# Antenatal Hydronephrosis and its Management

Reyaz Ahmed Wani

## Introduction

Antenatal hydronephrosis (ANH) is the most frequently detected abnormality on routine prenatal ultrasonography, with an incidence ranging from 1% to 5% of all pregnancies depending on diagnostic thresholds and screening practices. [1,2] The widespread adoption of high-resolution obstetric ultrasound has shifted the clinical paradigm from late postnatal presentation of obstructive uropathy to early prenatal detection, surveillance, and risk stratification. [3] For pediatric surgeons and urologists, ANH represents a heterogeneous clinical entity encompassing transient physiological dilatation, vesicoureteral reflux (VUR), developmental anomalies like multicystic dysplastic kidneys (MCDK) or duplex kidneys and significant obstructive uropathies such as ureteropelvic junction (UPJ) obstruction, vesicoureteral junction obstruction (VUJO) and posterior urethral valves (PUV). [3,4] Although majority of cases resolve spontaneously, a subset is associated with progressive renal damage, underscoring the importance of judicious evaluation and timely intervention. [5].

## Epidemiology

Population-based studies report antenatal hydronephrosis in approximately 1–5% of fetuses undergoing routine mid-trimester ultrasound evaluation. [1,6] Male predominance is consistently observed, particularly in moderate to severe hydronephrosis, reflecting the higher incidence of UPJ obstruction and PUV in male infants. [7] Bilateral hydronephrosis is present in 20–40% of cases and is associated with a greater likelihood of clinically significant pathology and impaired renal outcomes. [8] Despite its prevalence, only 10–30% of infants with antenatally detected hydronephrosis ultimately require surgical intervention, highlighting the risk of overtreatment if management is not appropriately stratified [2,9].

## Embryology and Developmental Basis

Renal development begins at approximately the fifth week of gestation with the interaction between the ureteric bud and the metanephric mesenchyme. The ureteric bud undergoes multiple

divisions and ultimately forms the entire collecting system by 20 weeks gestation after about 15 divisions, while the metanephric mesenchyme starts differentiating into nephrons from the seventh week of gestation by ureteric bud-mesenchymal interactions. [10] About one-third of nephrons are formed by the time complete collecting system is formed, and the process continues at rapid pace and is complete by 36 weeks of gestation. Mesonephric duct fuses with the urogenital sinus (future bladder) and part of it gets incorporated into posterior wall of bladder. Urine formation begins between the 5$^{th}$ and 9$^{th}$ weeks of gestation and keeps on increasing throughout gestation, and is the main contributor towards amniotic fluid volume after the first trimester of pregnancy.

Disruptions in ureteric bud branching, canalization, or integration with the bladder may result in congenital anomalies of the kidney and urinary tract (CAKUT), many of which present as antenatal hydronephrosis. [11] Obstruction occurring during active nephrogenesis, which continues until 34–36 weeks' gestation, may impair nephron formation and lead to irreversible renal dysplasia. In contrast, obstruction occurring later in gestation primarily affects renal growth and functional maturation. [12] Lower urinary tract obstruction affects both kidneys and can result in decreased urine output from fetal kidneys and oligohydramnios.

## Definitions and Classification Systems

*Anteroposterior Renal Pelvic Diameter (APD)*

Measurement of the anteroposterior diameter (APD) of the renal pelvis in the transverse plane remains the most widely used quantitative method for defining antenatal hydronephrosis. [2] Gestational age–specific thresholds are essential, as physiologic pelvic dilatation increases with advancing gestation (Table 1). Persistence or progression of hydronephrosis into the third trimester, particularly with APD >15 mm, is strongly predictive of postnatal pathology and need for surgical intervention. [1,13]

**Table 1. Definition of ANH by APD[3]**

| Degree of ANH | Second Trimester | Third Trimester |
|---|---|---|
| Mild | 4 to <7 mm | 7 to <9 mm |
| Moderate | 7 to ≤10 mm | 9 to ≤15 mm |
| Severe | >10mm | >15mm |

