ONLINE FIRST

This is a provisional PDF only. Copyedited and fully formatted version will be made available soon.

NICOLAUS COPERNICUS UNIVERSITY IN TORUŃ

MEDICAL RESEARCH JOURNAL

ISSN: 2451-2591

e-ISSN: 2451-4101

A study to estimate the prevalence of Asymptomatic Bacteriuria (ASB) and identify risk factors and causative microorganisms relating to ASB and identify the microorganisms and their sensitivity pattern in type 2 diabetes mellitus (T2D)

Authors: Asis Mitra, Saswati Roy, Arjun Baidya, Rishad Ahmed, Mridul Bera

DOI: 10.5603/mrj.104220

Article type: Original article

Submitted: 2024-12-25

Accepted: 2025-01-29

Published online: 2025-03-12

ORIGINAL ARTICLE

Asis Mitra[1], Saswati Roy[2], Arjun Baidya[3], Rishad Ahmed[4], Mridul Bera[5]

[1]Department of Internal Medicine, Ruby General Hospital, Kolkata, India
[2]Department of Physiology, Jagannath Gupta Institute of Medical Science and Hospital, Kolkata, India
[3]Department of Endocrinology, Nil Ratan Sircar Medical College & Hospital, Kolkata, India
[4]Department of Medicine, KPC Medical College and Hospital, Kolkata, India
[5]NH Narayana Multi Speciality Hospital, Howrah, West Bengal, India

A study to estimate the prevalence of Asymptomatic Bacteriuria (ASB) and identify risk factors and causative microorganisms relating to ASB and identify the microorganisms and their sensitivity pattern in type 2 diabetes mellitus (T2D)

Short title: Asis Mitra et al., Microbiological pattern and risk factors for asymptomatic bacteriuria (ASB)

Correspondence author:
Arjun Baidya
Department of Endocrinology
Nil Ratan Sircar Medical College & Hospital, Kolkata, India
e-mail: drarjun.baidya@rediffmail.com

DOI: 10.5603/mrj.104220

ABSTRACT

Introduction: The microbiological pattern and risk factors for asymptomatic bacteriuria (ASB), which is thought to occur before symptomatic urinary tract infections (UTI) in diabetes mellitus, vary by region. Data from India's eastern region is still missing, though.

Materials and methods: In order to (1) estimate the prevalence of ASB and its association with age, gender, duration of diabetes, and renal and glycemic status, and (2) identify the antibiotic sensitivity pattern of uropathogens and assess the utility of microbial pattern as a predictor of symptomatic UTI, a prospective longitudinal study involving 80 otherwise healthy type 2 diabetes patients with a one-year follow-up was conducted.

Results: In the present study, ASB was common in 21.25% of people with type 2 diabetes. The most frequent cause among males was found to be *Klebsiella sp*. It was discovered that having type 2 diabetes for a long time was the only risk factor for ASB. Age, gender, and recent glycemic status did not correlate. Patients with bacteriuria who had lower baseline HbA1C levels were more likely to get a UTI. Within a year, female diabetes patients with *Escherichia coli*-induced ASB were much more likely to get a UTI.

Conclusions: It will be truly unnecessary to reassess guidelines for screening for ASB caused by *E. coli* in females with chronic diabetes and poor glycemic control if a large-scale prospective trial replicates comparable results. In these patients, symptomatic UTI can be avoided by implementing a strict HbA1C reduction plan early on and taking steps to enhance genital hygiene.

Keywords: urinary tract infection, type 2 diabetes mellitus, asymptomatic bacteriuria, asymptomatic bacteriuria

Introduction

The presence of a freshly voided midstream urine specimen that yields positive cultures (≥ 105 CFU/ml) of the same bacterium in a patient who does not exhibit symptoms of a urinary tract infection (UTI), such as dysuria, urgency, frequency, or fever, is referred to as permissive asymptomatic bacteriuria (ASB), which is the definition used in many studies. In contrast, some studies define ASB in females based on two positive urine culture samples [1]. Why the same uropathogens that cause UTIs are less virulent in these people is not well understood. This lack of symptoms can be explained by lower uroepithelial adherence and, more generally, diminished host reactivity in diabetes [2]. Due to its detrimental impact on glucose control and patients' overall health, ASB is thought to occur before a symptomatic urinary tract infection (UTI; relative risk [RR] 1.65, 95 percent confidence interval [CI] 1.02–2.67). Therefore, it is vital to determine the risk factors in order to prevent UTI [34]. Screening for ASB in individuals with type 2 diabetes is not advised by the current Infectious Disease Society of America (IDSA) guideline. In India, where poor genital cleanliness is still a problem, particularly for female patients from lower socioeconomic backgrounds, this advice might not be totally applicable. Even though two recent investigations on ASB in diabetic patients from North and South India [5] and [6] have been carried out, there are currently no comparable studies from Eastern India. This work aims to close that data gap in light of the shifting prevalence of ASB, the emergence of treatment resistance, and regional differences in the drug susceptibility pattern of uropathogens

