ORIGINAL ARTICLE

Prevalence and Microbiological Characteristics of Bacterial Prostatitis in Patients with Erectile Dysfunction

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ABSTRACT

Background: *Prostatitis is common in men and causes physical discomfort and pain which suggested to have an impact on sexual dysfunction, including erectile dysfunction.* ***Objectives****: To identify the prevalence of bacterial prostatitis (BP) among Egyptian erectile dysfunction (ED) patients. Also, to identify the causative bacterial species.* ***Methodology****: A total number of 397 men with ED were included. Three samples were taken from each patient; two urine and one expressed prostatic secretion (EPS); which were subjected to bacterial culture as well as PCR (for Chlamydia trachomatis) with subsequent sensitivity test in case of positive cultures.* ***Results****: BP was diagnosed in 94/397 patients (23.7%) (92 cases were diagnosed by culture and 2 cases by PCR). There was no significant difference in the clinical data between ED patients with and without BP, except for age and lower urinary tract symptoms. The most common isolate was E. coli (46.8%). Antibiotic sensitivity revealed that the most effective tested class in decreasing order are; levofloxacin (81.5%), ciprofloxacin (75. %) norfloxacin (73.9%), nitrofurntation (69.6%) and cotrimoxazol (31.5%).* ***Conclusions****: In ED patients, 23.7% was found to have BP. The commonest isolates were E. coli followed by S. aureus and E. fecalis. The majority of the isolates were sensitive to levofloxacin, ciprofloxacin and norfloxacin.*

INTRODUCTION

The National Institute of Health (NIH) classified prostatitis into; acute bacterial prostatitis (ABP), chronic bacterial prostatitis (CBP), chronic pelvic pain syndrome/chronic prostatitis (CPPS/CP), and asymptomatic inflammatory prostatitis[1]. CBP is characterized by repeated lower urinary tract infection symptoms (LUTIS) due to the persistent hidden bacterial infection inside the prostate[2]. Several researchers suggested that Gram-negative bacteria, especially *E. coli*, usually cause the great majority of cases of CBP whereas other researchers suggested the preponderance of Gram-positive bacteria[3]. Therefore, the proper management of BP needs antibiotics with high tissue penetration into the prostate[4].

ED is defined as the persistent inability to attain/maintain an erection sufficient to permit satisfactory sexual performance[5]. Some investigators reported that prostatitis does not necessarily affect any aspect of sexual function[6]. whereas others are associated between prostatitis and ED[7]. In their study, Trinchieri et al.[8] reported that the prevalence of ED is 39% in patients with CBP. Smith et al.[9] pointed out that, compared to healthy controls, men with CP/CPPS reported significantly more sexual dysfunction and symptoms of depression. Besides, the partners of these men reported significantly more pain upon intercourse, vaginismus, and depressive symptoms compared to control females. Wagenlehner et al.[10] suggested that sexual dysfunctions in CBP cases add too many positive symptom phenotypes and correlate with increasing symptom scores in these patients. However, although some pieces of evidence are raising a relation between CBP and ED, limited studies are explaining the possible mechanisms causing these two conditions together[11]. Our objectivesis to identify the prevalence and microbiological characteristics of bacterial prostatitis among Egyptian men complaninig of erectile dysfunction.

METHODOLOGY

This is a Cross-Sectional Study, 397 men complaining of ED, and meeting the requested criteria, were recruited from Dermatology and Andrology

Department or Out-patients Clinic in Mansoura University Hospital (from Nov 2022 to March 2024) after informed consent from all participant. The study was approved by institutional review board of Mansoura faculty of medicine (R/24.03.2542, 2024). The inclusion criteria were; married patients with ED (a score of ≤21), according to the Sexual Health Inventory For Men (SHIM)[12]. Patients with conditions affecting either bacterial virulence or host response (e.g., immunodeficiencies, abnormalities of the urogenital system) or who received antibiotics/immunosuppressive drugs within the previous 3 months were excluded. All men were subjected to history taking, clinical examination, and bacteriological evaluation.

