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Guideline

AAUS guideline for acute bacterial prostatitis 2021

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1. Executive summary

1.1. Epidemiology and pathogenesis

1) Prostatitis syndromes are a very common presentation in the clinical setting and tend to occur in young and middle-aged men.

1.2. Diagnosis

1) Patients with acute bacterial prostatitis (ABP) present with typical signs and symptoms of an acute urinary tract infection including irritative and/or obstructive voiding complaints and have additional symptoms of systemic infections such as malaise, nausea, vomiting, chills, and fever and sometimes present with signs of urosepsis symptoms. They also complain of perineal and suprapubic pain due to a painful swollen prostate and may have associated pain or discomfort of the external genitalia (grades of guideline recommendations [GR]: B).
2) Digital rectal examination (DRE) reveals a hot, boggy, and exquisitely tender prostate gland (GR: B).
3) Prostatic massage for the prostatic fluid expression is not indicated and perhaps even harmful because it could precipitate bacteremia or sepsis (GR: B).
4) A midstream urine specimen is sufficient and will show prominent leukocyturia and bacteriuria with typical uropathogens (GR: B).
5) The sonographic determination of residual volume is an important diagnostic procedure because infravesical obstruction may play an important pathogenic role in ABP (GR: B).
6) Transrectal ultrasound (TRUS) does not need to be performed on every patient with suspected ABP (GR: B) but can aid in the diagnosis or exclusion of a prostatic abscess (GR: B).
7) Computed tomography (CT) and magnetic resonance imaging (MRI) offer no advantage over TRUS unless the abscess has penetrated the confines of the prostate gland or further abscess foci is suspected (GR: B).
8) Prostate-specific antigen (PSA) is moderate to markedly elevated in a patient with ABP. However, it is not a diagnostic requirement (GR: C).
9) Serial measurement is recommended as a useful tool for follow-up in patients with elevated PSA (GR: B).
10) *Escherichia coli* is the most common pathogen encountered in ABP (GR: B).
11) A significantly higher rate of mixed infection in prostatitis from prior manipulation exist compared with spontaneous ABP (GR: B).

1.3. Treatment

1) Rapid initiation of broad-spectrum parenteral antibiotics and symptomatic support are mandatory for patients with ABP (GR: B).
2) Supportive measures include intravenous hydration and catheter drainage if the patient cannot void (GR: B).
3) Insertion of a suprapubic cystostomy tube is the optimal therapy for relief from urinary obstruction (GR: B).
4) In-and-out catheterization to relieve the initial obstruction or short-term (<12 h) indwelling catheterization with a small-caliber Foley catheter is appropriate (GR: B).

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5) The selection and course of antibiotics should be adjusted according to the isolated pathogens and the results of bacterial susceptibility testing (GR: B).

1.4. Prostatic abscess

1) Suspicion of a developing abscess is raised if no response is noted in appropriate antibiotic therapy confirmed by TRUS (GR: B).
2) Initiation of broad-spectrum antibiotics and prompt surgical drainage is crucial if a prostatic abscess is discovered (GR: B).

2. Introduction

This guideline is supported by the Asian Association of Urinary Tract Infection and Sexually Transmitted Infection (AAUS). This is the second version of the AAUS guideline for acute bacterial prostatitis (ABP) (the first version has not been published). ABP constitutes a urologic emergency. It is uncommon and usually occurs in concert with a urinary tract infection (UTI) but can have a dramatic presentation. Many clinicians usually diagnose and empirically treat ABP. A correct diagnosis has therapeutic implications because ABP may require a longer treatment course than other forms of UTI and because the choice of antibiotic to complete treatment after fever subsides should be based on its ability to penetrate prostatic tissue. Thus, defining detailed and consensual diagnosis and ABP treatment criteria is of great necessity.

