UROLOGIC EMERGENCIES

Keywords: Urinary obstruction, obstructive pyelonephritis, clot retention, priapism, penile fracture, Fournier’s gangrene, paraphimosis

Learning Objectives:
At the end of medical school, the medical student will be able to:
1. Describe the most frequent conditions that are considered urologic emergencies requiring immediate recognition and treatment.
2. Distinguish, through the history and physical examination, the key features of urinary obstruction, obstructive pyelonephritis, gross hematuria with clot retention, priapism, penile fracture, Fournier’s gangrene, and paraphimosis.
3. Appropriately order imaging studies and lab tests to help evaluate the patient presenting with a urologic emergency.
4. Formulate a treatment plan for the most common urologic emergencies.

Introduction:
Any physician caring for patients must be able to rapidly recognize, diagnose, and treat urologic emergencies. Failure to recognize true urologic emergencies may result in renal failure, organ damage, or loss of sexual function. Often the diagnosis is obvious and the course of treatment self-evident. Other situations may be more subtle and the optimal treatment less obvious. Given the nature of urologic problems, patients may delay treatment or may present in a very urgent fashion. Following the completion of this module, readers should be confident in their ability to diagnose and formulate a treatment plan for the most common urologic emergency conditions.

Lower Urinary Tract Obstruction:
Acute urinary retention (AUR) is the most common urologic emergency, and presents as a sudden, complete inability to void. It is typically associated with considerable suprapubic and lower abdominal discomfort and can cause significant distress for the affected individual.

AUR is thirteen times more common in men than in women, and the incidence increases with age. It has been estimated that nearly 10% of men >70 years of age and nearly 33% of men >80 years of age will develop AUR in a five-year period.

AUR is most often caused by bladder outflow obstruction, which commonly occurs in aging men due to benign prostatic hyperplasia (BPH) and accounts for the observed epidemiologic pattern of disease. Risk factors for AUR in men with BPH include severity of urinary symptoms,
increased prostate volume, decreased urine flow rate, and a PSA >2.5. Other etiologies of outflow obstruction in men include urethral stricture or other anatomical occlusion of the urethra, prostate or bladder cancer, constipation in men, phimosis and paraphimosis, or urolithiasis. In women, the most common causes of AUR are pelvic organ prolapse, or a urethral diverticulum.

Additional etiologies of AUR include neurogenic impairment and detrusor muscle insufficiency. AUR may develop immediately after general anesthesia or following acute spinal cord injury such as infarction or demyelination. It may develop acutely after gradual deterioration of the sensory or motor neurons supplying the detrusor muscle – as a result, the patient may not sense bladder distention or may be unable to efficiently contract the detrusor to express urine. AUR can also be a result of incomplete relaxation of the external urethral sphincter during voiding, a phenomenon known as dyssynergia. Anticholinergic and sympathomimetic drugs can result in neurologic dysfunction and AUR. Urinary tract infection can also result in AUR. Lastly, patients with obstructive symptoms at baseline are at risk for post-operative urinary retention after general or epidural anesthesia if a urinary catheter is not in place.

The diagnosis can be confirmed by bladder ultrasound, bladder scan, or providers can proceed directly to bladder catheterization, which is both diagnostic and therapeutic. Catheterization is usually urethral but may also be suprapubic. After bladder decompression, most patients can be managed as outpatients, unless there are indications of sepsis, malignant obstruction, acute myelopathy, or acute renal failure, in which case the patient should be admitted for additional care.

Patients who have 2,000 cc’s or more drained immediately from the bladder are at increased risk for developing post-obstructive diuresis. This is defined as >200 cc’s per hour of urine for 3 consecutive hours immediately after relief of urinary obstruction. It is more common in men with chronic urinary retention and in those with existing fluid overload. Immediate treatment usually includes monitoring and limited fluid replacement of about 75% of urinary output with IV normal saline.

**Upper Urinary Tract Obstruction:**

Effective voiding and the preservation of renal function rely on the unobstructed flow of urine from the kidneys to the bladder, and from the bladder through the urethra. Upper urinary tract obstruction or hydronephrosis may occur at any level of the ureter but is most commonly found at the ureteropelvic junction, over the iliac bifurcation or at the ureterovesicle junction. Causes of upper tract obstruction include renal or ureteral stones, congenital abnormalities, ureteral strictures, or extrinsic compression of the ureter secondary to malignancy or inflammatory conditions. Bilateral hydronephrosis is most often caused by bladder outlet obstruction and acute or chronic urinary retention, although there are a number of other causes (Table 1).

