Chronic bacterial prostatitis

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INTRODUCTION

The prostate is subject to various inflammatory disorders [1]. One of these syndromes is chronic bacterial prostatitis, which is characterized by chronic or recurrent urogenital symptoms in the setting of documented or suspected bacterial infection of the prostate.

Definitions of inflammatory conditions of the prostate and the syndrome of chronic bacterial prostatitis will be reviewed here. Other prostatic syndromes, including acute bacterial prostatitis and chronic prostatitis/pelvic pain syndrome, are discussed separately. (See "Acute bacterial prostatitis" and "Chronic prostatitis and chronic pelvic pain syndrome".)

DEFINITIONS

While inflammatory or irritative conditions of the prostate are common clinical presentations, they often represent distinct pathogenic processes that may benefit from different management approaches. In order to standardize definitions, improve diagnosis and treatment, and facilitate research, the United States National Institutes of Health (NIH) established an International Prostatitis Collaborative Network to devise a classification approach for prostatitis [2]. The scheme developed by this group is the currently accepted categorization of prostatitis and defines the following syndromes (table 1):

- I. Acute bacterial prostatitis – Acute urogenital symptoms with evidence of bacterial infection of the prostate (see "Acute bacterial prostatitis")

- II. Chronic bacterial prostatitis – Chronic or recurrent urogenital symptoms with evidence of bacterial infection of the prostate
- IIIA. Chronic prostatitis/chronic pelvic pain syndrome, inflammatory – Chronic or recurrent urogenital symptoms with evidence of inflammation, but not bacterial infection of the prostate (see "Chronic prostatitis and chronic pelvic pain syndrome")
- IIIB. Chronic prostatitis/chronic pelvic pain syndrome, noninflammatory – Chronic or recurrent urogenital symptoms without evidence of inflammation or bacterial infection of the prostate (formerly designated prostatodynia) (see "Chronic prostatitis and chronic pelvic pain syndrome")
- IV. Asymptomatic inflammatory prostatitis – Absence of urogenital symptoms with evidence of inflammation of the prostate found incidentally (eg, biopsy performed for a different purpose)

Evidence of inflammation or bacterial infection is usually determined by the presence of inflammatory cells in, or bacterial growth from, expressed prostatic secretions, post-prostate massage urine, or seminal fluid. Maneuvers performed in the urology office can help refine the categorization of patients. As an example, including post-massage urine and seminal fluid for the assessment of inflammatory cells effectively doubles the number of people in the inflammatory subset (compared with using purulent prostatic secretions alone) [3].

PATHOGENESIS

The pathogenesis of chronic bacterial prostatitis is the same as in acute infection. 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). Chronic prostatitis may be a complication of acute prostatitis following inadequate and/or too short treatment. (See 'Risk factors' below.)

EPIDEMIOLOGY

Overall, prostatitis is a very common presentation in the clinical setting and tends to occur in young and middle-aged men. 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 a reason for the visit [4]. Extrapolation of the data estimated that the diagnosis of prostatitis was associated with two

million visits annually. However, actual bacterial infections of the prostate account for a minority of these cases [2].

In a retrospective study of 409 men with prostatitis syndromes, bacterial cultures of prostatic fluid were positive only in 10 percent [5]. The observed prevalence of bacterial prostatitis may be underestimated, as most studies infrequently evaluate for atypical organisms, such as *Ureaplasma urealyticum*, which in the above study was cultured in 20 percent of prostatic fluid samples. However, the role of atypical bacteria, like *Ureaplasma* in genitourinary infections, is not fully defined since it can be isolated from asymptomatic men as well [6].

Risk factors — The risk factors for the development of chronic bacterial prostatitis have not been clearly defined. Chronic bacterial infection of the prostate can develop following an episode of acute prostatitis. In a retrospective review of 480 patients with acute prostatitis, the 49 men (10 percent) who developed chronic infection were more likely to have a history of prior manipulation of the urinary tract, voiding symptoms, diabetes, and smoking, and on average had higher prostate volumes [7]. Furthermore, men who had been treated for acute prostatitis, but did not develop chronic prostatic symptoms, had received a longer treatment duration compared with men who developed chronic prostatic infection (average 36.5 versus 27.5 days). However, it is unknown whether these factors contribute independently to the development of chronic infection.

