

# Lower Urinary Tract Obstruction

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**Abstract:** Lower urinary tract obstruction is defined as partial or complete obstruction of the urinary tract at the level of the urethra and the bladder. The posterior urethral valves are the most common cause, 9 percent of the fetal uropathies. The incidence range is wide, because of different databases show different populations. The most severe obstructive uropathy is in patients with kidney damage, oligohydramnios and pulmonary insufficiency. If no prenatal intervention is performed, 45 percent of these patients die in the first 3 weeks of life and 25 percent have renal failure. *In utero* therapy is indicated to prevent renal damage and pulmonary hypoplasia in cases associated with progressive development of oligohydramnios. The treatment is usually limited to male fetuses with bladder outlet obstruction. The selection criteria (contraindications of fetal intervention) are: normal amniotic fluid volume, suggestion of nonobstructive dilatation of the urinary tract, sonographic evidence of renal cystic dysplasia, abnormal fetal urinary parameters, abnormal karyotype, presence of associated major congenital anomalies, fetal urinary parameters are above threshold. At the follow up after a prenatal intervention showed an apparent statistically significant improvement in perinatal survival with prenatal intervention relative to no intervention. Furthermore, there was no significant difference between the proportions of survivors with normal renal function who underwent bladder drainage and those who did not. Improved perinatal survival was also suggested in those fetuses with a poor prognosis. A nonsignificant improvement in perinatal survival for those fetuses with a good prognosis was also seen.

**Key words:** posterior urethral valves, fetal uropathy, fetal intervention, long-term outcome, prenatal diagnosis, prenatal surgery

## LOWER URINARY TRACT OBSTRUCTIONS

Fetal urinary tract obstruction is defined as partial or complete obstruction of the urinary tract from the kidney to the urethra. The definition excludes conditions that result in dilatation of the urinary tract secondary to neurologic etiologies. The site of the obstruction is usually distinguished into those that occur in the lower tract, including the urethra and the bladder (Table 1), as compared with the upper portion, which is composed of the ureter and renal pelvis.

### Incidence

The incidence of fetal obstructive uropathies is about 2 percent. Severe pathology occurs 1 in 500 fetuses. Posterior urethral valves are the most common cause, 9 percent of the fetal

**Table 1:** Possible causes of lower urinary tract obstruction

- Posterior urethral valves
- Anterior urethral valves
- Megalourethra
- Urethral duplications
- Urethral atresia
- Obstructive ureterocele
- Cloacal dysgenesis
- Prune-belly syndrome (Eagle-Barrett syndrome)
- Megacolon microcystis intestinal hypoperistalsis syndrome
- Obstructive ureteroceles

uropathies.<sup>1</sup> The incidence range is wide, because of different databases show different populations because of voluntary termination of pregnancy or miscarriage without pathologic confirmation.

### Embryology

The bladder and urethra are formed during the second and third months of gestation. During the fourth to seventh weeks of development, the cloaca, which is located at the proximal end of the allantois and is the precursor to the urinary bladder and urethra, is divided by the urorectal septum into the primitive urogenital sinus (anterior portion) and the anorectal canal (posterior portion). The primitive urogenital sinus develops into the bladder (upper portion), prostatic and membranous urethra (pelvis portion) in males, and the penile urethra (males) or urethra and vestibule (females). As the cloaca develops, the caudal portion of the mesonephric ducts is absorbed into the bladder wall. Similarly, the caudal portions of the ureters, which originate from the mesonephric ducts, enter the bladder. During these processes, the ureteral orifices move cranially and the mesonephric ducts move closer together to enter the prostatic urethra, forming the trigone of the bladder. At the end of the third month of gestation, the epithelial proliferation of the prostatic urethra forms outbuddings that constitute the prostate gland in males. In females, the cranial portion of the urethra forms buds that develop into the urethral and paraurethral glands.<sup>2</sup>

