Original Research Article

Comparative study of common organism growth in urinary tract infection in type I and type II diabetes mellitus among womens in Salem District

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Abstract

Background: Specifically asymptomatic bacteriuria occurring in diabetes mellitus can cause serious complications like renal and perirenal abscess, gas forming infections such as emphysematous pyelonephritis and cystitis, fungal infections, xanthogranulomatous pyelonephritis, and renal papillary necrosis. Though there is no consensus on the treatment of asymptomatic bacteriuria in various population groups, it is reasonable to treat asymptomatic bacteriuria in diabetes mellitus patients because of its potential complications.

The aim of the study: To compare the common organism growth in type I and type II diabetes mellitus among womens.

Materials and methods: 150 Diabetes mellitus patients without any urinary complaints such as dysuria, frequency, urgency, strangury, tenesmus, nocturia, nocturnal enuresis, incontinence, urethral pain, bladder pain, renal colic, who attended diabetology department as outpatients and in patients in various wards of Govt. Government Mohan Kumuramanglam Medical College, between 2015- 2016 were enrolled for this study.

Results: Of the total 150 patients in study group 44 patients had a positive urine culture in two consecutive urine samples. Most of the patients had blood sugar levels between 151 mg% to 250 mg%. 60% of patients had blood sugar in that range. Another 20% of patients had blood sugar valve between 251 mg% to 300 mg%. In the study group, 25 cases were Escherichia coli isolates. Next

common pathogen was Klebsiella (15 cases). There was no significant correlation in renal profile in type 1 and type 11 diabetes mellitus.

Conclusion: High incidence of asymptomatic bacteriuria has been observed in Diabetic woman. High incidence of asymptomatic bacteriuria occur in both Type I Diabetes Mellitus and Type Diabetes Mellitus. Causative organisms in diabetic and non-diabetic asymptomatic bacteriuria are similar. E.coli is the commonest organism.

Key words

Urinary Tract Infection, Diabetes Mellitus, E.Coli, Klebsiella.

Introduction

Diabetics are more prone to infections than their non-diabetic counterparts. The urinary tract is the most common site of infection in diabetic patients [1]. Most of the urinary tract infections (UTIs) in diabetic patients are relatively asymptomatic, which can lead to severe kidney damage and renal failure [2]. Bacteriuria is more common in diabetics than in non-diabetics due to a combination of host and local risk factors. Disturbances (low complement factor 4, decreased cytokine response) in humoral innate immunity have been described in diabetic patients. However, the clinical relevance of these findings is not clear [3]. Concerning cellular innate immunity, most studies show decreased function in diabetic polymorphonuclear cells and monocytes/macrophages compared to controls [4]. Improved control of the diabetes mellitus (DM) can lead to an improvement in these cellular functions [5]. As well, some microorganisms become more virulent in a high glucose environment. Therefore, screening for UTI in diabetic patients is very important to enable bacteruria to be properly treated, and prevent the development of renal complications of diabetes and eventually severe renal damage and failure [6]. However, controversies exist with respect to incidence, prevalence and microbiological features of UTI between diabetic and non-diabetic patients. Hence the study was planned to compare clinical, microbiological and predisposing features of UTI in diabetics and non-diabetics [7].

Materials and methods

150 diabetes mellitus patients without any urinary complaints such as dysuria, frequency, urgency, strangury, tenesmus, nocturia, nocturnal enuresis, incontinence, urethral pain, bladder pain, renal colic, who attended Diabetology Department as outpatients and in patients in various wards of Govt. Mohan Kumuramanglam Medical College, Between 2015- 2016 were enrolled for this study.

Exclusion criteria: Pregnancy, Recent hospitalization or surgery (within the past 4 months), Known urinary tract abnormalities (including cytopathy or recent urinary tract instrumentation), Symptoms of UTI, The use of antimicrobial drugs during the previous 14 days. WHO criteria was applied to diagnose diabetes mellitus. Then a detailed examination of patients carried out, particularly with regard to complications of diabetes routinely. A detailed gynecological examination carried out to rule out any gynecological problems, cystocele etc. Then all patients were subjected to radiological investigations i.e. plain x-ray KUB and ultrasonogram of the urinary tract to rule out any structural abnormalities, obstruction.

Method of urine specimen collection: Clean-catch midstream urine collection method adopted. Patients were explained about the method of collecting clean-catch midstream urine and elderly female patients were provided with a nursing assistant for cleaning the external genitalia. Urine was collected in a sterile wide-mouthed screw cap bottle for culture purpose and another sample collected for microscopic examination of pyuria. Like these two

consecutive urine specimens were obtained, and refrigerated immediately, because it was not possible to plate all the samples of urine immediately. First, the number of pus cells/mm^3 of urine was counted by using hemocytometer in the microscope.

