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Acute Bacterial Prostatitis – a review of current literature

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SUMMARY

Introduction and purpose

Acute bacterial prostatitis (ABP) is a troublesome prostate infection that manifests with a range of irritative urinary symptoms, pelvic discomfort, potentially progressing to urinary retention. ABP can cause severe complications, including prostatic abscess and sepsis. Hence the need to collect and share current knowledge on risk factors, diagnosis and treatment.

A brief description of the state of knowledge

ABP is in most cases caused by *E. Coli*. Symptoms are non-specific and many patients lack identifiable risk factors. It is important to perform cautious digital rectal examination, urine analysis and culture when diagnosing acute bacterial prostatitis. Oral fluoroqinolones are the first-line outpatient treatment for non-complicated cases. Patients presenting symptoms indicative of urosepsis should be hospitalized and treated with intravenous antibiotics. Increasing antimicrobial resistance observed among *Enterobacterales* necessitates the search for new therapeutic agents. Lack of clinical improvement after initial treatment may indicate resistant pathogens or the development of prostatic abscess (PA). PA can be diagnosed with transrectal ultrasound and may require surgical intervention. Without proper treatment ABP can transform to chronic bacterial prostatitis. Transrectal prostate biopsy is associated with higher risk of ABP therefore antibiotical prophylaxis should be used.

Conclusions

ABP is not the most common type of prostatitis, but it should not be overlooked. Better knowledge about risks associated with ABP and available treatment leads to more accurate therapy choices. Awareness of the challenges posed by antibiotic resistance leads to the development of new, more effective treatments.

Keywords: prostate; prostatitis; abscess; prostatic abscess; prostate biopsy; acute bacterial prostatitis.

1. Introduction and purpose

Prostatitis is a frequent and troublesome urinary tract condition, that manifests with a wide range of irritative urinary symptoms, pelvic discomfort, and sexual dysfunction causing significant distress for both patients and healthcare providers worldwide. [1] It is the third most common male urinary tract disorder and probably the most common urinary tract disease in men under the age of 50. [2]

According to The National Institutes of Health (NIH) prostatitis can be classified into four categories based on clinical characteristics. Chronic prostatitis/chronic pelvic pain syndrome (category III) is the most prevalent form and affects nearly 90% of prostatitis patients. [1]

This article focuses on acute bacterial prostatitis (category I) - acute prostate infection that represents up to 10% of all prostatitis cases and typically affects men aged 20 to 40 years, as well as those over 70. [3] Despite the fact that acute bacterial prostatitis (ABP) is not the most common type among NIH prostatitis categories, its significance cannot be overlooked. Without proper management it can cause severe complications, including abscess, sepsis, and septic shock. [4] Therefore the means of this article is to broaden the knowledge on acute bacterial prostatitis in accordance with current literature, thus leading to more precise diagnosis, successful treatment and prevention of complications. The authors of this article aim to gather the most up to date knowledge from various scientific research papers, in order to summarize available information on ABP including its risk factors, etiology treatment and therapeutic options

2. Description of the state of knowledge

Risk factors

Multiple risk factors leading to prostatitis have been identified, but it is worth noting that many patients with ABP lack identifiable predisposing factors. Researchers found that underlying functional or anatomical abnormalities that predispose individuals to urogenital infections can influence the onset of prostatitis. Populations at heightened risk for ABP include those with diabetes, liver cirrhosis, or immunosuppression. [4] Additional risk factors include high-risk sexual behavior, history of sexually transmitted diseases and immunodeficiency. Benign prostatic hypertrophy, epididymitis, orchitis, urethritis, phimosis may be associated with higher risk of prostatitis. [3] Prior interventions involving the lower urinary tract, such as chronic indwelling bladder catheterization, intermittent catheterization, cystoscopy, urodynamic testing, transurethral surgeries, and transrectal prostate biopsies are among contributing factors. [4] The occurrence of ABP following transrectal biopsy, has risen over the past decade, likely due to the growing prevalence of quinolone-resistant *Escherichia coli* within the community. Large study conducted on the incidence of ABP after transrectal biopsy showed that 1.1% patients developed acute prostatitis. On average symptoms manifested 1.28 days after undergoing a prostate biopsy. Researchers also found that the incidence of acute prostatitis was significantly higher in patients who underwent a second biopsy compared to those who had a single procedure. [5]