Three samples were collected from each patient according to Nickel et al.[13] after 3–5 days of sexual abstinence in labeled sterile screw-capped containers. These patients were asked to wash the glans penis with regular soap and water. The first sample was midstream urine (VB1). The second sample was an expressed prostatic secretion (EPS), obtained by prostatic massage. The third sample was a post-prostatic massage urine sample (VB2). These samples were transferred directly for analysis and culture in Microbiology Diagnostics and Infection Control Unit, Microbiology and Immunology Department, Mansoura University. EPS samples were inoculated over blood, chocolate, and MacConkey's agar (Oxoid, Hampshire, UK). Urine samples were inoculated on CLED agar (Oxoid, UK) plates that were incubated aerobically at 37°C for 24-48 hours. Used criteria for diagnosing BP were; positive EPS culture for bacteria with negative VB1 or the bacterial count in EPS and/or in VB2 be 10-folds greater than in midstream urine.

The identification of isolated bacterial colonies was carried out according to Koneman et al.[14]. The colonies were identified provisionally (klebsiella: mucoid pink colonies on MacConkey's and/or mucoid yellow colonies on CLED agars; *E. coli*: pink colonies on MacConkey's and/or yellow colonies on CLED agar. Staphylococci, Corynebacterium, and Enterococci: detect the type of hemolysis on blood agar and colonies character (all are γ-hemolytic colonies except *S. aureus* which gives B-hemolysis). Suspected Coryneform colonies were identified as non-hemolytic white-yellowish circular convex colonies.

- Gram-stained films were carried out from the growing colonies to detect the bacteria genus.

- Biochemical reactions include; catalase test, coagulase test, indole test (Oxoid, UK), methyl red test (Oxoid, UK), Voges Proskauer's reaction (Oxoid, UK), and Analytical Profile Index (BioMerieux, France) were carried out.

- PCR was carried out for the detection of *C. trachomatis* by DNA extraction using mini kits (Qiagen Hilden, Germany). It was positive for the 297 bp DNA band and reported positive for *Chlamydia trachomatis* infection in EPS.

Antimicrobial susceptibility testing was done according to Bauer et al.[15] by the disk diffusion method for levofloxacin, ciprofloxacin, norfloxacin, nitrofurantoin, and co-cotrimoxazole.

Besides, venous blood samples were estimated for total testosterone (T), serum prolactin (PRL), lipid profile, and random blood sugar.

Sample size calculation and power of the study:

At a level of confidence of 95 % with power of the study 0.8 and expected prevalence of infection was 15 percent with maxium 29 percent, the calculated sample size was 295, the research team increased the sample by thirty percent to guard against drop out, the total sample size was 397 https://www.qualtrics.com/blog/calculating-sample-size/

Statistical analysis:

Data were analyzed using the Statistical Package of Social Science (SPSS) program for Windows (Standard version 26). The normality of data was first tested with a one-sample Kolmogorov-Smirnov test.

Qualitative data were described using numbers and percentages. Association between categorical variables was tested using the Monte Carlo test when the expected cell count less than 5. Continuous variables were presented as mean ± SD (standard deviation) for normally distributed data and more than two groups were compared with the ANOVA test while the median (min-max) was used for nonparametric data and the Kruskal Wallis test was used to compare more than two medians.

RESULTS

A series of 464 patients diagnosed to have ED were screened where 50 patients did not meet the requested criteria. Also, 17 patients declined to participate in the study. Therefore, 397 patients were enrolled (mean age 44.45±10.45). Overall, 94 patients (23.7%) were diagnosed to have BP (92 cases by culture and 2 cases by PCR). Samples from 99 patients showed positive bacterial cultures (7 patients were positive for *Staphylococcus epidermidis* that were reported as contaminants and were excluded). Table (1) shows patients characteristics among the studied subjects with/without BP. Table (2) shows causative organisms and antibiotic sensitivity/resistance.

