Variation of Urine Parameters among Diabetic Patients: A Cross-Sectional Study

Molla Abebe¹, Tiruneh Adane¹, Kassa Kefyalew¹, Tesfahun Munduno¹, Alebachew Fasil¹, Belete Biadgo¹, Sintayehu Ambachew¹, Saira Shahnawaz²

ABSTRACT

BACKGROUND: Diabetic kidney disease is a common and severe microvascular complication of diabetes mellitus (DM). There are limited data regarding alteration of urine parameters other than proteinuria among DM patients.

METHODS: Institution based cross-sectional study was conducted from February to May 2017 to assess alteration of urine parameters among DM patients at the University of Gondar Hospital, Northwest Ethiopia. A Systematic random sampling technique was used to recruit adult (≥18 years) diabetic participants. Data were collected after ethical requirements had been fulfilled. The degree of association between variables was evaluated through bivariable and multivariable logistic regression models.

RESULTS: The majority (69.4%) of the study participants were type 2 DM patients. The prevalence of altered urine chemical parameters was 11.3% proteinuria, 4.5% ketonuria, 13.6% hematuria, 53.8% glucosuria, 24.9% leukocyturia and 1.7% positive for nitrite. Diastolic blood pressure and poor glycemic control were significantly associated with proteinuria. Male participants were 2.4 times more likely to have leukocyturia than female participants. The prevalence of abnormally increased microscopic findings was red blood cells 3.1%, white blood cells 12.5%, epithelial cells 27.5%, yeast cells 1.7%, bacteria 17.8%, casts 3.7% and crystals 29.2%.

CONCLUSIONS: The prevalence of altered urine parameters among DM patients is found to be considerable. These increased prevalences of altered urine parameters are potential indicators for diabetic kidney disease.

KEYWORDS: Diabetes mellitus, Diabetic kidney disease, Proteinuria

INTRODUCTION

Diabetes mellitus (DM) represents a cluster of metabolic disorders characterized by increased serum glucose level which is caused by insulin defects in terms of secretion or action (1). The worldwide prevalence and incidence of DM has grown significantly (2). It was estimated that there were 382 million diabetic individuals in 2013,
and DM patients are predicted to reach up to 592 million in 2035 worldwide. Among these, most people with DM are living in developing countries. In Ethiopia, among adults (20–79 years), the estimated prevalence of diabetes was 4.4% in 2013, and it is expected to rise to 5.1% in 2035 (3). In the world, the number of diabetic adults raised from 108 million (1980) to 422 million (2014) (4). The global annual health expense attributable to DM approximately ranged from USD 612 to 1,099 billion (5).

Diabetes associated hyperglycemia causes long-standing damage, dysfunction and collapse of many vital organs; mainly kidneys, eyes, nerves, heart and blood vessels (6). Long-term complications of DM include nephropathy which leads to renal failure, retinopathy which potentially causes loss of vision, autonomic neuropathy which causes gastrointestinal and cardiovascular dysfunction and peripheral neuropathy which causes foot ulcers (7). The majority of type 1 and type 2 DM patients develop such complications through time (8). There is also high prevalence of hypertension, cardiovascular and peripheral vascular diseases in diabetic patients. Diabetes has a psychosocial impact on diabetic individuals and family members because of its social influence and demands of high treatment cost (9). Complications of DM can be prevented or delayed by consistent checkup and management of serum glucose, hypertension or serum lipid levels (10).

Diabetic kidney disease (DKD) is a familiar and serious complication of DM. It is the primary cause of renal failure as well as mortality and morbidity in diabetic patients. DKD is caused by environmental and genetic factor interactions. DKD has an effect on 15–25% of type 1 and 30-40% of type 2 DM patients (11). Sustained serum glucose level drastically reduces DKD incidence and prevalence. DKD progression can be also slowed down effectively by blood pressure management (12).

The diagnosis of DKD at initial stage allows immediate management which improves disease prognosis. It is challenging to identify biomarkers for the management of kidney disorder progress in DM patients (13). Proteinuria is the marker of DKD and a primary indicator of kidney disorder progress (14). Microalbuminuria is a key biomarker of kidney injury (15). It is the predictor of kidney disorder in DM individuals and associates with premature mortality and morbidity in diabetic, hypertensive and healthy people (16). Many research findings have demonstrated that decreasing urine albumin level reduces the risks of adverse kidney problems (17,18).

