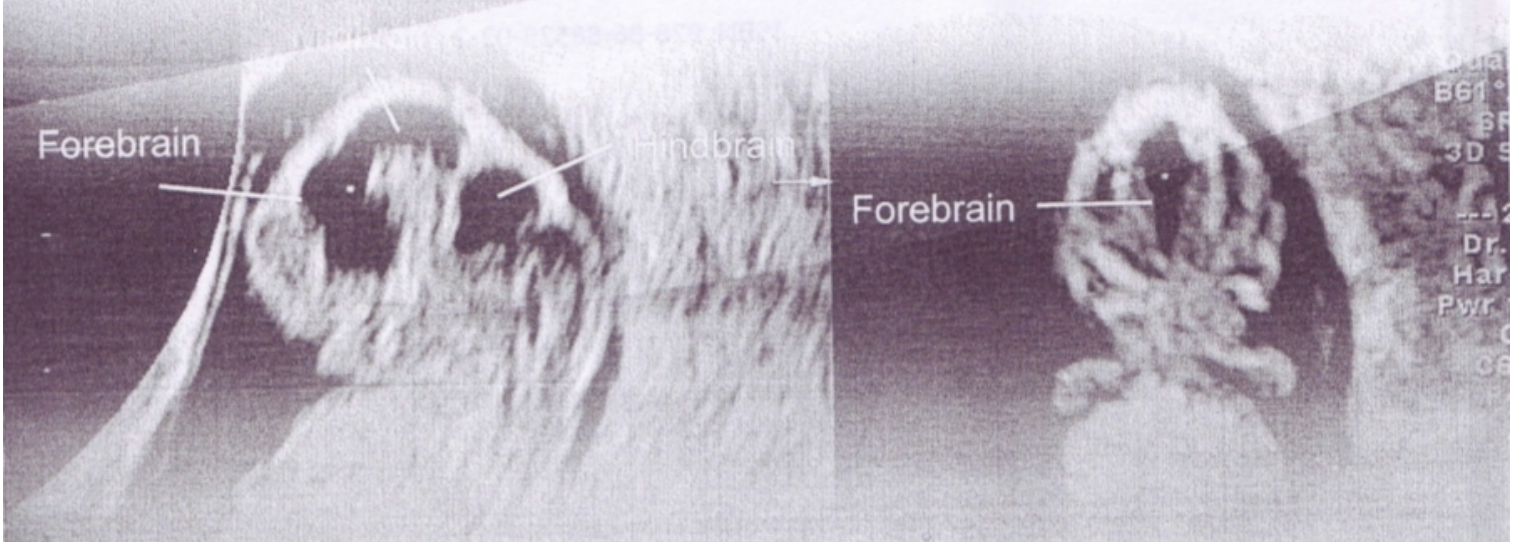
Mediterranean Association for Ultrasound in Obstetrics and Gynecology

SERBIAN ASSOCIATION OF PERINATAL MEDICINE

Ultrasound in Obstetrics and Gynecology – What is New?



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FETAL OBSTRUCTIVE UROPATHY DIAGNOSIS AND TREATMENT

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Introduction

Abnormalities of the kidneys and urinary tract are the most common abnormalities detected during routine antenatal ultrasound imaging and obstructive uropathies account for the majority of cases [1, 2].

The fetal urinary tract was first displayed by ultrasound in the late 1960s, and the first pathological ultrasound abnormality was reported in 1970.

The overall prevalence of the renal tract anomalies is estimated at 5/1000 births [3, 4]. The percentage is likely higher when considering transient anomalies. Anomalies involving the urinary tract are numerous and variable, and can be isolated or appear in association with other organ system's anomalies. Over the last decade, recommendations for postnatal evaluation of these abnormalities have been under intense scrutiny.

Antenatal renal abnormalities

There is a variety of renal abnormalities that may be detected on antenatal sonogram. Commonly found renal abnormalities are listed in Table 1, and common pathologic diagnoses in neonates are listed in Table 2.

Table 1.

Abnormalities of the kidneys and urinary tract detectable on antenatal sonogram
Hydronephrosis [unilateral or bilateral]
Hydroureter [unilateral or bilateral]
Cystic kidney
Small or dysplastic kidney
Renal agenesia

Table 2.

Common renal abnormalities detected in neonates
Ureteropelvic junction [UPJ] obstruction
Vesicoureteral reflux [VUR]
Posterior urethral valves [PUV]
Ureterovesical junction [UVJ] obstruction
Multicystic dysplastic kidney [MCDK]
Prune belly syndrome [PBS]
Duplicated collecting system with ureterocele
Solitary kidney
Urethral atresia or stricture
Autosomal recessive polycystic kidney disease [ARPKD]

Obstruction uropathy may be at different levels, at the ureteropelvic junction [upper level] or ureterovesical junction [mid-level] or urethral [lower level], lower urinary tract obstruction LUTO.

The upper and mid-urinary tract obstructions may be unilateral or bilateral with different consequences depending on the laterality. When the obstruction is bilateral or at the level of urethra, lower urinary tract obstruction [LUTO] , (bladder outflow obstruction), the prognosis is poor as a result of reduced fetal urine production and urine retention, leading to severe oligohydramnios or anyhydramnios and renal damage [5].

