

Full Length Research Paper

Prevalence of asymptomatic bacteriuria in prostatitis subjects attending University of Port Harcourt Teaching Hospital

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This study was carried out to determine the prevalence of asymptomatic bacteriuria in Prostatitis subjects attending urology clinic at University of Port Harcourt Teaching Hospital (UPTH). Fifty eight (58) prostatitis subjects attending urology clinic at University of Port Harcourt Teaching Hospital and 54 apparently normal subjects working at University of Port Harcourt Teaching Hospital (UPTH) were used for this study. Inclusion and exclusion conditions were observed in patients' selection. All the subjects had their blood Prostate specific antigen (PSA) determined by enzymes linked immunosorbent assay (ELISA). The urine samples were cultured on Cystine Lactose electrolyte deficient medium (CLED) and Chocolate agar by Standardized wire loop technique, incubated overnight at 37°C and the isolates identified. There was significant difference in PSA concentration of prostatitis subjects and controls ($P < 0.01$). Prostatitis subjects had significant bacteriuria compared with the controls ($P < 0.05$). *Klebsiella* spp was identified to be the predominant etiologic agent in prostatitis subjects while *S. aureus* was the prevalent uropathogen in controls. The result of this study suggests that bacteriuria is prevalent among elderly men in some pathological conditions such as Prostatitis. This may be caused by inflammation of prostate leading to non secretion of prostatic fluid which is bactericidal.

Key word: Prostatitis, asymptomatic, bacteriuria, uropathogen.

INTRODUCTION

Urine is the liquid waste excreted from the body. It is composed of various chemical substances dissolved in the excess water filtered from the blood by the kidney. Urine secreted in the kidney is sterile unless the kidney is infected. Bacteriuria is defined as presence of bacteria in urine. Significant bacteriuria is the occurrence of 10^5 or more bacteria per ml of a voided midstream urine (Kass, 1956). The use of significant bacteriuria to confirm the presence of urinary tract infection (UTI) has been validated in several studies (Kass, 1956; Clauge and

Horan, 1994). Infection of the urinary tract occurs when there is introduction of an appropriate organism and a reason for its persistence and multiplication (Becker, 1986). Because of physiologic changes related to aging and comorbid illnesses, asymptomatic bacteriuria (ASB) is a common occurrence in older adults. Neither short-term nor long-term adverse outcomes attributable to the high incidence and prevalence of ASB have been shown in this population (Manisha Juthani-Mehta, 2009; 2007). Population studies throughout the world have shown a rise in the prevalence of asymptomatic bacteriuria with age. ASB is very uncommon in young men, but for men over the age of 65, the prevalence ranges from 5 to 21% and is highest in those men over the age of 90 (Nicolle, 1997).

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The Prostate is an organ which lies at the base of the bladder around the urethra in males. Prostate enlargement causes increased frequency of micturition, nocturia and difficulty in initiating micturition. With advance age it tends to enlarge (benign prostatic hyperplasia) and may result in obstruction of the urethra. Occasionally malignant changes occur. The prostate gland in men may be a focus of infection and can give rise to fever, dysuria, increased frequency and perineal pain (Toohey, 1994). Prostatitis is a common syndrome characterized by infection, pain and /or inflammation of the prostate or surrounding tissue. Prostatitis has been reported as the most common urologic diagnosis for men under the age of Fifty and the third most common urologic diagnosis for those over Fifty (Collins et al., 1998). Severe benign prostatic hyperplasia is often implicated as a risk for recurrent UTI. When possible, resection of the prostate can assist in reducing recurrent episodes (Manisha Juthani-Mehta, 2009; 2007). Laboratory confirmation of UTIs with significant bacteriuria ($\geq 10^5$ cfu/ml on urine culture) and pyuria (≥ 10 white blood cells on urinalysis) was agreed on as minimum necessary but not sufficient criteria for diagnosis of UTI in this population (Garner et al., 1988).