The clinical profile of ASB in individuals with type 2 diabetes mellitus was the focus of this investigation. This study's specific goals were to: (1) determine the prevalence of ASB in patients with type 2 diabetes mellitus in Eastern India; (2) investigate the relationship between ASB and age, gender, renal and glycemic status, and length of diabetes; (3) identify the microorganisms and their sensitivity pattern in ASB in patients with diabetes mellitus; and (4) assess the utility of urine culture microbiological patterns as a predictor of symptomatic UTI.

Material and methods

Study population and setting

The study looked at 135 individuals with type 2 diabetes mellitus who were over 18 and saw the diabetes clinic in Kolkata. Exclusions from the study were pregnant women, patients with UTI symptoms and an indwelling urinary catheter, patients who had recently received antibiotic treatment, and patients who had used antiseptics before urine sample collection. Additionally excluded were patients whose urine samples were contaminated, as indicated by the presence of three or more distinct microorganisms in a single sample.

Study design

Following the exclusion of 25 patients based on the exclusion criteria, 110 patients were included in a prospective longitudinal study with a one-year follow-up. After selecting the first patient at random using a two-digit random number table, patients were enrolled using systemic random sampling.

Method of data collection

Each person's informed consent was obtained before a clinical examination and pertinent investigations were conducted. Measurements were made of HbA1c, urea, creatinine, and fasting and postprandial blood glucose. The Modification of Diet in Renal Disease Study (MDRD) equation was used to compute the patient's estimated GFR (eGFR), which was used to evaluate their renal health. Bacteriuria screening was performed on asymptomatic diabetic patients chosen based on sample design. Following appropriate cleaning of the male glans penis and female labia using swabs soaked in clean tap water, a single random clean-catch midstream urine specimen was obtained. After being inoculated into blood agar, MacConkey agar, and nutrient agar, the collected samples were aerobically incubated for 18 to 24 hours at 37 degrees Celsius. Gram staining, biochemical responses, and colony morphology were used to identify the species [8]. In accordance with CLSI recommendations, the Kerby-Bauer disc diffusion method was used to conduct the antibiotic sensitivity test for positive organisms [9]. For a comparison analysis, asymptomatic patients with diabetes mellitus were then split into two groups: Group 1 (ASB-positive patients) and Group 2 (ASB-negative patients).

Follow-up process

Each person was monitored for a full year to watch for UTI episodes. As part of their usual care, they were using insulin and/or oral antidiabetic medications. Simple cystitis and complex upper UTIs with systemic symptoms including fever, chill, stiffness, malaise, or flank pain are both referred to by the general term "UTI". In the present study, a urinary tract infection (UTI) was defined as the presence of any of the traditional symptoms, such as suprapubic discomfort, frequency, urgency, or dysuria, with or without systemic symptoms, and supported by more than five urine pus cells per high power field.

Statistical analysis

At the conclusion of the study, SPSSv22 was used to compile, tabulate, and analyze the data using the proper standard statistical procedure. The student's t-test was used to compare the means of the continuous variables between the ASB and non-ASB populations and the UTI and non-UTI subgroups. For data that was dispersed between groups and evaluated using the Chi-Square test or Fisher's exact test for small sample sizes, contingency tables were created. A P value of less than 0.05 was deemed significant.

Results

53.5% of the 110 participants in the study were between the ages of 40 and 59, while 2.5% were older than 80. Women made up the majority of the study population (53.6%).

