Acute bacterial prostatitis

Authors: Alain Meyrier, MD, Thomas Fekete, MD
Section Editor: Stephen B Calderwood, MD
Deputy Editor: Allyson Bloom, MD

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Literature review current through: Aug 2022. | **This topic last updated:** Oct 25, 2021.

INTRODUCTION

The prostate is subject to various inflammatory disorders [1]. One of these syndromes is acute bacterial prostatitis, an acute infection of the prostate, usually caused by gram-negative organisms [2]. The clinical presentation is generally well defined, and antimicrobial therapy remains the mainstay of treatment [3].

Acute bacterial prostatitis will be reviewed here. Chronic bacterial prostatitis and chronic prostatitis/chronic pelvic pain syndrome are discussed in detail elsewhere. (See "Chronic bacterial prostatitis" and "Chronic prostatitis and chronic pelvic pain syndrome".)

Other causes of dysuria in men, including cystitis, urethritis, and epididymitis, are also discussed elsewhere. (See "Acute simple cystitis in adult males" and "Urethritis in adult males" and "Acute scrotal pain in adults", section on 'Acute epididymitis or epididymo-orchitis'.)

PATHOGENESIS

Entry of microorganisms into the prostate gland almost always occurs via the urethra. In most cases, bacteria migrate from the urethra or bladder through the prostatic ducts, with intraprostatic reflux of urine (figure 1). As a result, there may be concomitant infection in the bladder or epididymis. Uropathogenic bacterial isolates that cause prostatitis may have a higher accumulation of specialized virulence factors than those involved in cystitis alone [4,5].

Acute prostatitis can also occur from direct inoculation after transrectal prostate biopsy and transurethral manipulations (eg, catheterization and cystoscopy) [6,7]. (See "Prostate biopsy", section on 'Infection'.)

EPIDEMIOLOGY

Overall, prostatitis syndromes are a very common presentation in the clinical setting and tend to occur in young and middle-aged men [3,8]. However, acute bacterial prostatitis accounts for a minority of these cases.

In a study of 58,955 ambulatory visits to clinicians by men over the age of 18 years reported to the United States National Ambulatory Medical Care Surveys from 1990 to 1994, 5 percent listed genitourinary tract symptoms as one of the reasons for the visit [9]. Prostatitis was listed as a diagnosis in an estimated two million visits annually. Acute bacterial prostatitis, however, accounted for only 4 percent of prostatitis diagnoses.

Risk factors — Acute prostatitis can occur in the setting of cystitis, urethritis, or other urogenital tract infections. Thus, underlying conditions such as functional or anatomical anomalies (eg, urethral strictures), that predispose to other urogenital infections can increase the risk of prostatitis.

Prostate infections following urogenital instrumentation, including chronic indwelling bladder catheterization, intermittent bladder catheterization, and prostate biopsy are well documented [10-12]. As an example, in a retrospective study of 1339 men who underwent transrectal ultrasound-guided prostate biopsy, 28 men (2.1 percent) developed acute prostatitis a mean of three days post-biopsy, despite receiving periprocedural fluoroquinolone prophylaxis [12]. In another study, infection with drug-resistant bacteria was more common in prostatitis following transrectal prostate biopsy compared with spontaneous acute bacterial prostatitis [13]. Periprocedural antibiotic prophylaxis prior to prostate biopsy is generally recommended [14]. (See "Prostate biopsy", section on 'Prophylactic antibiotics'.)

Lower urinary tract symptoms, including those caused by bacterial prostatitis, also occur more frequently in patients with HIV infection than in the general population [15,16]. Why this occurs is not clear. While immunosuppression associated with HIV certainly increases the risk of prostatitis, HIV-infected men without AIDS-defining illnesses or low CD4 cell counts also report lower urinary tract symptoms more frequently than uninfected patients [15].

Many patients with acute bacterial prostatitis have no clear risk factors. Anecdotally, trauma (eg, bicycle or horseback riding), dehydration, and sexual abstinence have been thought to

predispose to prostatitis. However, these factors have not been established by well-controlled studies.

