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# **Current Views on Diagnostic Approach and Treatment of Chronic Bacterial Prostatitis**

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#### Abstract

Prostatitis is a prevalent condition that encompasses a large array of clinical symptoms with significant impacts on men's life. The diagnosis and treatment of this disorder presents numerous challenges for urologists, most notably, a lack of specific and effective diagnostic methods. Chronic bacterial prostatitis is successfully treated with appropriate antibiotics that penetrate the prostate and kill the causative organisms. National Institutes of Health (NIH) classification four categories. Prostatitis accounts for 25% of all visits to urologists. Of patients diagnosed with prostatitis syndrome, 5 to 10% are suffering from chronic bacterial prostatitis (CBP). This condition is often triggered by an infection of the urinary tract. The pathogen spectrum includes that of complex urinary tract infections, with gram-negative and gram-positive bacteria, although the latter often occur only transiently. Animal experiments have shown that chronic infection leads to the formation of a biofilm in the prostatic acini, leading to pathogens forming colonies with enhanced growth conditions. We reported the clinical evaluation, diagnostic work-up and successful management of chronic bacterial prostatitis.

Keywords: Bacteria, prostatic gland, men

#### Introduction

Prostatitis is a prevalent, confusing and frustrating presentation for both patients and clinicians. It is an inflammatory condition of the prostate that presents with urethral symptoms, prostatic symptoms and sexual dysfunction. Up to 25% of men receive a diagnosis of prostatitis in their lifetime, but <10% have a proven bacterial infection(1,2). The causes and treatment of nonbacterial prostatitis are largely unknown, but bacterial prostatitis is caused by infection with uropathogens, especially aerobic gram-negative bacilli, E. coli cause 50%-80% of cases; other pathogens include Enterobacteriaceae (eg, *Klebsiella* and *Proteus*, which account for 10%–30% of cases), *Enterococcus* species (5%-10% of cases), and nonfermenting gram-negative bacilli (eg, Pseudomonas species; <5% of cases). Some debate the role of gram-positive organisms other than enterococci, but most accept Staphylococcus and Streptococcus species as pathogens (3). The increasing prevalence of gram-positive pathogens may represent changing disease epidemiology (perhaps related to fluoroquinolone therapy) or acceptance of their pathogenicity by health care providers. Acute bacterial prostatitis is easily diagnosed (by abrupt urogenital and often systemic symptoms, along with bacteriuria) and treated (by systemic antibiotic therapy). Chronic bacterial prostatitis is characterized by prolonged or recurrent symptoms and relapsing bacteriuria; diagnosis traditionally requires comparing urinary specimens obtained before with specimens obtained after prostatic massage (5, 6). Of patients diagnosed with prostatitis syndrome, 5 to 10% are suffering from chronic bacterial prostatitis (CBP). This condition is often triggered by an infection of the urinary tract. The pathogen spectrum includes that of complex urinary tract infections, with gram-negative and gram-positive bacteria, although the latter often occur only transiently. Animal experiments have shown that chronic infection leads to the formation of a biofilm in the prostatic acini, leading to pathogens forming colonies with enhanced growth conditions (7, 8, and 9).

Prostatitis is chronic when symptoms have been present for at least 3 months. Chronic bacterial prostatitis is a clinical syndrome, defined primarily on the basis of urologic symptoms and/or pain or discomfort in the pelvic region. It is a common condition among men of a wide age range, with detrimental effects on quality of life. The etiology, pathogenesis, and optimal treatment of CBP still remain relativelly unknown, although significant progress has been made in the last few years in the understanding and management of this disorder (10, 11).

Antibiotics are the most common therapy used to treat chronic bacterial prostatitis (CBP). Eradication of bacteria is associated with clinical success in the short and long term with CBP caused by both traditional and nontraditional bacteria. Antibiotic therapy can be used in an attempt to cure CBP but relapses are common. CBP in men with prostatic calculi is more difficult to cure (12).