The complex nature of diabetes needs consistent management through multi-factorial risk reduction approaches (19). A comprehensive management approach including serum glucose and blood pressure control with appropriate treatment integrated to reduced blood lipid level, low protein consumption, reduced salty diet, continuous physical exercise, weight loss and no smoking habit decreases kidney disease progress rate in DM patients (20,21). Thus, this study has presented baseline data regarding alteration of urine parameters among diabetic patients, which will potentially support in DKD assessment and monitoring.

**METHODS AND MATERIALS**

**Study setting and population:** An institution based cross-sectional study was conducted from February to May 2017 to assess alteration of urine parameters among DM patients at the University of Gondar Hospital (UoGH), Northwest Ethiopia. Gondar is located 738 kms northwest of Addis Ababa, the capital of Ethiopia. UoGH serves more than 5 million residents in northwestern Ethiopia. Approximately, 8,000 DM patients are registered at Chronic Illness Clinic for medical care service.

Adult DM patients (≥18 years) who attended UoGH Chronic Illness Clinic for diabetes follow-up and volunteered to give informed written consent were included. Critically troubled DM patients who were unable to communicate, catheterized patients and pregnant and menstruating women were excluded from this study.

**Sampling procedure:** A Systematic random sampling technique was used to recruit adult (≥18 years) DM study participants. The sample size
was determined based on a single population proportion formula by considering assumptions of 95% level of confidence, 5% margin of error and 50% prevalence of proteinuria. The total calculated sample size was 384. However, the total source population (diabetic patients) was approximately 8,000 (<10,000) and a sample size calculation correction factor (384/8000 =0.048) was used. Therefore, the corrected sample size was 366 (384/1+0.048). Among those, 353 (96.4% response rate) adult DM participants were included in the study. The remaining 3.6% of the population did not volunteer to participate.

**Data collection:** Prior to the actual data collection, a three-days training and demonstration was given for the data collectors (nurse and medical laboratory professionals) about the study participants’ rights, the objectives of the study, confidentiality, the procedure of urine sample collection and measurements, and how to approach and interview participants. Socio-demographic, clinical, behavioral and measurement data of the study subjects were collected by nurses using a pre-tested structured questionnaire. In addition, the urine samples were analyzed by medical laboratory technologist. All aspects of data collection process were supervised by experienced professionals to ensure data quality.

The study participants were communicated early in the morning when they came to UoGH Chronic Illness Clinic for their regular medical follow-up. Primarily, the objective of the study and related issues were explained to study participants by data collectors. Written informed consents were signed by volunteered study participants. Finally, demographic, clinical and measurement data were collected, and at the same time, participants were instructed to collect and transport urine specimen to urinalysis laboratory. Ten ml of urine specimen was collected in a clean, dry and leak-proof urine cap. Chemical parameters of urine were semi-quantitatively assessed by urine dipsticks immediately. Then, the sample was centrifuged at 3,000 rpm for 5 minutes, and the sediment was transferred to clean slide and evaluated microscopically. All the data obtained from chemical and microscopic examination of urine specimen were recorded on registration sheet prepared by authors.

Anthropometric measurements were done by nurses using calibrated equipment and standardized techniques. Every participant’s weight was recorded using weight balance. Stadiometer was used for height measurement. Thus, Body Mass Index (BMI) was calculated as weight/height² (kg/m²). BMI was categorized into <18.5 kg/m² (underweight), 18.5–24.9 kg/m² (normal), 25–29.9 kg/m² (overweight) and ≥30 kg/m² (obese). Waist Circumference (WC) was measured midway of lower rib and iliac crest. WC ≥88 cm for females and >102 cm for males was considered abnormal (22).

Mercury sphygmomanometer instrument was used to measure blood pressure (BP). BP was taken two times after participants relaxed for at least 15 minutes. Five minutes interval was recommended between the two measurements. In the end, the average of the two measurements was used as the final result. Systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg or getting histories of taking anti-hypertensive drugs were taken into account to classify hypertension. The current fasting blood glucose (FBG) data were collected from the patients’ registration book to group participants into good glycemic control (FBG ≤130 mg/dl) and poor glycemic control (FBG >130 mg/dl) (19).