MICROBIOLOGY

The pathogens associated with acute prostatitis reflect the spectrum of organisms causing cystitis, urethritis, and deeper genital tract infections (such as epididymitis). Thus, gram-negative infections, especially with Enterobacterales, are the most common [17]. Of Enterobacterales, *Escherichia coli* is the most typical, followed by *Proteus* species. As examples, in retrospective studies of men with acute bacterial prostatitis, such pathogens have been identified in positive urine cultures at the following frequencies [6,7,18-21]:

- *E. coli* – 58 to 88 percent
- *Proteus* species – 3 to 6 percent
- Other Enterobacterales (*Klebsiella*, *Enterobacter*, and *Serratia* species) – 3 to 11 percent
- *Pseudomonas aeruginosa* – 3 to 7 percent

Certain gram-positive cocci (including *Staphylococcus aureus*, streptococci, and enterococci) have also been implicated in acute bacterial prostatitis [18,19,22]. Acute *staphylococcal prostatitis*, in particular, may result from bacteremia that accompanies a remote *S. aureus* infection elsewhere. Isolation of *S. aureus* from prostatic secretions should trigger an evaluation for a remote or endovascular staphylococcal infection.

Instrumentation of the prostate has been associated with subsequent acute bacterial prostatitis due to organisms with broad resistance to antibiotics, including fluoroquinolone-resistant *E. coli* and *Pseudomonas aeruginosa* [11,18]. This is likely related to the use of periprocedural prophylactic fluoroquinolones.

Sexually active men may have sexually transmitted urogenital infections, such as urethritis and epididymitis, which also acutely involve the prostate, in which case *Neisseria gonorrhoeae* and *Chlamydia trachomatis* are important pathogens. (See "Urethritis in adult males".) This problem tends to occur more often in younger men, but age is not a specific risk factor for these sexually transmitted infections.

In HIV-infected patients, the microbiology typically reflects that seen in the general population; however, involvement with other pathogens, including *Salmonella typhi* and *N. gonorrhoeae*, has also been described [16].

In patients or travelers from regions where *Burkholderia pseudomallei* is endemic (eg, southern and southeast Asia or northern Australia) this uncommon organism has also been described as a cause of acute prostatitis and prostatic abscess [23,24]. (See "Melioidosis: Epidemiology, clinical manifestations, and diagnosis".)

Coccidioides fungal prostatitis has been observed in hosts receiving immunosuppressive therapy [25].

Recurrent infection after completion of therapy is usually caused by the same organism that was found in the original infection [26].

CLINICAL MANIFESTATIONS

The clinical presentation of acute prostatitis is generally not subtle. Patients are typically acutely ill, with spiking fever, chills, malaise, myalgia, dysuria, irritative urinary symptoms (frequency, urgency, urge incontinence), pelvic or perineal pain, and cloudy urine. Men may also complain of pain at the tip of the penis. Swelling of the acutely inflamed prostate can cause voiding symptoms, ranging from dribbling and hesitancy to acute urinary retention. In a retrospective review of 614 cases of men who presented to an emergency department in Spain and were diagnosed with acute bacterial prostatitis, irritative symptoms were observed most commonly (93 percent), with obstructive symptoms (poor stream, hesitancy) and fever reported in 25 and 34 percent of patients, respectively [18]. No specific characteristics of urinary symptoms (eg, end-stream dysuria) have been clearly associated with prostatitis.

Rarely, patients lack these local symptoms and present instead with constitutional symptoms or a flu-like illness.

On exam, the prostate is often firm, edematous, and exquisitely tender. Common laboratory findings include peripheral leukocytosis, pyuria, bacteriuria, and, occasionally, positive blood cultures. Inflammatory markers (erythrocyte sedimentation rate, C-reactive protein) are elevated in most cases. Inflammation of the prostate can also lead to an elevated serum prostate specific antigen (PSA) level [27]. Thus, if serum PSA testing for prostate cancer screening is planned, it should be deferred for one month following resolution of acute prostatitis. A detailed discussion of the risks and benefits of prostate cancer screening is found elsewhere. (See "Measurement of prostate-specific antigen".)

