Fetal Hydronephrosis

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Introduction

Ultrasound of the Fetal Genitourinary Tract

The incidence of identifying genitourinary abnormalities during prenatal ultrasound (US) screening is approximately 2–9 per 1,000 births, accounting for 17 % of all anomalies diagnosed prenatally. With the more common use of improved high-resolution US scanners, this incidence is rapidly increasing. The use of prenatal US allows children with congenital abnormalities of the genitourinary tract to be detected prior to developing signs and symptoms such as urinary tract infection, abdominal mass, hematuria, kidney stones, and pain. These children benefit from early diagnosis with the goal of preventing these complications and to preserve renal function when possible. However, not all findings on prenatal US represent pathology; many have no clinical significance. The dilemma is to be able to differentiate which children require intervention from those who do not. Specific findings on prenatal US can help to make this differentiation. Some important time points and US findings of the fetal urinary tract are listed in Table 1.1.

Hydronephrosis: The Scope of the Problem

Prenatal hydronephrosis affects 1–2 % of all pregnancies and is one of the most common prenatally detected anomalies. Although the use of prenatal ultrasound as a screening tool for birth defects has not been shown to improve perinatal outcome, more patients are undergoing prenatal counseling for the discovery of prenatal hydronephrosis. Children diagnosed with this entity on routine ultrasound often undergo extensive prenatal imaging that may include serial ultrasound and magnetic resonance imaging (MRI). In addition, they also undergo numerous postnatal examinations including serial renal ultrasound, voiding cystourethrogram (VCUG), diuretic renogram, intravenous pyelogram, and MRI urogram. Although current prenatal testing is mostly noninvasive, much of the postnatal assessment is invasive and exposes the child to radiation or anesthesia that may be unnecessary. The diagnosis of antenatal hydronephrosis (ANH) causes significant parental anxiety and physician uncertainty with regard to prenatal and postnatal management. Consequently, the efficacy and social

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Table 1.1 US key time points of the fetal GU tract

Fetal kidneys:
Ureteral bud formation at 5th week of gestation (weeks)
Urine formation at 5th–8th week
5 cm^3/h at 20th week
50 cm^3/h at 40th week
Can be visualized at 12th–13th week
Visualization of hydronephrosis at 12th–18th week
Distinct renal architecture at 20th week
Detailed examination is better in the second and third trimesters
Renal measurements: 12–40 weeks
AP diameter: 0.8–2.6 cm
Transverse diameter: 0.9–2.6 cm
Length: 1.0–2.7 cm
Fetal bladder:
Can be visualized at 14 weeks
Emptying of the fetal bladder can be seen at 15th week
Size:
10 cm^3 at 30th week
50 cm^3 at term
Amniotic fluid:
Early = transudate of amnion
Later = fetal urine + lung fluid-swallowing
Amniotic fluid volume:
380 cm^3 at 20th week
800 cm^3 at 28th week
800 cm^3 at 40th week
Amniotic volume dependent on urine production at 16th week
Etiology of polyhydramnios (>1.5 L)
Esophageal obstruction
Multicystic kidney
Mesoblastic nephroma
Some obstructive processes
Etiology of oligohydramnios (<0.5 L)
Amnion nodosum
Amniotic fluid leak
Urinary tract obstruction
Consequences of oligohydramnios
Pulmonary hypoplasia
Potter's syndrome: flat nose, recessed chin, low-set ears, bowed legs, small chest, talipes equinovarus, and hypoplastic hands
Limb deformities

health-care costs of routine prenatal ultrasound as a screening tool for potential postnatal health risks such as urinary tract pathologies remain undefined and quite controversial.

Table 1.2 Classification system for prenatal hydronephrosis

Date of detection	Degree of hydronephrosis	Pelvic diameter (mm)
Second trimester	Mild	4–7
	Moderate	8–10
	Severe	>10
Third trimester	Mild	7–10
	Moderate	10–15
	Severe	>15

Definitions

Hydronephrosis is used when describing the dilatation of renal pelvis (pelviectasis) and/or calyces (caliectasis). It can be physiologic and have no clinical consequences whatsoever or be caused by urinary tract pathologies such as obstruction or vesicoureteral reflux (VUR). It is important to identify the etiology of the hydronephrosis, because in itself it is merely a finding, not a diagnosis. The number of children diagnosed with prenatal hydronephrosis has increased in the past decade due to the more common use of fetal US imaging. Hydronephrosis presents in a spectrum, ranging from severe renal pelvic dilatation to small changes only noticeable to the trained eye. The anteroposterior (AP) diameter of the renal pelvis taken in the axial plane is the most commonly used measurement in defining prenatal hydronephrosis. It has been found to be the most simple and reliable parameter and is dependent on the gestational age of the fetus when the dilation is detected. While there remains controversy on the exact AP diameter considered being abnormal, there is a commonly accepted classification system (Table 1.2) used in describing prenatal hydronephrosis.