*Society for Fetal Urology (SFU) Grading System*

The SFU grading system classifies hydronephrosis from Grade 0 to Grade 4 based on the degree of pelvic and calyceal dilatation and renal parenchymal thinning (Table 2). [14] Although widely used postnatally, its antenatal utility is limited by interobserver variability and lack of assessment of ureteral and bladder abnormalities. [15]

**Table 2. SFU Grading system of Hydronephrosis [14]**

| SFU Grade | Renal Pelvis | Calyces | Renal Parenchyma | Key surgical implication |
|---|---|---|---|---|
| Grade 0 | No dilatation | None | Normal thickness and echogenicity | Normal kidney |
| Grade 1 | Mild dilatation of renal pelvis only | No calyceal dilatation | Normal | Usually transient, observation |
| Grade 2 | Moderate pelvic dilatation | Few calyces visualized, not dilated | Normal | Low risk, follow up imaging |
| Grade 3 | Marked pelvic dilatation | Uniform dilatation of all calyces | Parenchyma preserved | Intermediate risk; functional assessment needed |
| Grade 4 | Severe pelvic dilatation | Ballooned calyces | Parenchymal thinning ± increased echogenicity | High risk; likely obstruction, surgical evaluation |

*Urinary Tract Dilation (UTD) Classification System*

To address limitations of prior systems, a multidisciplinary consensus group developed the Urinary Tract Dilation (UTD) classification system, which integrates APD, calyceal dilatation, renal parenchymal appearance, ureteral dilatation, bladder abnormalities, and amniotic fluid volume. [3] This system provides standardized prenatal (UTD A1–A3) and postnatal (UTD P1–P3) risk stratification and is increasingly adopted in clinical practice (Figure 1 and 2).

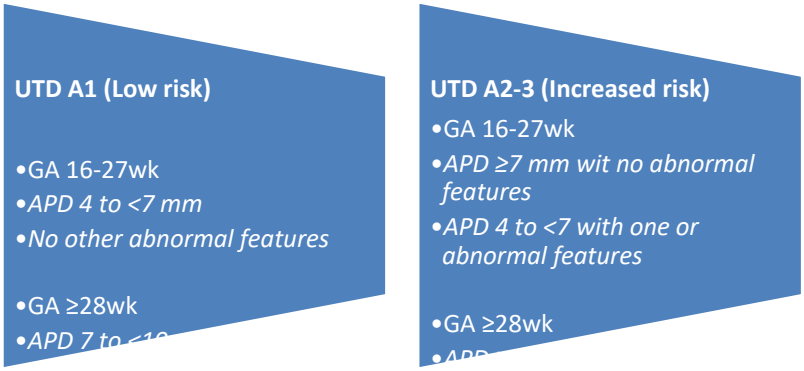


**Figure 1. Antenatal UTD Risk Stratification: UTD A1 (low risk) and UTD A2-3 (increased risk)**

Note: Abnormal features include *Peripheral calyceal dilation*, *parenchymal thinning*, *abnormal parenchymal appearance*, *abnormal ureters or bladder* and *unexplained oligohydramnios*