The epidemiological literature reported that 9-50% of all men will be diagnosed with Prostatitis at some time in their life (Stamey, 1980; Roberts et al., 1998). National Institutes of Health (NIH) consensus classification of prostatitis syndrome include (a) acute bacterial prostatitis (b) Chronic bacterial prostatitis (c) Chronic Prostatitis (i) Inflammatory and (ii) Non Inflammatory (d) Asymptomatic inflammatory Prostatitis. Acute or chronic bacterial prostatitis are based on symptoms and identification of bacteria in the urine or expressed prostate secretions, chronic pelvic pain syndrome is based on symptoms of chronic pelvic pain with the last group referring to asymptomatic patients with coincidental findings of prostate inflammation in patients undergoing evaluation for benign prostatic hyperplasia, Prostate cancer or infertility (Schaeffer, 2000). The diagnosis and treatment of Prostatitis remains appropriate localization of inflammation and infection to the prostate (Meares and Stamey, 1968). This study was designed to compare the prevalence of asymptomatic bacteriuria in Prostatitis and non prostatitis subjects attending University of Port Harcourt Teaching Hospital.

MATERIALS AND METHODS

Subjects

Fifty eight (58) subjects who has Prostate Specific antigen (PSA) value greater than 4ng/ml (>4.0 ng/ml) attending urology clinic at University of Port Harcourt Teaching hospital (UPTH) were selected as test subjects in this study. Fifty four (54) control subjects were volunteers at University of Port Harcourt Teaching hospital (UPTH) whose PSA values were below 4.0 ng/ml (<4 ng/ml). The age range of both controls and prostatitis subjects was 45-80 years. Five of

the Prostatitis subjects were hospitalized while none was hospitalized nor show symptoms of urinary tract infection among the controls. The Prostatitis subjects were diagnosed by physicians at the hospital after rectal examination. Inclusion criteria in this study include pain or discomfort in the pelvic area (penis, scrotum, perineum, or thereabouts) for at least 3 months, tender, tense prostate on rectal exam consistent with a diagnosis of acute prostatitis or soft, tender prostate without nodules consistent with a diagnosis of chronic prostatitis and one or more symptoms from the following group: Disturbances of urination, including frequency, urgency, dysuria, and/or lower urinary tract obstruction (more commonly seen in patients with chronic disease), hesitancy, decreased stream, urinary retention; perineal or low back pain; fevers; or chills. Exclusion criteria in this study include the presence of cancer of the genitourinary tract, active urinary stone disease, herpes of the genitourinary system, bacteriuria (100,000 colonies in a midstream urine) within the past 3 months, antibiotic therapy within the past 3 months, peri-rectal inflammatory disorders, inflammatory bowel disease, history of pelvic radiation or systemic chemotherapy, history of intravesical chemotherapy, documented gonorrhea, chlamydia, mycoplasma, or trichomonas infection of the urinary tract within the past 3 months, clinical epididymitis within the past 3 months, urethral stricture of 12 French or smaller, neurological disease or disorder affecting the bladder and prostate surgery (not including cystoscopy) within the past 3 months. Blood samples were collected from subjects by venepuncture for PSA determination in both the control and prostatitis subjects.

Clean catch midstream urine specimen of all subjects were collected aseptically into sterile universal bottles and taken immediately into the laboratory. The urine was cultured on cysteine lactose electrolyte deficient (CLED) and chocolate agar by standardized wire loop technique. The microscopic examination of the urine was then carried out. The Chocolate agar culture plate was incubated in the absence of oxygen overnight at 37°C while the CLED culture plate was incubated aerobically overnight at 37°C. The isolates were identified by standard methods such as Gram staining procedure, oxidase test, motility test, catalase test, coagulase test, indole test, methyl red test, Voges-Proskauer and citrate utilization test (Cheesbrough, 2000). Discrete colonies were counted to determine significant bacteriuria (300 colonies and above per plate were considered significant). For microscopic examination, each urine sample was mixed properly in a test tube and spun at 3000 rpm for 5 min. The supernatant was discarded and sediment loosened gently by tapping the bottom of the tube. A drop of sediment was placed on a clean glass slide, covered with cover slip and viewed under the microscope using the 10x and 40x objectives.