Fluoroquinolones are the mainstay in the treatment of CBP. Fosfomycin has been shown to have good activity against extended-spectrum beta-lactamase producing organisms. Azithromycin may be more effective for *Chlamydia* infections. Most other antimicrobial agents are unlikely to eradicate the infection.

Although bacteria are cultured in only 5%-10% of prostatitis cases, bacteria may still be the cause of the chronic prostatitis in many patients with the syndrome (13, 14).

We reported the clinical evaluation, diagnostic work-up and successful management of chronic bacterial prostatitis.

# **Material and Methods**

This study consists of 105 patients (age 27to 50 years) with diagnosis of CBP who visited at TSMU the First University Clinic Urology Department from 2017 january – until august 2017 with compliance of lower urinary tract symptoms: dysuria, pain in lower back and perineal area radiating to the testicle. The following analysis were performed:

- 1. International prostate symptom score(IPSS)
- 2. Uroflowmetry (Mediwatch)
- 3. Ultrasound of urinary system with waste urine(MINDEY DC-N3)
- 4. Prostate-spesific antigen (PSA) for patients older 40 years.
- 5. Bacteriology of prostate fluid.
- 6. Bacteriology of first steam urine after prostate massage.
- 7. Digital-rectal examination.
- 8. Safe blood analysis: Anti-HCV, Anti-TP, Anti-HIV, HBsAg.

Bacteriological examination of prostate fluid demonstrated significant growth of bacteria (>10<sup>5</sup>ml<sup>-1</sup>). The microorganisms were identified by gram stain, oxidase, catalase and other biochemical tests using Bio-Mérieux products (API Staph, API 20E, API20 Strep, API 20 NE, Bio-Mérieux). The cultures which grew only rare coagulase-negative staphylococci or diphtheroids were interpreted as negative, as these organisms were considered non-pathogenic and probably represented contaminants.

Sensitivity of microorganisms to antibiotics was defined with Kirby-Bauer disc-diffusion method using standard discs (EUCAST guidelines 2017). Antibiotic susceptibility test was done on following antibiotics: Amoxicillin+Clavulanic acid, Ampicillin+Sulbactam, Amikacine, Norfloxacine, Ciprofloxacine, Levofloxacine, Moxifloxacine, Fosfomycine, Doxycycline, Azithromycine, Nitrofurantoin, Thrimethoprim-Sulfamethoxazole.

# Results

After getting a results of appropriate analysis we subdivided three groups according to patient age: 27-30years (35 patient), 30-40year (55 patient), 40-50year (15patient) (Table1).

Table: 1

	27-30year	30-40 year	40-50year
IPSS	8-19	20-30	20-30
Prostate mass	22-27gr	24-31gr	30-80gr
<b>Residual urine</b>	15-20ml	15-40ml	15-60ml
Qmax	15-20	15-20	10-18

Safe blood analysis results were negative for all patients. Bacteriological investigation of prostatic fluid yelded in 56 patients Enterococcus faecalis (53, 3%), Staphylococcus aureus 32(30, 5%), Streprococcus anginosus 5(4.8%), Enterobacter cloacae 6(5, 7%), Escherichia coli 5(4, 8%), Klebsiella pneumonia 1(0, 9%). Only two patients had positive urine culture with same bacterial isolate which in prostatic were fluid (Enterococcus faecalis). Polymicrobial growth was observed in 6 cases with Enterobacter cloacae and Escherichia coli (3cases), Enterococcus faecalis and Staphylococcus aureus (2 cases), Enterococcus faecalis and Escherichia coli (1 case). Both gram positive and gram negative organisms were sensitive to ampicillin-sulbactam, amoxicillin-clavulanic acid and amikacin. There was a total of 82% resistance to ciprofloxacin and levofloxacin and only 56% was resistant to moxifloxacine, 94% resistant to co-trimoxazole, only 5% were shown resistance to fosfomycine and nitrofurantoin. 59% were resistant to doxycycline. Urine culture were negative in most patients (98%).

