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ORIGINAL ARTICLE

Subtle Urinary Tract Infection in Type-1 Diabetic Children attending Aswan University Hospital

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ABSTRACT

Keyword: T1DM, UTIs, HbA1c, ASB, DKA.

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Background: In Egypt, people with diabetes have four times the prevalence of Asymptomatic bacteriuria compared to the general population. It may cause pyelonephritis or cystitis. Unfortunately, it may be difficult to distinguish pyelonephritis from cystitis clinically, especially in infants and young children. This study aimed to identify the risk factors of subtle Urinary Tract Infection in Type 1 diabetic children. **Methodology:** this case-control study was conducted in the Pediatrics Department, Aswan University Hospital, on 212 children, 106 diabetic patients and 106 controls. All the studied groups carried out urinalysis or urine cultures. **Results:** the prevalence of UTI by urine analysis in our cases was 76.42% vs 16% in the controls, with a significant p value <0.001. Although urine culture was insignificant between 2 groups. Further, abnormal ultrasound findings were significantly increased in T1DM groups than in controls. **Conclusion:** T1DM group had a higher prevalence of UTI by urine analysis compared to the controls. However, urine culture results were insignificantly different between the groups, suggesting a potential discrepancy between the presence of asymptomatic bacteriuria and the development of overt UTI symptoms.

INTRODUCTION

Even with appropriate care and glycemic control, patients with type 1 diabetes may experience several diabetic sequelae **(1)**. Urinary tract infections (UTIs) are among the most common infections, and they are one of the sequelae that raise the incidence of morbidity and mortality in children with diabetes. Additionally, both asymptomatic and symptomatic bacteriuria are more common in diabetics **(2)**. This can be explained by the defective host immune factors and the inhibition of neutrophil phagocytosis and diapedesis caused by elevated serum and urine glucose levels. Furthermore, other factors that contribute to the development of diabetic UTIs include renal papillary necrosis, vesicourethral reflux, nephropathy, and urine retention in diabetic neuropathy **(3)**.

The presence of $\geq 10^5$ colony-forming units/ml of one or more bacterial species in a culture of clean-voided midstream urine taken from a patient who does not exhibit symptoms of a urinary tract infection is known as asymptomatic bacteriuria (ASB) **(4)**. The prevalence of ASB in diabetic patients in Egypt is four times higher than the general population **(5)**. Both the lower

urinary tract (i.e., cystitis) and the upper urinary tract (i.e., pyelonephritis) may be impacted by the infection. Unfortunately, based on clinical symptoms and signs, it may be difficult, if not impossible, to differentiate between cystitis and pyelonephritis, particularly in infants and young children **(6)**.

These two conditions are covered under the general heading of UTI. The high frequency, propensity for recurrence, related morbidity, and difficulties in obtaining an uncontaminated urine sample pose serious difficulties for the physician **(7)**. So, the current study aimed to identify the risk factors of subtle UTI in Type 1 diabetic children.

PATIENTS AND METHODS

This observational case-control study was conducted on diabetic children attending the pediatric endocrinology clinic at Aswan University Hospital, Egypt from May 2023 till April 2024.

The minimum required sample size was calculated using G*Power statistical package program Version 3.1.9.7 (Franz Faul, Universitat Kiel, Germany) using two population means formula using independent t-test and the following assumptions were considered: α= 95%; Power (1 - β): 99%; Effect size = 0.5; Two tail test. A total of 212 children were included in the study and divided into 2 equal groups.

Children between one and 15 years, with type-1 DM (both controlled and uncontrolled) and non-diabetic were included. On the other hand, those with symptomatic UTI at the time of the study, history of urologic disease, other autoimmune diseases, and chronic diseases were excluded.

Sampling

The recruited sample was divided into two groups:

Group-I (cases, no = 106), including children with type 1 diabetes who had regular follow-up in the pediatric endocrinology outpatient's clinic of Aswan University Hospital. All of them have asymptomatic bacteriuria with positive urinalysis or urine culture

Group-II (control, no = 106), including children matched to cases in age and sex, confirmed to be non-diabetic by measuring their fasting blood glucose. All of them have asymptomatic bacteriuria with positive urinalysis or urine culture.

