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The incidence of asymptomatic urinary tract infections amongst female university students in Owerri-South East Nigeria

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ABSTRACT

Investigation into the incidence of asymptomatic urinary tract infections was carried out using a total of 100 samples (50 early morning midstream urine and 50 high vaginal swab samples, respectively), from undergraduate female students of Imo State University and Federal University of Technology, Owerri. Isolation, characterization and identification of the microorganisms were done according to standard methods and reference manuals. A total of 48 organisms (48.0%) were obtained, which include: Escherichia coli (25.0%) Enterobacter aerogenes (12.5%), Pseudomonas aeruginosa (14.6%), Klebsiella pneumoniae (18.8%), Proteus mirabilis (4.1%), Staphylococcus aureus (20.8%) and Candida albicans (4.2%). The isolates showed variations in susceptibility to antibiotics used. This study has shown that asymptomatic bacteriuria is present in this seemingly healthy population of female students of the two Universities. There is need, therefore for routine checks for these infections and their earlier treatment, in order to avoid complications.

Keywords: Incidence, asymptomatic, females, bacteriuria, treatment and complications.

INTRODUCTION

The urinary tract is made up of the kidney, ureter, bladder and urethra. Urinary tract infections may be defined as all those conditions characterized by significant amount of pathogenic or opportunistic agents in the urinary tract [1].

Asymptomatic urinary tract infections (covert infections) are common in apparently healthy populations[2,3] and are detectable in the laboratory as 'significant bacteriuria'. When a person has no symptoms of infection but significant numbers of bacteria have colonized the urinary tract, the condition is called asymptomatic urinary tract infection(UTI), also called asymptomatic bacteriuria[4]. The condition is harmless in most people and rarely persists, although it does increase the risk for developing symptomatic UTIs. Asymptomatic Urinary Tract Infection (UTI) is defined as the presence in the urine of more than 10⁵ colony forming units of organisms per ml of urine in the absence of symptoms referable to the tract [5, 6]. It has been suggested by [6], that these sub-clinical conditions may represent an early stage of the natural history of UTI. It may lead to acute infections, chronic infections and eventual death from kidney failure. In other instances; the bacteriuria may disappear spontaneously in pregnancy. It is well established that bacteria even when asymptomatic predisposes to the development of acute pyelonephritis[7]. In addition, there is a suspicion that it increases the risk of fetal death as well.

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When an infection is confined to a particular site such as the prostrate, it is known as prostatitis. When it is the kidney, it is pyelonephritis, the urethra and bladder are the most common sites of infection in the urinary tract. Infection may be limited to one or the other but frequently co-exist.

Females far exceed males in the prevalence of asymptomatic bacteriuria because of the relative proximity of the anus to the vaginal introitus, the latter is susceptible to colonization with enteric bacteria [4, 8]. Factors reported to facilitate introital colonization include a relative high pH of vaginal secretions (more than 4.4), the presence of vaginal epithelial cell receptors that might aid bacterial adherence and decrease production of specific cervico-vaginal antibody [4, 8].

If four percent (4%) of females have symptomless bacteriuria, they are extremely unlikely to develop renal damage. But about one third(1/3) will have episodes of symptomatic bacterial cystitis, acute infections, infertility, chronic infection, hypertension, heart attack, premature birth, still birth, toxemia, and eventual death from kidney failure[6, 9]. This report is aimed at establishing the prevalence of asymptomatic UTI amongst female undergraduate students in Owerri, South East, Nigeria.