Table 1: Patients characteristics among the studied group

Patients characteristics	All patients (n=397)	ED with BP (n=94) (23.7%)	ED without BP (n=303)(76.3%)	P value
Age (years)				
Mean ± SD	**44.45±10.45**	**42.67±10.31**	**45.01±10.45**	
<30 y	23 (5.8%)	5 (5.3%)	18 (5.9%)	
30-40 y	114 (28.7%)	39 (41.5%)	75 (24.8%)	0.037*
40-50 y	154 (38.8%)	31 (33.0%)	123 (40.6%)	
50-60 y	60 (15.1%)	10 (10.6%)	50 (16.5%)	
>60 y	46 (11.6%)	9 (9.6%)	37 (12.2%)	
Duration of ED months				
Median (Min-Max)	**14 (3-300)**	**12 (3-300)**	**16 (3-240)**	
<12 m	155 (39.0%)	37 (39.4%)	118 (38.9%)	0.691
12-24 m	82 (20.7%)	22 (23.4%)	60 (19.8%)	
>24 m	160 (40.3%)	35 (37.2%)	125 (41.3%)	
Lower urinary tract symptoms				
Present	182 (45.8%)	58 (61.7%)	124 (40.9%)	≤0.001*
Absent	215 (54.2%)	36 (38.3%)	179 (59.1%)	

*significant p≤0.05

There was no significant difference in the clinical data between ED patients with/without BP except for age which was significantly lower (p= 0.037) in BP patients and also, lower urinary tract symptoms, which showed a significant difference (p = ≤0.001) with a prevalence of 61.7% in patients with BP.

Table 2: Causative organisms and antibiotic sensitivity/resistance

Causative organisms	94 patients (100%)
1-By culture of EPSE (92 patient)	
E coli	44 (46.8%)
Staphylococci	23 (24.5%)
Enterococci	16 (17.0%)
Corynebacteria (*C. seminale*)	5 (5.3%)
Klebsiella pneumoniae	4 (4.3%)
2-By PCR for Chlamydia (2 patients)	2 (2.1%)
Antibiotic sensitivity/resistance	**n=92**
Levofloxacin	
Sensitive	75 (81.5%)
Resistant	17 (18.5%)
Norfloxacin	
Sensitive	68 (73.9%)
Resistant	24 (26.1%)
Ciprofloxacin	
Sensitive	69 (75.0%)
Resistant	23 (25.0%)
Nitrofurntation	
Sensitive	64 (69.6%)
Resistant	28 (30.4%)
Co-trimoxazole	
Sensitive	29 (31.5%)
Resistant	63 (68.5%)

The antibiotic sensitivity in case of positive culture revealed that 81.5% of the isolates were sensitive to levofloxacin, 75.0% of the isolates were sensitive to ciprofloxacin , 73.9% of the isolates were sensitive to norfloxacin , 69.6% of the isolates were sensitive to nitrofurntation and 31.5% of the isolates were sensitive to cotrimoxazol .

Figure (1) shows age distribution between different organisms. Enteroccoci–induced prostatitis occurred in the highest age (48.68±11.04); while Chlamydia–

induced prostatitis occurred in the lowest age (30.5±0.71).

There was a significant difference regarding age of the patient, duration of ED and causative bacteria.

Enterococci , *S. auerus* and *K. pneumoniae*–induced prostatitis ocuured in higher age compared to others (Figure 1). However, *S. auerus*–induced prostatitis showed a longer duration of ED compared to others.