ASB was present in 20.91% of the research participants. The majority of ASBs happened to people over 40. Age and the existence of ASB in type 2 diabetes, however, did not statistically significantly correlate (Tab. 1). Among the 50 male participants, 18% had ASB, while 22% of the females did. The present investigation did not find a significant correlation between female gender and the incidence of ASB in type 2 diabetes (Tab. 1)
E. coli was the most frequent cause of ASB in the study population (47%) and *Klebsiella sp.* was the second most prevalent cause (35.3%). Additionally, one instance of coagulase-negative Staphylococcus and two cases of Enterococcus sp. were discovered. The most prevalent organism in the male population was *Klebsiella sp.*

Resistance to ciprofloxacin and amoxicillin was highest in this study sample (77.243 and 70.5%, respectively). Cotrimoxazole (41.17%) and ampicillin (52.94%) showed intermediate resistance. Ipenem resistance was nonexistent, but cefepime (5.88%), piperacillin-tazobactam, amikacin (both 11.7%), levofloxacin, and nitrofurantoin (17.6%) all showed minimal resistance.

Patients with diabetes for more than 15 years had the highest prevalence of ASB (50%), followed by those with 11–15 years (33.33%) and 6–10 years (26.31%). Patients with fewer than five years of diabetes had just 5.5% ASB. Patients with HbA1c levels between 6.5

and 7.4 had the highest ASB, but those with HbA1c levels beyond 8 did not. Although there was no significant difference in the mean HbA1C level between the two groups, patients with ASB had a considerably longer mean duration of diabetes than the non-ASB population (Tab. 1). Most ASB patients reported an eGFR between 30 and 59 ml/min/m2. The mean eGFR of the ASB and non-ASB populations did not differ significantly (Tab. 1).

Patients with ASB who have type 2 diabetes have a markedly increased chance of getting a UTI in the future, according to the present study (Tab. 1). The risk of UTI was significantly higher for patients with ASB and lower baseline HbA1C values. Interestingly, in this case, the length of diabetes had no discernible impact (Tab. 2). To remove the confounding bias, treatment compliance was guaranteed in both the ASB and non-ASB groups. Additionally, there was no statistically significant difference between the two groups' baseline and follow-up HbA1C readings (Tab. 2).

Every UTI case on follow-up had *E. coli* as the causal bacterium. Women made up 29% of diabetic patients with ASB who experienced a UTI during follow-up within a year. During the follow-up period, no UTIs occurred in any of the male ASB patients. When compared to the second most common cause, *Klebsiella*, ASB caused by *E. coli* was found to have a statistically significant increased risk of UTI at follow-up ($p < 0.05$, Fisher's exact statistic value 0.031).

Discussion

The study population's prevalence of ASB (20.91%) was comparable to many other studies that reported prevalence estimates between 8 and 26% [10, 11], which was higher than the 12.5% prevalence rate found in Renko et al.'s meta-analysis [7] and lower than the 28–32% prevalence rate found in recent Indian studies [5, 6, 12]. The majority of ASB cases in those over 40 were in line with findings from other investigations on otherwise healthy individuals [13–15]. According to multiple studies, women are more likely to have ASB (23.25% vs 18.91%) because of their small urethra, which is situated near warm, wet, vulvar, and perianal areas where enteric bacteria are present.

E. coli was the most prevalent causal bacterium of ASB in the study population, in line with other research. Intriguingly, *Klebsiella sp.* was more prevalent in the male population [16, 17]. In a similar vein, Janda et al.'s investigation revealed that adult males were more vulnerable to *Klebsiella sp.* infection [18]. This could be explained by the fact that men are more likely than women to have phimosis, chronic drunkenness, and other risk factors.

Despite growing reports of resistance, especially in *Klebsiella sp*., the present investigation demonstrated high sensitivity to cephalosporin [20, 21]. Levofloxacin sensitivity was higher in this study than ciprofloxacin sensitivity, suggesting that ciprofloxacin resistance is increasing, as one study found [22]. However, the present investigation found that Enterobacteriaceae in Eastern India had remarkable sensitivity to imipenem, which is noteworthy given the frequency of pan-drug-resistant Enterobacteriaceae described by Kumarasamy et al. [23].

Similar to a study by Bahl et al., ours also demonstrated a substantial correlation between the occurrence of bacteriuria and the length of diabetes [24]. The absence of correlation with HbA1c aligned with research conducted by Renko et al. [7] and Zhanel et al. [10]. This implies that the occurrence of ASB is not significantly influenced by glycosuria.