The PSA determination was done using TECO prostate specific antigen enzymes linked immunosorbent assay (ELISA) reagent produced by TECO diagnostics, USA. The PSA reagents were brought to room temperature while the coated wells were secured in the holder. Twenty five (25 ul) microlitre of standards, sample and controls were pipetted and dispensed into respective well while 100 ul of conjugate reagent was pipetted into each well, mixed thoroughly and incubated for 30 min. The contents of each well were discarded by decanting, blotted dry with absorbent paper. The wells were washed using 300 ul wash solution. The washing was repeated twice then 100 ul of tetramethylbenzene (TMB) solution was added into each well, mixed and incubated at room temperature in the dark for 15 min. Fifty microlitre (50 ul) of stop solution was added to each well, mixed till colour changes from blue to yellow and absorbance read at 450 nm using spectrophotometer. The result of the unknown was extrapolated from the curve prepared from the standards (Chen, 1995; Junker et al., 1997; Horton et al., 1988).

The data were subjected to some statistical analysis and reported as Percentage (%), mean and Standard deviation (SD). PSA Values were reported as Mean \pm SD while student's t-test was

Table 1. Prevalent bacterial isolates in prostatitis and control.

Isolates	Prostatitis No (%)	Control No (%)	P Value
<i>Klebsiella</i> sp	21(36.20)	2(3.70)	<0.05
<i>Staphylococcus aureus</i>	8 (13.80)	14(25.90)	<0.05
<i>Proteus</i> sp	7 (12.10)	1 (1.90)	<0.05
<i>E. coli</i>	8 (13.80)	8 (14.80)	>0.05
<i>P. aeruginosa</i>	0 (0.00)	1(1.90)	>0.05
No bacteria growth	14(24.10)	28 (51.80)	<0.05

Table 2. Age distribution and prevalence of significant bacteria in prostatitis subjects.

Group	Age group (Years)	Status	No of subjects	Significant bacteriuria No (%)	No of isolates (%)					No bacteria growth
					<i>Klebsiella</i> spp	<i>S. aureus</i>	<i>Proteus</i> spp	<i>E. coli</i>	<i>P. aeruginosa</i>	
A	45-55	Prostatitis	28	21(75.00)	15(53.57)	5(17.86)	0(0.00)	1(3.57)	0(0.00)	7(25.00)
		Control	30	9(30.00)	0 (0.00)	7(23.33)	0(0.00)	2(6.67)	0(0.00)	21(70.00)
B	56-65	Prostatitis	10	6(60.00)	2 (20.00)	1(10.00)	2(20.00)	1(10.00)	0(0.00)	4(40.00)
		Control	10	6(60.00)	0(0.00)	3(30.00)	0(0.00)	3(30.00)	0(0.00)	4(40.00)
C	66-75	Prostatitis	16	14(87.50)	3(18.75)	2(12.50)	3(18.75)	6(37.50)	0(0.00)	2(12.50)
		Control	12	10(83.33)	2 (16.67)	4(33.33)	1(8.33)	3(25.00)	0(0.00)	2(16.67)
D	76-80	Prostatitis	4	3(75.00)	1(25.00)	0(0.00)	2(50.00)	0(0.00)	0(0.00)	1(25.00)
		Control	2	1(50.00)	0(0.00)	0(0.00)	0(0.00)	0(0.00)	1(50.00)	1(50.00)

used to test for differences between treatment groups using Statistical Package for Social Sciences (SPSS) version 16.A value of $P < 0.05$ was accepted as significant.

RESULTS

Klebsiella spp was isolated in 21(36.20%) in prostatitis subjects while only 2(3.70%) had the organism isolated from their urine in control subjects. The incidence of *Staphylococcus aureus* was 14(25.90%) in control while in Prostatitis subjects it was 8(13.80%). *Proteus* spp isolated from the urine of Prostatitis subject was 7(12.10%) while only 1(1.90%) was the incidence in the control. *E. coli* had incidence of 8(13.80%) and 8(14.80%) in both prostatitis and control respectively while *Pseudomonas aeruginosa* had 1(1.90%) occurrence in control without any occurrence in Prostatitis subjects (0(0.00%)) as shown in Table 1.