The most common renal abnormality is hydronephrosis [also known as renal pelvic dilatation] with an incidence estimated between 0.5 and 1% [3, 4, 5].

Various ultrasound criteria are used in order to objectively evaluate renal dilatation using, axial [transverse] scan through the spine and fetal abdomen, and longitudinal [sagittal and coronal]

view [6]. The first is being the best sight for evaluation of the renal pelvis diameter or anteroposterior diameter [APD] of the renal pelvis and the second is representing the best view for evaluation of renal length and ruling out calicectasis.

Two grading systems exist for fetal hydronephrosis:

1. Society of fetal urology grading system of congenital hydronephrosis [table 3] [Figure 1]

2. Measurement of APD of the renal pelvis in fetuses of 20 weeks gestation or greater – Grignon's criteria [table 4]

Upper urinary tract obstruction [UUTO]

1. MILD PYELECTASIS [MP]

Mild pyelectasis is a term used to describe an ultrasound finding on the fetal kidney which comprises only dilated renal pelvis below significant threshold, with no concomitant calyceal dilatation. [Figure 2], [Figure 3]

Several classification systems have been suggested for determination of the degree of pyelectasis, mostly through measurement of the AP diameter of the renal pelvis in transverse view [table 5] including AP diameter of the kidney and their mutual ratio, but most commonly used is given in table 6.

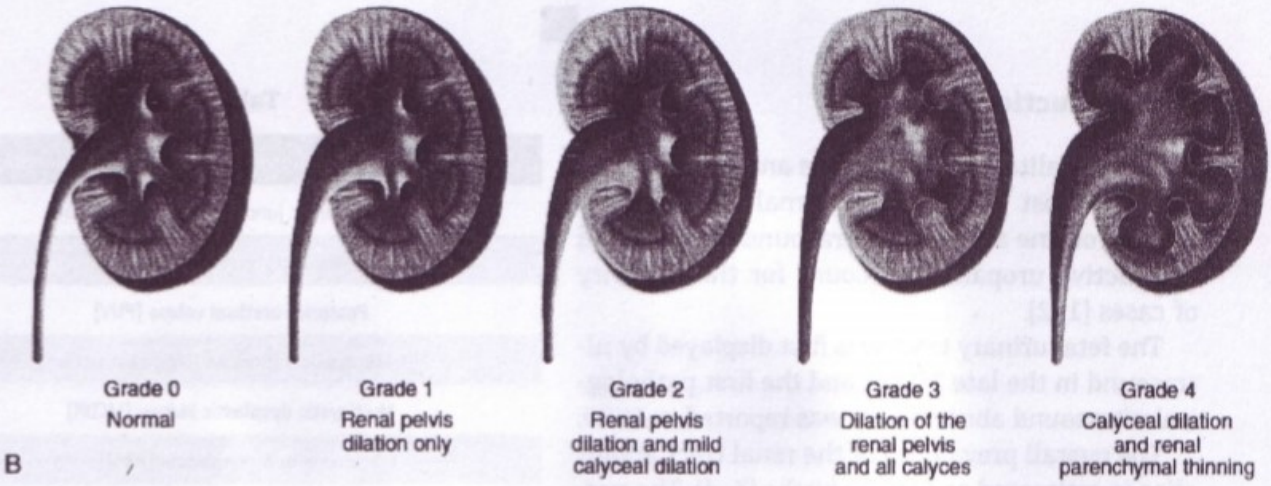


Figure 1. Ilustration demonstrating the ultrasound grading system of hydronephrosis from the Society of Fetal Urology (Ilustration by James A. Cooper, MD, San Diego. CA)

Table 3. Society of fetal urology grading system of congenital hydronephrosis

Grade	Central renal complex	Renal parenchyma thickness
0	Intact	Normal
I	Mild splitting of pelvis [dilatation]	Normal
II	Moderate splitting of pelvis and calyces, but complex, confined within renal border	Normal
III	Marked splitting, pelvis dilated outside renal borders and calyces dilated	Normal
IV	Further pelvicalyceal dilatation	Thinned

Table 4. Grignon's classification of hydronephrosis

Grade	Criteria
Gr. I [physiological]	Pelvis APD up to 10 mm
Gr. II	Pelvis APD 10 – 15 mm with normal calyces
Gr. III	Pelvis APD > 15 mm, slight calyceal dilatation
Gr. IV	Pelvis APD > 15 mm, moderate calyceal dilatation
Gr.V	Pelvis APD > 15 mm, severe calyceal dilatation with atrophic cortex

Many studies use an APD greater than or equal to 5 mm at any gestational age as abnormal [7, 8, 9], necessitating postnatal evaluation.

Table 5.

