Clinical Infectious Diseases

STATE-OF-THE-ART REVIEW

State-of-the-Art Review: Diagnosis and Management of Acute and Chronic Bacterial Prostatitis

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Acute bacterial prostatitis (ABP) and chronic bacterial prostatitis (CBP) are poorly defined clinical entities, and diagnosis can be challenging. A clinical diagnosis of ABP can be made in the setting of an acute urinary tract infection (UTI) with systemic illness and evidence of prostatic involvement as defined by prostatic tenderness or fluctuance on digital rectal examination or prostatic abscess identified on imaging. Management includes a minimum of 2 weeks of antibiotics with surgical intervention reserved for refractory cases or prostatic abscess (depending on size). Chronic bacterial prostatitis should be suspected in a male patient with chronic urinary symptoms or recurrent UTIs. Diagnostic evaluation should include a 4- or 2-glass Meares–Stamey test, with a positive test confirming the diagnosis. Management includes 6 weeks of antibiotics. Surgery can be considered for particularly refractory cases. Future research into ABP and CBP can address questions about epidemiology, role of radiographic imaging, and duration of antimicrobial therapy.

Keywords. acute bacterial prostatitis; chronic bacterial prostatitis; Meares–Stamey test; digital rectal examination; urinary tract infection.

CASE 1

A 65-year-old man with diabetes mellitus presents to the emergency department with 4 days of fevers, chills, dysuria, and difficulty starting his urinary stream. He does not report flank pain, back pain, nausea, or vomiting. He has not previously had such symptoms. He has a temperature of 38.7° C, a blood pressure of 109/67 mmHg, a heart rate of 121 beats per minute, and a respiratory rate of 16 breaths per minute. Cardiopulmonary examination is normal other than tachycardia. There is no abdominal or costovertebral-angle tenderness. A brief, gentle digital rectal exam is performed and reveals a tender prostate. The peripheral white blood cell (WBC) count is 16 500/μL with 87% neutrophils. The creatinine is 1.54 mg/dL, which is the patient's baseline. There are 312 WBCs per high-powered field (HPF) on the urine microscopy. Urine culture (obtained prior to prostatic examination) and blood culture ultimately grow pan-sensitive *Escherichia coli*.

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CASE 2

A 48-year-old man presents to the outpatient clinic with dysuria, urinary frequency and urgency, and mild pelvic discomfort. He has had 3 prior episodes of acute cystitis over the last 4 months. Each episode involved acute urinary symptoms, pyuria identified on urine microscopy, and urine cultures growing *E. coli*. Each episode was treated with 7 days of oral antibiotics. Although his bladder symptoms resolve, a vague pelvic discomfort persists between episodes. His vital signs are normal. The physical exam is unrevealing. A repeat urinalysis shows 58 WBCs/HPF. Urine culture from an initial voided specimen grows 10^4 colony-forming units (CFUs)/mL of *E. coli*. A second urine specimen is collected after prostatic massage, and this urine culture grows 10^5 CFU/mL of *E. coli*.

Acute bacterial prostatitis (ABP) and chronic bacterial prostatitis (CBP) are poorly defined clinical entities, and uncertainties about the clinical presentation and diagnostic criteria might lead to either under- or overdiagnosis. Data about the prevalence of these infections are limited, and evidence from clinical trials to inform optimal treatment approaches is sparse.

The clinical presentation is key to correctly identifying ABP and CBP but is not specific enough to make the diagnosis without supporting physical examination, laboratory, or radiographic data. Additionally, knowing when to consider these clinical entities and how to evaluate them is not always straightforward.

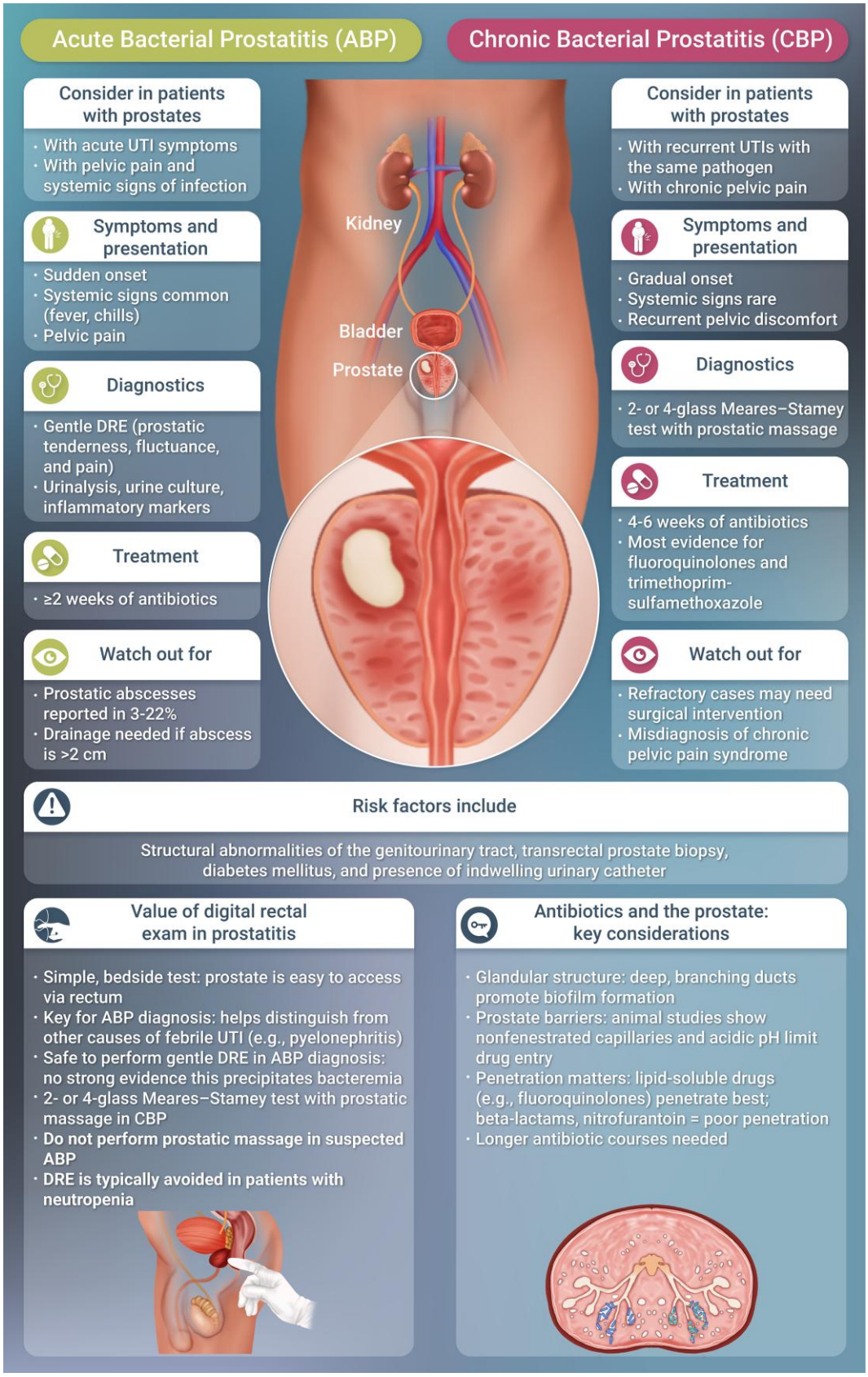

Table 1. Summary of Microbiologic Epidemiology of Acute and Chronic Bacterial Prostatitis