MATERIALS AND METHODS

Fresh early morning midstream urine and high vaginal swab (HVS) samples were collected from 100 female undergraduate students of the Imo State University and Federal University of Technology, Owerri within the age bracket of 16-30 years. Students showing clinical symptoms of urinary tract infections and those on antibiotic therapy were excluded from the sample collection. The method used was that suggested by [10] and [11], in which the urethra was flushed with the passage of about 100 ml of urine before the specimen was collected in sterile universal specimen bottles. These bottles were given to the female students and they were instructed to wash the urethral meatus with soap and rinse thoroughly with copious amount of water. Swab sticks were used for the HVS samples, where the students were told to expand the vagina, insert the swab stick and swirl it round and cover immediately to avoid contamination. The HVS and urine samples were taken to the laboratory for microbial analysis. Microscopy of the urine samples were carried out to detect the presence of bacteria, white cells, red cells, yeast cells and epithelial cells. Also, cultural technique was employed using growth media. The urine was uncentrifuged, however, before culturing. Using a standard platinum wire loop, a loop full of the urine was aseptically collected and uniformly spread on the dry surface of MacConkey agar, chocolate agar, nutrient agar and blood agar and the plates were incubated at 37°C for 24 hours aerobically. Also the HVS samples were cultured on chocolate agar, MacConkey agar and SDA and incubated at 37°c for 24 hours. Biochemical tests were also carried out for identification of the isolates. Also sensitivity tests were done to show the susceptibility of these isolates using Kirby-Bauer disk diffusion method [4].

RESULTS

A significant growth of microorganisms, 10⁵ cfu/ml of sample was recorded in 48 (48.0%) out of the 100 samples of midstream urine and high vaginal swab investigated. Such students were considered to have asymptomatic bacteriuria. The relative frequency of occurrence of the isolates in students with significant bacteriuria shows that of the 48 isolates, 10 were gram positive cocci,36 gram negative rods and 2 *Candida* species; giving prevalence rates of 20.8% ,75.0%, and 4.2% respectively. The gram positive cocci were identified to be *Staphylococcus aureus*. *Escherichia coli* occurred at 25.0%, *Enterobacter aerogenes* 12.5%, *Klebsiella pneumoniae* 18.8%, *Proteus mirabilis* 4.1%, *Pseudomonas aeruginosa* 14.6 % and *Candida albicans* 4.2% (Tables 1.0 and 2.0)

ORGANISM	NUMBER (%) OFFEMALES POSITIVE
Gram Negative Rods	36 (75.0 %)
Escherichia coli	12 (25.0%)
Pseudomonas aeruginosa	7 (14.6%)
Proteus mirabilis	2(4.1%)
Klebsiella	9(18.8%)
pneumoniae	
Enterobacter aerogenes	6(12.5%)
Gram positive cocci	10 (20.8%)
Staphylococcus aureus	10(20.8%)
Candida albicans	2(4.2%)
TOTAL	48 (100.00%)

TABLE 1.0 Relative frequency of the species of the isolates in students with asymptomatic urinary tract infections

The results of the antimicrobial sensitivity testing are shown in Table 3.

DISCUSSION

The observation that 48 out of 100 females are infected by urinary tract pathogens, out of which 38 were of the gram negative rods (75.0%), 10, gram positive cocci (20.8%) and 2, *Candida albicans* (4.2%) is in line with the report from [4 12 and 13], that gram negative bacilli of the enterobacteriacae family are responsible for almost 90% of all cases of UTI. It has been suggested that the higher incidence of gram negative rod may be accounted for by the fact that members of the entrobacteriaceae from a greater proportion of the gastro-intestinal tract (GIT).

Also, among the bacterial species isolated in this work, the most encountered Escherichia coli, isolated from 12 females (25.0%), agree with the work of [5,14 and 15] that *E.coli* is the most implicated causative agent of UTI. This is also due to the shortness of the urethra that is in close proximity to the peri-rectal area [5].E.coli is generally a harmless microorganism originating in the intestines. If it spreads to the vaginal opening, it may invade and colonize the bladder, causing an infection. The spread of E.coli to the vaginal opening most commonly occurs when women or girls wipe themselves from back to front after urinating; or after sexual activity [4]. Enterobacter aerogenes was isolated from 6 females giving a relative frequency of 12.5%. This is in agreement with reports by [15] that predominantly E.coli, Proteus spp and less commonly Klebsiella spp, Enterobacter spp, and Pseudomonas spp are usually responsible for infections originating in the urinary tract. The spectrum of organisms causing UTI varies. Escherichia coli accounts for 75% to 90% of isolates, Staphylococcus saprophyticus for 5% to 15% and Klebsiella, Proteus, Enterococcus species and other organisms account for 5% to 10% of isolates [9]. Candida albicans was isolated from 2 females and maybe accounted for by the nature of the urinary tract opening and its location in relation to the position of the anal orifice. Candida species are part of the normal intestinal flora in humans, the possibility of trans-infection into the urinary tract from the anus is quite high. The susceptibility of isolates to most antibiotics used for the treatment of UTI, for example trimethoprimsulfamethoxazole against urinary isolates of *E.coli* can vary considerably by geographic location [16,17]. Drugs of choice for UTI treatment are Cephalexin, Erythromycin, Nitrofurantoin, Augmentin, and Nalidixic acid[18,19].Nitrofurantoin has 90% efficacy and is a good choice because of its high urinary concentrations; as well as trimethoprim-sulfamethoxazole[20].