In group A, 21 (75%) subjects had significant bacteriuria in prostatitis. *Klebsiella* spp. accounted for 15(53.57%), *S. aureus* 5 (17.86%), *Proteus* Spp. and *E. coli* 1(3.57%) while 7(25%) had no bacteria growth. The respective control (A control) had 9(30.00%) prevalence of significant bacteriuria with *S. aureus* 7 (23.33%) and *E. coli* 2(6.67%) being the isolates.

The prevalence of significant bacteriuria in prostatitis subjects in group B was 6(60%) with prevalence of *Klebsiella* spp. at 2 (20.00%), *S. aureus* 1 (10.00%), *Proteus* spp. 2 (20.00%) and *E. coli* 1(10.00%) while 4(40%) had no bacteria isolated in their urine. In the control subjects of group B, 6(60) had significant bacteriuria consisting of *S. aureus* at 3(30.00%), and *E. coli* 3(30.00%).

The prevalence of *E. coli* in Prostatitis subjects in group C subject was 6(37.50%), while other isolates include *Klebsiella* spp. 3 (18.75%), *Proteus* spp. 3 (18.75%) and *S. aureus* 2 (12.50%) in 14(87.5%) subjects with significant bacteria growth. In the corresponding control subjects, 10(83.33%) had significant bacteria growth. *S. aureus* has the highest prevalence at 4 (33.33%), others include *E. coli* 3(25.00%), *Klebsiella* sp. 2(16.67%) while *Proteus* sp. was the least prevalent at 1(8.33%).

In group D subjects, the prevalence with significant bacteria growth was 3(75%). *Klebsiella* spp. was isolated in 1(25.00%) and *Proteus* spp. 2(50.00%) in the urine. The respective control showed significant bacteriuria in 1(50%) with *P. aeruginosa* occurring in 1(50.00%) as shown in Table 2.

The total PSA concentration (ng/ml) in Prostatitis subject of 13.55 ± 7.80 was significantly higher than 1.74

Table 3. Prostate specific antigen (PSA) concentration in prostatitis subjects.

Group	Age group (Years)	Prostate specific antigen(PSA) ng/ml		
		Prostatitis	Control	P value
A	45-55	13.15 ± 7.75	1.74 ± 0.60	<0.01
B	56-65	10.70 ± 4.32	1.68 ± 0.68	<0.01
C	66-75	13.56 ± 7.33	1.56 ± 0.40	<0.01
D	76-80	23.43 ± 12.50	2.53 ± 2.33	<0.01
	Total	13.55 ± 7.80	1.70 ± 0.65	<0.01

Mean ± SD.

± 0.60 in control. In group A, the PSA concentration (ng/ml) was 13.15 in Prostatitis subject while it was 1.74 ± 0.60 in control (P<0.01). The PSA concentration (ng/ml) of 10.70 ± 4.32 in Prostatitis subject was significantly different from 1.68 ± 0.68 of the control (P<0.01) in group B. Also the PSA concentration (ng/ml) of 13.56 ± 7.33 in Prostatitis subjects in group C was significantly higher than 1.56 ± 0.40 of the control (P<0.01) while the PSA concentration (ng/ml) of 23.43 ± 12.50 in Prostatitis subject was significantly different from 2.53 ± 2.33 obtained in controls in group D (P<0.01) as shown in Table 3.

DISCUSSION

The study was carried out to ascertain prevalence of bacteriuria in prostatitis. With the criterion of 10⁵ colonies/ml of urine for significant bacteriuria, this study revealed a higher prevalence of urinary tract infection (UTI) in prostatitis subjects compared to the controls (P<0.05). The prevalence obtained in this study was higher than study by Stamey and Pfau (1970). This may be due to absence of good marker like PSA for proper diagnosis before this time. The use of PSA as a better marker in diagnosis and management of adenocarcinoma of the prostate has been reported (Stamey et al., 1987) while Dalton (1989) reported elevated PSA in patients with acute bacteria Prostatitis. Prostatic fluid has bactericidal activity which helps prevent infection. In Prostate hypertrophy or Prostatitis, there is no secretion of the prostatic fluid which makes the prostate gland prone to infection by microorganism.