The presence of prostate stones may also contribute to the persistence of infection. In one study of men with chronic bacterial prostatitis, patients with prostate stones were more likely to experience relapse following antimicrobial therapy than patients without stones [8].

Otherwise, the risk factors for chronic infection appear to be similar to those observed for acute infection. (See "Acute bacterial prostatitis", section on 'Risk factors'.)

MICROBIOLOGY

Gram-negative rods are the most common etiologic agents, with *Escherichia coli* causing approximately 75 to 80 percent of episodes [9]. *Enterococcus faecalis*, *Klebsiella pneumoniae*, *Proteus mirabilis*, *Pseudomonas aeruginosa*, and other gram-negative bacilli are the next most commonly reported organisms [10-12]. *Staphylococcus aureus* and streptococcal species are occasional pathogens. The isolation of other gram-positive organisms, such as coagulase negative staphylococci and corynebacteria from prostatic fluid, is of uncertain clinical significance as illustrated by studies in which isolation of these organisms was neither

associated with inflammatory cells in prostatic secretions nor reproducible even in the absence of antibiotic therapy [13,14].

Fastidious sexually transmitted organisms, such as *Chlamydia trachomatis*, have also been associated with chronic prostatic infection, although this attribution remains speculative. *C. trachomatis* has been isolated from the prostate and in such cases appears to reside in prostate tissue as opposed to represent a contaminant from the urethra [15,16]. Some studies have shown that men with chronic prostatitis without a clear bacterial etiology had detectable chlamydial antigen in urine or prostatic secretions more frequently than men with pelvic pain but no signs of prostatic inflammation (21 to 25 versus 0 to 6 percent, respectively) [17-19].

Rarely, fungi or *Mycobacterium tuberculosis* may be involved in chronic prostatitis [20,21]. Patients with underlying immunosuppression, in particular, may be more likely to have prostatic involvement with organisms other than the usual bacteria that tend to cause urinary tract infection. As an example, in a series of patients with HIV-related immunosuppression and a history of treated cryptococcal meningitis, *Cryptococcus neoformans* could be isolated from prostatic secretions at a time when the organism was absent in blood or cerebrospinal fluid [22].

CLINICAL PRESENTATION

The presentation of chronic bacterial prostatitis can be quite subtle. Classically, men present with symptoms of recurrent urinary tract infection (frequency, dysuria, urgency, perineal discomfort, and perhaps a low-grade fever) with repeated isolation of the same organism from the urine. However, this presentation is reported by the minority of patients [11]. Most patients have only one or some of these features.

Some men may be asymptomatic and have only incidentally noted persistent or recurrent bacteriuria. Other symptoms can include pain (in the perineum, lower abdomen, testicles, penis, and with ejaculation), bladder irritation, bladder outlet obstruction, and sometimes blood in the semen. Sexual dysfunction may accompany chronic bacterial prostatitis, although it does not clearly occur more commonly than in men of a similar age without prostatitis [23-25].

On rectal examination, there may be prostatic hypertrophy, tenderness, edema, and nodularity. However, the prostate exam is frequently normal.

Laboratory findings that suggest inflammation or infection, such as elevated serum leukocytes or inflammatory markers, may be absent. In an analysis of participants in a trial of treatment for

documented chronic bacterial prostatitis, an elevated prostatic specific antigen (>4 ng/mL) was detected in only about 25 percent [26].

DIAGNOSIS

The diagnostic standard for bacterial prostatitis is the finding of bacteria at higher levels in prostatic fluid compared with urethral and bladder specimens. However, maneuvers to express prostatic fluid can be cumbersome and are rarely performed in clinical practice. Instead, chronic bacterial prostatitis is often presumptively diagnosed and empirically treated with antimicrobials when men present with chronic (eg, longer than three months) or recurrent urogenital symptoms, particularly if bacteriuria is also present.