Procedure

All the studied children were subjected to:

- History taking including demographic data, diabetes-focused history (age of disease onset, disease duration, presentation of diabetes, insulin therapy i.e., type of insulin, dose and frequency), history suggestive of UTI (burning micturition, change of urine color or odor, frequency or urgency), history suggestive of chronic diabetic complication (ocular and cardiovascular system (CVS) complications), and history of other medications i.e. antibiotics or any other associated disease.
- Clinical examination including anthropometric measurements (weight, height, and body mass index (BMI)), general and systemic examination.
- Laboratory investigation including complete blood count (CBC), Renal function test (serum BUN and serum creatinine), HbA1c, CRP, urine albumin/creatinine ratio (UACR) and urine analysis (via urinary catheterization). Also, in cases with infection-positive urinalysis, urine culture and pelvic-abdominal
- Full ultra-sound (US) was performed.

Statistical analysis

Data analysis was undertaken using IBM-SPSS version 26 **(8)**. Shapiro-Wilks test and histograms were used to evaluate the normality of the distribution of data. Quantitative parametric data were presented as mean and standard deviation (SD) and were analyzed using independent sample t-test. Quantitative non-parametric data were presented as median and interquartile range (IQR) and were analyzed using Mann Whitney U-test. Qualitative variables were presented as frequency and percentage (%) and were analyzed utilizing the chi-square test. A two tailed p-value < 0.05 was considered significant.

Ethical considerations

All the regulations of the ethical committee of the faculty of medicine were followed. Each patient had a private file with non-disclosure policy at data presentation where all presented data don't contain any personal information specifying the identity of any of the patients. All participants' guardians were required to sign a written consent after reading the patient information sheet or having it read to them.

The World Medical Association's 1964 Declaration of Helsinki, which addresses the research ethics guidelines involving humans and/or animals, was followed **(9)**. The study adhered to the STROBE guidelines for observational studies **(10)**. Also, no incentives or rewards for the participants or their caregivers were provided.

RESULTS

A total of 212 children with asymptomatic UTIs were included for the current study, which was carried out in the Pediatrics Department of Aswan University Hospital.

As shown in **Table 1**, both groups were matched for age (p=0.067), sex (p=0.345), weight (p=0.102), height (p=0.097) and BMI (p=0.224).

Table 1: Baseline Demographic and Clinical data of the studied groups.

		Group I (n=106)	Group II (n=106)	P-value
Age (years)	• **Mean ± SD**	10.61 ± 3.24	7.99 ± 4.05	= 0.067*
	• **Range**	3 – 15	1 - 15	
Gender	• **Male**	58 (54.72%)	55 (51.89%)	= 0.345**
	• **Female**	48 (45.28%)	51 (48.11%)	
Weight (kg)	• **Mean ± SD**	33.97 ± 10.46	27.05 ± 12.21	= 0.102*
	• **Range**	13 – 55	9 - 55	
Height (cm)	• **Mean ± SD**	132.02 ± 16.93	117.16 ± 21.84	= 0.097*
	• **Range**	94 – 157	70 - 152	
BMI (kg/m^2)	• **Mean ± SD**	18.98 ± 2.33	18.72 ± 2.79	= 0.224*
	• **Range**	13.46 - 24.06	14.49 - 26.2	

***Mann-Whitney U-test compares median between two groups**
****Chi-square test compares proportion between groups**

Regarding diabetic disease related data among Group-I (Diabetic group) (n=106), the age of onset ranged between 2 and 12 years with a mean of 8.6 ± 2.2 years. Also, disease duration ranged between 0 and 6 years. About 72% (n=76) of cases had DKA, and about 28% (n=30) had hyperglycemia. Further, the mean insulin daily dose was 0.9 ± 0.2 with a range of 0.6-1.2 IU **(Table 2)**.

Table 2: Disease characteristics of the diabetic patients.

		Group I (n=106)
Age of onset (years)	**Mean ± SD**	8.64 ± 2.24
	Range	2 - 12
Duration of disease (years)	**Mean ± SD**	1.99 ± 2.03
	Range	0 - 6
Type of presentation	**DKA**	76 (71.7%)
	Hyperglycemia	30 (28.3%)
Insulin dosage (IU)	**Mean ± SD**	0.92 ± 0.2
	Range	0.6 - 1.2