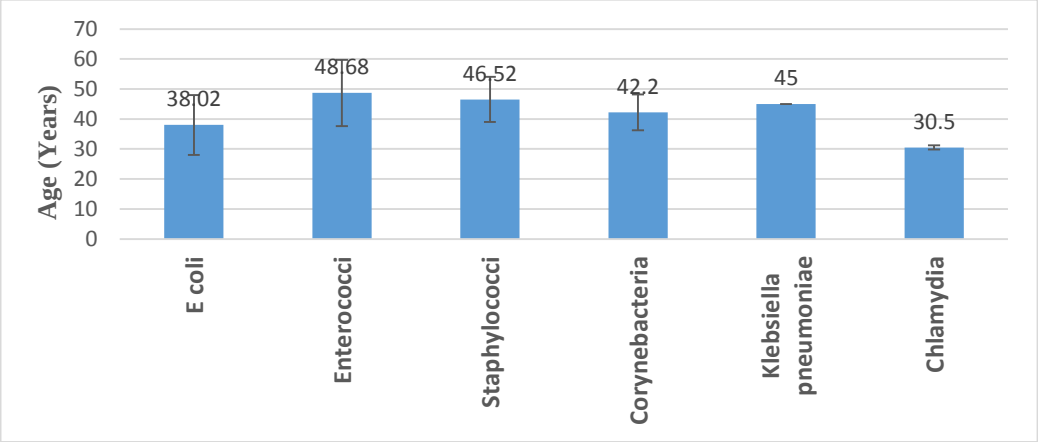

Fig. 1: Age distribution between different organisms

Enteroccoci–induced prostatitis occurred in the highest age (48.68±11.04);while Chlamydia–induced prostatitis occurred in the lowest age (30.5±0.71)

Levofloxacin was virtually effective (100%) against all BP induced by Enterococci and *Klebsiella pneumoniae* with significance compared to other isolates. *E. coli* and Enterococci were highly sensitive (86.4% and 81.2% respectively) to norfloxacin; while *K. pneumoniae* was virtually resistant to it. *E. coli* and *K. pneumoniae* were sensitive to ciprofloxacin (93.2% and 100% respectively) with a significance compared to other isolates. *K. pneumoniae* was virtually resistant to nitrofurantoin; while Enterococci, *S. auerus*, and *E. coli* were sensitive to it (81.2%, 78.3%, and 70.5% respectively). Co-cotrimoxazole was virtually ineffective against *Corynebacteria* and *K. pneumoniae* with no significance compared to other isolates (Table 3).

Table 3: The relation between each organism with the patients' age, ED duration, and antibiotic sensitivity/resistance

Patients characteristic	*E. coli*	Enterococci	Staphylococci	Corynebacteria	*K. pneumoniae*	Chlamydia	P value
Age Mean ± SD	38.02±10.4	48.68±11.04	46.52±7.5	42.2±6.2	45±0.0	30.5±0.71	0.001*
Duration of ED months Median (Min-Max)	10 (3-120)	12 (8-300)	24 (4-120)	12 (11-12)	12 (12-14)	4.5 (4-5)	0.003*
Levofloxacin Sensitive Resistant	38 (86.4) 6 (13.6)	16 (100%) 0 (0%)	13 (56.5) 10 (43.5)	4 (80%) 1 (20%)	4 (100%) 0 (0%)	-	0.006*
Norfloxacin Sensitive Resistant	38 (86.4) 6 (13.6)	13 (81.2) 3 (18.8)	15 (65.2) 8 (34.8)	2 (40%) 3 (60%)	0 (0%) 4 (100%)	-	≤0.001*
Ciprofloxacin Sensitive Resistant	41 (93.2) 3 (6.8)	12 (75%) 4 (25%)	10 (43.5) 13 (56.5)	2 (40%) 3 (60%)	4 (100%) 0 (0%)	-	≤0.001*
Nitrofurntation Sensitive Resistant	31 (70.5) 13 (29.5)	13 (81.2) 3 (18.8)	18 (78.3) 5 (21.7)	2 (40%) 3 (60%)	0 (0%) 4 (100%)	-	0.008*
Cotrimoxazol Sensitive Resistant	17 (38.6) 27 (61.4)	3 (18.8) 13 (81.2)	9 (39.1) 14 (60.9)	0 (0%) 5 (100)	0 (0%) 4 (100%)	-	0.131

*significant p ≤0.05

DISCUSSION

In the current study, *E. coli* was the commonest single isolate followed by *S. aureus* and *E. fecalis*, *C. seminale*, and then *K. pneumoniae* and *C. trachomatis*. Naber[16] reported that *E. coli* was the commonest cause of CBP whereas Lipsky et al.[17] reported a higher prevalence of *E. coli* (50–80%) and *K. pneumoniae* (10%), and a lower percentage for Enterococcus species (5-10%). Karlowsky et al.[18] reported that *E. coli* continues to be the most common cause of uncomplicated and complicated urinary tract infections, and acute and chronic BP. Also, Stamatiou and Pierris[19] reported that the most frequent pathogen from patients with BP for the first time and from relapsing patients with a history of BP and previous antibiotic treatment was *E. coli*.