Anatomy, histology, pathogenesis and etiology

Anatomy and histology

Acute bacterial prostatitis (ABP) is an inflammatory disease of the prostate – complex gland comprising diverse histological structures. It is located beneath the urinary bladder, anterior to the rectum and has seminal vesicles positioned on both sides of its foundation. The structure of the prostate gland consists of the central zone, peripheral zone, and transitional zone.
The central zone encircles the ejaculatory ducts and forms a significant part of the prostate's base. The central area and the distal part of the urethra are mostly enveloped by peripheral zone. The peripheral zone is associated with the majority of prostatitis and prostate cancer cases. The glandular epithelium consists of acini and ducts. The ducts consist of three types of

cells: basal cells, neuroendocrine cells and luminal cells that are specialized for secreting substances such as prostate-specific antigen into the lumen. [6]
The secretion of antibacterial substances and the mechanical flushing of the prostatic urethra during urination and ejaculation are natural defenses against infections of the prostate gland. [1]

Pathogenesis

Ascending urethral infections or intraprostatic reflux cause most cases of ABP. [3] Bacteria can cause ABP through retrograde infection from the external urethral meatus, backflow of contaminated urine into the ejaculatory and prostatic ducts following transurethral procedures. Alternative pathogenic mechanisms include hematogenous spread via bacterial sepsis. Less frequently ABP may result from direct or lymphatic spread from the rectum. [1]
Animal models show that in the first days after infection lesions in the dorsolateral and ventral lobes appear alongside inflammation in the glandular lumens and interstitial tissue. The severity of ABP is mainly influenced by the intensity and concentration of the invading pathogens. [6]

Etiology

Acute bacterial prostatitis is typically caused by Gram-negative bacteria with most infections being a result of a single pathogen infection. In 2,4% cases of ABP mixed cultures were identified. *Escherichia coli* is the most common pathogen in ABP. It is responsible for 50%–87% of ABP cases. [4] In cross-sectional study prostate biopsy confirmed *E. Coli* as the most common pathogen in prostate disease patients. [7] *Pseudomonas aeruginosa* is frequently responsible for prostatitis after transurethral manipulation and it is considered the second most common cause of ABP. *Klebsiella*, *Proteus*, and *Serratia* species are among other common causes of ABP. *Neisseria gonorrhea* and *Chlamydia trachomatis* are typical pathogens responsible for prostatitis in patients with history of high-risk sexual behavior. [2**,** 4]

Diagnostics

Diagnostics of acute bacterial prostatitis may pose a challenge since there are no universally consistent key symptoms, and its presentation can vary widely among patients. Moreover

several conditions, including benign prostatic hyperplasia, chronic bacterial prostatitis (CBP), chronic pelvic pain syndrome, cystitis, diverticulitis, epididymitis, orchitis, proctitis, and prostate cancer, may present with overlapping symptoms and require careful differentiation from acute bacterial prostatitis. [4]

A thorough history and physical examination are adequate for diagnosing acute bacterial prostatitis, but physicians should perform a urinalysis and collect a midstream urine culture to confirm the clinical diagnosis prior to initiating antibiotic treatment**.** Prostate biopsy is contraindicated in these scenarios to prevent the risk of inducing septicemia [3]

Medical history

Obtaining a detailed medical history plays a critical role in the diagnostic process. Patients typically complain of symptoms such as malaise, chills, and sweats, alongside signs of a urinary tract infection, including dysuria, urinary frequency, and urgency. Additionally, obstructive urinary symptoms, such as a weak urinary stream, dribbling, and hesitancy, may occur, potentially progressing to urinary retention. [8] Suprapubic, rectal, or perineal discomfort is also a common symptom. [3] Patients may experience painful ejaculation, hematospermia, and painful defecation. [4] A detailed sexual history may help assess the risk of sexually transmitted infections and allow for the optimal treatment selection. [8]

Physical examination

Abdominal examination has an important role in physical examination of patients with suspected ABP since it allows to identify bladder distension and costovertebral angle tenderness. [3] The evaluation of the external genitalia, including the perineum and rectal area, is essential. [2] Digital rectal examination (DRE) is commonly performed to assess the prostate for tenderness, swelling, or induration, which are indicative of infection. DRE has to be with caution and prostate massage is not recommended. Forceful palpation of the prostate may trigger the release of bacteria and inflammatory cytokines, potentially resulting in abrupt clinical decompensation and exacerbating complications. [2, 8].