Natural History

In most of the cases, the etiology of prenatal hydronephrosis is considered to be physiological. It will most likely resolve at the end of the pregnancy or within the first year of life. This spontaneous resolution may be due to several factors related to the maturation of the urinary tract. Fetal urine production is 4–6 times greater than after birth, due to the higher renovascular

resistance, greater glomerular filtering rate (GFR), and lower concentrating ability. This high urine output can overwhelm the capacity of the collecting system, resulting in dilation. As the kidneys mature, the urine output decreases and the hydronephrosis improves. In addition, the collecting system is more compliant during fetal development compared to that after birth, due to the composition and orientation of elastin and collagen. As the collecting system matures, alterations in its composition allow for accommodation of greater volume of urine without significant dilation. Finally, dilation of the proximal collecting system can also result from partial or transient anatomical or functional obstructions, such as persistent ureteral folds or delays in normal peristalsis, that resolve during fetal development.

Sairam et al. reviewed 11,465 scans at 18–23 weeks and observed the resolution of prenatal hydronephrosis antenatally and after birth. When the AP diameter was less than 7 mm, all patients had spontaneous resolution of the prenatal hydronephrosis antenatally or shortly after birth. In contrast, approximately 45 % of those with AP diameter greater than 7 mm had resolution of the prenatal hydronephrosis. Other authors noted similar findings with approximately 30 % resolving antenatally and 50–60 % resolving postnatally.

Differential Diagnosis

The etiology for prenatal hydronephrosis includes transient or physiologic hydronephrosis, ureteropelvic junction (UPJ) obstruction, VUR, ureterovesical junction (UVJ) obstruction, multicystic dysplastic kidney, and posterior urethral valves (PUV). Their incidences are listed in Table 1.3. Less common causes include ureterocele, ectopic ureter, duplex system, and urethral atresia. The degree of hydronephrosis observed on the first prenatal US is a good predictor of postnatal pathology. In a recent meta-analysis of the literature, we found that risk of postnatal pathology positively correlated with the degree of hydronephrosis, from 12 % in the mild group to 88 % in the severe group (Table 1.4). With regard to the specific diagnosis, all pathologies except for VUR were positively correlated with the increasing degree of prenatal hydronephrosis. This supports the observation that US is a poor predictor of reflux (i.e., high grade of reflux may be present despite the absence of hydronephrosis).

When the degree of prenatal hydronephrosis is not known, we found that the degree of hydronephrosis is also a good predictor of postnatal pathology. When evaluating 1,441 children with a history of prenatal hydronephrosis, the risk of postnatal pathology increases from 19 % in the no hydronephrosis group to 96 % in the severe group (Table 1.5). With regard to the specific

Table 1.3 Differential diagnosis for PNH and their incidence

Diagnosis	Incidence
Transient/physiologic	50–70 %
UPJO	20–40 %
VUR	15–25 %
UVJO/megaureter	1–20 %
MCDK	2–5 %
PUB	1–5 %
Ureterocele, ectopic ureter, duplex system, urethral atresia	Less common

Table 1.4 Predictive value of the prenatal US in the risk for postnatal pathology

	Mild (%)	Mild-moderate (%)	Moderate (%)	Moderate-severe (%)	Severe (%)	Trend *p*-value
Any pathology	12	39	45	72	88	<0.001
UPJ	5	14	17	37	54	<0.001
VUR	4	11	14	12	9	0.10
PUV	0.2	0.9	0.9	6.7	5.3	<0.001
Ureteral obstruction	1	12	10	11	5	0.25
Others	1	2	3	6	15	0.002

Table 1.5 Predictive value of the postnatal US in the risk for postnatal pathology