Results

Patient from all the age groups were enrolled for this study. Our study youngest patient was 9 years old, and the eldest patient was 71 years old. Most of the patients were in 41 to 60 years age group (**Table – 1**).

Table – 1: Age distribution of diabetes patients.

Age in years	Type I DM	Type II DM	Total	
			No. of Patients	Percentage
0-10	1		1	0.66
11-20	3		3	2.00
21-30	14	1	15	10.00
31-40	7	18	25	16.66
41-50	4	34	38	25.33
51-60	1	49	40	26.66
Above 60		28	28	18.66
Total			150	100.00

Table – 2: Random blood sugar values.

Blood Sugar level mg/dl	Type I DM	Type II DM	No. of Patients	Percentage
Up to 120	-	5	5	03.33
121 to 150	2	8	10	06.66
151-200	4	41	45	30.00
201 to 250	11	33	44	29.33
251 to 300	7	24	31	20.66
301 to 350	4	9	13	08.66
>350	2	-	2	01.33
Total			150	100.00

Table – 3: Culture positivity among type - I and type II diabetes mellitus.

Type I DM			Type II DM			Total		
No. of Patients	No. of culture positive	%	No. of Patients	No. of culture positive	%	No. of Patients	No. of culture positive	%
30	8	26.66	120	36	30	150	44	29.33

Table – 4: Break up of organisms grown.

Organisms	Type I DM	Type II DM	No. of Patients	Percentage
E.coli	4	21	25	56.81
Klebsiella	3	12	15	34.09
Pseudomonas		2	2	04.76
Proteus		1	1	02.38
Staphylococcus Aureus	1		1	02.38.
Total	8	36	44	100.00

Most of the patients had blood sugar levels between 151 mg% to 250 mg% (**Table – 2**). 60% of patients had blood sugar in that range. Another 20% of patients had blood sugar valve between 251 mg% to 300 mg%.

Most of the culture-positive patients (55%) were in the age group of 41-60 years (**Table – 3**).

In study group, 25 cases were Escherichia coli isolates. Next common pathogen is Klebsiella (15 cases) as per **Table - 4**.

Discussion

Worldwide the prevalence of diabetes mellitus is increasing day by day. The association of diabetes with an increased propensity of infection has in general, been well recognized. The reason for this increase include incompletely defined abnormalities in cell-mediated immunity and phagocyte function associated with hyperglycemia, as well as diminished vascularization secondary to long standing diabetes [8]. Hyperglycemia facilitates the colonization and growth of a variety of organism including fungal infection. Many common infections like UTI are more frequent and severe in the diabetic population, whereas several rare infections like rhinocerebral mucormycosis, emphysematous infections of the gall bladder and urinary tract and "malignant" or invasive otitis externa are seen almost exclusively in the diabetic population [9]. The spectrum of pathogens causing UTI in patients with a neurogenic bladder differs from that in patients with normal bladder function and is much broader. The majority of UTIs are generally caused by Gram-negative bacilli and enterococci, though sometimes by exogenous bacteria from the hospital environment and often by polymicrobial pathogens. Thus, urine culture and susceptibility testing should be performed before initiating antimicrobial therapy. For the selection of antimicrobial agents, regional differences in antibiotic resistance patterns must be taken into consideration [10]. The optimal duration of antimicrobial therapy ranges generally from 5 days to 14 days depending on severity, with 7 days most commonly used.SGLT2 inhibitor-induced glucosuria likely plays a facilitating role in raising the risk of developing genital infections and, to a lesser extent, UTIs [11]. Upper UTIs (pyelonephritis) is not increased with SGLT2 inhibitor treatment. The characteristics of the infections associated with SGLT2 inhibition are similar to those in any population with diabetes, as these infections respond to standard treatment and recur infrequently. In the case of genital infections, increased infection rates appear to be related to increased urinary glucose concentrations [12]. For long-term management of neurogenic bladder dysfunction, the method used for bladder emptying is the most important issue [13]. A lower rate of UTI and fewer complications are associated with intermittent catheterization as compared with indwelling urethral catheterization, supporting the notion that clean intermittent catheterization (CIC) is the best voiding method for reducing bacteriuria and urinary tract infections in patients with a neurogenic bladder. Thus, CIC should be employed as a standard routine treatment for patients unable to empty their bladder [14]. Some studies have reported beneficial effects of antimicrobial prophylaxis in patients with a neurogenic bladder when given with limited duration and under restricted conditions. However, that therapy is generally not recommended, because its benefits are unproven and it has been associated with the development of antimicrobial resistance. Bowel management aimed at regular emptying of the bowels often relieves constipation and fecal soiling, and also reduces the risk of UTI [15].