### Pathophysiology

Harrison's group was able to demonstrate that early obstruction resulted in more histological renal damage than obstruction

later in gestation.<sup>3</sup> Carr *et al* have shown, that mid-gestation is the period of greatest renal growth and development.<sup>4</sup> They suggest, that significant obstruction and hydronephrosis in early mid-gestation would have its greatest impact on renal development. Intrarenal physiological changes secondary to obstruction may alter the mechanisms of cellular proliferation, potentially irreversibly, and promote the dysplastic process. *In utero* urinary tract obstruction may cause urine retention in functioning nephrons and lead to multiple cyst formation in nephrogenic zone. Expansion of cysts with tubular dilatation disturbs the subsequent nephrogenesis by deregulated expression of growth and transcription factors, and may contribute to the maldevelopment of the fetal kidneys. Some observations imply that early obstructed urine flow results in morphological abnormalities of the developing proximal nephron rather than elements that develop into collecting ducts. Increased apoptosis of renal tubular cells as a result of the obstruction has also been implicated as a potential mechanism of renal damage. This phenomenon is critically dependent on the time at which the obstruction occurs.<sup>5,6</sup> Johnson *et al* found, that the epithelial growth factor's level in urine obtained at the time of vesicocentesis, in human fetuses was increased in cases of good prognosis fetuses with less renal damage than cases in which more advanced renal impairment and damage was present.<sup>2</sup> Pulmonary hypoplasia is a known potential complication of fetuses with obstructive uropathy. The association may result from the attendant oligohydramnios and not the result of impaired renal function *per se*. Decreased diaphragmatic excursion as a result of a large megacystis has also been implied as a potential mechanism.

## Diagnosis

The assessment of fetuses with presumed obstructive uropathy begins with a thorough ultrasound examination. Ultrasound may assist in determining the level of dilatation, the differential diagnosis, presence or absence of hydronephrosis or renal cystic dysplasia, and to rule out possible associated congenital anomalies.

The different causes of lower tract obstruction generally have similar ultrasound appearances and natural histories, they differ in their prognoses and treatments. They appear on a prenatal scan as bladder enlargement, ureteral and renal dilatation, and oligohydramnios. The presence and degree of hydronephrosis and oligohydramnios vary considerably. Obstruction can occasionally be present in cases with normal amount of amniotic fluid and without hydronephrosis.

## Sonographic Definitions and Findings

### *Hydronephrosis and Pyelectasis*

Hydronephrosis is defined as a dilatation of the renal pelvis of 10 mm or more measured in the anteroposterior diameter,

regardless of the gestational age (Fig. 1). The anteroposterior diameter increases with gestational age, but should not be more than 6 mm after 32 weeks gestation. Pyelectasis is defined as renal pelvis dilatation of > 3 mm in the second trimester and > 6 mm after 32 weeks.

### *Hydroureter*

The ureters are not normally seen on ultrasound. Hydroureters are thus defined as any dilatation of the ureters that enable them to be sonographically. Hydroureters may be markedly tortuous, giving the sonographic appearance of multiple cystic structures between the kidney and bladder.

### *Megacystis*

The diagnosis of megacystis has been mostly subjective, comparing the size of the fetal bladder relative to the fetal abdomen (Fig. 2). Normally, the bladder should not reach the level of the abdominal wall insertion of the umbilical cord. The fetal urethra is not normally seen on ultrasound.<sup>7</sup>



Fig. 1: Mild hydronephrosis

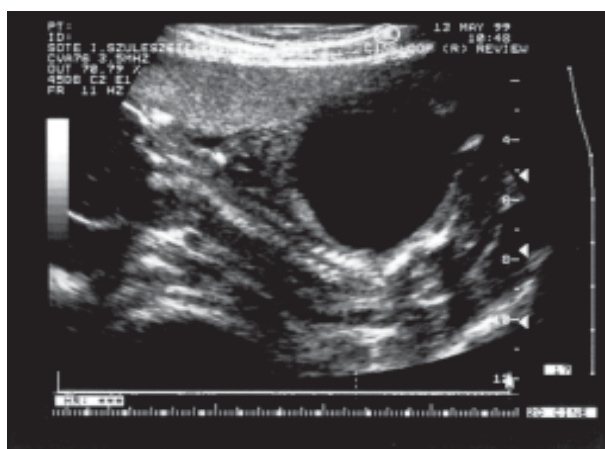


Fig. 2: Extreme megacystis (16th gestational week)

Bladder outlet obstruction may produce a so-called keyhole sign, which consists of a U-shaped dilatation of the urethra bellow the level of the bladder, in the medial plane (Figs 3 and 4). The length of the keyhole sign reflects the level of the urethral obstruction. Fetuses with posterior urethral valves or with urethral atresia may present with a keyhole sign and thus may be indistinguishable. The bladder wall hypertrophy is visible only after relief of the obstruction. Fetuses with prune-belly syndrome may present with funneled urethra, not U-shaped, different than that of posterior urethral valves or urethral atresia. The urethra will have a variable dilatation in cases of anterior urethral valves, depending on the location of the valve along the length of the urethra.