	Normal (%)	Mild (%)	Mild-moderate (%)	Moderate (%)	Moderate-severe (%)	Severe (%)	Trend *p*-value
Any pathology	19	30	44	62	92	96	<0.05
UPJ	1	8	17	23	53	61	<0.05
VUR	15	19	24	26	25	25	<0.05
Ureteral obstruction	3	4	6	18	22	25	<0.05
Others	1	1	2	2	1	2	<0.05

Table 1.6 Other prenatal US findings that suggest potential postnatal pathology

Kidney:	Renal parenchyma—echogenicity and thickness
	Calyceal dilation
	Unilateral versus bilateral hydronephrosis
	Variation in the degree hydronephrosis
Ureter:	Ureteral dilation
Bladder:	Size and emptying
Urethra:	Posterior urethral dilation
Other:	Amniotic fluid volume
	Extra renal fluid
	Other anomalies
	Gender
	Overall growth and development

diagnosis, all pathologies except for VUR were positively correlated with the increasing degree of prenatal hydronephrosis. With respect to VUR, there is a statistical difference in the normal and mild group compared to all other groups, but there was no positive trend with the increasing degree of hydronephrosis.

It is very important to describe the fetal hydronephrosis not only by its severity but also on other renal, ureteral, bladder, and urethral US findings (Table 1.6). The presence of bilateral hydronephrosis suggests the presence of PUV, VUR, bilateral UPJ obstruction, urethral aplasia, prune belly syndrome, or megacystis-megaureter complex. The association with hydroureter increases the risk of VUR, UVJ obstruction, or PUV. A thickened bladder that does not empty completely with an associated dilated urethra (keyhole sign) is highly suggestive of PUV. Oligohydramnios and renal parenchymal changes such as increased echogenicity and thinning suggest the presence of associated compromise in renal function.

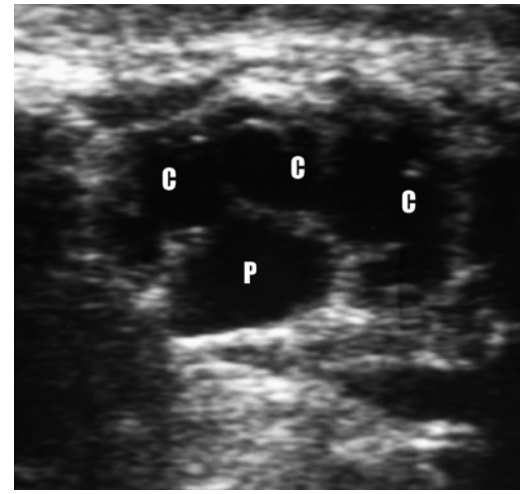

Fig. 1.1 Fetal ultrasound of a UPJ obstruction. Coronal imaging through the right kidney demonstrating dilated calyces (C) and a dilated renal pelvis (P), suggestive of a right UPJ obstruction

Ureteropelvic Junction Obstruction (UPJO)

Obstruction of urinary flow from the renal pelvis into the ureter is one of the more common causes of prenatal hydronephrosis, with an incidence ranging from 20 to 40 %. It is characterized on fetal US by the presence of renal pelvic dilatation and a normal bladder and the absence of a dilated ureter (Fig. 1.1). When occurring unilaterally, the amniotic fluid volume is unaffected. UPJ obstruction should be suspected in cases of moderate or severe dilatation. UPJ obstruction is unilateral in 70 % of the cases. It is usually sporadic, although familiar cases have been reported. The etiology of the obstruction can either be functional (i.e., abnormal peristalsis segment) or anatomic (i.e., caused by crossing vessels, fibrous bands, kinks, or polyps in the ureter).