The chemical parameters of urine (protein, glucose, blood, leukocyte, ketone, nitrite, bilirubin, urobilinogen, pH and specific gravity) were determined and reported semi-quantitatively as normal, 1+, 2+, 3+ and 4+ based on the manufacturer’s instruction of urine dipstick test (ALDE Diagnostic Co., Ltd, China). Therefore, the above reports were classified as normal and altered (1+, 2+, 3+ and 4+) for the purpose of this study. The microscopic examination of urine was also performed to evaluate abnormally increased number of urine sediment components (red blood cell (RBC), white blood cell (WBC), yeast cell, epithelial cell, casts, and parasite). RBCs, WBCs and epithelial cells found >5 cells per high power field were considered as abnormal and any yeast cells, bacteria, cast and crystal observed were defined as abnormal. The study followed
standard operation procedures (SOPs) to produce reliable results.

**Data analysis:** The data were cleared, edited and entered into EPI info version 3.5.3 (CDC, USA) and then transferred to SPSS version 20 (IBM, USA) software for statistical analysis. Bivariate and multivariable logistic regression models were used to determine the degree of association between variables. Variables having a p-value of ≤0.2 in the bivariate model were subjected to multivariable analysis to avoid confounding variables’ effect. In addition, crude and adjusted odds ratios, with their 95% confidence interval, were used to evaluate the associations between variables. P-value <0.05 was taken as statistically significant.

**RESULTS**

**Socio-demographic and clinical characteristics:** The study recruited 353 Diabetic patients. The mean age was 49.3±15.2 (range: 18-85 years). From all participants, 138 (39.1%) had no education, 112 (31.7%) were housewives, 234 (66.3%) were married, 245 (69.4%) were type 2 DM patients and 122 (34.6%) were hypertensive (Table 1).

| Variable                        | Category | Number | Percent |
|---------------------------------|----------|--------|---------|
| Sex                             | Male     | 168    | 47.6    |
|                                 | Female   | 185    | 52.4    |
| Age                             | ≤49      | 155    | 43.9    |
|                                 | >49      | 198    | 56.1    |
| Marital status                  | Single   | 39     | 11.0    |
|                                 | Married  | 234    | 66.3    |
|                                 | Divorced | 39     | 11.0    |
|                                 | Widowed  | 41     | 11.6    |
| Occupation                      | Employed | 96     | 27.2    |
|                                 | Housewife| 112    | 31.7    |
|                                 | Farmer   | 52     | 14.7    |
|                                 | Private  | 61     | 17.3    |
|                                 | Other    | 32     | 9.1     |
| Educational status              | No education | 138 | 39.1 |
|                                 | Primary  | 76     | 21.5    |
|                                 | Secondary| 61     | 17.3    |
|                                 | Higher   | 78     | 22.1    |
| Type of diabetes                | Type 1   | 108    | 30.6    |
|                                 | Type 2   | 245    | 69.4    |
| Family history of DM            | Yes      | 40     | 11.3    |
|                                 | No       | 313    | 88.7    |
| Hypertension                    | Present  | 122    | 34.6    |
|                                 | Absent   | 231    | 65.4    |
| Waist circumference             | Low risk | 230    | 65.2    |
|                                 | High risk| 123    | 34.8    |
| Blood glucose                   | ≤130     | 111    | 31.4    |
|                                 | >130     | 242    | 68.6    |
| Body mass index                 | <25      | 218    | 61.8    |
|                                 | 25-29.9  | 94     | 26.6    |
|                                 | ≥30      | 41     | 11.6    |
| Total                           |          | 353    | 100     |

**Prevalence of altered urine chemical parameters:** The 259 (73.4%) study participants had at least one altered chemical parameter. Mixed alteration was: 59.8% (155/259) single,
Variation of Urine Parameters

Molla A. et al.

