CHAPTER 100
BLADDER OUTLET OBSTRUCTION

Lukman O. Abdur–Rahman
Rowena Hitchcock

Introduction

Bladder outlet obstruction (BOO) is the impedance or blockage of urine outflow from the bladder into the urethra. This may be due to anatomical or functional causes. The anatomical causes can be intraluminal, intramural, or extrinsic. The functional causes may be neurogenic or nonneurogenic. The causes are frequently overlooked in children because they present typically with lower urinary tract symptoms that are associated with other more common forms of voiding disorders, which are often wrongly treated and delay presentation until adulthood.[1,2] Many of the underlying conditions lend themselves to medical or minimally invasive therapy, but in recent years, sophisticated techniques have become available to accurately diagnose the site and extent of obstructive uropathy.[2] These techniques are out of reach at many centres in African countries, and the diagnosis still may be missed. Subsequent treatment with a variety of empiric modalities may ultimately fail, leading to permanent damage of the urinary system, renal failure, or detrusor failure.

Demographics

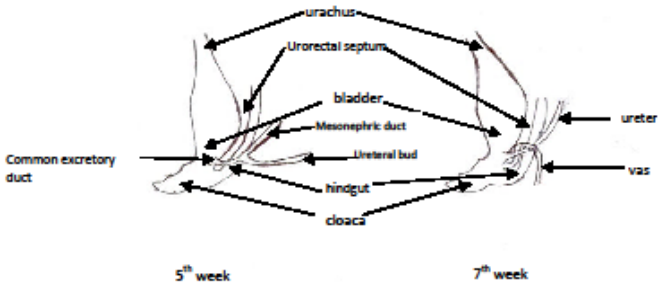
The causes of BOO are numerous and vary in incidence from one region to another. In Africa, reports on the incidence are scanty, probably due to lack of documentation. Voiding dysfunction is a general term to describe abnormalities in either the filling and/or emptying of the bladder. It is a common problem in children and constitutes up to 40% of paediatric urology clinic visits.[3] The International Children's Continence Society (ICCS) has issued standardised definitions for voiding dysfunction symptoms to facilitate classification and treatment.[4] The peculiar demography of each cause is discussed next.

Embryology of Lower Urinary Tract

The development of the lower urinary tract is closely interrelated with that of the genital tract and the hindgut.[5] By the 3rd week of gestation, the cloaca, an endodermal structure, meets the ectoderm of the body wall at the cloacal membrane, and by the 5th week, the cloaca is divided by the urorectal septum to form the primitive rectum posteriorly and urogenital sinus anteriorly (Figure 100.1). The allantois, bladder, pelvic, and phallic portions of the urogenital system are recognisable in the 6th week. By the 7th week, the mesonephric ducts (vas deferens) are shifted further caudal in the sinus and come to lie close to each other at Müller's tubercle. The metanephric buds (ureters) arise from the mesonephric ducts, and are incorporated and shift cephalad and laterally into the bladder, forming the trigone.[6]

By the 9th week of gestation, the bladder cavity expands and the urachus elongates and continues with the allantoic stalk at the umbilicus. The extra embryonic allantois degenerates, and the urachus closes by the 12th week, forming the median umbilical ligament. Closure of the urachus permits any bladder outlet obstruction to become manifest in some foetuses, but delayed closure may protect others.

The early bladder epithelium initially consists of a single cell layer, but by this point has become transitional. Meanwhile, the bladder muscle arises from mesenchyme as a longitudinal layer on the dorsal



Source: Drawing by Abdur-Rahman, LO.

Figure 100.1: Embryonic development of lower urinary tract

surface of the bladder and spreads cephalad from the bladder to the intrarenal collecting system.[7] The bladder epithelium over the ureteral orifice—Chwalla's membrane—temporarily covers and occludes the ureteral orifice, but later perforates, and the ureter becomes continuous with the bladder.[8] By the 16th week of gestation, the bladder is completely muscularized and the urachus is closed.

Agenesis of the bladder is a rare anomaly that may arise because the allantoic stalk fails to develop. Most urachal anomalies (e.g., patent urachus, urachal cyst, urachal sinus, and urachal diverticula) seem to result from delayed closure of the urachus, which may also arise from lower urinary tract obstruction at less than 12 weeks gestation. Some anomalies result from a general mesodermal failure (the urachal diverticulum of the Prune belly syndrome).[6] Intravesical (simple) ureterocoeles are thought to be due to the persistence of Chwalla's membrane beyond the time when urine flow begins.[8]

Bladder outlet obstruction caused by the urethral obstruction of posterior urethral valves or urethral hypoplasia may be detected from 4 weeks gestation concurrent with the absorption of the mesonephric duct and the resorption of the urogenital membrane. Abnormal dilatation of Cowper's glands may give rise to an obstructive urethral syringocele.

