trimester (26th–27th week) [11]. Renal calyces are not visible within normal kidneys.

The normal bladder can be visualized from the onset of urine production, which occurs around 9–10 w.G.A. At the 18- to 20-week routine scan, the bladder should be identified in 100% of cases during the 15- to 20-min examination. The cyclic pattern of fetal bladder filling

and emptying becomes slower during the last weeks of pregnancy, particularly in female fetuses, who consequently tend to have a larger bladder than males [11]. Bladder diameter threshold measurements are as follows: first trimester <1 cm, second trimester <3 cm, third trimester <6 cm [11] (Fig. 1). The bladder wall itself should not be thicker than 2–3 mm [12, 13]. Under normal conditions, the fetal ureters and urethra are not visible.

During the first trimester the placenta (chorion and amniotic membrane) is the principal source of amniotic fluid, but after 15 w.G.A. fetal kidneys contribute to the majority of amniotic fluid. Therefore, a normal amount of amniotic fluid after 15 weeks of gestation and normal development of lungs can be considered indirect signs of normally functioning kidneys.

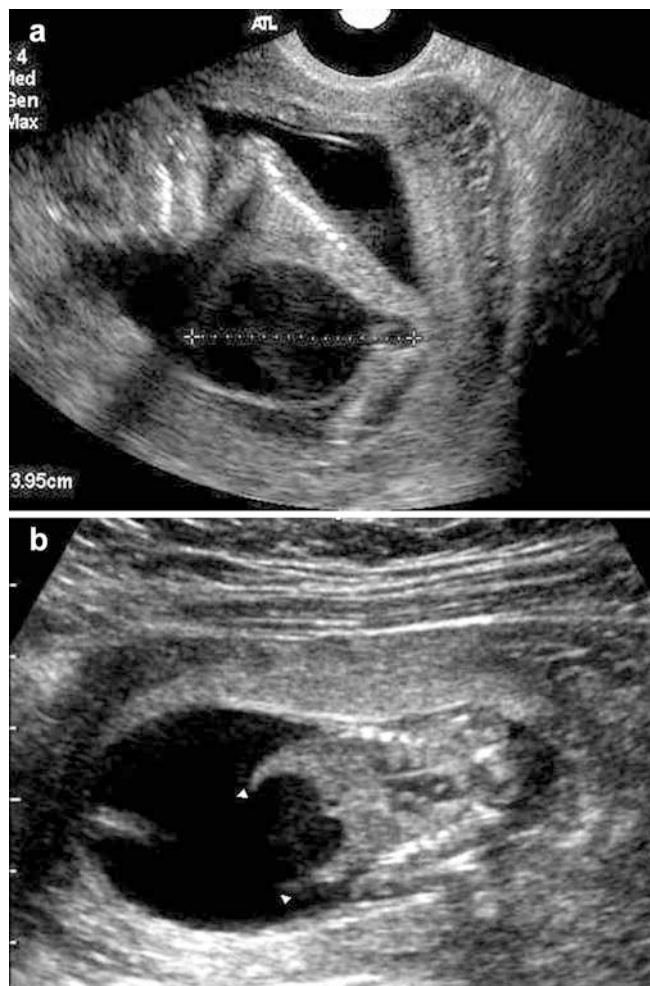


Fig. 1a, b Megabladder in the first trimester. **a** A sagittal scan shows a huge bladder with opened bladder outlet (in between calipers) caused by PUV or urethral atresia, 13 w.G.A. **b** Bladder rupture (arrowheads) in the same patient at 14 w.G.A. with evidence of ascites (longitudinal scan)

Physiology

There are several physiological factors in fetuses that account for the sonographic visibility of the renal pelvis.

The fetal urine production is characterized by both the low rate of tubular reabsorption and the 10- to 12-fold increase of urinary output from 20 to 40 weeks of gestation. This develops as a result of a threefold increase in fetal urine production between 20 and 25 w.G.A., and a two- to threefold increase between 30 and 40 w.G.A. [14, 15].

Maternal hydration [16, 17] as well as the extent to which fetal bladder is full play a role in fetal pyelectasis; when the fetal bladder was empty, there is a significant decrease in the size of the fetal renal pelvis [18].

Over time there are significant variations when measuring fetal renal pelvis anteroposterior (AP) diameter. Of 20 fetuses (8 in the second trimester and 12 in the third trimester) in which multiple observations were made over a 2-h period, 70% exhibited both normal (<4 mm) and abnormal values (4 mm or more) [19].