### Renal Dysplasia

Renal dysplasia can occur with both small and enlarged kidneys. The most important sonographic signs of renal dysplasia are

multiple cysts and the hyperechogenicity of the renal parenchyma. The detection of renal cysts is relative insensitive, but their presence indicates renal dysplasia. Renal echogenicity is more sensitive but also less specific. Hydronephrosis is the weakest of the renal signs in the prediction of renal dysplasia.<sup>8</sup>

### Urine Ascites

Urine can extravasate into the peritoneal cavity or the perirenal space. The mechanisms responsible for urine extravasation are often unknown. Rupture of the bladder can account for some cases, but the rest are attributed to transudation of urine into the peritoneal cavity. The degree of ascites is variable and can reach extreme proportions, leading to atrophy of the abdominal wall muscles (the prune-belly sequence). On occasion, there is no free-floating ascites, but an isolated perirenal urinoma persists.<sup>9</sup>

### Sonographic Visualisation of Fetal Micturition

In a prospective study 25 male fetuses (21 with bilateral pyelectasis, one ureterocele, three hypospadiasis) and five female fetuses (with bilateral pyelectasis) micturition was studied. A midline sagittal scan of the fetal pelvis, perineum and external genitalia was obtained and observed continuously during fetal micturition. In 19 of the 21 male fetuses and the five female fetuses with bilateral pyelectasis micturition was normal, with visualization of urinary bladder contraction, slight fluid distention of the urethra and a urinary stream from the external urethral meatus. In three male fetuses, two with bilateral pyelectasis and the one with ureterocele, the posterior urethra was normal at rest and it ballooned out during micturition, diagnostic of posterior urethral valves. The observation of fetal micturition may be of value in the diagnosis of posterior urethral valves and hypospadiasis.<sup>10</sup>

### Other Diagnostic Possibilities

#### Fetal Magnetic Resonance Imaging

Fetal magnetic resonance imaging can be used as a problem solving tool when ultrasonic findings are equivocal. It is possible with modern technology to complete a high-quality MRI examination in less than 30 minutes. The cost of MRI remains in impedance to its more widespread use, however. MRI is not believed to be hazardous to the fetus. A survey of pregnant MRI workers found no adverse fetal outcome. MRI is superior to ultrasound in examination of the fetal pelvis, which may be obscured by acoustic shadowing from pelvis bones. It helps assessing the anatomy of rare complex lower urinary tract and perineal malformations such as megacystis microcolon syndrome and cloacal extrophy.<sup>11</sup>



Fig. 3: Megacystis “keyhole” sign



Fig. 4: Megacystis (as on Fig. 3) in a male fetus

### *Fetal Cystoscopy*

The technique can be performed with submillimetric or standard diagnostic or operative fetoscopy endoscopes. It is impossible to distinguish definitively prenatal posterior urethral valves from other causes of bladder distension and oligohydramnios. Cystoscopy may help to define the underlying conditions responsible for sonographic findings and allow the introduction of treatment.<sup>12,13</sup>

### *Chromosomal Analysis*

The incidence of karyotypic abnormalities in fetuses with sonographic findings suggestive of obstructive uropathy is 8 to 23 percent. Karyotype analysis is thus part of the basic assessment of these fetuses. If megacystis and oligohydramnios are both present, successful chromosomal analysis can be achieved from fetal urine in up to 95 percent of cases and in 65 percent of fluorescent *in situ* hybridization studies. Because of the high incidence of chromosomal abnormalities, therapy should be withheld until results of the chromosome analysis become available.<sup>14</sup>