Complications — Complications of acute bacterial prostatitis include bacteremia, epididymitis, chronic bacterial prostatitis, prostatic abscesses, and metastatic infection (eg, spinal or sacroiliac infection) [28]. Patients with underlying valvular heart disease or a valvular prosthesis

are at risk for endocarditis when prostatitis is caused by certain bacterial pathogens, particularly, but not exclusively, gram-positive bacteria. These complications are more likely to occur if diagnosis and antimicrobial therapy is delayed.

Prostatic abscess — The incidence of prostatic abscess is currently low with the use of appropriate antibiotic therapy [29]. In a prospective study of transrectal ultrasonography of the prostate in 45 men hospitalized for acute bacterial prostatitis, no lesions with sonographic characteristics of prostatic abscesses were identified, despite detection of other, often transient, prostatic lesions in almost half of the men [30]. Certain underlying conditions, such as diabetes mellitus or HIV-related immunosuppression, may predispose to the development of prostatic abscesses [31,32].

Signs and symptoms of a prostatic abscess are similar to those of bacterial prostatitis in general, but may persist despite appropriate antibiotic therapy; in addition, fluctuance of the prostate on gentle digital exam can suggest an underlying abscess. On transrectal ultrasound, abscesses appear as hypoechoic or anechoic areas with thick walls or peripheral edema [32,33]. Computed tomography (CT) findings include nonenhancing fluid-density collections that can be multiseptated or rim-enhancing lesions.

DIAGNOSIS

The presence of typical symptoms of prostatitis should prompt digital rectal exam, and the finding of an edematous and tender prostate on physical exam in this setting usually establishes the diagnosis of acute bacterial prostatitis. Digital rectal examination should be performed gently; vigorous prostate massage should be avoided since it is uncomfortable, allows no additional diagnostic or therapeutic benefit, and increases the risk for bacteremia. In patients who present with constitutional symptoms only, establishing a diagnosis of acute prostatitis is challenging. Laboratory findings of leukocytosis, pyuria, bacteriuria, or an elevated serum prostate specific antigen (PSA) level can support the diagnosis, and should prompt consideration of digital rectal exam. (See 'Clinical manifestations' above.)

In order to establish the microbial etiology, a urine Gram stain and culture should be obtained in all men suspected of having acute prostatitis. Gram stain of the urine, if positive, can be used as a guide to initial therapy (see 'Antimicrobial therapy' below). Urine culture typically reveals a causative organism in acute prostatitis, unless antibiotics were recently used.

Blood cultures usually are not necessary for microbial diagnosis, and we typically do not perform them for this reason alone. In a retrospective review of 261 men with acute prostatitis

in whom urine and blood cultures were collected, blood cultures provided additional microbial information in 14 patients (5 percent) [20]. However, blood cultures are useful to assess for complications in patients with underlying cardiac valvular disease or clinical evidence of impending or severe sepsis.

EVALUATION

Evaluation for complications — Blood cultures to evaluate for bacteremia are warranted in patients with signs suggestive of severe sepsis (eg, hypotension, hematologic derangements). Additionally, a high level of suspicion for bacteremic complications of acute prostatitis is warranted in patients with underlying conditions that may predispose to them (eg, endocarditis in patients with valvular disease or prostheses).

In particular, when *S. aureus* is recovered from a urine culture, it is important to perform blood cultures to evaluate whether the bacteriuria reflects seeding of the prostate or urine from bacteremia. (See "Clinical manifestations of Staphylococcus aureus infection in adults".)

Imaging studies are generally not indicated in acute bacterial prostatitis, unless there is clinical suspicion for a prostatic abscess. In patients who have persistent clinical or laboratory abnormalities despite appropriate antimicrobial therapy, an abscess can be diagnosed radiographically with prostate ultrasonography or computed tomography (CT) scan [34]. (See 'Complications' above.)

Evaluation for anatomical abnormalities — Following the acute episode, underlying anatomical abnormalities that may have predisposed to an acute prostatic infection may be sought and, if possible, remedied to decrease the risk of recurrence. Consultation with a urologist for this evaluation may be useful in patients with recurrent infections.