DRE in patients with ABP typically reveals a prostate gland that is warm, boggy, extremely tender, and tense. The presence of fluctuation upon palpation raises suspicion for a prostatic abscess. Perineal pain and anal sphincter spasm may complicate the procedure. [4]

Laboratory tests

It is essential to perform urine analysis and culture when diagnosing acute bacterial prostatitis. It is worth noting however, that urine cultures in 35% of individuals with acute prostatitis may fail to grow pathogen. [3] A midstream urine sample should be collected and analyzed for the presence of white blood cells. The diagnosis will be considered positive if the number of white blood cells per high-power field exceeds 10. In cases where a patient presents with a palpable bladder or symptoms suggestive of incomplete emptying, it is important to document the residual urine volume. [4]

In Patients with fever, a potential hematogenous infection source, complex infections, or those with compromised immune systems blood culture should be obtained. [3]

Serum laboratory tests for ABP typically show elevated levels of inflammatory markers, including white blood cells, neutrophils, C-reactive protein, and erythrocyte sedimentation rate. [2] Studies identified that white blood cell counts exceeding 18,000 per mm^3 ($18 * 10^9/L$) and blood urea nitrogen levels greater than 19 mg/dL (6.8 mmol/L) are independently associated with severe cases of ABP. [9]

Approximately 70% of patients demonstrate abnormally high prostate-specific antigen (PSA) levels due to inflammatory damage to epithelial cells in the prostate ducts. [1] However, the role of serum PSA in the differential diagnostic evaluation of ABP remains unproven. Some experts suggest that high levels of serum PSA associated with ABP usually leads to confusion and fear therefore it is not recommended. [2] On the contrary, effective antibiotic treatment leads to a significant reduction in serum PSA levels. PSA levels typically return to normal after 1–2 months of treatment; if they do not, PCA should be considered [1] As a result, some authors advocate for the use of PSA as a concise, accurate, rapid, and cost-effective tool for diagnosing ABP and monitoring treatment progress. [4]

The Meares–Stamey four-glass test (considered as the gold standard for the diagnosis of CBP) is not recommended for men with suspected ABP because it increases risk of sepsis. [1]

Studies have been conducted on the use of semen samples instead of urine samples for diagnosing prostatitis. With new enrichment diagnostic technique for semen culture proposed, further testing is required to determine the utility of this method in cases of ABP. [10]

Imaging

Imaging studies are not always required during the initial assessment but may be beneficial in cases where the diagnosis is uncertain or if there is inadequate response to appropriate antibiotic therapy. [3]

Ultrasound

Prostatic ultrasound is a primary imaging modality in the diagnostic evaluation of ABP. Transrectal (TRUS) and transperineal (TPUS) approaches offer greater diagnostic utility compared to transabdominal ultrasound. With its higher accuracy, TRUS can be very helpful in distinguishing parenchymal abscesses from other differential diagnoses. Advanced techniques such as color Doppler imaging, tissue harmonic imaging, and contrast-enhanced ultrasound can enhance diagnostic precision [11] Especially Color Doppler sonography could serve as an effective tool for assessing treatment response and predicting clinical outcomes. Intraprostatic color flow in patients with ABP is higher compared to normal prostate tissue or cases of chronic inflammation or carcinoma since most patients exhibit increased vascularity of the prostate during the acute inflammatory phase. [4]

Multiparametric MRI

Multiparametric Magnetic Resonance Imaging is an important technique for evaluating different prostatic disorders, especially a prostate cancer. This advanced imaging modality technique offers detailed visualization of prostate anatomy and potential abnormalities, provide insights into tissue density, cellular architecture and vascular dynamics. It integrates high-resolution T2-weighted imaging with functional modalities, including diffusion-weighted imaging, dynamic contrast-enhanced imaging, and magnetic resonance spectroscopic imaging. [11]