3. Methodology

The extensive literature regarding ABP available in Medline via PubMed, Google Scholar, and Scopus was surveyed. Other relevant publications up to December 2020 are also considered. The terms *prostatitis*, *bacterial prostatitis*, and *ABP* were used combined with the terms *diagnosis*, *evaluation*, *management*, or *treatment* for the search strategy. Moreover, 70 possibly eligible publications, which were screened by title and abstracts, were included in the analysis for this review. The limitations for this literature search were the English language. The papers were rated according to the level of evidence and the strength of recommendations according to the International Consultation on Urological Diseases standards [1,2].

Level of evidence and grade of guideline recommendation was made according to the following strategy.

3.1. Levels of evidence (LE)

Level	Type of evidence
1a	Evidence obtained from meta-analysis of randomized trials
1b	Evidence obtained from at least one randomized trial
2a	Evidence obtained from one well-designed controlled study without randomization
2b	Evidence obtained from at least one other type of well-designed quasi-experimental study
3	Evidence obtained from well-designed nonexperimental studies (e.g., comparative studies, correlation studies, and case reports)
4	Evidence obtained from expert committee reports or opinions or clinical experience of respected authorities

3.2. GR grades

Grade	Nature of recommendations
A	Based on clinical studies of good quality and consistency addressing the specific recommendations and including at least one randomized trial
B	Based on well-conducted clinical studies but without randomized clinical trials
C	Made despite the absence of directly applicable clinical studies of good quality

4. Definition of the disease

4.1. Overview

ABP represents an acute infection of the prostate gland. It is a male UTI that has some features in common with lower UTI in females including the etiological organisms involved and some of their urovirulence factors. However, the host response is very different from simple cystitis, and the treatment course is more complicated [3].

Prostatitis syndrome is a common healthcare issue affecting 10%–14% of men of all ages and ethnicities. Moreover, ABP is associated with severe, mainly Gram-negative infection [4].

In 1999, the National Institutes of Health (NIH) consensus statement on prostatitis, prostatitis, and prostatitis-like symptoms was classified into four broad categories [5]. Type I prostatitis refers to ABP.

4.2. Epidemiology

Overall, prostatitis syndromes are a very common presentation in the clinical setting and tend to occur in young and middle-aged men. However, ABP accounts for the rarest category among the NIH classification. It is diagnosed in less than 0.02% of all patients seen for prostatitis [6]. However, the potential morbidity and mortality of ABP constitute a true urologic emergency. It is characterized by an acute pain onset combined with irritative and obstructive voiding symptoms in a patient with manifestations of a systemic febrile illness. The hospital admission rate for ABP in the USA in 1994 was 12.7 in 100,000 as of the first, second, or third principal diagnosis for hospital admission [7] (LE: 4).

5. Characterization

5.1. Etiology

ABP is the result of severe infection of mainly Gram-negative bacteria, which can be easily isolated from the urine. In addition, *E. coli* is the most common pathogen encountered in ABP, accounting for 50%–87% of cases. Other pathogens include Enterobacterales (e.g., *Klebsiella* and *Proteus* species; 10%–30% of cases), nonfermenting Gram-negative bacilli (e.g., *Pseudomonas* species; 5%–15% of cases), and *Enterococcus* species (5%–10% of cases) [8–15] (LE: 3). Infections are commonly caused by a single organism, but occasionally by two or more organisms [15]. Mixed cultures were isolated in 2.4% of the ABP [14] (LE: 3). *Neisseria gonorrhoeae* and *Chlamydia trachomatis* should be considered in sexually active men [13,16,17] (LE: 4).

5.2. Risk factors

The ABP mechanisms are reflux of infected urine into the ejaculatory and prostatic ducts, an ascending urethral infection from the distal urethra, meatus and bladder direct or lymphatic invasion from the rectum, and hematogenous infection [15,18] (LE: 4). Although many patients with ABP have no clear risk factors, the underlying functional or anatomical anomalies that predispose to urogenital infections may affect the development of prostatitis. Prior manipulation of the lower urinary tract including chronic indwelling bladder catheterization, intermittent bladder catheterization, urodynamic study, transurethral surgery, and transrectal prostate biopsy (TRPB) could be predisposing factors [14,16,19–22] (LE: 3). Patient populations who are at especially high risk of ABP include those with diabetes, cirrhosis, and suppressed immune systems [23] (LE: 4).