DIFFERENTIAL DIAGNOSIS

The most common alternate diagnosis to consider in a man who presents with dysuria, frequency, and/or urgency, and who has pyuria and bacteriuria, is an isolated lower urinary tract infection (UTI), or cystitis. UTIs in men generally occur in the presence of a predisposing functional or anatomic abnormality, such as prostatic hypertrophy or genitourinary instrumentation, which increases the risk of infection, although they can also uncommonly occur in otherwise healthy men. While isolated UTIs likely involve some amount of bacterial contamination along the prostatic ducts, this prostatic contamination may remain superficial without overt prostatic inflammation or suppuration. In such cases, fever, chills, and

constitutional symptoms are generally absent, and there is no prostatic tenderness on digital rectal exam. Men who lack this evidence of clinically significant prostatic involvement can be managed as having acute simple cystitis but should be monitored for lack of clinical response, which would warrant reevaluation for the possibility of underlying prostatitis. (See "Acute simple cystitis in adult males", section on 'Treatment'.)

Tenderness of the prostate on exam is also generally not found in other acute causes of dysuria (eg, urethritis, epididymitis) in the absence of coexisting prostatitis. (See "Approach to infectious causes of dysuria in the adult man".)

Patients with conditions, such as benign prostatic hyperplasia and overactive bladder, may also initially present with lower urinary tract symptoms, but typically do not have fever, pyuria, and other signs or symptoms of infection. These conditions are discussed in detail elsewhere. (See "Clinical manifestations and diagnostic evaluation of benign prostatic hyperplasia" and "Lower urinary tract symptoms in males".)

MANAGEMENT

Treatment of acute prostatitis includes antimicrobial therapy and supportive measures to reduce symptoms. Rarely, more invasive intervention is indicated to manage complications.

Indications for hospitalization — Not all patients with acute bacterial prostatitis warrant inpatient hospitalization. Patients who have no major comorbidities, no signs or symptoms of severe sepsis, and who can reliably take and tolerate oral antibiotics, can likely be managed appropriately in the outpatient setting. A short hospital stay in patients suspected of having bacteremia, or for monitoring purposes in those patients who would not otherwise be able to promptly return to medical care in the case of decompensation, may be prudent. Acute urinary retention should also warrant very close observation or hospitalization for bladder catheterization.

Antimicrobial therapy

Regimen selection — A variety of antimicrobials may be used for the treatment of acute prostatitis, which should be treated empirically pending culture results. Data on the treatment of acute prostatitis are limited, and there are no comparative trials evaluating the optimal antimicrobial choice. Thus, recommendations for empiric therapy are based on the likelihood of the infecting organism. Although not all antibiotics can penetrate into prostatic tissue, the presence of acute inflammation generally allows entry of drugs that would not otherwise

achieve therapeutic levels. (See "Chronic bacterial prostatitis", section on 'Antimicrobial penetration into prostatic tissue'.)

Empiric antibiotic therapy should adequately treat gram-negative organisms unless a urine Gram stain is available and suggests an alternate bacterial cause. For patients with acute prostatitis who can take oral medications, we suggest trimethoprim-sulfamethoxazole (one double-strength tab orally every 12 hours) or a fluoroquinolone (ciprofloxacin 500 mg orally every 12 hours or levofloxacin 500 mg orally once daily) as empiric therapy. We typically choose one of these antimicrobial agents because they achieve high levels in prostatic tissue. Although this may not be an issue in the acute setting, where prostatic inflammation allows penetration of a broader range of antibiotics, the ability of an antibiotic to penetrate prostate tissue is thought to be important during prolonged therapy, while inflammation is resolving [35]. The choice between these should take into account patient tolerance and regional patterns of Enterobacteriaceae drug resistance. (See "Acute simple cystitis in women", section on 'Resistance trends in E. coli'.)

Men younger than 35 years who are sexually active and men older than 35 years who engage in high-risk sexual behavior should be treated with regimens that cover *N. gonorrhoeae* and *C. trachomatis* [35]. (See "Treatment of uncomplicated Neisseria gonorrhoeae infections", section on 'Preferred regimen' and "Treatment of Chlamydia trachomatis infection", section on 'Doxycycline as preferred agent'.)