Study	Definition
Arger et al	AP diameter ³ 10 mm Ratio of AP diameter of pelvis/kidney > 0,5
Corteville, Gray,	AP diameter ³ 7 mm at < 33 weeks Ratio of AP diameter of pelvis/kidney > 0,28
Mandell et al	AP diameter ³ 5 mm at < 20 weeks AP diameter ³ 8 mm at 20 to 30 weeks AP diameter ³ 10 mm at > 30 weeks

Table 6. Renal pelvis diameter criteria for diagnosis of Mild pyelectasis

First trimester	> 3 mm
	> 4 mm
22-32 g.w	> 5 mm
After 32 g.w	> 7 mm
Any gestation	> 10 mm [pathologic finding]

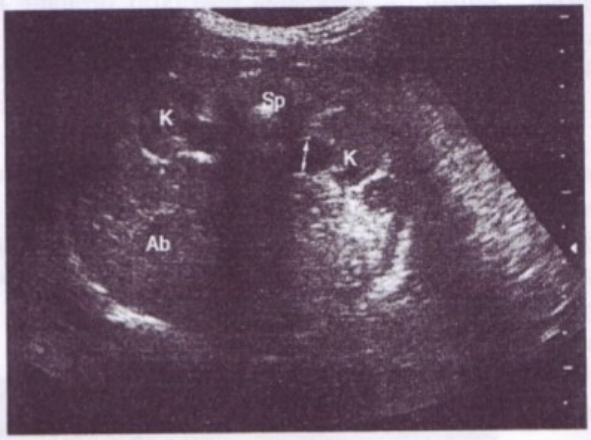


Figure 2. Transverse sonographic scan through both kidneys (k) demonstrating a midly distended renal pelvis and method of measuring the renal pelvis (arrows). Ab, fetal abdomen; Sp, spine.

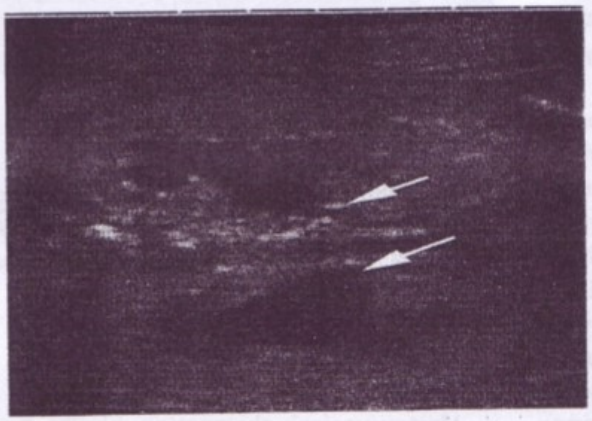


Figure 3. III degree bilateral hydronephrosis

The sonographic criteria for diagnosis of hydronephrosis in a fetus differ from those in children and adults because pyelectasis is relatively common in normal fetuses [5-8%]. But one should be alerted by the possibility of the concomitant presence of other structural anomalies. Mild pyelectasis is referred as a minor marker for anuploidy, mostly trisomy 21. Approximately 1,7% of all fetuses with MP have aneuploidy, and 0,46% of fetuses with isolated MP have aneuploidy.

Most common etiological factors for MP are:

- Mild obstruction of the uretero-pyelic junction
- Vesicoureteral reflux
- Maternal hyper hydration
- Extra renal pelvis
- Hormonal effect on the smooth muscle of the urinary tract

The likelihood of significant renal abnormality correlates with the severity of APD dilatation [7, 10, 11]. A meta-analysis of 17 studies reported the risk of renal abnormality for three classifications of antenatal hydronephrosis: mild, moderate, and severe [11]. *Mild hydronephrosis* [APD ≤7 mm in the second or ≤9 mm in the third trimester] had an 11.9% risk of postnatal abnormality. The risk of postnatal abnormality increased to 45.1% in the *moderate hydronephrosis* group [APD 7–10 mm in the second or 9–15 mm in the third trimester], and the risk further increased to 88.3% in the *severe hydronephrosis* group [APD ≥10 mm in the second or ≥15 mm in the third trimester] [12]. [Figure 4]. The probability of ureteropelvic junction [UPJ] obstruction increased with increasing APD, and there was no association of vesicoureteral reflux [VUR] with APD measurement

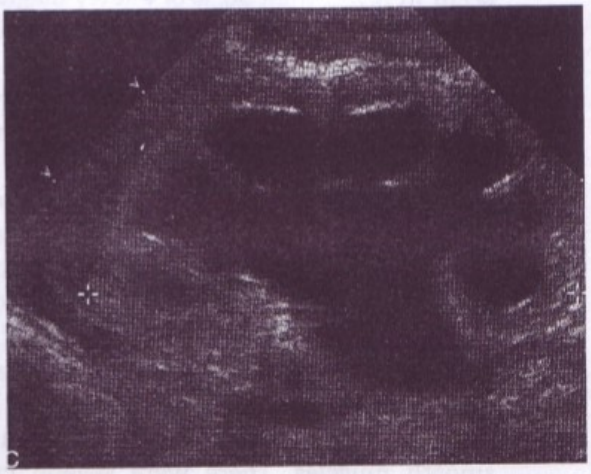


Figure 4. Severe dilatation of the renal pelvis with caliceal involvement

2. URETEROPELVIC JUNCTION OBSTRUCTION [UPJ]

Ureteropelvic junction obstruction has an incidence of 1 in 1000–1500 newborns and is the most common cause of antenatal hydronephrosis [12, 13]. Bilateral obstruction is present in 20–25% of cases, and it is three times more common in males [7].