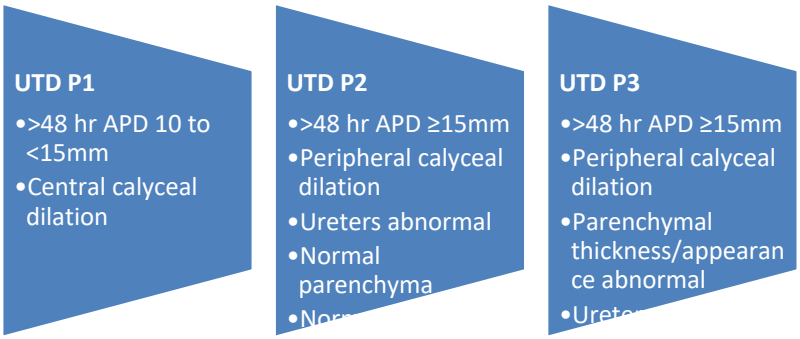


**Figure 2. Postnatal UTD Risk Stratification: UTD P1 (low risk), UTD P2 (intermediate risk), and UTD P3 (high risk)**

## Etiology and Differential Diagnosis

UPJ obstruction is the most common cause of clinically significant antenatal hydronephrosis, accounting for up to 50% of cases requiring intervention. [16] It is typically unilateral and characterized by renal pelvic and calyceal dilatation without ureteral involvement. Vesicoureteral reflux accounts for approximately 10–20% of antenatal hydronephrosis cases, particularly those with mild to moderate dilatation. [17] Posterior urethral valves represent the most severe etiology, characterized by bilateral hydronephrosis, hydroureter, bladder wall thickening, and often oligohydramnios, with a high risk of chronic kidney disease. [18] Other less common causes include MCDK, duplex anomalies, ureterocele, Prune belly syndrome and polycystic kidney disease (Table 3).

**Table 3. Etiology of Antenatal Hydronephrosis (ANH) [19]**

| S. No. | Etiology | Incidence (%) |
|---|---|---|
| 1. | Transient/Physiologic | 50-70 |
| 2. | Ureteropelvic junction obstruction | 10-30 |
| 3. | Vesicoureteral reflux | 10-40 |
| 4. | Ureterovesical junction obstruction/megaureter | 5-15 |
| 5. | Multicystic dysplastic kiudney | 2-5 |
| 6. | Posterior urethral valves | 1-5 |
| 7. | Ureterocele, ectopic ureter, duplex system, urethral atresia, Prune belly syndrome, polycystic kidney diseases, cysts | Uncommon |

## Prenatal Diagnosis

Prenatal ultrasound is the primary diagnostic modality for antenatal hydronephrosis. Comprehensive evaluation includes APD measurement, assessment of calyceal involvement, renal parenchymal thickness and echogenicity, ureteral dilatation, bladder morphology, and amniotic

fluid volume. [6] Serial ultrasound examinations (4-6 weekly) are essential, as progressive dilatation is a stronger predictor of obstruction than a single measurement. [1] Fetuses with suspicion of lower urinary tract obstruction or significant postnatal pathology like bilateral hydroureteronephrosis, distended bladder, progressive calyceal dilatation, parenchymal thinning, increased cortical echogenicity and oligohydramnios) are candidate for more frequent sonographic evaluation. [19-22] Fetal MRI may be useful in selected complex cases but is not routinely indicated. [23]

Antenatal Management and Counseling

Antenatal management is primarily observational, with intervention reserved only for cases of severe lower urinary tract obstruction threatening fetal viability. [24] The main objectives are accurate diagnosis, risk stratification, and parental counseling. Families should be informed that most cases resolve spontaneously, while moderate to severe hydronephrosis requires structured postnatal evaluation. [5] The general recommendations of prenatal monitoring schedule are as follows (Figure 3): (a) Fetuses with low risk of postnatal pathology (UTD A1) diagnosed before 32 weeks, should undergo ultrasound at or beyond 32 weeks to look for the progress. In cases of documented resolution with normal renal parenchyma, ureters and bladder, no further prenatal or postnatal follow up is warranted. In cases with persistent UTD A1 or A2-3, postnatal evaluation is recommended. (b) Cases that fall in high risk category (UTD A2-3) at the time of initial prenatal diagnosis, should undergo repeat US after 4-6 weeks and subsequent interval assessments as decided by the clinician. The risk of aneuploidy is minute in isolated ANH, however, when associated with any major structural anomaly (cardiac, neurological, skeletal or gastrointestinal) or one or more additional soft signs (absent nasal bone, increased nuchal translucency, shortened femur/humerus, echogenic bowel, choroid plexus cyst, ventriculomegaly, intracardiac echogenic focus), the risk increases substantially and further evaluation/counseling and decision of karyotyping is taken at specialized centres. [25,26]

