Urinary Tract Infection: Prevalence, Risk Factors, Bacterial Etiologies and Antimicrobial Resistance Profile among Egyptian Diabetic Patients

Dalia E. Desouky, Hala M. Gabr, Mohammed El-Helbawy, and Hanan M. Hathout

***Abstract* — Diabetic patients are more prone to infections due to impaired immune status. One of most frequent infections in diabetic patients are urinary tract infection (UTI). The aim of the work was to study the prevalence and associated risk factors of UTI among diabetic patients attending the outpatient clinics of Menoufia university hospital, and to assess the pattern of antimicrobial sensitivity of isolated organisms. A pre-designed questionnaire was used to collect information about age, sex and residence, smoking habits, and type and duration of diabetes. Laboratory investigations including blood analysis for glucose level, HBA1c, leucocytic count, urine culture and antimicrobial sensitivity testing were done. The prevalence of UTI was 51.3%, and the most significant risk factors associated with infection were older age, being female, BMI > 30, duration of diabetes > 10 years, together with uncontrolled diabetes. Residence, smoking, and type of diabetes were found to be insignificantly associated with UTI. Age, duration of diabetes, and HBA1c were found to be independently associated with UTI. Common isolated organisms in order of frequency were E. coli, Klebsiella, and Coagulase negative staph. More than 50% of isolates were resistant to one or more antibiotic on antimicrobial antibiotic sensitivity testing. The study concluded a high prevalence of UTI among studied patients. Proper control of diabetes with regular screening for HBA1c and UTI among diabetic patients is needed.**

***Index Terms*— Egyptian, infection, risk, urinary.**

I. Introduction

Diabetes mellitus (DM) is one of the top ten causes of death worldwide [1]. The incidence and prevalence of diabetes are increasing as by 2025, the number of diabetic patients is expected to double. [2] This number is expected to be 593 million by 2035 [3]. Diabetic patients have a high burden of the increased susceptibility to bacterial infections, increased risk for hospitalization, and increased mortality due to infection [4]. The most common infection among diabetic patients is urinary tract infection (UTI) [5], which contribute to the overall medical costs [6] and is a leading cause of end-stage renal disease [7]. It was reported that diabetes is associated with longer hospitalization, bacteremia, and septic shock in patients having UTI. In addition, it was found that UTI mortality is five times higher in diabetic patients aged 65 and older, as compared to control patients [8].

A lot of abnormalities in the host system predispose to the development of UTI among diabetic patients [9]. These factors include the immunologic deficiencies such as imperfect migration, phagocytic alteration of chemotaxis in polymorpho¬nuclear leukocytes [3]. These factors make the diabetic patients at increased risk of acute pyelonephritis, renal abscess, and pyelitis. [10] Risk factors for UTI among diabetic patients include glycosuria, low immunity, and bladder dysfunction [11]. The most common pathogen isolated from urine of diabetic patients with UTI is Escherichia coli, other aggressive pathogens include Klebsiella spp., Proteus spp., Enterobacter spp., and Enterococci [12]. In Egypt Diabetes is a growing health problem with a prevalence of type 2 diabetes of 15.6% among adults aged 20 - 79 years [13]. In an Egyptian study done in 2015, the prevalence of UTI among 500 diabetic patients studied was 52.2%, and Escherichia coli was the most prominent uropathogen isolated [14]. This study aimed to assess the prevalence and associated risk factors of UTI among diabetic patients attending the outpatient clinics of Menoufia university hospital. And to determine the antimicrobial sensitivity patterns of isolated organisms.

II. Subjects And Methods

A. Study Design

A cross-sectional study was done on 922 diabetic patients who attended the outpatient clinic of the Internal Medicine Department at Menoufia University Hospital from January to April 2019.

B. Sampling methodology

Patients were chosen by systematic random sample (every other day) for all attendances at that day. Total of 1345 patients attended the clinic at the time of the study. All patients, who agreed to participate in the study after explaining its purpose, were the study participants.

C. Exclusion criteria

Exclusion criteria were:Diabetic patients on antibiotics for the last two weeks, pregnancy, hospital staff, medical personnel, medical students, patients who could not speak or listen or who had mental health problems, and all emergencies and critically ill patients. After exclusion, a total of 922 patients were the study group.