Table 3 presented the comparison of laboratory findings between the studied groups. Insignificant difference was found regarding the level of Hgb (p=0.063). On the other hand, significantly (p=0.004) higher mean platelet count was found in Group-I (260.3 ± 88.9*10^9/L) compared with Group-II (255.9 ± 83.5*10^9/L). Also, Group-I had higher mean TLC (13.0 ± 4.1*10^9/L) compared with Group-II (8.8 ± 4.9*10^9/L) (p<0.001). Likely, Group-I had significantly (p<0.001) higher mean level of blood urea (55.7 ± 13.7mg/dl) than Group-II (31.9 ± 10.1mg/dl). Likewise, Group-I had higher mean serum creatinine (1.2 ± 0.2mg/dl) vs. Group-II (0.8 ± 0.3mg/dl) (p<0.001). In contrast, the median CRP was significantly (p<0.001) lower among the diabetic group (20 [12-32]) than the non-diabetic one (34 [24-60]). Consistently, the mean HbA1c level was significantly (p<0.001) higher in Group-I (10.1 ± 2.5%) than Group-II (5.1 ± 0.4%).

Table 3: Laboratory investigations Differences Between groups

	Group I (n=106)	Group II (n=106)	P value

Hb (g/dl)	**Mean ± SD**	11.46 ± 0.96	11.78 ± 1.46	0.063*
	Range	9.3 - 13.2	7 - 16	
Platelets ($x10^9$/L)	**Mean ± SD**	260.29 ± 88.77	225.96 ± 83.53	**0.004***
	Range	135 – 433	98 - 430	
TLC ($x10^9$/L)	**Mean ± SD**	13.03 ± 4.13	8.77 ± 4.99	**<0.001***
	Range	6.3 – 22	2.5 - 22	
Urea (mg/dl)	**Mean ± SD**	55.66 ± 13.75	31.97 ± 10	**<0.001***
	Range	21 – 87	18 - 55	
Creatinine (mg/dl)	**Mean ± SD**	1.15 ± 0.21	0.78 ± 0.26	**<0.001***
	Range	0.6 - 1.6	0.2 - 1.3	
CRP (mg/dl)	**Median**	20	34	**<0.001****
	IQR	12 – 32	24 - 60	
HbA1c (%)	**Mean ± SD**	10.06 ± 2.47	5.07 ± 0.39	**<0.001***
	Range	6.8 - 14.5	4.2 - 5.8	

***Independent Sample T-test compares the mean between two groups**
****Mann-Whitney U-test compare median between two groups**
Hb: hemoglobin, TLC: total leucocyte count, CRP: C-reactive protein, HbA1c: glycated hemoglobin, *: significant as P value ≤ 0.05

As shown in **Table 4 (Urine analysis parameters)**, significantly (p<0.001) higher percentage of patients with pus cell count > 5/HPF was recorded in diabetic group vs. non-diabetic group (84% vs 23.6%, respectively). For crystals, diabetics had a higher proportion of amorphous urates (33%) than non-diabetics (7.6%), while a higher proportion of uric acid and co-oxalate (56.6% and 3.8%) in non-diabetics in comparison with diabetics (51.9% and 0%) (p<0.001). Diabetic cases had significantly (p<0.001) higher frequency of positive detection of glucose (100%), ketones (100%) and casts (granular [23.6%] and hyaline [11.3%]) compared with non-diabetic cases glucose (0%), ketones (0%) and casts (granular [5.7%] and hyaline [3.8%]). Further, positive urinary leucocyte was significantly (p<0.001) among Group-I (15.1%) than Group-II (7.5%) **(Fig. 1).**

Table 4: Comparison of Urinalysis Results among studied groups.

		Group I (n=106)	**Group II (n=106)**	**P value**
pH	• **Mean ± SD**	5.86 ± 0.22	5.82 ± 0.24	0.240*
	• **Range**	4.8 – 6	4.9 – 6	
Pus cells	• **0-5**	17 (16.04%)	81 (76.42%)	**<0.001****