Conclusion

Prevalence of UTI was significantly higher in diabetics than non-diabetics where E.coli was the predominant pathogen in both the group of patients. Fluoroquinolones resistance was more common in diabetic than non – diabetics. We would like to recommend restriction of empirical use of fluoroquinolones in diabetic patients. Carbapenem, Amikacin, Piperacillin-tazobactam, and Nitrofurantoin still has acceptable sensitivity against uropathogenic E. coli in both the group of patients and can still be used in treatment failure.

References

1. Diabetes Mellitus. In: Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL, et al (eds). Harrison's principles of internal medicine, 17th media, New York: McGraw-Hill Medical Publishing Division; 2008, p. 2292-3.
2. Wheat LJ. Infection and diabetes mellitus. Diabetes Care, 1980; 3: 187-97.
3. Thornton GF. Infections and diabetes. Med Clin North Am 1971; 55: 931-8.
4. Joshi N, Caputo GM, Weitekamp MR, Karchmer AW. Infections in patients with diabetes mellitus. N Engl J Med., 1999; 341: 1906–12.
5. Patterson JE, Andriole VT. Bacterial urinary tract infections in diabetics. Infect Dis Clin North Am., 1997; 11: 735-50.
6. Wright SW, Wrenn KD, Haynes M, Haas DW. Prevalence and risk factors for multidrug-resistant uropathogens in emergency patients. Am J Emerg Med., 2000; 18: 143-6.
7. Abdul HZ, Sharqi RM, Bashir AL, Akhter MA. Incidence and Pattern of Infections in Diabetes Mellitus A Retrospective Study. Int J Diab Dev Countries, 1994; 14: 82-4.
8. Geerlings SE, Meiland R, Hoepelman AI. Pathogenesis of bacteriuria in women with diabetes mellitus. Int J Antimicrob Agents, 2002; 19: 539-45.
9. Geerlings SE, Meiland R, van Lith EC, Brouwer EC, Gaastra W, Hoepelman AI. Adherence of type 1-fimbriated Escherichia coli to uroepithelial cells: more in diabetic women than in control subjects. Diabetes Care, 2002; 25: 1405-9.
10. Brauner A, Flodin U, Hylander B, Ostenson CG. Bacteriuria, bacterial virulence and host factors in diabetic patients. Diabet Med., 1993; 10: 550-4.
11. Tahir N, Uddin QT. The frequency of urinary tract infection in diabetic females. KUST Med J., 2009; 1: 55-8.
12. Jha N, Bapat SK. A study of sensitivity and resistance of pathogenic microorganisms causing UTI in Kathmandu valley. Kathmandu Univ Med J (KUMJ), 2005; 3: 123-9.
13. Hasan MK, Nazimuddin K, Ahmed AKMS, Sarker RSC, Haque M, Musa AKM. Differences in bacteriological and antibiotic sensitivity pattern in UTI among hospitalized diabetic and non-diabetic patients. J Medicine, 2007; 8: 10-3.
14. Shill MC, Huda NH, Main FB, Karmakar UK. Prevalence of Uropathogens in Diabetic Patients and Their Corresponding Resistance Pattern: Results of a Survey Conducted at Diagnostic Centers in Dhaka, Bangladesh. Oman Med J., 2010; 25: 282-5.
15. Carson C, Naber KG. Role of fluoroquinolones in the treatment of serious bacterial urinary tract infections. Drugs, 2004; 64: 1359-73.
16. Patterson JE, Andriole VT. Bacterial urinary tract infections in diabetes. Infect Dis Clin North Am., 1997; 11(3): 735–50.
17. Geerlings SE, Hoepelman AI. Immune dysfunction in patients with diabetes mellitus. FEMS Immunol Med Microbiol., 1999; 26: 259–65.
18. Kunin CM. Detection, prevention, and management of urinary tract infections. 4th edition, Philadelphia, PA: Lea and Febiger; 1987.
19. Brauner A, Flodin U, Hylander B, Ostenson CG. Bacteriuria, bacterial virulence and host factors in diabetic patients. Diabet Med., 1993; 10(6): 550–4.
20. Bonadio M, Costarelli S, Morelli G, Tartaglia T. The influence of diabetes mellitus on the spectrum of uropathogens and the antimicrobial resistance in the elderly adult patients with urinary tract infection. BMC Infect Dis., 2006; 6: 54.