Some patients with acute bacterial prostatitis may need to be hospitalized for parenteral antibiotic therapy if they cannot tolerate oral medication, demonstrate signs of severe sepsis, or have bacteremia. In such cases, intravenous levofloxacin or ciprofloxacin may be given with or without an aminoglycoside (gentamicin or tobramycin 5 mg/kg daily, if the creatinine clearance is normal). An intravenous beta-lactam with activity against Enterobacteriaceae with or without an aminoglycoside is an alternate initial regimen for hospitalized patients. The choice between these should take into account patient tolerance and regional patterns of Enterobacteriaceae drug resistance.

Empiric treatment with an intravenous carbapenem or broad-spectrum penicillin or cephalosporin (with or without gentamicin) pending culture and sensitivity data is appropriate for patients who develop nosocomial prostatitis (eg, following a procedure for which they received a prophylactic fluoroquinolone) or who have a history of infections with drug-resistant pathogens, because of the increased risk of infection with a quinolone-resistant organism.

If available, a Gram stain of the urine can be helpful to further guide the empiric antibiotic choice:

- Patients with gram-negative rods on urine Gram stain should be treated as above.

- Gram-positive cocci in chains usually indicate enterococcal infection, which can be treated with amoxicillin (500 mg orally every eight hours) or ampicillin (2 g intravenous every six hours) if parenteral therapy is indicated. Of note, these regimens are not active against most *Enterococcus faecium* or other ampicillin-resistant strains. (See "Treatment of enterococcal infections".)

- Gram-positive cocci in clusters are most often due to *Staphylococcus aureus* or coagulase-negative staphylococci (eg, *S. epidermidis* or *S. saprophyticus*). Effective oral antibiotics for strains that are not methicillin-resistant include cephalosporins (eg, cephalexin 500 mg orally every six hours) or penicillinase-resistant penicillins (eg, dicloxacillin 500 mg orally every six hours). Choices for parenteral therapy include cefazolin (1 g intravenous every eight hours) or nafcillin (2 g intravenous every four to six hours). If there are risk factors for, or a history of methicillin-resistant *S. aureus*, vancomycin (table 1) can be used. (See "Methicillin-resistant Staphylococcus aureus (MRSA) in adults: Epidemiology", section on 'Risk factors'.)

Further changes to the empiric antibiotic regimen can be made based on susceptibility data of the isolated organism and clinical response. Of note, although nitrofurantoin is commonly used for lower urinary tract infections in women, we avoid this agent in men with prostatitis because of concern about poor tissue penetration and risk of adverse effects from prolonged use.

Duration of therapy — Patients initiated on parenteral antibiotics can be switched to oral antibiotics, if drug susceptibility and patient tolerance allow, 24 to 48 hours following improvement in fever and clinical symptoms. We treat with antibiotics for up to six weeks to ensure eradication of the infection [36]. For patients who have no prostatic abscess and, with treatment, have a painless rectal examination, sterile urine, and normal inflammatory markers (erythrocyte sedimentation rate and C-reactive protein), stopping treatment at four weeks is reasonable.

Clinical data on the duration of treatment for acute bacterial prostatitis are limited, and some experts endorse a shorter duration [22]. We favor a relatively prolonged course because of limited antimicrobial penetration into the prostate and the development of protected microcolonies deep within the inflamed gland that may be difficult to reach with antimicrobials. Shorter durations of therapy have been associated with progression to chronic symptoms. (See 'Prognosis' below.)

Nonantimicrobial therapy — Rarely, acute urinary retention develops during an episode of acute prostatitis. In this setting, we favor bladder drainage by suprapubic catheterization.

Passage of a catheter through the inflamed urethra into the bladder in a patient with acute prostatitis risks septic shock or rupture of a potential abscess. Nevertheless, some experienced urologists do cautiously attempt urethral catheterization in these uncommon situations. (See "Acute urinary retention", section on 'Urethral catheterization'.)

In a patient with a prostatic abscess, urological referral is indicated if the abscess is persistent after one week or more of antimicrobial therapy. In some cases, ultrasound-guided or surgical drainage may be warranted.