D. Study instrument

A pre-designed questionnaire was used to collect

Published on July 28, 2020.
Dalia E. Desouky, Menoufia University, Egypt.
(corresponding e-mail: daliadesouky1234@gmail.com)
Hala M. Gabr, Menoufia University, Egypt.
Mohammed El-Helbawy, Menoufia University, Egypt.
Hanan M. Hathout, Menoufia University, Egypt.

information about age, sex and residence, smoking habits, and type and duration of diabetes. Patients' height and weight were measured, and the body mass index (BMI) was calculated as weight divided by the square of the height (in kilograms per square meter) [15].

E. Laboratory investigations

Blood analysis: Venous blood samples were taken from all patients and collected samples were investigated for: random blood glucose, total leukocytic count, serum creatinine, and glycated hemoglobin (HbA1c). HbA1c was quantified spectrophotometrically using HbA1c test kits. HbA1c levels less than 7 % were considered good metabolic control and above 7.5 % considered as poor control according to the American Diabetes association [16].

Urine analysis: Urine samples were obtained from all patients by clean voided midstream technique into 20 ml calibrated sterile screw-capped containers. The sample container was labeled with the sample number, date and time of collection and transferred to the microbiology laboratory of the medical college of Menoufia University for the culturing, isolation, biochemical test and drug-resistance test. Until culture time, urine samples were stored at 2–8◦C in refrigerator.

Uropathogens identification of urine samples: Urine samples were cultured on blood agar, MacConkey agar and Cysteine Lactose Electrolyte Deficient Agar (CLED) and the plates were incubated at 37ºC for 24 h. Significant bacteriuria was defined as urine cultures grew >105 colony-forming unit /ml midstream urine [17].

Antimicrobial sensitivity testing: Antimicrobial sensitivity testing of all isolates was done on sensitivity test agar using disc diffusion methods following the definition of the National Committee of Clinical Laboratory Standards (NCCLS). [18] The antimicrobial agents tested were Ampicillin (AMP), Amoxicillin (AMC), Ciprofloxacin (CIP), Gentamycin, Co-trimoxazole, Amikacin, Nalidixic acid, Penicillin, Cefotaxime. The antimicrobial discs were tested in the following concentrations: Gentamycin (10 µg), Co-trimoxazole (25 µg), Amikacin (10µg), Nalidixic acid (30 µg), Penicillin (10 µg), Cefotaxime (30 µg), AMP (10 µg), AMC (10 µg), and CIP (5 µg).

Diameter of the zone of inhibition around the disc was measured to the nearest millimeter using a metal caliper and the isolate were classified as sensitive and resistant according to NCCLS [18].

F. Statistical analysis

Data were coded, tabulated and analyzed using (SPSS) version 20 (Armonk, NY: IBM Corp.). Qualitative data was expressed as numbers and percentages, and Chi- squared test ($\chi 2$) was applied to test the relationship between variables. Quantitative data was expressed as mean and standard deviation (Mean ± SD), and Student-t-test was used to study association between normally distributed quantitative variables. Stepwise binary logistic regression analysis was done for the risk factors Two-sided. A p-value of <0.05 was considered as statistically significant.

III. Results

A total of 922 patients were the study participants, their mean age was (51.08 ±11.79 years), 50.9% (No=469) were females, and 42.9% (No.=396) had a rural residence. The prevalence of UTI among the studied patients was 51.3%.

Table 1 shows that a significant relationship was found between patients who had UIT and who had not according to their age, gender BMI, and the duration of DM. Patients with UTI had a significantly longer age compared with those without UTI (p=0.028). Female patients had a significant higher percent of those having UTI compared to males (54.12% vs. 47.44%) (p= 0.042). Patients with a BMI higher than 30 had a significant higher percent of those having UTI (52.43% VS. 47.5%) (p ≤0.001). Patients who had DM for more than 10 years had a significant higher percent of those having UTI compared with those who had disease duration less than 10 years (72.73% vs. 27.27%) (p ≤ 0.001). A non-significant difference was found between patients who had UIT and who had not according to their residence and type of diabetes (p > 0.05).

TABLE 1: Comparison between patients with and without UTI according to their age, gender, residence, BMI, and type and duration of DM

Variable	UTI (No.= 473)	No UTI (No.= 449)	Chi squared test	p-value
Age in years (Mean ± SD)	51.36±10.42	49.78±11.47	2.19*	0.028
Gender				
- Male	217(45.88%)	236(52.56%)	4.12**	0.042
- Female	256(54.12%)	213(47.44%)		
Residence				
- Urban	231(48.83%)	221(49.22%)	0.01**	0.907
- Rural	242(51.17%)	228(50.73%)		
Smoking				
- Present	71(15.01%)	53(11.80%)	2.03**	0.153
- Absent	402(84.99%)	396(88.20%)		
BMI				
- < 30	248(52.43%)	295(65.70%)	17.34**	<0.001
- >30	227(47.57%)	154(34.30%)		
Type of diabetes				
- Type I	22 (4.65)	17(3.78%)	0.43**	0.51
- Type II	451(93.35%)	432(96.22%)		
Duration of diabetes mellitus			61.59**	<0.001
- < 10 years	129(27.27%)	236(52.56%)		
- >10 years	344(72.73%)	213(47.44%)		

N.B.: * t-test; ** Chi-square test.