Because of the insensitivity of these clinical findings and the therapeutic implication of a prolonged course of antibiotics, we favor obtaining prostatic specimens for analysis and culture to confirm the prostate as the site of infection in men with chronic symptoms of prostatitis, incidental bacteriuria, or recurrent urinary tract infections in the absence of other risk factors, such as bladder catheterization. In most cases, this is best performed in an urologist's office, where there is a greater level of experience in obtaining prostatic secretions and thus a greater likelihood of a microbial diagnosis.

Obtaining and testing prostatic specimens — Prostatic specimens can be obtained by collecting expressed prostatic fluid and a urine sample expressed following prostatic massage. For primary care doctors and internal medicine subspecialists, obtaining fluid or tissue from the prostate is difficult and semen cultures are not standard. Thus, referral to an urologist can be helpful for men with long-standing or refractory prostatic symptoms to obtain such specimens for diagnosis.

The classic method for localizing pathogens in the lower urinary tract and thus evaluating for bacterial prostatitis is the Meares-Stamey four-glass test [9]. It involves first cleaning the periurethral area and then collecting the first 5 to 10 mL void of urine (VB1, urethral sample) and a midstream sample (VB2, bladder sample). The patient should stop voiding before the bladder is empty and the prostate is then digitally massaged by applying gentle pressure moving from the superior portion to the apex for about one minute. Any prostatic secretions that are expressed (EPS, prostatic sample) and the first 5 to 10 mL of subsequently voided urine (VB3, prostatic sample) are collected. (See "Sampling and evaluation of voided urine in the diagnosis of urinary tract infection in adults".)

The finding of pathogens on culture of prostatic samples (EPS and VB3) exclusively or at a level 10 times higher than in urethral and bladder samples (VB1 and VB2) is diagnostic of bacterial prostatitis. For the test to be interpretable, the colony count in VB2 must be less than 10^3/mL, since bladder bacteriuria prevents identification of the frequently small number of organisms from the prostate. Chronic prostatitis is suspected when VB3 has more than 12 leukocytes per high power field; more than 20 leukocytes per high power field is generally diagnostic unless leukocytes were also present in VB2 [23].

Although the four-glass test is described extensively in the literature, it appears to be infrequently used in practice. In one survey of urologists in which 64 percent responded, 33 and 47 percent, respectively, said that they never or rarely performed the four-glass test [27]. Furthermore, the results of the test apparently did not influence the use of antibiotics, since urologists who used the test routinely did not differ in antibiotic prescribing from others who used it less often.

A simpler, "two-glass" method, in which cultures from only the post-prostatic massage urine (VB3) are compared with the pre-massage bladder urine sample (VB2), has been suggested as an alternate method, with a 100 percent positive and 96 percent negative predictive value when compared with the four-glass test [28]. Although this test has slightly lower sensitivity than the four-glass test, it is a preferred alternative compared with not performing any cultures of prostatic samples.

Evaluating the microbial etiology — Cultures of post-prostatic massage urine or expressed prostatic secretions are almost always positive in chronic bacterial prostatitis (table 1) and thus reveal the bacterial etiology. The repeated isolation of the same organism from urine cultures over time also suggests the etiologic agent. However, sexually transmitted organisms that may play a role in chronic bacterial prostatitis, such as *C. trachomatis*, will not grow in routine culture. Thus, in men who are sexually active and have clinical evidence of chronic prostatitis, but negative results of urine and prostatic secretion cultures, nucleic acid amplification testing for *C. trachomatis* on urine or urethral swabs can be diagnostically useful [15,16]. (See 'Microbiology' above and "Clinical manifestations and diagnosis of Chlamydia trachomatis infections", section on 'Diagnosis of chlamydial infections'.)