<table>
<thead>
<tr>
<th>Table 1: Causes of Bilateral Hydronephrosis</th>
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<tr>
<td>Acute urinary retention (BPH)</td>
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<tr>
<td>Cervical, Prostate or Uterine Cancer</td>
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<tr>
<td>Iatrogenic (Accidentally or deliberately tying off both ureters)</td>
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<td>Kidney stones (simultaneous, bilateral)</td>
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<td>Neurogenic bladder</td>
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<td>Posterior urethral valves</td>
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<td>Prune belly syndrome</td>
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<td>Retroperitoneal fibrosis</td>
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<td>Ureteropelvic junction obstruction (bilateral)</td>
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<td>Vesicoureteric reflux (severe, bilateral)</td>
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<td>Uterine prolapse</td>
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Upper urinary tract obstruction may lead to either acute or chronic renal failure depending upon the cause and duration of the obstruction. Renal failure results from the development of back-pressure on the kidney and ureter resulting in hydroureronephrosis and or hydronephrosis. In the acute phase of obstruction, the rise in intrarenal pressure will reduce the glomerular filtration rate and the renal plasma flow. This in turn will reduce the urinary concentrating mechanism resulting in decreased renal function. Long term obstruction may result in irreversible hypertrophy of the ureteral musculature with the associated development of fibrous bands that may cause a kink to develop in the ureter.

The muscle in the renal pelvis may also hypertrophy but then will become atonic. If left untreated the renal parenchyma will eventually atrophy.

Urinary obstruction (hydronephrosis) may be symptomatic or silent. Acute upper tract obstruction may lead to complaints of renal colic, flank pain, or pressure. Upper tract obstruction which occurs gradually over time, may be asymptomatic. On physical exam patients may have costovertebral tenderness or a palpable mass. Lab studies include CBC, electrolytes, BUN, creatinine, and a urine analysis. Appropriate imaging is critical to both the diagnosis and treatment of upper tract obstruction. If a patient has normal renal function, a CT urogram is the imaging study of choice. Many patients have an elevated creatinine which would preclude the administration of iodinated contrast material. In these cases, a non-contrast CT scan or a renal and bladder ultrasound may be necessary. The CT scan is usually preferred as it provides more information and anatomical detail if the etiology of the obstruction is unclear.

Rapid relief of the urinary obstruction is necessary to prevent the development or worsening of renal injury and to limit the progression of chronic renal failure. The options for relieving upper tract obstruction include the cystoscopic placement of a ureteral "double-J" stent or the percutaneous placement of a nephrostomy tube into the kidney. Once adequate decompression of the obstructed kidney is achieved, non urgent treatment of the cause of the obstruction may be undertaken.

**Obstructive Pyelonephritis:**

Obstructive pyelonephritis develops from a bacterial infection in an obstructed kidney. The site of obstruction can occur at any level along the ureter and may result from a stone, a tumor, a ureteral stricture, or a congenital obstruction. *E. coli* is the most common infecting organism,
cultured in over 80% of cases. Other likely causative organisms include *Klebsiella, Proteus, Enterobacter, Pseudomonas*, and *Citrobacter*.

Patients with obstructive pyelonephritis may have variable symptoms upon presentation. Many will present with classic symptoms such as renal colic, fever, chills, dysuria, and costovertebral tenderness. Some may also exhibit evidence of sepsis such as hypotension and tachycardia. If a patient is seen early in the course of the infection, the symptoms may be more subtle. A high index of suspicion is necessary, as a missed diagnosis of obstructive pyelonephritis may lead to septic shock and delayed treatment.

Basic laboratory evaluation of the patient with obstructive pyelonephritis includes a CBC with differential, BMP, urine analysis, and culture. Depending upon the severity of the infection, a lactate level, coagulation studies, and a blood gas may be necessary. Imaging of the entire GU tract is of paramount importance. A non-contrast CT scan of the abdomen and pelvis, or stone protocol CT, should delineate the location and probable cause of the obstruction. Other vital information obtained from the study may include the presence of gas in the collecting system, the presence of a renal abscess, or the development of a perinephric fluid collection. Although a renal and bladder ultrasound may be more readily available, sonography lacks detail and may miss small ureteral stones or other ureteral pathology. A plain film of the abdomen, or KUB, may be used in addition to other imaging but should not be the sole study ordered.