	• **>5**	89 (83.96%)	25 (23.58%)	
RBCs	• **0-4**	94 (88.68%)	100 (94.34%)	0.139**
	• **>4**	12 (11.32%)	6 (5.66%)	
Crystals	• **Nil**	16 (15.09%)	34 (32.08%)	**<0.001****
	• **Uric acid**	55 (51.89%)	60 (56.6%)	
	• **Urate**	35 (33.02%)	8 (7.55%)	
	• **Ca oxalate**	0 (0%)	4 (3.77%)	
Glucose	• **Positive**	106 (100%)	0 (0%)	**<0.001****
	• **Negative**	0 (0%)	106 (100%)	
Ketones	• **Positive**	106 (100%)	12 (11.32%)	**<0.001****
	• **Negative**	0 (0%)	94 (88.68%)	
Casts	• **Nil**	69 (65.09%)	96 (90.57%)	**<0.001****
	• **Granular**	25 (23.58%)	6 (5.66%)	
	• **Hyaline**	12 (11.32%)	4 (3.77%)	
Protein	• **Nil**	97 (91.51%)	100 (94.34%)	0.126**
	• +	5 (4.72%)	6 (5.66%)	
	• ++	4 (3.77%)	0 (0%)	
Nitrite	• **Nil**	106 (100%)	104 (98.11%)	0.498**
	• +	0 (0%)	2 (1.89%)	
Leucocytes	• **Nil**	52 (49.06%)	80 (75.47%)	**<0.001****
	• +	38 (35.85%)	18 (16.98%)	
	• ++	16 (15.09%)	2 (1.89%)	
	• +++	0 (0%)	4 (3.77%)	
	• ++++	0 (0%)	2 (1.89%)	

***Independent Sample t-test compare mean between two groups**
****Chi square test was used to compare proportions between groups**
RBCs: red blood cells, *: significant as P value ≤ 0.05

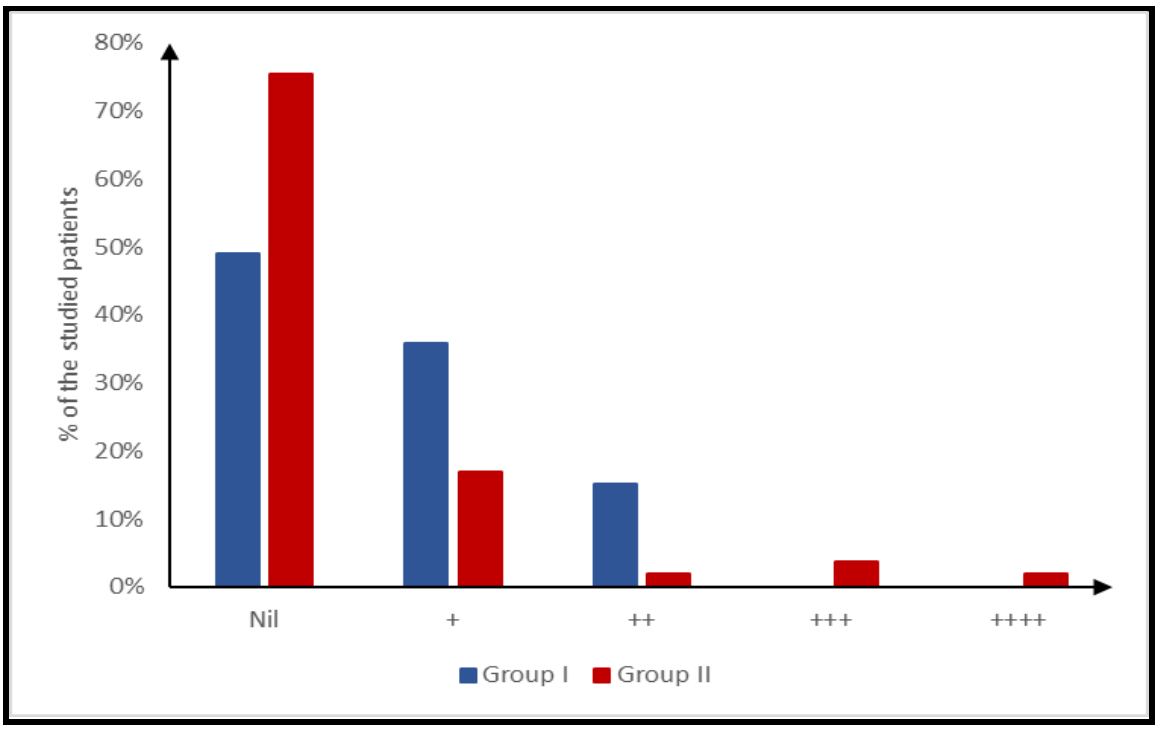

Fig. 1: Urinary Leucocytes among the studied NT cases

Regarding the urine culture results **(Fig. 2),** there was insignificantly (p=0.622) higher percentage of positive findings (E. coli) among diabetic group (9.4%, n=10) vs non-diabetic group (7.6%, n=8).