Enterobacteriacea isolated from this study were most sensitive to Gentamicin, Ciproxin, Septrin(trimethroprimsulfamethoxazole), and Tarivid. However, *Staphylococcus aureus* was most sensitive to Septrin, Ciproflox, Erythromycin and Rimfampin.

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Colony No.	Nature of colony formation on plate	Gram stain and microscopy	Motility	H ₂ S	Catalase	oxidase	coagulase	Indole	MR	VP	Citrate	Urease	Glucose	Lactose	Sucrose	Fructose	xylose	Suspected or ganism
А	Cream coloured colonies on NA, pink on MA, little shiny smooth not easily emulsified	Gram negative short rods to cocco bacilli	+	-	+	-	-	+	+	-	-	-	AG	AG	+	+	AG	Escherichia coli
В	Green coloured colonies on NA and pink on MA	Gram negative shorts rods	+	-	+	+	-	-	-	+	+	+-	0	AG	0	0	0	Enterobacter aerogenes
С	Raised mucoid colonies and cream coloured on NA	Gram negative rods	+	-	+	-	-	-	+	+	+	+	AG	+	AG	AG	AG	Klebsiella pneumoniae
D	Mucoid raised and Irregular colonies, greenish blue pigment on NA	Gram negative slemder rods	+	-	+	+	-	-	-	-	+	+	A	-	-	-	-	Pseudomonas aeruginosa
E	Cream coloured and swarming growth on NA, MA. Colonies are yellow, swarming and flat	Gram negative rods	0	+	+	-	0	+	+	-	+	+	AG	-	0	0	0	Proteus mirabilis
F	Colonies appear yellow in colour, circular with entire edges and raised on edges and raised on NA. On MA the colories are round smooth and shiny.	Gram positive cocci clusters	-	0	+	0	+	0	0	+	0	-	+(A)	+(A)	0	0	0	Staph aureus

TABLE 2.0: CULTURAL AND BIOCHEMICAL CHARACTERIZATION OF THE ISOLATES

KEY: + Positive, - Negative, NA Nutrient Agar, MA MacConkey's Agar, AG Acid and gas production, A Acid production, 0 Not determined, +(-) Some strains can be positive and others negative.

Anti- microbial Agent	disc potency (mcg)	Diameter of zone of inhibition (mm)									
		Staphyloclccus	Proteus	Escherichia	Klebsiella	Psuedomonas	Enterobacter				
		aureus	species	coli	pneumoniae	aeruginosa	aerogenes				
CN	10	9	20	30	22	15	28				
AU	30	-	-	-	-	-	-				
CPX ₁	10	-	30	24	28	30	24				
SXT	30	26	15	16	-	5	15				
CEP	10	-	11	-	7	10	12				
S	30	-	14	7	15	22	10				
PN	30	12	15	15	10	-	20				
OFX	10	-	20	27	21	28	18				
PEF	10	-	24	17	28	-	17				
CH	20	22	19	21	20	26	-				
Е	30	29	-	-	6	-	25				
CPX ₂	10	24	9	-	8	18	26				
APX	30	11	-	-	-	-	-				
RD	10	25	-	-	-	4	-				
TG	50		-	-	-	-	-				
NB	30	21	-	-	-	-	-				

TABLE 3.0 ANTI-MICROBIAL SUSCEPTIBILITY PATTERN OF ISOLATES

KEY: CN Gentamycin, E Erythromycin, AUAugumentin, CPX2 Ciproflox, CPX1 Ciproxin, CH Choronphenicol, SXT Septrin, APX Ampiclox, CEP Ceporex, RD Rimfampin, S Streptomycin, TG Togamycin, PIN Ampecillin, NB Norbactin, OFX Tarivid, PEF Peflacine, - Resistant.