Acute pyelonephritis cannot be clinically differentiated from obstructive pyelonephritis; an imaging study is required for a definitive diagnosis. This is important as acute pyelonephritis can be treated medically but obstructive pyelonephritis and pyonephrosis require urgent surgical intervention and drainage. Acute pyelonephritis that fails to respond to antibiotics and all septic patients with pyelonephritis require imaging to identify those with an obstructive uropathy that require immediate drainage.

Patients with obstructive pyelonephritis will require hospital admission. Appropriate initial management includes the obtaining urine and blood cultures, administration of broad spectrum IV antibiotics, fluid resuscitation, correction of electrolyte abnormalities, and rapid decompression of the obstructed kidney. The method of decompression depends upon the stability of the patient and the cause of the obstruction. For the majority of patients who are stable enough to go to the operating room, cystoscopy with placement of a ureteral stent will provide adequate drainage. Percutaneous drainage via a nephrostomy tube is another option and is preferred when the patient’s anatomy is not conductive to easy ureteral stenting or if the patient is too unstable for the OR. Once the patient is stable and has completed their course of antibiotics, the cause of the obstruction may be addressed.

**Gross Hematuria with Clot Retention:**

Gross hematuria, or the passage of frank blood, generally prompts urgent medical attention. The causes of hematuria are myriad and may include both urological and medical etiologies. Common urologic causes of gross hematuria include renal tumors both benign and malignant, bladder tumors, prostate cancer, prostatic enlargement, renal and/or ureteral stones, trauma, and urinary tract infections. Medical causes include nephritis, anticoagulation, and various
inflammatory conditions. Although gross hematuria is startling, it is seldom a true urologic emergency unless the patient becomes anemic, hypotensive, or develops obstruction of the bladder or ureters secondary to the blood clots.

In general, the evaluation of gross hematuria starts with a thorough history and physical exam. Necessary lab studies include a CBC with differential, BUN and creatinine, coagulation studies, urine analysis and culture, and a urine cytology. Optimal imaging of the urinary tract includes a CT scan of the abdomen and pelvis without and with contrast (CT Urogram). For patients who cannot receive IV contrast, other imaging options include a renal and bladder ultrasound, or noncontrast CT scan with imaging of the upper tracts via retrograde pyelograms. For patients with a contrast allergy, an MRI urogram is an option if kidney function is still sufficient. Finally, all hematuria patients will need cystoscopy to complete the evaluation and help determine the exact etiology of the bleeding as well as to identify any serious urological conditions requiring additional therapy. Even if a renal cause for hematuria is found, a cystoscopy is still recommended as there could also be hidden pathology in the bladder that would not otherwise be diagnosed.

The acute management of gross hematuria with obstructing clots requires the placement of a large urethral Foley catheter, typically 22-24 French in size. Using a Toomey syringe and saline, the bladder may be hand irrigated to evacuate the obstructing clots. Blood clots remaining in the bladder are digested by urinary urokinase leaving fibrin fragments that act as natural anticoagulants which is why removing the blood clots are so important in controlling bleeding. For many cases of gross hematuria, this will be sufficient treatment. If ongoing bleeding occurs, continuous bladder irrigation via a 3-way catheter may be necessary. Patients may also require transfusion and / or correction of a coexisting coagulopathy along with stopping or reversing any blood thinners the patient may be taking. Finally, patients who fail the above interventions may require urgent cystoscopy in the operating room to diagnose and treat the underlying cause of the bleeding.

Priapism:

Priapism is defined by the American Urologic Association: 
https://www.auanet.org/guidelines/priapism-(2003-reviewed-and-validity-confirmed-2010) as a persistent penile erection that continues hours beyond, or unrelated to, sexual stimulation and lasting for at least 4 hours. The incidence of priapism has been estimated to be 1.5 cases per 100,000 men per year with a bimodal peak of distribution of incidents occurring in children 5 to 10 years and in adults 20 to 50 years.