Monitoring during therapy — In most cases, fever abates and dysuria disappears within two to six days after the start of therapy. Acute phase reactants (eg, sedimentation rate, C reactive protein) and the PSA, if obtained, return to normal more gradually [27]. Clinical studies using a fluoroquinolone suggest that a negative urine culture at seven days following initiation of therapy predicts cure at the conclusion of the full course of therapy [37]. We typically repeat urine culture at seven days. If it is still positive at that time, alternative therapy should be initiated, based upon in vitro susceptibility tests of the most recent isolate.

Patients with persistently positive urine cultures should be further evaluated and treated for chronic bacterial prostatitis. (See "Chronic bacterial prostatitis".)

PROGNOSIS

Progression from acute to chronic bacterial prostatitis or inflammatory chronic pelvic pain syndrome (CPPS) is poorly understood. In a prospective cohort of 437 Korean men who presented with a confirmed diagnosis of acute bacterial prostatitis, 82 percent recovered without subsequent development of chronic infection three months or longer after treatment [38]. Development of chronic bacterial prostatitis or inflammatory CPPS (observed in 1.3 and 10.5 percent, respectively) was associated with higher rates of alcohol consumption, diabetes, voiding symptoms, enlarged prostate volume, catheterization, and short (two weeks) duration of antibiotic treatment.

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more

sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Bacterial prostatitis (The Basics)")

SUMMARY AND RECOMMENDATIONS

- Acute bacterial prostatitis is an acute infection of the prostate that typically occurs in young and middle-aged men. In most men, it is generally caused by the same organisms that cause urinary tract infections, most commonly gram-negative bacteria, especially Enterobacteriaceae (typically *Escherichia coli* or *Proteus* species). Sexually transmitted pathogens, such as *Neisseria gonorrhoeae* and *Chlamydia trachomatis*, are possible etiologies in sexually active men, who may have concomitant urethritis or epididymitis. (See 'Epidemiology' above and 'Microbiology' above.)

- Patients with acute bacterial prostatitis are typically acutely ill. The most common symptoms and signs include fevers, chills, dysuria, pelvic or perineal pain, and cloudy urine. Obstructive symptoms, such as dribbling of urine, can also occur. On exam, the prostate is often firm, edematous, and exquisitely tender. Common laboratory findings include peripheral leukocytosis, pyuria, and bacteriuria. (See 'Clinical manifestations' above.)

- Delay of antimicrobial therapy can increase the risk of secondary complications, including bacteremia, prostatic abscess, and metastatic infection (eg, spinal or sacroiliac infection, endocarditis). Certain underlying conditions, such as diabetes mellitus or HIV-related immunosuppression, may also predispose to the development of prostatic abscesses. (See 'Complications' above.)

- The presence of typical symptoms of prostatitis and the finding of an edematous and tender prostate on physical exam usually establishes the diagnosis. Digital rectal examination should be performed gently; vigorous prostate massage should be avoided since it is uncomfortable, allows no additional diagnostic or therapeutic benefit, and increases risk for bacteremia. Tenderness of the prostate on exam is generally not found in

isolated UTIs or other acute causes of dysuria (eg, urethritis, epididymitis) in the absence of coexisting prostatitis. (See 'Diagnosis' above and 'Differential diagnosis' above.)

- A urine Gram stain and culture should be obtained in all men suspected of having acute prostatitis to identify the bacterial etiology. Imaging studies are generally not warranted in acute bacterial prostatitis, unless there is clinical suspicion for a prostatic abscess (ie, when there are persistent clinical or laboratory abnormalities despite appropriate antimicrobial therapy). (See 'Diagnosis' above and 'Evaluation for complications' above.)

- Most cases of acute bacterial prostatitis are caused by gram-negative organisms, and empiric antibiotic therapy should be directed against this class. We suggest empiric treatment with trimethoprim-sulfamethoxazole or a fluoroquinolone (**Grade 2C**), unless drug resistance is suspected. The choice between these should take into account patient tolerance and regional patterns of Enterobacteriaceae drug resistance. If a urine Gram stain is available and suggests an alternate bacterial cause, initial antibiotic therapy should be directed against the identified class of organism. Parenteral antimicrobial therapy is warranted in patients who cannot tolerate oral medications, demonstrate signs of severe sepsis, or have bacteremia. Further changes to the empiric antibiotic regimen can be made based on susceptibility data of the isolated organism and clinical response. (See 'Antimicrobial therapy' above.)