Physiology of Voiding

The control of voiding is at three levels: the central nervous system (CNS), the spinal cord, and the peripheral nervous systems from the sacral cord.

Normal voiding essentially is a spinal reflex modulated by the CNS (brain, pontine micturition centre, and spinal cord), which coordinates the functions of the bladder and urinary sphincter. The bladder and sphincter are innervated by three sets of peripheral nerves arising from the autonomic nervous system (parasympathetic (S2–S4) and sympathetic (T11–L2)) and somatic nervous system (pudendal nerve (S1–S4)).

In infants, the higher CNS network that controls voiding is not sufficiently mature to command the bladder, and control of urination in infants and young children comes from signals sent from the sacral cord. When urine fills the infant bladder, an excitatory signal is sent to the sacral cord (a spinal reflex center), which automatically triggers

the detrusor to contract involuntary, and voiding results. As the child's brain matures and develops, it gradually dominates the control of the bladder and the urinary sphincters to inhibit involuntary voiding until complete control is attained. Voluntary continence is affected by the environment, but it is usually attained by age 3–4 years.

Bladder filling is primarily a passive event determined by its intrinsic viscoelastic properties and inhibition of the parasympathetic nerves reducing detrusor tone. The sympathetic nerves facilitate urine storage by inhibiting the parasympathetic nerves, thereby causing relaxation and expansion of the detrusor muscle and closing the bladder neck by constricting the internal urethral sphincter.

As the bladder fills, the pudendal nerve becomes excited, resulting in contraction of the external urethral sphincter. The continence mechanism is achieved by contraction of the external sphincter and the internal sphincter, which maintains urethral pressure (resistance) above normal bladder pressure. The storage phase of the urinary bladder can be switched to the voiding phase either involuntarily (reflexively) or voluntarily.

Aetiology

The primary causes of BOO in children are anatomical or functional and may be congenital or acquired in both males and females (Table 100.1). The causes may be grouped further into primary urogenital tract abnormalities and secondary causes from adjacent structures, and may be neurogenic or nonneurogenic.[9]

Congenital Causes of BOO

Congenital urethral developmental anomalies may present early with features of congenital obstructive uropathy, or later with voiding dysfunction. These include urethral hypoplasia, urethral agenesis, urethral valves, syringocele, and urethral duplications. The severity of presentation depends on the gestational age at onset and the degree of obstruction. In surviving patients with severe obstruction, an accompanying vesicorectal fistula or patent urachus may be present,[10–12] as this would have been protective of the foetus.

Table 100.1: Causes of bladder outlet obstruction.

Congenital urinary anomalies	
Male	**Female**
Posterior urethral valve Anterior urethral valve Urethral diverticulum Urethral duplication Urethral atresia Posterior urethral polyp Urethral stricture Ureterocoele Cowper's gland duct cysts Prune belly syndrome Congenital urethral hypoplasia Congenital giant diverticulum of the bladder Hypertrophic utriculus masculinus	Vaginal obstruction (e.g., vaginal atresia with hydrometrocolpos, haematometrocolpos) Urogenital sinus Cloacal abnormalities Urethral atresia Urethral diverticulum Urethral duplication Ureterocoele Prune belly syndrome
Acquired urogenital tract anomalies	
Anomaly	Example
Bladder neck fibrosis	Schistosomiasis and other infections
Stones	
Tumours	Soft tissue sarcomas (rhabdomyoblastoma), squamous cell cancer of bilharziasis
Urethral stricture	Trauma and infection
	Postinstrumentation (endoscopy, catheterisation)
Marian's disease	A neural disorder of the urethral sphincters due to excessive fibrosis
Neurogenic bladder dysfunction	Associated with hypertrophy of the bladder neck
Postoperative obstruction	Ablation of posterior urethral valve (PUV) or ureterocoele, internal urethrotomy, bladder neck sling
Trauma	Haematoma, urethral transaction
Gastrointestinal tract (GIT)	Severe constipation including Hirschsprung's disease with faeculoma
	Rectal duplication
Pelvic tumours	Neuroblastomas
	Sacrococcygeal teratomas (types III, IV)
	Anterior (pelvic) meningocele/myelolipoma
	Currarino's triad (anal stenosis, sacral defect, anterior meningocele)
Some dysfunctional voiding disorders	
Neuropathic bladder with fixed sphincter resistance or detrusor sphincter dyssynergia	
Dysfunctional voiding: sphincter dyssynergia with no apparent neurological lesion (Hinman's syndrome)	

Posterior urethral valves

Posterior urethral valves occur in 0.25–0.5 per 10,000 births. In Europe, this condition may represent 10% of all urological anomalies detected by prenatal ultrasound, and the overall mortality is 25–50%. Many foetuses are lost antenatally, and renal failure is present in 45% of survivors. Foetuses with obstructive uropathy can also have other associated anomalies, such as chromosomal abnormalities (especially trisomies 13, 18, and 21), and some deformations related to the oligohydramnios. In Africa, the severity of this diagnosis and the complexity of treatment may preclude effective management in many infants.