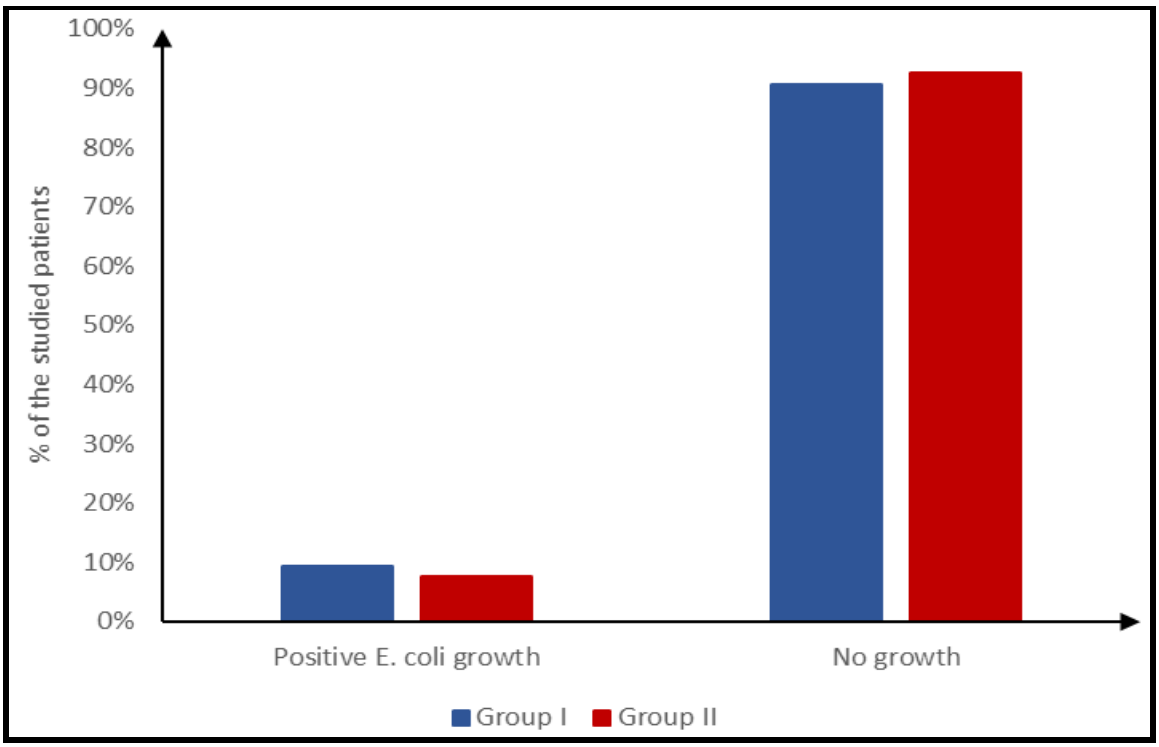

Fig. 2: Urine culture among studied cases

Furthermore, there was a significant (p<0.001) difference respecting the US findings between groups i.e., higher percentage of gaseous distention, nephropathy grades and IPF in diabetic group (28%, 4.7%, 3.8% and 1.9%) vs. non-diabetic group (0%, 3.8% and 0%). In contrast, a higher percentage of hepatomegaly and ascites in the non-diabetic group (5.7% and 1.9%) than the diabetic group (0%) **(Table 5 and Fig. 3)**.

Table 5: Difference in US findings between studied groups

	Group I (n=106)	**Group II (n=106)**	**P value**
Normal	65 (61.32%)	94 (88.68%)	**<0.001***
Gaseous distention	30 (28.3%)	0 (0%)	
Grade 1 nephropathy	5 (4.72%)	4 (3.77%)	
Grade 2 nephropathy	4 (3.77%)	0 (0%)	
Hepatosplenomegaly	0 (0%)	6 (5.66%)	
Ascites	0 (0%)	2 (1.89%)	
IPF	2 (1.89%)	0 (0%)	

***Chi square test was used to compare proportions between groups**

US: ultrasound, IPF: idiopathic pulmonary fibrosis, *: significant as P value ≤ 0.05