Many potential causes have been proposed for UPJ obstruction [11]:

- intrinsic stenosis,
- insertion anomaly of the ureters,
- peripelvic fibrosis,
- peristaltic abnormalities, and
- blood vessels crossing the ureter

The diagnosis of UPJ obstruction in utero is based on the following criteria: presence of one of the described criteria for pyelectasis, there is no evidence of ureterectasis, ectopic ureterocele or bladder or posterior urethral dilation. Obviously the diagnosis of UPJ obstruction in utero is based more heavily on negative than on positive observations. Ultrasound has a good record of identifying the degree of obstruction. Quite helpful is the shape of dilated pelvis – rounded caudal end of the pelvis. [Figure 5]

When obstruction is incomplete and occurs later in pregnancy, varying degrees of pelvocalicectasis are present without histologic evidence of renal dysplasia. Incomplete obstruction earlier in gestation may result in dysplastic parenchymal changes with or without cyst formation. Multicystic dysplastic kidney is thought to result from complete obstruction at the level of the UPJ before 8 to 10 weeks.

In the praxis, the progressive hydronephrosis occur in fewer than 50 % of kidney diagnosed as having UPJ obstruction in utero. However, postnatal surgical intervention is likely in a kidney that shows progression of the dilation in utero. The progression of hydronephrosis is a very rare cause for preterm delivery.

The volume of amniotic fluid may be normal, but there may be poly- or olygohydramnios. Olygohydramnios secondary to UPJ obstruction is rare and usually a symptom of renal damage because of the secondary development of the bilateral renal dysplasia. The cause for dilation of the fetal collector system may be a high level of mother hormones that leads to dilation of the smooth muscles. There were several cases in which hydronephrosis detected in utero demonstrated no evidence of hydronephrosis in the first days of life. This phenomenon is due to the relative neonatal dehydration. Thus, the sonograms to confirm and evaluate in utero findings should be delayed at least the third and preferably the seventh postnatal day. The most severe complication is paranephritic urinoma that is result of the rupture of the collector system. In such cases, the evaluation must focus on the contralateral kidney because the associated urinoma will be functionless. Ex utero evaluation often discloses that the obstruction does not warrant surgical intervention. The most of the cases with this anomaly and AP diameter < 10 mm does not warrant surgical intervention. But the cases categorized as mild, may in the future progress to a more severe state. Thus we recommend extensive monitoring. The problem of discriminating between physiologic dilation, vesicoureteral reflux and morphological obstructions is multifaceted. These false-positive diagnoses result in paternal anxiety, numerous in utero sonographic follow-up, extensive postnatal urologic evaluation and potentially long-term follow-up.

Prophylactic coverage with systemic antibiotic was traditionally used pending the results of the workup. However most recent recommendations do not advocate routine administration of antibiotics. Instead, families should be counselled on signs and symptoms of urinary tract infections in order to allow prompt diagnosis and treatment [14].

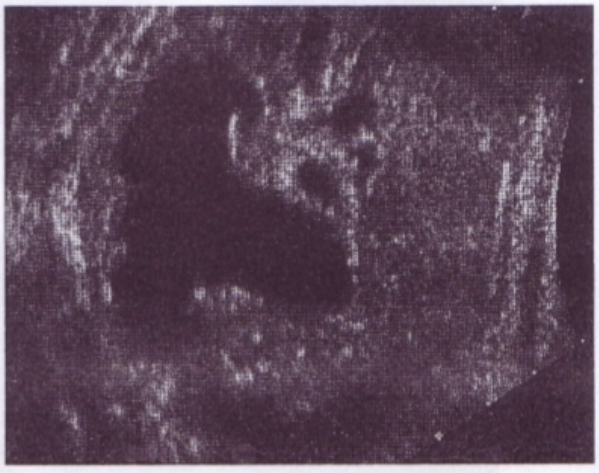


Figure 5. Moderate dilatation and eversion of calices typical for UPJ obstruction

3. URETEROVESICAL JUNCTION OBSTRUCTION [UVJ]

Ureterovesical obstruction occurs more frequently on the left and four times more frequent-

ly in males. Twenty-five percentage of cases are bilateral. Ultrasound findings include ureteral dilatation [megaureter] and dilated renal pelvis. UVJ obstruction results from an aperistaltic segment of the distal ureter, near the UVJ. Many urologists promote conservative management [15, 16], but surgical repair may be necessary. Reference to a pediatric urologist is recommended to determine surgical need which typically includes excision of the distal ureteric segment, tapering, and reimplantation of the ureter [17].