Antimicrobial monotherapy was initiated depends on the local susceptibility test results. 27-30 year patient underwent two week course of antibiotic with significant improvement of IPSS, subjective complaints were reduced, uroflowmetry results were improved. Q max were increased (25ml/sec).

30-40 year patients were treated with appropriate antibiotics from 3 to 4 weeks, at the same time antifungal drugs with prophylactic dosage were administrated despite this in 4 patients were developed dysbacterioses. After treatment IPSS were normal, Q max were 20-25ml/sec

40-50 year patients were treated with antibiotics 3-4 weeks course, urine flow by uroflowmetry were improved. In this age group use of alpha- blokers after two weeks with combination of antibiotics were highly effective rather than antimicrobial monotheraphy.Q max were 18-24 ml/sec

# Conclusion

Our study show current views on effective diagnostic approach and successful treatment of chronic bacterial prostatitis with antibiotic monotherapy depends on local susceptibility pattern and determination of effective treatment duration in different age groups.

### References

- 1. Shoskes DA, Nickel JC, Rackley RR, Pontari MA. Clinical phenotyping in chronic prostatitis/chronic pelvic pain syndrome and interstitial cystitis: a management strategy for urologic chronic pelvic pain syndromes. Prostate Cancer Prostatic Dis. 2009;
- 2. Shoskes DA, Nickel JC, Kattan MW. Phenotypically directed multimodal therapy for chronic prostatitis/chronic pelvic pain syndrome: a prospective study using UPOINT. Urology. 2010;
- 3. Nickel JC, Narayan P, McKay J, and Doyle C. Treatment of chronic prostatitis /chronic pelvic pain syndrome with tamsulosin: a randomized double blind trial. J Urol. 2004;
- 4. Yang G, Wei Q, Li H, Yang Y, Zhang S, Dong Q. The effect of alpha-adrenergic antagonists in chronic prostatitis/chronic pelvic pain syndrome: a metaanalysis of randomized controlled trials. J Androl. 2006;
- 5. Nickel JC, Krieger JN, McNaughton-Collins M, Anderson RU, Pontari M, Shoskes DA, et al. Alfuzosin and symptoms of chronic prostatitis-chronic pelvic pain syndrome. N Engl J Med. 2008;
- 6. Chuang YC, Yoshimura N, Wu M, Huang CC, Chiang PH, Tyagi P, et al. Intraprostatic capsaicin injection as a novel model for nonbacterial prostatitis and effects of botulinum toxin A. Eur Urol. 2007;
- Kiyota H, Onodera S, Ohishi Y, Tsukamoto T, Matsumoto T. Questionnaire survey of Japanese urologists concerning the diagnosis and treatment of chronic prostatitis and chronic pelvic pain syndrome. Int J Urol. 2003;
- 8. Pontari MA, Joyce GF, Wise M, McNaughton-Collins M. Prostatitis. J Urol. 2007;
- S. Krieger JN, Riley DE, Cheah PY, Liong ML, Yuen KH. Epidemiology of prostatitis: new evidence for a world-wide problem. World J Urol. 2003;
- 10. Yoo YN. Prostatitis. Korean J Urol. 1994; 35:575–585.
- 11. McNaughton Collins M, Pontari MA, O'Leary MP, Calhoun EA, Santanna J, Landis JR, et al. Quality of life is impaired in men with chronic prostatitis: the Chronic Prostatitis Collaborative Research Network. J Gen Intern Med. 2001;
- 12. Staud R. Fibromyalgia pain: do we know the source? Curr Opin Rheumatol. 2004;
- Dadabhoy D, Clauw DJ. Fibromyalgia: progress in diagnosis and treatment. Curr Pain Headache Rep. 2005;
- Diatchenko L, Nackley AG, Slade GD, Fillingim RB, Maixner W. Idiopathic pain disorders--pathways of vulnerability. Pain. 2006