Congenital urethral polyps

Congenital urethral polyps are a rare anomaly of the male urethra that may present with features of voiding dysfunction or obstruction. The exact incidence is unknown because many cases are asymptomatic; their diagnosis requires a high index of suspicion due to the variability of presentation.[13] Many reports favour a congenital aetiology of the polyps, though; infective, irritative, traumatic, and obstructive causes have also been proposed.[13–15]

Congenital giant diverticulum of the bladder

Congenital giant diverticulum of the bladder is a consequence of deficiency in the detrusor musculature and has been reported in infants as a rare cause of bladder outlet obstruction.[16] A giant congenital bladder diverticulum, when noted on voiding cystourethrogram (VCUG) to descend below the bladder neck, may lead to bladder outlet obstruction. Children with connective tissue disorders may be predisposed to this disorder.

Ureterocoeles

A ureterocoele is an abnormal dilatation of the terminal intravesical or extravesical portion of the ureter, most commonly associated with distally ectopic and upperpole duplex ureters. The aetiology may include:

- incomplete dissolution of Chwalla's membrane;[8]
- inadequate muscularisation;
- infection (especially schistosoma haematobium);
- trauma leading to fibrosis and subsequent stenotic ureterocoele; and
- incomplete distal ureteral obstruction by tumour or calculus, causing a pseudoureterocoele due to fibrosis.

Ureterocoeles are common in females (the male-to-female ratio is 1: 4–7), with incidence of 1 in 4,000 live births, but could be as high as 1 in 500 in autopsies.[17] Ureterocoeles are more common in caucasian than black infants. A ureterocoele is the most common cause of bladder outlet obstruction in the female, and 80% are associated with the upper pole ureter of a duplex kidney. A ureterocoele may be confused with a bladder base mass or bladder diverticulum, as it may evert on filling where the associated bladder wall is thin; early filling phase images on MCUG are therefore recommended.

Urethral duplication

Urethral duplication is a rare congenital anomaly with varied presentation, including urinary tract infection (UTI), infertility, penile deviation during erection, urinary incontinence, and abnormal urethra. The embryologic development is poorly understood, but proposed theories suggest cloaca membrane–genital tubercle and urogenital sinus anomalies.[18,19] Effmann types IIA2 and IIB are known to cause outflow obstruction due to a mucous plug in the orthotopic urethra or by proximal dilatation of the dorsal urethra that compresses the ventral urethral during micturition.[19]

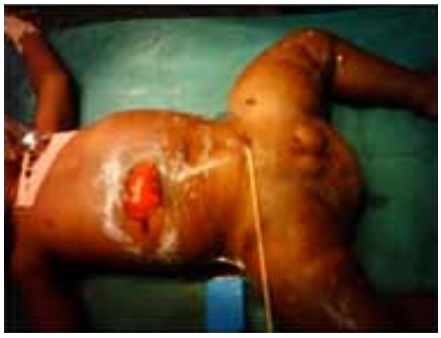
Acquired and Extrinsic Causes of BOO

Bladder stones

Bladder stones forming in the absence of underlying uropathy are termed primary or endemic bladder stones. Children account for only 2–3% of all calculous disease patients, endemic in the developing nations of Asia (Turkey, India, and Thailand) and northern Africa.[20] Until recently, bladder stones were relatively rare in the Western Hemisphere. Boys and girls are equally affected. The mean age at presentation is 6.9 years for girls and 5.2 years for boys.[21,22] Insufficient diuresis, stasis, and infection associated with malnutrition seem to be the most common causes. Bladder schistosomiasis and foreign bodies may form niduses for stone formation.[23] The foreign bodies can be either iatrogenic or noniatrogenic (e.g., suture material, shattered Foley catheter balloons, staples, ureteral stents, and regimen of clean intermittent catheterisation (CIC) of Mitrofanoff conduit).[23] Endemic paediatric bladder stones are not usually associated with renal stones and are relatively less likely to reoccur after treatment.[24] In the paediatric population, the rate of recurrence of stones ranges from 3.6% to 68%, with the highest rates in children with underlying metabolic risk factors.[25] Endemic stones are composed mainly of ammonium acid urate, calcium oxalate, or an impure mixture of ammonium acid urate and calcium oxalate with calcium phosphate. The schema proposed by Smith and Segura[26] is one of the most comprehensive and useful systems for classification.

Extrinsic bladder outlet compression

Extrinsic compression of the bladder outlet is not common but may occur in association with sacrococcygeal teratoma, anterior sacral myelolipoma/meningocele, hydrometrocolpos, faeculoma of constipation or Hirschsprung's disease, pelvic neurofibromatosis or neuroblastoma, and genitourinary rhabdomyosarcoma. These conditions present a great challenge to the surgeon[27–29] (Figures 100.2 and 100.3).