DOI: http://dx.doi.org/10.4314/ejhs.v29i1.9

30.9% (80/259) double, 8.9% (23/259) triple and 0.4% (1/259) quadruple. The prevalence of altered urine chemical parameters was 11.3% (95% CI: 8.2-15) proteinuria, 4.5% (95% CI: 2.5-6.8) ketonuria, 13.6% (95% CI: 10.2-17.3) hematuria, 53.8% (95% CI: 48.4-58.9) glucosuria, 24.9% (95% CI: 20.7-29.5) leukocyturia and 1.7% (95% CI: 0.6-3.1) positive for nitrite (Figure 1).

![Figure 1: Alteration of urine chemical parameters of DM patients attending at University of Gondar Hospital, Gondar, Northwest Ethiopia, 2017 (n= 353)](image)

**Figure 1**: Alteration of urine chemical parameters of DM patients attending at University of Gondar Hospital, Gondar, Northwest Ethiopia, 2017 (n= 353)

Risk factors associated with altered urine chemical parameters: Elevated diastolic blood pressure and poor glycemic control were significantly associated with proteinuria. The odds of hematuria was 2.4 times higher among female participants compared to their male counterparts. On the other hand, male participants were 2.4 times more likely to have leukocyturia than female participants. Furthermore, the odds of glucosuria was 3.4 times higher among study participants with poor glycemic control than good glycemic control (Table 2).

Alteration of urine microscopic parameters: The study participants, 217 (61.5%), had at least one urine microscopic parameter abnormality. Mixed abnormality was: 60% (130/217) single, 27.6% (60/217) double, 9.7% (21/217) triple and 2.7% (6/217) quadruple. Observed casts were 13: 9 casts were granular and 3 casts were cellular (RBC, WBC or epithelial cell) and 1 was hyaline cast. The frequently reported crystals were calcium oxalate (87/103) and uric acid (13/103) (Figure 2).
| Variable | Proteinuria | COR | P-value | AOR | P-value |
|----------|-------------|-----|---------|-----|---------|
| SBP | <140 | 257 | 26 | 1.013 | 1.573(0.623-3.971) | 0.338 |
| | ≥140 | 257 | 14 | 2.471(1.214-5.032) | 1.004 | 0.256 |
| DBP | <90 | 257 | 28 | 1.003 | 1.573(0.623-3.971) | 0.019 |
| | ≥90 | 257 | 12 | 3.197(1.498-6.824) | 1.003 | 0.256 |
| BG | ≤130 | 105 | 6 | 1.022 | 1.573(0.623-3.971) | 0.019 |
| | >130 | 208 | 34 | 2.861(1.164-7.029) | 1.003 | 0.256 |

| Hematuria | | |
|----------|----------|----------|----------|----------|----------|----------|----------|----------|----------|
| Sex | Male | 152 | 16 | 1.036 | 1.573(0.623-3.971) | 0.338 |
| Female | 153 | 32 | 1.987(1.047-3.771) | 1.573(0.623-3.971) | 0.338 |
| TDM Type 1 | 88 | 20 | 1.761(0.943-3.291) | 1.573(0.623-3.971) | 0.338 |
| Type 2 | 217 | 28 | 1.076 | 1.573(0.623-3.971) | 0.338 |
| HTN Present | 111 | 11 | 1.003 | 1.573(0.623-3.971) | 0.338 |
| Absent | 194 | 37 | 1.003 | 1.573(0.623-3.971) | 0.338 |