DIFFERENTIAL DIAGNOSIS

The main differential diagnosis to consider in a patient with symptoms and signs consistent with chronic bacterial prostatitis is chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS), in whom the symptoms may be the same, but there is no clear evidence of a bacterial infection

(see 'Definitions' above). This distinction is not always readily established; however, as cultures of prostatic specimens are infrequently performed in clinical practice and atypical, fastidious pathogens have been implicated as occasional causes of chronic bacterial prostatitis. (See 'Microbiology' above and 'Evaluating the microbial etiology' above.)

Furthermore, some patients with CP/CPPS respond to antibiotic therapy despite lack of a clear bacterial infection [29]. Thus, an initial antimicrobial course is often given to patients with symptoms of prostatitis to treat a potential bacterial infection, and the diagnosis of CP/CPPS is entertained among those patients who do not respond or relapse and continue to have no clear evidence of infection. (See "Chronic prostatitis and chronic pelvic pain syndrome", section on 'Diagnosis' and "Chronic prostatitis and chronic pelvic pain syndrome", section on 'Management'.)

Noninflammatory disorders of the prostate, bladder, and urinary tract can also lead to persistent irritative (urgency, frequency, nocturia) and obstructive (slow stream, hesitancy, dribbling) urinary symptoms that can be seen with chronic bacterial prostatitis. The evaluation of men with lower urinary tract symptoms is discussed in detail elsewhere. (See "Lower urinary tract symptoms in males".)

MANAGEMENT

Antibiotic therapy — Prolonged antibiotic therapy (eg, at least six weeks) with an agent that has good penetration into the prostatic tissue is generally necessary for treatment of chronic bacterial prostatitis. Nevertheless, the infection frequently recurs. A fluoroquinolone is generally the drug of choice for both initial and recurrent episodes, if organism susceptibility and patient tolerance allow. Trimethoprim-sulfamethoxazole is an adequate alternative regimen.

Antimicrobial penetration into prostatic tissue — The barrier between the microcirculation and the prostate gland stroma limits drug entry to passive diffusion, which only permits non-protein-bound, lipophilic antimicrobial agents to reach therapeutic levels within the gland. In addition, the low pH of prostatic fluid permits antibiotics with alkaline pK_as (such as fluoroquinolones and sulfonamides) to achieve high concentrations in prostatic tissue more readily than antibiotics with acidic pK_as. However, antibiotic prostatic penetration in the setting of inflammation occurs more readily [30]. Nevertheless, ideal choices for therapy of bacterial prostatitis include those agents that have optimal prostatic penetration.

In addition to fluoroquinolones and sulfonamides, other agents with good to excellent penetration into prostatic fluid and tissue include tetracyclines and macrolides [31].

Fosfomycin also appears to achieve reasonable intraprostatic concentrations in uninflamed prostate. In a prospective pharmacokinetic study of 26 healthy men undergoing a transurethral resection of the prostate for benign prostatic hyperplasia, serum, urine, and prostatic tissue fosfomycin concentrations were assessed following a single 3-g oral fosfomycin dose within 17 hours of surgery [32]. The mean overall prostate fosfomycin level was 6.5 mcg/g (range, 0.7-22.1 mcg/g), with therapeutic concentrations detectable up to 17 hours following the dose. Only one patient had a mean prostatic fosfomycin concentration of <1 mcg/g, whereas the majority (70 percent) had concentrations ≥4 mcg/g. Further data are warranted before fosfomycin can be routinely recommended for use in prostatic infections, as tissue levels after multiple doses and in the presence of inflammation or microabscesses remain to be determined.

Initial antibiotic therapy — For men with an initial episode of chronic bacterial prostatitis caused by a susceptible organism, we suggest antibiotic treatment with a fluoroquinolone. In cases of patient intolerance, contraindications or concerns about fluoroquinolone use, or bacterial drug resistance, antimicrobial choice should be guided by susceptibility testing. Trimethoprim-sulfamethoxazole is the primary alternative. Other possibilities include a macrolide, doxycycline, or oral beta-lactams.