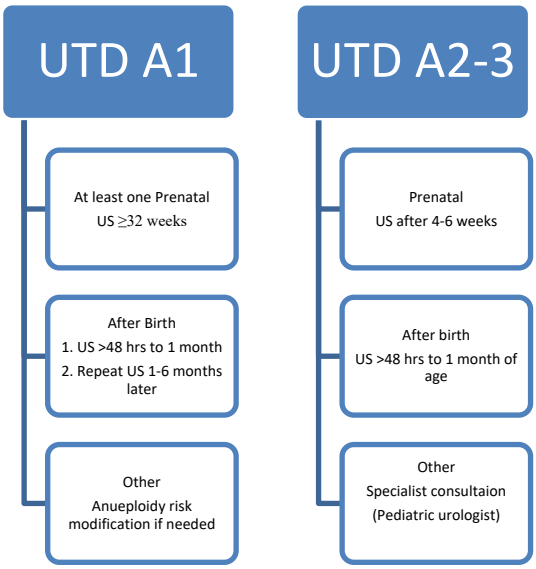


**Figure 3. Management schema based on UTD classification system's risk stratification of UTD A1 and UTD A2-3** [3]

Prenatal Intervention for Lower Urinary Tract Obstruction

Fetal lower urinary tract obstruction (LUTO) is characterized by bilateral hydroureteronephrosis with dilated/ thick-walled bladder with dilated posterior urethra (key-hole sign). The underlying etiology is posterior urethral valve (PUV) in most of the cases, however, the alternate diagnosis like anterior urethral valve (AUV), Prune belly syndrome, prolapsed ureterocele or urethral atresia are also seen. [24,27] The in-utero and post-natal course is quite variable with severely affected ones presenting with severe oligohydramnios or anhydramnios, renal dysplasia or even pulmonary hypoplasia. [28] The sonographic indicators of possible end stage obstructive uropathy are small hyperechoic kidneys with subcortical cysts.[29] It is important to distinguish severe form that might require intervention from milder disease with minimal clinical sequelae [30]. Besides assessment of fetal anatomical abnormalities and amniotic fluid volume, genetic evaluation in LUTO is paramount as 10% cases are associated with trisomies 13, 18 or 21. [26] Ever since the first evidence of improved survival from VAS over conservative management in a randomized controlled (Percutaneous Vesicoamniotic Shunting for fetal Lower Urinary Tract Obstruction-PLUTO) trial, antenatal intervention (vesicoamniotic shunting-VAS or cystoscopic valve ablation using flexible fetoscope) is being used selectively in LUTO [31-33]. Fetal urinary biochemical

analysis has prognostic significance in such patients and can help select subjects for intervention (Table 4). [34] A more comprehensive grading system and need for fetal intervention was proposed by Ruano et al in 2015 (Table 5). [35] Potential complications of fetal intervention are intrauterine death, termination of pregnancy, chorioamnionitis, blocked shunt, premature rupture of membranes, shunt migration, bowel perforation or herniation. [31,32]

**Table 4. Favourable prognostic criteria using fetal urine [34]**

| | |
|---|---|
| Sodium (Na) | Less than 100 mEq per liter |
| Chloride (Cl) | Less than 90 mEq per liter |
| Osmolarity (Osm) | Less than 210 mEq per liter |
| Calcium (Ca) | Less than 2 mmol per liter |
| Beta-2 microglobulin | Less than 2 mg per liter |

**Table 5. Staging system proposed by Ruano et al [35]**

| | Stage 1 | Stage 2 | Stage 3 | Stage 4 |
|---|---|---|---|---|
| Amniotic fluid | Normal | Oligohydramnios or Anhydramnios | Oligohydramnios, but usually Anhydramnios | Anhydramnios |
| Echogenicity of fetal kidneys | Normal | Echogenic | Echogenic | Echogenic |
| Renal cortical cysts | Absent | Absent | Can be present | Present |
| Renal dysplasia | Absent | Absent | Can be present | Present |
| Fetal urinary biochemistry | Favourable | Favourable within three consecutive evaluations | Not favorable after three consecutive evaluations | Unfavorable, poor bladder re-filling after vesicocentesis |
| Fetal intervention | Not indicated | Indicated to prevent | May be indicated to prevent | Not indicated amino infusion only |

| | | | | |
|---|---|---|---|---|
| | | pulmonary hypoplasia and severe renal impairment (VAS or cystoscopy) | pulmonary hypoplasia but not postnatal renal impairment (VAS with amino-infusion); further studies are necessary | |