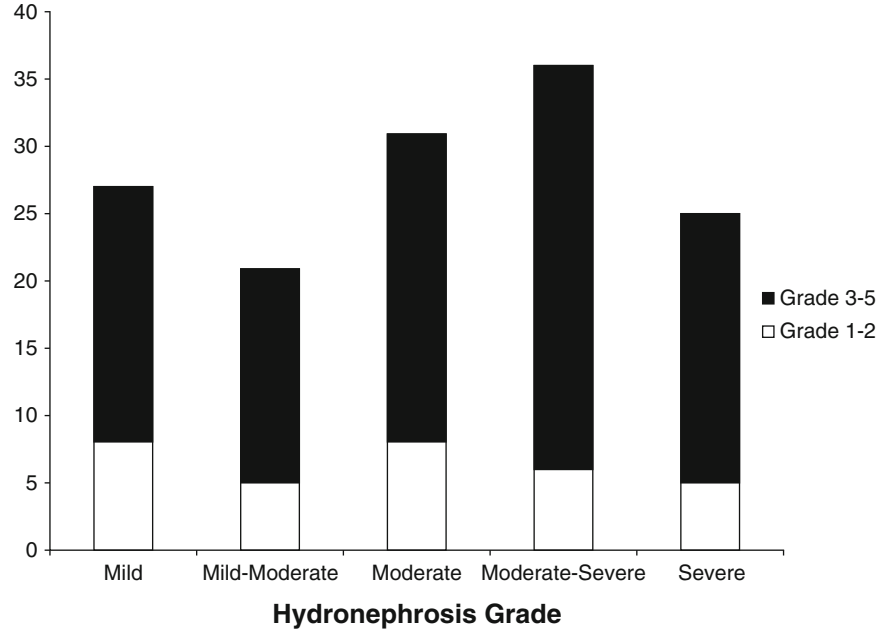

Fig. 1.2 The incidence and grade of VUR in children with PNH

Functional obstruction occurs in most cases of UPJ obstruction that were detected in the evaluation for prenatal hydronephrosis.

VUR

VUR occurs when urine flows from the bladder back to the kidney. The incidence of VUR in children with a history of PNH ranges from 15 to 25 %. On prenatal US, VUR is suggested when there is varying degree of hydronephrosis or hydroureteronephrosis during the scanning (Fig. 1.2). In a study performed in our institution, we observed that the degree of prenatal hydronephrosis did not correlate with the incidence or the grade of VUR (Fig. 1.3). However, the presence of a dilated ureter did increase the likelihood of VUR (odds ratio of 1.52). We could not identify any specific US or clinical predictors that can exclude VUR. Thus, there are no reliable findings to predict reflux on fetal US. The presence of significant hydroureteronephrosis, a large thin-walled bladder, and normal renal architecture and amniotic fluid in a male fetus may correspond to significant reflux, which has been termed as megacystis-megaureter association. VUR is bilateral in 60–70 % of the cases. It is the most commonly inherited anomaly of the genitourinary tract with a 33–40 % incidence in siblings.

Ureterovesical Junction Obstruction (UVJO)/Megaureter

Obstruction at the level of the junction between the ureter and the bladder impairs urinary flow from the distal ureter into the bladder, resulting in dilation of the entire collecting system from the distal ureter to calyces. The incidence of UVJ obstruction/megaureter in children with a history of PNH ranges from 10 to 20 %. On fetal US, UVJ obstruction is suggested when there is dilation of renal pelvis and ureter to the level of the bladder (Fig. 1.4). This diagnosis should be considered when a significantly dilated ureter is visualized. It is not uncommon for the dilated ureter to be mistaken as fetal bowel. The etiology of UVJ obstruction/megaureter may be due to a deficiency of smooth muscle in the ureterovesical ureter, resulting in an adynamic distal segment that impedes normal peristalsis of urine through the ureter (primary) or due to extrinsic compression of the ureter by a thick bladder wall

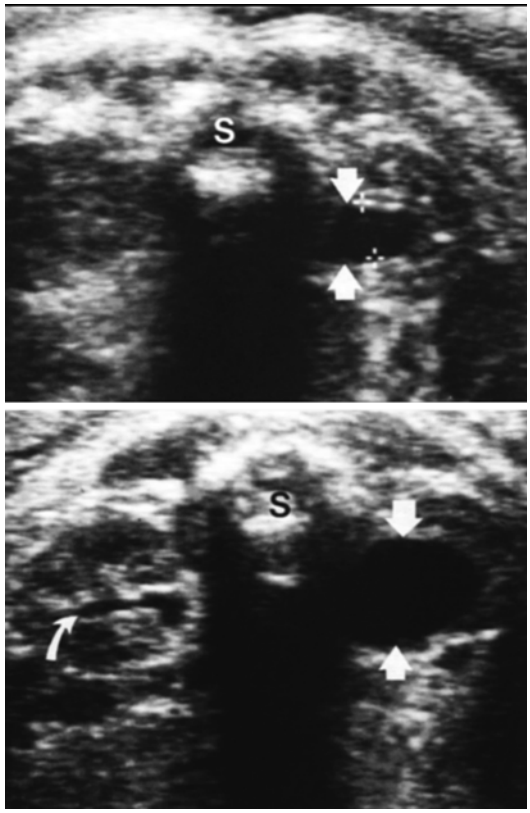

Fig. 1.3 Fetal US of VUR. Transverse imaging through the right and left kidney demonstrates fluctuation in the degree of hydronephrosis (*arrows*) during the same examination, suggestive of VUR. S = Spine