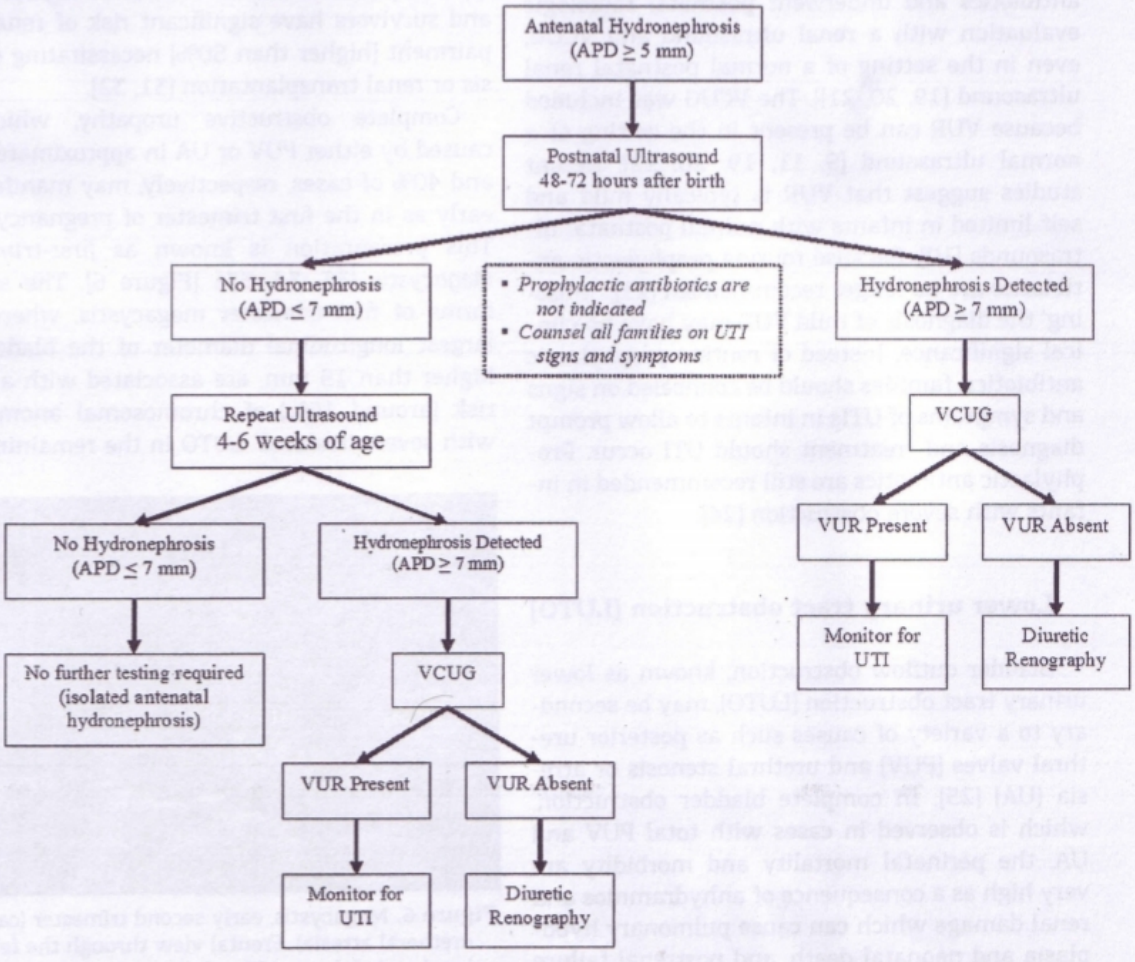
4. VESICOURETERAL REFLUX [VUR]

Vesicoureteral reflux is an upward flow of the urine out of the bladder into the ureters during voiding. VUR is a common cause of antenatal hydronephrosis. It is found in 38% of infants even in the settings of normal postnatal US. [7, 1] VUR is graded in V degrees by the International Reflux Study in Children [Table 7]

Table 7.

VUR grade	Description
Gr. I	Reflux of the urine into a nondilated ureter
Gr. II	Reflux of the urine into a nondilated renal pelvis
Gr. III	Reflux of the urine into a dilated ureter up to the renal pelvis with possible blunting of the calyceal fornices
Gr. IV	Reflux of the urine into a grossly dilated ureter with moderate blunting of the calyces
Gr. V	Reflux of the urine into a massively dilated and tortuous ureter with loss of the papillary impression

It is expected that 78-90% of cases with VUR, grades I-III, will resolve spontaneously. On the other hand, most of the cases with grade IV and V VUR will require surgical intervention, although spontaneous resolution can be seen in some of the patients. Current recommendations for treatment of VUR do not include routine pro-



Algorithm 1. Algorithm for postnatal evaluation of antenatal hydronephrosis gr I APD, anterior–posterior diameter, VCUG, voiding cystourethrogram, VUR, vesicoureteral reflux.

phylactic antibiotics. Instead, parents should be counselled on signs and symptoms of urinary tract infections [UTIs] such as fever, irritability, and foul-smelling urine.

Postnatal evaluation

The postnatal evaluation of an infant with antenatally detected renal abnormalities should always begin with a physical examination.

Radiologic evaluation of all infants with antenatal renal abnormalities begins with a postnatal renal ultrasound. Results of the postnatal sonogram will dictate subsequent evaluations. Infants with severe bilateral hydronephrosis or severe unilateral hydronephrosis in a single functioning kidney warrant immediate postnatal evaluation with a renal sonogram and voiding cystourethrogram [VCUG]. [Algorithm 1]

Traditionally, all infants with antenatal hydronephrosis [APD ≥5 mm] received prophylactic antibiotics and underwent postnatal radiologic evaluation with a renal ultrasound and VCUG, even in the setting of a normal postnatal renal ultrasound [19, 20, 21]]. The VCUG was included because VUR can be present in the setting of a normal ultrasound [9, 11, 19, 20, 22]. Recent studies suggest that VUR is typically mild and self-limited in infants with normal postnatal ultrasounds [10]. Because routine prophylactic antibiotics are no longer recommended [23], `missing' the diagnosis of mild VUR may have no clinical significance. Instead of routine prophylactic antibiotics, families should be counseled on signs and symptoms of UTIs in infants to allow prompt diagnosis and treatment should UTI occur. Prophylactic antibiotics are still recommended in infants with severe obstruction [24].