This report has shown that asymptomatic bacteriuria is present in the seemingly healthy population of students at the Imo State University and Federal University of Technology, Owerri especially females in the age ranges of 16-30 years. This may be attributed to their social behavior with regards to drug (antibiotics) abuse, toilet habits, physiological conditions and sexual behaviors. It is hoped that this study would provide the necessary data to aid the planners of our health care delivery systems in the management of UTI (both symptomatic and asymptomatic), as well as create awareness in our young females about the possible complications that may arise if not properly handled.

REFERENCES

[1] WE Stamm, and TM Hooton, Engl.J.Med 1993, 329, 1328-1334.

[2] R Cruickshank, JP Dugnid, BP Marmion and RHA, Swain, 1992. Medical Microbiology. 12th edition, vol 2. Longman group London.pp 86-104.

[3] B Eugene, F Anthony, LK Fennis, LH Stephen, Dan, LL Stephen, and J J Larry, 2001. Principles of Internal Medicine 15th Edition, Mc Graw Hill Medical Publisher U.S.A.Pp 1620-1626.

[4] N Shree, JS Sharma, M Kumar, and Y Sharma, International Journal of Microbiology Research,2014, 6(1),545-552.

[5] JB Ellen, RP Laurice, and MF Sydney, 1994, Diagnostic Microbiology. 9th Edition, Mosby Company, St Louis, U.S.A.Pp 249-255.

[6] R Colgan, LE Nicolle, A McGlore, TM Hooton, Am. Fam Physician, 2006, 74(6), 985-90.

[7] SR Norrby, 2007, Approach to the patient with Urinary tract Infection. In. Goldman L, Ausiello, D., eds. *Cecil Medicine* 23rd ed. Pa: saunders Elsevier Chap.306.

[8] M Sussman, AW Asscher, WE Waters, JAS Evans, H Campbell, KT Evans and JE Williams, British MedicalJournal 1969, 1, 799-803.

[9] TM Hooton, D Schooles, AE Stapleton, N.Engl.J.Med., 2000, 343,992.

[10] EH Kass, Trans Assoc. Am. Phy. ,1965, 9,56-64.

[11] AR Roland, 1989. Urethritis and Cystitis. In Infectious Diseases. A modern Treatise of Infectious Processes (Hoeprich, P.D. and Jordan, M.C. Eds. J.P. Lippincott Co.) Philadelphia U.S.A. pp 538-46.

[12] P Boutros, H Mourtada, and AR Roland, 1972. Urinary Infection Localization Am. J. Obstet. Gynecol 112: 379-381.Campbell's Urology, 5th edition. Walsh, P.C. Gittes, R.F. Perlmutter, A.D. Philadelphia U.S.A.pp 868-887.

[13] IEK Mba, ,C Aluka, and ANR Amadi, Journal of Hygiene and Sanitation.2002, 2:27-29.

[14] EM Meares Jr, **1986**. Prostatitis and Related Disorders. *In* Medical Microbiology. 12th ed.vol 2.The Practice of Medical Microbiology. Longman Group London.

[15] B Foxman, American Journal of Medicine, 2002, 1, 5-13.

[16]JA Karlowsky,RSL Philippe,PJ Simner,RD Meldin,JA Heathe,W Andre,JH Daryl, and GZ George,*Antimicrob.Agents Chemother*,**2011**,55(7),3169-3175.

[17] S Smita, and G Ravi, Indian J. Community Med., 2012, 37(1), 39-44.

[18] E Gratacos, PJ Torres, J Villa, PL Alonso, and V Cararach, J. Infect. Dis., 1994, 169, 1390-1392.

[19] M Akram, M Shahid, and AU Khan, Annals of Clinical Microbiology and Antimicrobials, 2007, 6, 4.

[20] MP Mishra, NK Debata, and RN Padhy, Asian Pac.J.Trop.Biomed., 2013, 3(4), 315-324.