All of these incidents happened in females, and ASB was found to be substantially related to UTI during the 12-month follow-up. During their 18-month or 14-year follow-up, certain prospective cohort studies of female diabetics found no difference in the rates of symptomatic urinary infections between those who were originally bacteriuric and those who were not [25, 26]. However, ASB was linked to a higher incidence of hospitalization for urosepsis in one prospective observational analysis [27].

In the present investigation, *E. coli* significantly outperformed *Klebsiella sp.* in producing symptomatic UTI, suggesting a distinct pathogenetic pathway for UTI. Because *Klebsiella sp.* are nonmotile and lack flagella, unlike other coliforms, they may be the source of the lower incidence of UTI in ASB. The main component of the bacterial flagellum, flagellin, also known as FliC, is a primary antigen of the adaptive immune response in addition to its function in innate immunity. Flagellin has been shown to act as an adhesin in a variety of pathogens, including Clostridium difficile, Pseudomonas aeruginosa, and *Escherichia coli*. It was recently discovered that Shiga-toxigenic *E. coli's* FliC contributes to cellular invasion through lipid rafts [28].

Treatment of ASB did not lower the likelihood of symptomatic UTI, according to a seminal randomized controlled trial on ASB conducted by Harding et al [29]. It was hypothesized that preemptive antibiotic medication might be helpful in ASB patients because of *E. coli* with poor glycemic control, as the trial was not conducted on a specific high-risk population. However, it is stressed that strict HbA1C lowering and hygiene instructions are necessary for this patient group due to the possibility of recurrent bacteriuria and treatment resistance. The necessity to reassess guidelines for screening for ASB in females with chronic diabetes and poor glycemic control will really be eliminated by a large-scale prospective investigation that replicates comparable results.

Conclusions

In people with type 2 diabetes, the prevalence of ASB was reported to be 20.91%, with the majority occurring in women over 40. The most prevalent uropathogen in the male population is *Klebsiella sp*. ASB screening is recommended for female patients in the present group who have had diabetes for a long time, given the noticeably increased incidence of UTI in female patients with ASB caused by *E. coli*. These patients can avoid UTIs by implementing stringent glycemic control early on and taking steps to enhance genital hygiene.

Article information

Data availability statement: *The original contributions presented in the study are included in the article/supplementary material, further inquiries can be directed to the corresponding author.*

Funding: *The author received no financial support for the research, authorship, and/or publication of this article.*

Author contributions: *Asis Mitra: Conceptualization; Formal analysis; Methodology; Writing — original draft; data collection. Saswati Roy: Conceptualization; Formal analysis; Methodology; Writing — original draft; data collection. Arjun Baidya: Conceptualization; Formal analysis; Methodology; Writing — original draft; data collection. Rishad Ahmed: Conceptualization; Supervision; data collection, Writing — review and editing. Mridul Bera: Conceptualization; Investigation; Resources; Formal analysis; data collection.*

Conflict of interest: *The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.*

Acknowledgments: *The authors gratefully appreciate all departmental staff for their support throughout the research and the study participants for their meticulous information. The authors are also thankful to intigent research for their help in medical writing, data collection and data analysis.*

References

1. Nicolle LE, Bradley S, Colgan R, et al. Infectious Diseases Society of America guidelines for the diagnosis and treatment of asymptomatic bacteriuria in adults. Clin Infect Dis. 2005; 40(5): 643–654, doi: 10.1086/427507, indexed in Pubmed: 15714408.

2. Abdel-Aziz Elzayat M, Barnett-Vanes A, Dabour MF, et al. Prevalence of undiagnosed asymptomatic bacteriuria and associated risk factors during pregnancy: a cross-sectional study at two tertiary centres in Cairo, Egypt. BMJ Open. 2017; 7(3): e013198, doi: 10.1136/bmjopen-2016-013198, indexed in Pubmed: 28325856.

3. Geerlings SE, Stolk RP, Camps MJ, et al. Consequences of asymptomatic bacteriuria in women with diabetes mellitus. Arch Intern Med. 2001; 161(11): 1421–1427, doi: 10.1001/archinte.161.11.1421, indexed in Pubmed: 11386891.

4. Feleke Y, Mengistu Y, Enquselassie F. Diabetic infections: clinical and bacteriological study at Tikur Anbessa Specialized University Hospital, Addis Ababa, Ethiopia. Ethiop Med J. 2007; 45(2): 171–179, indexed in Pubmed: 17642174.