Fetal pyelectasis is five to six times more likely to occur in fetuses of mothers who themselves demonstrate pyelectasis [20]. The relaxant effect of progesterone on the smooth muscle of the urinary tract is considered a cause for maternal hydronephrosis in pregnancy. The same hormonal effect is likely to influence the fetal urinary tract.

Fetal pyelectasis has a marked sex difference with a male-to-female ratio of around 2:1 [21–23]. It is possible that the explanation of sex difference is anatomical [24]; the increased voiding pressure in male fetuses can cause

the distortion of the vesico-ureteral junction in utero, hence the prevalence of VUR in male fetuses. Over 80% of children with prenatally diagnosed VUR are boys [25–27] (Fig. 2).

There are also variations in the configuration of pelvicalyceal system. Even though the renal pelvis is most often intrarenal in fetuses and newborns, it can also be situated extrarenally. The prevalence of extra-renal pelvis ranges from 10 to 15% of cases [28, 29]. A renal pelvis AP diameter measuring 10–15 mm with no evidence of calyceal dilatation may represent an extra-renal pelvis (Fig. 3).

The recognition of fetal urinary tract dilatation

Numerous cut-off values for the AP diameter of fetal renal pelvis have been proposed for the diagnosis of antenatal hydronephrosis. They vary from 4 to 10 mm in the second trimester and 7 to 10 mm in the third. Some authors [30] in earlier studies have also used the ratio of renal pelvic diameter and the renal diameter (PD/RD) to define hydronephrosis.

The choice of cut-off values depends on the initial objectives, namely the sensitivity of detection of hydronephrosis and the resulting false-positive rate (Table 1). For example, strict criteria (≥ 4 mm <33 w.G.A., ≥ 7 mm >33 w.G.A.) [31] will lead to a 100% detection of hydronephrosis with a resulting false-positive rate of 30–80% of cases [32]. Such a high rate of false-positive cases is not surprising, considering the fact that, at least 3–5% of normal fetuses meet these criteria for fetal

Fig. 2a–c VUR prenatally and postnatally. **a** A sagittal scan (26 w.G.A.) of the right kidney displays grade III/V pelvocalyceal dilatation (*arrows*). **b** Associated with an ipsilateral megaureter (in between calipers). **c** Oblique neonatal VCUG demonstrates bilateral VUR, grade IV–V/V in the right kidney (*arrow*), II/V in the left (*arrowhead*)

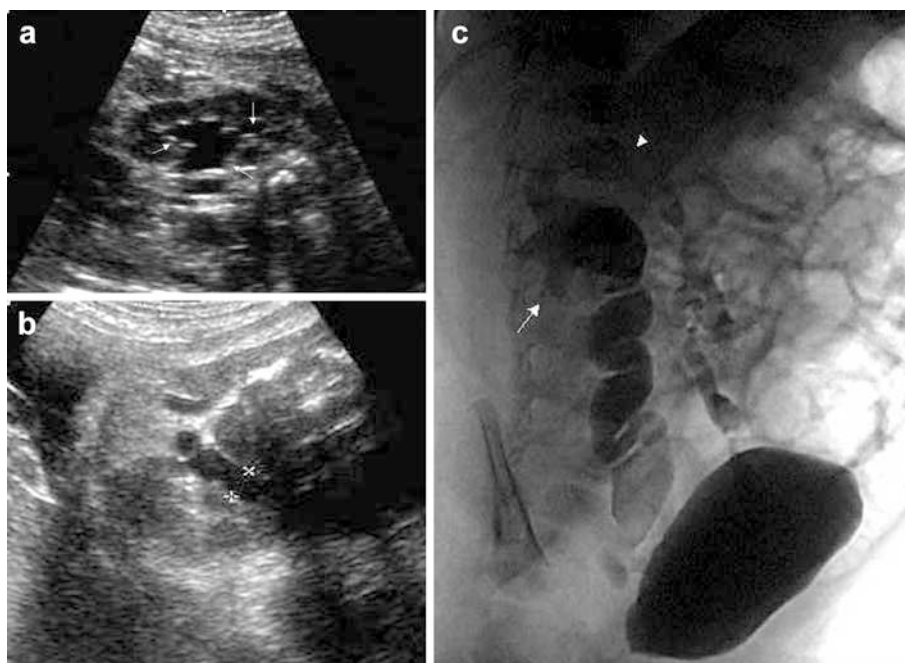




Fig. 3a–c Prenatal and postnatal appearance of extrarenal pelvis. **a** A third-trimester transverse sonogram shows a left renal pelvis AP diameter of 15 mm without caliectasis. **b, c** On neonatal US, transverse (**b**) and longitudinal (**c**) scans make an extrarenal pelvis most likely. VCUG and isotopic renography (not shown) were normal