The normal physiology of an erection begins with nitric oxide or neuroendocrine induced-relaxation of the smooth muscles of the cavernous arteries and tissues ultimately resulting in increased penile blood inflow. As the corpus cavernosum fills with blood, the veins that drain the corpus cavernosum are compressed resulting in maintained turgidity. While the exact underlying cause of priapism is often unknown, priapism generally occurs when there is a failure of the corpus cavernosum to drain either due to impaired relaxation or paralysis of the cavernosal smooth muscle or occlusion of the venous outflow. This failure of the corpus cavernosum to
drain can result in one of two types of priapism: ischemic or non-ischemic priapism. Ischemic priapism is the more common type. The AUA guideline on priapism: https://www.auanet.org/guidelines/priapism-(2003-reviewed-and-validity-confirmed-2010) differentiates between these two subtypes with differential management strategies employed.

**Ischemic Priapism:**
In ischemic priapism as the corpus cavernosum fills with blood, the increased intracavernosal pressure eventually begins to decrease arterial inflow. This low inflow and low outflow dynamic results in hypoxia, acidosis, and eventually penile compartment syndrome. Men with ischemic priapism present with a painful, prolonged, and fully rigid erection. In ischemic priapism, microscopic tissue edema begins to occur at four to six hours while wide structural damage of cavernous smooth muscles occurs after 12 hours. Irreversible damage is seen after 24 hours as cavernosal smooth muscles begin to demonstrate necrosis and fibroblast proliferation. Ultimately irreversible fibrosis of the corpus cavernosum is seen after 48 hours.

On physical exam, men will present with erythematous, tender, and a fully erect corpus cavernosum with a soft glans and corpus spongiosum.

There are several causes of ischemic priapism:
- Sickle cell trait or disease (most common cause in children)
- Malignant tumor (notably leukemia)
- Drugs (PDE5i (sildenafil and similar medications), intracavernous injections, alpha blockers, anticoagulants, trazodone, buproprion, cocaine)
- Neurologic shock

Ischemic priapism is a medical emergency that requires immediate treatment. Untreated ischemic priapism can result in permanent erectile dysfunction and corporal tissue damage. If ischemic priapism lasts for 12 hours, 50% of men experience permanent erectile dysfunction. If it lasts for longer than 24 hours, 90% of men experience permanent erectile dysfunction.

**Non-Ischemic Priapism:**
Non-ischemic priapism occurs far less commonly than ischemic priapism and usually is the result of a fistula between the cavernosal artery and corpus cavernosum. This dynamic results in a consistently high inflow of blood into the corpus cavernosum without reduced outflow (high inflow, high outflow). The resulting clinical picture is a partial and non-tender erection. The penis is usually much less rigid than the ischemic type. As the blood flow is maintained, the tissue damage, irreversible necrosis and fibrosis seen in ischemic priapism is not seen in the non-ischemic type.

The causes of non-ischemic priapism revolve around the creation of the fistula.
- Needle injury resulting in a hole in the cavernosal artery
- Blunt trauma
- Congenital arterial malformations
- Iatrogenic (fistula can be created during penile surgery)
Non-ischemic priapism is not an emergency and 62% of cases will resolve without treatment. Non-ischemic priapism is not generally related to permeant erectile dysfunction.

The diagnosis of priapism can often be performed after clinical evaluation and a thorough history and physical exam. The history should focus on duration of erection, severity of the pain, medications, history of hematologic disease (especially sickle cell), and any recent penile trauma. To distinguish between ischemic and non-ischemic priapism, a blood gas can be drawn from the corpora cavernosum. Dark blood with hypoxemia, hypercarbia, and acidemia is indicative of ischemic subtype while bright red blood with a normal blood gas is indicative of non-ischemic subtype. A diagnosis can also be made with penile Doppler allowing for the identification of high or low or high cavernosal arterial flow states.

First line treatment of ischemic priapism lasting greater than 4 hours is corporal aspiration where an 18 or 19-gauge needle is placed at 3 or 9 o’clock and 5 mL blood is aspirated. Diluted phenylephrine, an alpha-1 adrenergic agonist, can be injected after aspiration to reverse the smooth muscle relaxation contributing to the disorder. (Typical dilution is usually 1 cc of 10 mg phenylephrine diluted in 19 cc of normal saline. 1 cc of diluted phenylephrine is injected every 15-20 minutes and the patient’s vital signs are monitored. Typically, up to 3 injections may be used.) If the priapism does not resolve after this treatment, the creation of a surgical shunt by Urology is the next step in treatment. A fistula is deliberately made between the corpus cavernosa and the corpus spongiosum or glans with a biopsy needle, allowing for the direct venous drainage of the corpus cavernosum. In those rare cases where even this is not successful, dilation of the corpora with sounds may be the only remaining option available.