- Patients initiated on parenteral antibiotics can be switched to oral antibiotics, if drug susceptibility and patient tolerance allow, 24 to 48 hours following improvement in fever and clinical symptoms. We typically use a prolonged antibiotic course (eg, at least four weeks) to try to ensure eradication of the infection. If the urine culture is still positive at seven days, alternative therapy should be initiated, based upon in vitro susceptibility tests of the most recent isolate. (See 'Duration of therapy' above and 'Monitoring during therapy' above.)

- Adjunctive therapies include management of complications, such as acute urinary retention and prostatic abscesses. Passage of a catheter through the inflamed urethra into the bladder is contraindicated in patients with acute prostatitis. If needed, bladder drainage must be done by suprapubic catheterization. (See 'Nonantimicrobial therapy' above.)

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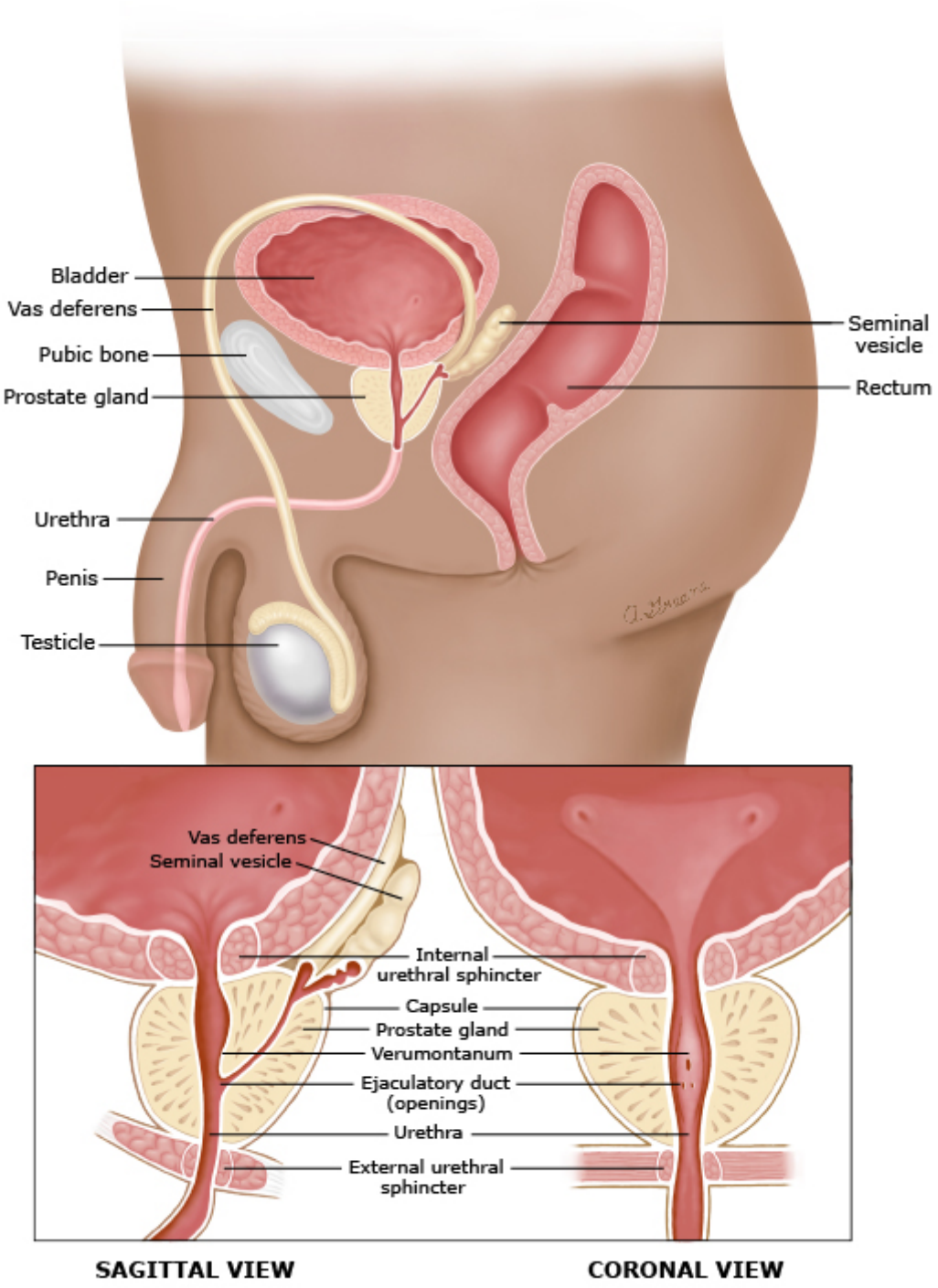
GRAPHICS