In patients with ABP Multiparametric MRI shows focal or diffuse areas of low T2 signal intensity with patchy enhancement in the peripheral zone. Inflammatory cellular infiltrates result in mild to moderate diffusion restriction, which is reflected by signal loss on apparent diffusion coefficient maps. Morphological patterns of prostatitis include a diffuse, band-like, or wedge-shaped appearance. However, ABP can occasionally present in the transitional zone, where its regular low signal intensity may mimic the 'erased charcoal' sign characteristic of prostate carcinoma. [4]

Treatment

ABP treatment is based on outpatient antibiotic therapy in most non-complicated cases. [2**,** 12] Percentage of patients hospitalized due to ABP varies between studies. [1, 3, 4] Risk of hospitalization increases with intolerance of oral antibiotic, previous unsuccessful treatment with fluoroquinolone, transurethral or transrectal prostate manipulation, Hospitalization is required for patients presenting with prolonged vomiting, severe dehydration, hyperpyrexia, tachycardia, hypotension, tachypnea, or other symptoms indicative of urosepsis. Additionally, hospitalization is recommended for high-risk individuals, including those with diabetes and immunosuppression. In patients with prostatic abscesses surgical drainage may be required. [2] Aside form antibiotic therapy nonsteroidal anti-inflammatory drugs are recommended in ABP to relieve the pain and lower fever. [2, 4]

Antibiotics

Lack of large reliable randomized controlled trials on antibiotics selection leads to empiric antibiotic therapy based on patients' factors such as recent immunosuppressive therapy, high-risk sexual practices, benign prostatic hyperplasia, prior infections, the antibiotics' concentration in prostatic fluid and local antibiotic susceptibility patterns. [4] Treatment protocols rely primarily on clinical expertise and data derived from observational studies. Prompt therapy with broad-spectrum, antibiotics is typically sufficient since significant prostatic inflammation helps with adequate antibiotics penetration (with the exception of nitrofurantoin). [1]

Patients presenting with acute illness, clinical signs of sepsis or systemic inflammatory response syndrome should be treated with parenteral antibiotics. After regaining clinical stability, in patients without fever or urinary retention intravenous antibiotics should be transitioned to oral therapy for a duration of 2–4 weeks. [1, 4, 12] Some experts advocate for a longer treatment duration of 4 to 6 weeks, which is commonly employed in the management of chronic prostatitis. [13] Patients with fever should typically become afebrile within 36 hours of initiating antibiotic therapy. [3] If clinical improvement cannot be observed, it is important to perform follow-up urine cultures to evaluate bacterial eradication. [8] One week after treatments completion urine cultures should be repeated to confirm the eradication. [8, 12]

Adequate antibiotic treatment is the most important independent factor in prevention of relapse. Furthermore patients treated with co-trimoxazole had higher risk of relapse after adequate antibiotic therapy than patients treated with ciprofloxacin. Intravenous dosing of beta-lactam resulted in lower rate of relapse in comparison with oral beta-lactams. [14] It is worth noting however, that those statistics refers to hospitalized patients only and further studies including outpatient cases are necessary.

Antibiotics selection

Non-complicated cases could be adequately treated with a 2-week course of oral fluoroquinolones. [4] Patients with low risk of sexually transmitted infections should receive 500mg of Ciprofloxacin or 500-750mg of Levofloxacin orally twice a day for 10 to 14 days as first-line therapy. [3] Trimethoprim-sulfamethoxazole, broad-spectrum penicillin derivative, or third-generation cephalosporin are viable alternatives to quinolones as the first choice for empirical outpatient therapy. It is worth noting however, that fluoroquinolones can achieve intraprostatic concentrations that are three to four times higher than those of β-lactam antibiotics. Men over 35 who engage in high-risk sexual activities, as well as men under the age of 35 who are sexually active and are suspected of being infected with *Neisseria gonorrhoeae* or *Chlamydia trachomatis*, should be treated using standard protocols. [1] First-line therapy for those patients should include single dose of ceftriaxone (250 mg intramuscularly) or a single dose of cefixime (400 mg orally) followed by 100 mg of Doxycycline, orally twice daily for 10 days. [3]