6. Clinical evaluation/risk assessment

6.1. Diagnostic and staging procedures

ABP may be difficult to diagnose. Several conditions (e.g., benign prostatic hypertrophy, chronic bacterial prostatitis (CBP), chronic pelvic pain syndrome (CPPS), cystitis, diverticulitis, epididymitis, orchitis, proctitis, and prostate cancer) present with similar symptoms and must be differentiated from ABP [16]. Patients with ABP symptoms have to undergo urinalysis and urine culture. Initial prostate imaging is suggested to exclude prostatic abscess [24] (LE: 4).

1) Symptoms and signs

No common key ABP symptoms exist. The NIH revised the classification of prostatitis to deal more with the pathophysiology and laboratory diagnosis and describes the clinical features of ABP only briefly as *acute symptoms of a UTI* [5] (LE: 4). Irritative and/or obstructive voiding complaints including dysuria, urinary frequency, and urgency are typical [5] (LE:4). Obstructive voiding complaints including hesitancy, poor interrupted stream, and even acute urinary retention are common. The patients complain of perineal and suprapubic pain and may have associated pain or discomfort of the external genitalia. The patients have a swollen prostate that is extremely painful when investigated. Patients with ABP are often acutely ill and distressed. Symptoms of systemic infection such as malaise, nausea, vomiting, chills, and fever may vary. These patients may even be systemically toxic, i.e., flushed, febrile, tachycardic, tachypneic, hypotensive, and present even with all signs and symptoms of urosepsis [5,8,15,16,25] (LE: 4).

DRE reveals a hot, boggy, exquisitely tender, and tense prostate gland. Fluctuation during palpation is suspicious for prostatic abscess. Painful ejaculation, hematospermia, and painful defecation may be present as well [3,16]. Older patients also had fewer signs and symptoms (less burning micturition, less painful DRE, and less hematuria) [13] (LE: 4). Perineal pain and anal sphincter spasm may complicate DRE [26] (LE: 4). Although a gentle rectal examination can be performed in patients who have suspected ABP, prostatic massage for the expression of prostatic fluid is not indicated and perhaps even harmful because it is painful for the patient and could precipitate bacteremia or sepsis [4,8,14,16,27] (LE: 4).

2) Clinical findings

A midstream urine specimen will show prominent leukocyturia and bacteriuria in patients presenting with ABP. Midstream urine culture is considered the only laboratory evaluation of the lower urinary tract and usually shows typical uropathogens [24] (LE: 4).

Blood culture and complete blood count are useful in ABP [27] (LE: 4). In particular, blood cultures should be collected before initiating antibiotics in patients with high fever [28] (LE: 3), with a possible hematogenous infection source (e.g., endocarditis with *Staphylococcus aureus*), with complicated infections (e.g., sepsis), or immunocompromised [16] (LE: 4). A study suggested that a temperature of >38.4 °C may be a predictor of positive blood culture in patients with ABP [28] (LE: 3). However, blood culture should be obtained if patients are systemically ill.

3) Functional findings

The sonographic determination of residual volume is an essential diagnostic procedure because intravesical obstruction may play an important pathogenic role in ABP [3] (LE: 3). The difference in voiding problems may reflect age differences and prostate volume. In addition, the frequency of voiding problems, particularly urinary retention, was remarkably higher in the group that underwent prior manipulation [22] (LE: 3). Some investigators suggested that bladder outlet obstruction may not be the main cause of ABP [14] (LE: 3). Thus, more evidence is necessary to clarify the relationship between obstructive bladder dysfunction and ABP.

