URINARY TRACT INFECTIONS

Perspective

Background

Urinary tract infection (UTI) describes an inflammatory response of urothelium to microorganisms in the urinary tract, resulting in clinical symptoms including dysuria, frequency, urgency, hematuria, and suprapubic or costovertebral angle discomfort self-reported by the patient or elicited on physical examination. This term does not differentiate between upper and lower tract infections. Traditionally the emphasis has been on distinguishing lower tract from upper tract infections. Although this distinction may seem sensible from an anatomic perspective, it often does not provide additional information for making important management and disposition decisions. Bacteriuria is the presence of bacteria in the urine but is not considered to represent a UTI in the absence of clinical manifestations. Bacteriuria accompanied by symptoms should be treated, whereas bacteriuria in the absence of symptoms should be treated only in select patients (e.g., pregnant women, immunosuppressed patients).1

It is more useful to designate UTIs as being either uncomplicated or complicated, rather than as lower or upper tract infections. An uncomplicated infection is one involving a structurally and functionally normal urinary tract. The causative pathogen generally can be eradicated with a short course of standard antibiotics.2 This type of infection usually occurs in nonpregnant, sexually active, young women who have no evidence of an obstructive process. Complicated infection is that associated with underlying neurologic, structural, or medical problems, all of which may reduce the efficacy of standard antimicrobial therapy. These types of infections often require a prolonged course of antibiotic therapy and a more in-depth approach to testing and anatomic evaluation.

Urethritis refers to the inflammation of the urethra secondary to either an infection or trauma. Frequently, urethritis may be a manifestation of a sexually transmitted disease (STD), such as gonococcal urethritis in Neisseria gonorrhoeae infection, but may occur in other clinical scenarios as well. Cystitis generally refers to inflammation of the bladder resulting in increased urinary frequency, urgency, dysuria, and suprapubic pain. Cystitis can be separated into bacterial and nonbacterial (e.g., radiation) etiologic categories. Acute pyelonephritis is a UTI of the renal parenchyma and collecting system manifesting with the clinical syndrome of fever, chills, and flank pain. Management and disposition of patients with acute pyelonephritis depend on whether the infection is simple or complicated.

Epidemiology

UTI is a problem that affects all age groups.3,4 It is considered the most frequently occurring bacterial infection, accounting for 7 to 8 million annual outpatient visits, 1 million annual emergency department (ED) visits, 100,000 annual hospitalizations, and more than one third of all hospital-acquired infections.5 These statistics notwithstanding, gauging the actual extent of the disorder is very difficult because it is not a reportable disease in the United States and the definition of a UTI is not exact.

Women have a 50% chance of experiencing a UTI during their lifetime.2 The annual incidence of UTIs is 2 to 4% among young, sexually active women and gradually increases to 5 to 10% by the age of 70 years and to approximately 50% by the age of 80 years.

UTIs account for 5 to 14% of pediatric ED visits in the United States. UTI is more common in boys during the neonatal period but becomes more common in girls during infancy and thereafter.6 When a UTI is seen in preschool boys, it is almost always associated with congenital anomalies of the urinary tract. The overall risk for all children with unexplained fever having a UTI is 7 to 9%.7

UTIs in adult men are uncommon unless cystoscopy or catheterization has been performed. The prevalence is less than 1% from childhood through middle age but increases to 1 to 3% by the age of 65 years and to 10% by the age of 80 years. Among institutionalized men and women, prevalence rates for bacteriuria and UTI are increased to approximately 25% and 40%, respectively.8 UTIs associated with presence of an indwelling catheter constitute the most common nosocomial infection in the United States, accounting for more than 1 million cases annually.4

Principles of Disease

Physiology

The urine is sterile along the entire urinary tract from the glomerulus to the external sphincter in men and to the bladder neck in women. The urinary tract maintains its sterility by means of various defenses. A major mechanism is complete emptying. Free, unobstructed flow of urine within the kidney and down the ureter, coupled with complete evacuation of the bladder, is essential. Abnormal anatomy or physiology or the presence of a foreign body may compromise host defense mechanisms and predispose the patient to infection.5

In men the distal end of the urethra is inhabited by staphylococci, streptococci, and diphtheroid organisms. Nevertheless, men
generally do not become infected without an underlying obstruction of normal urinary flow.

In women the urethra is short and opens close to the vulvar and perirectal areas. The organisms that cause UTI in women usually arise from the fecal reservoir and initially colonize the vaginal introitus and periurethral area. These factors contribute in part to the much higher incidence of UTI in women.

Pathophysiology

Bacteria most often enter the urinary tract by ascent through the urethra and into the collecting system. Infrequently, bacterial infection of the urinary tract arises from hematoogenous or lympathic sources. This is the usual pathologic mechanism in debilitated and chronically ill patients who are immunosuppressed. Organisms from distal foci of infection such as endocarditis or soft tissue sources may make their way through lympathic or hemogematic channels, resulting in a UTI. In these patients, it is important to identify and treat the primary source of infection.

Numerous abnormalities of the urinary tract interfere with its innate ability to resist infection. Obstruction from any cause, with resultant stasis of urine, is the major causative factor. Any obstruction or impediment to the free flow of urine or complete bladder emptying results in an increased incidence of infection. Urinary calculi may cause obstruction and increased susceptibility to the development of UTI. It is crucial that obstruction in the setting of infection be relieved promptly.

Vesicoureteral reflux in children plays an important role in the pathogenesis of UTIs, particularly upper tract infections. Reflux caused by congenital abnormalities or by bladder overdistention (as seen in advanced prostatic hypertrophy) also predisposes affected patients to infection. Subgroups of patients who are more susceptible than the normal population to UTIs include diabetic patients, pregnant women, the elderly, patients with spinal cord injury and indwelling urinary catheters, patients with multiple sclerosis, and those with immunodeficiency disorders such as human immunodeficiency virus (HIV) infection.

In young men, asymptomatic bacteriuria is rare and may signify urinary tract disease. UTIs in men generally begin to appear at 50 years of age (concomitant with the onset of prostatic hypertrophy) and slowly increase in incidence. The occurrence of UTIs in men of any age warrants referral to a urologist for further evaluation.

Bacteriology

The organisms that cause UTIs generally come from enteric flora colonizing the perineum and urethra. *Escherichia coli* is the dominant pathogen in more than 80% of first infections in women, men, and children, as well as in 50% of nosocomial UTIs. *Staphylococcus saprophyticus*, a coagulate-negative gram-positive organism, is the second most common pathogen in UTI and accounts for approximately 11% of cases. This species is present in normal skin flora, including the perineal area, but only in low numbers, and it does not appear to be of fecal origin. Occasionally, it is falsely identified as *Staphylococcus albus* or *Staphylococcus epidermidis*. Other, less common bacteria that may be responsible for infection include *Proteus, Klebsiella*, and *Enterobacter*. Unusual microorganisms may be found in institutionalized or hospitalized populations and with complicated UTIs. Such settings and conditions predispose the patient to alterations in the normal gastroinestinal (GI) flora, leading to complex UTIs. The uropathogens in these patients include more resistant strains of *Escherichia*, *Klebsiella*, *Proteus*, and *Enterobacter*, as well as *Pseudomonas*, *Enterococcus*, *Staphylococcus*, *Providencia*, *Serratia*, *Morganella*, *Citrobacter*, *Salmonella*, *Shigella*, *Haemophilus*, *Mycobacterium tuberculosis*, and fungi. Although not necessary with uncomplicated UTIs, urine culture and sensitivity testing are recommended for all patients with complicated UTIs. Antibiotic therapy based on the most likely pathogen should be initiated immediately.

Uropathogenic organisms may elaborate various factors that affect their virulence, including aerobactin, hemolysins, and fimbralae (pili). Fimbrialae, also called *adhesions*, are proteinaceous structures that can attach to specialized receptor sites on host cells. Attachment of bacteria to vaginal and uroepithelial cells ultimately leads to a higher incidence of UTI.

Clinical Features

Signs and Symptoms

UTI is suspected in adolescents and adults on the basis of clinical findings, including dysuria, frequency, urgency, and hematuria in the setting of suprapubic or costovertebral angle (CVA) discomfort. Symptoms and signs vary with age. In infants, initial manifestations may include irritability, fever, vomiting, diarrhea, and failure to thrive. Preschool children with UTI have vomiting, diarrhea, generalized abdominal pain, and febrile seizures. History of fever alone is not an adequate indicator of severity of infection in children because it may be historically absent in pediatric patients subsequently found to have significant renal scarring.

In general, clinical signs and symptoms associated with lower UTIs are localized to the genitourinary system and include urgency, dysuria, frequency, and suprapubic pain. In addition to these problems, a patient with an upper UTI may have back and flank pain as well as constitutional symptoms and signs such as fever, nausea, vomiting, and malaise.

As discussed previously, it is more important to determine if the infection is simple or complex. Simple, uncomplicated infections do not require urine culture, may be treated on an outpatient basis, and may not always necessitate urinalysis when thought to represent isolated cystitis. Complicated infections require urine culture with antibiotic sensitivity and also may necessitate inpatient therapy with further diagnostic evaluation. Distinction between upper and lower tract infections becomes important for understanding the differences in pathology and the pharmacokinetics of antibiotic delivery. Infection of the bladder generally involves only the superficial mucosa, and high urinary concentrations of antibiotics can easily be achieved with a short course of antibiotic therapy. The kidney, by contrast, tends to become infected in the medullary tissue, where achieving therapeutic concentrations of antimicrobial agents is far more difficult. As a result, parenteral antibiotics and longer courses of therapy are needed.

Men who report dysuria must be evaluated for the presence of urethral discharge before urinalysis is performed. In these patients, in whom the diagnosis of UTI is rare, the most likely causative disorder is an STD such as gonococcal or nongonococcal urethritis. If the patient has purulent urethral discharge, he should undergo testing for *Chlamydia trachomatis*, *N. gonorrhoeae*, and syphilis, as well as empirical treatment for STDs. If the patient does not have urethral discharge and complains predominantly of dysuria, frequency, urgency, and suprapubic or CVA discomfort, a urinalysis with culture should be performed. Male patients who have bacteriuria in the absence of clinical signs of urethritis or prostatitis should be considered to have a complicated UTI and should be treated with antibiotic therapy and undergo urologic follow-up for further evaluation.

Diagnostic Strategies

Laboratory Tests

Urine Collection Methods. The diagnostic value of microscopic examination depends on the quality of the specimen obtained. In neonates and children younger than 6 months, urethral
catheterization is the preferred method of urine collection. Aseptic technique ensures a low risk of introducing bacteria. Before catheterization is attempted in these younger patients, an ultrasound or bladder scanner can be used to confirm the presence of urine in the bladder. Suprapubic bladder aspiration is a reliable method if urine is available to be withdrawn, but it is less commonly used owing to the invasiveness of the procedure. For patients 12 months or younger, ultrasound-guided suprapubic bladder aspiration is a useful method and carries an incidence of adverse effects similar to that of urethral catheterization. Plastic bag collection is a reasonably reliable method, but the perineum (in girls) and the glans (in boys) should be properly cleansed before application of the bag. It is the least invasive method but is useful only if culture results are negative, because of high associated contamination rates. A clean-catch urine specimen is preferred in cooperative and continent male patients.

In older children, a sterile midstream urine sample can be collected from boys. In girls, if the voided specimen is free of cellular elements (epithelial cells), it is acceptable for analysis. Catheterization often is a traumatic experience for children and one that invades their sense of privacy. An alternative approach to catheterization in a young girl with classic symptoms of UTI who has epithelial cells in the urine suggesting contamination is to evaluate the urine for other signs of infection. If the urine contains bacteria, red blood cells (RBCs), and white blood cells (WBCs) and is either nitrite or leukocyte esterase positive, the most likely cause of the symptoms is an uncomplicated UTI, for which antibiotic therapy and referral for follow-up care with the child’s pediatrician are indicated. If the clinical picture remains unclear and a definitive diagnosis is mandatory, straight catheterization is performed after examination of the child’s bladder with an ED ultrasound machine has confirmed the presence of urine.

Recommendations regarding urine collection methods in women vary widely. Midstream-voided specimens rarely escape perineal contamination, because for most adult women, adequate preparatory self-cleansing of the perineal area is difficult to achieve. It has been shown that in up to 50% of women with sterile bladder urine, a midstream clean-catch specimen grows 1000 to 100,000 bacterial colony-forming units (CFUs) per milliliter. This finding assumes major significance in the ED evaluation, in which accurate, initial supportive evidence for the diagnosis of UTI is crucial.

Sterile catheterization is the quickest and most accurate method of obtaining a urine specimen from an adult woman and may be the best solution for achieving a reliable urinalysis if the patient is actively menstruating. It is safe and relatively atraumatic and carries a remarkably low risk of infection. This risk increases if the patient is pregnant, elderly, or debilitated. If the clinician decides against catheterization, a clean-catch, midstream urine specimen should be sought. A predominance of epithelial cells suggests that the specimen is contaminated. The lower the ratio of leukocytes to vaginal epithelial cells, the more likely it is that the leukocytes are vaginal contaminants.

In men the specimen is not affected significantly by lack of cleansing or by the timing of specimen collection. Therefore it is not appropriate to catheterize an adolescent or adult man simply for the purpose of collecting a urine specimen.

Urinalysis. Urine cultures constitute a majority of cultures performed by microbiology laboratories, and various screening tests have been developed for the purpose of reducing this burden and its attendant costs. The goal of urine screening tests is reliable selection of specimens that will provide negative cultures. This allows the laboratory to focus more appropriately on higher-yield studies.

The most commonly used screening tests measure urinary leukocyte esterase and nitrite. Leukocyte esterase is an enzyme found in neutrophils, and nitrite is produced from urinary nitrate by nitrate reductase, which is present in gram-negative bacteria. Both can be detected by a color change on dipstick testing. The two tests often are combined to improve overall accuracy. Indirect urine dipstick tests for pyuria or bacteriuria are inexpensive and easy to perform and may aid in establishing the diagnosis of UTI. They should be used with caution, however, because they can be less sensitive than microscopic examination of urine (urinalysis). Urine dipstick testing for leukocyte esterase has shown a sensitivity of 75 to 96% in detecting pyuria associated with UTI. By contrast, a meta-analysis of screening tests for UTI in children has demonstrated that dipstick testing for leukocyte esterase and nitrite may be equivalent to microscopic urinalysis for detection of UTI.

Symptomatic patients who have normal host defenses and demonstrate a positive result on leukocyte esterase testing (in the absence of other indications for urine culture) can be treated empirically without culture. In symptomatic patients, a negative result on tests for both leukocyte esterase and nitrite should be followed by urine microscopy. In adults, urine culture should be performed only if findings on the microscopic analysis also are negative or if the patient is at risk for bacteremia.

Urine Microscopy. Urine microscopy is another commonly used method of providing rapid results, thereby reducing the number of urine cultures performed. Up to 96% of infected urine specimens contain 10 or more WBCs per cubic millimeter when counted by a hemocytometer. Various counting chamber methods detect pyuria with an accuracy approaching that of the hemocytometer. Unfortunately, these tests are not widely available, so direct microscopy commonly is used.

The accuracy of direct microscopy is compromised by a lack of standardization of the technique. Common sources of variability include specimen collection and transport, centrifugation speed and duration, decanting and resuspension techniques, staining, and the threshold used for significant numbers of WBCs or bacteria. One method, the slide centrifuge test, avoids many of these sources of error, and high sensitivity and specificity have been reported.

Although no accepted level of pyuria is diagnostic of UTI, on careful quantitation with a hemocytometer chamber, pyuria will be found in nearly all cases of acute UTI caused by coliforms. In patients with a low-count coliform infection, those with fewer than 8 WBCs/mm$^3$ of urine will have no demonstrable infection. In patients with more than 8 WBCs/mm$^3$, 85% will have documented infection (by the presence of coliforms, staphylococci, or chlamydiae). Despite these controversies and limitations, microscopic examination of urine to identify bacteria remains the most readily available and reliable test for a presumptive diagnosis of UTI in most patient populations. Any analysis of a urine sample must be performed immediately after collection. Urine specimens that are allowed to sit for longer than 2 hours become alkaline, with subsequent dissolution of the cellular elements and multiplication of bacteria, thus providing the clinician with markedly unreliable results.