### *Fetal Renal Function Assessment*

Fetal renal function may be assessed by analysis of fetal urinary parameters through vesicocentesis. Renal damage from obstructive uropathy is associated with salt-wasting. Fetal urinary sodium decreases normally with gestational age, consistent with increased renal maturation. Several urinary parameters have been assessed as predictors of renal cystic dysplasia, including sodium, calcium, total protein, microalbumin, phosphate, N-acetyl-beta-D-glucosaminidase, osmolality, and B-2-microglobulin. Stagnant fetal urine within the fetal bladder is apt to become concentrated with time as a result of preferential loss of water through the bladder wall. As a result, old fetal urine is less likely to reflect the actual fetal renal function at the time of the vesicocentesis. If an initial vesicocentesis yields urinary values below the threshold for renal cystic dysplasia, no further vesicocentesis is necessary. However, if the values are above threshold, a second vesicocentesis within 48 hours is recommended. Some authors recommend a third vesicocentesis if the second sample still yields values above threshold.<sup>13,15</sup>

Fetal renal function status may also be assessed from fetal serum through cordocentesis. Potential advantages of this approach over fetal vesicocentesis include performance of a single procedure and faster karyotype results. Disadvantages include potential increased risk of fetal death, increased degree of technical difficulty, and decreased access.

Beta-2 macroglobulin and creatinine are both serum markers of renal function in children and adults. Beta-2 macroglobulin does not cross the placenta to a significant extent and the fetal

values remain unchanged or decrease with advancing gestation. Thus, increased serum beta-2-microglobulin values correlate with decreased renal function. Different threshold values have been proposed. A beta-2-microglobulin above 5.6 mg/L has been associated with a sensitivity of 80 percent, specificity of 98.6 percent, a positive predictive value of 88.9 percent, and a negative predictive value of 97 percent, for the prediction of renal cystic dysplasia. As unilateral renal damage is also associated with elevated beta-2-microglobulin values, cordocentesis should not be used to differentiate unilateral to bilateral renal damage.

## **Malformations Causing Lower Urinary Tract Obstruction**

### *Posterior Urethral Valves*

Posterior urethral valves, first described by Yung, constitute the most common cause of lower urinary tract obstruction in male neonates, with an incidence of one in 8000 to one in 25,000 live birth.<sup>16</sup> The lesions occur only in males because the female counterpart of the verumontanum, from which the valves originate, is the hymen. Posterior urethral valves are of heterogeneous embryologic origin. Young type I embryologically, is thought to result from high insertion of the posterior urethral folds or plicae colliculi. Other valves (Young type III) develop because of abnormal canalization of the urogenital membrane. Obstruction of the urinary flow due to the urethral valves results in compensatory hypertrophy of the detrusor. The portion of the detrusor within the bladder neck undergoes hypertrophy and may give the appearance of a bladder neck contracture in voiding cystourethrograms. The bladder neck may, in itself, obstruct the urine outflow. A persistent cloacal membrane, the most common cause of a lower urinary tract obstruction in girls, is usually a component of syndromic abnormalities for which prenatal therapy has not been shown to be beneficial.

### *Anterior Urethral Valves*

Anterior urethral valves are rare anomalies, occurring 7 to 8 times less frequently than posterior urethral valves. They probably represent an attempt at duplication of the urethra in the first 12 to 14 weeks of intrauterine life. The most common type is cusp-like and most frequently located in the bulbar urethra. Anterior urethral valves may also result from congenital urethral diverticula, secondary to incomplete formation of the ventral corpus spongiosum, an incomplete urethral duplication, or a congenital cystic dilatation of a periurethral gland.

### *Megalourethra*

Megalourethra can be described as a urethral diverticulum affecting the entire penile urethra, occurs only in boys because



of the absence of either the corpus spongiosum alone, or in combination with an absent corpora cavernosa. The penile urethra develops a cystic mass, leading to an enlarged phallus that can cause urinary obstruction (Fig. 5). Megalourethras often have been associated with other severe anomalies such as the prune belly syndrome, cloacal malformations, and vertebral, anal, cardiac, tracheal, esophageal, renal, limb association of congenital anomalies.<sup>17,18</sup>

#### *Urethral Atresia or Agenesis*

Urethral atresia or agenesis may be sonographically indistinguishable from posterior urethral valves. It is frequently associated with other genitourinary anomalies. The prognosis is uniformly fatal unless a fistulous tract develops or prenatal vesicoamniotic drainage is performed.

