SIGNIFICANT ASYMPTOMATIC BACTERIURIA AMONG NIGERIAN TYPE 2 DIABETICS

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Significant asymptomatic bacteriuria is a risk factor for symptomatic urinary infection and septicemia among predisposed individuals such as diabetics. We investigated the pattern of asymptomatic bacteriuria among our type 2 diabetics with a view to documenting the prevalence, type of organisms responsible and the antibiotic susceptibility pattern.

One hundred and twenty-four type 2 Nigerian diabetics (55 males and 69 females) submitted midstream urine specimens for culture. Thirty-three patients had significant bacteriuria (9 males and 24 females), showing the frequency of occurrence of asymptomatic bacteriuria to be 26.6%. The most common organism isolated was Klebsiella pneumonia at 42.4%. Gram-negative bacilli made up about 23 (69.7%)of the isolates. Isolates were poorly sensitive to the readily available antibiotics (ampicillin, tetracycline and cotrimoxazole), but a large number of the organisms isolated were sensitive to nitrofurantoin, gentamicin, ciprofloxacin and ofloxacin. Sensitivity to erythromycin, nalidixic acid and cefuroxime was moderate.

Asymptomatic bacteriuria is, thus, more prevalent among the Nigerian diabetic population than in the non-diabetics. A changing pattern of disease is observed with Klebsiella sp. now accounting for the majority of asymptomatic bacteriuria among diabetics. The organisms are not sensitive to the commonly available antibacterial agents. (*J Natl Med Assoc.* 2003;95:344-351.)

Key words: asymptomatic bacteriuria ♦ diabetes mellitus ♦ Nigeria

Urinary Tract Infection (UTI) is common in women, elderly men, and children.[1] Significant bacteriuria is taken as 10^5 organisms per millimeter of a fresh "clean catch" urine specimen in any patient. It indicates active infection of the urinary tract.[2]

Kass[3] was the first to highlight the importance of asymptomatic bacteriuria (AB)—he observed that 6 to 7% of pregnant women have AB and almost half of these develop pyelonephritis subsequently. AB also is of importance in the elderly and individuals who are known to be at high risk for recurrent symptomatic infection including diabetics, patients with cystic renal diseases, anatomic or neurologic urinary abnormalities and patients with a history of urethral catheterization.[4] Untreated AB predisposes the individual to recurrent UTI, which cause considerable morbidity if complicated and can also cause severe renal disease.

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Morbidity and mortality from UTI at all ages is still considerable.[5] Patients with diabetes mellitus have an increased risk of infections,[6,7] with UTI being the most prevalent infection.[8,9] Serious complications of urinary infection, such as emphysematous cystitis, pyelonephritis, renal or perinephric abscess, bacteremia, and renal papillary necrosis occur more commonly in diabetic patients.[10]

It has been postulated[11] that both altered bacterial adhesion to the uroepithelium, due to abnormality of Tammhorsfal protein, and granulocyte dysfunction contribute to the pathogenesis of UTI in diabetics. Several polymorphonuclear defects, such as impaired migration, phagocytosis, intracellular killing, and chemotaxis, (which may be due to decreased polymorphonuclear membrane f luidity)[12,13] occur in diabetic subjects. The pattern of AB has been studied in various populations.[14-21] Although more controlled studies have examined the prevalence of bacteriuria in diabetic compared with nondiabetic subjects than any other infection,[14,22-32] few workers have carried out similar work among diabetics in Nigeria.[24,40] Of the highlighted studies, 75% reported a higher (two- to four-fold increase) bacteriuria prevalence in diabetic subjects.

This study aims to investigate the prevalence of AB among Nigerian diabetic patients; document the susceptibility to AB among Nigerian diabetics, and to document the organisms' responsible and antimicrobial sensitivity pattern of such organisms in this environment.

METHODOLOGY

The study had the approval of the Ethics Committee of the Olabisi Onabanjo University Teaching Hospital, Sagamu. All patients gave their informed consents. The study group consists of consecutive known diabetic patients with AB presenting at the Medical Out-Patient Department of the Hospital.