Urine Culture. Definitive diagnosis of UTI is based on isolation of significant numbers of bacteria on urine culture. Traditionally, 10$^5$ CFUs/mL has been used as the statistically significant number for the presence of UTI. However, use of an absolute number is fraught with limitations. The presence of 10$^5$ CFUs/mL of bacteria in cultures from urine is associated with a 95% likelihood of infection, whereas 10$^4$ CFUs/mL is associated with a 50% likelihood of infection. It makes best clinical sense to put these results in clinical context regarding the presence of symptoms suggestive of a UTI. The symptom complex of dysuria, frequency, urgency, and suprapubic pain may be caused by a wide variety of infectious organisms in numbers far less than the traditional 10$^5$ CFUs/mL. In addition, these same symptoms may represent a significant upper tract infection or may be caused by urethritis.
The presence of bacteria on culture in the absence of clinical manifestations does not always indicate infection. Women often carry large numbers of pathogenic bacteria on the perineum, and uncircumcised men may harbor large quantities of uropathogenic bacteria on the foreskin. The presence of bacteria in these regions may contaminate otherwise sterile bladder urine during collection.

The decision to perform a urine culture should be assessed for its relevance to patient care. Patients with frequency, dysuria, urgency, and suprapubic pain should be treated on the basis of its relevance to patient care. Patients with frequency, dysuria, and suprapubic pain should be treated on the basis of its relevance to patient care. Patients with frequency, dysuria, and suprapubic pain should be treated on the basis of its relevance to patient care. Patients with frequency, dysuria, and suprapubic pain should be treated on the basis of its relevance to patient care. Patients with frequency, dysuria, and suprapubic pain should be treated on the basis of its relevance to patient care.

**Imaging.** The majority of patients with acute cystitis or pyelonephritis do not need emergency imaging of the urinary tract. In certain clinical settings, however, emergency imaging is indicated. Patients with either unusually severe signs and symptoms or an atypical clinical presentation are candidates for genitourinary imaging. For example, a patient with the classic signs and symptoms of pyelonephritis but unremarkable urinalysis findings may have an obstructive process that has prevented the leukocytes and bacteria from reaching the bladder. Another example is that of a patient with a known history of UTI, currently receiving antibiotic therapy, who has persistent fever, chills, and general toxicity. Perhaps one of the most sensitive predictors of a complicated infection (e.g., abscess) is the persistence of fever beyond 72 hours after the institution of antimicrobial therapy. Pyelonephritis with obstruction from any cause can rapidly lead to abscess formation with resultant deterioration of renal function and sepsis. Emergency imaging is indicated to rule out this condition or to identify a suspected renal stone serving as a nidus for infection.

First episodes of UTI in selected patients, such as children younger than 4 years, generally require evaluation after resolution of the UTI. These patients are more likely than those in the general pediatric population to have structural anomalies and, if untreated, are at increased risk for recurrent UTI or for the development of complications such as hydronephrosis, renal scarring, and ultimately renal failure. The female patient with multiple episodes of complex infection, the patient with diminishing renal function, and the patient with renal colic and a possible obstructing stone, which progresses to sepsis, all require imaging.

Several imaging studies may be useful in these patients. Historically, intravenous or excretory urography—typically, intravenous pyelography (IVP)—was used because such studies provide both structural and functional information about the upper urinary tract. Recent work has focused on obtaining this information through safer, less invasive, and less costly methods. Ultrasonography compares favorably with IVP but is inferior to computed tomography (CT). Radionuclide cystograms compare favorably with voiding cystourethromgrams in the diagnosis of vesicoureteral reflux and give less ionizing radiation to the gonads by a factor of 50 to 100. Voiding cystourethrography is the traditional method for initial evaluation of the genitourinary tract. CT scan is exceptional for diagnosing upper tract complications such as various degrees of pyelonephritis, abscesses, pyonephrosis, granulomatous infections, and infected cysts. As with IVP, its disadvantages include higher cost, radiation exposure, and potential for contrast-induced reactions.

**Ultrasonography.** Ultrasonography is useful in the evaluation of patients with potential urinary obstruction. It is a sensitive tool for detecting intrarenal and perinephric abscess and the presence of hydroureret and hydrenephrosis. It is less accurate in determining the presence of a partially obstructing ureteral stone. Ultrasound examination also can detect the presence of pyelonephritis and congenital anomalies. Regardless of patient age group, this procedure is relatively inexpensive and avoids the hazards of contrast and radiation exposure.

**Intravenous Pyelography.** IVP, previously considered one of the mainstays in the evaluation of the genitourinary tract, has been nearly replaced by helical CT scanning and ultrasonography. IVP has higher sensitivity and specificity for determining the presence of obstruction than ultrasound examination but is inferior to CT scanning, and it is not sensitive for detecting the presence of pyelonephritis and renal abscess.

**Radionuclide Scans.** Radionuclide scans also are gaining popularity for the early evaluation of UTI. A dimercaptosuccinic acid scan is the most sensitive method of identifying pyelonephritis and is the imaging study of choice in infant girls with UTI and fever.

**Computed Tomography of the Abdomen.** A contrast-enhanced CT scan of the abdomen is perhaps the best test for assessing the kidneys, ureters, and bladder. It has the highest sensitivity for detecting abscess, obstruction, and acute inflammation. Its disadvantages include cost, radiation exposure, and potential for contrast-induced reactions and radiocontrast agent–induced acute kidney injury, which occurs infrequently in patients with a serum creatinine less than 1.5 mg/dL and can be further avoided by intravenous hydration with normal saline. CT without contrast can be performed in patients with renal insufficiency and is the preferred study in patients with clinical concern for urolithiasis.

### Complicated Urinary Tract Infection in High-Risk Populations

**Pregnancy**

UTI during pregnancy represents a special situation. The incidence of UTI in pregnancy is approximately 2 to 7%. Maternal complications include acute pyelonephritis, increased incidence of...

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**Box 99-1 Patient Groups in Which Urine Culture Is Indicated**

- Children
- Adult men
- Immunocompromised patients
- Patients with “treatment failure” (i.e., with persistent urinary symptoms despite recently completed course of antibiotics)
- Patients with duration of symptoms more than 4 to 6 days
- Elderly patients at risk for bacteremia
- Ill-appearing patients with signs and symptoms suggestive of pyelonephritis or bacteremia
- Pregnant women
- Patients with known chronic or recurrent renal infection
- Patients with known anatomic urologic abnormalities
- Patients in whom urinary tract obstruction is suspected (e.g., stones, benign prostatic hypertrophy)
- Patients with serious medical diseases, including diabetes mellitus, sickle cell anemia, cancer, and other debilitating diseases
- Patients with alcoholism or drug dependence
- Recently hospitalized patients
- Patients taking antibiotics
- Patients who recently have undergone urinary tract instrumentation (e.g., cystoscopy, catheterization)
postpartum chronic pyelonephritis, preterm delivery, and low newborn birth weight. The physiologic changes that occur within the urinary tract of pregnant women include ureteral and renal pelvis dilation, as well as reduced peristalsis throughout the collecting system. During the last trimester, minimal ureteral contractions occur in many patients.

Unlike bacteriuria in nonpregnant females, bacteriuria in pregnant women, even if they are asymptomatic, should be treated. Complications that may result from untreated bacteriuria in pregnancy include premature labor, perinatal mortality, maternal anemia, and maternal pyelonephritis.

Reasonable antibiotic choices include amoxicillin, cephalixin, and nitrofurantoin. Trimethoprim-sulfamethoxazole (TMP-SMX) may be used before the third trimester. Single-dose therapy is not recommended, because these are not considered simple infections. Hospital admission should be considered in patients who are in their last trimester, who appear ill, or who have evidence of pyelonephritis and would benefit from treatment with parenteral antibiotics and intravenous fluids. Although pregnant patients with UTI are being treated on an outpatient basis more frequently than in the past, conservative treatment and close follow-up are warranted.

Diabetic and Sickle Cell Disease

Diabetic patients with bacteriuria also have an increased risk for the development of pyelonephritis, but treatment of asymptomatic bacteriuria has not been proved to be beneficial and should not be standard therapy at this time. Papillary necrosis, perinephric and renal abscess formation, and emphysematous cystitis represent grave complications for this patient group. Manifestations of these complications may include altered vital signs, systemic signs and symptoms such as nausea and vomiting with dehydration, and a toxic appearance suggesting bacteremia and sepsis. These patients require aggressive fluid resuscitation, intravenous antibiotics, and a thorough diagnostic investigation including CT scanning.

Patients with sickle cell anemia also have shown a predilection for the development of papillary necrosis and generalized renal microvascular compromise. In these patients, compromised renal function is thought to be secondary to microvascularule damage from the chronic sickling of the erythrocytes. The renal damage from a UTI can compound the chronic renal insufficiency, leading to rapid worsening of the condition of these patients. They and all patients with underlying kidney disease or renal compromise should be approached conservatively, with hospitalization as deemed necessary for intravenous hydration and antibiotics.

Indwelling and Temporary Urinary Catheters

Treatment of asymptomatic bacteriuria in patients with indwelling catheters is not indicated. Antibiotic treatment results in the development of resistant microorganisms, whereas removal of the catheter leads to the spontaneous elimination of bacteria in many patients. Treatment of patients with a UTI in whom removal of the catheter is contraindicated includes urine culture and sensitivity, antibiotic therapy, replacement of the catheter, and strong consideration for hospitalization in those who exhibit altered vital signs, systemic symptoms, or a toxic appearance. These patients are at high risk for infection with an unusual pathogen and subsequent bacteremia. Urine culture with antibiotic sensitivity testing will help guide antibiotic therapy in this patient population.

The prevention of catheter-associated urinary tract infections (CAUTIs) has become an area of recent interest in the hospital setting since the Centers for Medicare and Medicaid Services (CMS) identified this condition as one of the six unacceptable diagnoses for payment if UTI was not present on admission. CAUTIs are responsible for more than 80% of nosocomial UTIs and 40% of all nosocomial infections. Less than half of urinary catheters placed in hospitalized elderly patients have been found to be clinically indicated. The most effective strategy for addressing CAUTIs is to prevent the infection from occurring by placing urinary catheters only when indicated and considering the use of intermittent catheterization and condom catheters when appropriate.

Differential Considerations

Bacterial UTI is the most common cause of dysuria with low-count infections. It is important, however, to consider the possibility of acute urethritis or acute vaginitis in these patients, as well as mechanical trauma or irritation (Tables 99-1 to 99-3; Fig. 99-1). Urethritis caused by Chlamydia may be seen in patients with acute dysuria; in fact, C. trachomatis may be present in up to 20% of women with dysuria. In general, if historical information includes contact with multiple sexual partners, a recent change in sexual partners, or a sexual partner with dysuria or discharge, C. trachomatis infection should be strongly considered. A pelvic examination should be performed, and culture specimens should be obtained to detect C. trachomatis and N. gonorrhoeae. Other causes of acute dysuria include infections with Trichomonas vaginalis and herpes simplex virus.

The dysuria of vaginitis most often is described as “external,” the sensation being caused by the passage of urine over inflamed introital tissue. Elderly women may report dysuria secondary to atrophic vaginitis. In either case, a pelvic examination may be required. Urinary frequency and urgency are seldom if ever associated with a vaginal cause of dysuria.

Bacterial infection of the bladder is the most likely cause of dysuria in female patients. Most demonstrate positive results on urine cultures, with growth of more than 10^5 CFUs/mL of bacteria. This number is not absolute because 30 to 50% of patients have low-count bacterial infections as a cause of their symptoms. It has been suggested that low bacterial counts may represent an early phase of UTI.

Management

Simple Urinary Tract Infection

Options for treating uncomplicated lower UTI include single-dose therapy, short-course therapy (3-5 days), and the more traditional 7- to 10-day course of therapy (Table 99-4). E. coli remains the most common urinary tract pathogen and is susceptible to many antibiotic regimens. Emerging resistance to TMP-SMX has been noted in 15 to 32% of organisms. In some areas of Europe, resistance to TMP-SMX is approaching 50%. Risk factors for UTI from TMP-SMX-resistant E. coli include recent use of antibiotics (especially TMP-SMX), recent travel to areas with a high prevalence of resistance, and age younger than 3 years with daycare attendance.

Three days of therapy is more effective than single-dose therapy. Three days of therapy have the advantages of improved compliance, lower cost, and reduced side effects. It currently is the recommended regimen for treatment of uncomplicated lower UTI. Despite emerging resistance, TMP-SMX remains the best first-line agent for 3-day regimens when compared with other commonly used antibacterials, owing to its low cost and effectiveness. Short-course 3-day therapy also is effective for asymptomatic bacteriuria in pregnancy. It is unclear whether this regimen can be used for symptomatic lower UTIs in pregnancy, so longer-course therapy is recommended. A regimen of 7- to 10-day therapy generally offers no benefit over shorter courses in uncomplicated
Table 99-1  Differential Diagnosis of Dysuria Syndromes: Laboratory Findings

<table>
<thead>
<tr>
<th>SYNDROME OR DISORDER</th>
<th>MICROSCOPIC HEMATURIA OR PYURIA</th>
<th>BACTERIURIYA</th>
<th>URINE CULTURE (&gt;10^5 CFUs/ML)</th>
<th>POSITIVE FLUID OR CERVICAL SMEAR</th>
<th>POSITIVE CULTURE OF GENITAL LESIONS, CERVIX, OR URETHRA*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute pyelonephritis</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Acute cystitis</td>
<td>+</td>
<td>±</td>
<td>+</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Urethritis caused by sexually transmitted disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Herpes simplex virus</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>±</td>
<td>+</td>
</tr>
<tr>
<td>Neisseria gonorrhoeae</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>Chlamydia trachomatis</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>Vulvovaginitis (bacterial vaginosis, trichomoniasis, yeast, genital herpes simplex)</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>±</td>
</tr>
<tr>
<td>Noninflammatory dysuria (trauma, irritant, allergy)</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
</tbody>
</table>


*Positive for herpes simplex virus, Neisseria gonorrhoeae, or Chlamydia trachomatis.

Table 99-2  Differential Diagnosis of Dysuria Syndromes: Physical Examination

<table>
<thead>
<tr>
<th>SYNDROME OR DISORDER</th>
<th>VAGINAL OR CERVICAL DISCHARGE, VULVAR LESIONS</th>
<th>SUPRAPUBIC TENDERNESS</th>
<th>FLANK TENDERNESS, FEVER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute pyelonephritis</td>
<td>−</td>
<td>±</td>
<td>−</td>
</tr>
<tr>
<td>Acute cystitis</td>
<td>−</td>
<td>±</td>
<td>−</td>
</tr>
<tr>
<td>Urethritis caused by sexually transmitted disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Herpes simplex virus</td>
<td>+</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Neisseria gonorrhoeae</td>
<td>+</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Chlamydia trachomatis</td>
<td>+</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Vulvovaginitis (bacterial vaginosis, trichomoniasis, yeast, genital herpes simplex)</td>
<td>+</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Noninflammatory dysuria (trauma, irritant, allergy)</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
</tbody>
</table>


Table 99-3  Clinical Differentiation among Major Causes of Dysuria

<table>
<thead>
<tr>
<th>CAUSE</th>
<th>CLINICAL FEATURES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinary tract infection</td>
<td>Internal dysuria Frequency, urgency, voiding small volumes Abrupt onset Suprapubic pain Often associated with diaphragm use Presence of pyuria Presence of hematuria (50% of patients)</td>
</tr>
<tr>
<td>Sexually transmitted disease</td>
<td>Internal dysuria Occasional history of frequency, urgency, voiding small volumes Gradual onset History of new or multiple sexual partners Vaginal discharge</td>
</tr>
<tr>
<td>Vaginitis</td>
<td>External dysuria Gradual onset Vaginal discharge Vaginal odor Pruritus</td>
</tr>
</tbody>
</table>


UTIs; however, it remains the standard of care in complicated infections (in patients with pregnancy, diabetes, or sickle cell anemia) for which cure rates are lower with shorter regimens.

The fluoroquinolones are considered first-line agents in regions in which the incidence of TMP-SMX resistance has approached 10 to 20%. Ciprofloxacin is the most commonly used drug and requires twice-daily dosing. Although more expensive than ciprofloxacin, gatifloxacin and levofloxacin offer once-daily administration, have the broadest activity, and have same-dose bioequivalence between oral and parenteral administration. Fluoroquinolones damage developing cartilage in animal studies and should be avoided in children.

Nitrofurantoin and trimethoprim are excellent drugs for the treatment of acute bacterial cystitis. Nitrofurantoin is inexpensive and maintains low serum and high urine levels, with a bacterial resistance pattern that remains unchanged. Adverse reactions are primarily secondary to GI disturbance, but they may be alleviated by use of the macrocrystalline form (Macrobid). Folate antagonists such as trimethoprim have a broader spectrum of activity than does nitrofurantoin. The addition of sulfamethoxazole further broadens the spectrum to include coverage for Proteus and Klebsiella. Folate antagonists carry a higher incidence of adverse
Figure 99-1. Diagnostic protocol for women with dysuria. CFUs, colony-forming units; STD, sexually transmitted disease; UTI, urinary tract infection. (From Stamm WE: Protocol for diagnosis of urinary tract: Reconsidering the criterion for significant bacteriuria. Urology 32[Suppl 2]:6, 1988.)