#### *Cloaca*

Cloaca a condition commonly associated with obstruction of both the genito- and gastrointestinal tracts, results from failure or separation of the urogenital sinus. The diagnosis may be suspected on ultrasound. Free-floating particles within the presumed bladder, which represent meconium, can be seen on ultrasound and confirmed during fetal cystoscopy.<sup>19</sup>

#### *Prune-belly Syndrome (Eagle-Barrett Syndrome)*

Prune-belly syndrome (Eagle-Barrett syndrome) is a poorly understood condition characterized by a dilated, unobstructed urinary tract; underdeveloped abdominal wall muscles, megacystis, hydroureters, hydronephrosis, and bilateral cryptorchidism. The estimated incidence is 1 in 35,000 to 50,000 live births, with more than 95 percent of the cases involving boys. It is presumed to result from mesodermal arrest possibly between the 6th and 10th weeks of gestation. Renal failure may



**Fig. 5:** Megalourethra in a male fetus

result from renal dysplasia and complications of urinary stasis. Prenatal diagnosis can be suspected by noting the presence of a large distended bladder, bilateral hydroureters, and hydronephrosis in the presence of normal amniotic fluid volume or oligohydramnios. A keyhole sign, as seen in posterior urethral valves or urethral atresia, is typically not seen on ultrasound.<sup>20</sup>

#### *Megacystis-microcolon Hypoperistalsis Syndrome*

Megacystis-microcolon hypoperistalsis syndrome was first described by Berdon in 1976. The syndrome is a familial condition of unknown etiology, autosomal-dominant or recessive, and most prevalent in females. The syndrome is characterized by microcolon, megacystis, intestinal hypoperistalsis, hydronephrosis, and dilated small bowel. The amniotic fluid is either normal or increased. Surgical pathology shows an abundance of ganglion cells in both dilated and narrow areas of the intestine. Ultrasound may be misleading in that only the urinary tract abnormalities may be apparent. Drainage of the fetal bladder may subsequently disclose dilated loops of bowel, suggestive of the syndrome. The syndrome is a universally lethal condition that afflicts female 4 times more often than male.<sup>21</sup>

#### *Obstructive Ureterocele*

An ureterocele is an obstructive dilatation of the submucosal or intravesical portion of an ureter. Ureteroceles can be either simple, involving a single ureter, or ectopic, with a duplex system. Approximately 15 percent are bilateral. Ureteroceles are 4 to 7 times more common in females. Depending on their implantation, ureteroceles may obstruct one or both ureters and the urethra. Bilateral ureteral obstruction from an obstructive ureterocele without urethral obstruction does not result in megacystis and may represent a diagnostic dilemma.

In cases of urethral obstruction, ultrasound findings are: megacystis, hydroureters, membrane-like structure within the bladder. Fetal cystoscopy allows identification of the ureterocele within the bladder. In the absence of oligohydramnios the therapy is not indicated, because this suggests that the obstruction is not complete.<sup>2,3,13</sup>

#### *In Utero Therapy*

There is no clinical consensus about fetal therapy for lower urinary tract obstruction.

The natural history of obstructive uropathy is highly variable and dependent on etiology, severity, duration and age of onset of the obstruction. The reported natural history remains widely conflicting due to the variability induced by voluntary termination, age at diagnosis, degree of oligohydramnios, obstetrical risks, multiple underlying etiologies and lack of functional measurements. In a large series all patients who met

the selection criteria and were not treated died because of pregnancy termination or pulmonary hypoplasia. It is fair to state that these newborns had a poor prognosis. Outcomes are usually measured in terms of newborn survival and are dependent on two factors: pulmonary development and renal function. Pulmonary hypoplasia is the leading cause of mortality in obstructive uropathy. An overall 45 percent mortality rate for posterior urethral valves that can be directly attributed to pulmonary insufficiency. Early midgestation oligohydramnios carries a poor prognosis for the fetus, and when associated with urethral obstruction, the mortality rate estimated as high as 95 percent.<sup>13,22,23</sup> Currently oligohydramnios is the best predictor of an adverse prognosis. Mahony *et al*<sup>24</sup> reported 100 percent survival in infants without oligohydramnios and bilateral obstruction, while Hobbins *et al*<sup>25</sup> noted 13 percent survival of fetuses with bilateral hydronephrosis and oligohydramnios. A majority of correctable fetal malformations are treated postnatally, and some patients do well and maintain enough renal function to avoid transplantation but demonstrate stunted growth and development. Also, some patients treated postnatally appear to do well initially but end-stage renal failure may develop in 25 percent. However, the most severe obstructive uropathy is in patients with kidney damage, oligohydramnios and pulmonary insufficiency. If no prenatal intervention is performed, 45 percent of these patients die in the first 3 weeks of life and 25 percent have renal failure. With prenatal intervention the reported neonatal mortality rate is 20 to 40 percent. A good example of the natural course of the obstructive uropathy the observation of Sepulveda *et al*.<sup>23</sup> In their study five twin pregnancies discordant for lower urinary tract obstruction were diagnosed between 11th and 15th weeks of gestation. There were three dichorionic and two monochorionic pregnancies. The dichorionic pregnancies were managed conservatively, resulting in a pregnancy loss of both twins in 1 case, a single fetal death at 29 weeks in 1 case, and an early neonatal death due to lung hypoplasia of an affected twin in 1 case. On the other hand, both monochorionic twin pregnancies were managed with several vesicocentesis. In both cases, the prenatal course was complicated, one by premature rupture of the membranes and the other by cord entanglement, requiring delivery at 29th and 31st weeks. Among the four continuing pregnancies with complete perinatal outcome, none of the affected twins survived, and the structurally normal twins were delivered between 29th and 36th weeks and discharged from the hospital in good condition.