Table 1 The sensitivity and the false-positive rate in detection of hydronephrosis

Renal pelvis AP diameter	Sensitivity (%)	False-positive rate (%)
≥4 mm prior to 33 weeks	100	30–80
≥7 mm after 33 weeks		
> 4 mm prior to 24 weeks	80	15
> 10 mm after 24 weeks		

hydronephrosis [33]. Conversely, our own cut-off values in Sainte-Justine Hospital (4 mm prior to 24 w.G.A., 10 mm after 24 w.G.A.) will, indeed, decrease the sensitivity for detecting hydronephrosis to 80%, but the resulting false-positive rate of 15% becomes clinically more acceptable.

The grading of the fetal urinary tract dilatation is therefore crucial in order to correlate prenatal findings with postnatal outcome. Two classifications have been proposed which are practically identical [34, 35] (Table 2).

Most authors agree that a renal pelvic AP diameter greater than 10–15 mm is highly predictive of significant urinary tract pathology, especially in the presence of caliectasis [34, 36, 37]. As an example, Barker et al. [38] have correlated renal pelvic dilatation at 19 weeks' gestation with the mean differential renal function in pelvi-ureteric junction (PUJ) obstruction patients (Table 3).

Table 2 Classification of fetal hydronephrosis (after 24 w.G.A.)

Grade	Grignon et al. [34]	Grade	SFU [35]
I	AP pelvis < 10 mm	0	No dilatation
II	AP pelvis 10–15 mm	I	Renal pelvis dilatation
III	AP pelvis > 15 mm, mild calyceal dilatation	II	Renal pelvis dilatation with visible calyces (mild)
IV	AP pelvis > 15 mm, moderate calyceal dilatation	III	Renal pelvis and dilated calyces (moderate)
V	AP pelvis > 15 mm, severe calyceal dilatation	IV	Features of grade III with parenchymal thinning (severe)

There is a general consensus about fetal renal collecting system dilatation in the following situations:

1. *Prior to 24 w.G.A.*, the fetal urinary tract is *normal* if the renal pelvis AP diameter is 4 mm or less, the calyces and ureters are non-visible and the bladder is normal.
2. *After 24 w.G.A.*, the fetal urinary tract is *abnormal* if the renal pelvis AP diameter exceeds 10 mm, if the calyces are visible, if the ureter is dilated and if the bladder or the urethra is enlarged and/or thick-walled.

Besides the grade of fetal renal collecting system dilatation, other prognostic predictors of outcome include:

1. The involvement of both kidneys (Fig. 4).
2. The presence of coexisting renal dysplasia, sonographically characterized by increased parenchymal echogenicity and/or cortical cysts. At least 90% of renal dysplasias are related to fetal urinary tract obstruction [39].
3. A decreased amount of amniotic fluid.
4. Pulmonary hypoplasia.
5. Significant modification of normal fetal urine biochemistry.
6. Coexisting associated anomalies, which may eventually lead to karyotyping.

On the other hand, mild pyelectasis (renal pelvic AP diameter 4–10 mm) has led to *ongoing controversies* and numerous cut-off values (Table 4).

A comparison between mean renal pelvic diameter of obstructed and nonobstructed fetal kidneys has shown overlapping between 16 and 21 w.G.A. and diversion of corresponding curves after 22–24 w.G.A. (Fig. 5) [42]. Indeed, follow-up examinations are indicated in equivocal cases and most cases amenable

Table 3 Mean differential renal function in PUJ obstruction patients [38]

Dilatation at median of 19 week (mm)	Mean differential function (%)
Normal: < 4	48.2
Mild: 5–8	42.7
Moderate: 10–15	37.3
Severe: > 15	26.6