in pathological states such as PUV or neurogenic bladder (secondary). UVJ obstruction/megaureter is bilateral in 10–25 % of the cases, and most cases are sporadic without a genetic component.

Posterior Urethral Valves (PUV)

PUV are redundant folds that arise from the verumontanum on the floor of the urethra, extending toward the bulbomembranous junction and attaching to the urethra throughout its circumference. They have no active function but create a barrier to urine flow, leading to bladder outlet obstruction. This anomaly occurs exclusively in males. The incidence of PUV in children with a history of PNH ranges from 1 to 5 %. On fetal US, the diagnosis of PUV is suspected when there is uni- or bilateral hydroureteronephrosis, a thick wall bladder with persistent dilatation and a fusiform or pear-shaped appearance, and a dilated posterior urethra (keyhole sign) (Fig. 1.5). Increased renal echogenicity or cysts and varying degrees of oligohydramnios may be present. During the first trimester, echogenic kidneys and dilated renal pelves can be seen, though the amniotic fluid is usually normal since this fluid is not primarily of renal origin before 16 weeks. Variable US findings are seen due to the wide spectrum of severity of the disease. The etiology is hypothesized to be

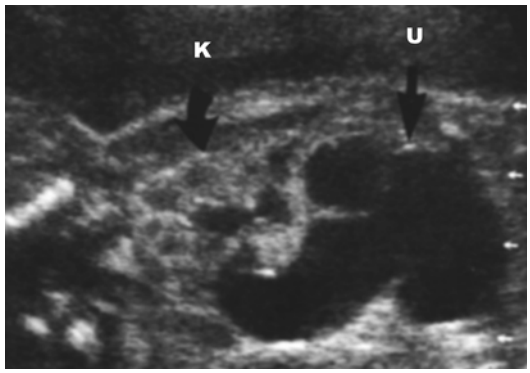

Fig. 1.4 Fetal US of UVJ obstruction/megaureter. Transverse imaging of the left kidney (K) demonstrates a dilated ureter (U) with mild pelviectasis. Together these findings suggest the diagnosis of UVJ obstruction/megaureter

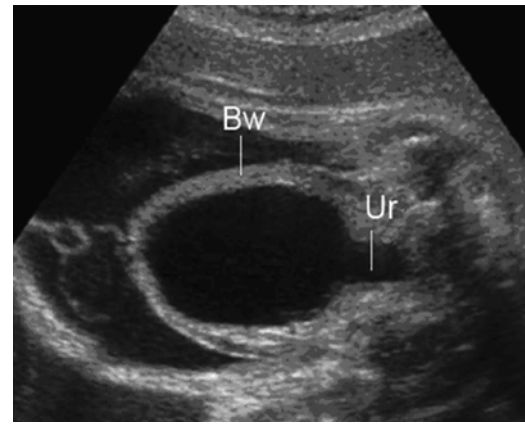

Fig. 1.5 Fetal US appearance of PUV. Sagittal imaging through the bladder demonstrates a thick bladder wall (Bw) and a dilated proximal urethra (Ur)—keyhole sign, suggestive of PUV

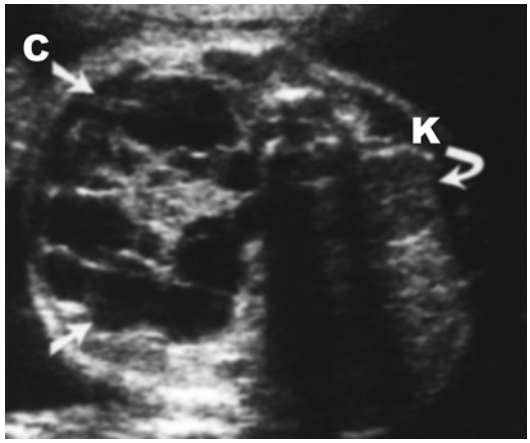

Fig. 1.6 Fetal US of MCDK. Transverse imaging of the left and right kidney demonstrates a normal left kidney (K) and multiple noncommunicating cystic structures and little parenchyma in the right kidney, suggestive of MCDK