Patients diagnosed with UTI had a significant higher level of random blood glucose, total leukocytic count, serum creatinine, and HbA1c levels. Compared to patients without UTI (P ≤ 0.05) (Table 2).

TABLE 2: Comparison between patients with and without UTI according to their random blood glucose (mg/dl), total leukocytic count/1000, serum creatinine (mg/dl), and HbA1c levels

Variable	UTI (No.= 473)	No UTI (No.= 449)	Test	P value
Random blood glucose (mg/dl)	378.9±79.9	244.1 ± 48.1	30.84*	<0.001
HBA1c (%)				
- ≤ 7	207(43.76%)	229(51.00%)	4.84**	0.027
- >7	266(56.24%)	220(49.00%)		
Serum creatinine (mg/dl)	1.27 ± 0.52	0.8 ± 0.12	18.69*	<0.001
Total leukocytic count /1000	15.4 ± 1.6	13.23 ± 0.32	28.21*	<0.001

N.B.: * t-test; ** Chi-square test.

Binary logistic regression analysis was done to detect the independent predictors for UTI among the studied patients.

The present study found that having an older age, a duration of DM more than 10 years, and having a higher level of total leukocytic count and HbA1c were independent predictors for (Table 3) .

TABLE 3: BINARY LOGISTIC REGRESSION ANALYSIS OF RISK FACTORS OF URINARY TRACT INFECTION AMONG DIABETIC PATIENTS

Parameter	Wald	p-value	Odd's ratio	CI 95%
Age	6.84	0.008	1.43	1.10-1.86
Female sex	1.37	0.242	1.18	0.91-1.53
Random blood glucose	2.370	0.124	1.021	0.994-1.049
Total leukocytic count	12.968	<0.001	27.895	5.113-51.988
BMI >30 Kg	1.931	0.165	0.056	0.001-3.271
Duration of diabetes mellitus >10 years	7.345	0.021	2.95	2.25-3.89
HbA1c	4.56	0.032	1.34	1.03-1.73

Table 4 shows the antimicrobial sensitivity of different bacteria isolated from urine culture of the studied patients.

E. coli was sensitive to all studied antibiotics except for CIP, with the highest sensitivity was to cefotaxime (80.3%) and the lowest was to AMP (27.8%). About the remaining isolated organisms, all showed sensitivity to all tested antibiotics.

For klebsiella spp, the highest sensitivity was to AMC (90.1%) and the lowest was to AMP (46.5%). For P. aeruginosa the highest sensitivity was to Nalidixic acid (90.9%) and the lowest was to Cefotaxime. For S. typhi the highest sensitivity was to Cefotaxime (90.9%) and the lowest was to Nalidixic acid (27.2). For Proteus, the highest sensitivity was to Cefotaxime and Nalidixic acid with 100% sensitivity, and the lowest sensitivity was to Penicillin (20.3%). Regarding S. aureus, the highest sensitivity was to CIP and Nalidixic acid with 100% sensitivity, followed by Cefotaxime with 88.4% sensitivity and its sensitivity to all remaining antibiotics was 50%. Enterococcus organism showed 100% sensitivity to AMC and Nalidixic acid and showed the lowest sensitivity to Co-trimoxazole with 41.6% sensitivity. As for Coagulase negative staph, it showed 100% sensitivity to AMC, AMP and Cefotaxime, and its lowest sensitivity was to amikacin (31.4%).

As shown in Table 4, the reported high rate of antibiotic resistance (> 50%) of different isolated organisms is as follows:

E. coli: CIP (100%), AMP (72.1%), amikacin (68.3%), and Co-trimoxazole (50.8) %.

Klebsiella spp.: AMP and amikacin (53.4%).

Coagulase negative staph: amikacin (68.5%).

Proteus: CIP and Co-trimoxazole (59.3%), AMP (69.4%), and Penicillin (79.6%).

Enterococcus: Co-trimoxazole (58.3%).