*In utero* therapy is indicated to prevent renal damage and pulmonary hypoplasia in cases associated with progressive development of oligohydramnios. The treatment is usually limited to fetuses with bladder outlet obstruction with two exceptions: bilateral ureteroplevic obstruction or ureteral

obstruction secondary to an ectopic ureter without megacystis. The application of selection criteria before *in utero* therapy is essential. These are: normal amniotic fluid volume, suggestion of nonobstructive dilatation of the urinary tract, sonography evidence of renal cystic dysplasia, abnormal karyotype, presence of associated major congenital anomalies, fetal urinary parameters are above threshold.<sup>1,13,26-30</sup>

### Vesicocentesis and Vesicoamniotic Shunting

The vesicocentesis and vesicoamniotic shunting should be withheld until at least the 16th week to allow membrane fusion and avoid chorioamniotic dissection or gross ruptured membranes. Kim SK *et al* report on a fetus (it was the first report of successful vesicoamniotic shunt placement using a double-basket catheter in the first trimester of pregnancy) with posterior urethral valves treated using vesicoamniotic shunting at 13 + 5 week's gestation.<sup>30</sup> A double-basket catheter was used for shunting. A 2,582 g male neonate was delivered at 33rd week of gestation, and the infant continued to show normal renal function at 3 years of age.

Before the vesicoamniotic shunt emplacement necessary three percutaneous bladder drainages performed at 48 to 72 hours intervals, analyzing for electrolytes, osmolality and protein composition. Good prognostic group: sodium < 100 mmol/L, chloride < 90 mmol/L, calcium < 8 mg/dl, osmolality < 200 mmol/L, beta-2-microglobulin < 6 mg/L, total protein < 20 mg/dl. Borderline prognostic group: maximum of 2 values above good prognostic criteria, poor prognostic group > = 3 values above good prognostic criteria.

### Complications of Vesicoamniotic Shunting

Malfunction in 60 percent, the shunt may pull from the skin into fetal abdomen, resulting iatrogenic ascites, or out of the fetal bladder, with no further derivation of urine. The shunt may pull out of the fetus altogether as well. The malfunctioning is probably the result of the changes in the anatomic relationship of the bladder wall and fetal skin after shunting. Replacement of the shunt is associated with an additive risk of fetal death, chorioamnionitis, premature rupture of membranes, and miscarriage or preterm delivery, for a total perinatal loss of approximately 5 percent per instance.<sup>31</sup>

### Complications of Vesicocentesis

Iatrogenic vesicoperitoneal fistula has been reported with subsequent fetal urinary ascites. Premature rupture of membranes, chorioamnionitis, miscarriage, intestinal evisceration, amniorrhaxis and fetal death may also occur. The frequency of these complications is additive as repeated procedures are performed.

## Fetal Cystoscopy and Endoscopy

The technique can be performed with submillimetric or standard diagnostic or operative fetoscopy endoscopes, connected with laser ablation of posterior urethral valves.