		Acute Bacterial Prostatitis (%) (Across 14 Studies)	Chronic Bacterial Prostatitis (%) (Across 13 Studies)
Gram-negative organisms	*Escherichia coli*	68	17
	Klebsiella spp.[a]	7	2
	Other *Enterobacterales* spp.[b]	8	11
	Pseudomonas aeruginosa	7	1
	Other Gram-negative organisms[c]	2	1
Gram-positive organisms	*Enterococcus* spp.[d]	3	41
	Staphylococcus spp.[e]	2	19
	Streptococcus spp.[f]	2	6
	Other Gram-positive organisms[g]	<1	<1
Other	*Candida* spp.	<1	
	Mycoplasma hominis		<1
	Ureaplasma urealyticum		1
	Other[h]	2	1

The table includes studies published from July 1999 to December 2024, which investigated National Institutes of Health category I and/or II prostatitis, reported urine culture results, included at least 20 samples, and utilized culture (as opposed to polymerase chain reaction [PCR] or metagenomic testing) for microbiological identification. The table excludes case reports, case series, and studies, which selectively excluded large proportions of organisms (eg, studies of Gram-positive or Gram-negative organisms only). The numbers represent weighted averages (in percent) of all included studies. Weighted averages for each individual organism/category were calculated by multiplying the total number of cultures in a study by the percentage of organism present in that study (= # of cultures of that organism in that specific study), summing across all studies (= # of cultures of that organism across all studies), and then dividing by the total number of cultures across all included studies (=weighted average of that organism). Organisms related to sexually transmitted infections (*Chlamydia trachomatis*, *Neisseria gonorrhea*, and *Trichomonas vaginalis*) were excluded from culture results and overall percentages. No criteria were applied to restrict the inclusion of possible contaminants outside of those already used in the methodology of included manuscripts. In particular for chronic bacterial prostatitis, there might be an overestimation of certain organisms (such as coagulase-negative staphylococci), which might actually represent contaminants.

[a]*K. ornithinolytica, K. oxytoca, K. pneumoniae,* and unspecified *Klebsiella* spp.

[b]Includes *Citrobacter freundii, Citrobacter koseri,* unspecified *Citrobacter* spp., *Enterobacter aerogenes, E. cloacae, E. intermedius,* unspecified *Enterobacter* spp., *Klebsiella, Enterobacter, Serratia* (KES) group, *Morganella morganii, Proteus mirabilis, P. penneri, P. vulgaris,* unspecified *Proteus* spp., *Serratia marcescens,* unspecified *Serratia* spp., and unspecified other *Enterobacterales.*

[c]Includes *Acinetobacter baumannii,* unspecified *Acinetobacter* spp., *Haemophilus parainfluenzae, Pseudomonas putida,* and unspecified *Pseudomonas* spp.

[d]*Enterococcus faecalis, E. faecium,* and unspecified *Enterococcus* spp.

[e]*Staphylococcus aureus, S. epidermidis, S. haemolyticus, S. hominis, S. saprophyticus, S. warneri,* unspecified coagulase-negative staphylococci, and unspecified *Staphylococcus* spp.

[f]*Streptococcus agalactiae, S. anginosus, S. intermedius, S. mitis, S. sanguinis,* and unspecified *Streptococcus* spp.

[g]Includes *Aerococcus urinae, Corynebacterium renale, Corynebacterium seminale,* unspecified *Corynebacterium* spp., unspecified *Lactobacillus* spp., and unspecified other Gram-positive organisms.

[h]Includes unspecified "atypical pathogens," unspecified fungi, *Gardnerella vaginalis,* unspecified "mixed infections," and unspecified "other."

Acute bacterial prostatitis should be considered in a male patient presenting with acute urinary symptoms in combination with fever and/or other signs and symptoms of systemic illness [1]. Pelvic pain or obstructive urinary symptoms in this clinical setting suggest ABP, but their absence does not exclude the diagnosis. While pyelonephritis and bacteremia are also frequently diagnosed in urinary tract infections (UTIs) associated with systemic symptoms, the presence of either pyelonephritis or bacteremia does not inherently exclude the possibility of ABP. Similarly, ABP can also occur in patients with a chronic indwelling urinary catheter [2].

In contrast to ABP, CBP presents indolently, with most patients diagnosed and treated in the ambulatory setting [1]. CBP should be suspected in men with recurrent UTIs due to the same causative organism. In addition to typical lower urinary tract symptoms, some patients may report pelvic pain or pressure [1]. Like ABP, CBP can occur in patients with a chronic indwelling urinary catheter; in these scenarios, removing the indwelling catheter if clinically feasible, along with antibiotic therapy, is the ideal strategy to decrease risk of recurrent prostatic infection.

Of note, patients who have previously undergone radical prostatectomy (ie, removal of the entire prostate) are not at risk for developing ABP or CBP. This is in contrast to those who have only had partial resection of the prostate, such as with transurethral resection of the prostate (TURP); such patients could still potentially develop prostatitis.

This narrative review will summarize available case series information, observational studies, expert opinion, and consensus guidelines on diagnosis and treatment of ABP and CBP from the 1960s through the present.

WHAT ARE THE EPIDEMIOLOGY AND MICROBIOLOGY OF ACUTE BACTERIAL PROSTATITIS AND CHRONIC BACTERIAL PROSTATITIS?