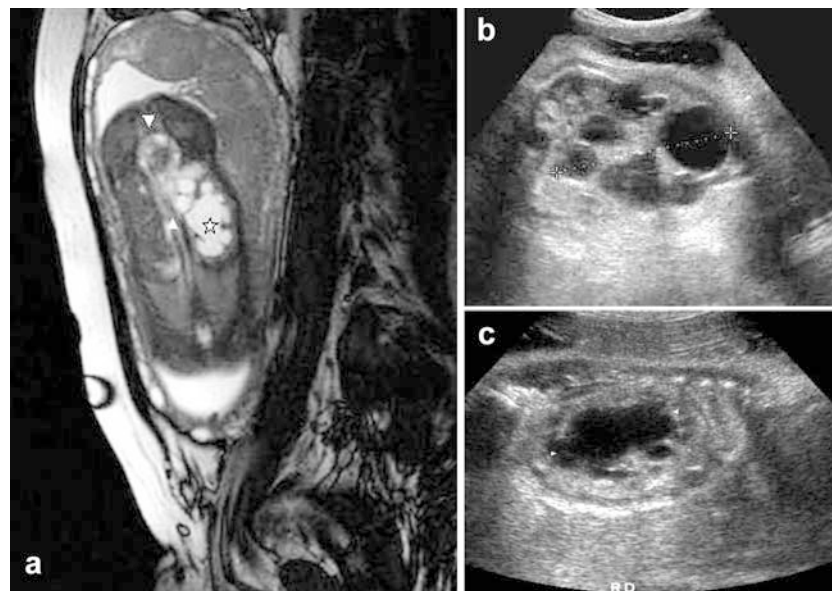
to treatment are detected during the third trimester [11].

Causes of urinary tract dilatation

Neonatal urinary tract dilatation has many aetiologies, including structural abnormalities and reflux (Tables 5 and 6). However, structural and non-structural causes of fetal “hydronephrosis” reported in newborns and infants include [45]:

- Transient hydronephrosis in 48% of cases
- Physiological hydronephrosis, e.g. extrarenal pelvis in 15%

Fig. 4a–c Prenatal MRI and US with bilateral involvement of kidneys. **a** Patient was referred to our hospital because of complex anatomy and bilateral abnormalities detected in the second trimester US (not shown) in another hospital. MRI was performed (25 w.G.A.) in our institution. T2-weighted (TRUFI) coronal image demonstrates crossed fused kidneys with hydronephrosis in the right kidney (*star*) and multiple cysts in the ectopic left kidney (*arrowheads*). **b** A follow-up US examination (35 w.G.A. sagittal scan) showing ectopic left fused kidney with multiple cortical cysts and increased parenchymal echogenicity indicating MCDK. **c** and marked (grade IV/V) hydronephrosis (*between the arrowheads*) of the contralateral right kidney (sagittal scan)



- PUJ obstruction in 11%
- Vesico-ureteral reflux (VUR) in 9%
- Megaureter in 4%
- Ureteroceles and duplex kidney in 2%
- Multicystic kidney disease (MCDK) in 2%
- PUV in 1%

These figures may at first appear confusing, but they are not contradictory.

The entire cohort of fetuses with prenatally detected urinary tract dilatation reveals a considerable number with non-significant hydronephrosis [45]. The transient, non-significant nature of numerous fetal “hydronephroses” is of utmost importance when deciding on the appropriateness of investigating newborns and infants. If transient (48%) and physiological (15%) hydronephroses are excluded, PUJ obstruction and VUR are the leading causes of clinically significant urinary tract anomalies [11, 45, 46].

Postnatal investigations

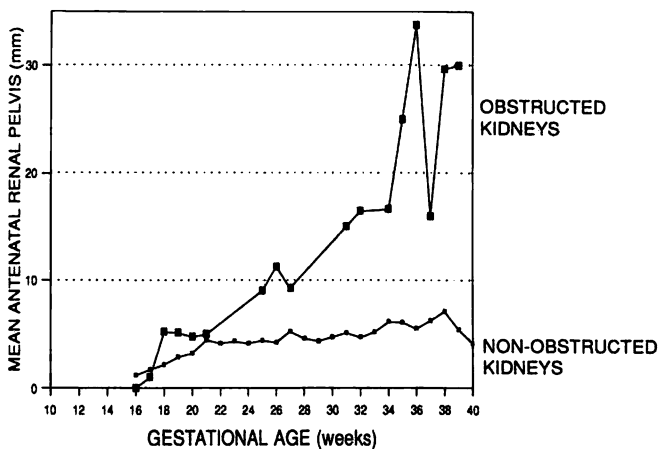
Classically, the prenatal diagnosis of hydronephrosis initiates postnatal investigations, including sonography, voiding cystourethrography (VCUG) and isotopic renography.