Hospitalized patients could be treated with intravenous doses of ciprofloxacin/ levofloxacin. Intravenous piperacillin/tazobactam every 6 hours should be considered in severe cases. Alternatively to piperacillin, intravenous dose of cefotaxime can be used every 4 hours. [3, 12] Aminoglycosides may be added if patient is clinically unstable. The choice of specific aminoglycoside may be influenced by local antibiotic management guidelines, accessibility, and pharmacoeconomic considerations. Some experts recommend aminoglycoside treatment durations up to 7 days. [15]

Considering the fact that in most cases ABP is caused by Gram negative pathogens, guidelines for treating ABP caused by Gram-positive bacteria are very limited. ABP caused by methicillin-susceptible Staphylococcus aureus could be successfully treated with dalbavancin due to dalbavancin favorable pharmacokinetic profile. However, it is worth noting that further research on dalbavancin is necessary as the impact of pH on dalbavancin's

activity is unknown and dalbavancin prostate penetration models are based on levofloxacin which has the same volume of distribution. [16]

Fosfomycin

Fosfomycin has been successfully utilized in extended treatment regimens for bacterial prostatitis, especially in cases involving multidrug-resistant bacteria, previous treatment failures, or intolerance to alternative antibiotics. Studies have shown that favorable pharmacokinetic and pharmacodynamic profile of oral fosfomycin, enable higher drug concentrations within the prostate, which are often sufficient to achieve bactericidal effects. It is worth noting however, that the available evidence is more representative of chronic bacterial prostatitis than ABP and primarily concerns *Escherichia coli* infections. Despite the current absence of randomized controlled trials fosfomycin may in the future be considered a valid therapeutic option, potentially even as a first-line treatment in some cases. [17, 18]

Oral fosfomycin could be important alternative to intravenous carbapenems in complicated cases of ABP caused by *Enterobacterales* producing extended-spectrum beta-lactamases. The availability of an alternative oral therapy to replace carbapenems could result in improved patient quality of life, reduced healthcare costs, and a lower ecological impact. [19]

Moreover oral fosfomycin is generally well tolerated. Patients should be monitored for diarrhea - the most frequently reported side effect. [17] In case of fosfomycin-related diarrhea the dosing frequency prolongation is often a viable solution.

Nowadays low resistance rate among commonly associated uropathogens is still an important benefit of employing fosfomycin. However, in the future development of resistance to fosfomycin caused by prolonged use should be expected since in vitro studies have indicated that fosfomycin can acquire antimicrobial resistance through mutational changes or the action of fosfomycin-modifying enzymes. [18]

Antibiotic resistance

Data from both Europe and United States indicate increasing antimicrobial resistance observed among *Enterobacterales.* [12] Study conducted on elderly patients (≥75 years old) shows that resistance rates in this group exceed 30% for ciprofloxacin, amoxicillin-clavulanic acid, and cotrimoxazole, Moreover it was found that 15,4% of strains produced extended-spectrum beta-lactamase. [20]

It is worth noting that recent studies show that *Escherichia coli* associated with post-prostate biopsy ABP (Bx-ABP) demonstrated a higher prevalence of antibiotic resistance compared to

community-acquired ABP (Ca-ABP). Remarkable variations in *E. coli* antibiotic sensitivity were noted between patients with Ca-ABP and Bx-ABP for fluoroquinolones, cephalothin, and gentamicin. Moreover the incidence of bacteremia was significantly higher in the group of patients with the Bx-ABP compared to the CA-ABP group. Amikacin, imipenem, meropenem, amoxicillin/clavulanic acid, and piperacillin/tazobactam demonstrated very high effectiveness against *E. coli* in both groups. [21] Due to high resistance fluoroquinolones should not be the first choice in empirical therapy of Bx-ABP. Carbapenems may be used instead but given the recent rise in carbapenem-resistant bacteria, piperacillin/tazobactam should be considered. Ceftolozam with tazobactam and may also exhibit activity against carbapenem-resistant *Pseudomonas aeruginosa*. [15] Oral administration of amoxicillin/clavulanic acid could be a viable alternative for patients who do not require hospitalization and are presenting mild symptoms. [21]