P. aeruginosa: gentamycin (54.5%), AMC (63.6%), and Cefotaxime (81.8%).

S. typhi: amikacin (63.6%), Nalidixic acid (72.7%), Co-trimoxazole (81.8%) and Penicillin (81.8%).

TABLE 4: FREQUENCY OF ISOLATED ORGANISMS FROM URINE CULTURES OF THE STUDIED PATIENTS AND THEIR ANTIMICROBIAL SUSCEPTIBILITY PATTERN

Isolated bacteria	No (%)	Sensi-tivity	AMP	AMC	CIP	gentamycin	Co-trimoxazole	Amikacin	Nalidixic acid	Penicillin	Cefotaxime
E coli	183 (38.69%)	S R	51(27.87%) 132(72.13%)	104(56.83%) 79(43.17%)	0(0.00%) 183(100.0%)	105(57.38%) 78(42.62%)	90(49.18%) 93(50.82%)	58(31.69%) 125(68.31%)	115(62.84%) 68(37.16%)	115(62.84%) 68(37.16%)	147(80.32%) 36(19.68%)
Klebsieilla spp	101 (21.35%)	S R	47(46.53%) 54(53.47%)	91(90.10%) 10(9.90%)	57(56.44%) 44(43.56%)	92(91.09%) 9(8.91%)	52(51.49%) 49(48.51%)	47(46.53%) 54(53.47%)	83(82.18%) 18(17.82%)	51(50.49%) 50(49.51%)	72(71.29%) 29(28.71%)
Coagulase negative staph	70 (14.8%)	S R	70(100.0%) 0(0.00%)	70(100.0%) 0(0.00%	58(82.86%) 12(17.14%	64(91.43%) 6(8.57%)	35(50.00%) 35(50.00%)	22(31.42%) 48(68.58%)	62(88.57%) 8(11.43%)	55(78.57%) 15(21.43%)	70(100.0%) 0(0.00%
Proteus	59 (12.47%)	S R	18(30.51%) 41(69.49%)	54(91.52%) 5(8.48%)	24(40.68%) 35(59.32%)	36(61.02%) 23(38.98%)	24(40.68%) 35(59.32%)	48(81.36%) 11(18.64%)	59(100%) 0(0.00%)	12(20.34%) 47(79.66%)	59(100.0%) 0(0.00%)
S. aureus	26 (5.50%)	S R	13(50.00%) 13(50.00%)	13(50.00%) 13(50.00%)	26(100.0%) 0(0.00%)	13(50.00%) 13(50.00%)	13(50.00%) 13(50.00%)	13(50.00%) 13(50.00%)	26(100%) 0(0.00%)	13(50.00%) 13(50.00%)	23(88.46%) 3(11.54%)
Enterococcus	12 (2.54%)	S R	10(83.33%) 2(16.67%)	12(100%) 0(0.00%)	9(75.00%) 3(25.00%)	7 (58.33%) 5(41.67%)	5(41.67%) 7(58.33%)	11(91.67%) 1(8.33%)	12(100%) 0(0.00%)	11(91.67%) 1(8.33%)	11(91.67%) 1(8.33%)
P. aerogenosa	11 (2.33%)	S R	9(81.82%) 2(28.18%)	4(36.36%) 7(63.64%)	9(81.82%) 2(28.18%)	5(45.45%) 6(54.55%)	6(54.55%) 5(45.45%)	8(72.73%) 3(27.27%)	10(90.91%) 1(9.09%)	8(72.73%) 3(27.27%)	2(28.18%) 9(81.82%)
S. typhi	11 (2.33%)	S R	8(72.73%) 3(27.27%)	6(54.55%) 5(45.45%)	6(54.55%) 5(45.45%)	8(72.73%) 3(27.27%)	2(28.18%) 9(81.82%)	4(36.36%) 7(63.64%)	3(27.27%) 8(72.73%)	2(28.18%) 9(81.82%)	10(90.91%) 1(9.09%)
Total	473 (100.0%)	S R	226(47.7%) 24 (52.3%)	345(72.9) 119(27.1)	189(40) 284(60)	330(69.8) 143(30.2)	227(48) 246(52)	211(44.7) 262(55.3)	370(78.3) 103(21.7)	267(56.5) 206(43.5)	394(83.8) 79(16.7)

N.B.: R = Resistant; S = Sensitive; AMP: Ampicillin; AMC: Amoxicillin; CIP: Ciprofloxacin.