The US examination should be performed after the physiological dehydration period (3–5 days after birth). In recent studies, the routine use of VCUG in mild pyelectasis appears questionable [47].

The role of MR urography in the work-up of hydronephrosis in infants and children is currently being investigated by several authors [48–52]. With the use of dynamic contrast-enhanced T1-weighted sequences, a single non-irradiating examination provides both mor-

Table 4 The definition of fetal hydronephrosis in different studies

Study	Definition of hydronephrosis
Arger et al. [30]	AP diameter ≥ 10 mm or PD/RD over 50%
Grignon et al. [34]	AP diameter ≥ 10 mm > 24 w.G.A.
Livera et al. [40]	AP diameter > 10 mm > 28 w.G.A.
Mandell et al. [41]	AP diameter ≥ 5 mm at 15–20 w.G.A. ≥ 8 mm 20–30 w.G.A. ≥ 10 mm > 30 w.G.A.
Corteville et al. [31]	AP diameter ≥ 4 mm < 33 w.G.A. ≥ 7 mm > 33 w.G.A.
Johnson et al. [37]	AP diameter > 10 mm (AP > 15 mm strongly predictive)
Lam et al. [29]	AP diameter > 10 mm (extrarenal pelvis in 15.6%)
Anderson et al. [42]	AP diameter ≥ 4 mm < 24 w.G.A. ≥ 6 mm at 24–30 w.G.A. ≥ 8 mm > 31 w.G.A.
Adra et al. [21]	AP diameter ≥ 8 mm > 28 w.G.A.
Barker et al. [38]	AP diameter > 5 mm
Ouzounian et al. [43]	AP diameter ≥ 5 mm
Dudley et al. [22]	AP diameter > 5 mm
James et al. [44]	AP diameter > 5 mm at 18 w.G.A. > 7 mm > 28 w.G.A.

**Fig. 5** Mean renal pelvic diameter of obstructed and non-obstructed kidneys in relation to gestational age (reproduced with permission from [42])**Table 5** Aetiologies of neonatal hydronephrosis [11]

Aetiology	Percent
PUJ obstruction	45
VUR	30
VUJ obstruction	10
Duplex kidneys (\pm ureterocele)	7
PUV	4
Multicystic dysplasia	4

physiological and functional information of the urinary tract. The future of MRI urography remains to be seen; however, MRI is likely to challenge the present imaging protocols, especially in the case of complicated uropathies. The value of cyclic VCUG and contrast sonogra-

Table 6 The final urologic diagnoses in infants with significant prenatally detected urinary tract anomalies [46] in percent

PUJ obstruction	35
VUR	20
MCDK	15
VUJ	10
PUV	9
Duplex system	8

phy (echocystography) in the search of VUR is beyond the scope of this review.

When to investigate?

The timing of postnatal examinations depends on the suspected pathology. As a rule, immediate neonatal investigations are required together with antibiotics in cases of:

- Severe bilateral urinary tract dilatation
- Dilatation of a single (functioning) kidney
- Posterior urethral valves
- Obstructed duplex kidneys.

Conversely, in the cases of mild or moderate dilatation, the investigations may be delayed and performed after 3–4 weeks because of the high prevalence of transient VUR and resolute hydronephrosis. Antibiotic prophylaxis is then a matter of institutional preference. In our institution antibiotic prophylaxis is initiated only after VUR has been demonstrated.

Table 7 Prenatal sonographic findings in Sainte Justine Hospital indicating the need for postnatal investigations

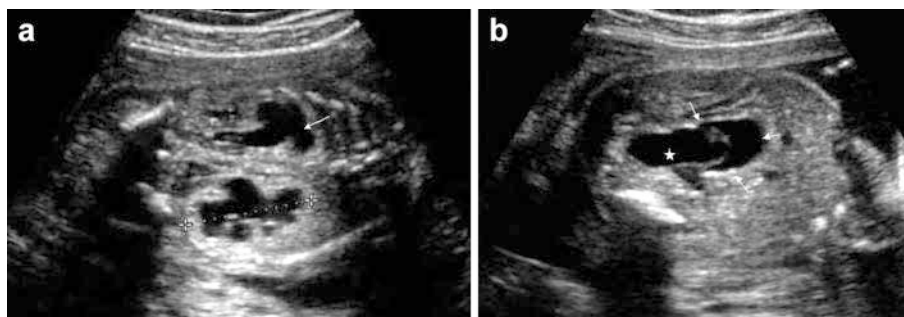
Prenatal US findings
Renal pelvic dilatation (AP > 10 mm after 24 w.G.A.)
Calyceal dilatation (isolated or associated with pyelectasis)
Ureteral dilatation (with or without ureterocele)
Bladder enlargement or bladder wall thickening
Urethral dilatation

Who should be investigated?