Lower urinary tract obstruction [LUTO]

Bladder outflow obstruction, known as lower urinary tract obstruction [LUTO], may be secondary to a variety of causes such as posterior urethral valves [PUV] and urethral stenosis or atresia [UA] [25]. In complete bladder obstruction, which is observed in cases with total PUV and UA, the perinatal mortality and morbidity are very high as a consequence of anhydramnios and renal damage which can cause pulmonary hypoplasia and neonatal death, and postrenal failure and renal impairment. On the basis of these facts, fetal intervention has been proposed for severe LUTO. Different therapeutic surgical methods have been performed for LUTO such as vesicocentesis, vesico-amniotic shunting, and fetal cystoscopy [26-30].

There is very little information regarding the incidence of LUTO. It is estimated to account for less than 10% of congenital uropathies diagnosed *in utero*. Despite the low incidence, LUTO has a significant impact on perinatal and child health among the congenital uropathies [30].

The natural history of LUTO is dependent on the degree of bladder outflow obstruction [complete or partial] and gestational age at presentation. These two facts are probably related since complete bladder obstruction is associated with earlier manifestations of the obstructive uropathy.

LUTO is associated with a high mortality rate which is predominantly due to severe oligohydramnios which can cause severe pulmonary hypoplasia leading to neonatal death. The perinatal mortality rate is estimated to be as high as 90%, and survivors have significant risk of renal impairment [higher than 50%] necessitating dialysis or renal transplantation [31, 32].

Complete obstructive uropathy, which is caused by either PUV or UA in approximately 60 and 40% of cases, respectively, may manifest as early as in the first trimester of pregnancy [25]. This presentation is known as *first-trimester megacystis* [33, 34, 35]. [Figure 6]. The severe forms of first-trimester megacystis, where the largest longitudinal diameter of the bladder is higher than 15 mm, are associated with a high risk [around 10%] of chromosomal anomalies, with severe 'isolated' LUTO in the remaining fe-

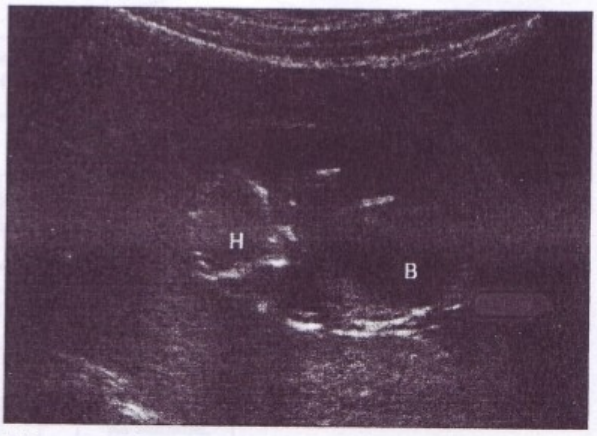


Figure 6. Megacystis, early second trimester (case of uretheral atresia). Frontal view through the fetal head and abdomen. The abdomen is filled by the distended urinary bladder (B). H. fetal head

tuses [36,37]. Pathological studies suggest that the frequency of the diagnosis of UA increases in earlier gestational age a condition that has been considered lethal until now [38].

Besides pulmonary hypoplasia due to severe oligohydramnios, experimental and clinical studies have demonstrated that urine retention may cause severe bladder and renal damage and impairment. The bladder becomes dilated with a thick wall [megacystis], and progressive bilateral hydroureter and severe hydronephrosis. This presentation is associated with renal dysplasia and impairment of renal function [39].

However, a very few fetuses may have spontaneous resolution of LUTO, which may be related to other diagnosis such as *'prune belly syndrome'* or commencement of spontaneous voiding, which may occur as a consequence of the increased pressure in the urinary bladder that overcomes the resistance of PUV [[38]

Prenatal diagnosis

LUTO can be accurately diagnosed by prenatal ultrasonography, with a high sensitivity [95%] and specificity [80%]. The characteristic ultrasound findings are a dilated bladder [DB] with a thick wall, bilateral hydroureter, and hydronephrosis, which are often associated with oligohydramnios or anyhydramnios. The dilated posterior urethra can be recognized by the characteristic 'keyhole sign' [Figure 7]. However, the definitive diagnosis of the underlying etiology causing LUTO is impossible using prenatal ultrasonography alone [40].

Prenatal ultrasonography is also essential to determine the presence or absence of associated anomalies as well as to evaluate the renal appearances which are important prognostic factors. However, it has significant limitations when severe oligohydramnios or anyhydramnios is present, and sometimes amnioinfusion is necessary to complete the assessment of fetal anatomy.

Fetal MRI, using single-shot fast-spin echo techniques, may be considered as a complementary method to prenatal ultrasonography, mainly when severe oligohydramnios/anyhydramnios is present. In addition, MRI may provide more detailed analysis of the urethral obstruction, although it has not been demonstrated to date that this method can determine the underlying etiology of the LUTO [41].