For men with non-ischemic priapism that does not resolve spontaneously, embolization of the artery containing to fistula is effective for 75% of men.

Penile fracture:

Penile fractures occur when the erect penis is forcibly bent, causing a rupture of the tunica albuginea of the corporal bodies of the penis. Penile fractures generally occur during sexual intercourse but may be self-inflicted during vigorous masturbation or may occur with other types of blunt trauma to the erect penis.

The diagnosis is based primarily on the history and physical exam. Men will generally describe a distinct feeling as a “pop” when the tunica ruptures, followed by immediate pain, and a sudden loss of the erection. This will be accompanied by the development of a significant ecchymosis involving the shaft of the penis and substantial penile swelling which has been referred to as an “eggplant deformity.” Depending upon the severity of the injury, patients may have difficulty urinating and may report gross hematuria, especially if the urethra is also torn.

Imaging plays a fairly limited role in the diagnosis and management of penile fracture. Retrograde urethrogram may be valuable in situations where patients have gross hematuria, voiding difficulty or there is a high index of suspicion that a urethral injury has occurred. Other imaging modalities such as ultrasound, MRI and cavernosography may be helpful but are not definitive in diagnosing a fracture.
The management of penile fracture, in general, is immediate surgical repair. Meta-analysis shows that immediate surgical repair is associated with significantly fewer complications when compared to conservative therapy. Surgical repair is done using either a subcoronal degloving incision or less often, an incision over the suspected site of the hematoma. The penis is completely examined to evaluate the extent of the injury. The hematoma is evacuated and the corporal bodies are repaired using either running or interrupted sutures. Repair of any concomitant urethral injuries over a Foley catheter is performed at the same time.

Possible complications attributable to penile fracture include erectile dysfunction, penile curvature, or the development of penile plaques. Urethral stricture is another possible complication.


**Fournier’s gangrene:**

Fournier’s gangrene is a life threatening, necrotizing infection of the male or female perineum. The condition is relatively rare with the estimated incidence being 1.6 cases per 100,000 males in the U.S. Fournier’s gangrene is a urologic emergency in adults. Many hospitals do not see a case of Fournier’s Gangrene in a given year. The causative event is usually a mucosal barrier breakdown in the urethra or colon. The bacterial infection is from mixed facultative and anaerobic organisms including *E. coli, Klebsiella, enterococci, Bacteroides, Fusobacterium,* and *Clostridium.* The majority of cases are seen in older men, but both men and women with diabetes mellitus are at higher risk for having Fournier’s. Other risk factors include obesity, AIDS, malignancy, alcoholism, smoking, and renal failure.

Signs and symptoms of Fournier’s are usually swift in onset and dramatic in presentation. The diagnosis is clinical and suspected Fournier’s must be treated promptly. Patients can present with both systemic and local symptoms. Local symptoms include blisters, bullae, edema, subcutaneous gas, and crepitus. Systemic symptoms include hypotension, fever, tachycardia, and shock. The infection can spread to the anterior abdominal wall, gluteal folds, and the genitals (penis, scrotum, and labia). The wounds should be cultured and sent for identification and susceptibility testing, but immediate empiric treatment is necessary.

Treatment includes broad spectrum antibiotics with emergent, extensive surgical debridement and drainage. Treatment with antibiotics alone typically has a 100% mortality. Surgical intervention may include cystostomy, colostomy, orchiectomy, and skin grafting. Surgical intervention is based on the depth and extent of the infection as well as the status of the patient. After surviving the initial infection, many patients will need multiple reconstructive procedures. Additionally, there is a role for hyperbaric oxygen to reduce the amount of debridement necessary. There is also evidence that vacuum-assisted devices may improve outcomes. Morbidity and mortality from Fournier’s gangrene is high. The mortality rate from sepsis has been reported to be between 22-40%. Morbidity is directly related to what procedures needed to
be done to stop the infection and can range from permanent colostomies and catheters to decreased fertility (as in orchietomy) and loss of sensation.

**Paraphimosis:**

Paraphimosis is an emergent urologic condition in which the foreskin of an uncircumcised or partially circumcised male becomes retracted behind the coronal sulcus of the glans penis and will not return to its normal position. When the foreskin remains retracted for an extended amount of time, venous and lymphatic outflow is obstructed and the foreskin and glans become increasingly edematous. With sufficient edema, arterial inflow to the foreskin and to the glans can become occluded. Over the course of days or even just hours, this can result in local skin necrosis, but may also result in infarction, gangrene, and autoamputation of the glans.