For most authors, postnatal investigations should be performed *according to the prenatal data* [53]. In our institution, US, VCUG and isotopic renography (with diuretic challenge) for assessing obstruction are indicated in patients in whom prenatal imaging has shown evidence of abnormality in urinary collecting system (Table 7).

Recently, some authors [54] have recommended that VCUG be performed only when *postnatal renal US is abnormal*. They define neonatal abnormality as: renal pelvis AP over 7 mm (with an empty bladder), a variable dilatation, calyceal dilatation, an ureteral dilatation above 3 mm, pelvic wall thickening, a megabladder, and renal parenchymal changes (loss of corticomedullary differentiation, small kidney, signs of dysplasia). A recent study [47] has shown that a normal-appearing urinary tract on two sequential neonatal US scans during the 1st week and at the 6th week rarely (only in 6.7% of cases) coexists with abnormal findings at VCUG. Such a protocol is supported by the local routine use of fetal sonography in the first, second and third trimester of pregnancy. Indeed, these authors have shown beyond any doubt that the third trimester US is the best predictor of postnatal uropathy [55].

Fig. 6a, b Prenatal US of duplex system with ureterocele and contralateral obstructive renal dysplasia. **a** A coronal scan (20 w.G.A.) shows a duplex right kidney with upper-pole hydronephrosis (*arrow*) and marked (grade IV/V) hydronephrosis of the contralateral left kidney (in between calipers) with hyperechoic parenchyma, suggesting obstructive parenchymal dysplasia. **b** A transverse scan of the fetal bladder (*arrows*) during the same examination displays the giant ureterocele (*star*) connected to the ureter of the upper moiety and causing the obstruction of the contralateral left kidney. The parents elected to terminate the pregnancy



Numerous authors have conversely emphasized the poor predictive value of US regarding VUR [45, 56–58] and have insisted on the need for VCUG to rule out VUR. These different guidelines are directly connected to the lack of routine third trimester sonography in North America.

Fetal hydronephrosis and postnatal follow-up

Despite the controversy regarding the diagnostic criteria of fetal hydronephrosis and the postnatal investigation protocols, practical conclusions can be drawn from the abundant literature on the topic:

1. The likelihood of a patient having a significant postnatal renal abnormality is proportional to the severity of the antenatal hydronephrosis [34, 38]. Let us repeat that the third trimester renal sonography is the best predictor of all. There is, however, no direct correlation between the degree of dilatation and renal function. The best marker of renal obstructive dysplasia and probable poor function is hyperechoic parenchyma with cortical cysts [54, 59] (Fig. 6).
2. The differential diagnosis relates to the degree of antenatal hydronephrosis [45]. In marked hydronephrosis PUJ (Fig. 7) is the most likely diagnosis (10% of all cases of fetal hydronephrosis) whereas in mild hydronephrosis (renal pelvis AP measurement 5–9 mm) transient hydronephrosis (in about 50% of cases), physiological hydronephrosis, e.g. extrarenal pelvis (in about 15% of cases) and VUR (10–15%) are the most common causes.
3. Spontaneous resolution of second-trimester pyelectasis is seen in 80% of cases, either in utero or during the first year of life [22, 33, 60] (Fig. 8).
4. Because many antenatally detected uropathies have shown the potential to resolve spontaneously, surgery is performed only after a period of observation. Nowadays only 5–10% of cases require surgical intervention. Surgical indications are often correlated with the progression of dilatation in utero on sequential examinations [61] (Fig. 9).
5. Only 10–30% of fetal pyelectasis in reported series proves to be related to VUR postnatally [26–28, 62]

Fig. 7a, b PUJ obstruction as cause of marked hydronephrosis. **a** A transverse sonogram at 33 w.G.A. with marked right hydronephrosis (renal pelvis AP diameter 51 mm, in between calipers) and a mild (renal pelvis AP diameter 12.5 mm) dilatation of the contralateral renal pelvis. **b** VCUG at birth reveals a right PUJ obstruction with associated ipsilateral VUR. Follow-up US (not shown) showed a normal left kidney