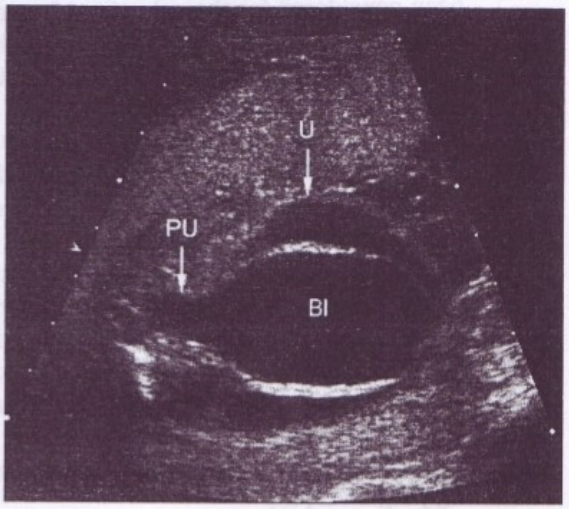


Figure 7. Dilated posterior urethra [PU], distended bladder [Bl] and characteristic 'keyhole sign'

Assessment of fetuses with obstructive uropathy for in utero therapy

The correct selection of potential subjects for fetal therapy is important to avoid unnecessary intervention in those unlikely to survive as well as complications related to the procedure in those that are likely to survive without any intervention

Today definition of, a suitable candidate for fetal intervention in LUTO is a fetus with normal karyotype without any other associated anomaly, with ultrasound findings of bladder obstruction [DB with the 'keyhole sign' and bilateral hydronephrosis], the presence of oligohydramnios or anyhydramnios and 'favorable' urine analysis. There is no consensus regarding the value of surgical intervention in a fetus with ultrasound findings of LUTO and normal amniotic fluid. These cases may have 'prune belly syndrome' particularly if the bladder is dilated and floppy rather than thick-walled and tense. However, early presentations of severe LUTO [before 18 weeks] are often associated with normal amniotic fluid volumes initially and will develop severe oligohydramnios or anyhydramnios as the pregnancy progresses. Only time and the progression of signs [oligohydramnios] in those cases will allow the definition of severe LUTO and the necessity of surgery.

'Favorable' urine analysis is considered when urinary sodium is < 100 mEq/L, chloride < 90 mEq/L, osmolarity < 200 mOsm/L and β2-microglobulin < 6 mg/L [42]. And therefore, these values have

been considered for the selection of cases for fetal therapy. However, fetal urinary biochemistry should be interpreted with extreme caution since these values cannot predict prognosis if they are measured early in gestation [before 19 weeks]. Thus fetal urinary biochemistry should not be considered in the evaluation of cases presenting before 20 weeks, particularly those with first-trimester megacystis. Furthermore, stagnate urine in the bladder may not represent the true fetal renal function, thus serial bladder sampling for repeated assessment of fresh urine is ideal but this increases the maternal and fetal risks [42,43.]. Finally, a recent meta-analysis showed low sensitivity of the urine analysis in determining prognosis [43].

Table 8. Fetal urinary markers of renal function-favorable urin parameter

Sodium	< 100 mEq/dl
Chloride	< 90 mEq/dl
Calcium	< 8 mEq/dl
β2 microglobulin	< 10/7 mg/L
Osmolality	< 210 mOsm/L
Total protein	< 20 mg/dL

Other urinary parametrer include: phosphate and N-acetyl-beta-D-glucosaminidase.

Fetal renal function status may also be assessed from fetal serum via cordocentesis. [43] Potential advantages of this approach over fetal vesicocentesis include performance of a single procedure and faster karyotype results. Beta 2-microglobulin and creatinine are both serum markers of renal function.

Fetal therapeutic options

Experimental studies have suggested that reversing the obstruction in LUTO allows the increase in amniotic fluid quantity, by improving renal function and preventing pulmonary hypoplasia Therefore, the rationale for *in utero* therapy for LUTO is to permit restoration of the amniotic fluid volume. Open fetal surgery has been almost completely abandoned because of the increased maternal and fetal risks. Minimally invasive procedures, performed under ultrasound guidance, have been proposed with the objective of decompressing the bladder obstruction and therefore improving survival rates and avoiding renal impairment. These fetal procedures should be considered as other options than termination of pregnancy for severe LUTO and should be offered in a specialized center with access to a multidisciplinary team including fetal medicine consultants, pediatric nephrologist and urologist. Therapeutic surgical methods that have been performed for LUTO are vesicocentesis, vesico-amniotic shunting, and fetal cystoscopy [26-30].

VESICOCENTESIS

The simplest procedure consists of puncturing the bladder and aspirating the urine. Many investigators have suggested that vesicocentesis in fetuses with severe megacystis in the first trimester may relieve the urethral obstruction. This approach is probably only effective in fetuses with no anatomical bladder obstruction in which the spontaneous resolution of the megacystis is highly likely to occur even in the absence of fetal intervention. Besides, in order to obtain sustained decompression, vesicocentesis should be performed several times per week, which increases significantly the risks of infection, abortion, preterm rupture of membranes and preterm labor. Therefore, vesicocentesis is considered a diagnostic method for fetal urinalysis with maternal–fetal risk almost similar to an amniocentesis.