Following the 1985 WHO criteria,[33] diabetes was defined as a fasting glucose concentration of 7.8mmol/l, a two-hour glucose concentration of 11.1mmol/l, or the use of glucose lowering medications (oral agents or insulin). We defined AB as the presence of at least 10^5 colony forming units/ml of 1or 2 bacterial species in a culture of clean-voided urine from an individual without symptoms of a UTI.[34] We defined contaminated urine as the presence of at least three different micro-organisms in one urine specimen.[35] The inclusion criteria are a prior diagnosis of diabetes mellitus without recent symptoms of UTI (dysuria, frequency, fever, etc). These patients should also have received no therapy with antibiotics, non-steroidal anti-inflammatory drugs, and immunosuppressives in the preceding two weeks. The exclusion criteria include pregnancy, presence of cystic renal disease, recent hospitalization or surgery (within the past four months), recent instrumentation (within the past two months), known anatomic and neurologic urinary tract abnormalities, the presence of urinary symptoms (the presence of dysuria, frequency or urgency, strangury, abdominal discomfort, or fever) or recent use of antimicrobial drugs (within the previous two weeks).

Clinical parameters including the age, gender, duration of diabetes mellitus, drug therapy, clinical symptomatology—especially urinary complaints, last menstrual period (for female patients) were recorded. Fasting blood sugar and two-hour postprandial blood sugar (on two occasions) were estimated.

Mid-stream urinary specimens were collected for urinalysis, microscopy, culture, sensitivity and gram staining. All patients were asked to start with a full bladder. Female patients separated the labia using the left hand and cleansed the vulva from the front to the back with sterile swabs before passing urine. The sample was collected by plunging the sterile container into the urinary stream, without stopping the urine flow, with the right hand.

Male patients were required to clean the penis with a sterile swab, void half way and plunge the sterile container into the stream. The specimens were refrigerated (immediately after

Table 1. AGE AND SEX DISTRIBUTION OF DIABETICS WITH ASYMPTOMATIC BACTERIURIA

AGE (YRS)	MALES%	FEMALES%	TOTAL NOS%
30-39	1(11.1)	1(4.2)	2(6.0)
40-49	1(11.1)	9(37.5)	10(30.3)
50-59	1(11.1)	5(20.8)	6(18.2)
60-69	4(44.4)	6(25)	10(30.3)
>70	2(22.2)	3(12.5)	5(15.2)
Total	9(27.3)	24(72.7)	33(100)

collection) and cultured within two hours.

All urine samples were cultured on Blood and MacConkey agar plates using standard platinum wire loop. The plates were incubated at 37° C aerobically for 24 hours. Cultures with colony counts ≥ 10^5/ml were considered as significant bacteriuria.[34,37] The organisms were identified using standard bacteriological techniques.[36] Antimicrobial sensitivity was determined by the agar diffusion method using multodiscs (Abtek Biologicals Ltd., Liverpool, UK). Escherichia coli NCTC 6571 and staphylococcus aureus NCTC were used as controls. Urinalysis was performed using a dipstick (Medi-Test Combi 9 Macherey, Nagel, Germany).

A well-structured questionnaire was administered to each patient to obtain his or her demographic data, biodata, diabetic history, associated medical/surgical conditions and detailed urinary symptoms.

RESULTS

One hundred and twenty-four type 2 diabetics formed the study group (55 males and 69 females). Thirty-three patients (26.6%) had significant bacteriuria (9 males and 24 females). Their ages ranged from 36 to 70 years with a mean of 56±6.4. Table 1 shows the age and sex distribution of diabetics with AB. The peak incidence is in the forth and the sixth decade of life. The mean fasting blood glucose and two-hour postprandial blood glucose levels in the population studied were 5.5±1.3mmol/l (range 4.4-6.1mmol/l) and 7.8±0.1mmol/l (range 7.3-8.7mmol/l), respectively.