Prostate anatomy



The prostate gland is a walnut-shaped structure located at the base of the urinary bladder. The prostate gland is composed of both glandular and muscular tissue. Secretions from the prostate, vas deferens, and seminal vesicle empty into the prostatic urethra.

Graphic 54803 Version 9.0

Approach to vancomycin dosing for adults with normal kidney function*

Loading dose (for patients with known or suspected severe *Staphylococcus aureus* infection)¶	Load 20 to 35 mg/kg (based on actual body weight, rounded to the nearest 250 mg increment; not to exceed 3000 mg). Within this range, we use a higher dose for critically ill patients; we use a lower dose for patients who are obese and/or are receiving vancomycin via continuous infusion.
Initial maintenance dose and interval	Typically 15 to 20 mg/kg every 8 to 12 hours for most patients (based on actual body weight, rounded to the nearest 250 mg increment). In general, the approach to establishing the vancomycin dose/interval is guided by a nomogram.Δ
Subsequent dose and interval adjustments	Based on AUC-guided (preferred for severe infection)[1] or trough-guided serum concentration monitoring.◇

AUC: area under the 24-hour time-concentration curve.

* Refer to the UpToDate topic on vancomycin dosing for management of patients with abnormal kidney function.

¶ For patients with known or suspected severe *S. aureus* infection, we suggest administration of a loading dose to reduce the likelihood of suboptimal initial vancomycin exposure. Severe *S. aureus* infections include (but are not limited to) bacteremia, endocarditis, osteomyelitis, prosthetic joint infection, pneumonia warranting hospitalization, infection involving the central nervous system, or infection causing critical illness.

Δ If possible, the nomogram should be developed and validated at the institution where it is used to best reflect the regional patient population. Refer to the UpToDate topic on vancomycin dosing for sample nomogram.

◇ Refer to the UpToDate topic on vancomycin dosing for discussion of AUC-guided and trough-guided vancomycin dosing. For patients with nonsevere infection who receive vancomycin for <3 days (in the setting of stable kidney function and absence of other risk factors for altered vancomycin kinetics), vancomycin concentration monitoring is often omitted; the value of such monitoring prior to achieving steady state (usually around treatment day 2 to 3) is uncertain.

Reference:

1. *Rybak MJ, Le J, Lodise TP, et al. Therapeutic Monitoring of Vancomycin for Serious Methicillin-Resistant Staphylococcus Aureus Infections: A Revised Consensus Guideline and Review by the American Society of Health-System Pharmacists, the Infectious Diseases Society of America, the Pediatric Infectious Diseases Society, and the Society of Infectious Diseases Pharmacists. Am J Health Syst Pharm 2020; 77:835.*

Graphic 128911 Version 5.0

Contributor Disclosures

Alain Meyrier, MD No relevant financial relationship(s) with ineligible companies to disclose. **Thomas Fekete, MD** No relevant financial relationship(s) with ineligible companies to disclose. **Stephen B Calderwood, MD** Consultant/Advisory Boards: Day Zero Diagnostics [Whole genome sequencing for microbial identification and determination of antimicrobial susceptibility]. All of the relevant financial relationships listed have been mitigated. **Allyson Bloom, MD** No relevant financial relationship(s) with ineligible companies to disclose.

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