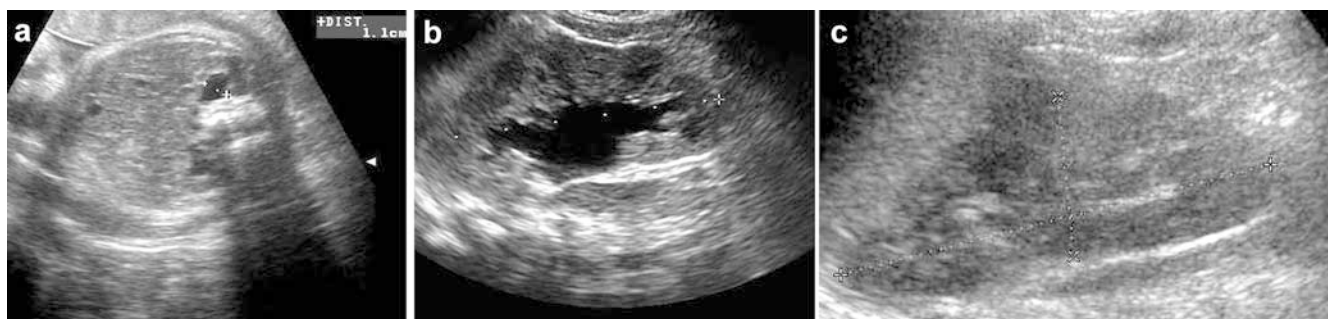
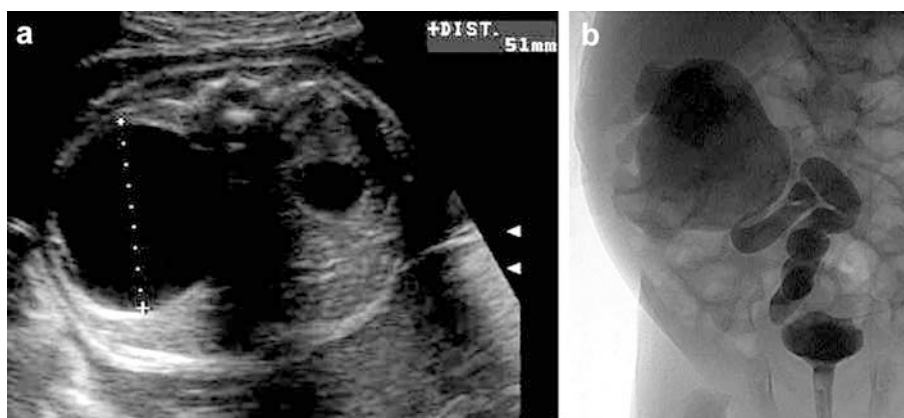


Fig. 8a–c Spontaneous resolution of prenatal pyelectasis during the first year of life. **a** A 35 w.G.A. transverse scan shows a right renal pelvic AP diameter of 11 mm (in between calipers). **b** Neonatal US (longitudinal scan) of the right kidney shows a mild dilatation (grade II–III/V) of a renal collecting system. VCUG (not shown) was normal. **c** US of the same patient at 9 months of age shows no residual sign of hydronephrosis (kidney in between calipers)

(Fig. 10). In our institution, 14% of newborns undergoing VCUG for antenatal hydronephrosis showed evidence of VUR.

6. Spontaneous resolution of VUR occurs in 65% of refluxing units within 2 years, including 20% of grade IV and V [63]. Studies have shown a lower incidence of renal scarring in infants with prenatally detected VUR when compared with infants presenting postnatally with infection [64].
7. Even though prenatal screening has led to a drastic decrease of urinary tract infections in infants, postnatal infections still occur occasionally [65]. In the study of Dacher et al. [66] on infants with urinary tract infection, in spite of a prenatal diagnosis of hydronephrosis, four explanations are suggested: failure to communicate the prenatal findings, failure to prescribe prophylactic antibiotics, failure to administer the prescribed medication, and finally, the onset of infection in spite of adequate continuous antibiotic prophylaxis.

Conclusions

Prenatal US has made it possible to recognize fetal hydronephrosis and to treat obstructive and/or refluxing uropathies before the onset of clinical symptoms. The controversy about cut-off values, the variable standards of routine obstetrical ultrasound, and postnatal ultrasound triage are all closely interconnected.