Table 2 shows the microorganisms isolated from the urine specimens of the study population. Gram-negative bacilli were the predominant organisms isolated-23 (69.7%). They

Table 2. THE MICROORGANISMS ISOLATED FROM THE URINE OF DIABETIC PATIENTS WITH ASYMPTOMATIC BACTERIURIA

MICROORGANISMS	NUMBER OF PATIENTS (%)
Klebsiella pneumonia	14(42.4)
E. coli	7(21.2)
E. faecalis	4(12.1)
S. pyogenes	3(9.1)
Coagulase negative Staphlyloccocus	3(9.1)
Atypical coliforms	2(6.1)
Total	33(100)

included Klebsiella pneumonia 14 (42.4%), E. coli 7 (21.2%), and 2 (6.1%) atypical coliforms that could not be identified further. Gram-positive cocci isolated were E. faecalis, S. pyogenes, and Coagulase negative staphylococcus epidermidis and they account for 10 (30.3%).

Table 3 shows the sensitivity pattern of each organism. A large number of the organisms isolated in this study were sensitive to Nitrofurantoin, gentamicin, ciprofloxacin and ofloxacin. Very few isolates were sensitive to ampicillin, septrin and tetracycline. The two coliforms that were not identified were resistant to all antibiotics tested.

In the nondiabetic control group (124 patients, 55 males and 69 females), 7 (5.6%) of the patients had significant bacteriuria. E.coli was the causative microorganism in 5 (71.4%) of these patients. The remaining 2 (28.6%) had E. faecalis. The prevalence of AB was significantly higher among the diabetics than in the nondiabetic control group ($p<0.0001$). There is a positive significant association between the duration of disease (diabetes mellitus, $p<0.01$) and the age of the patients ($p<0.001$) and the presence of significant bacteriuria. The degree of diabetic control did not influence the risk of development of significant bacteriuria, $p=0.05$.

DISCUSSION

In this study, 33 out of 124 type-2 diabetics (26.6%) had AB. This is comparable to previous reports from Africa[27] (22.5%), Netherlands[35] (26%), and Turkey[43] (25.5%); although these studies were carried out on a larger population of diabetics. In some parts of Nigeria, the prevalence of AB in two different studies was 6.3%[24] and 20%.[40] Other centers worldwide have reported much lower values varying from 5.8%,[23] 7.9%,[18] 10%,[14] 11.1,[4] 17%,[29] to 19%.[31] The lower figures recorded in these studies could be explained in that the studies based the diagnosis of AB on two urine cultures, as against single urine culture in the present study; the later may increase the observed prevalence of AB.

Many workers have demonstrated the presence of AB in different groups such as school age girls,[20,37,39] sexually active young women,[19,38,39] pregnant women,[3,21] institutionalized elderly[39] and catheterized patients.[39] This shows that AB is quite common. In this study, AB was more common among diabetic women. This is comparable to previous reports in many countries including Italy, Scandinavia, Netherlands, South Africa, etc.[7,10,15,17,18,23,30,31,32,35] In this study, most of the diabetic women with AB were between 40 and 59 years of age, while most of the diabetic men were above 60 years. Only two patients in the study population below 40 years had AB. This supports other studies where UTI was found more commonly in the elderly.[38,39]

K. pneumonia was the most common isolate from the urine samples. This is in contrast to other studies where E. coli was the most common organism isolated.[24,40] This suggests a changing pattern in organisms causing infection in the population. At the University College Hospital in Ibadan, Nigeria, Klebsiella species were reported to occur more commonly than E. coli as the causative agent of bacteraemia amongst diabetics.[41] Klebsiella species also was identified as the predominant organism causing UTI in the general population in the same environment.[42] This observation is, however, not surprising since Klebsiella species were known to be an important cause of both nosocomial and community acquired UTI.[42]

In this study, the presence of AB was related to the duration of diabetes and the age of the patient, but not related to the degree of control of diabetes. This is comparable to previous reports worldwide. A statistically significant longer diabetes duration has been documented for diabetic subjects with bacteriuria than without (9.9vs5.4); while bacteriuria prevalence increased 1.9-fold with each 10-year increase in diabetes duration.[14,23,35]

There was a high prevalence of multidrug resistant organisms in this study. A large number of the isolates were resistant to ampicillin, cotrimoxazole and tetracycline, which are very common and often purchased without prescription. The isolates were, however, susceptible to quinolones, gentamicin and nitrofurantoin.