5. Hiamanshu D, Singhal S, Vaish A, et al. A study of asymptomatic bacteriuria in North Indian type 2 diabetic patients. Int J Diabetes Dev Ctries. 2015; 6(11), doi: 10.1007/s13410-015-0430-0.

6. Venkatesan KD, Chander S, Loganathan K, et al. Study on asymptomatic bacteriuria in diabetic patients. Int J Contemp Med Res. 2017; 4: 480–483.

7. Renko M, Tapanainen P, Tossavainen P, et al. Meta-analysis of the significance of asymptomatic bacteriuria in diabetes. Diabetes Care. 2011; 34(1): 230–235, doi: 10.2337/dc10-0421, indexed in Pubmed: 20937688.

8. Colles JG, Duguid JP, Fraser AG. Laboratory strategy in diagnosis of infective syndromes. In: Colle JG, editors. Mackie & McCartney Practical Medical Microbiology.Colles JG, Duguid JP, Fraser AG. ed. 14th ed Pun Elsevier; 2006:86–90.

9. CLSI. Clinical & Laboratory Standards Institute: CLSI Guidelines. [Online] Available: http://clsi org.

10. 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. Clin Infect Dis. 1995; 21(2): 316–322, doi: 10.1093/clinids/21.2.316, indexed in Pubmed: 8562738.

11. Schneeberger C, Kazemier BM, Geerlings SE. Asymptomatic bacteriuria and urinary tract infections in special patient groups: women with diabetes mellitus and pregnant women. Curr Opin Infect Dis. 2014; 27(1): 108–114, doi: 10.1097/QCO.0000000000000028, indexed in Pubmed: 24296584.

12. Aswani SM, Chandrashekar Uk, Shivashankara Kn, et al. Clinical profile of urinary tract infections in diabetics and non-diabetics. Australas Med J. 2014; 7(1): 29–34, doi: 10.4066/AMJ.2014.1906, indexed in Pubmed: 24567764.

13. Matthiopoulou G, Ioannou P, Mathioudaki A, et al. Asymptomatic Bacteriuria in Patients with Type 2 Diabetes Mellitus. Infect Dis Rep. 2023; 15(1): 43–54, doi: 10.3390/idr15010005, indexed in Pubmed: 36648859.

14. Colgan R, Nicolle L, McGlone A, et al. Asymptomatic bacteriuria in adults. Am Fam Physician. 2006; 74(6): 985–990, indexed in Pubmed: 17002033.

15. Nicolle LE, et al. Asymptomatic bacteriuria. Curr Opin Infect Dis. 2014; 27(61): 90–96, doi: 10.1097/QCO.0000000000000019, indexed in Pubmed: 24275697.

16. Evans DA, Williams DN, Laughlin LW, et al. Bacteriuria in a population-based cohort of women. J Infect Dis. 1978; 138(6): 768–773, doi: 10.1093/infdis/138.6.768, indexed in Pubmed: 368263.

17. Kunin CM, McCormack RC. An epidemiologic study of bacteriuria and blood pressure among nuns and working women. N Engl J Med. 1968; 278(12): 635–642, doi: 10.1056/NEJM196803212781201, indexed in Pubmed: 4866351.

18. Janda JM, Abbott SL. The Genera Klebsiella and Raoultella. In: The Enterobacteria. 2nd ed. ASM Press, Washington, USA 2006: 115–129.

19. National Nosocomial Infections Surveillance System. National Nosocomial Infections Surveillance (NNIS) System Report, data summary from January 1992 through June 2004, issued October 2004. Am J Infect Control. 2004; 32(8): 470–485, doi: 10.1016/S0196655304005425, indexed in Pubmed: 15573054.

20. Abigail S, Mathai E, Jesudason MV, et al. Ceftazidime resistance among Klebsiella pneumoniae in south India. Indian J Med Res. 1995; 102: 53–55, indexed in Pubmed: 8834813.

21. Hansotia JB, Agarwal V, Pathak AA, et al. Extended spectrum beta-lactamase mediated resistance to third generation cephalosporins in Klebsiella pneumoniae in Nagpur, central India. Indian J Med Res. 1997; 105: 158–161, indexed in Pubmed: 9145597.

22. Astal ZE. Increasing ciprofloxacin resistance among prevalent urinary tract bacterial isolates in the Gaza Strip. Singapore Med J. 2005; 46(9): 457–460, indexed in Pubmed: 16123829.