There was a significant difference in the age of the patient and causative bacteria. Enterococci, *S. auerus*, and *K. pneumoniae* –induced prostatitis occurred at higher age compared to others (Figure 1). However, Chlamydia prostatitis occurred at the lowest age (30.5±0.71).

Regarding *E. coli*, 93.2 % of isolates were sensitive to ciprofloxacin, 86.4% to norfloxacin and levofloxacin, 70.5% to nitrofurantoin, and 38.6% to co-cotrimoxazole. In their study, Cai et al.[20] reported that 90.7% and 88.5% of cases were sensitive to ciprofloxacin, and levofloxacin, respectively, and a higher sensitivity to nitrofurantoin (86.2%), co-trimoxazole (77.5%), and a lower one for norfloxacin (82%).

All isolates of *E. fecalis* were sensitive to levofloxacin. Meanwhile, it was sensitive to norfloxacin and nitrofurantoin in 81.2% of cases; and to ciprofloxacin and co-trimoxazole in 75% and 18.8% of cases respectively. In their studies, Farajnia et al.[21] and Sharifi et al.[22] generated similar results in urinary tract infections.

S. aureus was sensitive to nitrofurantoin in 78.3% of isolates and norfloxacin, levofloxacin, and ciprofloxacin in 65.2 %, 56.5%, and 43.5% of isolates respectively. Cai et al.[20] reported sensitivity of 89%, 70.8%, 65.8%, and 70% to nitrofurantoin, ciprofloxacin, norfloxacin, and levofloxacin, respectively in the EPS.

Isolates of *C. seminale* were sensitive to levofloxacin in 80% of cases. Meanwhile, it was sensitive to ciprofloxacin, norfloxacin, and nitrofurantoin in 40% of cases; and all isolates were resistant to co-trimoxazole. Mashaly et al.[23] found considerable resistance to clindamycin, doxycycline, erythromycin, and nitrofurantoin. In their study, Türk et al. [24] reported that 35% of Coryneforms were resistant to doxycycline and more than half were resistant to clindamycin (63%), nitrofurantoin (62%), and erythromycin (53%). They also reported a sensitivity of 75%) for ciprofloxacin in seminal fluid. Mashaly et al.[23] reported 41.6% for erythromycin and 75%, 95% for ciprofloxacin, and norfloxacin in seminal fluid.

Only 4 cases of *K. pneumoniae* were isolated which was virtually sensitive to ciprofloxacin and levofloxacin. However, it was virtually resistant to nitrofurantoin, norfloxacin, and co-trimoxazole. Cai et al.[20] results in EPS were close to ours regarding ciprofloxacin (91.1%) and levofloxacin (92.5%), but it was in disagreement regarding nitrofurantoin (83.6% of isolates were sensitive).

Several hypotheses tried to explain ED in patients with BP as decreased arterial inflow due to pelvic floor spasm, and inflammation in endothelial tissues that can induce ED through impaired nitric oxide (NO) availability[25]. Shoske et al.[11] reported that the incidence of vascular endothelial cell damage is higher in patients with CP/CPPS than in the healthy population. Thus, vascular endothelial damage could disturb the arterial perfusion of the penis which may cause ED in patients with CP/CPPS. At the molecular levels, Huang et al.[26] showed increased levels of inflammatory mediators IL-8, IL-1β, and ICAM-1 in the prostatic fluid of CP/CPPS patients where those mediator levels were negatively correlated with IIEF-5 scores. Besides, Alkan et al.[27] suggested that superoxide anion ($O_2^{\bullet-}$) and total reactive oxygen species (ROS) overproduction could be one of the important mechanisms in the etiology of ED development in CP/CPPS patients. Another important detrimental effect on erectile function is the Rho/Rho-kinase pathway where ROS activation of the RhoA/Rho kinase pathway results in vasoconstriction and inhibition of eNOS expression[28,29] .