Urinary obstruction

Alpha-blockers could be used in case of high risk of urinary retention which is very common in patients with ABP. [2] It is worth noting that the therapy with alfa-blockers may be considered only if bladder scanning shows that residual urine is below 100ml. Patients with severe urinary retention (>100ml) should instead be treated with catheterization. Small-caliber urethral catheter could be used for the short time (<12h). Alternative methods include placement of a suprapubic tube or single catheterization with the trial of voiding. [2, 4] Retained Foley catheter may exacerbate urethral duct obstruction increasing risk of the formation of prostatic abscesses, so the suprapubic cystostomy has traditionally been considered as the best option. [4]

Prevention

No established methods for preventing community-acquired acute bacterial prostatitis exist, but the risk of nosocomial infections can be minimized by limiting unnecessary prostate interventions, such as transrectal biopsies or urethral catheterizations. [3]

Post-prostate biopsy prostatitis

The prophylactic use of antibiotics can be effective strategy to prevent post-prostate biopsy ABP (Bx- ABP). Previously mentioned rise in severe infectious complications following

biopsy coincide with increasing resistance to quinolones. However oral quinolones, either alone or in combination with other antibiotic agents, remain the most common prophylactic approach. [5] Typically patients take two prophylactic oral doses - the first 500mg of ciprofloxacin is administrated 12 hours before transrectal prostate biopsy and the second dose is repeated at the time of the biopsy. It is worth noting however, that this method cannot entirely eliminate Bx-ABP. Moreover overuse of similar antimicrobial agents for prophylaxis, has been linked to the emergence of resistant strains. Selecting appropriate prophylactic antibiotics by collecting data on antibiotic sensitivity of pathogens causing ABP in the region became crucial. [3].

To improve the prevention of prostatitis following biopsy the effectiveness of different prophylactic protocols was tested. The protocol that involved the use of ciprofloxacin alone was compared with the protocol incorporating ciprofloxacin with addition of ornidazole and pre-biopsy enema. No statistically significant difference was observed between the two regarding the rate of acute prostatitis. [5] Preoperative enemas alone also turned out to be ineffective in reducing infection rates. [3]. Advanced biopsy techniques, such as magnetic resonance imaging-transrectal ultrasound (MRI-TRUS) fusion-guided 3D-targeted biopsies, may help minimize the need for repeated biopsies therefore reducing risk of the post-biopsy ABP. [5]

Stool cultures

Preoperative stool cultures may be helpful to optimize antibiotic therapy by enabling the identification of patients with antibiotic-resistant strains. It is worth noting however, that the normal rectal microbiota presents a challenge in isolating and analyzing specific fluoroquinolone-resistant specimens. Employing selective media can help minimize false-negative results since using a standard media when isolating a single colony among numerous others may lead to the misclassification of the strain as fluoroquinolone-sensitive. [22]

Future perspectives

Nowadays many different supportive treatments, such as natural antipyretics and anti-inflammatory agents are tested in vitro and in vivo on animals. Study that explored the effects of levofloxacin combined with tamsulosin for treating ABP in animals showed that tamsulosin could be useful to increase the levofloxacin’s therapeutic effect. [4]

In recent years more and more reports suggest that the effectiveness of fluoroquinolones has progressively diminished due to the rise of multidrug-resistant bacteria therefore search for new treatment strategies becomes necessary. Researchers are working on targeting drug delivery systems that may allow to use natural flavonoid compound – luteolin. Despite luteolins' antibacterial and anti-inflammatory properties, it has not been used in ABP yet due to its poor water solubility and low structural stability. It is worth noting that recently new Hyaluronic Acid-Modified Luteolin−Copper Complex (Lut−Cu@HA) Nanodelivery System was designed. Naturally macromolecular glycosaminoglycan was grafted with luteolin and copper ions that can coordinate to form structurally stable complexes. In vitro testing showed that the system exhibits vey good blood compatibility and high safety while in vivo tests on rats proved it could be effective in treating bacterial prostatitis. [23] Due to limited evidence and lack of proper testing on humans Lut−Cu@HA system has not been included in recommendations for ABP treatment yet.