Appropriate fluoroquinolone agents include ciprofloxacin 500 mg orally every 12 hours or levofloxacin 500 mg orally daily, each given for four to six weeks. In studies of men with documented chronic bacterial prostatitis, these and similar fluoroquinolone regimens have six-month clinical cure rates of about 60 to 70 percent when given for four weeks or longer [33-37]. There does not appear to be a clinically meaningful efficacy difference between specific fluoroquinolone agents [37]. However, shorter courses (less than four weeks) of higher doses of fluoroquinolones have been associated with an increased likelihood of relapse [38]. Courses longer than six weeks may be warranted for patients who have a relatively difficult to treat organism. Patients should be counselled about and monitored for potential adverse effects associated with prolonged use of fluoroquinolones. (See "Fluoroquinolones", section on 'Adverse effects'.)

Trimethoprim-sulfamethoxazole (one double-strength tablet [160 mg trimethoprim and 800 mg sulfamethoxazole] orally twice daily) is an appropriate alternative for infection caused by susceptible organisms. It is not as extensively studied as the fluoroquinolones and is generally given for longer than six weeks [9,11]. In a review of small studies of men with documented chronic bacterial prostatitis treated with trimethoprim-sulfamethoxazole for three months, initial cure rates were 68 to 100 percent (weighted average 85 percent), although the relapse rate over several years of follow up was approximately 64 percent [39].

The rising rate of resistance of Enterobacteriaceae to fluoroquinolones and trimethoprim-sulfamethoxazole (see "Acute simple cystitis in women", section on 'Resistance trends in E. coli') and the frequency of allergies, contraindications, or intolerance to these agents complicate treatment decisions for chronic bacterial prostatitis. There are limited data on management of such cases. Potential options include the following; they are typically given for six or more weeks:

- Doxycycline and macrolides are expected to penetrate into prostate tissue well, although they generally have little activity against the genitourinary pathogens that cause prostatitis. Nevertheless, one study suggested that doxycycline levels achieved in the prostate can overcome high minimum inhibitory concentration of presumptively resistant organisms [40]. Furthermore, doxycycline and azithromycin are the agents of choice in prostatic infections associated with *C. trachomatis* [41,42]. In a trial of 89 men with chronic prostatitis and laboratory evidence of *C. trachomatis* infection, men randomly assigned to azithromycin (500 mg daily for three days each week for three weeks) had higher rates of bacterial eradication (80 versus 39 percent) and clinical cure (69 versus 34 percent) compared with those who received ciprofloxacin (500 mg twice daily for 20 days) [43].

- Prolonged courses of cephalosporins chosen based on susceptibility testing, with close monitoring for clinical response and relapse, is also a reasonable approach.

- Fosfomycin is expected to penetrate into prostate tissue well, and some studies have reported good outcomes with fosfomycin-trometamol, in some cases at unconventionally high doses, for treatment of prostatitis with organisms resistant to other agents [40,44-46]. As an example, in a prospective study of 44 patients with chronic bacterial prostatitis (59 percent due to multidrug-resistant organisms), three- and six-month cure rates were 80 and 73 percent with fosfomycin dosed at 3 g once daily for one week then 3 g every 48 hours for a total of 6 to 12 weeks [45]. However, about 20 percent of patients experienced diarrhea on this regimen.

In some cases, the resistance pattern may only leave intravenous agents as effective options. In cases of complicated drug resistance or intolerance, consultation with an expert in the treatment of prostatitis is advised.

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 poor efficacy; there is also a risk of pulmonary and hepatic adverse effects from prolonged use.

The selection of agents and duration of therapy for chronic bacterial prostatitis have not been extensively studied using comparative trials with antimicrobial agents from varying classes. Moreover, studies of antimicrobial treatment of chronic prostatitis often include men with inflammatory chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS) in which an infectious etiology is not clearly established. Shorter courses (eg, two to four weeks) of antibiotics are sometimes used when the diagnosis is uncertain and the possibility of CP/CPPS is being entertained. (See 'Definitions' above.)