IV. DISCUSSION

The present study showed that the prevalence of UTI among the studied diabetic patients was 51.3%. This prevalence is going on with other studies done in Egypt and showed also high prevalence of UTI among diabetic patients. [14], [19] Patients with diabetes have a 10-fold increased risk of UTI when compared to non-diabetics in a previous study [20]. Prevalence of UTIs infection and asymptomatic bacteriuria in diabetic patients ranges from 11% to 68 % in some of international studies [21], [22]. This high prevalence among diabetic patients could be attributed to the autonomic neuropathy in those patients that leads to bladder dysfunction, incomplete bladder emptying and stagnation of urine that constitutes a favorable medium for microbial growth [6], [23]. Also, diabetic patients have impaired immune system functions as leucocyte adherence, chemotaxis, and phagocytosis, impaired neutrophil function, low levels of prostaglandin E, thromboxane B2, leukotriene B4, decreased T cell-mediated immune response, etc. leading to increased risk for infection in general, and especially UTIs. In addition, high glucose level in the urine favors the growth of bacteria as enriched media. Study of Akter et al., found that different species of bacteria have been colonizing the urinary tract of diabetic patients due to low immunity, glucosuria, bladder dysfunction, and depletion of local urinary cytokines [24].

In the present study, the prevalence of UTI was associated significantly with increasing mean age, and this was in accordance with other studies [25]. On the other hand, other studies did not find any relationship between age and increased risk of UTI among diabetic patients [26].

This study showed a significantly high prevalence of UTIs among diabetic females compared to diabetic males (54% vs. 45%, $P < 0.042$). This finding agrees with many other studies [25], [26], [27]. Anatomically, women have shorter urethra than men. In addition, bacteria from rectum can easily travel up to the urethra and cause urinary tract infections [28]. Risk factors like pregnancy, frequent sexual intercourse, shorter urethra and perineal colonization of common pathogens like Escherichia coli and S. aureus also increase the risk of UTI infections in female compared to males [29].

A Canadian study demonstrated that diabetic females were 6–15 times more frequently hospitalized for acute pyelonephritis than non-diabetic females, and diabetic males were hospitalized 3.4–17 times more than non-diabetic males. Asymptomatic bacteriuria was reported to have an increased prevalence in diabetics by about 8% to 25% and was also found to have amplified occurrence among patients with longer duration of diabetes [23].

In the present study, there was a significant increase in the risk of UTI among patients with diagnosed diabetes more than 10 years. This may be attributed to the long-term effects of diabetes like immured immune system and neuropathy. Previous studies detected similar increase in the risk for patients with diabetes >15 years [27]. Long standing diabetes may develop cystopathy, nephropathy, and renal papillary necrosis, that predispose to UTI [30]. On the other hand, another study has found that the duration of diabetes did not influence the risk of UTI in diabetic patients [26].

This study revealed that a none-significant association was detected between the type of diabetes and increased infection risk. A previous study has found that people with diabetes, particularly T1DM, are at increased risk of serious infections, but the risk for UTI was higher among T2DM [31]. Another study has found that 64.2% of T1DM patients had positive urine culture with bacteriuria and pyuria, and 77.7% of T2DM patients had positive urine culture and pyuria. The study revealed that Escherichia coli was the most common isolated uropathogen [19].

HbA1c reflects average plasma glucose over the previous six to eight weeks [16]. Concentrations of blood levels of HbA1c among diabetes mellitus (DM) patients are increased when there is poor glycemic control causing renal disease and predisposition to UTI [32]. HbA1c values reflect poor glycemic control and may be a proxy indicator for screening for UTI among women with diabetes mellitus [33].

The present study found a significant association between uncontrolled diabetes in the form of elevated random blood glucose level and HBA1c and increased risk of UTI. this result goes with finding from previous studies [27]. Hyperglycemia contributes to the colonization of different kind of microorganisms in the urinary system [27]. The longer the duration and the greater the severity of diabetes were found to increase the chance to develop UTI [34]. Different results regarding effect of HbA1C was detected by a previous study that found that HBA1c >8 was not found to be associated with UTI [27].

Among the studied diabetic patients, the most commonly isolated in order of frequency were: E. coli (39%), Klebsiella (21%), Coagulase negative staph (15%), and Proteus (12.5%). Other less frequently isolated organisms were Pseudomonas aeruginosa, S. aureus, and Enterococcus.

Both gram positive and gram-negative bacteria are implicated as common causes of UTI, and E. coli was found to be the most common causative agent in both DM and non-DM patients [35]. E-coli was encountered as the leading organism causing UTI in diabetic patients in other studies [27].