For the patient with dysuria, initial evaluation includes a pelvic exam and a Gram’s stain, followed by a urine culture. If symptoms persist, a second- or third-line antibiotic may be necessary. Patients with severe UTI may require hospitalization.

Complex Urinary Tract Infection

Mild-to-moderate pyelonephritis can be safely treated on an outpatient basis with a fluoroquinolone for 10 to 14 days (first-line agent) or TMP-SMX (second-line agent) as long as the patient is able to eat and drink, has achieved adequate pain control, and has appropriate social support in the home. In many clinical centers, observation units have evolved to offer a short-stay (less than 24 hours) option for moderate cases in which immediate discharge for outpatient therapy may not be the optimal approach to management.

Severe upper tract UTI necessitating hospitalization initially should be treated with parenteral antibiotics, with transition to oral therapy after the patient has been afebrile for 24 to 48 hours. Oral therapy should be continued for 2 weeks. Because 20% of cultures are resistant to ampicillin, cephalothin, and sulfonamides, antibiotic therapy should be initiated with a fluoroquinolone.

Hospitalization is required in the presence of clinical toxicity (fever, tachycardia, hypotension, vomiting), inability to take oral medications, an immunocompromised state, third-trimester pregnancy, inadequate social circumstances, failure of oral outpatient therapy, or urologic abnormalities or in patients with significant comorbid conditions, including heart failure or renal insufficiency. A subgroup of patients with upper tract UTI do not require immediate hospital admission but may benefit from intravenous hydration and pain and fever control, along with a first dose of an intravenous fluoroquinolone before discharge from the ED. If these patients do not have any contraindications as previously discussed and they improve clinically and are able to tolerate food and drink, they can be safely discharged home on a 10- to 14-day course of an oral fluoroquinolone with close primary physician follow-up. Urine culture with sensitivity testing and further diagnostic evaluation are not necessary in this patient population.

**URINARY TRACT INFECTION IN CHILDREN**

**Perspective**

UTI is a major bacterial disease of childhood; it is estimated that 0.8 to 1.5% of children have bacteriuria. The risk for development of UTI before 11 years of age is 3% in girls and 1% in boys.4 The incidence of UTI in the neonatal period is higher in boys, but the infection becomes more prominent in girls during infancy and thereafter. In children aged 1 to 3 months, UTI is associated with a high incidence of sepsis (30%).7 After the age of 3 months, the incidence of sepsis associated with UTI decreases (to 5%). Vesicoureteral reflux is a common risk factor for UTI and renal scarring in children.12 Data suggest that the incidence of scar formation after acute pyelonephritis may be as high as 37%.
Table 99-4  Treatment Regimens for Bacterial Urinary Tract Infections (UTIs)

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>CHARACTERISTIC PATHOGENS</th>
<th>MITIGATING CIRCUMSTANCE(S)</th>
<th>RECOMMENDED EMPIRICAL TREATMENT*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute uncomplicated cystitis in women</td>
<td><em>Escherichia coli</em>, <em>Staphylococcus saprophyticus</em>, <em>Proteus mirabilis</em>, <em>Klebsiella pneumoniae</em></td>
<td>None</td>
<td>Use 3-day regimens: oral TMP-SMX, trimethoprim, norfloxacin, ciprofloxacin, ofloxacin, lomefloxacin, or enoxacin†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diabetes, symptoms for longer than 7 days, recent UTI, use of diaphragm, age &gt;65 yr</td>
<td>Consider 7-day regimen: oral TMP-SMX, trimethoprim, norfloxacin, ciprofloxacin, ofloxacin, lomefloxacin, or enoxacin†</td>
</tr>
<tr>
<td>Acute uncomplicated pyelonephritis in women</td>
<td><em>E. coli</em>, <em>P. mirabilis</em>, <em>K. pneumoniae</em>, <em>S. saprophyticus</em></td>
<td>Mild to moderate illness, no nausea or vomiting—outpatient therapy</td>
<td>Oral† TMP-SMX, norfloxacin, ciprofloxacin, ofloxacin, lomefloxacin, or enoxacin for 10-14 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe illness or possible urosepsis—hospitalization required</td>
<td>Parenteral† TMP-SMX, ceftriaxone, ciprofloxacin, ofloxacin, or enoxacin for 14 days</td>
</tr>
<tr>
<td>Complicated UTI</td>
<td><em>E. coli</em>, <em>Proteus species</em>, <em>Klebsiella species</em>, <em>Pseudomonas species</em>, <em>Serratia species</em>, enterococci, staphylococci</td>
<td>Mild to moderate illness, no nausea or vomiting—outpatient therapy</td>
<td>Oral† norfloxacin, ciprofloxacin, ofloxacin, lomefloxacin, or enoxacin for 10-14 days</td>
</tr>
</tbody>
</table>


 TMP-SMX, trimethoprim-sulfamethoxazole.

*Recommended treatments are those to be prescribed before the causative agent is known (Gram staining can be helpful); they can be modified once the agent has been identified. These recommendations are limited to drugs currently approved by the U.S. Food and Drug Administration, although not all of the regimens listed are approved for these indications. Fluoroquinolones should not be used in pregnancy. TMP-SMX, although not approved for use in pregnancy, has been widely used. Gentamicin should be used with caution in pregnancy because of its possible toxicity to eighth nerve development in the fetus.

†Multiday oral regimens for cystitis are as follows: TMP-SMX 160-800 mg every 12 hr; trimethoprim 100 mg every 12 hr; norfloxacin 400 mg every 12 hr; ciprofloxacin 250 mg every 12 hr; ofloxacin 200 mg every 12 hr; lomefloxacin 400 mg every day; enoxacin 400 mg every 12 hr; macrocrystalline nitrofurantoin 100 mg four times a day; amoxicillin 250 mg every 8 hr; and cefpodoxime proxetil 100 mg every 12 hr.

‡Parenteral regimens are as follows: TMP-SMX 160-800 mg every 12 hr; norfloxacin 400 mg every 12 hr; ciprofloxacin 500 mg every 12 hr; ofloxacin 200-300 mg every 12 hr; lomefloxacin 400 mg every day; enoxacin 400 mg every 12 hr; amoxicillin 500 mg every 8 hr; and cefpodoxime proxetil 200 mg every 12 hr.

§Parenteral regimens are as follows: TMP-SMX 160-800 mg every 12 hr; ciprofloxacin 200-400 mg every 12 hr; ofloxacin 200-400 mg every 12 hr; gentamicin 1 mg/kg of body weight every 8 hr; ceftriaxone 1-2 g every day; ampicillin 1 g every 6 hr; imipenem-cilastatin 250-500 mg every 6-8 hr; tetracycline-clavulanate 3.2 g every 8 hr; and aztreonam 1 g every 8-12 hr.

Principles of Disease

As in adults, *E. coli* is the predominant pathogen. Age-related differences are recognized: In older boys *Proteus* often is isolated during UTI, whereas in newborn children, *Klebsiella* is more often the causative agent. The route of infection also is age related. It is thought that in the newborn period the bacteria are blood-borne (and often associated with generalized sepsis). In the older age group, as in adults, the ascending urethral route is primarily responsible for generating infection of the urinary tract.

Clinical Features

UTI often is overlooked in children because of inappropriate emphasis on classic signs and symptoms, with little regard to age variables. Nonspecific findings should be considered the rule and not the exception (Table 99-5). Pyelonephritis may be present without overt symptoms. A UTI in a febrile patient usually indicates pyelonephritis. An elevated blood urea nitrogen (BUN) level or hypertension in a child older than 2 months strongly suggests bilateral hydronephrosis or advanced renal parenchymal disease.

Neonates

Generalized septicemia often is the major manifestation of neonatal UTI. Classically, feeding difficulties, irritability, and sluggishness are seen in this age group. Bacteremia is present in nearly 50% of cases.

Age 1 Month to 3 Years

The 1-month to 3-year age group has the most deceptive clinical presentation of UTI. Nonspecific findings are typical: fever, irritability, abdominal pain, vomiting, and failure to thrive. Occasionally, gross hematuria may be present.

Table 99-5  Signs and Symptoms of Urinary Tract Infection by Age Group

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>INFANT</th>
<th>PRESCHOLER</th>
<th>SCHOOL-AGE CHILD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor feeding</td>
<td>Vomiting</td>
<td>Jaundice</td>
<td>Hypothermia</td>
</tr>
<tr>
<td>Poor feeding</td>
<td>Vomiting</td>
<td>Diarrhea</td>
<td>Fever</td>
</tr>
<tr>
<td>Poor feeding</td>
<td>Vomiting</td>
<td>Diarrhea</td>
<td>Fever</td>
</tr>
<tr>
<td>Poor feeding</td>
<td>Vomiting</td>
<td>Diarrhea</td>
<td>Fever</td>
</tr>
<tr>
<td>Poor feeding</td>
<td>Vomiting</td>
<td>Diarrhea</td>
<td>Fever</td>
</tr>
</tbody>
</table>
As described previously, urine collection often poses a challenge in a child with a suspected UTI. Catheterization or suprapubic bladder aspiration provides the most reliable specimen for determination of UTI.

**Management and Disposition**

As in adults, many therapeutic options are available for children with UTI. Sulfonamides, nitrofurantoin, TMP-SMX, cephalosporins, and aminopenicillins are effective. Newborns and young infants should be treated with ampicillin and gentamicin on an inpatient basis. Sulfonamides and fluoroquinolones should be avoided in pediatric patients. Traditionally, inpatient treatment with parenteral antibiotics has been the standard of care for young children with suspected pyelonephritis. Oral therapy is acceptable in children with uncomplicated infections. These patients should be managed conservatively, and hospital admission is advised for children who are dehydrated, severely ill, or not tolerating oral fluids and for those with underlying structural abnormalities of the genitourinary system. In addition, family dynamics, which could affect compliance with medication, should be taken into consideration.

The appropriate duration of therapy currently is a subject of debate. Many experts believe that in children with uncomplicated lower UTI, short-course therapy can be used instead of the traditional 10-day regimen. Short-course (3-day) therapy is more widely accepted in adolescent girls. Once the decision to discharge a child has been made, the parents should be advised of signs of toxicity and the importance of compliance with medications.
Parents should bring the child to the ED immediately if signs of toxicity develop and should arrange routine follow-up with the pediatrician if the child improves. The child should be seen for follow-up 2 to 3 days after the ED visit and again 2 to 3 weeks later (or 7 to 10 days after completion of the antibiotic course).

**URINARY TRACT INFECTION IN MEN**

**Perspective**

The incidence of UTI is much lower in men than in women. The route of infection in men is usually ascending, from the urethra to the prostate, to the bladder, and then to the kidney. Pathogenic organisms responsible for UTI in men are similar in type, regardless of the site of infection in the genitourinary tract. E. coli causes 80% of infections in men. UTI in men should be considered a complicated infection and requires a more extensive evaluation as well as a longer course of antibiotics.

**Specific Disorders**

**Cystitis**

Cystitis is rare in male patients in the absence of blunt trauma, prostatic hyperplasia with obstruction, and previous instrumentation. Chronic prostatitis, prostatic hyperplasia with obstruction, and previous instrumentation are the most common predisposing factors. Lack of circumcision and homosexuality are other recognized risk factors. Commonly, men with cystitis have symptoms of urinary urgency, frequency, dysuria, nocturia, suprapubic pain, and often low back pain. Gross hematuria occasionally occurs, but fever, chills, and flank pain generally are absent. On physical examination, suprapubic tenderness to palpation may be elicited. Pneumaturia may be present and is indicative of an infection with gas-forming bacteria. It also may indicate the presence of a vesicoenteric fistula, which often is caused by diverticulitis, although rectosigmoid carcinoma and regional enteritis are associated diseases as well. If fever and chills are present in association with irritative symptoms and difficulty voiding, acute bacterial prostatitis should be strongly considered. The most common pathogens found in men with cystitis are E. coli, Proteus, and Providencia.

A voided urine specimen should reveal pyuria, bacteriuria, and a variable degree of hematuria. Urine culture is indicated. If there are no signs of toxicity, the patient can be treated on an outpatient basis with any of the urinary antibacterial agents (TMP-SMX, nitrofurantoin, sulfonamides, or fluoroquinolones).

Three qualifying factors should be addressed in dealing with UTIs in men:

1. **Obstruction.** It is imperative that urinary obstruction be ruled out as a pathogenic mechanism. Infection and obstruction together can be catastrophic and lead to sepsis. Obstruction at the level of the prostate in older men is common and should be considered. Catheterization or bedside ultrasound examination may help to rule out retention. Suggestion of obstruction of the upper tracts based on lack of response to medical therapy necessitates performance of an abdominal noncontrast CT scan or ultrasound.

2. **Genitourinary tract anomalies.** UTIs in men may be secondary to underlying diseases of the genitourinary tract. Therefore all of these patients should be referred to a urologist for diagnostic studies.

3. **Catheterization.** Urethral catheterization should not be used to collect a urine specimen in men unless they are experiencing urinary retention. An inability to produce a specimen in the presence of infectious symptoms should be a major clue regarding the cause of the infection. If retention is suspected, catheterization for collection of residual urine is indicated. Referral for urologic consultation is recommended and hospital admission is prudent for a majority of these patients.

**Pyelonephritis**

Clinical features in men with acute pyelonephritis include flank and costovertebral angle pain, chills and fever, urinary frequency, urgency, and dysuria. Generalized malaise, nausea, and vomiting are early signs of systemic toxicity, suggesting impending gram-negative sepsis. These patients require hospitalization for intravenous hydration and antibiotic therapy, as well as pain and fever control.

A voided urine specimen usually reveals leukocytes, occasional leukocyte casts, a variable number of RBCs, and bacteria. Urine culture is essential in this patient population. Blood cultures should be performed if the clinical picture suggests sepsis. A complete blood count, renal function studies, and electrolyte studies are recommended. Uncomplicated pyelonephritis should not produce detectable alterations in the BUN level. In males, imaging with ultrasonography or an abdominal CT scan without contrast is required because the cause typically involves obstruction secondary to a stone, prostate pathology, stricture, or tumor. Catheterization for collection of residual urine may be indicated if urinary retention is suspected.

**Prostatitis**

Bacterial prostatitis is an infection of the prostate caused primarily by gram-negative organisms. More than 80% of cases are caused by strains of E. coli; the remainder are caused by Klebsiella, Enterobacter, Proteus, and Pseudomonas species. Patients with prostatitis may report dysuria and perineal and low back pain associated with fever, chills, and malaise. A rectal examination should be part of the workup in all men with symptoms suggestive of cystitis. Bacterial prostatitis is not a subtle disease, and patients will have an exquisitely tender and boggy prostate.

Acute bacterial prostatitis is an acute febrile illness characterized by chills, low back pain, and perineal pain. Irritative symptoms of voiding, including frequency, urgency, and dysuria, are present, along with various degrees of bladder outlet obstruction and retention. Patients often have constitutional symptoms of arthralgia, myalgia, and generalized malaise. Prostate examination reveals a tender, swollen gland that is firm and warm to touch. Palpation of an acutely inflamed prostate should be limited, to avoid the possibility of precipitating bacteremia or sepsis. Cystitis usually accompanies acute bacterial prostatitis; thus culture of voided bladder urine generally reveals the responsible pathogen.

Antimicrobial therapy has been shown to be beneficial and is recommended. In patients without signs of systemic toxicity, a prolonged course of an antibiotic for 4 to 6 weeks is required. This may need to be repeated if only partial success is achieved. The following list represents an appropriate selection of drugs for nontoxic prostatitis:

1. Ciprofloxacin 500 mg orally (PO) twice daily; norfloxacin 400 mg PO twice daily; ofloxacin 400 mg PO twice daily or enoxacin 400 mg PO twice daily for 30 days
2. Trimethoprim with sulfamethoxazole (Bactrim), one double-strength tablet PO twice daily for 30 days

If the patient is toxic appearing with fever, chills, or urinary retention, hospitalization and parental antibiotics are warranted. The following antibiotic choices are appropriate for toxic prostatitis:

1. Ciprofloxacin 400 mg intravenously (IV) every 12 hours, or levofloxacin 500 mg IV every 24 hours
2. Ceftriaxone 2 g IV every 24 hours with or without gentamicin 3 to 5 mg/kg/day

Some experts have recommended alpha-blocker therapy because the bladder neck and prostate are rich in these receptors and alpha-blockade may improve outflow obstruction and diminish prostatic reflux. Although this may facilitate the patient’s recovery course, clinical trials are ongoing and this is not considered standard of care.²⁸

If the patient is experiencing painful urinary retention, urethral catheterization should be avoided. Suprapubic needle aspiration or catheterization is much safer and more comfortable than urethral catheterization for initial management. A urologist should be consulted regarding management of all patients requiring suprapubic tube placement.