Investigations in an in vitro model of BP underscore the potential therapeutic advantages of the formulation composed of pumpkin extract, bromelain and the probiotic strain *L. rhamnosus* in managing bacterial prostatitis. Specifically, the active compounds in the formulation effectively target critical cytokines released by pathogen-stimulated macrophages, thereby mitigating the inflammatory response in prostate cells. [24]

Complications

Frequent oversight of acute bacterial prostatitis due to its nonspecific symptoms can pose real danger for patients' health. Initial treatment failure may lead to formation of prostate abscess and sepsis. [13, 25] In addition, transition to chronic bacterial prostatitis (CBP) or chronic pelvic pain syndrome (CPPS) is also possible. [13] High rates of alcohol consumption, diabetes, voiding symptoms, previous medical procedures, enlarged prostate volume, catheterization history, and shorter durations of antibiotic treatment are among risk factors of ABP transition into CBP. In a retrospective analysis of 437 ABP patients, 1.3% progressed to CBP, while 10.5% developed inflammatory CPPS. [26]

Success of initial treatment does not eliminate the risk as preemptive switching from intravenous to oral antibiotics may result in CBP caused by surviving causative pathogens.

Epididymitis is a common complication of ABP. It may transform to chronic inflammation and result in chronic pain and obstruction of the sperm passageway leading to male infertility. [13]

Prostatic abscess

Prostatic abscess (PA) is an uncommon yet serious complication of ABP. Without early treatment of PA additional complications such as extra-prostatic spread are difficult to prevent. Studies show that the risk of sepsis becomes noticeably increased in patients with prostatic abscess in comparison with non-complicated ABP. [27] Diabetes mellitus and immunodeficiency are among risk factors contributing to the development of PA. Immunocompromised patients diagnosed with AIDS, have the risk of severe complications (including PA) increased up to 14%. [4] The factors with the most significant impact on likelihood of prostatic abscess also include recent history of prostate biopsy, weight loss or malnutrition and urethral structure. [27]

Diagnostics

Approximately 13% of patients diagnosed with acute bacterial prostatitis experience a recurrence, often requiring an extended course of antibiotic therapy. [8] Frequent recurrences despite adequate treatment should undergo further evaluation to rule out the presence of prostatic abscesses. [3] Transrectal ultrasound (TRUS) should be performed in patients who do not show improvement after appropriate antibiotic treatment of ABP for 48 hours. [4] TRUS offers direct contact with the prostate therefore it is favored over transperineal or transabdominal ultrasound. For well-defined, larger walled-off abscesses TRUS can achieve 80-100% diagnostic accuracy, but in the initial stages of abscess formation, it may yield uncertain results. Abscesses are generally confined to the transitional and central zones, frequently resulting in anatomical distortion of the prostate. Typical TRUS findings consist of one or more hypoechoic regions, featuring internal septations, thick well-defined walls, enhanced color Doppler flow signals, and the presence of intraglandular calcifications. [28] If PA could not be diagnosed with TRUS, CT and MRI imaging techniques can be useful. [4]

Treatment

Developed abscess (>20mm) cannot be typically treated with antibacterial therapy alone and surgical interventions such as drainage become necessary. Studies show that almost half of the patients diagnosed with PA require surgical treatment. [25, 27] Conservative treatment of prostatic abscesses larger than 2cm may lead to recurrence and eventually resulted in resection of the prostate. Surgical interventions, help shorten the duration of antibiotic therapy

and may alleviate voiding symptoms. It is worth mentioning however that the duration of antibiotic therapy is longer in cases of prostatic abscess compared to those without an abscess, irrespective of whether surgical intervention was performed. [25]

Historically, TURP was regarded as the definitive intervention for managing prostatic abscesses, particularly in older patients with coexisting benign prostatic hyperplasia. This procedure has been associated with a significant risk of transient urinary incontinence, affecting up to 50% of cases. Nowadays, with advancements in minimally invasive techniques, TURP is typically reserved for refractory cases involving multiple abscesses.