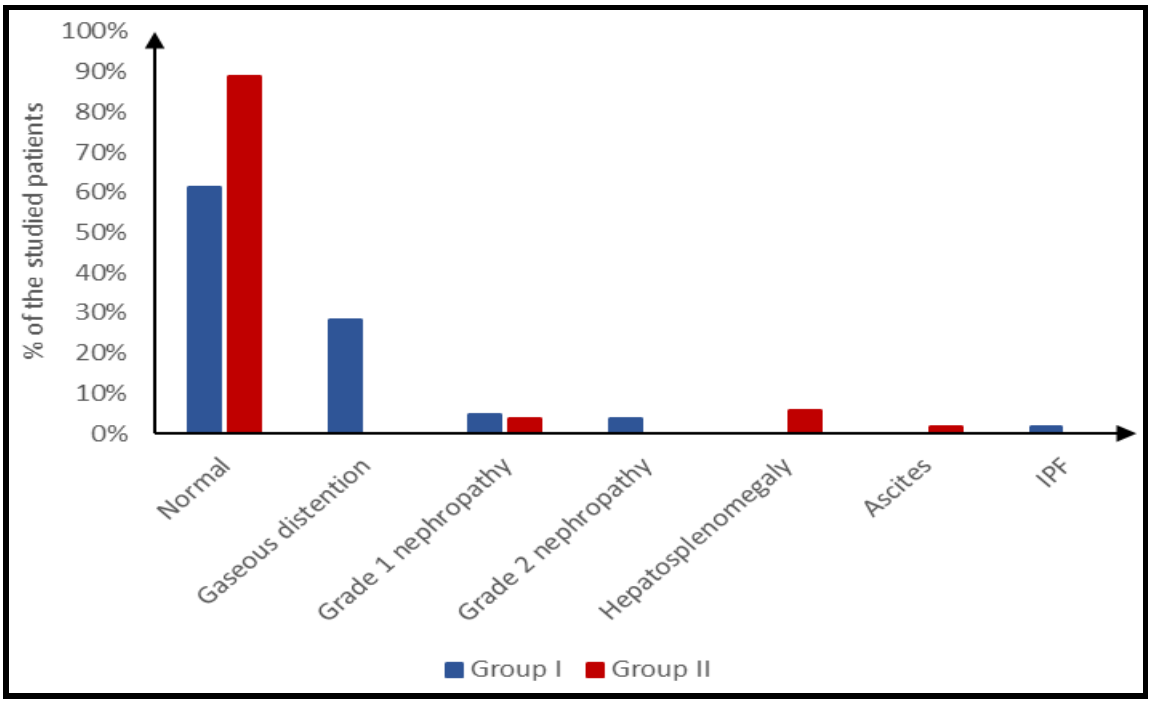

Figure 3: Comparison of US Findings among studied group

DISCUSSION

There is a lot of interest in the medical literature regarding the link between infections and DM. There is a proof that in diabetic cases, proper glycemic control enhances immune function and lowers morbidity and mortality linked to serious infections **(11)**. It's crucial to keep in mind that DM is categorized based on its etiopathogenesis. Type 2 diabetes (T2D) is the most common kind, particularly in adults, and type 1 diabetes (T1D) is the most common type, particularly in children and adolescents. Studies evaluating the incidence and prevalence of infections don't usually specify the kind of DM. T1D is caused by an autoimmune process that targets the β-cells of the pancreas, which produce and secrete insulin. This process destroys the cells, resulting in insulin insufficiency and hyperglycemia. It is mostly linked to microvascular problems (retinopathy, nephropathy, and neuropathy) **(12)**.

People with DM are more likely to get UTIs, which can have serious consequences. An impaired immune response, inadequate bladder emptying, and altered metabolic control are some of the variables that increase the risk of UTI. Escherichia coli and other enterobacteria are the most prevalent agents, just like those found in the general population. Asymptomatic bacteriuria, upper urinary tract infections (pyelonephritis), lower urinary tract infections (cystitis), and urosepsis are the most prevalent illnesses. Additionally, complications such as renal abscess, emphysematous cystitis, and renal papillary necrosis could occur **(13)**.

Our results revealed that the prevalence of UTI by urine analysis in our cases was 76.42% vs 16% in the controls, with a significant p value <0.001. Although urine culture was insignificant between 2 groups.

According to previous research, the prevalence of asymptomatic bacteriuria and UTI infections in diabetic patients varies from 11% to 68% **(14)**. This high prevalence may be related to autonomic neuropathy, which causes bladder dysfunction, inadequate bladder emptying, and urine stagnation, which creates an environment that is conducive to the growth of microorganisms.

This study's objectives were to determine the risk of asymptomatic UTIs in children with T1D and identify the risk factors for developing DKA. A total of 212 children (106 diabetic cases and 106 controls) were enrolled in this case-control study.