23. Kumarasamy KK, Toleman MA, Walsh TR, et al. Emergence of a new antibiotic resistance mechanism in India, Pakistan, and the UK: a molecular, biological, and epidemiological study. Lancet Infect Dis. 2010; 10(9): 597–602, doi: 10.1016/S1473-3099(10)70143-2, indexed in Pubmed: 20705517.

24. Bahl AL, Chugh RN, Sharma KB. Asymptomatic bacteremia in diabetics attending a diabetic clinic. Indian J Med Sci. 1970; 24(1): 1–6, indexed in Pubmed: 4912511.

25. Geerlings SE, Stolk RP, Camps MJ, et al. Consequences of asymptomatic bacteriuria in women with diabetes mellitus. Arch Intern Med. 2001; 161(11): 1421–1427, doi: 10.1001/archinte.161.11.1421, indexed in Pubmed: 11386891.

26. Semetkowska-Jurkiewicz E, Horoszek-Maziarz S, Galiński J, et al. The clinical course of untreated asymptomatic bacteriuria in diabetic patients--14-year follow-up. Mater Med Pol. 1995; 27(3): 91–95, indexed in Pubmed: 8935144.

27. Karunajeewa H, McGechie D, Stuccio G, et al. Asymptomatic bacteriuria as a predictor of subsequent hospitalisation with urinary tract infection in diabetic adults: The Fremantle Diabetes Study. Diabetologia. 2005; 48(7): 1288–1291, doi: 10.1007/s00125-005-1794-3, indexed in Pubmed: 15918016.

28. Haiko J, Westerlund-Wikström B. The role of the bacterial flagellum in adhesion and virulence. Biology (Basel). 2013; 2(4): 1242–1267, doi: 10.3390/biology2041242, indexed in Pubmed: 24833223.

29. Harding GK, Zhanel GG, Nicolle LE, et al. Antimicrobial treatment in diabetic women with asymptomatic bacteriuria. N Engl J Med. 2002; 347(20): 1576–1583, doi: 10.1056/NEJMoa021042, indexed in Pubmed: 12432044.

30.

Table 1. Relationship between baseline parameters and urinary tract infections (UTI) and asymptomatic bacteriuria (ASB)

Parameters	**Total (n = 110)**	**ASB (n = 23)**	**Non-ASB (n = 87)**	**P value**
Age (years)	55.11 ± 10.9	54.18 ± 9.48	56.26 ± 11.23	0.789
Male n (%)	50 (45.5%)	9 (39.1%)	41 (47.1%)	$p > 0.05$
Female (n%)	59 (53.6%)	13 (56.5%)	46 (52.9%)	
Duration of diabetes (years)	9.80 ± 6.12	12.13 ± 5.23	7.21 ± 6.42	0.027
HbA1C (%)	7.59 ± 1.21	7.52 ± 1.18	7.7 ± 1.11	0.931
eGFR (ml/min/m^2)	78.8 ± 32.61	72.62 ± 32.58	83.71 ± 32.34	0.281
UTI on follow-up	14 (13%)	7 (30%)	7 (8%)	$p < 0.05$
No UTI on follow-up	96 (87%)	16 (70%)	80 (92%)	

ASB — asymptomatic bacteriuria; eGFR — estimated glomerular filtration rate; UTI — urinary tract infections

Table 2. Relationship between HbA1C and the duration of diabetes and urinary tract infections (UTI) in study participants with and without asymptomatic bacteriuria (ASB)

Characteristics	**ASB (n = 17)**			**Non-ASB (n = 63)**			**p**
	UTI (n = 5)	**No UTI (n = 12)**	P	**UTI (n = 5)**	**No UTI (n = 58)**	***P***	
Duration of diabetes (years)	10 ± 7.21	11.61 ± 6.11	0.710	8.8 ± 7.92	7.69 ± 7.13	0.826	> 0.05
Baseline HbA1C (%)	8.32 ± 0.69	7.2 ± 0.91	0.034	8.3 ± 0.91	7.41 ± 0.42	0.424	> 0.05
HbA1C on follow up (%)	7.7 ± 0.25	7.2 ± 0.5	0.001	7.4 ± 0.28	7.26 ± 0.63	0.741	> 0.05

ASB — asymptomatic bacteriuria; UTI — urinary tract infections