4) Imaging studies

Only a few studies that describe ultrasonographic findings in non-abscessed ABP exist [29–32]. In a prospective study of 45 patients with a clinical ABP diagnosis, TRUS was performed upon admission as well as 1 month after antibiotic therapy. Moreover, the findings correlated with the clinical disease presentation [33]. The authors conclude that TRUS does not need to be performed on every patient with suspected ABP (as only 47% had sonographically demonstrable lesions on admission and 61% had improved or disappeared lesions post-treatment), but TRUS would be indicated to exclude the presence of prostatic abscess (LE: 2a, GR: B). In conclusion, carefully performed TRUS can aid in the diagnosis or exclusion of a prostatic abscess without increasing the risk for urosepsis [33] (LE: 2a, GR: B).

Some reports indicate that the more cost-intensive CT and MRI offer no advantage over TRUS unless the abscess has penetrated the confines of the prostate gland or further abscess foci is suspected [3,34] (LE: 3).

Color Doppler sonography is a useful tool in monitoring the response to treatment and in predicting clinical outcomes [35] (LE: 3). In some papers, investigators stated that intraprostatic color flow in patients with ABP was greater than in the normal prostate or those with chronic inflammation or carcinoma [36] (LE: 3). According to the study, the vascularity of the prostate in most of the patients was increased in the acute inflammation phase (15-spot scale). Color flow in the prostate decreased to 15 spots in healthy men with infection recovery [37] (LE: 3).

Hypoechoic areas in the peripheral prostate zone can persist for a long time in patients with ABP. Color Doppler ultrasonography of these areas can help differentiate them from those with carcinoma [38].

Two interesting imaging studies also exist—one performed with prostatic indium-labeled leukocyte scintigraphy and one performed with a combination of PSA levels and TRUS-provided evidence—supporting the frequent involvement of the prostate in male UTI [13,39,40] (LE: 2a).

The former was carried out to determine whether indium-111 (111In)-labeled leukocytes (ILLs) accumulated in the infected tissue and whether uptake responded to treatment [41]. Scintigraphy before antibiotic treatment showed uptake in prostates of all patients with ABP. No uptake was noted in nine of 10 patients after treatment. Moreover, one of 10 patients had markedly decreased uptake. No uptake occurred in prostates in patients with UTI if no involvement in the prostate exists. Thus, ILLs could be useful for detecting ABP in the future [24].

The latter showed that the prostate is concurrently involved in men with febrile UTI with a transient increase in prostate volume and serum PSA during the acute disease stage [32,39] (LE: 3). The presence of an inflammatory reaction within the prostate with these two concepts can provide information on ABP when the diagnosis is unclear [13].

5) Serum PSA

Although PSA testing is not usually recommended for ABP, PSA levels are generally moderately to markedly elevated in the ABP setting [42–44] (LE: 3). The role of serum PSA in the differential diagnosis and evaluation of ABP is unclear. However, elevated PSA levels have been described in 70% of men with ABP [45] (LE: 3) as a consequence of increased vascular permeability and disrupted gland epithelium. In a prospective study of 39 men with pyrexia (>38.3 °C), serum PSA levels were used to categorize patients according to an initial diagnosis of ABP, pyelonephritis, urogenital infection, or fever of unknown origin. All of the 20 cases with pyrexia and elevated PSA were diagnosed and treated as ABP [42].

Game' et al. demonstrated a decreased free-to-total (f/t) PSA ratio up

to 30 days following adequate antimicrobial therapy, indicating the significance of increased-bound PSA in ABP [46] (LE: 3). The decrease of f/t PSA ratio has been correlated to systemic inflammation as measured by serum C-reactive protein (CRP) levels. This marker has been proven useful to assess prostatic infection in a prospective study of 70 men with febrile UTI with prostatic involvement as measured by total PSA (tPSA). Effective treatment with antibiotics resulted in significantly reduced serum PSA. Moreover, a decline in tPSA levels in patients after appropriate antimicrobial treatment has been suggested to indicate a healing process [32] (LE: 3).

Thus, the authors recommend PSA as a concise, accurate, rapid, and cost-effective tool for identifying ABP and for follow-up [24] (GR: B).