In the United States and in Canada, a mid-second trimester ultrasound is routinely performed and further examinations are warranted only if there is evidence of an abnormality or an equivocal finding. In several European countries, three routine examinations are performed, one in each trimester. The importance of third-trimester ultrasound has been well demonstrated both for the screening of urinary tract dilatation and for the selection of patients needing postnatal investigations [55]. For the time being, the cost-effectiveness of routine third-trimester ultrasound is still being debated in North America. There is no doubt that many unnecessary VCUGs are performed postnatally, based upon non-significant mild pyelectasis in the second trimester, that would have resolved by the 32-week sonogram. In our institution an abnormal US according to the criteria already mentioned leads to postnatal investigations. When the renal pelvis AP diameter prior to 24 w.G.A. is ≥ 4 mm, a third trimester follow-up US is indicated. If it is within normal

Fig. 9a–c Progression of hydronephrosis in utero. **a** A 25-w.G.A. transverse scan shows a unilateral left hydronephrosis (pelvic AP diameter 16 mm). **b** Hydronephrosis has progressed with parenchymal thinning in 34 w.G.A. US (longitudinal scan). **c** In the same patient antegrade pyelography through a nephrostomy tube is suggestive of VUJ obstruction (*arrows*)

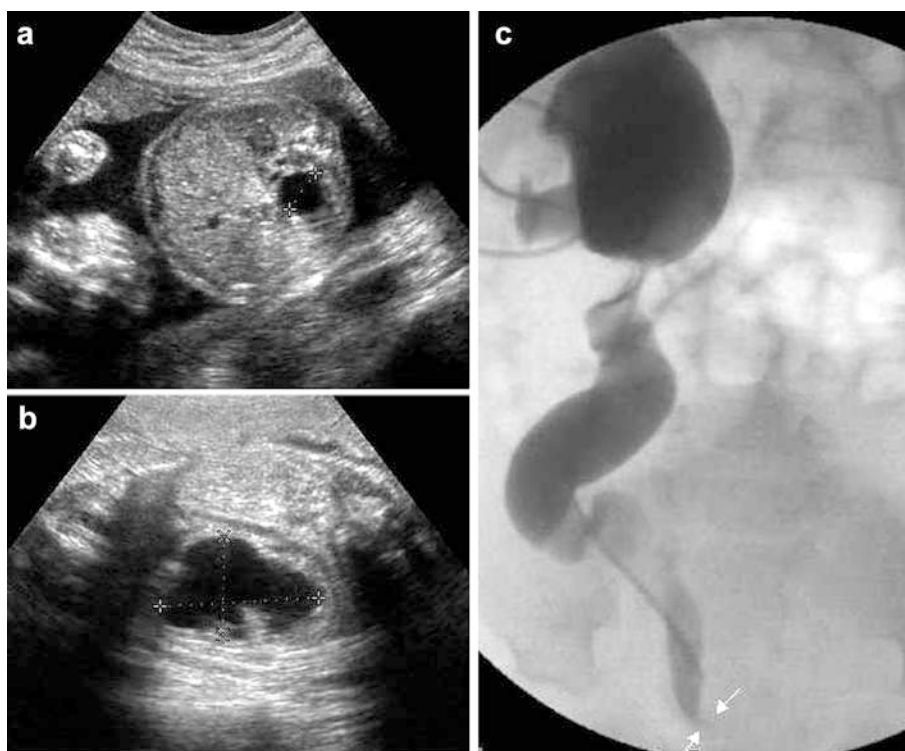


Fig. 10a–c VUR in duplex kidney. **a** A 35-w.G.A. sagittal scan shows hydronephrosis (grade IV–V/V) of the lower pole (*arrowheads*) of a left duplex kidney (in between calipers). **b** A corresponding longitudinal US scan in the same patient displays evidence of grade V/V hydronephrosis. **c** The neonatal VCUG demonstrates vesico-ureteral reflux (IV–V/V) to the lower pole of a duplex system. Isotopic renography (not shown) revealed non-function of the left lower pole and the patient underwent partial lower pole nephrectomy at the age of 7 months



range (<10 mm), no postnatal investigations are performed.

Prenatal imaging and screening go along with parental information in order to alleviate the resulting anxiety; the type of anomaly, the natural history, and the postnatal significance should be addressed in detail,

within a multidisciplinary framework including obstetricians, radiologists, geneticists, neonatologists, surgeons and paediatricians. Prenatal data should be available on site for appropriate postnatal investigations. Such continuity in care is best achieved in mother-child institutions.

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