Transurethral drainage with its high effectiveness in abscess evacuation was the preferred approach among urologists. However, its limitations (invasive nature and the need for general anesthesia) have led to its replacement by the transrectal approach or percutaneous ultrasound-guided drainage. Both these techniques employ TRUS to guide a needle and offer a reduced risk of complications such as retrograde ejaculation and urinary incontinence. [28]

Unusual organ complications

APB-related bacteriemia may lead to uncommon but dangerous complications such as acute myocarditis. It presents a diagnostic and therapeutic challenge as literature shows that myocarditis in the setting of urosepsis can been associated with poor cardiac outcomes and high morbidity. [29]

Liver abscesses are typically caused by bacteria and remain rare in patients with acute prostatitis. Possibility of metastatic infection in other organs should be considered in individuals with ABP and abnormal liver function tests. Overlapping of systemic symptoms, such as fever and muscle pain can complicate the diagnosis, but early detection of liver abscesses is crucial. Liver abscess is associated with various types of liver cancer and require proper drainage. Research on the epidemiology and characteristics of liver abscesses in the context of AP is limited, but studies show that in patients with prostatitis and abscesses affecting other organs 50% of them were located in liver. *Klebsiella pneumoniae* was found in most of these cases. [30]

Klebsiella pneumoniae could also compromise visual function. There is a case of ABP-related endogenous endophthalmitis with retinal necrosis in older, but immunocompetent male. Loss of vision occurred during hospitalization and treatment for prostatitis. [31]

The broad range of complications cited should emphasize the necessity of appropriate ABP treatment.

Cancer and benign prostatic hyperplasia

There is no consensus on the definitive link between prostatitis and prostate cancer (PCa) but studies suggest significant association between them. However, further research is required as previous studies do not include specific classification of prostatitis. [32]

There is some evidence that chronic prostatitis may cause benign prostatic hyperplasia as cytokines and growth factors released by inflammatory cells could stimulate hyperproliferation of stromal and epithelial cells. Alterations in the prostate environment related to inflammation can change gene expression leading to prostatic hyperplasia and morphological changes of stromal tissue [32**,** 33] It remains unclear if those mechanisms are applicable exclusively to chronic bacterial prostatitis or ABP as well.

Conclusions

Acute bacterial prostatitis (ABP) is an acute infection that can present diagnostic and therapeutic difficulty due to its nonspecific symptoms and the risk of severe complications. Patients with ABP may often require rapid and decisive treatment to avoid sepsis, and septic shock. Risk factors are strongly associated with prior interventions involving the lower urinary tract. ABP can be caused by ascending urethral infections as well as hematogenous or lymphatic spread. *Escherichia coli* remains the primary pathogen responsible for ABP *while Pseudomonas aeruginosa* is a common cause of prostatitis after transurethral manipulation. Increasing prevalence of antibiotic-resistant strains pose an additional challenge in recent years. New anti-inflammatory agents and advanced drug delivery systems targeting prostate tissues may provide solutions to this issue in the future. Further research is necessary before they could be utilized in treatment. Correct diagnosis mainly relies on a medical history and physical examination, but laboratory tests—particularly urine and blood cultures— as well as imaging studies can be very helpful. Outpatient empirical antibiotic therapy is usually sufficient, but patients with severe symptoms and the high risk of complications should be hospitalized. Oral fluoroquinolones for 10 to 14 days remain the first-line therapy. In the coming years, fosfomycin may find broader application in the treatment of ABP. No clinical improvement after initial treatment may indicate resistant pathogens or the development of prostatic abscess (PA) —a serious complication that may require surgical intervention. PA can usually be diagnosed with the help of transrectal ultrasound.

Preventing ABP is a significant concern in the context of transrectal prostate biopsy. Proper prophylactic measures can reduce the incidence of post-prostate biopsy ABP.
Knowledge about risks associated with ABP and available prophylaxis helps making better therapeutic decisions. Therefore it improves patients' prognosis, enhances quality of life, and reduce healthcare costs.

Disclosure

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