Epidemiology

Acute bacterial prostatitis and CBP lack standardized and clinically applicable diagnostic criteria, resulting in significant heterogeneity in epidemiological studies of prostatitis, a problem further exacerbated by between-study heterogeneity in the geographic regions, time periods, and age groups examined. That said, surveys from 1998 to 2003 indicated that 2%–10% of adult

Table 2. NIH Consensus Definitions and Classification of Prostatitis[35]

Classification	Definition	Associated Clinical Symptoms [35, 36]
Category I—acute bacterial prostatitis	Acute bacterial infection of the prostate gland	Acute onset of malaise, fever, obstructive lower urinary tract symptoms, dysuria, and perineal or prostatic pain
Category II—chronic bacterial prostatitis	Chronic (>3 m) infection of the prostate gland	Recurrent episodes of bacterial UTI caused by the same organism, often associated with prostatic pain
Category III—chronic pelvic pain syndrome	Chronic pelvic pain consistent with prostatitis but without pathogenic bacterial growth in the urine and prostatic fluid samples	Pelvic or prostatic pain
IIIA—inflammatory	Elevated level of white blood cells in postprostatic massage urine and prostatic fluid samples	
IIIB—noninflammatory	No WBC elevation in postprostatic massage urine and prostatic fluid samples	
Category IV—asymptomatic inflammatory prostatitis	Absence of genitourinary symptoms but presence of elevated WBCs in postprostatic urine and prostatic fluid samples or evidence of inflammation on prostate biopsy	No genitourinary pain

Abbreviations: NIH, National Institutes of Health; UTI, urinary tract infection; WBC, white blood cell.

men reported ever having prostatitis symptoms (ie, without distinction between bacterial and nonbacterial prostatitis) [3–7].

The incidence of ABP increases with age; <10% of patients with ABP were under 35 in a US cohort from 2008, while over 50% were 65 years and older [8]. For CBP, the mean age was 48 years in a European cohort published in 2016 [9]. The prevalence of prostatitis symptoms was over 3 times greater in men aged 50–59 years compared with 20–39 years in a separate European cohort from 2000 [10].

Other risk factors for ABP and CBP include benign prostatic hyperplasia, other anatomical urological disorders, a history of prior sexually transmitted infections (STIs), and a past history of UTIs [8, 11]. A particularly high prevalence of diabetes mellitus (37.5%–75%) is reported in patients with prostatic abscess [12–14]. In the preantiretroviral therapy (ART) era, HIV/AIDS was also described as a risk factor for bacterial prostatitis and prostatic abscess; however, whether HIV/AIDS is an independent risk factor for prostatitis in the era of effective ART is unclear [15].

Lower urinary tract manipulation (eg, transrectal prostate biopsy, urologic surgery, catheter placement, etc.) can precipitate ABP and/or prostatic abscess, with a reported frequency of prior urinary tract manipulation in 10% of patients in one study of 614 patients with ABP [2, 8, 12, 16, 17]. In a separate study of 923 patients who underwent prostate biopsy, ABP occurred in 2% of patients after the procedure [18]. Receipt of a fluoroquinolone within the 6 months prior to the biopsy in comparison with having received no antibiotic was significantly associated with postbiopsy prostatitis (17.1% vs 4.5%, $P = .042$) in one study, likely due to colonization with drug-resistant pathogens leading to ineffective preprocedural antimicrobial prophylaxis [19].

The possibility of using preprostate biopsy rectal-swab culture to direct prophylactic-antimicrobial choice was evaluated in Dutch randomized trial involving 1379 patients in which the control arm received prophylaxis with ciprofloxacin, while the intervention arm received directed antibiotic therapy depending upon results of a rectal swab culture [20]. Although there was a trend toward a decreased 7-day infection rate in the intervention arm (2.5% vs 4.3%), this finding did not meet statistical significance ($P = .08$). In the control group, having an organism resistant to fluoroquinolones on a rectal swab was a risk factor for infection within 7 days after the biopsy.

There are few studies on the long-term outcomes of patients with ABP and CBP. In one study of 480 patients with ABP, 1.4% progressed to CBP; 4.1% had subsequent epididymo-orchitis; 5.8% had "persistent pyuria and bacteriuria"; and 9.6% went on to develop a chronic, nonbacterial inflammatory prostatitis [21]. In another study of 473 patients with ABP, out of 296 whose information was available for follow-up for at least 6 months, 24 (8.1%) had reported progression to chronic prostatitis (not specified as bacterial vs noninfectious), while the 6-month recurrence rate of ABP was 9.5% (34/358 patients) [17]. Progression to CBP was more common in patients with histories of smoking, prior manipulation of the urinary tract, and diabetes mellitus. Data on long-term mortality related to bacterial prostatitis are not available.

Microbiology

In the modern antibiotic era, pathogens predominantly associated with ABP and CBP reflect those known to typically cause UTIs (as opposed to the preantibiotic era, in which gonorrhea was a major contributing pathogen) [22, 23]. However, the reported microbiology of prostatitis varies greatly among studies over time and by geographic region and syndrome (ie, ABP vs CBP) (Table 1). *Escherichia coli* was the etiological pathogen in half or more of ABP cases, with other common causes including *Klebsiella* spp., *Pseudomonas aeruginosa*, *Proteus* spp., and *Enterococcus faecalis* [8, 24–26]. When and where the sample is acquired affects the microbiologic epidemiology, with healthcare-associated infections yielding higher proportions of *P. aeruginosa* and lower proportions of *E. coli* [2]. For CBP,

E. faecalis was the most common organism, reported in 38%–44% of cases, and appears to be becoming more common or at least more commonly reported [9, 27, 28]. *E. coli* remains the most commonly identified Gram-negative pathogen for CBP.

Other less common causes of ABP and CBP are described in the literature, such as fungal (ie, *Candida* spp., *Cryptococcus* spp., *Histoplasma* spp., *Coccidioides* spp., *Blastomycosis* spp.) and mycobacterial organisms [23, 29–32]. In these cases, patients are typically immunocompromised (eg, *Cryptococcus* spp. in HIV) or have a clear exposure risk factor (eg, *Mycobacterium bovis* prostatitis after intravesical bacillus Calmette–Guérin immunotherapy for nonmuscle-invasive bladder cancer). The finding of *S. aureus* as the causative agent in prostatitis (often associated with prostatic abscess) should prompt consideration of hematogenous metastatic infection. Prostatitis due to multidrug resistant organisms, such as extended spectrum beta-lactamase producers (ESBLs), is an increasing problem. In one study of postprostate biopsy ABP, nearly 43% of recovered *E. coli* isolates were ESBL-positive [24].

Finally, in young sexually active men with prostatitis, *Chlamydia trachomatis* and *Mycoplasma genitalium* are often detected in studies specifically evaluating for these organisms [33, 34]. The pathogenesis of disease is also potentially different for prostatitis arising through the UTI pathway versus STIs. Additionally, the potential connection between *M. genitalium* and prostatitis is less well defined than that between *C. trachomatis* and prostatitis. *Mycoplasma hominis* and *Ureaplasma urealyticum* might also contribute, although their clinical significance remains debated.

WHAT ARE THE DIAGNOSTIC CRITERIA FOR ACUTE BACTERIAL PROSTATITIS AND CHRONIC BACTERIAL PROSTATITIS?