Also the result of this study showed *Klebsiella* spp. as the prevalent uropathogen in Prostatitis subjects, followed by *S. aureus*, *E. coli* and Proteus Species. The occurrence of *Klebsiella* spp. and *E. coli* may be from subjects own gut while Proteus spp which is associated with the use of instrument or obstruction may be from renal abnormalities (Brooks et al., 1998a). Several authors, Becker (1986), Drucker et al. (1983) and Olusanya and Olutiola (1984) have noted that most common uropathogens in UTI such as *E. coli* are enteric. In the control subjects *S. aureus* was the most prevalent

organism, followed by *E. coli*, *Klebsiella* spp, Proteus spp. and *P. aeruginosa*. The control subject with *P. aeruginosa* reported a history of surgical operations suggesting the occurrence of the organism might be from the use of instrument (Smith and Easman, 1990). Ubiquitous nature of *S. aureus* (Brooks et al., 1998b) may be the reason for its high prevalence in both Prostatitis and control subjects. The prevalence of bacteriuria in Prostatitis subjects may be fatal with decline in renal function and increase mortality rates. The cause of asymptomatic bacteriuria in the elderly is multifactorial, but incomplete emptying of the bladder is believed to be the primary source (Brooks et al., 1998b; Ribeiro et al., 2002; Baldassarre and Kaye, 1991; Nicolle, 2003; Reid and Nicolle, 1999) especially in pathological condition like Prostatitis. Bacteria gain access to the urinary tract by the ascending route from the perineum and are typically eliminated by urine flow. CLED agar was used as a culture medium in this study because its electrolyte deficiency prevents the swarming of Proteus species and good colonial differentiation with most urinary pathogen (Baker et al., 2000).

The PSA concentration in Prostatitis subject in group D years was the highest, followed by group C, suggesting that PSA concentration increased as age increases (Table 3). PSA levels tend to increase with age; this increase is related to prostate volume. Most PSA is made in the transition zone (TZ) of the prostate and this region of the prostate increases in volume in men with Benign Prostate Hyperplasia (BPH). Studies have also shown an increase in PSA level in men with signs and symptoms of urinary tract infection (UTI) and a positive bacterial culture. These studies demonstrated that most of these men had a median PSA level increase of 14.1 ng/mL during the acute phase of an infection, which remained elevated for some time, taking up to 6 months to return to baseline levels (Zackrisson et al., 2003; Bañez et al., 2007). Toohey (1994) reported that prostate tend to enlarge with advance age causing fever, dysuria, increased frequency and perineal pain while severe benign prostatic hypertrophy has often been implicated as a risk for recurrent UTI (Manisha Juthani-Mehta, 2009; Manisha Juthani-Mehta, 2007). Therefore this group of subjects should always be screened for Prostatitis and

bacteriuria.

Also group C years had the highest prevalence of bacteriuria in Prostatitis subjects, with *E. coli* being the most prevalent uropathogen. In group A; *Klebsiella* spp. was the prevalent uropathogen followed by *S. aureus*. These 2 groups of subjects had the highest PSA concentrations and bacteriuria while subjects in group B had the least PSA concentration vis-à-vis bacteriuria. This confirms study of Dalton (1989) showing elevation of PSA concentration in acute bacteria Prostatitis.

This may be as a result of non secretion of prostatic fluid in this group of subjects as confirmed by the elevation of PSA thus making the prostate gland prone to infection by microorganism. Population studies throughout the world have shown a rise in the prevalence of asymptomatic bacteriuria with age. Asymptomatic bacteriuria (ASB) is very uncommon in young men, but for men over the age of 65, the prevalence ranges from 5 to 21% and is highest in those men over the age of 90 (Nicolle, 1997). UTIs are the second most common cause of infectious disease hospitalization in adults 65 years or older after lower respiratory tract infections (Curns et al., 2005).