Current evidence suggests fetal intervention in the form of VAS in selected cases of LUTO offers perinatal survival benefit. However, parental counseling regarding long term outcome like renal functional impairment or mortality is very vital. [33]

Postnatal Management

Postnatal renal ultrasound should be performed after 48–72 hours of life to avoid underestimation of hydronephrosis due to neonatal dehydration. [13] Early scans are likely to miss abnormalities of kidney and urinary tract due to low urine flow rate secondary to physiological dehydration and low glomerular filtration rate (GFR). [36] However in suspected cases of infravesical obstruction like PUV, early USG followed by voiding cystourethrogram (VCUG) is a norm to expedite evaluation and initiate timely management. Two normal postnatal ultrasounds invariably rule out significant renal pathology. [37] The scheme of management is guided by the risk stratification proposed by UTD classification system (Fig 3). [3]

1. *Low risk hydronephrosis (UTD P1)*
    - No routine antibiotic prophylaxis in most of the cases
    - Repeat renal ultrasound at 4-6 weeks of age
    - Continued surveillance at 3–6-month intervals until resolution or stability is confirmed
    - VCUG is not routinely indicated unless there are recurrent UTIs, ureteral dilatation or worsening hydronephrosis
2. *Intermediate risk hydronephrosis (UTD P2)*

- Initiate antibiotic prophylaxis in selected infants, especially in cases of ureteric dilatation or bilateral disease
- Perform repeat ultrasound at 4-6 weeks
- VCUG is recommended when ureteral dilatation, bladder abnormalities, or bilateral hydronephrosis are present to rule out VUR or posterior urethral valves (PUV)
- Diuretic renography (MAG3 or EC or DTPA scan) is performed at 6-12 weeks of age if hydronephrosis persists, to assess drainage pattern and differential renal function (DRF)

3. *High risk hydronephrosis (UTD P3)*
    - Early postnatal ultrasound and repeat imaging in 1-2 weeks
    - Prophylactic antibiotics are recommended due to increased risk of UTI
    - VCUG to exclude VUR and PUV
    - Early diuretic renography to evaluate drainage and DRF
    - To decide early surgical intervention (if indicated)