In 1999, the International Prostatitis Collaborative Network published the National Institutes of Health (NIH) Consensus Definition and Classification of Prostatitis (Table 2) [35]. This NIH classification of prostatitis originated from an 18-member expert panel of urologic researchers, physicians, and patient advocates convened by the NIH during the mid-1990s [35, 37]. This system, which was developed by consensus and was intended primarily to standardize prostatitis research, divides prostatitis into bacterial and nonbacterial forms and distinguishes between acute and chronic disease. Of note, Category IV, asymptomatic inflammatory prostatitis, is a laboratory-based diagnosis requiring histology from a prostate biopsy and is usually detected incidentally during urologic evaluation.

The authors of the NIH classification system noted its limitations, including reliance on evaluation of prostate-specific specimens (eg, expressed prostatic fluid) that most clinicians do not obtain in actual practice and a lack of standardized diagnostic cutoffs for leukocytosis or presence of bacteria in prostatic samples [37]. Subsequent studies have suggested that the NIH classification system is not fully accurate in discriminating between patients who do or do not have prostatic infection. For example, observational and prospective randomized studies have found an association between antibiotics and clinical improvement in at least a subset of patients with the noninfectious entity chronic pelvic pain syndrome (CPPS) [36]. Distinguishing CBP from CPPS can be clinically challenging [1].

If we seek to apply this NIH classification system to the 2 case examples, the findings that support the diagnosis of ABP compared with other febrile UTI syndromes include pelvic pain, difficulty initiating a urinary stream, and prostatic tenderness. In the absence of these findings, the clinician would have more difficulty differentiating ABP from other forms of acute, complicated UTI, such as pyelonephritis or urinary-source bacteremia.

For the second case, the patient has recurrent UTI, but whether the source is the prostate, reinfection due to an organism living in his gut, or reinfection from a nidus within the urinary tract (eg, intracellular biofilms in the bladder wall or kidney stones in upper or lower urinary tract) is not clear from the clinical presentation.

The difficulty in determining whether the prostate is involved to a clinically significant extent in male UTI parallels the unsuccessful efforts in the 1970s to identify a biomarker reliably identifying upper versus lower urinary tract disease in women. The field of UTI research in general suffers from a lack of laboratory testing that reliably distinguishes the various UTI syndromes, which may reflect in part our growing understanding that the states of asymptomatic bacteriuria and UTI are more of a continuum than a dichotomy [38]. The continuum model might also be applicable to the extent of prostate involvement in men with UTI.

Role of Prostate-Specific Antigen in Diagnosis of Prostatitis

The role of prostate-specific antigen (PSA) in diagnosing prostatitis is unclear. Elevated PSA was observed in only 74/320 (23%) of patients with CBP in a large clinical trial [39]. PSA elevation might be more sensitive for ABP than CBP, but cohort studies have found PSA to be elevated in just 58%–71% in patients with ABP [40, 41]. The European Association of Urology (EAU) guidelines on urologic infections specifically recommend against using PSA for the diagnosis of prostatitis [42].

Imaging Studies in Diagnosis of Prostatitis

The diagnostic utility of imaging in ABP and CBP has not been adequately studied. One cohort suggested that prostate abscess complicates ABP in 31/142 patients (22%), while other studies have reported a lower prevalence ranging from 2.7% to 3.5% [2, 8, 43]. Prostatic abscess might be more common in postprocedural cases versus spontaneous cases of ABP (17.6% vs 1.6% in one study) [2]. In clinical practice, it is often the case that the

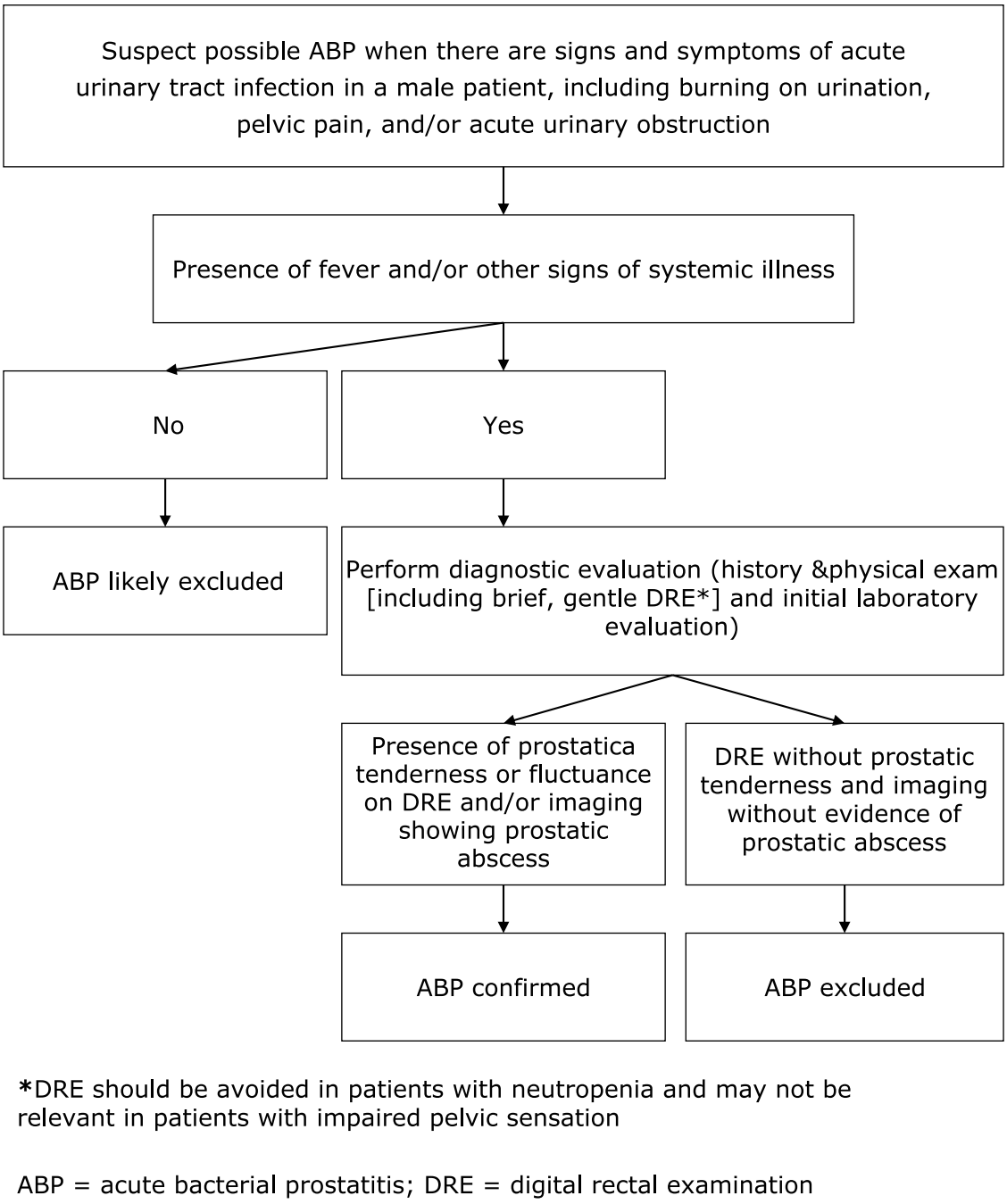

Figure 1. Diagnostic approach to acute bacterial prostatitis.

clinician is admitting a patient who has already undergone a computed tomographic (CT) scan or magnetic resonance imaging (MRI), and the more salient issue is what to do with a radiographic read suggesting prostatic involvement (nonspecific edema) when neither clinical nor physical exam findings suggestive of prostatitis are present. We did not find data addressing this specific question. In general, in the absence of clinical suspicion for prostatitis, further evaluation is typically not pursued, especially if the patient is asymptomatic or improving clinically.