Eleven (78.6%) of the Klebsiella isolates were sensitive to nitrofurantoin, thus, indicating that the drug is still a useful urinary antiseptic in our environment. This finding is comparable to reports on antimicrobial sensitivity pattern of isolates from various centers in the country.[24,40-42] Ciprofloxacin is quite expensive and, therefore, not likely to be purchased without prescription. Gentamicin is cheap and effective but requires parenteral administration and, therefore, will not be suitable for treating outpatients.

There are conflicting views as to the rationale of screening diabetics routinely for significant bacteriuria. In the mid-1950s, at a time when urinary infection was considered to be an important contributor to chronic renal failure, hypertension, toxaemia of pregnancy, routine screening for bacteriuria was advocated in diverse groups of patients.[3,22] Thereafter, it was observed that schoolgirls and women with AB were no more likely to have progression to renal failure than those without.[1,10,16,20,38] Interest in AB later shifted to pregnant women, men or women undergoing invasive genitourinary procedures, renal transplant recipients, post transplantation period, and diabetics.[14,15,17-19,23-32] Studies in all these groups have consistently confirmed that patients with AB also are at increased risk for symptomatic urinary infection. This suggests that the diabetic patients with AB in this study also are at risk. It is, therefore, necessary to study the pattern and the prevalence of symptomatic bacteriuria among type 2 diabetic patients in Sagamu, Nigeria.

In conclusion, AB is more prevalent among female type 2 diabetics in Sagamu, Nigeria, than in non-diabetics. Diabetologists should emphasize the importance of urine microscopy and culture in the clinical care protocols of patients. It appears that there is a changing pattern in the organisms causing infection among diabetics. This may be a result of antibiotic abuse, which is prevalent in the population. This is reflected in various hospitals nationwide. There is a need for continuous surveillance of organisms causing infections in the hospital and the community.

REFERENCES

1. Andriole VT. Urinary tract infections and pyelonephritis—In Cecil Textbook of Medicine 17th Edition. Wyngaarden JB and Smith LH (eds). W.B. Saunders, 1985; 619–623.

2. Stamm WE, Turck M. Urinary tract infection,pyelonephritis, and related conditions. In Harrisons Principles of Internal Medicine, 11th Edition. Braunwald E, Isselbacher KJ, Petersdorf RG, Wilson JD, Martin JB, Fauci AS (eds). McGraw-Hill Book Co.; 1987; vol 2:1189-1195.

3. Kass EA. Bacteriuria and pyelonephritis of pregnancy. *Arch Intern Med.* 1960; 105:194-198.

4. Kayima J.K; Otieno L.S, Twahir A, Njenga E. Asymptomatic bacteriuria among diabetics attending Kenyatta National Hospital. *East African Medical Journal.* 1996; 73: 524-526.

5. Glauser MP, Kuylens R, Mollby R, et al. Infections. Current

Table 3. URINE ISOLATES FROM DIABETIC PATIENTS AND THEIR ANTIBIOTIC SENSITIVITY PATTERN

ORGANISM	NOS OF ISOLATES	NUMBER OF ISOLATES SENSITIVE TO ANTIBIOTICS																	
		A	COT	NIT	G	TET	CAR	NAL	TOB	CIP	O	CXM	CAZ	CHL	ERY	CEP	NOV	STR	CLO
K. pneumonia	14	0	4	11	12	3	0	7	7	12	10	3	4	0	NT	0	0	0	0
E. coli	7	0	1	7	6	3	0	4	2	5	7	2	0	0	NT	0	0	0	0
E. faecalis	4	2	2	1	0	1	1	0	0	2	4	3	0	1	2	0	0	0	0
S. pyogenes	3	1	0	0	2	0	1	1	0	1	2	0	0	1	1	1	0	0	0
C-ve Staph.	3	0	0	0	1	0	0	0	0	2	1	1	0	1	2	0	1	1	1
A. coliforms	2	0	0	0	0	0	0	0	0	0	0	0	0	0	NT	0	0	0	0

C–ve Staph: Coagulase negative Staphyloccocus bacilli; A. coliforms: Atypical coliforms; NT:NOT TESTED; A-Ampicillin; COT- Cotrimoxazole; NIT-Nitrofurantoin; G-Gentamicin; TET-Tetracycline; CAR-Carbenicillin; NAL-Nalidixic acid; TOB-Tobramycin; CIP-Ciprofloxacin; O-Ofloxacin; CXM-Cefuroxime; CAZ-Ceftazidine; CHL-Chloramphenicol; ERY-Erythromycin; CEP-Cephalexin' NOV-Novobiocin STR-Stretomycin; CLO-Cloxacillin. (ABTEK BIOLOGICALS LIMITED)

concepts in the management of urinary tract infections workshop. Proc 1Xth Int. Congress Nephr. 1984; 1441–44.