6.2. Principles of management and treatment

1) Hospitalization criteria

ABP management should be based on the severity of symptoms, risk factors (benign prostatic hypertrophy, genitourinary infections, high-risk sexual behavior, history of sexually transmitted diseases, immunocompromised states, phimosis, prostate manipulation, and urethral stricture), and local antibiotic resistance patterns [16]. Most patients can be treated with outpatient antibiotics, and fewer than one in six patients will require hospitalization [14,16]. Hospitalization should be considered in failed outpatient management, inability to tolerate oral intake, resistance risk factors, recent fluoroquinolone use, recent transurethral or transrectal prostatic manipulation, systemically ill or septicemia, and urinary retention [16] (LE: 4).

2) Use of antibiotics

No randomized controlled trial exists on antibiotic selection and antibiotic use duration [47]. The treatment regimen for ABP is based on clinical experience and a number of uncontrolled clinical studies. Parenteral antibiotic therapy is preferable for systemically ill patients with ABP [27,48] (LE: 4).

Appropriate ABP management includes rapid initiation of broad-spectrum parenteral antibiotics and symptomatic support. Current guidelines for ABP treatment have been worked out by the European Association of Urology, the Korean Society of Infectious Diseases/Korean Society for Chemotherapy, the National Institute for Health and Care Excellence, and the Japanese Association for Infectious Disease/Japanese Society of Chemotherapy [27,47–49].

Pharmacologic penetration of antibiotics in the acutely inflamed prostatic tissue is considered to be sufficient in the case of susceptible bacteria. In severe cases, parenteral administration of high doses of bactericidal antibiotics (e.g., a broad-spectrum penicillin derivative and a third-generation cephalosporin with or without aminoglycosides) or a fluoroquinolone is required until fever and other parameters of acute infection are normalized. It can be performed alone or in combination with supportive measures including intravenous hydration and catheter drainage if the patient cannot void [16,22,24,50] (LE: 4).

An oral fluoroquinolone for 10 days may be sufficient in less severe cases [3] (LE: 4). The selection and course of antibiotics can be adjusted according to the isolated pathogens and the results of bacterial susceptibility testing [16,27,47,48].

The fever resolves in 36–48 h in most cases [38,51] (LE: 2a-4). Switching to an oral regimen (e.g., fluoroquinolone) is appropriate after a successful initial therapy. Moreover, oral antibiotic therapy should be continued at least for 2–4 weeks [16,27,47–49] (LE: 4) although no consensus currently exists on optimal treatment duration.

3) Antibiotic resistance

In recent guidelines for antibiotic ABP treatment, the administration of cephalosporins or a quinolone alone or in combination with an aminoglycoside has been recommended [27,47–49] (LE: 4). Third-generation cephalosporins, a broad-spectrum beta-lactam/beta--lactamase inhibitor (BLI), or carbapenem are recommended for patients with ABP requiring hospitalization or if the resistance of the causative bacteria to fluoroquinolone is considered [47] (LE: 4).

The *E. coli* susceptibility to ciprofloxacin was shown to be relatively low (76.2%) for ABP in some Korean areas between 2001 and 2005 [22] (LE: 3). Such a result probably reflects the increase in resistant bacteria owing to the excessive use of ciprofloxacin in that local area.

As in the previous report, ciprofloxacin alone may be an inadequate choice, especially in patients with prior urinary tract manipulation. Considering the very low susceptibility to cephalosporins (<60%) in pathogens other than *E. coli* and the relatively high isolation rates (>40%) of pathogens other than *E. coli*, cephalosporins, as single therapeutic agents, may have limited use in this community. Antibiotic combination therapy for ABP most commonly includes a cephalosporin and an aminoglycoside. The second- and third-generation cephalosporins have been relatively prescribed frequently for this purpose. Administration of an aminoglycoside must be confined to the group of patients without prior manipulation owing to their susceptibility. In the group of patients with prior manipulation in which pathogens other than *E. coli* constitute a substantial number of isolates, a combination of cephalosporin and amikacin should be recommended for empirical therapy [22] (GR: B).