VESICO-AMNIOTIC SHUNTING

The most common method to relieve urinary tract obstruction is to perform an ultrasound-guided percutaneous vesico-amniotic shunt [Figure 8]. The classical technique consists of placing a double pig-tailed catheter [Figure 9] under ultrasound guidance and local anesthesia, with the distal end inside the fetal bladder and the proximal part in the amniotic cavity. This procedure allows drainage of fetal urine. Before inserting the trochar and cannula, it is recommended that the entry site is examined using color-Doppler ultrasonography to prevent vascular traumas. Severe oligohydramnios and anhydramnios represent the main technical difficulty to place a catheter in severe LUTO.

For this reason, amnioinfusion immediately before placing the vesico-amniotic shunt usually becomes necessary. It is also important to place the shunt as low as possible in the bladder, in order to prevent the catheter displacement after bladder decompression.

Figure 8. Technique for vesico-amniotic

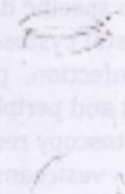


Figure 9. Double pig-tail catheter shunt shunting under ultrasound guidance

Vesico-amniotic shunting has been very widely performed for at least 25 years. There have been more than 300 cases of shunts reported in the literature. Overall, neonatal survival rates after vesico-amniotic shunting is approximately 40%, with about 50% of survivors having renal impairment [4, 26, 27, 28]

Vesico-amniotic shunting is associated with many complications. Complications related to bladder shunts occur in up to 45% of cases which include shunt blockage [25%], and shunt migration [20%] as well as urinary ascites, preterm labor, chorio-amnionitis, and iatrogenic gastroschisis. The most serious maternal complication related to vesico-amniotic shunting is infection which increases the risk for fetal death. Shunt displacement from the bladder may cause urine ascites, massive fetal abdomen distention, diaphragmatic elevation, intra-abdominal and intra-thoracic hemodynamic changes, and even fetal hydrops. With those complications, re-shunting of the bladder or even placing an abdominal-amniotic shunt may become necessary.

FETAL CYSTOSCOPY

Because of the limitations of percutaneous vesico-amniotic shunting, fetal cystoscopy was introduced by Quintero *et al.* in 1995[44, 45] The advantages of fetal cystoscopy may be to permit a more physiological drainage of the obstructed bladder and an endoscopic examination of the dilated bladder and posterior urethra allowing the direct diagnosis of the cause of the obstructive uropathy [Posterior Urethral Valve or Urethral Artesia]. A further clinical advantage over bladder shunting is the fact that there is no need for amnioinfusion prior to the procedure.

It is recommended that the procedure should be done under maternal and fetal anesthesia. [38, 46] The trochar needs to be inserted in the upper part of the fetal bladder under ultrasound guidance. If a membrane-like obstruction of the urethral lumen is seen, the diagnosis of PUV is confirmed and at this time the valves can be treated. Different methods are used to perforate the valves using hydroablation, guide-wire or laser fulguration. However, if a non-membrane-like structure is found, even with the fluid injection, the UA is diagnosed and no attempt to perforate this structure is performed. Therefore, percutaneous fetal cystoscopy is useful for diagnostic as well as therapeutic purposes in LUTO. [Figure 10]

Disruption of the valves was achieved by laser fulguration in 50% [10/20] of fetuses with a neonatal survival rate of 70% [7/10], hydroablation in 20% [4/20] of cases with a survival rate of 75% [3/4], guide-wire in 20% [4/20] with no survival, monopolar fulguration of the valves in one [5%] fetus and 'urethral probe' in another one [5%] both with no survivors. Therefore, it seems that laser fulguration and hydroablation are the best option for urethra disruption, with neonatal survival rate of approximately 70 to 75%. Further studies with larger series and perhaps with randomized comparison between fetal cystoscopy and vesico-amniotic shunting are still necessary to confirm these findings. [47]

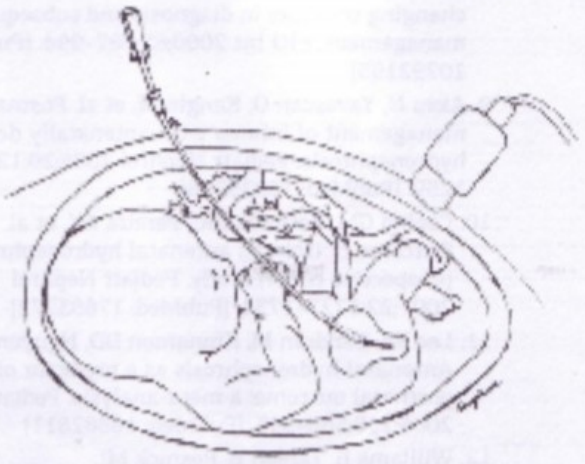


Figure 10. Technique for fetal cystoscopy

Since there are few reports in the literature, there are no specific data concerning the complications of fetal cystoscopy. Possible risks include maternal infection, premature rupture of the membranes and peripheral bladder damage.

Fetal cystoscopy requires greater skilling comparison to vesicoamniotic shunting. Further studies with long-term follow-up are still necessary to compare both surgical methods for the treatment of severe LUTO. Ideally this should be evaluated by a randomized controlled trial with stratification of cases based on the prenatal ultrasound findings, fetal urine biochemistry, and the etiological diagnosis of the obstruction.

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