General support measures for outpatients should include bed rest, analgesics, antipyretics, hydration, and stool softeners.

**Chronic Prostatitis**

Patients with chronic prostatitis typically visit the ED when they experience an acute exacerbation of the disease. Clinical manifestations vary widely, but most patients report some degree of irritative voiding symptoms (frequency, urgency, dysuria), low back and perineal pain, and occasionally myalgias.²⁷ Fever and chills are uncommon except during an acute exacerbation of the chronic infection. A history of previous episodes of acute prostatitis may be absent. Findings on the physical examination, including examination of the prostate, often are unremarkable. The hallmark of chronic bacterial prostatitis is relapsing UTI caused by the same organism. Chronic bacterial prostatitis is the most common cause of recurrent UTI in men.

Antimicrobial therapy is recommended for the treatment of chronic prostatitis.²⁹ Most antimicrobials diffuse poorly from plasma into prostatic fluid. The fluoroquinolones achieve the highest concentrations in the prostate and are the drugs of choice, with cure rates of approximately 64%. The recommended dosages are as follows: ciprofloxacin 500 mg twice daily for 30 days; norfloxacin 400 mg twice daily for 30 days; enoxacin 400 mg twice daily for 30 days; or ofloxacin 300 mg twice daily for 6 weeks. TMP-SMX also is useful, with cure rates of 44 to 50%. The dosage is one double-strength tablet twice daily, but the optimal duration of therapy is unclear and may range from 4 to 16 weeks.

**RENAL CALCULI**

**Perspective**

Background

Urolithiasis constitutes a common clinical problem seen in the ED and affects 5 to 15% of the population worldwide, and recurrence rates are close to 50%.³⁰ From 1994 through 2000, presentations for urolithiasis nearly doubled, and all indicators point to a rise in the incidence of kidney stones in the general population.²⁷ Renal calculi are seen commonly in young adults and middle-aged men, with nearly 70% of all ureteral calculi occurring in individuals aged 20 to 50 years. Most ureteral calculi originate in the kidney and then pass into the collecting system.

Epidemiology

Various clinical syndromes involve metabolic alterations and are therefore associated with an increased likelihood of stone formation (Box 99-2). Risk factors include age, male gender, and family history. In the United States, prevalence rates for renal calculi are 10% in men and 5% in women.³⁰

**Box 99-2  Risk Factors for Urolithiasis**

- Metabolic disease or disturbance
- Crohn’s disease
- Milk-alkali syndrome
- Primary hyperparathyroidism
- Hyperuricemia
- Hyperuricosuria
- Sarcoidosis
- Recurrent UTI
- Renal tubular acidosis (type I)
- Gout
- Laxative abuse
- Positive family history
- Hot arid climates (southeast United States)
- Male gender (white men affected more commonly than black men)
- Previous kidney stone
- Dehydration

UTI, urinary tract infection.

**Principles of Disease**

**Overview**

Multiple pathogenic factors interact to cause the formation of renal calculi. Renal calculi can be stratified into the following types: calcium, struvite, uric acid, and cystine.

Most stones (75%) are composed of calcium oxalate, alone or in combination with calcium phosphate. Hyperexcretion of calcium is a major contributor to stone formation and occurs in various clinical settings. The major dietary sources of calcium are cheese and milk, and hypercalciuria may occur in adults who ingest more than 1 quart of milk daily. Many conditions are associated with the development of hypercalciuria with increased risk of stone formation. The most common is hyperparathyroidism, in which calculi develop in 67% of patients. Peptic ulcer disease also may be a predisposing factor. These patients tend to ingest large amounts of calcium with food, in addition to absorbed alkali sources (sodium bicarbonate) and antacids. The other major component of calcium stones, oxalate, is influenced by diet. Hyperoxaluria occurs in the presence of small bowel disease, Crohn’s disease, ulcerative colitis, and radiation enteritis.

Magnesium-ammonium-phosphate (struvite) stones account for approximately 15% of all renal calculi. Struvite stones occur almost exclusively in patients with UTI and often are referred to as “infection stones.” They form as a result of urea-splitting organisms such as *Proteus*, *Providencia*, *Klebsiella*, *Pseudomonas*, and *Staphylococcus*. Distinctive features of these stones include common occurrence as staghorn calculi and the formation of “coffin-lid” crystals, often in the presence of alkaline urine.

Uric acid stones account for 10% of all stones in the United States. The basic causative factor is excessive excretion of uric acid in urine. Approximately 25% of patients with symptomatic gout have uric acid calculi, and the incidence of uric acid stones increases with the use of uricosuric agents. A distinctive feature of uric acid stones is their radiolucency. These stones infrequently take the form of staghorn calculi.

Cystine stones are rare and account for only 1% of stones. They are caused by an inborn error of metabolism that results in increased secretion of cystine and often occur as staghorn calculi.

**Pathophysiology**

Impaction along the genitourinary tract is a serious complication of renal calculi and can cause several physiologic changes. Once
obstruction occurs, a rapid redistribution of renal blood flow results in a decrease in the glomerular filtration rate. As glomerular and tubular function decreases, renal excretion shifts to the unaffected kidney. Obstruction also causes a rapid decrease in ureteral peristaltic activity. In the presence of infection, both renal and ureteral function may be impaired. Complete obstruction of the ureters may lead to loss of renal function, with an increased incidence of irreversible damage after 1 to 2 weeks, including rupture of the renal calyx. Partial obstruction is associated with a lower likelihood of renal injury, but it may still result in irreversible damage.

Although calculus size and location are important determinants of the degree of disease, the major cause of progressive renal damage is associated infection. The stone behaves as a foreign body and leads to stasis and obstruction, decreasing host resistance and increasing the incidence of infection. Subsequent infectious complications include pyelonephritis, perinephric abscess, and gram-negative bacterial sepsis.

The three primary predictors of stone passage without the need for surgical intervention are calculus size, location, and the degree of patient pain at discharge. The most important factor that relates to passage of a calculus though the genitourinary tract is its size. Approximately 90% of stones smaller than 5 mm pass spontaneously within 4 weeks. This percentage decreases to 15% for stones 5 to 8 mm. Up to 95% of stones larger than 8 mm become impacted along the genitourinary tract, and lithotripsy or surgical removal may be required. Unless the stone is infected, renal damage is considerable, or there is obstruction of a solitary kidney, surgical intervention can be performed on an outpatient basis, provided the patient is able to tolerate oral intake and have adequate pain control. Spontaneous passage is more frequent with stones located below the midureter than with those located above the midureter. Patients with well-controlled pain on discharge are less likely to require surgical intervention than those who do not have well-controlled pain.

Renal calculi seldom cause complete obstruction. There are five sites along the ureter at which calculi are likely to become impacted (Fig. 99-3). First, a stone may lodge in the calyx of the kidney or pass into the renal pelvis and become lodged at the ureteropelvic junction. The relatively large renal pelvis (1 cm) narrows abruptly at its distal portion, where it is equal in diameter to its adjoining ureter (2-3 mm). The third region is near the pelvic brim where the ureter arches over the iliac vessels posteriorly into the true pelvis. The most constricted area along the ureter, and a common location for impaction, is at the ureterovesicular junction. This is the site at which the ureter enters the muscular coat of the bladder (intramural ureter). At the time of diagnosis, up to 75% of stones are located in the distal third of the ureter. Finally, calculi may become lodged in the vesical orifice.

**Clinical Features**

**Signs and Symptoms**

The onset of pain usually is abrupt, with a crescendo of extreme pain that begins in the flank, extends laterally around the abdomen, and radiates into the groin. Pain may radiate to the testicles in men and the labia majora in women. A constant, underlying dull ache in the flank is common between episodes of colic. The cause of colicky, severe flank pain is hyperperistalsis of the smooth muscle of the calyces, pelvis, and ureter, whereas the cause of a dull ache can be acute obstruction and renal capsular tension.

Autonomic nerve fibers that serve the kidney, testicle, and ovary are involved in the transmission of pain with renal calculi, and the location of the stone may be suggested by the pattern of pain. A stone located high in the ureter may cause pain that radiates to the testicle (or ovary). As the stone approaches the bladder, the pain may shift to the scrotum or vulva. Symptoms of urinary urgency and frequency often develop as the stone nears the bladder.

GI symptoms of nausea and vomiting are common in patients with renal colic. A third of patients experience gross hematuria, with or without blood clots in the urine. A history of fever and chills strongly suggests superimposed infection; such cases should be regarded as true urologic emergencies.

**Physical Examination**

A patient with renal colic often is in severe pain and paces or writhes in pain on the stretcher, unable to find a comfortable position. The skin usually is pale, cool, and clammy. Fever generally is not noted but if present strongly suggests infection. The abdominal examination may reveal signs of an early ileus with hypoactive bowel sounds. A decrease in peristalsis often accompanies renal colic, but abdominal tenderness usually is absent and flank tenderness is more common. It is essential that the abdomen be auscultated and palpated in search of bruits and thrills over the abdominal aorta and iliac vessels, because the clinical manifestations of aortic abdominal aneurysms may mimic those of renal colic. Patients commonly have intermittent pain that may nearly resolve between episodes of severe discomfort. The clinical picture may be misleading, with the patient’s repeated attempts to secure relief from the pain misinterpreted as drug-seeking behavior, whereas actual pathology requiring medical attention is present.
Diagnostic Strategies

Laboratory Tests

Urinalysis. The initial diagnostic step in the management of suspected renal colic is urinalysis. This simple, noninvasive test provides helpful information on various aspects of the patient’s condition. In general, a dipstick test is performed first to evaluate for the presence of blood and infection. If results are abnormal, it is usually followed by microscopic analysis.

Sediment Analysis. RBCs generally are found in the urine of patients with urolithiasis. However, the absence of RBCs in urine does not exclude the diagnosis. From 10 to 20% of patients with documented urolithiasis have no microscopic hematuria. Furthermore, there is no correlation between the degree of obstruction and the absence of hematuria. Although sterile pyuria can occur in the absence of infection as a result of ureteral inflammation, the presence of a UTI should be investigated if other clinical signs of infection are present, such as fever and chills. A urinalysis with culture should be performed to look for pyuria and bacteriuria and to measure nitrites and leukocyte esterase when infection is suspected.

Urinary pH. The kidney will not produce urine with a pH greater than 7.5 under normal conditions, so urinary pH greater than 7.5 should raise suspicion for the presence of urea-splitting organisms such as Proteus. Renal tubular acidosis and ingestion of absorbable alkali also may increase urine pH and should be considered in the differential diagnosis. A pH less than 5 often is associated with the formation of uric acid calculi.

Crystalluria. Historically, examination of urine crystals present on microscopic evaluation has provided a clue to the type of stone present. As a result of consolidation of urine testing to a central laboratory in most hospitals, this test is no longer routinely performed in most EDs.

Other Laboratory Tests. Serum uric acid levels are elevated in 50% of all uric acid stone formers, but this determination is not mandatory in the ED evaluation of the patient. Measurement of BUN and serum creatinine levels is not routine but should be done in patients who have a renal calculus with a solitary kidney, a transplanted kidney, or a history of renal insufficiency. A complete blood count may reveal a slightly elevated WBC count in patients with renal calculi that may be a result of demargination, but this is not a sensitive test and should be performed only in patients who are thought to be infected. A WBC count higher than 15,000/mm³ or a significant left shift on the differential suggests active infection. Serum calcium and phosphorus levels can help screen for hyperparathyroidism, sarcoidosis, and other disorders of calcium metabolism, but this metabolic workup is not a necessary component of the ED evaluation of nephrolithiasis.

Imaging

Imaging is not needed in all patients with renal colic. If the signs and symptoms are atypical, the diagnosis is in question, the patient appears toxic, high-grade obstruction is suspected, or it is the patient’s first episode of flank pain, imaging should be performed.

Computed Tomography. Non–contrast-enhanced helical (spiral) CT scanning is the standard imaging modality in the United States. It is sensitive and specific (98% and 100%, respectively, with a negative predictive value of 97%) in detecting both ureteral calculi and ureteral obstruction. Other advantages include its ability to detect calculi as small as 1 mm in diameter and to provide direct visualization of complicating conditions such as hydronephrosis (Fig. 99-4), and ureteral edema. The CT scan is superior to other imaging modalities in its ability to recognize other pathology (malignancy, renal abscess, abdominal aortic aneurysm). It also has the advantage of lack of contrast exposure, short duration of testing, and ease of interpretation.

In patients who have signs and symptoms consistent with renal colic without a history of nephrolithiasis, noncontrast CT should be performed to improve diagnostic accuracy and guide management. For unnecessary radiation to be limited and resource usage improved, patients who have a known history of nephrolithiasis and signs and symptoms consistent with renal colic in whom infection is not a concern do not need any form of imaging as long as they improve with medical management. Further imaging is appropriate in patients who have a history of nephrolithiasis who do not improve with treatment, who have a urinalysis showing infection, or in whom a more likely diagnosis than renal colic is suspected.

Contraindications to CT scan imaging are few. Obese patients may be unable to undergo CT scanning if their girth or weight is beyond the capacity of the scanner. CT scanning should be avoided in pregnant patients because of radiation exposure to the fetus, and ultrasonography is the preferred diagnostic modality. CT scans may underestimate the actual size of the stone compared with the scout film from an IVP or kidney, ureter, and bladder (KUB) study. This limitation may adversely affect the management of patients with 5-mm stones, for example, in whom conservative therapy would be chosen on the basis of CT findings, although specific intervention would be more appropriate.

Intravenous Pyelography. IVP is an accurate imaging modality to detect renal stones, but it is seldom used because CT scanning...
and ultrasonography have become first-line imaging modalities. It is very sensitive, capable of establishing the diagnosis of calculous disease in 96% of cases, and it can quantify the presence and severity of obstruction. Contraindications to the use of urographic contrast media include renal insufficiency with a serum creatinine greater than 1.5 mg/dL and previous reaction to radiocontrast material. The incidence of serious contrast reactions is extremely low, estimated to be 0.9 per 100,000. The value of IVP also is limited by the complexity and length of time needed to perform the procedure, which can take as long as 2 hours.

**Ultrasonography.** Ultrasonography is safe and easily performed, but it is much less reliable than CT scanning for detecting small (less than 5 mm in diameter) ureteral and midureteral stones. Although only 37 to 64% sensitive for detecting calculi, ultrasond examination shows hydronephrosis with a sensitivity of 85 to 94% and a specificity of 100% (Fig. 99-5). It is the study of choice for ruling out hydronephrosis in a pregnant patient with pyelonephritis, if obstructive urolithiasis is a concern, or in obese patients who cannot undergo CT scanning.

**Radiography of the Kidney, Ureter, and Bladder.** A KUB film is the standard initial radiographic study done before injection of contrast medium during IVP. It is of limited usefulness on its own except as a progress film after CT has already identified a radiopaque stone. A KUB film is not reliable for diagnosing urolithiasis because it provides only presumptive evidence of calculi (less than 70% specificity), so it should be followed by a more definitive study or be avoided altogether. The most common radiographic densities seen on KUB films are phleboliths in the pelvic veins, which are spherical with a hollow (lucent) center, whereas calculi usually are irregularly shaped and solid. Calcified mesenteric lymph nodes also may add confusion, although unlike phleboliths, these densities commonly change position on subsequent films.

Most calculi (90%) are radiopaque, including calculi composed of calcium oxalate, cystine, calcium phosphate, or magnesium-ammonium-phosphate (Figs. 99-6 and 99-7). Uric acid stones, blood clots, and sloughed papillae are seen as “negative” shadows on radiographs. The most commonly overlooked calculi lie in the region over the sacrum, where small stones often are obscured by this bony density.

**Differential Considerations**

A number of significant clinical diseases can produce flank pain (Box 99-3) and should be considered in patients with symptoms suggestive of renal colic. Such conditions include abdominal aortic aneurysm, pyelonephritis, renal abscess, carcinoma, renal tuberculosis, papillary necrosis, and vascular compromise.

Acute pyelonephritis can cause severe renal pain. Urinalysis will aid in the differential diagnosis by demonstrating pyuria and bacteriuria. However, infection also can occur concomitantly with an obstructive stone. This combination constitutes a true urologic emergency and requires imaging (renal CT, ultrasonography, IVP) to rule out hydronephrosis, which may warrant immediate

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**Figure 99-5.** Ultrasound images of the kidney in a patient with renal colic. A, A long 1-1 axis view demonstrates hydronephrosis. Continued.
Figure 99-5, cont’d. B, A transverse cut shows a calcification with an acoustic shadow in the renal calyx.

Figure 99-6. In a near-term pregnant woman with an obstructed left kidney, intravenous pyelography demonstrates a delayed nephrogram. The right kidney has physiologic hydronephrosis from ureteral compression by the fetal head.