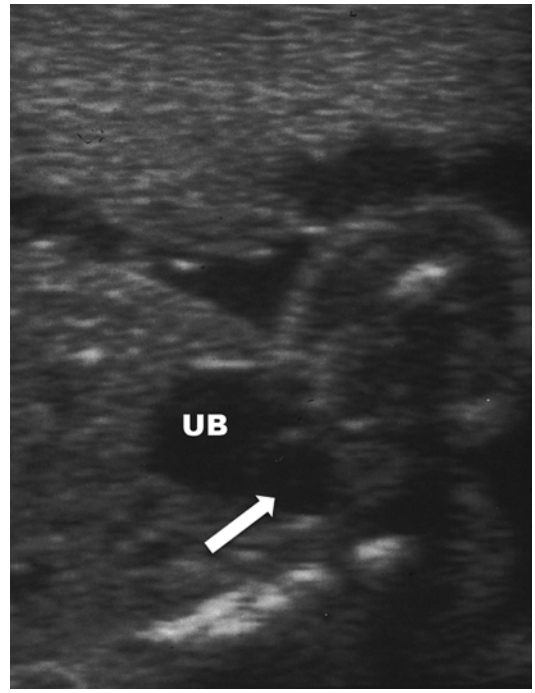

Fig. 1.7 Fetal US of ureterocele. Transverse imaging through the bladder (UB) demonstrates a cystic structure (*arrow*) at its base, suggestive of a ureterocele

that the terminal ends of the Wolffian ducts mismigrate and are integrated into the urethral wall abnormally, resulting in obliquely oriented ridges that act as one-way valve, impeding urine flow from the bladder. Most cases of PUV are sporadic without a genetic component.

Multicystic Dysplastic Kidney (MCDK)

MCDK is an anomaly of renal development, in which the renal parenchyma consists primarily of dysplastic elements (primitive ducts and metaplastic cartilage) with a preponderance of cysts encompassing the kidney. Not uncommonly, MCDK is mistaken for hydronephrosis on fetal US as. The incidence of MCDK in children with a history of PNH ranges from 2 to 5 %. On fetal US, the diagnosis of MCDK is suggested when a nonreniform structure with multiple noncommunicating fluid-filled cystic spaces without a central large cyst and little renal parenchyma is visualized in the renal fossa (Fig. 1.6). The etiology of MCDK is unknown, but it appears that it does not have a genetic component. VUR in the contralateral renal unit is found in 20–40 % of children with a unilateral MCDK.

Ureterocele

A ureterocele is a cystic dilatation of the intravesical submucosal ureter, usually associated with an obstructed orifice that impairs urinary flow into the bladder. When associated with a duplicated system (80 % of the cases), the ureterocele is associated with the upper pole collecting system (Weigert-Meyer Law). The incidence of ureterocele in children with prenatal hydronephrosis is less than 1 %. On fetal US, the diagnosis of a ureterocele is suggested when there is a thin-walled cystic structure in the base of the bladder with associated upper pole hydroureteronephrosis (Fig. 1.7). The etiology is unknown but is hypothesized that it results either from an incomplete breakdown of the ureteral (Chwalla) membrane present at the time of ureteral bud arising from the mesonephric duct or from a delay in the establishment of the lumen of the ureteral bud. There does not seem to be a genetic component; however, ureteroceles are more commonly seen in females than males (5–7 to 1). VUR is commonly found in association with the ureterocele, 50–70 % in the ipsilateral lower pole and 10–30 % in the contralateral renal unit.

Management of PNH: Which Patients Will Need Further Evaluation?

Once a fetal genitourinary abnormality is diagnosed, the management of the fetus is dependent upon the severity and the etiology of hydronephrosis and the gestational age at which it was diagnosed. When the hydronephrosis is severe (especially in bilateral cases) or is associated with oligohydramnios or echogenic kidneys, urgent referral should be made to help council the parents on diagnosis and management. Similarly, findings of bilateral hydroureteronephrosis, a thickened bladder wall, or dilated posterior urethra (suggestive of PUV) should be urgently evaluated. In most cases, the hydronephrosis is mild or moderate and is unilateral without associated changes in amniotic fluid or renal parenchymal abnormalities. Consequently, serial prenatal US should be performed to monitor for progression. There is no set recommendation on the frequency of US imaging but it may range from every 4–6 weeks.