(A)

(B)

Figure 100.2: (A) Seven-month-old girl with growing type III sacrococcygeal teratoma causing BOO and constipation, which necessitated suprapubic cystostomy and transverse loop colostomy. Note the gross vulva and bilateral thigh oedema. (B) Thirteen-year-old boy with genitourinary rhabdomyosarcoma. Note suprapubic fullness and overflow urinary and faecal incontinence.

Figure 100.3: Eleven-year-old girl with sacrococygeal mass (histology: neurofibromatosis) since birth. MRI (below) shows that the bladder and the rectum are surrounded and compressed by the mass, causing BOO and constipation.

Secondary Pathologies from UTI

Urethral stricture

Urethral strictures are not common in children. When they do occur, they are usually posttraumatic or iatrogenic from urethral catheterisation and instrumentation.[30,31] The use of equipment should be carefully monitored for the size and suitability for the patient and suitable lubrication and care taken to avoid this complication. The congenital form results from abnormal junction between the proximal and distal urethra and may have a neonatal and postpubertal presentation. Recurrent infection may lead to bladder neck fibrosis, contracture, and resultant BOO. This may also occur from urinary schistosomiasis.[32]

Dysfunctional voiding

Dysfunction of the lower urinary tract in children can be secondary to derangement of nervous control, disorder of detrusor and sphincteric muscle function, structural abnormalities, and unclassified conditions.[9] Where there is loss of coordinated relaxation of the sphincter when there is detrusor contraction, a functional BOO occurs. This may be associated with congenital pathologies of the spinal cord, spinal trauma, or presacral nerve injury associated with surgery or tumour, or may be physiological in origin with no identifiable neurological deficit (nonneurogenic bladder dysfunction).[9,33,34] Nonneurogenic functional bladder disorders in children are observed in 5–15% of the paediatric population. Depending on the balance between the detrusor activity and the sphincter leak pressure, the patient may present with retention, overflow incontinence, or, in rare cases, a small, high-pressure bladder with vesicoureteric reflux and obstructive uropathy, Hinman's syndrome, or nonneurogenic neurogenic bladder dysfunction).[9,35,36] Video urodynamics is useful in the diagnosis and management of these conditions but is not available in many parts of Africa.[33,34]

Pathophysiology of BOO: Obstructive Uropathy

An obstruction of the bladder outlet (mechanical or functional) results in an elevation of intravesical pressure, which is followed by excessive force generation by the detrusor muscles against outlet resistance. This results in massive hypertrophy of the detrusor muscles with resultant sacculation, trabeculation, and ultimately diverticula formation. The trigone may be preserved, but in many instances the antireflux mechanism of the ureterovesical junction becomes incompetent, ureteral peristalsis is overcome, and increased hydrostatic pressures are transmitted directly to the nephron, causing resultant impairment of renal development and function. The ureters respond to the outlet obstruction by changes similar to the detrusor macroscopically by showing dilatation, elongation, and tortuousity.[33]

As pressures in the proximal tubule and Bowman space increase, glomerular filtration rate (GFR) falls. After 12–24 hours of complete obstruction, intratubular pressure decreases to preobstruction levels. If complete obstruction is not relieved, a depressed GFR is maintained by decreases in renal blood flow mediated by thromboxane A2 and angiotensin II. With continued obstruction, there is a progressive fall in renal blood flow, ischaemia, and nephron damage. GFR falls, but tubular function is particularly severely affected, with a high water and sodium loss resulting in the high output renal failure of obstructive nephropathy.

In the foetus, the placenta functions as the primary excretory organ in place of the kidney throughout gestation. Hence, in the newborn with BOO, the renal function is usually similar to the normal maternal levels initially because of the placental function. The kidneys commence gradual glomerular filtration by the 11th to 12th weeks of gestation. About 90% of amniotic fluid is produced in the kidneys, and only 10% comes from the GIT, lungs, and skin. In the absence of adequate foetal urine production, oligohydramnios results, restricting lung movement and decreasing fluid in the bronchial tree, and thereby causing poor acinar growth and decreased surfactant production. The newborn may have compressed limbs, Potter's facies, and pulmonary hypoplasia presenting with respiratory distress and pneumothorax.

The renal compromise affects the homeostatic, hormonal, and enzymatic functions of the kidney and may manifest as disturbances of water, electrolytes and acid-base balance, anaemia, UTI, septicaemia, circulatory collapse or hypertension, growth retardation, azotaemia, chronic acidosis, and renal osteodystrophy.