6. Pozilli P, Leslie RDG. Infections and diabetes: mechanisms and prospect for prevention. *Diabet Med.* 1994; 11: 935-941.

7. Carton JA, Maradona JA, Nuno FJ, Fernandez-Alvarez R, Perez-Gonzalez, Asensi V. Diabetes mellitus and bacteremia: a comparative study between diabetic and nondiabetic patients. *Eur J Med.* 1992; 1: 281-287.

8. Mac Farlane IA, Brown RM, Smyth RW, Burdon DW, FitzGerald MG. Bacteremia in diabetics. *J Infect.* 1986 12: 213-219.

9. Wheat LJ; Infection and diabetes mellitus. *Diabetes Care.* 1980;3:187-197.

10. Editorial. Asymptomatic bacteriuria in diabetic women. *Diabetic Care.* 2000:23;722-723.

11. Geerlings SE, Brouser EC, van Kessel CPM, et al. Cytokine secretion is impaired in women with diabetes mellitus (abstract). In proceedings from the 39th Interscience Conference of Antimicrobial Agents and Chemotherapy, San Francisco, CA; 1999;p.388.

12. Valerius NH, Eff C, Hansen NE, et al. Neutrophil and lymphocyte function in patients with diabetes mellitus. *Acta Med Scand.* 1982;211:463-67.

13. Masuda M, Markami T, Egawa H, Murata K. Decreased fluidity of polymorphonuclear leukocyte membrane in streptozocin-induced diabetic rats. *Diabetes.* 1990;39:466-70.

14. Shmitt JK, Fawcett CJ, Gullickson G. Asymptomatic bacteriuria and haemoglobin A1. *Diabetes Care.* 1986;9:518–20.

15. Kelestimur F, Unal A, Pasaoglu H, et al. Asymptomatic bacteriuria in patients with diabetes mellitus. *Mikrobiyoloji Bulteni.* 1990;24: 126–32.

16. Perez–Luque EL, dela Luz Villalpando M, Malacara JM. Association of sexual activity and bacteriuria in women with non-insulin dependent diabetes mellitus. *J of Diabetes.* 1992:6;254–7.

17. Zhanel GG, Harding GK, Nicolle LE. Asymptomatic bacteriuria in patients with diabetes mellitus. *Review of Infectious Diseases.* 1991;13; 150-4.

18. Zhanel GG, Nicolle LE, Harding GK. Prevalence of asymptomatic bacteriuria and associated host factors in women with diabetes mellitus. The Manitoba Diabetic Urinary Infection Study Group. *Clinical Infectious Diseases.* 1995;21: 316–22.

19. Hermida Perez JA, Vento Remedios TE, Perez Fernandez L, et al. [Asymptomatic bacteriuria or "detected" bacteriuria in the female. Incidence in our health area]. [Spanish]. *Archivos Espanoles de Urologia.* 1998; 51: 145–9.

20. Okafor HU, Okoro BA, Ibe BC, Njoku–Obi NU. Prevalence of asymptomatic bacteriuria among nursery school children. *Nigerian Journal of Paediatrics.* 1993;20: 84–88.

21. Olusanya O, Ogunledun A, Fakoya TA. Asymptomatic significant bacteriuria among pregnant and non–pregnant women in Sagamu, Nigeria. *Cent–Africa J. Medicine.* 1992:38;297-302.

22. Kass EH: Asymptomatic infections of the urinary tract. *Trans Assoc Am Phys.* 1956;69:56-64.

23. Keane EM, Boyko EJ, Reller LB, Hamman RF. Prevalence of asymptomatic bacteriuria in subjects with NIDDM in San Luis Valley of Colorado. *Diabetes Care.* 1998:11;708-12.