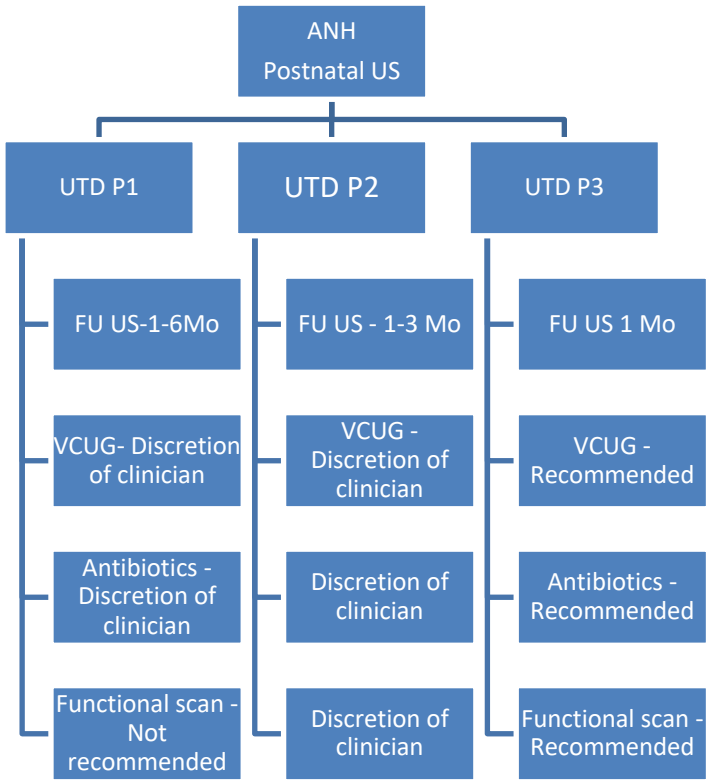

**Figure 3. Postnatal management schema based on Urinary Tract Dilation (UTD) classification** [3]

## Role of Voiding Cystourethrography

VCUG is the gold standard for diagnosing VUR. Routine VCUG for all cases of ANH is no longer recommended; instead, it is reserved for infants with moderate to severe hydronephrosis, ureteral dilatation, abnormal bladder findings or recurrent febrile UTIs. Selective use of VCUG reduces unnecessary invasive testing without compromising detection of clinically significant reflux.

Posterior urethral valves should be strongly suspected in male infants with bilateral hydronephrosis, dilated posterior urethra, thick-walled bladder, or oligohydramnios antenatally. Early VCUG in these patients allows timely confirmation of diagnosis, valve ablation and prevention of progressive renal damage.

## Diureteric Renography and Functional Assessment

Diureteric renography using technetium-99m mercaptoacetyltriglycine (MAG3) or Ethylene Dicysteine (EC) or diethylenetriamine pentaacetic acid (DTPA) is the cornerstone for evaluating drainage and differential renal function. It is typically performed after 6-12 weeks of age, when renal maturation allows reliable functional assessment. Indications include:

- Persistent moderate or severe hydronephrosis
- Increasing APD on serial ultrasounds
- Suspected PUJO or VUJO
- Declining renal parenchymal thickness

A DRF <40% in the affected kidney or a progressive decline on serial studies suggests significant obstruction warranting surgical correction.

**Antibiotic Prophylaxis-** The role of continuous antibiotic prophylaxis (CAP) has evolved, and current evidence suggests selective use rather than routine prophylaxis for all ANH cases. [8] CAP is generally recommended in:

- High grade hydronephrosis
- Suspected VUR

- Bilateral disease or solitary kidney

**Indications for Surgical Intervention**

Approximately only 20-30% infants with ANH require surgery. The most common indication is PUJO followed by VUR, VUJO and ureterocele. Surgical decisions are based on a combination of imaging and functional criteria rather than APD alone.

**Outcomes and Prognosis**

With contemporary risk-based management, the prognosis for most children with antenatal hydronephrosis is excellent. [9] However, infants with severe bilateral disease, posterior urethral valves, or dysplastic kidneys require long-term follow-up due to the risk of chronic kidney disease, hypertension, and bladder dysfunction. [38,39]

**References**


1. Lee RS, Cendron M, Kinnamon DD, Nguyen HT. Antenatal hydronephrosis as a predictor of postnatal outcome: a meta-analysis. Pediatrics. 2006 Aug; 118(2):586-93.
2. Sidhu G, Beyene J, Rosenblum ND. Outcome of isolated antenatal hydronephrosis: a systematic review and meta-analysis. Pediatr Nephrol. 2006 Feb;21(2):218-24.
3. Nguyen HT, Benson CB, Bromley B, et al. Multidisciplinary consensus on the classification of prenatal and postnatal urinary tract dilation (UTD classification system). J Pediatr Urol. 2014 Dec;10(6):982-98.
4. Koff SA. Neonatal management of unilateral hydronephrosis. Role for delayed intervention. Urol Clin North Am. 1998 May;25(2):181-6.
5. Onen A. Treatment and outcome of prenatally detected newborn hydronephrosis. J Pediatr Urol. 2007 Dec;3(6):469-76.
6. Corteville JE, Gray DL, Crane JP. Congenital hydronephrosis: correlation of fetal ultrasonographic findings with infant outcome. Am J Obstet Gynecol. 1991 Aug;165(2):384-8.
7. Peters CA. Urinary tract obstruction in children. J Urol. 1995 Nov;154(5):1874-83; discussion 1883-4.