Clinical Presentation

History

Many of the cases of BOO may be asymptomatic for a long time or present with constitutional features that often are misleading to an unsuspecting practitioner. The presentation may be acute or chronic urinary retention, overflow incontinence, UTI, or renal failure. In the neonate, the presentation is characterised by abdominal distention; palpable suprapubic or flank masses (bladder and the kidneys); and urachal cyst, fistula, or abscess. Neonatal sepsis and respiratory distress

Figure 100.4: Six-week-old infant who presented with progressively reducing urine output, feed intolerance, failure to thrive, abdominal distenion, and gross lower limbs pitting oedema. Bilateral flank masses on abdominal palpation and ultrasound confirmed bilateral urinoma with hydroureteronephrosis.

may also be present. Parents may give a history of urine dribble, poor stream, and failure to thrive in the young infant.

In infants and other children, there may be enuresis, weight loss, vomiting, and diarrhoea. Constipation may be present in cases of dysfunctional voiding, faecal impaction, Hirschsprung's disease, or compression from pelvic masses. Other features include urine dribbling, incontinence, straining, frequency, and intermittency or "staccato" stream. The history should elicit prenatal health, birth and development, perinatal complication, and bowel and bladder habits.

Physical Examination

In the African setting, due to late presentation and lack of prenatal diagnosis, many children present with complications such as small stature for age, anaemia, gross pitting pedal oedema, ascites, and abdominal masses (Figure 100.4). Other features depend on primary or secondary causes. The spine should be examined for defects, and the lower extremities for reflexes, muscle mass and strength, sensation, and gait. The perineum should be checked for gluteal fold symmetry, natal cleft depth, absent coccyx, perineal sensation, tone and reflexes. The CNS should be checked for handedness as well as fine and gross motor coordination. A digital rectal examination should be done for anal sphincteric tone, faecal impaction, distended rectum, and presacral and pelvic masses.

Investigations

Before putting forward diagnostic pathways, consideration must be given to which investigative methods are available and what they can achieve. Hence, the algorithm shown in Figure 100.5 can be followed in the choice of investigative tools for diagnosis of the cause of BOO.

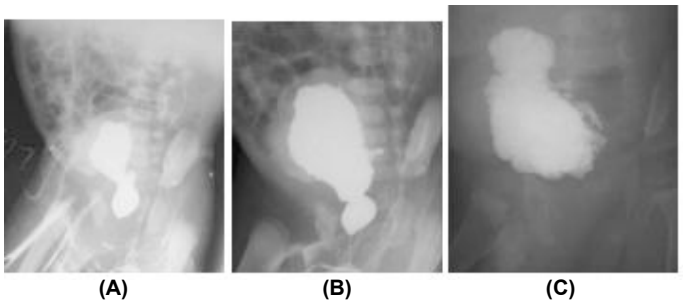


Figure 100.6: MCUG showing (A) dilated posterior urethral and bladder diverticulum around the neck; (B) multiple saculation and diverticula of the bladder wall; and (C) distorted bladder wall and giant fundal diverticulum (a multiple streak of contrast suggests extravasations).

A good bladder function diary and urinary flow rate form the basis of BOO diagnosis, but a variety of tests may be required to confirm the diagnosis and determine the extent of damage. Imaging studies such as ultrasonography (US), plain radiography, and contrast radiography such as MCUG, retrograde urethrocystogram, and intravenous urography (IVU) are relatively inexpensive and easily accessible modalities in many parts of Africa. Where available, magnetic resonance imaging (MRI), computed tomography (CT), scintigraphy, and angiography are modalities of choice. Such imaging may be sufficient to define:

- site and extent of an obstructing pathology;
- extent of urinary tract reaction to the BOO (renal pop-off mechanisms);