24. Abu-Bakare A, Oyaide SM. Asymptomatic bacteriuria in Nigerian diabetes. *J Trop Med Hyg.* 1986;89:29-32.

25. Bahl AL, Chugh RN, Sharma KB. Asymptomatic bacteriuria in diabetics attending a diabetic clinic. *Indian J Med Sci.* 1970;24:1-6.

26. Hansen RO: Bacteriuria in diabetic and non-diabetic outpatients. *Acta Med Scand.* 1964;176:721-30.

27. Joffe BL, Seftel HC, Distiller LA. Asymptomatic bacteriuria in diabetes mellitus. *S Afr Med J.* 1974;1: 306-308.

28. O'Sullivan DJ, Fitzgerald MG, Meynell MJ. Urinary tract infection: Comparative study in the diabetic and normal populations. *Br Med J.* 1962;1:786-88.

Ooi BS, Chen BTM, Yu M. Prevalence and site of bacteriuria in diabetes mellitus. *Postgrad Med J.* 1974;50:497-99.

30. Jaspan JB, Mangera C, Krut LJ. Bacteriuria in black diabetics. *S Afr Med.* 1977;51:374-76.

31. Vejlsgaard R. Studies on urinary infections in diabetics. I. Bacteriuria in patients with diabetes mellitus and in control subjects. *Acta Med Scand.* 1966;179:172-82.

32. Harding GKM, Zhanel GG, Nicolle LE, Cheang M. (Manitoba Diabetes Urinary Tract Infection Study Group). Antimicrobial Treatment in Diabetic Women with Asymptomatic Bacteriuria. *NEJM.* 2002;347; 1576-1583.

33. Wahl FW, Savage PJ, Psaty BM, et al. Diabetes in older adults: comparison of 1997 American Diabetes Association classification of diabetes mellitus with 1985 WHO classification. *Lancet.* 1998:352: 1012-1015.

34. Recommended terminology of urinary tract infection: a report by the members of the Medical Research Council Bacteriuria Committee. *Br Med. J.* 1979:2; 717-719.

35. Geerlings SE, Edith WMT ter Braak, Manuel C, Castro Cabezas, Timon W van Haeften, Ligtenberg-Oldenburg C, et al. The Diabetes Mellitus Women Asymptomatic Bacteriuria Utrecht (DWABU) Study Group: Asymptomatic bacteriuria may be considered a complication in women with diabetes. *Diabetes Care.* 2000;1-11.

36. Lennette EH, Balows A, Hausler WJ Jr., Truant JO. Manual of Clinical Microbiology (3rd ed). Washington, USA. 1980; American Society for Microbiology Publications.

37. Pryles CV, Eliot GR. Pyuria and bacteriuria in infants and Children, *Am J Dis Child.* 1965;110: 628–635.

38. Hooton TM, Scholes D, Stapleton AE, et al. A Prospective Study of Asymptomatic Bacteriuria in Sexually Active Young Women. *NEJM.* 2000;343: 992-997.

39. Lindsay E Nicolle. Asymptomatic bacteriuria-important or not? *NEJM.* 2000;343: 1037-1039.

40. Epoke J, Odigwe CO, Anyanwu GOC, Opara AA. The prevalence of significant bacteriuria in diabetic patients in Calabar, Nigeria. *Diabetes International.* 2000; 10:16-17, 2000.

41. Oni AA,Ogunkunle MO, Oke AA, Bakare RA. Pattern of gram-negative rods bacteremia in diabetic patients in Ibadan, Nigeria. *Afr. J. Med. Sci.* 2000:29; 207-210.

42. Ekweozor Ccand Onyemenem TN. Urinary tract infections in Ibadan: causative organisms and antimicrobial sensitivity patterns. *Afr. J. Med. Sci.* 1996:25;165-169.

43. Kelestimur F, Unal A, Pasaoglu H, et al. [Asymptomatic bacteriuria in patients with diabetes mellitus]. *Turkish. Mikrobiyoloji Bulteni.* 1990:24; 126-32.