Figure 99-7. In a near-term pregnant woman with an obstructed left kidney, intravenous pyelography demonstrates a delayed nephrogram. The right kidney has physiologic hydronephrosis from ureteral compression by the fetal head.
urologic intervention for placement of ureteral stents or decompression of the renal pelvis by percutaneous nephrostomy. Renal carcinoma also may produce flank pain, especially if hemorrhage has occurred within the tumor. An abdominal flat-plate radiograph (a KUB film) may demonstrate calcifications overlying the renal shadow, which often are seen in renal neoplasms. IVP may suggest the diagnosis, but CT scanning is the best imaging modality.

Papillary necrosis may cause renal colic as a result of passage of sloughed papillae down the ureter. It is most commonly seen in diabetics and in patients with a history of acute or chronic UTI. Sloughed papillae may be visualized on renal imaging and can be mistaken for an obstructive stone. Renal pain, either colicky or noncolicky, may be caused by acute vascular compromise of a kidney. The pain of renal infarction is severe and occasionally associated with microscopic or gross hematuria. The acute vascular changes may be secondary to renal artery embolism, renal vein thrombosis, dissection of the renal artery, rupture of a renal artery aneurysm, aortic dissection, or abdominal aortic aneurysm. If a vascular cause is suspected, a contrast-enhanced CT scan or an angiogram should be performed. The most common of these relatively rare processes is renal artery embolism, which most often is of cardiac origin (as with atrial fibrillation, subacute bacterial endocarditis, or mural thrombus).

An immediate traditional angiogram or CT angiogram scan is indicated because early diagnosis allows possible salvage of the ischemic kidney. Most renal artery aneurysms are small and seldom produce clinical manifestations. Dissection or rupture of a renal artery aneurysm is rare and causes shock and flank pain. Renal vein thrombosis often demonstrates microscopic hematuria and proteinuria. The KUB film may show an increased renal shadow, and in the early stages, contrast studies may show decreased function of the affected kidney. Predisposing factors for renal vein thrombosis include nephrotic syndrome, malignancies, and pregnancy.

A renal or perinephric abscess may cause flank pain, fever, and a palpable mass. Ultrasonography or a CT scan should be performed. A chest radiograph may demonstrate a pleural effusion or elevation of the diaphragm. Both renal and perinephric abscesses require hospitalization for drainage, intravenous fluids, antibiotics, and determination of the underlying cause.

Management

General Approach

Patients with renal stones generally are in severe pain and are unable to find a comfortable position. Often the history and physical examination, combined with the finding of hematuria, allow a presumptive diagnosis to be made and therapy to be initiated. The first priority is adequate pain control. Nonsteroidal anti-inflammatory drugs (NSAIDs) are first-line agents, but parenteral administration often is necessary because of nausea and vomiting. Intravenous ketorolac or dicyclafenc provides rapid effective analgesia with minimal side effects. In addition to their analgesic effects, NSAIDs decrease the pain of renal colic by decreasing ureterospasm and also reduce renal capsular pressure by diminishing the glomerular filtration rate in the obstructed kidney. Accordingly, caution is advised with use of these agents in patients with underlying renal insufficiency or peptic ulcer disease. An antiemetic may be useful if the patient has nausea, and intravenous fluids should be given to the vomiting patient who is unable to tolerate liquids and who is likely to undergo an imaging procedure. There are no definitive studies proving that high-volume fluid therapy in acute renal colic facilitates stone passage or improves outcomes.

Outpatient Management

Most patients with nephrolithiasis may be safely discharged from the ED. Current guidelines recommend urologic intervention in patients with symptoms persisting for longer than 2 months. The patient should be instructed to drink a moderate amount of fluids, to take analgesics as needed for pain, and to engage in activity as tolerated.

Medical expulsive therapy (MET) is another potentially useful treatment modality in the management of distal ureteral stones, although initial reports of its efficacy have recently been questioned. Because ureteral smooth muscle contraction is mediated by intracellular calcium and by the autonomic nervous system, it is thought that alpha-antagonists and calcium channel blockers facilitate distal stone expulsion and decrease the time to spontaneous stone passage. These agents are presumed to work by blocking ureteral smooth muscle contraction and improving antegrade
Indications for Hospital Admission

Hospital admission should be sought for patients who are severely dehydrated, are experiencing unremitting pain or vomiting, or have an underlying urinary infection (Box 99-4). Sepsis and renal damage are risks in the presence of obstruction and infection, so admission to the hospital for symptomatic therapy is inadequate. These patients require urologic consultation to evaluate the need for drainage and for relief of the obstruction. If signs of sepsis (tachycardia, fever, hypotension, shock) are present, antibiotics and fluid resuscitation should be administered pending urologic evaluation. Immediate operative intervention may be indicated to provide drainage and relieve the obstruction.

A patient returning to the ED with persistent colic does not need a second imaging study if the stone was identified previously. In such cases a KUB film may localize the stone. An intervention by the urologist may be necessary if the stone has not progressed along the genitourinary tract. Several interventional strategies are available to the urologist for the management of stones that do not pass spontaneously. Optimal therapy depends on the size, location, and composition of the stone. Extracorporeal shock wave lithotripsy (ECSWL) has proved to be effective for stones located in the kidney, with a greater than 85% clearance rate. Upper ureteral stones also may be cleared with a high success rate when ECSWL is performed after ureteroscopic manipulation of the stone to a more proximal position. Percutaneous nephrolithotomy, which establishes a tract from the skin to the collecting system, is used for stones too large or hard for ECSWL by removing them directly from the renal pelvis.

**BLADDER (VESICAL) CALCULUS**

Although calculi generally form in the kidneys, they also may originate in the bladder. Bladder stones are different from renal stones. In the United States, bladder stones occur almost exclusively in elderly men, often as a complication of other urologic disease. The most common cause is infection of residual bladder urine with urea-splitting organisms. The other common cause of vesical stones is an indwelling catheter. Disorders predisposing to the formation of bladder stones include bladder neck obstruction (usually secondary to prostatic hyperplasia), neurogenic bladder, vesical diverticula, damage from irradiation, and schistosomiasis.

The presenting manifestations most often are pain on voiding and hematuria. The patient may report a sudden interruption of the urinary stream, which strongly suggests a vesical stone that intermittently obstructs the bladder outlet. Frequency, urgency, and dysuria are described by up to 50% of patients, and UTI is common. Physical examination is rarely rewarding because signs may be minimal. Rectal examination may reveal an enlarged prostate or a prostatic malignancy. Poor sphincter tone may suggest a neurogenic bladder. Urinalysis generally reveals pyuria, bacteriuria, and hematuria. Plain radiographs of the pelvis reveal a bladder stone in 50% of cases. Contrast scans may demonstrate obstructive changes in the upper tracts or bladder diverticula. Ultrasonography also is useful in the diagnosis of bladder stones.

**ACUTE SCROTAL PAIN**

**Perspective**

Several unique disorders encountered in the ED setting produce scrotal pain. It is crucial to determine the exact causative disorder in such cases because testicular torsion, a common cause of scrotal pain, represents a surgical emergency requiring rapid recognition and treatment. Other causes of scrotal pain require less invasive and time-dependent therapies, such as antibiotics for epididymitis and observation for benign masses or torsion of the appendix of the testes.

**Principles of Disease**

**Anatomy**

Knowledge of testicular landmarks is essential for examination of the patient with an acute scrotal mass. Figure 99-8 demonstrates the normal anatomy of the scrotum and testis. A normal scrotum is relatively symmetrical, and both testicles are of equal mass and volume. The left testicle often is higher than the right, because its blood flow empties into the large, low-pressure vena cava, whereas the right drains into the relatively smaller, high-pressure renal vein. A normal testis is found in the vertical axis with a slight forward tilt, and the epididymis is above the superior pole in the posterolateral position.

**Physical Examination**

On examination of the testis, any tenderness to palpation, discrepancies in size, loss of testicular landmarks, or discoloration should be noted. The epididymis is located posterolateral to the testis and is normally nontender and soft. The cremasteric reflex is elicited by stroking or pinching the inner aspect of the thigh; more than 0.5 cm of elevation of the ipsilateral testicle is considered evidence of a normal reflex. This reflex normally is absent in 50% of male infants younger than 30 months of age.
Differential Considerations

Acute scrotal pain can arise with testicular torsion, epididymitis, torsion of the appendix of the testis, testicular tumor, or a hernia. Testicular torsion is the most important causative disorder to be excluded because any delay in treatment of this condition beyond 6 hours is associated with increased risk of testicular loss and infertility (Box 99-5).

Specific Disorders

Testicular Torsion

Perspective. Most studies suggest that testicular torsion is present in approximately 16 to 42% of patients coming to the ED with scrotal pain. However, this is likely an overestimation resulting from selection bias, as most studies include only patients for whom suspicion of torsion is high, such as those evaluated by a urologist. A more recent retrospective study identified torsion in only 3.2% of all children brought to the ED with scrotal pain. Nevertheless, torsion remains the most common cause of the acute scrotum in prepubertal boys, with a peak incidence in the first year of life. It also has a second peak incidence at puberty, when the rapid increase in testicular volume predisposes the testis to torsion. It can occur in adulthood, as evidenced by a retrospective review of data for 44 patients with torsion, of which 17 were older than 21 years of age. Factors that increase the likelihood of torsion include horizontal lie of the testis, increased length of the spermatic cord in the scrotum, and a history of cryptorchidism. It is interesting to note that low ambient temperature has also been associated with testicular torsion, presumably because it induces contraction of the cremasteric muscles.

Principles of Disease. With torsion, an extravaginal or intravaginal defect of the testis leads to twisting of the spermatic cord and decreased blood supply to the testicle. The cremasteric muscle surrounds the spermatic cord, and in the normal testicle, the testicle does not rotate when the cremasteric muscle contracts. In patients with torsion, however, the anatomic defect allows the tunica vaginalis to insert higher on the testicle, where it encircles the epididymis and distal spermatic cord. When the cremasteric muscle contracts in this abnormal scrotum, there is more room for the testicle to move, and it twists around the spermatic cord. Its movement resembles the ringing of the clapper in a bell—hence the description of a “bell clapper” deformity. By contrast, extravaginal defects occur almost exclusively in neonates and arise because the testicle lies outside of the tunica vaginalis, where it is prone to rotation. Torsion in neonates often is congenital, and by the time the condition is noted at birth, the testicle is not salvageable.

Testicular trauma also has been associated with torsion and has been noted in the literature to occur after motor vehicle collisions, straddle injuries, and athletic injuries. In these instances, the symptoms of torsion often are misattributed to the trauma itself, delaying the diagnosis. As a result, testicular salvage rates are only
40% after groin trauma. To prevent loss of the testicle, scrotal pain persisting for an hour after any traumatic injury should raise suspicion for concomitant torsion of the testis.

As the testicle twists around the spermatic cord in torsion, it initially hinders venous return. Persistent torsion produces arterial obstruction, leading to ischemia and eventual necrosis of the testicle. With increasing degrees of rotation of the cord, ischemia increases, and infarction of the testicle occurs much more rapidly. Unfortunately, no clinical method has been devised for determining the extent of rotation of the testicle and the resultant likelihood of rapid ischemia. The duration of vascular obstruction also affects the ability to salvage the testicle; torsion recognized within 6 hours is associated with testicular salvage rates of 80 to 100%, whereas persistence of symptoms for 24 hours or longer is associated with nearly universal loss of the testicle.

Clinical Features. Patients typically report sudden onset of scrotal pain that awakens them from sleep or develops several hours after physical activity. The pain arises in the scrotum, lower abdomen, or inguinal area. Patients with torsion tend to seek medical attention earlier than those with other causes of acute scrotal pain. However, the mean time of presentation is still 9.5 hours into the disease course, which makes expedient diagnosis and treatment very important for this time-sensitive condition. Up to 29% of patients with testicular torsion describe similar pain in the past, caused by previous intermittent torsion in a predisposed testicle. Patients often report nausea and vomiting or abdominal pain caused by reflex stimulation of the celiac ganglion. Although a history of scrotal trauma reduces a patient’s likelihood of having testicular torsion, approximately 10% of patients with testicular torsion report prior acute blunt trauma to the scrotum. There are no historical factors, including nausea, vomiting, abdominal pain, fever, urinary symptoms, or a history of trauma, that accurately or reliably differentiate torsion from other causative disorders (Table 99-6).

Table 99-6 Differentiation among Causes of the Acute Scrotum

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>TESTICULAR TORSION</th>
<th>APPENDIX TORSION</th>
<th>EPIDIDYMITIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>&lt;1 yr, puberty</td>
<td>7-14 yr</td>
<td>Adult</td>
</tr>
<tr>
<td>Onset</td>
<td>Hours</td>
<td>1-2 days</td>
<td>Days to weeks</td>
</tr>
<tr>
<td>Location of pain</td>
<td>Entire testicle</td>
<td>Upper pole</td>
<td>Epididymis</td>
</tr>
<tr>
<td>Systemic symptoms</td>
<td>Nausea</td>
<td>None</td>
<td>Fever</td>
</tr>
<tr>
<td>Cremasteric reflex</td>
<td>No</td>
<td>Intact</td>
<td>Intact</td>
</tr>
<tr>
<td>Pyuria</td>
<td>Rare</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Ultrasound findings</td>
<td>Diffusely hypoechoic</td>
<td>Symmetrical testicles</td>
<td>Hypoechoic epididymis</td>
</tr>
<tr>
<td></td>
<td>Asymmetrical testicles</td>
<td>Normal flow</td>
<td>Symmetrical testicles</td>
</tr>
<tr>
<td></td>
<td>Normal or decreased flow</td>
<td>Spermatic cord knot</td>
<td>Increased flow</td>
</tr>
<tr>
<td>Treatment</td>
<td>Surgery</td>
<td>Supportive</td>
<td>Antibiotics; prepuberty: supportive only</td>
</tr>
</tbody>
</table>

Note: No single finding in patients with an acute scrotum can reliably differentiate torsion from other causative disorders. When torsion is a diagnostic possibility, prompt urology consultation is mandatory.
as 50% of boys younger than 8 years do not show intratesticular flow. This hypovascularity can result in false-positive diagnoses, which could potentially lead to unnecessary surgical exploration. Comparison with the contralateral testicle can help avoid this misdiagnosis, as in normal patients, blood flow to the two testicles will be similar.

The color Doppler appearance of the testicle depends on the degree of twisting of the spermatic cord. With 180 degrees or less of twisting of the cord, venous flow from the testicle ceases but arterial flow persists. This leads to edema of the testicle on ultrasound that can be misinterpreted as inconsistent with torsion. Although technically more difficult to perform, direct visualization of the spermatic cord for torsion can prevent this misdiagnosis. With torsion, this should reveal a knot in the spermatic cord above the testis, often referred to as a “snail shell” or “whirlpool.”

A study of 919 children found this sign had a sensitivity and specificity close to 100%. In contrast, with more than 180 degrees of twisting of the cord, arterial flow also ceases, leading to a lack of Doppler signal on ultrasound.

Parenchymal echo texture on ultrasound may help predict the viability of the testicle. Homogenous echo texture of the parenchyma has been associated with higher likelihood of testicular salvage. A retrospective review of 25 cases of testicular torsion found a zero recovery rate in testes with heterogeneous echogenicity. In the future, parenchymal appearance may help determine patients who are appropriate candidates for emergent surgery.

Color Doppler ultrasound imaging has the advantage of being an inexpensive, rapid test, readily performed in the ED setting. It is helpful when it demonstrates torsion in cases with equivocal findings on the history and physical examination, but it does not have sufficient sensitivity to rule out a diagnosis of torsion. A recent analyses of 669 scrotal ultrasounds revealed a 98% negative predictive value for torsion. A urologist should evaluate any patient in whom ultrasound findings are negative but history and physical findings are suggestive of torsion. Moreover, ultrasound examination should never delay evaluation by a urologist in any patient with probable torsion.

Radioisotope scanning has the advantage of improved sensitivity compared with ultrasonography, but it is a time-consuming test inappropriate for use in the ED.

Management. The first step in management of very strongly suspected testicular torsion is immediate consultation with a urologist. The longer the spermatic cord remains twisted, the lower the likelihood of testicular salvage. More than 90% of affected testicles can be saved within 6 hours of symptom onset, but by 24 hours, 100% are lost. In addition, early consultation allows the urologist to accompany the patient to ultrasound—if imaging is obtained—where images can be reviewed in real-time with the radiologist. After consultation, intravenous access is established and analgesia is provided either systemically or with a block of the spermatic cord.