The Role of Fetal Intervention

Occasionally, in utero intervention may be required with the goal of preventing pulmonary hypoplasia (by restoring normal amniotic fluid), improving kidney and bladder function. The type of fetal intervention includes early delivery, open bladder decompression, vesicoamniotic shunts, and minimally invasive endoscopic and laparoscopic valve ablation and vesicostomy. All of these procedures have significant maternal and fetal risks; consequently, it has to be determined that the benefits of fetal intervention outweigh the risks before undertaking such procedures.

The indication for fetal intervention in fetuses with hydronephrosis is limited to those with evidence of severe bladder outlet obstruction with oligohydramnios but have a normal karyotype, have no other systemic anomalies, are singleton fetuses, and, most importantly, have favorable renal function. The use of serial fetal bladder aspiration and examination of urine components (β2-microglobulin, sodium, chloride, and osmolality) are the most helpful methods of evaluating fetal renal function. As glomerular filtration rate increases, the presence of urinary sodium and low molecular weight plasma proteins (β2-microglobulin) decreases. However, in the case of dysplastic kidneys, electrolytes and proteins cannot be retained. Good renal function is seen in fetuses with favorable urinary indices (Na <100, Cl <110, Osm <210, β2 microglobulin <10–20) by serial sampling over 3 days. Urine sodium <100 mEq/mL and echolucent kidney are associated with good outcome (81 % survival), while sodium levels >100 mEq/mL and echogenic kidneys have been associated with poor outcome (12 % survival).

Postnatal Follow-Up

If the hydronephrosis resolved during pregnancy, further postnatal radiological evaluation may not be necessary. In children with a history of prenatal hydronephrosis that resolved during pregnancy, the incidence of VUR is less than 5 % and the incidence of urinary tract obstruction would be rare. However, the parents should be advised that if their child develops a urinary tract infection later on, he/she should be evaluated for VUR. In contrast, infants with history of prenatally detected hydronephrosis that did not resolve during pregnancy should undergo further postnatal follow-up. The postnatal US and a VCUG should be performed within several days after birth in children with a solitary kidney, severe bilateral hydronephrosis, and/or ureterocele. Serum electrolytes and creatinine should be obtained to evaluate renal function. If the diagnosis of PUV is suspected or confirmed, urinary drainage should be instituted as soon as possible.

In cases of moderate prenatal hydronephrosis that did not resolve or a history of prenatal hydronephrosis of unknown severity and a postnatal US that demonstrated moderate or worse grade of hydronephrosis, a postnatal US and a VCUG would be indicated since the incidence of clinically significant urinary tract pathology is high enough to outweigh the risks of performing