The use of levofloxacin could be an ABP risk factor after TRPB owing to an increase in fluoroquinolone-resistant *E. coli* in the rectum. Treatment with cephalosporin or carbapenem is recommended for patients with ABP after prostate biopsy [52] (GR: C).

History of prior urologic manipulation was an independent risk factor for ciprofloxacin-resistant and extended-spectrum beta-lactamase (ESBL)-producing microbes. Advanced age (>60 years) was an independent risk factor for ciprofloxacin-resistant microbes [53] (LE: 4).

The ciprofloxacin susceptibility for *E. coli* in groups without prior manipulation was documented as 85.7% for ABP in a single Korean institution between 2006 and 2015. The susceptibility was 10.0% for groups with prior manipulation [53] (LE: 4).

For ABP in a single Korean institution between 2006 and 2015, the incidence of ESBL-producing microbes by pathogens for *E. coli* and *Klebsiella pneumonia* was 3.8% and 1.0% in the absence of a manipulation group and 20% and 33.3% in the presence of a manipulation group, respectively [53] (LE: 4). Initial ABP treatment must consider the patient's age and the possibility of prior manipulation to optimize patient treatment. With the high rate of fluoroquinolone resistance, cephalosporins with amikacin, carbapenems, or extended-spectrum penicillin with BLI should be considered as the preferred empirical ABP treatment in patients with history of prior urologic manipulation [53] (LE: 4).

Pseudomonas species were more dominant pathogens in the transurethral manipulation group than in the transrectal manipulation group. The susceptibilities to second-, third-, and fourth-generation cephalosporins, amikacin, carbapenem, and aztreonam were very low in the transurethral manipulation group [54] (LE: 3).

ESBL-producing bacteria accounted for 64.7% of culture-positive patients in the biopsy-related ABP compared to 13.3% in the spontaneous ABP for ABP in single Korean institutions between 2004 and 2013. Biopsy-related ABP showed a higher incidence of septicemia and antibiotic-resistant bacteria than did the spontaneous ABP. These results have important implications for the management and antimicrobial treatment of biopsy-related ABP, which may well deserve to be considered a distinct prostatitis category [55] (LE: 4).

For ABP in Korean institutions between 2005 and 2014, the ABP following TRPB group (59.1%) showed a higher bacteremia prevalence than did the community-acquired ABP group (13.2%). Significant differences in the antibiotic sensitivity to *E. coli* between the two groups were observed for fluoroquinolone, cephalothin, and gentamicin. The antibiotic sensitivity of fluoroquinolone in the ABP following TRPB group was only 27.3%. Amikacin, imipenem, meropenem, amoxicillin/

clavulanic acid, and piperacillin/tazobactam showed >95% antibiotic sensitivity in both groups. ABP following TRPB was an independent predictive factor for bacteremia by multivariate analysis. Furthermore, carbapenem may be a treatment of choice for patients suspected of having sepsis [56] (LE: 3).

4) Treatment for ABP

Initial empiric antimicrobials should be based on risk factors of the drug-resistant bacterium and clinical characteristics. Antimicrobials should be adjusted on the basis of the results of culture testing of the initial urine sample and antimicrobial susceptibility testing. Table 1 shows a summary of the ABP treatment according to the guidelines and previous ABP studies.

5) Additional points to be considered

Other supportive treatment options such as alpha-blockers, antipyretics, or anti-inflammatory agents may be beneficial, although current data are insufficient. Only one animal study exists investigating the effects of levofloxacin on tamsulosin for ABP. In the prostatic tissues, tamsulosin increased the C_{max}, prolonged the $t1/2$, and decreased the clearance rate of levofloxacin. These results indicate that tamsulosin may enhance the effect of levofloxacin in the treatment of bacterial prostatitis without changing the drug concentration in the liver and kidney [57] (LE: 3). Moreover, stool softeners are also recommended [15] (GR: C).

6) Follow-up and monitoring

Elevated PSA is common although the role of serum PSA in differential ABP diagnostic evaluation is not proven. Effective treatment with antibiotics results in significantly reduced serum PSA. Therefore, some authors recommend PSA as a concise, accurate, rapid, and cost-effective tool for identifying ABP and for the follow-up [24,58] (LE: 4).