If the urologist is not readily available, then manual detorsion should be attempted as follows. The operator stands at the supine patient’s feet and rotates the affected testicle away from the midline, as if turning the pages of a book. If this maneuver is successful, patients should report immediate improvement of symptoms. Most studies suggest that this technique untwists about one fourth of testicles under torsion; other studies, however, have achieved success rates as high as 80%. Some torsions involve rotation through 720 degrees, so continuing the untwisting maneuver past 360 degrees is recommended if no improvement is obtained initially, especially in cases in which torsion is strongly suspected. Analgesics should be administered before this procedure. Regardless of the outcome with manual detorsion, patients still require surgical evaluation. Often urologists will perform bilateral orchiopexy after successful manual detorsion, to prevent recurrence. In addition, evaluation by a urologist should never be delayed to perform this or any other test or maneuver.

Once the diagnosis of testicular torsion has been established, the clinician should determine if emergent removal of the torsed testicle is necessary. Many studies suggest that emergent surgical exploration is unnecessary when symptoms of testicular torsion persist beyond 24 hours because the testicle is not salvageable. Other studies suggest that persistence of symptoms beyond only 10 hours, combined with absence of flow on ultrasound, predicts irreversible testicular necrosis. Unfortunately, in the ED it is difficult to know if torsion is complete, and with incomplete torsion, surgery can achieve salvage beyond 24 hours. In addition, surgical removal of the necrotic testicle reduces pain and the duration of other symptoms. Therefore, emergency clinicians should not rely on the duration of symptoms to determine management; all patients require emergent urologic evaluation.

Disposition. Rapid diagnosis of testicular torsion is essential and should be followed by emergent surgical scrotal exploration and bilateral orchiopexy, if necessary. Loss of the testicle most commonly is a result of delay in seeking medical attention. However, almost 30% of cases of failed testicular salvage have been attributed to misdiagnosis, and another 13% to a delay in treatment after the proper diagnosis was established. Misdiagnosis nearly universally leads to orchiectomy and represents a common source of litigation.

Torsion of Appendages of the Testis

Perspective. A normal scrotum has several vestigial appendages that also can twist and become ischemic, with resultant scrotal pain. This process is most common between 7 and 14 years of age, with a mean age of 10 years. In retrospective analyses, torsion of an appendage rivals testicular torsion as the most common cause of the acute scrotum.

Principles of Disease. The appendix tes-tis, a remnant of the paronephric duct, is present in 92% of patients. It is located on the superior aspect of the testicle between the testis and the epididymis. The appendix epididymis, a remnant of the mesonephric duct, is the second most common testicular appendage, found in approximately 23% of patients. It typically is located on the tip of the epididymis (Fig. 99-10). These appendages are prone to torsion owing to their pedunculated shape. After several days of ischemia from torsion, they will undergo necrosis with eventual reabsorption. Their loss does not permanently affect fertility or have any impact on surrounding structures.

Clinical Features. As with testicular torsion, patients with torsion of an appendage complain of scrotal pain but report milder symptoms with a more gradual onset. These patients usually seek
medical attention in the ED later than patients with testicular torsion, commonly after 48 hours of symptoms. Also in contrast to testicular torsion, the pain often can be localized to one point on the testicle. These patients report nausea, vomiting, urinary symptoms, or previous episodes of similar pain less commonly than patients with testicular torsion.

On physical examination, twisting of the appendix testis leads to formation of a hard, tender, 2- to 3-mm nodule at the upper pole of the testicle. Unlike in testicular torsion, the entire testicle is not tender. The testicle also does not change in overall size, and the scrotum typically does not swell until late in the disease process. The cremasteric reflex typically is intact. On transillumination, the ischemic appendage may appear as a blue dot. Although highly specific for torsion of the appendix, this sign is found in as few as 10% of patients. A reactive hydrocele can form during the course of disease, obscuring the physical findings.

**Diagnostic Strategies.** Urinalysis does not show evidence of infection. On ultrasound imaging, the appendix under torsion will appear hypoechoic. When the appendix then enlarges, the “Mickey Mouse” sign may be apparent on the transverse view, as a result of juxtaposition of the testicle, enlarged appendix, and epididymis. Color Doppler ultrasound can show decreased flow in both normal and torsed appendages. With torsion of the appendix, a hypoechoic spherical nodule with a diameter greater than 5 mm is present over the superior aspect of the testicle.

**Differential Considerations.** Other diagnostic entities such as testicular torsion, epididymitis, and testicular tumor should be considered and excluded before a firm diagnosis of torsion of an appendage can be made.

**Management and Disposition.** If testicular torsion is ruled out, surgical excision of the appendix is rarely necessary. Treatment consists of scrotal support, ice, and NSAIDs. Resolution of symptoms can be expected within 7 to 10 days. Surgical excision is reserved for uncontrollable pain.

**Epididymitis**

**Perspective.** Epididymitis is the most common intrascrotal inflammatory disease. Nearly one half of the cases arise in young men aged 20 to 29 years, but the disease also has affected patients from 4 months to 76 years of age. If untreated, it can lead to orchitis, testicular abcess, and rarely sepsis.

**Principles of Disease.** The epididymis is a tightly coiled tubular area along the posterior aspect of the testes, where sperm mature before their transit to the vas deferens. It becomes infected when organisms travel retrograde from the vas deferens. With infection, the testis may become edematous secondary to passive congestion and inflammation. Resolution of epididymitis typically concludes without sequelae, but rarely, men will develop infertility that persists after bacteriologic cure.

**Pathophysiology.** The particular organisms involved in the infection depend on the sexual activity of the patient. Although the literature classically describes men younger than 35 years of age as prone to *C. trachomatis* and *N. gonorrhoeae* infections, all sexually active men, regardless of age, are at risk for epididymitis from these organisms. *C. trachomatis* is much more common, being identified in 47% of patients younger than 35 years, with *N. gonorrhoeae* seen in 20%. *Ureaplasma* also has been identified in patients with epididymitis, although whether it was the actual causative agent is unclear. In patients with syphilis, infection can spread to the epididymis during the secondary phase. In homosexual men who engage in anal intercourse, coliforms also can lead to sexually transmitted epididymitis.

In men older than 35 years, urinary tract pathogens become the predominant cause of epididymitis. *E. coli* is the most common, occurring in 32 to 55% in this age group. *Pseudomonas aeruginosa* and *Proteus mirabilis* also can infect the epididymis, and although much less common in the industrialized world, *M. tuberculosis* also has been implicated. In immunocompromised patients, fungal and other opportunistic infections have been reported.

Unlike younger patients, older men with epididymitis tend to have urinary tract abnormalities that predispose them to these infections of the epididymis. In one review, over half of men older than 60 years with epididymitis had lower urinary tract obstruction. Older men also are more likely to have undergone recent genitourinary instrumentation, another risk factor for epididymitis. This includes hernia repair, which often can lead to iatrogenic infection of the nearby spermatic cord and epididymis. Acute or chronic prostatitis also may increase the risk of epididymitis. Finally, several authorities have suggested a possible association between indwelling catheters and epididymitis. Each of these factors renders older men prone to epididymitis after they are no longer sexually active.

Epididymitis also occurs in children. Historically, epididymitis was considered uncommon in prepubertal children, but it has been increasingly identified in this age group. In children, epididymitis is most commonly idiopathic, although children can also have congenital genitourinary anomalies that predispose them to recurrent infection. The most commonly associated abnormality is neurogenic bladder, which produces increased pressure during urination and reflex into the ejaculatory ducts. In contrast to older children, in infants, bacterial causes are more common.

Amiodarone also has been implicated as a cause of epididymitis, with nearly 20 case reports in the literature. One study has suggested that amiodarone-induced epididymitis may be more common, arising in as many as 3 to 11% of patients receiving this agent. The effect is dose dependent, typically occurring after at least 4 months of treatment and with doses of 400 mg or more per day. Amiodarone concentrates in the testicle, resulting in lymphocytic infiltration and epididymal fibrosis. Unlike with infectious epididymitis, patients do not have fever, pyuria, or leukocytosis.

**Clinical Features.** Patients with epididymitis experience scrotal pain of gradual onset, prompting them to come to the ED later in the clinical course than patients with torsion, whose symptoms tend to be more abrupt in onset. Initially, this pain may reside in
the lower abdomen or flank, caused by inflammation of the vas deferens. In patients with STDs, urinary symptoms and urethral discharge are found in 10 to 30%. Nearly three fourths of patients report fever. In the early stages of the disease, tenderness is localized to the epididymis, but it quickly spreads to the ipsilateral testicle. Later in the course the scrotum can become edematous, erythematous, and extremely tender. Although Prehn’s sign—decrease in pain with elevation of the scrotum—has been touted as indicative of epididymitis, it has low sensitivity and specificity. Similarly, only 10% of patients with epididymitis from sexually transmitted organisms have urethral discharge on examination. None of these historical factors or physical findings have been shown reliably to differentiate torsion from epididymitis.

**Diagnostic Strategies.** Urinalysis typically demonstrates evidence of infection, with the finding of pyuria in 50 to 93% of patients with epididymitis (defined as the presence of more than four leukocytes per high-power field). If patients are at risk for STD, a urethral swab or urine sample should be obtained to test for C. trachomatis and N. gonorrhoeae; polymerase chain reaction (PCR) and other nucleic acid amplification tests have the greatest sensitivity and specificity. Studies suggest that this diagnostic regimen is underused, with less than 35% of adults with epididymitis undergoing testing for STDs.

The literature also describes epididymal aspiration, but this diagnostic modality has no role in the ED. It is difficult to use, and studies suggest that all patients with positive epididymal cultures have positive urine cultures. Systemic leukocytosis may be present but is a nonspecific finding and does not differentiate epididymitis from torsion. In prepubertal children, urinalysis and urine culture rarely are positive; a retrospective review of 151 children with epididymitis demonstrated bacteruria in only one patient. Nevertheless, in these patients, urine cultures should still be obtained to rule out bacterial infection, because untreated bacterial infections may lead to long-term complications.

In patients with an equivocal presentation for epididymitis versus testicular torsion, testicular ultrasound examination should be performed in the ED as rapidly as possible. As noted earlier, historical and physical examination features and laboratory results cannot reliably distinguish torsion from epididymitis or other diseases. With inflammation, the epididymis appears enlarged and hypoechoic (Fig. 99-11). On color Doppler ultrasound images, the affected testicle will show increased vascularity. These findings are 70% sensitive and 88% specific for epididymitis and can help differentiate the condition from torsion. However, a minority of patients with torsion have preserved flow that can appear similar to epididymitis. In these cases, the presence of a spermatic cord knot, indicative of torsion, should be sought.

Finally, prepubertal children with recurrent epididymitis should undergo renal ultrasound and cystography to identify any potential underlying urinary tract abnormalities. These are important to identify to reduce the risk of future inflammation.

**Differential Considerations.** Three intrascrotal processes are commonly confused with epididymitis: torsion, torsion of the testicular appendage, and tumor of the testicle. Epididymitis is the most common misdiagnosis in patients with torsion, and this error can lead to loss of the testicle.

**Management.** Empirical antibiotics should be selected in accordance with the patient’s age, sexual history, and any previous genitourinary instrumentation (Table 99-7). In patients with a suspected sexually acquired infection, ceftriaxone 250 mg intramuscularly (IM) should be given to treat possible N. gonorrhoeae infection. In conjunction, doxycycline 100 mg PO twice daily for 10 to 14 days should be started to treat C. trachomatis or Ureaplasma urealyticum infection. Although this regimen has never been studied in patients with chlamydial epididymitis, it is presumed to be more successful than the single-dose azithromycin that is used for uncomplicated urethritis. Treatment of sexual partners should be arranged even if the partner’s culture demonstrates no growth because female partners of men with epididymitis often harbor C. trachomatis infection, including partners of men with negative culture results. A recent retrospective review suggests that only 50% of patients 18 to 35 years of age receive appropriate antibiotic treatment for sexually transmitted causes of epididymitis.

In patients with infection by urinary tract pathogens, ciprofloxacin 500 mg PO twice daily has been shown to be the most successful regimen, with 80% of patients cured after 14 days of treatment. Ofl oxacin 200 mg PO twice daily for 14 days is an

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**Figure 99-11.** Ultrasound of the testicle showing an enlarged epididymis and increased blood flow on Doppler ultrasound imaging (white box). (From Blaivas M, Brannam L: Testicular ultrasound. In Rosen C, Wolfe R [eds]: Ultrasound in Emergency Medicine. Emerg Med Clin North Am 22:741, 2004 23 [Figure 23].)

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**Table 99-7** Treatment of Epididymitis

<table>
<thead>
<tr>
<th>Presumed Sexually Acquired Epididymitis</th>
<th>DRUG OF CHOICE</th>
<th>DOSE AND ROUTE</th>
<th>ALTERNATIVE REGIMEN(S)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ceftriaxone</td>
<td>250 mg IM</td>
<td>Ofl oxacin 500 mg PO</td>
<td></td>
</tr>
<tr>
<td>followed by</td>
<td></td>
<td>Ofl oxacin 400 mg PO</td>
<td></td>
</tr>
<tr>
<td>Doxycycline</td>
<td>100 mg PO bid ×10 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>or</td>
<td></td>
<td>Tetracycline 500 mg qid ×10 days</td>
<td></td>
</tr>
<tr>
<td>Presumed Nonsexually Acquired Epididymitis*</td>
<td>Trime thoprim-sulfamethoxazole</td>
<td>One double-strength tablet PO bid ×14 days</td>
<td>Ciprofloxacin 500 mg PO bid ×14 days Ofl oxacin 400 mg PO bid ×14 days</td>
</tr>
<tr>
<td>Prepuberty</td>
<td>Supportive care only</td>
<td>Obtain urine culture and administer antibiotics only if culture positive</td>
<td></td>
</tr>
</tbody>
</table>

*IM, intramuscularly; PO, orally.

*Adjust antibacterial therapy according to results of urine culture.
alternative that also will cover sexually transmitted organisms and therefore is the ideal treatment when it is unclear if a patient has sexually acquired or urinary tract pathogens. Treatment can later be adjusted in accordance with the results of the patient's urine culture. In addition to appropriate antibiotics, bed rest, scrotal support, analgesics, sitz baths, and ice packs may be beneficial. A urologist can block the spermatic cord with bupivacaine to provide pain relief. This maneuver also appears to hasten the healing process, perhaps by increasing blood flow to the spermatic cord that was anesthetized. On discharge the patient should be referred to a urologist for follow-up evaluation within 1 week.

In prepubertal boys in whom an idiopathic cause is suspected, antibiotics are not routinely recommended. This contrasts with prior teaching and practice; previously over 90% of boys received antibiotics. Urine cultures should be obtained for these boys, and only if the cultures reveal bacteria should antibiotic therapy be initiated. This graduated treatment approach is supported by studies showing that most epididymitis in boys is idiopathic. Boys should limit activity, elevate the scrotum with ice packs, and reduce inflammation with NSAIDs. In contrast, infants often have bacterial epididymitis and should be treated empirically with antibiotics pending urine culture results.

Typically, symptoms resolve in 2 weeks in patients with sexually acquired disease and in 4 weeks in patients with urinary tract pathogens. Complications are more common in older men, occurring in 39% of patients, and include intratesticular and epididymal abscess, testicular infarction, and late testicular atrophy. In adolescents, infection often reduces spermatogenesis, but the long-term effect on fertility is unclear. Finally, prepubertal children are at risk for developing testicular abscess, necrosis, and infertility.

Disposition. Patients with systemic signs of toxicity (fever, chills, nausea, vomiting) or complications of acute epididymitis should be hospitalized and treated with parenteral antibiotics. Well-appearing patients with uncomplicated epididymitis can be discharged from the ED and should be reassessed within 1 week to ensure that their symptoms are resolving. Spread of infection, continued scrotal edema, ongoing testicular pain, and scrotal wall fixation are indications that outpatient management has failed, and hospitalization and emergent urologic evaluation are required to rule out an alternate diagnosis such as testicular torsion or scrotal abscess. Prepubertal children with evidence of bacterial infection require outpatient urologic evaluation owing to a high incidence of genitourinary tract abnormalities.

Testicular Tumors

Perspective. Tumor of the testis is the most common malignancy in young men but accounts for only 1% of all cancers in men. These tumors are more common in infertile patients and whites. They also occur with increased frequency in the nondescended and descended testicles of patients with cryptorchidism. A number of simplified classification systems have been proposed for categorizing the different types of testicular tumors. Approximately 95% of tumors are germ cell tumors, with half of these being seminomas and the other half being mixed types, including teratomas, choriocarcinomas, and yolk sac tumors. The other 5% of testicular tumors are sex cord stromal tumors. The disease course will depend on the type of tumor present as well as the age of the patient.