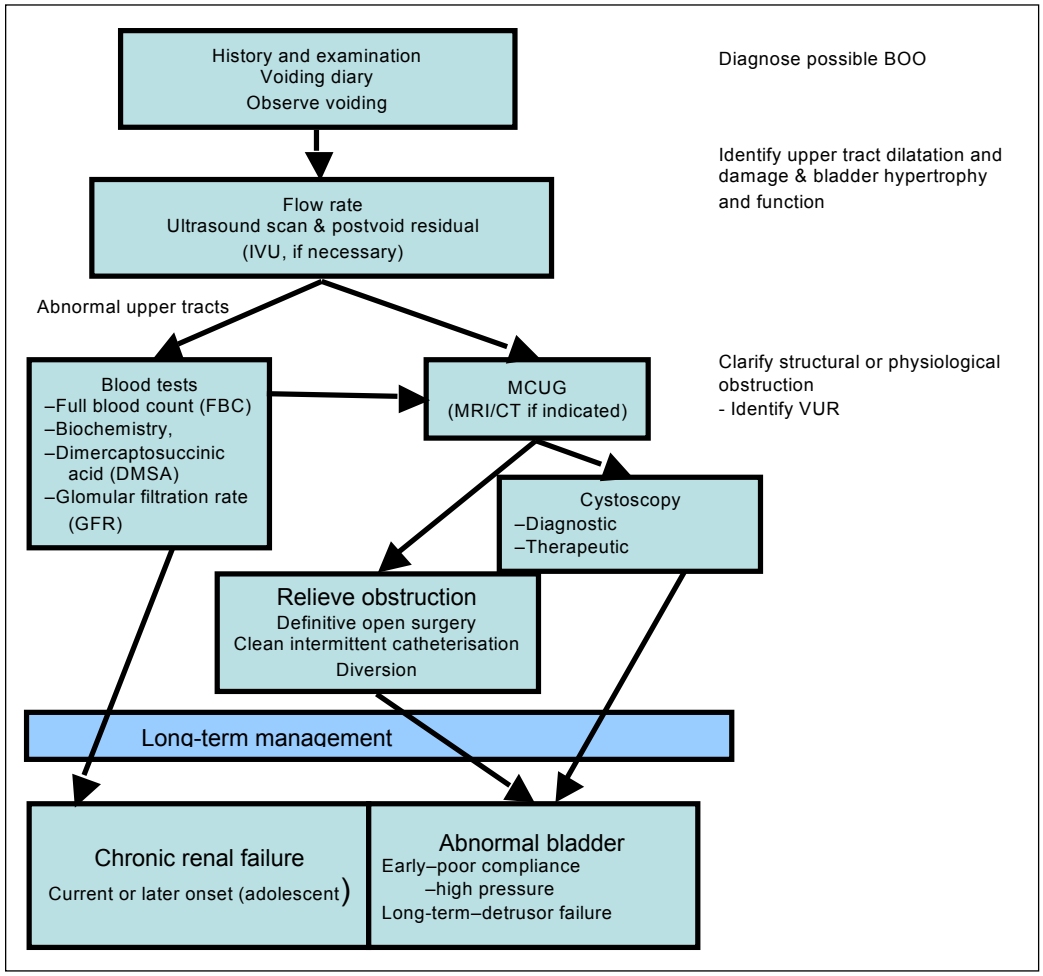


Figure 100.5: Algorithm for bladder outlet obstruction.

- split and total renal function and scarring;
- presence and degree of reflux; and
- associated anomalies, where present.

Useful Imaging Modalities

Plain radiography

Plain radiography can identify radio-opaque stones. It may suggest an enlarged or poorly emptying bladder, displacement of the bowel shadow from the hypochondrium by a large fluid-filled kidney, and/or outline associated vertebral and other bony anomalies.

Ultrasonography

Ultrasonography can demonstrate filling, dilated, or narrow posterior urethra or the presence of intraluminal lesions. US can demonstrate the size and shape of the kidneys and bladder as well as dilated pelvicaliceal system and ureter. US should be performed with a moderately filled urinary bladder, and if an anomaly is found, US should be repeated with the bladder empty. It can be used to estimate the post voidresidual volume of urine, and a spinal ultrasound in children younger than 3 months of age is useful for spinal lesions (tethering, defects, or masses)

Micturating cystourethrography

MCUG is essential for demonstration of bladder shape and capacity, including diverticulum (Figure 100.6) and vesicoureteric reflux and grading, posterior urethral dilatation, and possible filling defects and urinary incontinence. A filling phase is important to identify ureterocoele.

Intravenous urogram

An IVU study gives all the information needed about the morphology of the urinary tract, provided the kidney is working well enough. IVUs in young infants are often disappointing and sometimes dangerous. In the neonate, due to the low GFR, the urinary tract does not opacify well. The x-ray exposure for IVU is relatively high, and US scans are performed where possible.

Scintigraphy

Dimercaptosuccinic acid (DMSA) scintigraphy is a radionuclide technique that provides a functional cortical map of the kidney, quantifying renal tubular cell mass. It is particularly useful for identifying scars of reflux nephropathy and for estimating differential function, provided there is no obstruction.

In a diethylenetriamine penta-acetic acid (DTPA) scintigram, the DTPA is filtered by the glomerulus and gives a dynamic study similar to an IVU. It identifies dilatation and obstruction, and in the latter stages of the study it can give information concerning reflux. Differential renal function can also be quantified.

CT scan/MRI

CT scan and MRI with or without contrast enhancement are very precise in providing anatomical details of the lesion and the extent of damage, but they are quite expensive and not readily available in most African centres. Good-quality ultrasound with Doppler is a good substitute.

Urodynamic studies

Urodynamic studies of the physiologic function of the bladder mechanics during filling and voiding have been used to ascertain the aetiology and epidemiology of nonneurogenic bladder sphincter dysfunction (NNBSD) with the aid of x-ray screening.[35] Examples of such studies include uroflowmetry and cystometrogram.

Uroflowmetry

Uroflowmetry should precede urodynamics to determine the urine flow rate in the child. Structural BOO is associated with a low-amplitude plateau flow rate, detrusor sphincter dyssynergia may be associated with staccato voiding or nonsustained flow with detrusor failure. The careful observer can identify this just by watching.