## OUTCOME

There are growing number of case series about outcome after fetal vesicoamniotic shunt implantation. In study of Biard *et al* clinical outcomes in 20 pregnancies with a singleton male fetus, oligo/anhydramnios, and lower urinary tract obstruction were studied. Overall 1 year survival was 91 percent.<sup>22</sup> Two neonatal death occurred from pulmonary hypoplasia. Mean gestational age at delivery was 34,6 weeks, mean days from shunting to delivery were 84,4, and mean birth weight was 2,574 g. Prenatal prognosis was good in 13, borderline in 2, and poor in 3 of the survivors. Mean age at follow-up was 5,83 years. Posterior urethral valves were confirmed in 7 males, urethral atresia in 4, and prune-belly syndrome in 7. Eight children had acceptable renal function, 4 had mild insufficiency, and 6 required dialysis and eventual renal transplant. Eleven children had normal bladder function with spontaneous voiding, 6 required catheterization, and 1 child still had vesicotomy. Persistent respiratory problems were present in 8, musculoskeletal problems in 9, and frequent urinary tract infections were reported in 9. Health-related quality of life results in a group with lower urinary tract obstruction were similar to those in a healthy child population. In a study of Freedman *et al*, half of the children report ongoing respiratory concerns (asthma and frequent infections). The majority of patients with recurrent pulmonary infections had prune-belly syndrome, a predisposition previously described. Musculoskeletal abnormalities were found in 50 percent, more often in prune-belly syndrome. Children with urethral atresia had the worst renal outcomes, with 50 percent transplanted in infancy. They found, that proper assessment of the effect of prenatal intervention on postnatal renal function is still difficult. Despite advances in the use of fetal electrolytes and proteins, assessment can only estimate prenatal renal injury. Most importantly in the postnatal period, pre-existing prenatal renal injury may be exacerbated by febrile urinary tract infections, reflux, and residual bladder dysfunction.<sup>32</sup> Therefore long-term creatinine measurements or late progression to renal failure may not reflect antenatal injury or the effectiveness of prenatal intervention but may represent progressive morbidity caused by postnatal urinary tract dysfunction and infections.

In a study of Mclorie *et al* from 89 prenatally diagnosed bladder obstruction at 12 patients prenatal shunting was indicated.<sup>26</sup> Nine underwent vesicoamniotic shunt insertion between 20th and 28th weeks of gestation. None of the prenatal procedures was associated with preterm labor, chorioamnionitis or urine chemistry values greater than cut-off threshold on bladder tap. Shunts were extruded from two fetuses, which

required sequential insertion. After prenatal intervention one patient elected pregnancy termination and the others proceeded to term. Two neonates died after birth, and six survived. The diagnosis was posterior urethral valves in four newborns, urethral atresia in one, and prune-belly variant and urethral atresia in one. Of the patients three had relatively normal renal function, two had severe renal insufficiency and one had mild renal impairment. Five newborns are voiding freely and 1 patient had pulmonary problems at last follow-up. They found that the vesicoamniotic shunting was effective in reversing oligohydramnios, but its ability to achieve sustainable good renal function in infancy was variable. They did not find specific prenatal parameters to be predicting eventual good renal function, and on their opinion the pulmonary function cannot be assured with restoration of amniotic fluid. Bladder dysfunction with obstructive uropathy was common (75%). The exact mechanism, whether shunt placement prevents regular storage and evacuation, thus, inducing bladder dysfunction is not known. In a meta-analysis of Clark *et al* on lower urinary tract obstruction, 16 studies—9 case series (147 fetuses) and seven controlled series (195 fetuses) were identified.<sup>27</sup> There was apparent statistically significant improvement in perinatal survival (excluding intrauterine death and termination of pregnancy) with prenatal intervention relative to no intervention. Furthermore, there was no significant difference between the proportions of survivors with normal renal function who underwent bladder drainage and those who did not. Improved perinatal survival was also suggested in those fetuses with a poor prognosis. A nonsignificant improvement in perinatal survival for those fetuses with a good prognosis was also seen.

## CONCLUSION

Early and correct diagnosis and successful prenatal intervention is possible in cases of lower urinary tract obstructions. The prenatal intervention is done to prevent mortality and avoid disability. To achieve good results fetuses should be selected carefully, and counseling should be tailored to the gestational age and severity of the fetal anomalies. The option of pregnancy termination before 24 weeks of gestation can be considered. In cases when prenatal intervention is considered parents should be informed about possible long-term renal and pulmonary dysfunction despite successful prenatal intervention.

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