There is evidence that strains of multi drugs resistant (MDR) E. coli increased in both diabetic and non-diabetic [12], [36]. In the present study, the antimicrobial resistance patterns of bacteria isolated from UTIs differed for different bacteria and antibiotics, but there is markedly resistance of organisms to tested antibiotics. This observed increase of antibiotic resistance is a world-wide phenomenon that occurs due to the abuse of antibiotics.

Irrational drug use such as: long-term use, low-dose antibiotic use due to lack of protocol for antibiotic use, and empiric therapy due to lack of laboratory facility to determine sensitivity, are the possible reasons for resistance [37]. This resistance was found to be a leading cause of recurrent infections and complicated UTIs [37].

The emergence of resistant bacterial strains occurs due to indiscriminate usage of antibiotics as multiple courses of antibiotic therapy that are administered to asymptomatic or only mildly symptomatic UTI [38]. This leads to the increased resistance to commonly used antimicrobials. [38]

Patients with diabetes are more likely to have resistant organisms causing the UTI, including extended-spectrum β-lactamase Enterobacteriaceae, carbapenem-resistant Enterobacteriaceae, fluoroquinolone-resistant uropathogens, and vancomycin-resistant Enterococci. Type II diabetes was found to serve as a risk factor for fungal UTI [39].

This study revealed that K. pneumoniae was the second most commonly isolated organism. This finding is in agreement with a recent report from Nepal [36]. Other aggressive pathogens are highly prevalent in diabetic UTIs, such as fungal infections, Klebsiella, Gram negative rods, enterococci, group B streptococci, Pseudomonas, and Proteus mirabilis [12]. Misuse and overuse of antimicrobials is one of the world's most pressing public health problems. Infectious organisms adapt to the antimicrobials designed to kill them, making the drugs ineffective. People infected with antimicrobial-resistant organisms are more likely to have longer, more expensive hospital stays, and may be more likely to die because of an infection [40].

Limitations: One of the limitations of this study is collecting data from a tertiary care hospital that

cannot be representative to all Egyptian diabetic patients. Another limitation was the use of antibiotics that were available in the market and not all antibiotics used in the clinical practice.

V. Conclusion

The prevalence of UTI infection among diabetic patients was 51.3%. The most significant risk factors associated with infection were the older age, being female, BMI > 30,

duration of diabetes > 10 years together with the uncontrolled diabetes. Residence, smoking, and type of diabetes was found to be insignificantly associated with UTI. Age, duration of diabetes, HBA1c were found to be independently associated with UTI. the common isolated organisms in order of frequency were E. coli, Klebsiella, and Coagulase negative staph, with more than 50% of isolates resistant to one or more antibiotic on antimicrobial antibiotic sensitivity testing. This study calls for proper control of diabetes with regular screening for HBA1c and for infection especially UTI. Patients should be educated about the appropriate antibiotic use based on culture results. Implementation of stewardship program to rationalize the antibiotic use is needed.

ACKNOWLEDGMENT

All authors acknowledge the cooperation of the participant patients.

ETHICAL APPROVAL

The study was reviewed and approved by the Research Ethics Committee of Menoufia University. Verbal and written consents were obtained from all participants.

COMPETING INTERESTS

The authors declare that they have no competing interests.