Cystometrogram

Cystometrogram investigates the pressures during the filling and emptying phases of bladder function and may be used to measure external urethral sphincter activity. Bladder volumes and pressures allow detrusor stability and compliance, and voiding pressures may be assessed.

Cystourethroscopy

Cystourethroscopy is the approach of choice for the identification and treatment of structural abnormalities. Posterior urethral valves, syringoceles, and ureterocoeles may be definitively treated, and polyps may be biopsied or resected. In the African setting, however, appropriate scopes may not be readily available or accessible for either adults or paediatric surgical services.[37]

Ancillary Investigations

Ancillary investigations include the following:

- Haemogram to quantify anaemia, white cell counts, and differentials in UTI. The platelet count may also drop.
- Serum biochemistry to assess the electrolytes, urea, creatinine, and acid-base balance to reveal the level of renal function and allow prompt correction. Attention should be paid to the age-related, lower-than-adult creatinine levels in a child, lest significant renal impairment be missed.
- Urinalysis for blood or infection and urine specific gravity for renal concentration ability and albuminurea.
- Urine culture and antibiotic sensitivity.
- Biopsy and histology for potential bladder tumours and schistosoma granules.
- Urinary stone analysis to determine composite content and hence predict aetiology.

Management

The degree and duration of obstruction are the chief determinants of bladder dysfunction. Early recognition and treatment are the keys to preventing renal function loss. Bladder outlet obstruction should be viewed as a potentially curable form of lower urinary tract and renal disease.

Efforts should be made to probe the symptoms and signs of BOO in children.

Initial Treatment

Treatment of BOO depends on the underlying cause of the obstruction. For most cases, there is a need to control superimposed sepsis and urgently relieve the obstruction pending definitive diagnosis of the cause. The empirical choice of antibiotic includes intravenous amoxyl-clavulanic acid or an ampicillin-sulbactam combination, pending arrival of culture results. The cephalosporins and quinolones can be used as well. Aminoglycosides (e.g., gentamicin) are contraindicated in cases of compromised renal function.

A nonballoon catheter inserted through the urethra into the bladder will relieve the obstruction temporarily. The bladder may be drained by suprapubic catheter or vesicostomy or the upper tracts by ureterostomy or nephrostomy. Loop ureterostomy protects the kidneys from persistent reflux of infected urine and permits early recovery of the tone of dilated ureters while allowing the bladder to cycle normally. A postobstructive diuresis is possible following relief or bypass of obstruction, particularly chronic bladder outlet obstruction, and may lead to massive fluid depletion and electrolytes derangement.

In nonneurogenic dysfunctional voiding disorder, surgery is rarely indicated. Adoption of a conservative regimen, such as that presented below, may prevent further deterioration of this disorder.[9,36]

1. Behavioural modification and standard urotherapy, which includes good fluid intake and timed voiding schedules and double voiding,

and a nonpharmacologic and nonsurgical combination of cognitive, behavioural, and physical therapy with the aim to normalise the micturition pattern.

2. Biofeedback and pelvic floor rehabilitation to build self-perception on the detrusor contraction and pelvic floor relaxation in the patient.

3. Bowel management by use of diets, laxatives, enema, and regular elimination.

4. Clean intermittent catheterisation, which is indicated in upper tract dilatation, thickened bladder wall, and significant residue. This can be combined with medications.

5. Medications, such as anticholinergics and α-adrenergic blockers. Anticholinergics (e.g., oxybutynin, 0.2 mg/kg b.d.; propantheline, 0.5 mg/kg b.d.; and hyoscyamine) are used to reduce or abolish uninhibited bladder contracture and increase functional bladder capacity but do not overcome BOO and may make bladder empting worse. Thus, they may need to be used with CIC. α-adrenargic blockers (e.g., doxazin mesylate, prazosin, terazocin) are used to cause detrusor smooth muscle relaxation and decrease outlet resistance.

6. Botulinum A toxin injection into the urethral urinary sphincter has been used in children with NNBSD,

Surgical Treatment

The definitive treatment of mechanical causes of BOO is mostly surgical. Noninvasive and minimally invasive approaches have less morbidity compared with open surgery. Many centres in Africa still have to contend with an open approach to treat and relieve obstruction, which can be done with endoscopy, laparoscopy, or extracorporeal lithotripsy where available.

Surgical diverticulectomy, often with ureteral reimplantation, is the preferred treatment for bladder diverticulum, and this gives excellent long-term results if the underlying cause of BOO has been treated.[16]

Endoscopic incision of ureterocoeles (intravesical and ectopic) as a primary form of treatment diminishes the need for upperpole heminephroureterectomy. Open deroofing of a ureterocoele may require bladder wall reconstruction or in the presence of poor detrusor support of the ureterocoele, puncture is preferred.[17,38] Endoscopic subureteric injection of polytetrafluoroethylene (PTFE) gives good results for associated reflux after relief of BOO.