Management of recurrences — Chronic bacterial prostatitis often recurs and is usually treated with an additional course of antibiotics. Fluoroquinolones generally remain the treatment of choice for recurrent bacterial prostatitis, even if this class of drug was used for the initial treatment course, unless a resistant organism is suspected or detected. If the first course was four weeks or less, a longer second course of at least six weeks is recommended.

The efficacy of fluoroquinolones in recurrent chronic bacterial prostatitis has been suggested by several small studies [47-49]. In one study of 33 men who had failed therapy with trimethoprim, TMP-SMX, or norfloxacin, and were then retreated with ciprofloxacin (500 mg twice daily) for two to four weeks, the following results were noted [47]:

- Of 26 patients with *E. coli* as the pathogen, 17 were cured at greater than one year follow-up. In another two patients, a second treatment course with ciprofloxacin was successful. Two patients withdrew from therapy due to adverse drug reactions.
- Therapy was successful in two of five with pathogens other than *E. coli*.

Furthermore, failures of initial fluoroquinolone therapy are not necessarily due to bacterial resistance and instead can also be related to underlying prostate disease, incomplete adherence, drug interactions that reduce fluoroquinolone bioavailability, or to some other less understood component. Thus, in patients who have relapsed or failed to respond following a course of a fluoroquinolone, causes of impaired bioavailability of the fluoroquinolone should be sought and, if possible, remedied. (See "Fluoroquinolones", section on 'Drug interactions'.)

However, prolonged use of fluoroquinolones has been associated with several serious side effects, including *Clostridioides difficile* associated diarrhea, central nervous system toxicity, and tendinopathy. As examples, tendinitis and tendon rupture have been reported in patients receiving prolonged fluoroquinolone therapy, especially in patients >60 years of age [50]. Among patients in this age group, those receiving glucocorticoids are at the highest risk. Fluoroquinolones have also been associated with aortic dissection in patients with aortic arch aneurysms [51]. (See "Fluoroquinolones", section on 'Adverse effects'.)

In cases of bacterial resistance, patient intolerance, or concerns about prolonged use of fluoroquinolones, trimethoprim-sulfamethoxazole is an alternative. As above, a longer duration of therapy (at minimum, six weeks) may be needed to achieve clinical cure.

Fosfomycin may be a potential option for the treatment of multidrug-resistant gram-negative prostatitis, as the drug appeared in one study to achieve reasonable intraprostatic concentrations in the uninflamed prostate following a single 3-g oral dose [32], and has been successfully used in case reports [40,44], but further data are warranted. Other potential options for recurrent prostatitis in the setting of antimicrobial resistance or intolerances are similar to those used for initial therapy. (See 'Initial antibiotic therapy' above.)

Addressing urinary obstruction — Symptoms of difficulty initiating urination, a sensation of incomplete emptying, or post-void dribbling should lead to a work-up for urinary obstruction. This is discussed in detail elsewhere. (See "Lower urinary tract symptoms in males", section on 'Diagnostic testing'.)

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

- Prostatitis can be divided into the following categories (table 1) (see 'Definitions' above):
  - I. Acute prostatitis

- II. Chronic bacterial prostatitis
- IIIA. Chronic prostatitis/pelvic pain syndrome, inflammatory
- IIIB. Chronic prostatitis/pelvic pain syndrome, noninflammatory
- IV. Asymptomatic inflammatory prostatitis

- Overall, prostatitis is a very common presentation in the clinical setting and tends to occur in young and middle-aged men. However, actual bacterial infections make up a minority of these cases. Gram-negative rods and *Escherichia coli* in particular, are the most common etiologic agents of chronic bacterial prostatitis. Other organisms, including enterococci, staphylococci, streptococci, and *Chlamydia trachomatis* have also been associated with chronic prostatic infection. (See 'Epidemiology' above and 'Microbiology' above.)