Pyuria is defined as the presence of white blood cells in a person's urine. It is usually indicative of a host response to a stimulus, such as bacteria in the urine. Pyuria is common (>90% prevalence) even in asymptomatic bacteriuria, and therefore does not help distinguish asymptomatic bacteriuria from "true" urinary tract infection requiring treatment (Nicolle, 1997). However, the absence of pyuria in an immunocompetent host does make "true infection" unlikely. Pyuria was not used as a reliable index of the presence of disease because it may be absent during the process or may occur in disorders other than bacterial infection such as extreme dehydration, trauma secondary to instrumentation or calculi, chemical inflammation, renal tuberculosis, acute glomerulonephritis, non bacterial gastroenteritis and respiratory infection (Lennette et al., 1985). Alausa et al. (1979) also reported that factors such as urine PH and techniques of examination influence the detection of pyuria. This explains why PSA concentration was used to confirm cases of Prostatitis rather than pyuria.

The result of this study carried out in a teaching hospital in Nigeria showed the presence of symptomless UTI in prostatitis (prostate inflammation) probably caused by lack or low secretion of the prostatic fluid. However, duration of prostatitis was not taken into account during this study. Furthermore our study gives an insight into prevalent etiologic bacteria agents. Additionally, study on chemotherapeutic intervention could provide a good option in treatment of these uropathogen.

Conclusion

This study has shown that significant bacteriuria is prevalent in Prostatitis subjects hence Prostatitis subjects

should always be screened and treated for bacteriuria.

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REFERENCES

- Alausa KO, Montefiore, Sobayo E (1979). Problems in the diagnosis of urinary tract infections. *Niger. Med. J.*, 9(1):107-111.
- Baker FJ, Silverton RE, Pallister CJ (2000). Preparation of culture media in: *Introduction to Medical Laboratory Technology*. Bounty Press Limited Nigeria. pp. 269-270.
- Baldassarre JS, Kaye D (1991). Special problems of urinary tract infection in the elderly. *Med. Clin. North Am.*, 75(2):375-390.
- Bañez LL, Hamilton RJ, Partin AW (2007). Obesity-related plasma hemodilution and PSA concentration among men with prostate cancer. *JAMA*, 298(19):2275-2280.
- Becker G (1986). Urinary tract infection and reflux nephropathy in adults. *Med. int.*, 1337-1347.
- Brooks GF, Butel JS, Morse SA (1998a). Enterobacteriaceae in: *Jawetz, Melnick and Adelberg's Medical Microbiology*, 21st edition. Edited by Ransom James. Prentice Hall Int Inc/Appleton and Lange Stamford USA, pp. 217-228.
- Brooks GF, Butel JS, Morse SA (1998b). Staphylococci in: *Jawetz, Melnick and Adelberg's Medical Microbiology*, 21st edition. Edited by Ransom James. Prentice Hall Int Inc/Appleton and Lange Stamford USA, pp. 197-202.
- Cheesbrough M (2000). *Biochemical testing of Microorganisms in: District Laboratory Practice in Tropical Countries, Part 2*. Cambridge University Press, U.K., pp. 275.
- Chen Z (1995). A. Prestigiacomo, T. Stamey, *Clin. Chem.*, 41:1273-1282.
- Clauge JE, Horan MA (1994). Urine culture in elderly scientifically doubtful and practically useless. *Lancet*, 344:1035-1036.
- Collins MN, Stafford RS, O'Leary MP, Barry MJ (1998). How common is prostatitis? A national survey of Physician visits. *J. Urol.*, 159:1224-1228.
- Curns AT, Holman RC, Sejvar JJ, Owings MF, Schonberger LB (2005). Infectious disease hospitalizations among older adults in the United States from 1990 through 2002. *Arch. Intern. Med.*, 165: 2514-2520.
- Dalton DL (1989). Elevated serum prostate specific antigen due to acute bacterial prostatitis. *Urology*, 33(6):465.
- Drucker M, Nitzan Y, Maayan M, Wajzman M (1983). Urinary tract infection and drug resistance bacteria in different patients population. *Israeli J. Med. Sci.*, 19:1039-1045.
- Garner JS, Jarvis WR, Emori TG, Horan TC, Hughes JM (1988). CDC definitions for nosocomial infections, *Am. J. Infect. Control*, 16: 128-140.
- Horton GL, Bahnson RR, Datt M, Chfhan KM, Catalona WJ, Landenson JH (1988). Differences in values obtained with two assays of prostate specific antigen. *J. Urol.*, 139: 762-772.
- Junker R, Brandt B, Zechel C, Assman G (1997). Comparison of Prostate-specific antigen (PSA) measured by four combinations of free-PSA and total PSA assays. *Clin. Chem.*, 43:1588-1594.
- Kass EH (1956). Asymptomatic infections of the urinary tract. *Trans. Assoc. Am. Physicians*, 69:56-64.
- Lennette EH, Balows A, Hausler WJ (Jnr), Shadommy J (1985). Collection and handling of specimen in: *Manual of Clinical Microbiology*. 4th edition. American Society for Microbiology, Washington D.C., pp. 81-82.
- Manisha Juthani-Mehta (2009). *Urinary Tract Infections in Elderly Persons*. *Geriatric Nephrology Curriculum*, Am. Soc. Nephrol.,