The long-term response is unclear after antibiotic treatment. A prospective study found that the total serum PSA level was elevated up to 3 months after the ABP episode in 39% of patients [38] (LE: 2a). In this manner, patients with ABP tend to have a persistent infection. Moreover, ABP tends to persist, and bacterial localization cultures should be taken at subsequent follow-up visits for at least 3 months [38]. According to another prospective study, PSA levels could be high even up to 6 months after an acute episode [59] (GR: B).

Morote et al. [60] showed that acute inflammation contributed to PSA increases but did not influence the percentage of free PSA in patients with cancer-free biopsies (LE: 3). Moreover, some patients with carcinoma could be missed during the acute inflammation phase. Therefore, PSA and TRUS monitoring is strongly recommended (GR: B).

Of the 437 ABP patients, 1.3% and 10.5% progressed to CBP and inflammatory CPPS, respectively, according to a retrospective analysis [11] (LE: 3). Patients who developed chronic infection had higher alcohol consumption rate, diabetes, voiding symptoms, prior manipulation rate, enlarged prostate volume, catheterization history rate, and short duration of antibiotic treatment.

Table 1
Treatment for acute bacterial prostatitis.

a. Mild/moderate ABP (outpatient therapy regimen)
(Oral) Fluoroquinolone, cephalosporin, beta-lactams and BLI, trimethoprim/sulfamethoxazole
Choose other antibiotics where quinolone resistance is a concern
b. Severe ABP (inpatient therapy regimen)
(Intravenous) Broad-spectrum beta-lactams and BLI, third-generation cephalosporin, fluoroquinolone plus aminoglycosides, and carbapenem
c. ABP following transrectal prostate biopsy; consideration of fluoroquinolone resistance and ESBL-producing *E. coli*
(Intravenous) Broad-spectrum beta-lactams and BLI, and carbapenem
d. ABP following transurethral manipulation—consideration of *Pseudomonas* species
(Intravenous) Broad-spectrum beta-lactams and BLI, third-generation cephalosporin, and carbapenem

7) Relief from obstruction

Urinary obstruction is a very common symptom in ABP. Bladder scanning for postvoid residual urine is recommended because patients can have significant obstruction from an acutely inflamed prostate. The patient should be initiated on alpha-blocker therapy if the residual urine is < 100 mL. If the residual is large, consideration should be given to the placement of a small urethral or a suprapubic catheter if a short- or longer-term drainage is required, respectively [6,25] (GR: B).

The insertion of a suprapubic cystostomy tube has been traditionally suggested as the optimal therapy because an indwelling Foley catheter may further obstruct urethral ducts, resulting in the potential development of prostatic abscesses [60–62] (LE: 4). However, an in-and-out catheterization to relieve the initial obstruction or short-term (<12 h) indwelling catheterization with a small-caliber Foley catheter is appropriate in most patients [26] (GR: C).

6.3. Special considerations

1) Prostatic abscess

Prostatic abscesses are uncommon but potentially serious manifestations of acute infection of the prostate and demand prompt treatment. It represents a severe complication of ABP with an estimated incidence of 2%–18% [14] (LE: 3) and a mortality rate of 3%–16% [63] (LE: 3).

Antibiotic ABP treatment is simple, but abscess formation is well described and may have devastating sequelae. Its clinical diagnosis is somewhat difficult and a suspicion of a developing abscess is raised if no response to appropriate antibiotic therapy is noted, which can be confirmed by TRUS [33,34] (LE: 4). Moreover, TRUS should not be postponed for >48 h in patients who do not respond to appropriate antibiotic therapy [38] (GR B). CT and MRI are helpful when it is difficult to diagnose prostatic abscesses with TRUS or when TRUS cannot be performed because of pain or discomfort.