8. Braga LH, Mijovic H, Farrokhyar F, Pemberton J, DeMaria J, Lorenzo AJ. Antibiotic prophylaxis for urinary tract infections in antenatal hydronephrosis. Pediatrics. 2013 Jan;131(1):e251-61.
9. Leigh J, Rickard M, Sanger S, Petropoulos J, Braga LH, Chanchlani R. Antibiotic prophylaxis for prevention of urinary tract infections in the first year of life in children with vesicoureteral reflux diagnosed in the workup of antenatal hydronephrosis: a systematic review. Pediatr Nephrol. 2020 Sep;35(9):1639-1646.
10. Kagan M, Pleniceanu O, Vivante A. The genetic basis of congenital anomalies of the kidney and urinary tract. Pediatr Nephrol. 2022 Oct;37(10):2231-2243.
11. Walawender L, Becknell B, Matsell DG. Congenital anomalies of the kidney and urinary tract: defining risk factors of disease progression and determinants of outcomes. Pediatr Nephrol. 2023 Dec;38(12):3963-3973.
12. Chevalier RL. Pathophysiology of obstructive nephropathy in the newborn. Semin Nephrol. 1998 Nov;18(6):585-93.
13. Sinha A, Bagga A, Krishna A, Bajpai M, Srinivas M, Uppal R, Agarwal I. Revised guidelines on management of antenatal hydronephrosis. Indian J Nephrol. 2013 Mar;23(2):83-97.
14. Nguyen HT, Herndon CD, Cooper C, Gatti J, Kirsch A, Kokorowski P, et al. The Society for Fetal Urology consensus statement on the evaluation and management of antenatal hydronephrosis. J Pediatr Urol 2010;6:212e31.
15. Chalmers DJ, Meyers ML, Brodie KE, Palmer C, Campbell JB. Inter-rater reliability of the APD, SFU and UTD grading systems in fetal sonography and MRI. J Pediatr Urol. 2016 Oct;12(5):305.e1-305.e5.
16. Vemulakonda VM. Ureteropelvic junction obstruction: diagnosis and management. Curr Opin Pediatr. 2021 Apr 1;33(2):227-234.
17. Kangin M, Aksu N, Yavascan O, Anil M, Kara OD, Bal A, Kamit F. Significance of Postnatal Follow-up of Infants with Vesicoureteral Reflux Having Antenatal Hydronephrosis. Iran J Pediatr. 2010 Dec;20(4):427-34.
18. Eckoldt F, Heling KS, Woderich R, Wolke S. Posterior urethral valves: prenatal diagnostic signs and outcome. Urol Int. 2004;73(4):296-301.

19. Maizels M, Wang E, Sabbagha RE, Dinsmoor M, Seshadri R, Ginsberg N, *et al*. Late second trimester assessment of pyelectasis (SERP) to predict pediatric urological outcome is improved by checking additional features. J Matern Fetal Neonatal Med. 2006;19:295-303.
20. Sibai H, Salle JL, Houle AM, Lambert R. Hydronephrosis with diffuse or segmental cortical thinning: impact on renal function. J Urol. 2001;165:2293-5.
21. Estroff JA, Mandell J, Benacerraf BR. Increased renal parenchymal echogenicity in the fetus: importance and clinical outcome. Radiology. 1991;181:135-59.
22. Haeri S. Fetal Lower Urinary Tract Obstruction (LUTO): a practical review for providers. Matern Health Neonatol Perinatol. 2015 Nov 18;1:26.
23. Chauvin NA, Epelman M, Victoria T, Johnson AM. Complex genitourinary abnormalities on fetal MRI: imaging findings and approach to diagnosis. AJR Am J Roentgenol. 2012 Aug;199(2):W222-31.
24. Morris RK, Middleton LJ, Malin GL, Quinlan-Jones E, Daniels J, Khan KS, et al. Outcome in fetal lower urinary tract obstruction: a prospective registry. Ultrasound Obstet Gynecol. 2015;46(4):424–31.
25. Bromley B, Lieberman E, Shipp TD, Benacerraf BR. The genetic sonogram: A method of risk assessment for Down syndrome in the second trimester. J Ultrasound Med. 2002;21:1087–96.
26. Staebler M, Donner C, Van Regemorter N, Duprez L, De Maertelaer V, Devreker F, et al. Should determination of the karyotype be systematic for all malformations detected by obstetrical ultrasound? Prenat Diagn. 2005;25:567–73.
27. Anumba DO, Scott JE, Plant ND, Robson SC. Diagnosis and outcome of fetal lower urinary tract obstruction in the northern region of England. Prenat Diagn. 2005;25:7–13.
28. Ruano R. Fetal surgery for severe lower urinary tract obstruction. Prenat Diagn. 2011;31:667–74.
29. Kilby MD, Morris RK. Fetal therapy for the treatment of congenital bladder neck obstruction. Nat Rev Urol. 2014;11:412–9.
30. Glick PL, Harrison MR, Golbus MS, Adzick NS, Filly RA, Callen PW, et al. Management of the fetus with congenital hydronephrosis II: Prognostic criteria and selection for treatment. J Pediatr Surg. 1985;20:376–87.
31. Ruano R, Sananes N, Sangi-Haghpeykar H, Hernandez-Ruano S, Moog R, Becmeur F, et al. Fetal intervention for severe lower urinary tract obstruction: a multicenter case–control study