pp. 1-4.

- Manisha Juthani-Mehta (2007). Asymptomatic Bacteriuria and Urinary Tract Infection in Older Adults. *Clinics in Geriatric Medicine*, Elsevier. 23(3):585-594.
- Meares EM, Stamey TA (1968). Bacteriologic localization patterns in bacterial prostatitis and Urethritis. *Invest. Urol.*, 5:492
- Nicolle LE (1997). Asymptomatic bacteriuria in the elderly. *Infect. Dis. Clin. North Am.*, 11 (3):647-662.
- Nicolle LE (2003). Asymptomatic bacteriuria: When to screen and when to treat. *Infect Dis. Clin. North Am.*, (17):367-394
- Olusanya O, Olutiola PO (1984). Studies on bacteriuria in patients and students in Ile- Ife, Nigeria. *W. Afr. J. Med.*, 303:177-182.
- Reid G, Nicolle LE (1999). Asymptomatic bacteriuria in spinal cord patients and the elderly. *Urol. Clin. North Am.*, 26(4):789-795.
- Ribeiro RM, Rossi P, Guidi HG, Pinotti JA (2002). Urinary tract infections in women. *Int. Urogynecol. J. Pelvic Floor Dysfunct.*, 13:198-203.
- Roberts RO, Lieber MM, Rhodes T, Girman CJ, Bostwick DG, Jacobsen SJ (1998). Prevalence of a physician-assigned diagnosis of prostatitis: The Olmsted county study of urinary symptoms and health status among men. *Urology*, 51:578-584.
- Schaeffer AJ (2000). Diagnosis and management of Prostatitis. *Braz. J. Urol.*, 26(2):122-131.
- Smith MN, Easman TD (1990). In Topley and Wilson's Principle of Bacteriology, Virology and immunity. Volume 3, 8th edition, Edward Arnold London. pp. 199-205.
- Stamey TA, Yang N, Hay AR, McNeal JE, Friehe FS, Redwine E (1987). Prostate specific antigen as a marker for adenocarcinoma of the Prostate. *N. Engl J. Med.*, 317: 909-916.
- Stamey TA, Pfau A (1970). Urinary infections: A selective review and some observations. *Calif. Med.*, 113:16.
- Stamey TA (1980). Pathogenesis and treatment of Urinary tract infections. Baltimore, Williams and Wilkins.
- Toohey S (1994). Diseases of the urinary system in: Toohey's Medicine Edited by Stephen R. Bloom. Published by Churchill Livingstone/Longman Group UK., pp. 272-276.
- Zackrisson B, Ulleryd P, Aus G, Lilja H, Sandberg T, Hugosson J (2003). Evolution of free, complexed, and total serum prostate-specific antigen and their ratios during 1 year of follow-up of men with febrile urinary tract infection. *Urology*, 62(6):287; 281.