Synthesis

While the NIH classification system might help standardize much-needed research in the field of prostatitis, clinical criteria for rapid bedside diagnosis of bacterial prostatitis are needed. Men with UTI may have a spectrum of prostate involvement,

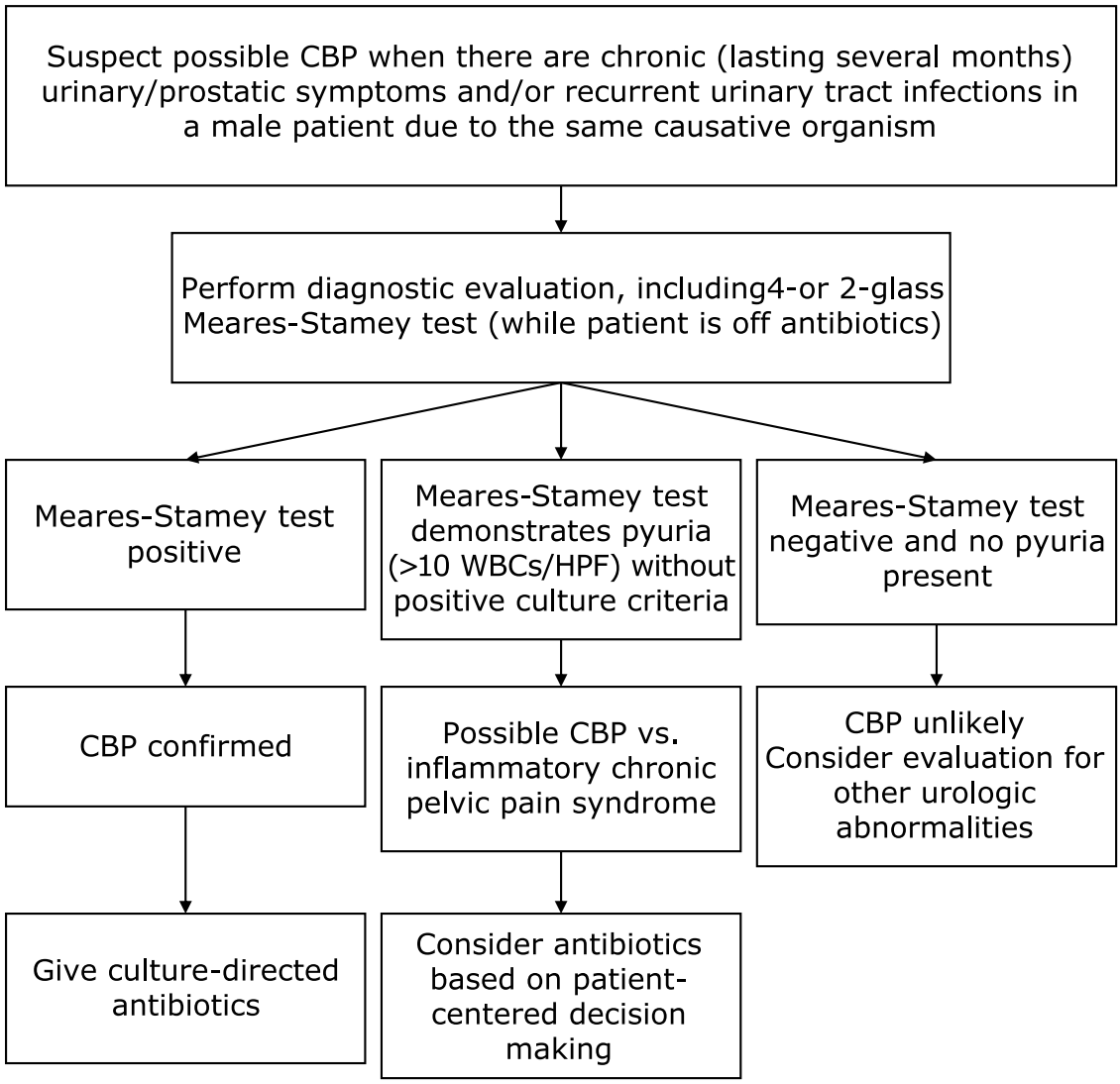

Figure 2. Diagnostic approach to chronic bacterial prostatitis.

including clinically apparent and symptomatic ABP, "subclinical prostatitis" (ie, visible on imaging or suggested by elevated PSA, but without pain on digital rectal examination [DRE]), or no prostatic involvement. The key (and not yet clearly answered) question is where along this spectrum is a longer course of antibiotics needed to clear bacteria from the prostate and prevent recurrent UTI or progression of ABP to CBP.

We propose that a clinical diagnosis of ABP be defined as acute UTI accompanied by clear evidence of prostatic involvement, meaning prostatic tenderness or fluctuance identified via brief, gentle DRE or imaging demonstrating a prostatic abscess. Although many patients with ABP will be febrile or bacteremic or have an elevated PSA in the context of an acute UTI, none of these variables are necessary or sufficient to diagnose ABP.

However, their presence should provoke further diagnostic inquiry (ie, DRE and/or pelvic imaging).

We further propose that a clinical diagnosis of CBP be defined as chronic (ie, usually months duration) urinary or prostatic symptoms *or* recurrent UTIs due to the same pathogen not adequately explained by another predisposing risk factor (eg, abnormal urologic anatomy). To note, prior literature has suggested the presence of a boggy prostate on DRE in patients with CBP, but this finding is not sensitive, and the presence of a boggy or tender prostate is not required to diagnose CBP. Additionally, patients with CBP may lack pyuria or bacteriuria prior to prostatic massage. PSA elevation is uncommon in CBP and should not be used for diagnosis.

When feasible, performing the Meares–Stamey 4-glass or 2-glass tests (described in detail below) while the patient is off antibiotics remains the best option for diagnosing CBP and guiding antimicrobial therapy through the acquisition of susceptibility data. Notably, if there is not a positive culture from a urine sample or prostatic fluid sample, the clinical scenario would technically be classified as NIH Category III, namely CPPS. However, from the viewpoint of a practicing clinician, CBP would be difficult to rule out entirely in this situation, and as discussed above, the NIH classification system imperfectly distinguishes infectious from noninfectious prostatitis.