The majority of vesical calculi in children are treated by open surgery because the removal per-urethrally with the use of ultrasonic or pneumatic lithotripsy devices is restricted in paediatric patients by the narrow calibre of the urethra.[20–24] A percutaneous suprapubic cystolithotripsy as an alternative to open surgery circumvents the problem of urethral calibre in children.[39] It is safe; in addition, it reduces morbidity and hospital stay, and thus reduces the cost of treatment.

In urinary schistosomiasis, medical treatment with praziquantel is used in the acute phase, whereas in the late forms, transurethral incision can be successful in treating bladder neck contracture.

Excision of a duplicated poorly functioning urethra and or urethroplasty is successful in urethral duplication. However, individual cases should be selected by adequate investigation to avoid postoperative incontinence or urinary retention.[18,19] The more hypospadiac urethra is usually the better functioning channel.

Extrinsic compression by masses should be treated accordingly by surgical excision and/or the use of cytotoxic drugs as either adjuvant or neoadjuvant where indicated. Some rhabdomyosarcomas respond to radiation therapy, but care must be taken to avoid postradiation damage with bladder neck contracture.

Subsequent reconstruction, including bladder augmentation and urinary diversion, may be necessary and demand expert care to reduce morbidity.

Concomitant medical treatment for correction of accompanying pathophysiologic derangements, such as control of cardiovascular risk factors, diabetic nephropathy, drug dosage adjustment, and renal replacement therapy, is very important for early recovery of function and patient recovery.[40]

Postoperative Complications

- Reparative operations will fail if the underlying cause of BOO has not been treated. Urinary fistula (vesicocutaneous, perineal) requires catheterisation and/or prompt repair. Treat UTI as much as possible before surgery.
- Wound infection requires adequate dressing, maintenance of sterile procedure rules, and appropriate use of antibiotics.
- Urinary incontinence from damage to pelvic innervation during pelvic dissection, weakness of the bladder neck, or bladder neck fibrosis and contracture require careful plane dissection during surgery.
- Urethral stricture may be treated by dilatation, optical urethrotomy, or urethroplasty.
- Vesicoureteric reflux may suggest a need for reimplantation.
- Psychological disturbance and disruption in the family and social cycle demands social and religious support.

Prognosis and Outcome

Early diagnosis and prompt appropriate treatment of most causes of BOO give good results. Any untreated obstruction to the developing renal tract can lead to irreversible damage to the developing kidneys and accounts for 25% of chronic renal failure seen in childhood.[40]

Even complete early relief of a severe structural obstruction such as posterior urethral valves may not prevent the later onset of long-term detrusor failure, increasing postvoid residual volumes, UTI, and upper tract dilatation with obstructive nephropathy.

Improved nutrition, better prenatal and postnatal diagnosis and care, and improved awareness of the problem improve the prognosis.

Prevention

Parents should be educated on proper toilet training of their children and to look out for any abnormal patterns. In all cases, early referral and a team approach improve the outcome. The routine antenatal US scan should be upgraded and is the key to early diagnosis. Follow-up should be life-long because urological status may change rapidly.

Ethical Issues

In view of the poor outcome of late diagnosed BOO, a general debate on ethical grounds concerns whether prenatal diagnosis, early and repeated postnatal screening, and even treatment can be used in children to prevent the occurrence of complications. The cost of the screening, unwarranted exposure to radiation, and sometimes invasive techniques in search of the pathology in a relatively small population of children calls for caution. More specifically, the question is whether every child with UTI needs a MCUG and where does the DMSA scan fit into our evaluation? In the UK, MCUGs are now more rarely performed, and national guidelines no longer routinely call for DMSA unless there is a US scan abnormality or the child is younger than 3 months of age. The questions of how long prophylactic antibiotics should be used or whether they are useful at all are as controversial in Europe as they are in Africa.

Key Summary Points

1. A high index of suspicion is needed for early detection and prevention of inappropriate treatment.
2. Dysfunctional elimination syndrome describes the association between voiding and bowel dysfunction.
3. Progressive bladder outlet obstruction (BOO) leads to obstructive nephropathy and renal failure.
4. Urethral instrumentation in children should be under a sterile technique, choosing appropriate-size material and using gentle manoeuvres to avoid strictures.
5. Patients should have sepsis controlled and obstruction relieved while investigating the cause of the obstruction to prevent further damage.
6. Treatment may consist of behavioural modification, standard urotherapy, biofeedback, pelvic floor rehabilitation, neuromodulation, bowel management, drug therapy, and/or surgery.
7. An open surgical approach may suffice to correct anatomical BOO in many instances.
8. There should be medical treatment of concomitant obstructive nephropathy.
9. Many patients would benefit from prolonged follow-up and a team approach.

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