Individuals with phimosis are at greatest risk for developing paraphimosis. Partial phimosis is common in uncircumcised infants or young boys, in whom the epidermis of the foreskin has not entirely separated from the epidermis of the glans, and the opening of the prepuce is smaller than the circumference of the coronal sulcus. In these individuals, paraphimosis can be physiologic or can occur when a caregiver or the individual retracts the foreskin for cleaning or urination and then fails to return it to its anatomical position.

In adult and adolescent men, paraphimosis typically occurs in four settings:
1) Iatrogenic: due to a healthcare provider’s neglect to return the foreskin after performing a cystoscopy, catheterization, or other urologic procedure.
2) Anatomic: due to phimosis, secondary to inflammation/infection or to the loss of skin elasticity in aging men.
3) Traumatic: due to sexual activity or penile trauma related to genital piercings.
4) Non-hygienic: due to the individual neglecting to reduce the foreskin after urination or cleaning.

Men will present with penile pain (or in an infant, general irritability), swelling of the foreskin and glans, a constricting band of tissue at the coronal sulcus, and a flaccid shaft. Healthcare providers should rule out any constricting foreign body, including hair, clothing, rubber bands, rings, or metal piercings. Skin may be erythematous only, but if ischemia has begun to occur (a rare complication), it may be blue or black and firm.

Depending on the degree of edema, patients may also have urinary obstruction, with concomitant bladder distention, retention, and tenderness.

Urgent pain control is essential for these patients. Adolescents and adults may tolerate application of topical lidocaine or injection of local bupivacaine or lidocaine without epinephrine (epinephrine and ice are contraindicated as they can cause additional vasoconstriction and ischemia). If these are insufficient, a dorsal penile nerve block can be performed. Young boys often require intranasal or intravenous opioids as well as light sedation, as well as local anesthesia.
Once analgesia is achieved, manual reduction can be attempted. The provider should squeeze the glans, foreskin, and shaft in a closed fist for several minutes to reduce swelling before attempting reduction. If additional reduction in swelling is necessary for manual reduction, various methods can be utilized, such as: application of osmotic dehydrating agents (granulated sugar, 20% mannitol, or 50% dextrose), or elastic compression bandages to the swollen foreskin and glans. Once the swelling has diminished sufficiently, the provider should generously apply water-soluble lubricant to the glans and foreskin. Then, he or she should place the pads of his/her thumbs on each side of the urethral meatus and while placing the first and second fingers proximal to the coronal sulcus and foreskin. With moderate pressure, the provider will usually be able to gently push the glans while pulling the foreskin over the glans.

If this is unsuccessful or if any tissue necrosis appears imminent, manual reduction should be abandoned and emergency reduction of the foreskin should occur with a dorsal slit. After sterilization with an antiseptic solution, the provider should create a 1-2 cm longitudinal incision along the dorsal side of the constricting, swollen foreskin. This will allow rapid reduction in edema of the foreskin and then the glans, permitting easy reduction of the foreskin back over the glans. After the foreskin is reduced, sutures can be place in the foreskin across the incision (typically using 3-0 or 4-0 absorbable suture material).

Patients should be instructed not to retract the foreskin for at least one week and to avoid forceful retraction in the future. Cleaning should occur with water and no potentially irritating chemicals. Patients should be aware of the possibility for secondary infection, and can be instructed to apply bacitracin to the foreskin without retracting it.

Any patients with paraphimosis should have a permanent, elective dorsal slit surgery or a full circumcision to definitively prevent any further recurrence of the paraphimosis.

Given the potential severity of the condition and the frequency of iatrogenic etiology, it is important for healthcare providers to ensure that a retracted foreskin is always reduced after any patient care activities and to properly instruct patients and nurses in the proper care of the foreskin to prevent paraphimosis.

**Testicular Torsion:**

*See Testicular Torsion sub-section within “Acute Scrotum” on the AUA Medical Student Curriculum page: [http://www.auanet.org/education/educational-programs/medical-student-education/medical-student-curriculum/acute-scrotum]*

**Summary:**

- Urologic emergencies are numerous and variable in presentation
- A thorough history and physical exam combined with timely lab studies and imaging are critical to properly diagnosis and treat urologic emergencies in a timely manner.
- A high index of suspicion is necessary to avoid the long-term damage often associated with the common urologic emergencies.
Reference


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