WHAT DIAGNOSTIC EVALUATION SHOULD BE PERFORMED FOR A PATIENT IN WHOM ACUTE BACTERIAL PROSTATITIS OR CHRONIC BACTERIAL PROSTATITIS IS BEING CONSIDERED?

Figures 1 and 2 provide suggested diagnostic and management flowcharts for ABP and CBP, respectively.

Acute Bacterial Prostatitis

Physical Examination for Acute Bacterial Prostatitis

DRE is an important part of the clinical evaluation for ABP. The concept that DRE should be avoided in ABP out of concern for precipitating bacteremia probably relates to confusion about the difference between a brief DRE (encouraged) and a more intense prostatic massage (discouraged) [44–47]. For reference, brief DRE would typically last <5 seconds, while prostatic massage involves massaging all lobes of the prostate with firm pressure over 30–60 seconds. Additionally, DRE findings have been used as a key clinical criterion for the diagnosis of ABP in the prostatitis literature for decades. A tender prostate is found in 63%–90% of cases of ABP [2, 8]. Prostatic fluctuance on DRE suggests presence of a prostatic abscess but is insensitive, with studies reporting fluctuance is found in 16%–71% of cases [12, 23]. Costovertebral angle (CVA) tenderness should also be assessed to evaluate for pyelonephritis, although the sensitivity and specificity of CVA tenderness for pyelonephritis is not well defined. Studies of men with febrile UTI report prostatic tenderness in 9%–27% of patients and CVA tenderness in 37%–58% of patients, suggesting both physical exam maneuvers should routinely be performed when examining men with fever or systemic illness and suspected UTI [48, 49].

Laboratory Studies for Acute Bacterial Prostatitis

Serum WBC count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) are commonly elevated in ABP but not specific for ABP. Pyuria is present in most but not all cases of prostatitis (72%–89%), which contrasts with UTI in women, where absence of pyuria (<10 WBCs/HPF) rules out UTI; we suspect this difference reflects the small proportion of hematogenous prostatitis (eg; prostatic abscess as a metastatic focus of infection in *Staphylococcus aureus* bacteremia). Urine cultures are positive in 65 with –83% of patients with ABP, with negative results often in the context of prior antibiotic administration [2, 8, 26]. Blood cultures, when collected concurrently with urine cultures prior to receipt of antibiotics, contribute to a microbiologic diagnosis in 5% of cases [50]. Practically speaking, when patients present with sepsis or are ill-appearing, blood cultures will likely (and appropriately) be collected in the emergency department. For prostatic abscess, it is important to obtain an operative culture of purulent material if surgically drained. One study found that only a minority of patients had matching organisms identified on urine culture and prostatic abscess culture [14]. However, in that study, urine culture was negative in approximately one-third of patients, and abscess culture was only obtained in 50% of patients. As discussed above, we do not obtain PSA in suspected prostatitis as it does not appear reliable for either ruling in or out ABP.

Imaging Studies for Acute Bacterial Prostatitis

The main utility of imaging in suspected ABP is identification of prostatic abscess. There are no studies reporting improved sensitivity/specificity for diagnosis of ABP with ultrasound, CT, or MRI. Additionally, no data are available to indicate whether nonspecific prostate edema on imaging indicates a patient is likely to benefit from more prolonged antibiotic therapy. Indium-labelled scintigraphy (ie, a tagged WBC scan) has been reported to potentially distinguish between acute prostatitis and men with lower urinary tract symptoms without prostatitis [51]. However, more robust clinical studies to demonstrate its utility—and whether routine use of scintigraphy to diagnose ABP would represent high-value care—are needed.

Chronic Bacterial Prostatitis

Physical Examination for Chronic Bacterial Prostatitis

Unlike ABP, the physical exam has limited utility in the diagnosis of CBP. The prostate may be normal, boggy, or tender on exam.

Laboratory Studies for Chronic Bacterial Prostatitis

Serum WBC, ESR, and CRP may be normal and do not have an established role in diagnosing CBP. PSA is elevated only in a

Table 3. Dosing of Intravenous Antibiotics in the Treatment of Bacterial Prostatitis[a]

Medication	Suggested Dosing Regimen[b]
Intravenous antibiotics	
Cefepime	1–2 g every 8–12 h
Cefiderocol	2 g (infused over 3 h) every 8 h
Cefotaxime	1–2 g every 8 h
Ceftazidime	1–2 g every 8 h
Ceftazidime–avibactam	2.5 g (infused over 2 h) every 8 h
Ceftolozane–tazobactam	1.5 g every 8 h
Ceftriaxone	1–2 g once per day
Ertapenem	1 g once per day
Imipenem–cilastatin	500 mg every 6 h or 1 g every 8 h
Imipenem–cilastatin–relebactam	1.25 g every 6 h
Meropenem	1 g every 8 h
Meropenem–vaborbactam	4 g (infused over 3 h) every 8 h
Piperacillin–tazobactam	4.5 g every 6–8 h
Oral antibiotics	
Amoxicillin	1 g 3 times per day
Amoxicillin–clavulanate	875/125 mg every 8–12 h
Azithromycin	500 mg 3 times per week
Ciprofloxacin	500–750 mg every 12 h
Doxycycline	100 mg every 12 h
Fosfomycin	3 g every 24–48 h
Levofloxacin	500–750 mg once per day
Trimethoprim–sulfamethoxazole	160 mg/800 mg (1 double-strength tablet) every 12 h

[a]Dosing guidance extrapolated from other studies; trials specifically of prostatitis are limited.
[b]Dosing should be modified for altered renal function as required.

minority of patients with CBP and is not a useful rule-in or rule-out test [39]. Further study is needed to determine if PSA could potentially be used to monitor for treatment failure, as a decrease in PSA while a patient is on antimicrobial therapy has been associated with microbiologic cure.

The historic gold standard for differentiating chronic bacterial from noninfectious prostatitis was the Meares–Stamey 4-glass test [52]. This procedure, not frequently performed due to the time involved and challenges for both patient and clinician, entails collection of 4 serial samples. The initial sample collected is 10 mL of first-void urine after cleansing the head of the penis; the next sample is 10 mL of midstream urine; after this, the clinician performs prostatic massage, and a third fluid sample (the expressed prostatic secretions) is collected; immediately after, the fourth and final sample, another 10 mL of urine, is collected. Each of these 4 samples is sent for culture. The 4-glass test is considered positive if at least 10-fold more bacteria are found in either the third or fourth samples (the postmassage fluid) versus the initial urine samples [53]. The test is also considered positive if the postmassage sample grows uropathogenic bacteria while the premassage sample was negative [53].