- The clinical presentation of chronic bacterial prostatitis is typically subtle. Classically, men present with symptoms of recurrent urinary tract infection with repeated isolation of the same organism from the urine. However, some men may be asymptomatic with only persistent or recurrent bacteriuria. Other lower urinary tract symptoms include pain and irritative and obstructive symptoms. (See 'Clinical presentation' above.)

- In clinical practice, chronic bacterial prostatitis is usually presumptively diagnosed in men with chronic or recurrent urogenital symptoms and/or persistent bacteriuria. Ideally, bacterial infection of the prostate should be confirmed with comparative analysis of cultures from prostatic secretions and urine obtained prior to and following prostatic massage. (See 'Diagnosis' above and 'Obtaining and testing prostatic specimens' above.)

- The bacterial etiology can usually be identified through culture of urine or expressed prostatic secretions. In sexually active men with suspected chronic bacterial prostatitis, but no bacterial organism on routine culture, nucleic acid amplification testing for *C. trachomatis* on urine or urethral swabs should be performed. (See 'Evaluating the microbial etiology' above.)

- The main differential diagnosis to consider in a patient with symptoms and signs consistent with chronic bacterial prostatitis is chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS). An initial antimicrobial course is often given presumptively to patients with symptoms of prostatitis without a documented bacterial infection, and the diagnosis of CP/CPPS is then entertained among those patients who do not respond or relapse and continue to have no clear evidence of infection. (See 'Differential diagnosis' above and "Chronic prostatitis and chronic pelvic pain syndrome".)

- For patients with an initial episode of chronic bacterial prostatitis, we suggest treatment with a fluoroquinolone as long as organism susceptibility and patient tolerance allow (**Grade 2C**). Trimethoprim-sulfamethoxazole is an alternative option. Fluoroquinolones are typically given for at least four to six weeks for probable or documented chronic bacterial prostatitis. Longer courses may be indicated for patients who are infected with a relatively difficult to treat organism or are given a non-fluoroquinolone antibiotic for therapy. Shorter courses (eg, two to four weeks) are sometimes used when the diagnosis is uncertain and the possibility of CP/CPPS is being entertained. Documented *C. trachomatis* infections can be treated with doxycycline or azithromycin. (See 'Initial antibiotic therapy' above.)

- Recurrences of chronic bacterial prostatitis are common and warrant a second course of antibiotics. Possible causes of treatment failure, including antibiotic resistance, incomplete adherence, and impaired drug absorption, should be evaluated. For men with a recurrent episode of chronic bacterial prostatitis, we suggest treatment with a fluoroquinolone regardless of the initial antibiotic choice unless there is suspicion of a resistant organism or poor drug bioavailability (**Grade 2C**). Trimethoprim-sulfamethoxazole is an alternate agent. If the initial course of therapy was less than six weeks, a longer subsequent course is indicated. (See 'Management of recurrences' above.)

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Topic 86802 Version 18.0

GRAPHICS

Classification of prostatitis with prostatic localization studies

Category	Mid-stream urine (VB2)		Expressed prostatic secretion		Organism
	White cells	Culture	White cells	Culture	
ABP	++	+	++	+	Enterobacteriaceae
CBP	+	+	+	+	Enterobacteriaceae
CP/CPPS, inflammatory	–	–	+	–	None
CP/CPPS, noninflammatory	–	–	–	–	None
Asymptomatic inflammatory prostatitis*	–	–	+	–	None