Patients who are immunocompromised, especially patients who have HIV/AIDS, seem to be more susceptible to ABP development and the occurrence of a potentially life-threatening prostatic abscess. The incidence rate rises to roughly 14% in those who have developed AIDS [61] (LE: 3). Initiation of broad-spectrum antibiotics and prompt surgical drainage is crucial if a prostatic abscess is discovered [6,62] (GR: C).

2) Microbiology of prostatic abscess

E. coli and *Staphylococcus* species are the most commonly isolated pathogens in prostatic abscess, although other pathogens (e.g., *Mycobacterium tuberculosis*, *Actinomyces*, *Citrobacter*, *Bacteroides fragilis*, *Aeromonas aerophyla*, and *K. pneumonia*) have been reported [10,34,63–68] (LE: 3). Moreover, *Burkholderia pseudomallei* overwhelmingly predominates in the Thai population [64]. Increasing cases as a result of methicillin-resistant *S. aureus* (MRSA), both nosocomial and community-acquired, is a growing concern with >30 cases of prostatic abscess as a result of *S. aureus* (methicillin-resistant and methicillin-sensitive) reported in the literature [63].

3) How to treat the prostatic abscess

The recommended treatment for prostatic abscess consists of broad-spectrum antibiotic coverage and, in most cases, drainage of the abscess. Several surgical procedures have been described to relieve abscess formation. Transurethral incision or resection, suprapubic adenectomy, perineal incision, transrectal or transperineal prostatic puncture, and drainage under sonographic guidance have been applied according to

the location and extent of the abscess [3,34] (GR: C and B).

Transperineal incision and drainage [69] must be considered when the abscess has penetrated beyond the prostatic capsule or penetrated through the levator ani muscle [26] (GR: C).

Although transurethral unroofing and perineal drainage were once the mainstays of surgical drainage, TRUS-guided aspiration of prostatic abscesses has been increasingly used as an effective means for drainage that may avoid potential morbidity associated with transurethral drainage [65,70] (GR: B). Some authors also support urinary diversion with a suprapubic catheter [34,68]. Moreover, the follow-up requires regular TRUS controls [3,34] (GR: C and B).

Patients may be treated conservatively in small abscesses by the administration of antibiotic agents together with the placement of a suprapubic catheter. According to a multicenter retrospective cohort study, patients with abscesses <20 mm in size did not undergo surgery and were cured without any complications [10] (LE: 3). In contrast, patients with abscesses >20 mm who underwent transurethral resection had a shorter duration of antibiotic treatment than those who did not have surgery. Early diagnosis is beneficial because prostatic abscesses require prolonged treatment protocols or even surgical drainage. Surgical drainage procedures (e.g., transurethral prostate resection) were not necessary for all patients with prostate abscesses. However, surgical intervention may have potential merits that reduce the antibiotic exposure period and enhance voiding function in patients with prostatic abscess [10] (LE: 3).

7. Abbreviation

AAUS Asian Association of Urinary Tract Infection and Sexually Transmitted Infection, *ABP* acute bacterial prostatitis, *BLI* beta-lactamase inhibitor, *CBP* chronic bacterial prostatitis, *CPPS* chronic pelvic pain syndrome, *CRP* C-reactive protein, *CT* computed tomography, *DRE* digital rectal examination, *EPS* expressed prostatic secretion, *ESBL* extended-spectrum beta-lactamase, *GR* grades of guideline recommendations, *LE* levels of evidence, *MRI* magnetic resonance imaging, *MRSA* methicillin-resistant *Staphylococcus aureus*, *PSA* prostate-specific antigen, *tPSA* total PSA, *TRPB* transrectal prostate biopsy, *TRUS* transrectal ultrasound, *UTI* urinary tract infection.

ICMJE statement for authorship

All authors meet the ICMJE authorship criteria.

M. Matsumoto was responsible for the acquisition of data, the analysis and interpretation of data, drafting the article or revising it critically for important intellectual content, and final approval of the version to be submitted.

S. Yamamoto was responsible for the conception and design of the study, revising it critically for important intellectual content, and final approval of the version to be submitted.

Declaration of competing interest

None.

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