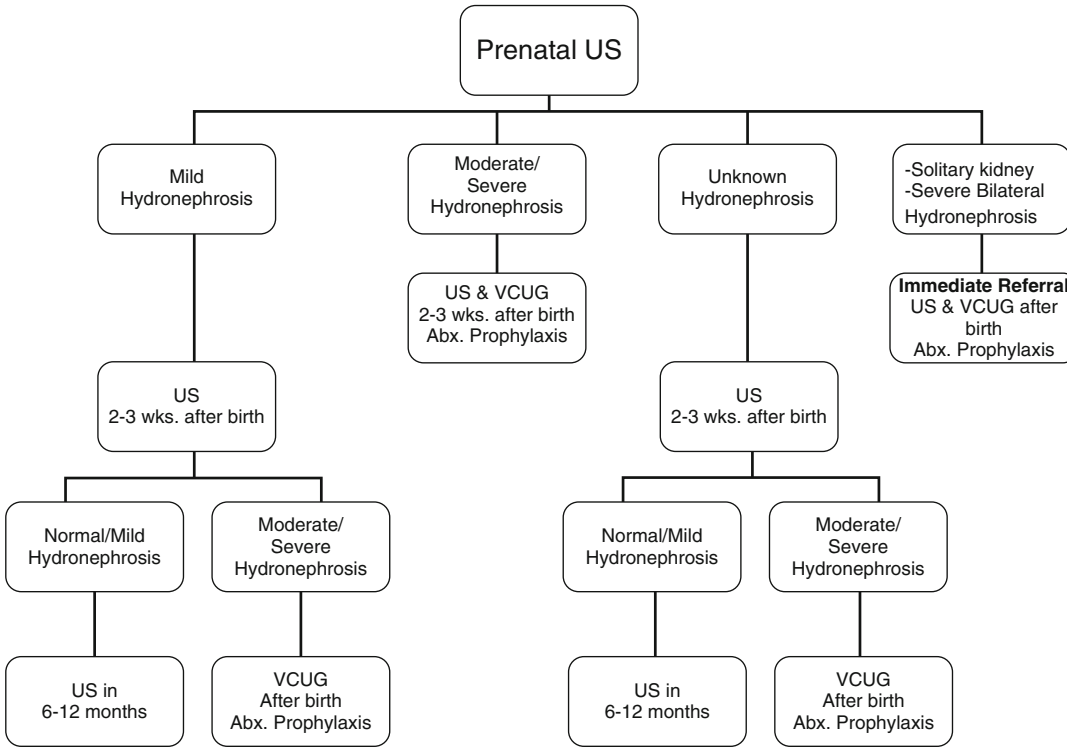

Fig. 1.8 Suggested follow-up according to ultrasound findings

the radiologic tests. The US should be performed after 48 h since the degree of dilation may be significantly underestimated during the first few days of life, due to neonatal oliguria. The management of children with mild prenatal hydronephrosis and no or mild hydronephrosis on postnatal US is more controversial. In general, it is recommended that a follow-up US be performed. There is no standard recommendation on the timing of the US, but it is usually suggested that it will be performed 6 months to a year after birth. A follow-up postnatal US is recommended since significant urinary tract obstruction has been documented in children with a normal postnatal US. In addition, since the US is noninvasive and does not require exposure to radiation, it has minimal associated risks. The need for a VCUG in these children is highly debated. The incidence of VUR ranges from 5 to 20 %. VCUG requires exposure to radiation and placement of a urethral catheter, making the procedure more invasive than US. Consequently, some practitioners routinely performed VCUG in these children, while others do not and recommend a VCUG only if subsequent UTI occurs. These recommendations are summarized in Fig. 1.8.

The Role of Antibiotic Prophylaxis

The advantages of using antibiotic prophylaxis in children with prenatal hydronephrosis have not been formally evaluated. It is expected that its use would prevent urinary tract infection and, consequently, prevent renal damage in the immature infant kidney. Some practitioners recommend antibiotic prophylaxis in all patients with confirmed postnatal hydronephrosis, while others only in cases with severe dilation. Oral amoxicillin (25 mg/kg once a day) is most commonly recommended during the first 3 months of life. Trimethoprim (2 mg/kg once a day) or nitrofurantoin (1–2 mg/kg once a day) may be utilized after 3 months of age. One practical approach

would be to use antibiotic prophylaxis only when VUR or lower urinary tract obstruction (such as UVJ obstruction/megaureter, ureterocele, and PUV) is suspected. The rationale for this approach is that children with VUR and lower urinary tract obstruction are at higher risks of developing UTIs than those with transient/physiologic hydronephrosis or upper urinary tract obstruction.

Antenatal Counseling

The diagnosis of prenatal hydronephrosis may cause significant parental anxiety with regard to its implication on renal function and fetal health and the need for prenatal and postnatal management. Consequently, it is important to assure the parents that prenatal hydronephrosis represents a spectrum of urinary tract anomalies with variable severity. The etiology of the hydronephrosis cannot be accurately determined by prenatal US. However, US findings such as AP size, the presence of oligohydramnios, and the onset on the dilatation may suggest its etiology and estimate the severity of the problem. In the majority of the cases (approximately 60 %), prenatal hydronephrosis is transient and has no significant clinical sequelae. Many will resolve during pregnancy or shortly after birth. Less than 5 % of the patients diagnosed with prenatal hydronephrosis require surgery for correction of VUR or urinary tract obstruction. Prenatal hydronephrosis may occur with subsequent pregnancy, occurring in 67 % of cases (relative risk of 6.1). Nevertheless, it is important to inform the parents about the importance of postnatal follow-up for diagnosis and appropriate management. It is possible in patients with mild prenatal hydronephrosis to avoid performing a VCUG; however, this must be done in selected cases with reliable families.

Recommended Reading

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