In the current study, the male/female ratio was 55%/45% in Group I, this was consistent with the Saudi Health Interview Survey (SHIS), which was conducted in 2013, where the male/female ratio was 15%/12% of women had diabetes. This difference was seen between the sexes **(15)**.

Similar to the findings of other studies, the characteristics of diabetic patients linked to their chance of getting UTIs were examined, and it was discovered that age and disease duration had a significant impact **(16)**. Longer duration of DM may increase the risk of diabetic chronic complications, hospitalization, and urinary tract catheterization, all of which raise the risk of urinary tract infections **(13)**.

Gorter et al. (17) reported several risk factors for recurrent urinary tract infection (rUTI) in females, including insulin treatment. Also, **Wilke et al. (18)** reported that insulin treatment was not associated with rUTI risk. Another study including 1157 Indian patients, showed a correlation between the percentage of patients with UTI and the duration of diabetes (41.8% < 10 years vs. 58.2% > 10 years) **(16)**.

Per the current study, **Desouky et al. (19)** reported that there was a significant increase in the risk of UTI among patients with diagnosed diabetes >10 years. This may be attributed to the long-term effects of diabetes like an impaired immune system and neuropathy. Long-standing diabetes may develop cystopathy, nephropathy, and renal papillary necrosis, which predispose to UTI **(20)**. In contrast, A Saudi study found that the DM duration did not influence the risk of UTI in diabetic patients **(15)**.

In our study, diabetics were insignificantly older compared with healthy controls. This was in agreement with **Al-Rubeaan et al. (14)** who did not find any relationship between age and increased risk of UTI among diabetic patients **(15)**. Contrarily, in Carrondo study, it was found that UTI rate in people aged 18 – 64 was 9%, compared with 27.5% in people over 85 years old and rate of UTI in females was higher than in males, which seems to be associated to bladder neurological malfunction, physiological bladder alterations brought on by aging or dyspnea, and female's close closeness to the anus **(21)**. Similarly, **Desouky et al. (19)** reported that the risk of UTI was associated significantly with increasing mean age.

In this study, HbA1c was significantly decreased in the healthy control group than the T1DM groups (P value < 0.001). These findings are in agreement with those in previous reports suggesting an association between elevated HbA1c levels and presence of UTIs **(22-24)**.

This contradicts the finding from a meta-analysis of 22 studies that the degree of HbA1c derangement does not necessarily impact the biological flora or play any role in UTI susceptibility **(25)**. In the same line, **Ahmed et al. (26)** in a study in Saudi Arabia reported that higher levels of HbA1c were unrelated to the patients' UTI status, either positive or negative, and had only a weak correlation. **Chiţă et al. (13**) demonstrated that glycemic control had no significant influence on the risk for UTIs in the univariate analysis, but appeared as a significant risk factor when multivariate logistic regression model was applied .

Moreover, in the current study, pus cells, glucose, ketones, casts and leucocytes were significantly increased in T1DM groups than control. Likewise, urine culture was significantly different between the studied groups.

It was reported that the main organism that causes UTI in diabetics was E. coli **(27)**. Septic bacteria causing UTI with apparent symptoms such as increased frequency of urination, dysuria, hematuria, and a painful touch, while aseptic bacteria causing UTI without obvious symptoms **(28)**. It was proposed that the risk of aseptic bacteria in people with diabetes is three times higher than in normal people. Several mechanisms were claimed to increase the UTI risk, such as diabetic nephropathy, autonomic neuropathy, immune system abnormalities, and glucosuria **(29)**. Abnormal US findings were significantly different between the studied subgroups in the current study. Consistently, a large retrospective cohort study including 179,580 subjects with T2DM, showed that both cystitis and pyelonephritis were more common in diabetic patients than control (1.34% vs. 0.9% for cystitis and 0.14% vs. 0.07% for pyelonephritis, respectively). Also, recurrence of UTI was higher in diabetics (1.6% vs. 0.6%) **(30)**.

Conclusion

In conclusion, the prevalence of UTI by urine analysis in diabetic patients was significantly higher than in the controls. Although , urine culture results were insignificantly different between the groups, suggesting a potential discrepancy between the presence of asymptomatic bacteriuria and the development of overt UTI symptoms. Further, abnormal US findings were significantly increased in T1DM groups than group II.

In recommendation, further studies with many patients should be done to support our results for a better outcome.

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