The limitation of this study is the need to find a cause-related effect by reassessment of erectile function after a sufficient period of anti-microbial therapy.

CONCLUSIONS

In the current study, the prevalence of BP in men with ED was 23.7%. *E. coli* was the commonest isolate followed by *S. aureus* and *E. fecalis*. Antibiotic sensitivity revealed that the most effective tested class in decreasing order are; levofloxacin (81.5%), ciprofloxacin (75.%), norfloxacin (73.9%), nitrofurantoin (69.6%) and cotrimoxazole (31.5%). However, the relationship between ED and BP should be dealt with caution for further investigations.

This manuscript has not been previously published and is not under consideration in the same or substantially similar form in any other reviewed media. I have contributed sufficiently to the project to be included as author. To the best of my knowledge, no conflict of interest, financial or others exist. All authors have participated in the concept and design, analysis, and interpretation of data, drafting and revising of the

manuscript, and that they have approved the manuscript as submitted.

REFERENCES

1. Holt JD., Garrett WA., McCurry TK, Teichman JM. Common questions about chronic prostatitis. Am Fam Physician 2016; 93:290-296.
2. Urkmez A., Yuksel OH, Uruc F, Akan S, Yildirim C, Sahin A, et al.The effect of symptomatic histological prostatitis on sexual function and lower urinary tract symptoms. Arch Esp Urol 2016; 69:185-191.
3. Liu L, Yang J, Lu F..Urethral dysbacteriosis as an underlying, primary cause of chronic prostatitis: Potential implications for probiotic therapy. Med Hypotheses 2009; 73:741-743.
4. Polackwich AS, Shoskes DA. Chronic prostatitis/chronic pelvic pain syndrome: A review of evaluation and therapy. Prostate Cancer Prostatic Dis 2016;19(2):132-8.
5. Hatzimouratidis K, Amar E, Eardley I, Giuliano F, Hatzichristou D, Montorsi F, et al. Guidelines on male sexual dysfunction: erectile dysfunction and premature ejaculation. Eur Urol 2010; 57:804-14.
6. Mizuno T, Hiramatsu I, Aoki Y, Shimoyama H, Nozaki T, Shirai M, et. al. Relation between histological prostatitis and lower urinary tract symptoms and erectile function. Prostate Int 2017; 5:119-23.
7. Zhang Y, Zheng T, Tu X'an, Chen X, Wang Z, Chen S, et al. Erectile Dysfunction in Chronic Prostatitis/Chronic Pelvic Pain Syndrome: Outcomes from a Multi-Center Study and Risk Factor Analysis in a Single Center. PLoS ONE 2016; 11(4):e0153054. doi:10.1371/journal.pone.0153054.
8. Trinchieri A, Magri V, Cariani L, Bonamore R, Restelli A, Garlaschi MC,et al. Prevalence of sexual dysfunction in men with chronic prostatitis/chronic pelvic pain syndrome. Arch Ital Urol Androl 2007;79: 68-9.
9. Smith KB, Pukall CF, Tripp DA, Nickel JC. Sexual and relationship functioning in men with chronic prostatitis/chronic pelvic pain syndrome and their partners. The Arch Sex Behav 2007; 36:301–11.
10. Wagenlehner F, Pilatz A, Linn T, Diemer T, Schuppe HC, Schagdarsurengin U, et al. Prostatitis and andrological implications. Minerva Urol Nefrol 2013;65:117-23.
11. Shoskes DA, Nickel JC. Quercetin for chronic prostatitis/chronic pelvic pain syndrome. Urol Clin North Am 2011; 38:279-84.
12. Rosen RC, Cappelleri JC, Smith MD, Lipsky J, Peña BM. Development and evaluation of an abridged, 5-item version of the International Index of Erectile Function (IIEF-5) as a diagnostic tool for erectile dysfunction. Int J Impot Res 1999; 11:319-26.
13. Nickel JC, Shoskes D, Wang Y, Alexander RB, Fowler JE, Zeitlin S, et al. How does the pre-massage and post-massage 2-glass test compare to the Meares-Stamey 4-glass test in men with chronic prostatitis/chronic pelvic pain syndrome? Urol J 2006; 176:119–24.
14. Koneman EW, Scheckenberger PC, Allen SD, Winn WC, Janda WM (4th ed.) . Color atlas and textbook of diagnostic microbiology. Philadelphia, Lippincott Comp; 1992; 243–77.
15. Bauer AW, Kirby WM, Sherris JC, Turck M. Antibiotic susceptibility testing by a standardized single disk method. Am J Clin Pathol 1966; 45: 493-96.
16. Naber KG. Management of bacterial prostatitis: what's new?. BJU International 2008; 101:7-10.
17. Lipsky BA, Byren I, Hoey CT.Treatment of bacterial prostatitis. Clin Infect Dis 2010; 50:1641–52.
18. Karlowsky JA, Denisuik AJ, Lagacé-Wiens PR, Adam HJ, Baxter MR, Hoban DJ, et al. In vitro activity of fosfomycin against Escherichia coli isolated from patients with urinary tract infections in Canada as part of the CANWARD surveillance study. Antimicrob Agents Chemother 2014; 58(2):1252–56.
19. Stamatiou K, Pierris N. Mounting resistance of uropathogens to antimicrobial agents: A retrospective study in patients with chronic bacterial prostatitis relapse. Investig Clin Urol 2017; 58 (4): 271-80.
20. Cai T, Mazzoli S, Meacci F, Boddi V, Mondaini N, Malossini G, Bartoletti R. Epidemiological features and resistance pattern in uropathogene isolate from chronic bacterial prostatitis. J Microbiol 2011; 49:448-54.
21. Farajnia S, Alikhani MY, Ghotaslou R, Naghili B, Nakhlband A. Causative agents and antimicrobial susceptibilities of urinary tract infections in the northwest of Iran. Int J Infect Dis 2009; 13:140-44.
22. Sharifi Y, Hasani A, Ghotaslou R, Naghil, B, Aghazadeh M, Milani M, et al. Virulence and antimicrobial resistance in enterococci isolated from urinary tract infections. Adv Pharm Bull 2013;3:197-201.
23. Mashaly M, Masallat DT, Elkholy AA, Abdel-Hamid IA, Mostafa T. Seminal Corynebacterium