Clinical Features. Unlike in torsion and epididymitis, patients with tumor typically have a painless scrotal mass. When pain develops, it most often is from acute hemorrhage within the tumor pushing against the nonpliable tunica albuginea. Presence of pain should be considered unusual with testicular tumor, and in such cases, torsion and epididymitis should be ruled out before a diagnosis of malignancy is considered. Unlike other painless scrotal masses, such as hydroceles and varicoceles, tumors cannot be separated from the underlying testicle. Palpable tumors are more likely to be malignant compared with tumors identified only with imaging. Patients with tumor also can have symptoms related to metastases from a previously undiagnosed testicular cancer; 15% of patients have metastases to regional lymph nodes at the time of diagnosis, and 5% have metastases to the abdomen or pelvis.

Diagnostic Strategies. All patients with a scrotal mass should undergo a scrotal ultrasound examination. This study can reveal a concomitant hydrocele or a homogeneous hypoechoic lesion. Intratesticular tumors are typically hypervascular with irregular branching vessels. Leydig cell tumors are unique, because they show hypervascularity around the lesion but no internal color Doppler flow. Although helpful for staging purposes, CT scans of the chest and abdomen are necessary in the ED only if the patient has complaints related to these parts of the body. Urinalysis findings typically are normal.

Management. Suspicion of a testicular tumor is an indication for urgent referral to a urologist because radical orchiectomy with high ligation of the spermatic cord may be required. The radiosensitive nature of seminomas renders the combined treatment of orchiectomy and radiation therapy highly successful for early-stage disease. The ultimate treatment strategy, however, should be determined by a urologist. Any patient with systemic symptoms should be admitted to the hospital.

Orchitis

Perspective. Orchitis is a rare acute infection of the testis. It is most common in prepubertal boys, with viral infections such as mumps causing a majority of cases. Orchitis rarely develops in prepubertal boys with mumps but is more common in adolescent males with mumps. It tends to arise several days after the onset of parotitis. Although vaccination has significantly reduced the incidence of mumps infection, sporadic outbreaks have occurred. Infections in vaccinated individuals are increasingly common, presumably resulting from vaccine failure or antigenic differences between the infecting and vaccine strains.

Owing to the testes’ relatively high threshold of resistance to infection, bacterial orchitis more commonly results from local bacterial spread from the epididymis. The most frequent bacterial pathogens are N. gonorrhoeae, C. trachomatis, E. coli, Klebsiella, and P. aeruginosa. These organisms tend to infect postpubertal males and men older than 50 years with benign prostatic hyper trophy (BPH).

Clinical Features. A patient with mumps orchitis has testicular pain and swelling that commonly begins 4 to 6 days after the onset of parotitis, although it can develop in the absence of parotitis. The clinical course varies, with adults having more severe symptoms. Clinical resolution generally occurs in 4 to 5 days. More than 50% of testes involved with mumps orchitis develop atrophy; however, this seldom results in infertility.

Patients with bacterial orchitis have fever and scrotal pain. They often have constitutional signs and symptoms including nausea, vomiting, myalgias, and malaise. The affected testicle (the disease is unilateral in 70% of patients) and the scrotum are swollen, tender, and erythematous.

Diagnostic Strategies. As with all causes of scrotal pain, the first priority is to exclude testicular torsion. If the patient clearly has mumps orchitis based on the clinical presentation and a history of preceding parotitis, then no other tests are necessary. For all other patients, urinalysis and urine culture should be performed. Patients in whom the diagnosis is unclear also should undergo color Doppler ultrasound examination to evaluate for torsion or concomitant epididymitis. On ultrasound, orchitis shows hypervascularity, commonly described as a “testicular inferno.” Blood
tests are typically not helpful, as false negatives are common with serologic testing, particularly in vaccinated individuals.

**Management.** In sexually active patients, ceftriaxone and doxycycline should be used as in epididymitis to cover *N. gonorrhoeae* and *C. trachomatis*. In older patients, fluoroquinolones provide the best coverage of gram-negative organisms. Treatment of viral orchitis is supportive only. Although steroids may improve symptoms, they can reduce testosterone levels. All patients should receive local scrotal care as described for epididymitis. Patients with marked pain, high fever, or constitutional symptoms merit hospitalization and parenteral antibiotics.

**Testicular Trauma**

The most concerning injury associated with trauma involves rupture of the testicle. The presentation can range from a large blood-filled scrotum to minimal swelling with mild pain of the testicle. If there is any concern for rupture, scrotal ultrasound is indicated. Disruption in the echogenic tunica albuginea is 100% sensitive and 65% specific for rupture. Surgical intervention is associated with higher rates of testicular salvage. Hematomas can also form in the testicle without overt rupture. Similar to rupture, these require surgical evaluation unless the tunica albuginea is intact. Trauma can also result in formation of a hematocoele with blood accumulating within the tunica vaginalis. This often leads to ecchymosis of the scrotal wall. Ultrasound can also identify this condition. Lacerations of the scrotum that do not violate the dartos fascia can be repaired in the ED.

**Inguinal Hernia, Acute Hydrocele, Varicocele, and Spermatocele**

Inguinal hernias, hydroceles, varicoceles, and spermatoceles are reasonable considerations in the differential diagnosis of an acute scrotal mass. These clinical entities are typically painless and readily identifiable on physical examination. Most children with inguinal hernias will not have any palpable mass on examination but will report a history of intermittent bulge in the groin that appears with straining or crying. Less commonly an inguinal mass is palpable, and it may extend into the scrotum. If this mass becomes incarcerated, it will be tender, and often the overlying skin will be edematous and erythematous. Children typically will develop irritability, vomiting, or abdominal distention. Incarcerated hernias should be reduced promptly to prevent bowel infarction from strangulation. Reduction can be accomplished by applying gentle pressure to expel the gas and stool in the bowel from the hernia. Pressure is then applied over the distal aspect of the hernia to reduce the bowel. If this technique fails, which is more likely in younger children with prolonged symptoms, then the patient should be placed in the Trendelenburg position, ice applied to the groin, and surgery consulted. After reduction of an incarcerated hernia, children typically require hospitalization and delayed surgical repair. In contrast, acute hydroceles typically are benign, are localized to the scrotum, and transilluminate. Varicoceles are enlarged spermatic cord veins that typically are painless or cause only minimal discomfort. On examination, they are often described as feeling similar to “a bag of worms” just superior to the testicle and decrease in size when the patient is supine. In contrast, a spermatocele is a sperm-containing cyst that is palpated as a nontender mass posterior to the testicle. Ultrasound is diagnostic of both conditions. No emergent treatment is necessary, but patients require outpatient urologic evaluation.

Regardless of the cause of the scrotal swelling, it remains crucial to rule out concomitant pathology by assessing the testicle thoroughly, as each of these conditions is often identified in patients with torsion, epididymitis, and tumors.

**ACUTE URINARY RETENTION**

**Perspective**

Acute urinary retention (AUR) is the sudden inability to pass urine voluntarily. In contrast to patients with anuria, in which the kidneys fail to produce urine, patients with AUR have normal renal function but cannot pass urine and typically have a distended bladder. The lifetime risk of AUR increases with age: 10% of men in their 70s and 33% of men in their 80s have experienced AUR. The retention most commonly is secondary to an obstructive lesion but also can be the presenting manifestation of other pathologic processes (Box 99-6). For example, in women it often develops from infrequent voiding leading to an atomic bladder. In younger patients, it often is suggestive of a serious underlying neurologic disease.

The most common cause of AUR seen in the ED is obstruction of the urinary tract distal to the bladder. In men, BPH is the typical precipitant, causing AUR in 53% of patients. Enlargement of the prostate coupled with constriction of the prostatic urethra from heightened alpha-adrenergic tone obstructs urinary output. Prostate cancer also can block the urethra by means of similar mechanisms, and urinary retention develops in 70% of patients with this cancer. Approximately 25% of men seen in the ED with AUR have prostate cancer, and often this cancer diagnosis was not known beforehand. Strictures of the urethra can arise after trauma from manipulation with a Foley catheter, cystoscopy, previous infections, or radiation therapy. Such strictures may then lead to obstructed urinary outflow. Other, less common obstructive causes of AUR include phimosis (inability to retract the foreskin over the glans penis) and paraphimosis (inability to reduce the foreskin over an edematous glans).

In women, the most frequent causes of obstructive AUR are pelvic masses and prolapse of pelvic organs such as the bladder, rectum, or uterus. These structures compress the urethra, thereby creating AUR. In both sexes, bladder neoplasms, fecal impaction,
GI masses, foreign bodies, or stones can block urine outflow. Finally, congenital posterior urethral valves are the most common source of AUR in children.

Infection also can cause AUR and should be considered in the differential diagnosis, because treatment of the condition through placement of a catheter will correct the retention but not the underlying infection. The most common infectious causative disorder is acute prostatitis, which usually is a result of infection of the prostate by *E. coli* or *P. mirabilis*. The resulting edema causes AUR, particularly in the setting of BPH or other underlying prostatic disease. Similarly, urethritis from a UTI or sexually transmitted infection can obstruct the urethra, and in women vulvovaginitis also can produce enough urethral edema to create AUR. In Elsberg syndrome, genital herpes involving the sacral nerves leads to AUR. Finally, in pediatric patients UTIs can induce sufficient dysuria that the child refuses to void, with consequent urinary retention.

Pharmacologic agents have been associated with AUR. Anticholinergic agents inhibit detrusor muscle contraction, whereas sympathomimetic agents increase α-adrenergic tone in the prostate. In addition, NSAIDs may inhibit prostaglandin-mediated detrusor muscle contraction, leading to a twofold increase in risk of AUR in men taking these drugs.74

Although less common in older patients, neurogenic causes of AUR constitute an important etiologic category. Upper motor neuron lesions create bladder spasticity through a deficit above the micturition center in the sacral cord. They result from spinal cord and cortical lesions caused by multiple sclerosis, trauma, Parkinson's disease, stroke, or neoplasms. Lower motor neuron deficits produce bladder flaccidity with lesions below S1. Such lesions typically are associated with spinal cord tumors, epidural abscesses, spinal trauma, Guillain-Barré syndrome, tabes dorsalis, and multiple sclerosis. Peripheral nerve lesions also can cause AUR. Diabetic peripheral neuropathy is the most commonly encountered peripheral lesion and occurs in 45% of patients with diabetes mellitus.75

Numerous alternate causes for AUR are recognized, as listed in Box 99-6. They include intervertebral disk herniation, recent surgery, and psychogenic causes. In the ED these are encountered much less frequently than the aforementioned disorders. Psychogenic urinary retention also is rare and is a diagnosis of exclusion made only after appropriate studies of bladder function have been performed by a urologist.

### Table 99-8 Presentation and Diagnosis of Acute Urinary Retention

<table>
<thead>
<tr>
<th>CAUSE</th>
<th>HISTORY</th>
<th>PHYSICAL EXAMINATION FINDINGS</th>
<th>DIAGNOSIS*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign prostatic hypertrophy</td>
<td>Frequency, urgency, hesitancy, Prior retention</td>
<td>Enlarged, firm prostate</td>
<td>UA</td>
</tr>
<tr>
<td>Prostate cancer</td>
<td>Frequency, urgency, hesitancy, Previous retention, Constitutional symptoms</td>
<td>Enlarged, firm prostate, Nodular prostate</td>
<td>UA</td>
</tr>
<tr>
<td>Phimosis, paraphimosis</td>
<td>Penile pain</td>
<td>Nonretractable foreskin Edematous penis</td>
<td>Clinical only</td>
</tr>
<tr>
<td>Prostatitis</td>
<td>Dysuria, frequency, urgency, Fever, chills</td>
<td>Warm, tender, boggy prostate, Penile discharge</td>
<td>UA</td>
</tr>
<tr>
<td>Urethritis, vulvovaginitis</td>
<td>Dysuria, frequency, urgency, Itching</td>
<td>Discharge</td>
<td>UA or Urine culture</td>
</tr>
<tr>
<td>Pelvic mass</td>
<td>Pelvic pain pressure</td>
<td>Prolapse of rectum, bladder, uterus</td>
<td>UA Ultrasound imaging, CT</td>
</tr>
<tr>
<td>Neurogenic bladder</td>
<td>Other neurologic complaints</td>
<td>Neurologic deficits</td>
<td>CT, MRI</td>
</tr>
</tbody>
</table>

*In the emergency department setting, each of these diagnoses is made primarily by the history and findings on the physical examination. Additional tests are needed as described.

### Box 99-7 Symptoms of Urinary Retention

#### Obstructive Symptoms
- Urinary hesitancy
- Straining to void
- Decrease in size and force of urinary stream
- Interruption of urinary stream
- Sensation of incomplete emptying
- Previous episode of urinary retention

#### Irritative Symptoms
- Urinary frequency
- Urinary urgency
- Dysuria (usually secondary to infection)
- Nocturia or nocturnal incontinence


### Clinical Features

Although the potential causes of AUR are many, the history and physical examination can considerably narrow the scope of the differential diagnosis (Table 99-8). Patients with AUR report sudden pain and have a distended bladder that is tender to palpation (Box 99-7). With lesions proximal to the bladder, patients typically note pain in the flank, whereas lesions distal to the bladder can produce pain radiating to the scrotum or labia. This pain is often quite severe, leading to significant distress. By contrast, with slowly developing or chronic obstructions, patients typically are older with multiple comorbid conditions; they have overflow incontinence and report little to no pain.

When obstruction is the cause of AUR, the patient often will recall multiple previous episodes of urinary retention. In addition to this history, patients with BPH report frequency, urgency, hesitancy, nocturia, difficulty initiating the urinary stream, decreased force of the stream, a sensation of incomplete voiding, and terminal dribbling. The prostate is enlarged, firm, and non-nodular.
Normal findings on the prostate examination do not exclude BPH. Patients with prostate cancer can have similar symptoms, but these often are accompanied by weight loss, bone pain, and other constitutional signs and symptoms. These patients generally will have an enlarged, nodular prostate. Examination of the penis also is crucial in men because phimosis will reveal edema of the penis with a nonretractable foreskin. In women with obstruction, pelvic pain and pressure are symptoms commonly associated with AUR. A prolapsed bladder, rectum, or uterus and enlarged ovaries or uterus can be identified on pelvic examination.

Patients with an infectious cause for their symptoms often complain of dysuria, frequency, urgency, hematuria, fever, chills, and low back pain. In acute prostatitis these symptoms can be associated with penile discharge and a tender, warm, and boggy prostate. Despite the obstruction, the patient may nevertheless be able to void small amounts of urine. In vulvovaginitis and urethritis, presenting complaints also may include discharge and pruritus.

Patients with a neurogenic cause for AUR may already have a history of neurologic disease. They require a thorough neurologic examination, focusing on strength, sensation, and reflexes in the lower extremities, for which innervation is similar to that of the bladder. The status of the bulbocavernous reflex, anal reflex, sphincter tone, and perineal sensation also should be assessed.

**Diagnostic Strategies**

The only two suggested procedures in the ED for AUR are placement of a Foley catheter and a urinalysis. The urinalysis can reveal infection, whereas presence of hematuria suggests infection, tumor, or calculi. Patients with hematuria require follow-up because the bleeding can arise from prostatic obstruction alone or may have other, more serious causes such as bladder cancer. A basic chemistry panel for assessment of renal function should be obtained only when renal damage or hydronephrosis is a concern. Historically, it was believed that the significant pain associated with AUR prompts patients to come to the ED readily before their creatinine elevates. However, a recent study of patients in the ED with AUR revealed that 24% of patients had an elevated creatinine on presentation and 7% had persistent elevations 1 month later.

Imaging studies are indicated when patients have evidence of concomitant infection or neurologic deficits or when the diagnosis of AUR is unclear. With an equivocal history or physical examination, bedside ultrasound can confirm AUR. Renal and bladder ultrasound studies provide visualization of any obstruction, hydronephrosis, or other causes of upper urinary tract disease. Pelvic ultrasound examination and CT scan evaluate for masses or malignancy causing obstruction. Magnetic resonance imaging of the spine evaluates for disk herniation, cord compression, and cauda equina syndrome. Cystoscopy and retrograde cystourethrogram can depict problems in the lower urinary tract and usually are performed as outpatient procedures.

Several other tests described in the literature provide little benefit in the ED management of patients with AUR. Prostate-specific antigen (PSA) levels frequently are elevated in the setting of AUR, so a PSA assay will not help in differentiating cancer from other causes of retention. Similarly, alternate imaging modalities have rendered IVP unhelpful in the ED.

**Management**

Immediate placement of a 14F to 18F Foley catheter should provide decompression of the bladder. If this fails, placement of an elbowed catheter (coudé catheter) should be attempted; its curved and firm tip allows it to pass more easily across any obstruction. The curved tip of the coudé catheter should point cephalad during insertion. If both of these techniques prove to be unsuccessful, a urologist should be consulted unless the ED personnel are well versed in the use of filiforms, followers, and metal sounds because such instrumentation without the requisite degree of skill may result in serious tissue damage. In addition, if a patient recently underwent urologic surgery, urethral catheterization should not be performed.