The modified 2-glass test, which compares a premassage midstream urine sample to postmassage urine sample, is simpler than the 4-glass test and can also be used for diagnosing CBP. The 2-glass approach, while less sensitive, still arrives at a result concordant with the 4-glass test in 96% of cases and retains 100% specificity for CBP [53]. The 2-glass test is diagnostic of CBP when the postmassage sample has ≥10-fold greater CFU/mL of a uropathogen compared with the premassage sample [53]. The test is also considered positive when the postmassage sample grows a uropathogen while the premassage sample is negative [53].

To note, the presence of pyuria (>10 WBCs/HPF) in urine samples can suggest ongoing prostatic inflammation but does not confirm a diagnosis of CBP [54]. Such information might be helpful in settings where patients who have received recent antibiotic therapy. Neither the 4-glass nor the 2-glass test should be performed within 72 hours of last ejaculation, which temporarily elevates the prostatic fluid leukocyte count [55].

A 5-glass version of the Meares–Stamey test has also been proposed, where the fifth culture is a semen culture. Studies report higher sensitivity with this method compared with the 4-glass test (97% vs 82%), with notable increased sensitivity for Gram-positive (100% vs 16%) versus Gram-negative (97% vs 82%) pathogens [9, 56]. While these studies report a significant improvement in the negative predictive value of 5-glass compared with the 4-glass test (99% vs 85%), this advantage must be weighed against an increased rate of false-positive results from Gram-positive skin-flora contamination due to the semen collection method. Moreover, treatment outcomes (microbiological eradication and long-term symptom relief) did not significantly differ between groups diagnosed with the 2-, 4-, or 5-glass version of the Meares–Stamey test in a retrospective cohort of 696 symptomatic patients [57]. In the case of growth of a nontypical uropathogen, repeat culture is typically warranted prior to initiating treatment. By the same token, semen culture alone has not been studied for and is not recommended for CBP diagnosis.

For practical diagnostic purposes, we recommend obtaining urinalyses and urine cultures using the Meares–Stamey 2-glass or 4-glass testing approach.

Imaging for Chronic Bacterial Prostatitis

No studies have demonstrated utility of imaging in the diagnosis of CBP. With MRI, even when abnormalities are present, it is difficult to distinguish infection from other etiologies such as prostate cancer [58]. Transrectal ultrasound (TRUS) can also be used to detect prostatic abnormalities, although this imaging modality is typically performed in the outpatient setting by a urologist or radiologist with expertise. Importantly, TRUS does not distinguish between CBP, inflammatory pelvic pain syndrome, and noninflammatory pelvic pain syndrome [59].

WHAT IS THE PREFERRED ANTIMICROBIAL THERAPY FOR ACUTE BACTERIAL PROSTATITIS AND CHRONIC BACTERIAL PROSTATITIS? WHAT IS THE RECOMMENDED DURATION OF TREATMENT?

The prostate gland has a nonfenestrated capillary bed. Only lipid-soluble drugs, such as fluoroquinolones, traverse these

capillaries readily if there is no acute inflammation [60, 61]. Additionally, pH differences can cause mean antibiotic concentrations in prostatic secretions to differ from those in the plasma at equilibrium. Particularly in CBP, because of relatively lower inflammation, selection of an appropriate antimicrobial agent with optimal pharmacokinetics for prostatic tissue is important [61].

Although there are no prospective randomized controlled trials for treatment of ABP, a variety of antibiotics can be used for initial empirical treatment, such as broad-spectrum penicillins (eg, piperacillin/tazobactam), third-generation cephalosporins, or a fluoroquinolone (Table 3). Antibiotics can subsequently be tailored based upon microbiologic results. Guidelines from EAU recommend that patients who initiated intravenous antibiotics can be transitioned to oral therapy after improvement in infection parameters, such as fever curve and other vital signs [42]. If the isolate is susceptible, fluoroquinolones or trimethoprim/sulfamethoxazole are considered the main oral options for step-down oral therapy for ABP.

The optimal duration of antimicrobial therapy for ABP is not established. In one retrospective analysis conducted in 437 patients with ABP, performing cystostomy, avoiding urethral catheterization, and a 9-day increase in duration of antibiotics (resulting in a mean of 36 days) were independently associated with a lower likelihood of developing chronic infection [62]. A recent multicenter trial of 7 versus 14 days of antibiotics for febrile UTI in men with symptoms of cystitis or prostatitis found the 7-day course of therapy inferior to 14 days [63]. However, more than 95% of patients in each arm experienced short-term clinical cure, and the overall outcome was driven by solely microbiologic failures (ie, persistently positive urine cultures in patients whose febrile illness had improved). This trial did not distinguish between pyelonephritis or prostatitis as possible reasons for febrile UTI, but did include patients with prostatitis, which many prior trials had not done. Additionally, Van Nieuwkoop et al [64] observed in a clinical trial (also including men with ABP) that antibiotic therapy for 7 rather than 14 days yielded similar rates of treatment success in febrile women with UTI, whereas febrile men with UTI had lower rates of clinical cure with shorter therapy. Both studies observed nonsignificant trends toward treatment failure with shorter courses of therapy amongst patients with bacteremia. These trials suggest that clinically significant involvement of the prostate can occur in febrile UTI, and when present, benefits from a longer (ie, at least 2 weeks) course of antibiotic therapy. Based on these data, we advocate for treating ABP for at least 2 weeks; longer durations might be reasonable for some patients, although prospective data demonstrating a benefit for durations longer than 2 weeks are lacking.

A Cochrane review from 2013 identified 18 randomized studies of antimicrobial therapy for CBP with a total of 2196 participants [65]. The review found that the microbiological and clinical efficacies and safety profiles of different oral fluoroquinolones were comparable. Fluoroquinolones represent the therapy for CBP with the largest evidence base, with far more limited data from randomized controlled trials to support alternative agents. The identified trials used fluoroquinolones for 4 or 6 weeks. Fluoroquinolones were also compared with nonfluoroquinolones, namely doxycycline, minocycline, carbenicillin, trimethoprim–sulfamethoxazole, and azithromycin (which was studied for CBP caused by nontraditional pathogens, such as *Chlamydia* or *Ureaplasma*); none of these agents were clearly noninferior or superior to fluoroquinolone antibiotics in the treatment of CBP caused by traditional pathogens, and the evidence supporting their use in this setting was limited. Despite these limitations, trimethoprim/sulfamethoxazole is considered a leading alternative agent to fluoroquinolones if use of a fluoroquinolone is not possible [66, 67]. For example, in a cost-effectiveness modeling paper that reviewed prior studies of trimethoprim/sulfamethoxazole for treatment of CBP, 5 small studies with 15–19 patients each had a clinical cure rate ranging from 68 with –100% [68].