strains in infertile men with and without leucocytospermia. Andrologia 2016;48: 355-59.

24. Türk S, Punab M, Mändar R. Antimicrobial susceptibility patterns of coryneform bacteria isolated from semen. Open Forum Infect Dis 2009;3: 31-36.
25. Blans MC, Visseren F.L Banga JD, Hoekstra JB, van der Graaf Y, Diepersloot RJ, et al. Infection induced inflammation is associated with erectile dysfunction in men with diabetes. Eur J Clin 2006; 36:497-502.
26. Huang TR, Li W, Peng B. Correlation of inflammatory mediators in prostatic secretion with chronic prostatitis and chronic pelvic pain syndrome. Andrologia 2018;50:e12860.
27. Alkan I, Yüksel M, Özveri H, Atalay A, Canat HL, Culha MG, et al. Semen reactive oxygen species levels are correlated with erectile function among chronic prostatitis/chronic pelvic pain syndrome patients. Int J Impot Res 2018; 30: 335-41.
28. Bivalacqua TJ, Champion HC, Usta MF, Cellek S, Chitaley K, Webb RC .RhoA/Rho-kinase suppresses endothelial nitric oxide synthase in the penis: a mechanism for diabetes associated erectile dysfunction.Proc Natl Acad Sci USA 2004; 101:9121–26.
29. Li WJ, Park K, Paick JS, Kim SW.Chronic treatment with an oral rho-kinase inhibitor restores erectile function by suppressing corporal apoptosis in diabetic rats. J Sex Med 2011;8:400-10.