comparing fetal cystoscopy with vesicoamniotic shunting. Ultrasound Obstet Gynecol. 2015;45:452–8.

32. Morris RK, Malin GL, Quinlan-Jones E, Middleton LJ, Hemming K, Burke D, Daniels JP, Khan KS, Deeks J, Kilby MD; Percutaneous vesicoamniotic shunting in Lower Urinary Tract Obstruction (PLUTO) Collaborative Group. Percutaneous vesicoamniotic shunting versus conservative management for fetal lower urinary tract obstruction (PLUTO): a randomised trial. Lancet. 2013 Nov 2;382(9903):1496-506.
33. Vinit N, Gueneuc A, Bessières B, Dreux S, Heidet L, Salomon R, Lapillonne A, De Bernardis G, Salomon LJ, Stirnemann JJ, Blanc T, Ville Y. Fetal Cystoscopy and Vesicoamniotic Shunting in Lower Urinary Tract Obstruction: Long-Term Outcome and Current Technical Limitations. Fetal Diagn Ther. 2020;47(1):74-83.
34. Abdennadher W, Chalouhi G, Dreux S, Rosenblatt J, Favre R, Guimiot F, Salomon LJ, Oury JF, Ville Y, Muller F. Fetal urine biochemistry at 13-23 weeks of gestation in lower urinary tract obstruction: criteria for in-utero treatment. Ultrasound Obstet Gynecol. 2015 Sep;46(3):306-11.
35. Ruano R, Dunn T, Braun MC, Angelo JR, Safdar A. Lower urinary tract obstruction: fetal intervention based on prenatal staging. Pediatr Nephrol. 2017 Oct;32(10):1871-1878.
36. Clautice-Engle T, Anderson NG, Allan RB, Abbott GD. Diagnosis of obstructive hydronephrosis in infants: comparison sonograms performed 6 days and 6 weeks after birth. AJR Am J Roentgenol. 1995;164:963-7.
37. Ismaili K, Hall M, Donner C, Thomas D, Vermeylen D, Avni FE; Brussels Free University Perinatal Nephrology study group. Results of systematic screening for minor degrees of fetal renal pelvis dilatation in an unselected population. Am J Obstet Gynecol. 2003;188:242-6.
38. Capone V, Persico N, Berrettini A, et al. Definition, diagnosis and management of fetal lower urinary tract obstruction: consensus of the ERKNet CAKUT-Obstructive Uropathy Work Group. Nat Rev Urol. 2022 May;19(5):295-303.
39. Robinson CH, Rickard M, Jeyakumar N, Smith G, Richter J, Van Mieghem T, Dos Santos J, Chanchlani R, Lorenzo AJ. Long-Term Kidney Outcomes in Children with Posterior Urethral Valves: A Population-Based Cohort Study. J Am Soc Nephrol. 2024 Dec 1;35(12):1715-1725.