When immediate bladder decompression is required and a urologist is not available or major urethral trauma is present, suprapubic bladder drainage should be performed. With use of ultrasound guidance and sterile technique, a 22-gauge spinal needle should be inserted two fingerbreadths above the symphysis pubis in the midline and directed toward the anus. This needle can be used to administer local anesthetic and should be advanced until urine is aspirated. Return of air suggests that the needle is in the bowel, and the needle should be retracted and moved more cephalad. Once the bladder has been located, the needle is left in place and the syringe is removed. A guidewire is then passed through the needle into the bladder, followed by removal of the needle. A 1-cm skin incision is made, and a dilator and sheath are inserted into the bladder. The guidewire and dilator are removed, and a Foley catheter can then be passed through the sheath. The Foley balloon is inflated and urine drained (Fig. 99-12). Alternatively, an open technique with a trocar can be used. Finally, bladder aspiration without placement of a catheter can also be performed as a temporizing measure pending urology consultation.

Historically, it has been recommended that initial drainage be limited to 1 L to prevent postobstructive diuresis and hypotension. Such postobstructive diuresis is uncommon after AUR. Moreover, limited drainage of the bladder can increase the risk of UTI. Therefore, complete drainage should be performed.

![Figure 99-12. Cystostomy tube placement. A. Anesthetizing trocar tract. After a skin wheal (a) is raised, the suprapubic tract for the trocar is anesthetized, including the rectus fascia (b). Anesthetizing until the bladder is penetrated will ensure total comfort for the patient during insertion of the trocar. B. Needle localization of the bladder. A spinal needle may be used even during insertion of the trocar to locate the bladder. C. Trocar position. The trocar is advanced until its sheath, as well as the point, is fully in the bladder. D. Tubing position. Enough tubing is inserted that it will not pull out of the bladder when the bladder empties. (From Robert JR, Hedges JR: Clinical Procedures in Emergency Medicine. Philadelphia, WB Saunders, 1985.)](image-url)
Complications

Catheterization has been associated with urethritis, cystitis, prostatitis, bacteremia, and sepsis. These infections are most common in patients with indwelling catheters and elderly debilitated female patients. For each day a catheter remains in place, there is an additional 4% risk of bacterial colonization of the bladder. Placement of a catheter also has been reported to cause postobstructive diuresis and hematuria. Such problems are believed to be related to rapid bladder decompression, so historically, gradual decompression has been recommended to prevent complications such as hematuria, hypotension, and postobstructive diuresis. Although hematuria occurs in 2 to 16% of patients after rapid bladder emptying, no major consequences from this bleeding have been documented.72 Similarly, postobstructive diuresis can cause transient hypotension, but it is responsive to fluid administration. The side effects of rapid bladder decompression are rare; therefore all patients with AUR should undergo rapid, complete decompression of the bladder.

Rarely, chronic obstruction may lead to development of a salt-wasting nephropathy that requires treatment. The patient’s urine output should be observed for 4 hours after catheterization. If output is greater than 200 mL/hour, the patient should be hospitalized for monitoring of blood pressure and sodium concentration.

Disposition

After bladder drainage, healthy and reliable patients can be safely discharged from the ED with an indwelling catheter and education about catheter management. Follow-up with a urologist should be arranged before the patient leaves. Patients with concomitant infection, significant comorbid illnesses, impaired renal function, neurologic deficits, or complications from catheterization require emergent urology consultation and probably admission.

Although the catheter is an inconvenience for the patient, and chronic use has been associated with UTI, trauma, stones, and urethral strictures, early removal of the catheter also is associated with heightened risk for recurrence of AUR. In a study of patients with suspected BPH, if the bladder was drained and a catheter was not left in place, recurrent AUR developed in 70%.74 In patients in whom the catheter remained in place for 3 days, AUR developed in 49%, and when the catheter was in place for 7 days, AUR recurred in only 38% of patients.79

Studies also suggest that initiation of an alpha-adrenergic blocker, such as tamsulosin, at the time of catheter insertion improves the likelihood of spontaneous voiding after catheter removal. In addition, a recent Cochrane review suggests that alpha-blockers may also improve the likelihood that a patient will not require ongoing catheter placement; 48 to 62% of patients on alpha-blockers successfully completed a trial without a catheter, compared with 23 to 28% of patients not treated with alpha-blockers.80 These medications should be provided only after consultation with the urologist or primary care physician because they increase the risk of orthostatic hypotension, particularly in the elderly. 5-Alpha-reductase inhibitors, another medication typically used for BPH, have not been shown to reduce recurrence of AUR.81

Prophylactic antibiotic therapy is not recommended in patients with AUR because although bacteruria often develops in patients with indwelling catheters, it typically is asymptomatic. Antibiotics only promote resistance among these organisms.

Definitive therapy often requires surgical correction of any underlying obstruction. This should not be performed in the acute setting, as early surgery is associated with increased morbidity.

HEMATURIA

Perspective

Although hematuria is commonly encountered in the ED, the specific underlying disorder often is elusive. Most cases seen in the ED result from detection of asymptomatic microscopic hematuria, as an incidental finding noted on routine examination through microscopic or dipstick analysis of the urine. The bleeding typically is transient and not indicative of serious underlying pathology. After an uneventful workup, patients often are referred for outpatient evaluation; however, the cause of the hematuria remains unknown in 61% of patients evaluated with laboratory and imaging studies.82 Less commonly, patients come to the ED complaining of gross blood in their urine. Unlike microscopic hematuria, gross blood in the urine often is the presenting symptom of an underlying malignancy. The risk of malignancy is greater in older patients; in those older than 60 years, gross hematuria has a positive predictive value for malignancy of 22.1% in men and 8.3% in women.83 Regardless of age or visibility of blood in the urine, patients with hematuria require evaluation in the ED to rule out life-threatening diagnoses such as malignancy and abdominal aortic aneurysm.

Principles of Disease

Blood in the urine can be gross or microscopic. As little as 1 mL of whole blood in 1 L of urine can produce gross hematuria, turning the urine red. Multiple other substances and reactions can turn the urine red, and centrifugation of the urine and microscopic analysis differentiate these false positives from true hematuria. After centrifugation, the red color persists only in the urine sediment with hematuria. By contrast, a red supernatant appears bloody but contains no RBCs on microscopic analysis, and the cause typically is a benign condition (Box 99-8). The supernatant should be tested for heme, and if positive suggests hemoglobinuria or myoglobinuria. The supernatant will then contain heme in addition to a red coloring agent.

Microscopic hematuria is the presence of more than three to four RBCs per high-power field in clear urine. The particular cutoff for number of RBCs per high-power field used is arbitrary. Lower numbers of RBCs per high-power field decrease specificity but increase sensitivity, because significant disease can occur with as little as one RBC per high-power field. Urine dipstick testing increases sensitivity to 91 to 100% for the presence of blood, detecting one or two RBCs per high-power field. A negative urine dipstick rules out the presence of hematuria, and urine microscopy is then not necessary. However, the dipstick is less specific, with false positives occurring when sperm are present and with alkaline or extremely concentrated urine.84 Accordingly, a positive result on urine dipstick testing should trigger at least reflex confirmation with microscopic analysis.

Bleeding from anywhere along the genitourinary tract can produce hematuria. In both the upper and lower portions of the urinary tract, infection, trauma, and renal calculi are the most

<table>
<thead>
<tr>
<th>Box 99-8</th>
<th>Etiology of Red-Colored Urine without Hematuria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenazopyridine</td>
<td>Bromide</td>
</tr>
<tr>
<td>Nitrofurantoin</td>
<td>Food coloring</td>
</tr>
<tr>
<td>Rifampin</td>
<td>Beets</td>
</tr>
<tr>
<td>Chloroquine</td>
<td>Berries</td>
</tr>
<tr>
<td>Hydroxychloroquine</td>
<td>Rhubarb</td>
</tr>
<tr>
<td>Iodine</td>
<td></td>
</tr>
</tbody>
</table>
common causative disorders. Patients also can have more serious causes of hematuria, such as malignancy or vascular lesions (e.g., abdominal aortic aneurysm), and these diagnoses should be excluded. Unfortunately, hematuria has poor specificity for malignancy; an analysis of 156,691 adults with hematuria revealed malignancy in only 0.7% of patients. Malignancy was more common in patients older than 40 years (odds ratio [OR] 17), male patients (OR 4.8), and patients who had more than 25 RBCs per high-power field (OR 4.0). Other, less common risk factors for malignancy include cigarette smoking, occupational exposure to chemicals such as aniline dye and benzidine, and excessive use of analgesics. When another cause for the hematuria, such as UTI or stone, is identified in the ED, close follow-up is still necessary because patients with these risk factors often prove to have an underlying malignancy. In the upper urinary tract, the glomerulus is the most frequent source of bleeding, which may result from glomerulonephritis, immunoglobulin A (IgA) nephropathy, or nephritis. The common causes of hematuria are further divided by patient age and gender in Box 99-9.

Occasionally, hematuria also has been attributed to warfarin use, BPH, and exercise. Supratherapeutic anticoagulant therapy can lead to blood in the urine, but therapeutic anticoagulation does not produce spontaneous hematuria. The incidence of hematuria was not significantly different between controls and 243 patients followed prospectively while receiving warfarin. Similarly, BPH can lead to increased vascularity of the prostate but does not increase the risk of hematuria. High-intensity exercise also can produce hematuria. This bleeding typically is transient and clinically inconsequential. However, a concomitant genitourinary tract lesion can be present, particularly if the bleeding persists for more than 72 hours after exercise. Because warfarin use, BPH, and exercise do not directly cause persistent hematuria, patients in whom these factors are present require evaluation identical to that for other patients.

<table>
<thead>
<tr>
<th><strong>Age 0-20 Years</strong></th>
<th><strong>Acute UTI</strong></th>
<th>Irritation of urethral meatus</th>
<th>Acute glomerulonephritis</th>
<th>Congenital urinary tract anomalies with obstruction</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age 20-40 Years</strong></td>
<td><strong>Acute UTI</strong></td>
<td>Bladder cancer</td>
<td>Urolithiasis</td>
<td></td>
</tr>
<tr>
<td><strong>Age 40-60 Years</strong></td>
<td><strong>Women</strong></td>
<td>Acute UTI</td>
<td>Bladder cancer</td>
<td>Urolithiasis</td>
</tr>
<tr>
<td><strong>Men</strong></td>
<td>Acute UTI</td>
<td>Bladder cancer</td>
<td>Urolithiasis</td>
<td></td>
</tr>
<tr>
<td><strong>Age 60 Years and Older</strong></td>
<td><strong>Women</strong></td>
<td>Acute UTI</td>
<td>Bladder cancer</td>
<td></td>
</tr>
<tr>
<td><strong>Men</strong></td>
<td>Acute UTI</td>
<td>Benign prostatic hyperplasia</td>
<td>Bladder cancer</td>
<td></td>
</tr>
</tbody>
</table>

**UTI**, urinary tract infection.

In children, gross hematuria typically arises from UTI or benign causes such as irritation of the urethral meatus or hypercalciuria without nephrolithiasis. However, when accompanied by trauma, edema, or hypertension, more serious causes should be considered such as rhabdomyolysis, tumors, glomerulonephritis, and hemolytic uremic syndrome.

**Clinical Features**

The timing and appearance of the blood in the urine can help narrow the scope of the differential diagnosis (Table 99-9). Repeated episodes of bleeding during and after menstruation in women suggests endometriosis of the urinary tract. Gross hematuria suggests lesions in the lower urinary tract. Passage of clots indicates a source below the kidney. Finally, passage of a large amount of congealed blood in the urine suggests malignancy of the kidney. Several other historical factors also are associated with particular causes of hematuria (Box 99-10). A careful history will identify a benign cause for hematuria, such as menstruation, recent heavy exercise, or sexual activity, as well as the use of agents that can produce red urine without blood (see Box 99-8). Patients may report frequency, urgency, and dysuria in the setting of infection. They may note flank pain with urolithiasis or pyelonephritis. IgA nephropathy arises days after a viral respiratory infection, whereas poststreptococcal glomerulonephritis develops in children 1 to 2 weeks after skin or throat infections.

The physical examination is crucial in establishing the diagnosis. Hypertension occurs with glomerulosclerosis and, in the setting of peripheral edema, suggests nephrotic syndrome. Atrial fibrillation increases the likelihood of embolic disease and renal infarction. An abdominal bruit may be caused by an arteriovenous fistula, whereas a palpable abdominal mass may represent an abdominal aortic aneurysm. Flank pain and tenderness can arise with pyelonephritis or nephrolithiasis. The external genital examination can show evidence of trauma or a tumor, and in children often a reveals a rectal or vaginal source for the bleeding, such as urethral prolapse. It is also important to assess for hypertension and edema in children, which can arise with nephritis.

**Diagnostic Strategies**

A clean-catch or catheterized urine specimen should be obtained in all patients with hematuria. Catheterization itself induces hematuria in approximately 15% of patients, but the amount of bleeding is inconsequential, rarely exceeding three RBCs per high-power field. Bedside urine dipstick testing of this urine should be performed. If positive for blood, then urine microscopy should be performed. Microscopy will reveal WBCs in addition to RBCs in the presence of infection. Proteinuria and RBC casts are seen with glomerular disease, and the urine is often brown or cola colored. Patients with these disorders do not require further
to a nephrologist. The exception is glomerular disease in children, evaluation in the ED beyond the urinalysis and should be referred diseases of the upper urinary tract. It is important to identify the cause for the hematuria, a CT scan with contrast or a renal ultrasound study. If findings fail to suggest a particular disorder as a result of hematuria in trauma, but its use now should be limited to unstable patients in whom CT is impractical.

CT is the appropriate imaging modality for both adults and children with traumatic hematuria. Its sensitivity and specificity exceed those of ultrasound. The exact level of hematuria that should trigger imaging is unclear, but it appears that patients without gross hematuria or evidence of coexisting abdominal or pelvic injuries are unlikely to have clinically significant injuries on

**Medical History for Patients with Hematuria**

- Exclude pseudo-hematuria—drugs, vegetable dyes, pigments
- Factitious—Munchausen syndrome, narcotic-seeking behavior
- Bleeding diathesis
- Clots—indicate nonglomerular bleeding; large, thick clots (bladder); small, stringy clots (upper tract)
- Gross hematuria—relationship to exercise, infection
- Relationship of gross hematuria to urinary tract—initial (urethra distal to the urogenital diaphragm), total (bladder proper or upper urinary tract), terminal (bladder neck or prostatic urethra)
- Painful hematuria—urinary tract infection or calculus, papillary necrosis, passage of clots, obstruction, loin pain—hematuria syndrome, glomerulonephritis
- Genitourinary history—flank trauma or pain frequency; nocturia; dysuria; previous stones, tissue passage, or infections; vaginal or penile discharge; sexual activity; presence of urinary catheter
- Relationship to menstruation—endometriosis
- Sickle cell disease or trait
- Medications
- Systemic symptoms—fever, rash, joint pain, weight loss
- Infectious cause—night sweats, sore throat, impetigo, recent tooth extraction or other invasive procedure, diarrhea, travel to areas endemic for Schistosoma haematobium
- Risk factors for urologic cancer—age older than 40 years, tobacco use, analgesic abuse, pelvic irradiation, cyclophosphamide, S. haematobium, occupational exposure to dyestuffs and rubber compounds
- Family history—hematuria, renal disease, sickle cell disease, deafness, bleeding diathesis
- Previous testing—blood pressure, urinalysis, serum chemistries, intravenous pyelogram
- Pregnancies—proteinuria, hypertension (with month of onset)

**Risk Factors Requiring Further Evaluation of Hematuria**

**Adults**
- Age older than 40 years
- Cigarette smoking
- Occupational exposure
- Analgesic abuse
- Persistent hematuria
- Abdominal pain
- Trauma

**Children**
- Trauma
- Hypertension
- Edema
- Proteinuria (>2+ on dipstick)
- Signs of coagulopathy
- Flank or abdominal pain

**KEY CONCEPTS**

- Acute scrotal pain should be considered a result of testicular torsion until proven otherwise.
- No historical or physical examination findings accurately and reliably exclude testicular torsion, and therefore patients with concern for this diagnosis should undergo a scrotal ultrasound.
- Sexually active males should receive ceftriaxone and doxycycline to treat epididymitis, whereas in young children, antibiotics are not typically required.
- Scrotal pain or swelling after trauma warrants a scrotal ultrasound to evaluate for testicular rupture.
- Patients with acute urinary retention and concomitant infection or neurologic deficits warrant imaging in the ED.
- Patients with acute urinary retention should have complete drainage of the bladder performed via catheterization or, if this is not possible, by suprapubic bladder aspiration.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.


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