REFERENCES

[1] World Health Organization (WHO). WHO The top 10 causes of death. Fact sheet Nr. 310. Updated June 2011. htttp://www.who.int/mediacentre/factsheets/fs310/en/index.html.
[2] NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in diabetes since 1980: a pooled analysis of 751 population-based studies with 4·4 million participants. Lancet 2016; 387:1513–30.
[3] Guariguata L, Whiting DR, Hambleton I, Beagley J, Linnenkamp U, Shaw JE. Global estimates of diabetes prevalence for 2013 and projections for 2035. Diabetes Res Clin Pract 2014;103(2):137–49.
[4] Benfield T, Jensen JS, Nordestgaard BG. Influence of diabetes and hyperglycemia on infectious disease hospitalization and outcome. Diabetologia 2007; 50:549–54.
[5] 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):108–14.
[6] Yu S, Fu AZ, Qiu Y, Engel SS, Shankar R, G. Brodovicz K, et al Disease burden of urinary tract infections among type 2 diabetes mellitus patients in the U.S. J Diabetes Complications 2014;28:621-26.
[7] American Diabetes Association. Position statement. Standards of medical care in diabetes 2012. Diabetes Care 2012; 35: S11-S63.
[8] Kofteridis DP, Papadimitraki E, Mantadakis E, Maraki S, Papadakis JA, Tzifa G, et al. Effect of diabetes mellitus on the clinical and microbiological features of hospitalized elderly patients with acute pyelonephritis. J Am Geriatr Soc 2009; 57(11):2125–8.
[9] Saleem M, Daniel B. Prevalence of Urinary Tract Infection among Patients with Diabetes in Bangalore City. International Journal of Emerging Sciences 2011; 1(2): 133-42.
[10] Hakeem LM, Bhattacharyya DN, Lafong C, Janjua KS, Serhan JT, Campbell IW. Diversity and complexity of urinary tract infection in diabetes mellitus. The British Journal of Diabetes & Vascular Disease 2009; 9(3): 119-25.
[11] Funfstuck R, Nicolle LE, Hanefeld M, Naber KG. Urinary tract infection in patients with diabetes mellitus. Clin Nephrol 2012; 77:40–8.
[12] Aswani SM, Chandrashekar UK, Shivashankara KN, Pruthvi BC. Clinical profile of urinary tract infections in diabetics and non-diabetics. Australasian Medical Journal 2014; 7(1): 29–34.
[13] Hegazi R, El-Gamal M, Abdel-Hady N, Hamdy O. Epidemiology of and Risk Factors for Type 2 Diabetes in Egypt. Annals of global health 2015;81(6):814-20.
[14] El-Nagara MMG, Dawooda AEE, Gabrb HM, Abd El-Nabya EEM. Prevalence of urinary tract infection in Damietta diabetic patients. Menoufia Medical Journal 2015; 28:559–64.
[15] Alberti KGM, Zimmet P, Shaw J. Metabolic syndrome: a new worldwide definition. A consensus statement from the International Diabetes Federation. Diabetic Med 2008; 23:469–80.
[16] American diabetes association. Executive Summary: Standards of Medical Care in Diabetes—2009. Diabetes care 2009; 32: Suppl 1: S6-12.
[17] Forbes B, Sahm D and Weissfeld A. (2007). Infection of the urinary tract. Bailey and Scott's Diagnostic Microbiology, 12th ed., Mosby, USA.
[18] National Committee for Clinical Laboratory Standards (NCCLS). Performance standards of antimicrobial susceptibility, NCCLS approved standards M 100-59 National Committee for Clinical Laboratory Standards, Wayne PA. 2002.
[19] Adly NN, Ragab YM, Hashem AM, Ahmady AK. Effect of diabetes on occurrence of urinary tract infection and asymptomatic bacteriuria among diabetic and ketacidosis patients in Egypt. International Research Journal of Microbiology (IRJM) 2015; 6(3):27-36.
[20] Goswami R, Bal CS, Tejaswi S, Punjabi GV, Kapil A, Kochupillai N. Prevalence of urinary tract infection and renal scars in patients with diabetes mellitus. Diab Res Clin Pract 2001; 53:181–6.
[21] He K, Hu Y, Shi JC, Zhu YQ, Mao XM. Prevalence, risk factors and microorganisms of urinary tract infections in patients with type 2 diabetes mellitus: a retrospective study in China. Therapeutics and Clinical Risk Management 2018;14 403–8.
[22] Hamdan HZ, Kubbara E, Adam AM, Hassan OS, Suliman SO, Adam I. Urinary tract infections and antimicrobial sensitivity among diabetic patients at Khartoum, Sudan. Annals of Clinical Microbiology and Antimicrobials 2015; 14:26-32.
[23] Nitzan O, Elias M, Chazan B, Saliba W. Urinary tract infections in patients with type 2 diabetes mellitus: review of prevalence, diagnosis, and management. Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy 2015, 8:129–36.
[24] Akter T, Mia Z, Shahriar M. Antibiotic Sensitivity of Pathogens Causing Urinary Tract Infection. Bangladesh Pharmaceutical Journal 2013;16(1): 53-8.
[25] Tektook NK, Al- Lehibi KI, Al-Husseinei RK. Prevalence Some Pathogenic Bacteria Causing UTI in Diabetic Patients in Specialized Center for Endocrinology and Diabetes of Baghdad City–Iraq. Medical Journal of Babylon 2017; 14(2):260 – 6.
[26] Al-Rubeaan KA, Moharram O, Al-Naqeb D, Hassan A, Rafiullah MR؛ Prevalence of urinary tract infection and risk factors among Saudi patients with diabetes. World J Urol 2013; 31: 573-8.
[27] Abdulla MC, Jenner FP, Alunga J. Urinary tract infection in type 2 diabetic patients: risk factors and antimicrobial pattern. Int J Res Med Sci 2015;3(10):2576-9.
[28] Njunda AL, Assob NJC, Nsagha SD, Nde FP, Kamga FHL, Nkume AF, Emmanuel TK, et al. Uropathogens from diabetic patients with asymptomatic bacteriuria and Urinary tract infections. Sci J Microb 2012; 1: 141-6.
[29] Pargavi B, Mekala T, Selvi AT, Moorthy K. Prevalence of Urinary Tract Infection (UTI) among diabetics' patients in Vandavasi, Tamil Nadu, India. IJBT 2011;2(2): 42-5.
[30] Nawaz M. Isolation and characterization of tetracycline resistant Citrobacter spp. from catfish. Food Microbiol 2008;25: 85-91.
[31] Carey IM, Critchley JA, De- Wilde S, Harris T, Hosking FJ, Cook DG. Risk of Infection in Type 1 and Type 2 Diabetes Compared with the General Population: A Matched Cohort Study. Diabetes Care 2018; 41:513–21.
[32] Kilpatrick ES, Bloomgarden ZT, Zimmet PZ. Is hemoglobin A1C a step forward for diagnosing diabetes?. BMJ 2009; 339: b4432.
[33] Olayemi-Abdul I, Osazuwa F, Osilume D. Association between elevated HbA1c levels and urinary tract infection among diabetic women. Zahedan J Res Med Sci2015; 17(6):e994.
[34] Abbott SL. Klebsiella, Enterobacter, Citrobacter, Serratia, Plesiomonas, and Other Enterobacteriaceae. In P.R. Murray, E. J. Baron, J. H. Jorgensen, M. A. Pfaller and M. L. Landry (Eds.), Manual of Clinical Microbiology 9th ed., 2007: 698-715.
[35] Geerlings SE. Urinary tract infections in patients with diabetes mellitus: epidemiology, pathogenesis and treatment. International Journal of Antimicrobial Agents 2008;31(1):54–7.
[36] Baral P, Neupane S, Marasini BP, Ghimire KR, Lekhak B, Shrestha B. High prevalence of multidrug resistance in bacterial uropathogens from Kathmandu, Nepal. BMC Res Notes 2012;19(5):38-47.
[37] Craig J, Simpson J, Williams G, Lowe A, Reynolds G, McTaggart S, et al. Antibiotic prophylaxis and recurrent urinary tract infection in children. N Engl J Med 2009; 361:1748-59.