Fosfomycin use for CBP has been reported in published cohort studies and a phase I-II trial using an oral dose of 3 g daily or every 48 hours for a duration ranging from 2 to 12 weeks, with ~80% microbiological cure achieved overall [69–72]. Therefore, oral fosfomycin could be an option in patients with CBP when other antibiotics cannot be used [73]. In patients with CBP caused by *C. trachomatis*, previous work suggested that macrolides had higher microbiological and clinical cure rates compared with fluoroquinolones [74]. More recent data suggest that use of doxycycline rather than azithromycin results in better outcomes for patients with urethritis and rectal infection due to *C. trachomatis*; whether this is also true for prostatitis is not known [75, 76].

WHEN IS UROLOGICAL INTERVENTION NECESSARY FOR ACUTE BACTERIAL PROSTATITIS AND CHRONIC BACTERIAL PROSTATITIS?

Most cases of ABP and CBP can be managed without an invasive procedure. The primary indications for surgery in these patients are (1) prostatic abscesses, many of which are unlikely to fully resolve with antibiotics alone; and (2) CBP that has been refractory to multiple courses of appropriate antimicrobial therapy or in which uropathogens are resistant to the antibiotics of choice.

Contemporary studies of prostatic abscess report that source-control interventions are performed in 57%–93% of cases, most often consisting of abscess aspiration, but also sometimes drain placement, surgical unroofing/debridement, and more rarely TURP or radical prostatectomy [77–82]. The size of prostatic abscess ranged from <1 to 5 cm. In these studies, successful nonoperative management of prostatic abscess was achieved

in a minority of patients (usually around 30%). We recommend nonoperative management of prostate abscess only for patients who have a small (ie, no more than 1–2 cm) single fluid collection, who are responding well to systemic antibiotics, and for whom short-term follow-up with repeat imaging is feasible.

In patients with recurrent UTIs and/or persistent prostatic symptoms due to proven CBP after an initial 6-week course of antibiotics, potential management options include repeating the same antibiotic regimen (potentially more than once), using the same antibiotic but increasing the duration (eg, to 12 weeks of therapy), or utilizing a different antibiotic from a different pharmacological class. Exactly which subsequent approach to take and when has not been systematically evaluated. In practice, a clinician will need to individualize the management approach for each patient depending upon the details of the case.

From a surgical standpoint, for refractory CBP cases, TURP or transurethral prostate enucleation can be offered. Radical prostatectomy represents a more invasive approach with the risk of postoperative erectile dysfunction and/or urinary incontinence. Radical prostatectomy should be performed only in patients with infections extending beyond the prostatic capsule, concomitant prostate cancer, prior failed endoscopic treatment, or prior life-threatening UTI. Data supporting the value of prostatectomy in CBP is limited to a handful of small noncomparative case series that are heterogenous in their documentation of the CBP diagnosis, treatment success criteria, and duration of follow-up [83–85]. Nevertheless, these studies suggest clinical cure (variously defined as freedom from recurrent UTIs or resolution of prostatic pain, dysuria, or sexual symptoms) can be achieved in 67%–85% of cases.

FUTURE RESEARCH DIRECTIONS

In developing this state-of-the-art review, we identified several key limitations of the current body of bacterial prostatitis research. Among these, the most pressing clinical questions include:

- What are updated risk factors for ABP and CBP?
- What is the modern microbiological epidemiology of ABP and CBP?
- What are the evidence-based symptoms and signs of ABP and CBP?
- What is the role of radiologic imaging in the diagnosis of ABP and CBP?
- How often does a man with febrile UTI have clinically significant involvement of the prostate?
- What is the randomized controlled trial-defined optimal duration of antibiotics for patients with ABP? (ie, do 2- vs 4-week regimens produce similar rates of long-term cure without progression to CBP?)
- What is the randomized controlled trial-defined optimal duration of antibiotics for patients with CBP? (ie, do 2-week, 4-week, or longer regimens produce similar rates of long-term cure?)
- What role, if any, can PSA serve in the diagnosis or monitoring of patients with ABP or CBP?
- What is the optimal antimicrobial therapy (ie, choice and duration of antibiotic) for patients with imaging findings of acute prostatic inflammation without abscess and a normal DRE?
- What is the optimal antibiotic regimen for CBP in patients who are unable to receive fluoroquinolones or trimethoprim/sulfamethoxazole because of side effects or infection due to resistant pathogens?

REVISITING THE TWO INTRODUCTORY CASES

The first case represents a typical clinical presentation for ABP. Physical examination identified prostate tenderness, and the organism recovered from both the urine and the blood was a pan-sensitive *E. coli* isolate. The patient was started on ceftriaxone while awaiting culture results. Because he was having difficulty voiding, Urology was formally consulted to evaluate the patient. A Coude catheter was placed with care (indiscriminate placement of a standard Foley catheter past a severely inflamed prostate can cause harm in some instances). By the time the sensitivity data had become available, the patient's fever had resolved, his vital signs were stable, and he was able to take oral medications. As this patient had previously suffered Achilles tendonitis, and since his renal function was normal, he received trimethoprim/sulfamethoxazole 160/800 mg twice per day to complete a 14-day course of antibiotic treatment. He passed a voiding trial prior to discharge and went home without an indwelling urinary catheter.

The second case was a representative example of CBP. The causative organism proved to be an *E. coli* expressing extended spectrum beta-lactamase but was sensitive to fluoroquinolones. He received a 6-week course of ciprofloxacin 750 mg twice daily. He also reported a history of 6–12 months of difficulty starting his urinary stream and a sensation of incomplete voiding for which he was referred to Urology. He was ultimately diagnosed with benign prostatic hypertrophy causing significant urinary retention. His pelvic pain symptoms resolved during the 6-week course of ciprofloxacin, and he subsequently underwent TURP to improve his voiding and lower urinary tract symptoms.

CONCLUSIONS

Accurately diagnosing and managing ABP and CBP (and understanding the distinction between the 2 clinical entities) are important for any clinician who takes care of men with UTI. It is possible for ABP to simply be labeled as "complicated

UTI" or "febrile UTI," but making a specific diagnosis of ABP has important implications regarding duration of antimicrobial therapy and follow-up for the patient. Although there is no single gold-standard test for diagnosing ABP, we propose the presence of acute urinary and systemic symptoms in a patient with a tender or fluctuant prostate on DRE as a simple and practical clinical definition. CBP can also be a challenging diagnosis to make, particularly because of some clinical overlap with CPPS. A practical way to diagnose CBP is the 2-glass Meares–Stamey test. Patients with CBP likely need a more prolonged course of antimicrobial therapy, and refractory cases might benefit from surgical intervention. Bacterial prostatitis research is currently quite limited, with even basic clinical management questions addressed primarily via small, observational, and often noncomparative studies; better quality evidence, including randomized clinical trials, are greatly needed.

Notes

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