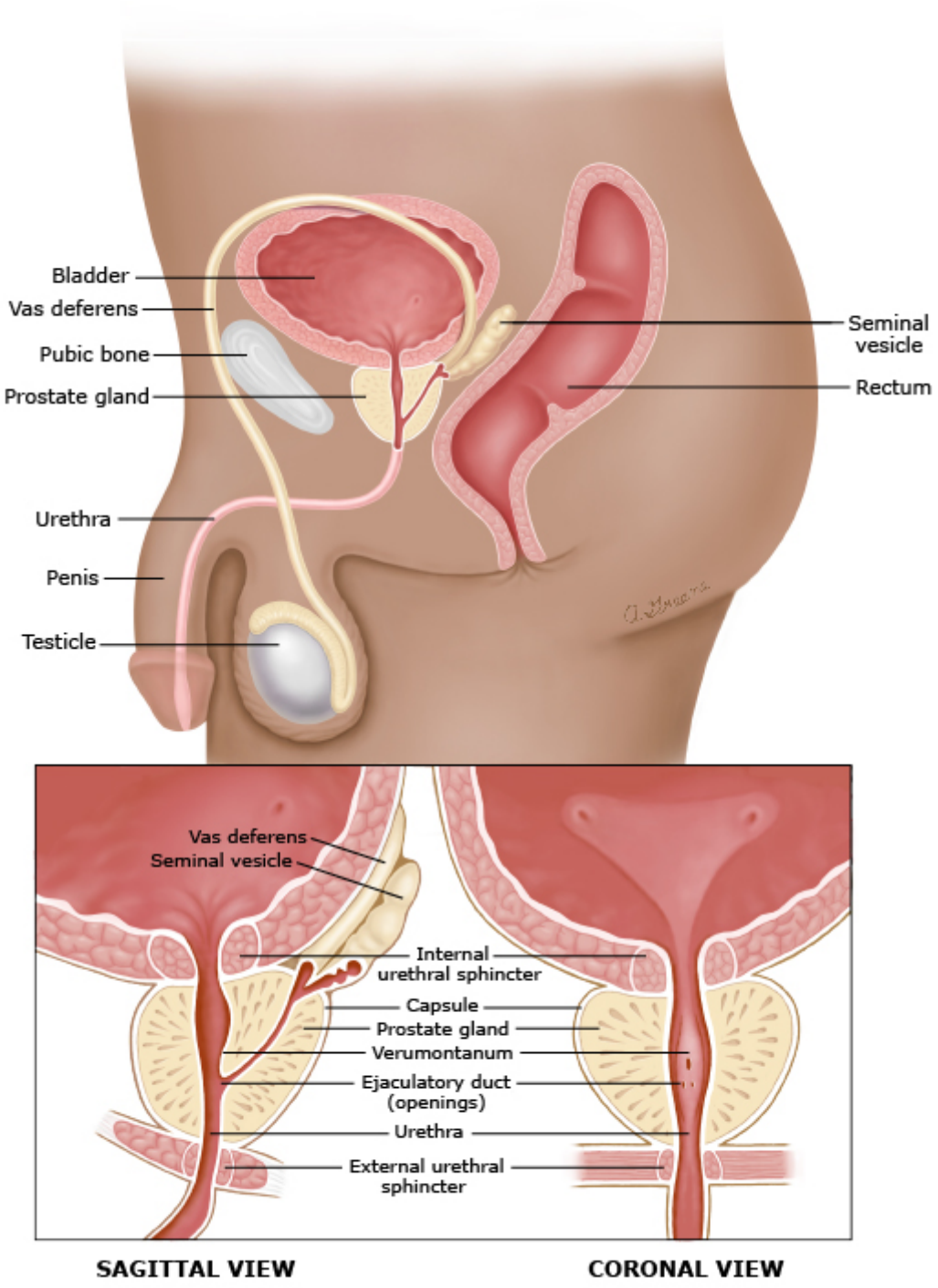
ABP: acute bacterial prostatitis; CBP: chronic bacterial prostatitis; CP/CPPS: chronic prostatitis/chronic pelvic pain syndrome; VB2: clean-catch mid-stream urine specimen.

* Asymptomatic inflammatory prostatitis is generally detected incidentally, for example, on a biopsy performed for a different purpose.

Adapted from: Doble A. Br J Urol 1994; 74:537 and Krieger JN, et al. JAMA 1999; 282:236.

Graphic 75162 Version 4.0

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

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