[38] Papadimitriou-Olivgeris M, Drougka E, Fligou F, Kolonitsiou F, Liakopoulos A, Dodou V, et al. Risk factors for enterococcal infection and colonization by vancomycin-resistant entero¬cocci in critically ill patients. Infection 2014;42(6):1013–22.
[39] Giesen LG, Cousins G, Dimitrov BD, van de Laar FA, Fahey T. Predicting acute uncomplicated urinary tract infection in women: a systematic review of the diagnostic accuracy of symptoms and signs. BMC Fam Pract 2010; 11:78-92.
[40] US department of health and human services (Centers for Disease Control and Prevention). Antibiotic Use in the United States, 2017: Progress and Opportunities. 2017. https://stacks.cdc.gov/veiw/cdc/46911.

Dr. Dalia E. Desouky: was borne in Saudi Arabia in 1975. She was graduated from medical school of Menoufia university in 1999 and had her PhD in public health and community medicine in 2011 from the same university. She worked as an assistant professor of public health and community medicine, in faculty of medicine, Taif University, KSA from 2011-2019.

Dr. Hala M. Gabr: was borne in Menoufia governorate, Egypt in 1979. She was graduated from medical school of Menoufia university in 2002 and had her PhD in public health and community medicine in 2011 from the same university. She worked as an assistant professor of public health and community medicine, in faculty of medicine, Menoufia University, from 2016 till now.

Dr. Mohammed El-Helbawy: was borne in Menoufia governorate, Egypt in 1982. He was graduated from medical school of Menoufia university in 2005 and had his PhD in clinical pathology in 2016 from the same university. He worked as a lecturer of clinical pathology in faculty of medicine, Menoufia University, from 2013 till now.

Hanan M. Hathout: was borne in Menoufia governorate, Egypt in 1973. She was graduated from medical school of Menoufia university in 1997 and had her PhD in in public health and community medicine 2007 from the same university. She worked assistant professor of public health and community medicine, in faculty of medicine, Menoufia University, from 2015 till now.