| Leukocyturia | | |
|----------|----------|----------|----------|----------|----------|----------|----------|----------|----------|
| Sex | Male | 140 | 28 | 1.001 | 2.4(1.442-3.994) | 0.338 |
| Female | 125 | 60 | 2.4(1.442-3.994) | 2.4(1.442-3.994) | 0.338 |
| Age | ≤49 | 122 | 33 | 1.016 | 1.573(0.623-3.971) | 0.338 |
| >49 | 143 | 55 | 1.422(0.867-2.332) | 1.573(0.623-3.971) | 0.338 |
| ALC | Yes | 31 | 5 | 0.455(0.171-1.208) | 0.114 | 0.609(0.22-1.682) | 0.338 |
| No | 234 | 83 | 0.163 | 1.573(0.623-3.971) | 0.338 |
| SBP | <140 | 217 | 66 | 1.016 | 1.573(0.623-3.971) | 0.338 |
| ≥140 | 48 | 22 | 1.507(0.848-2.678) | 1.573(0.623-3.971) | 0.338 |
| DBP | <90 | 232 | 72 | 1.016 | 1.573(0.623-3.971) | 0.338 |
| ≥90 | 33 | 16 | 1.562(0.813-3.002) | 1.573(0.623-3.971) | 0.338 |
| WC | Low risk | 185 | 52 | 1.016 | 1.573(0.623-3.971) | 0.338 |
| High risk | 87 | 36 | 1.416(0.862-2.327) | 1.573(0.623-3.971) | 0.338 |
| BMI | <25 | 173 | 45 | 0.103 | 1.573(0.623-3.971) | 0.338 |
| 25-29.9 | 62 | 32 | 1.984(1.158-3.399) | 1.573(0.623-3.971) | 0.338 |
| ≥30 | 30 | 11 | 1.415(0.656-3.029) | 1.573(0.623-3.971) | 0.338 |

| Glucosuria | | |
|----------|----------|----------|----------|----------|----------|----------|----------|----------|----------|
| Age | ≤49 | 57 | 98 | 1.982(1.289-3.044) | 0.002 | 1.729(1.081-2.764) | 0.022 |
| >49 | 106 | 92 | 1.352(0.888-2.058) | 0.16 | 1.388(0.879-2.191) | 0.159 |
| Sex | Male | 71 | 97 | 1.352(0.888-2.058) | 0.16 | 1.388(0.879-2.191) | 0.159 |
| Female | 92 | 93 | 1.352(0.888-2.058) | 0.16 | 1.388(0.879-2.191) | 0.159 |
| TDM Type 1 | 40 | 68 | 1.714(1.078-2.726) | 0.023 | 1.118(0.59-2.121) | 0.732 |
| Type 2 | 123 | 22 | 0.101 | 1.451(0.776-2.712) | 0.243 |
| DDM | <120 | 133 | 167 | 1.638(0.909-2.952) | 0.101 | 1.451(0.776-2.712) | 0.243 |
| ≥120 | 30 | 23 | 0.101 | 1.451(0.776-2.712) | 0.243 |
| ALC | Yes | 12 | 24 | 1.819(0.879-3.764) | 0.107 | 1.473(0.658-3.301) | 0.346 |
| No | 151 | 166 | 0.107 | 1.473(0.658-3.301) | 0.346 |
| HTN Present | 70 | 52 | 0.501(0.321-0.781) | 0.002 | 0.557(0.343-0.905) | 0.018 |
| Absent | 93 | 138 | 0.041 | 0.948(0.497-1.807) | 0.87 |
| SBP | <140 | 123 | 160 | 0.577(0.34-0.978) | 0.041 | 0.948(0.497-1.807) | 0.87 |
| ≥140 | 40 | 30 | 0.577(0.34-0.978) | 0.041 | 0.948(0.497-1.807) | 0.87 |
| BG | ≤130 | 73 | 38 | 3.244(2.026-5.195) | 0.000 | 3.393(2.092-5.502) | 0.000 |
| >130 | 90 | 152 | 3.244(2.026-5.195) | 0.000 | 3.393(2.092-5.502) | 0.000 |
| WC | Low risk | 98 | 132 | 0.067 | 0.931(0.504-1.722) | 0.821 |
| High risk | 65 | 58 | 0.067 | 0.931(0.504-1.722) | 0.821 |
| BMI | <25 | 93 | 125 | 0.307 | 0.911(0.532-1.559) | 0.733 |
| 25-29.9 | 46 | 48 | 0.307 | 0.911(0.532-1.559) | 0.733 |
| ≥30 | 24 | 17 | 0.048 | 0.717(0.338-1.52) | 0.385 |

ALC: Alcohol consumption, AOR: Adjusted odds ratio, BG: Blood glucose, BMI: Body mass index, COR: Crude odds ratio, DBP: Diastolic blood pressure.
**DISCUSSION**

This study demonstrated that 242 (68.6%) DM patients had poor glycemic control (FBG >130 mg/dl). DM patients who are unable to control their blood glucose level may develop complications, like diabetes kidney disease, diabetes ketoacidosis, and infection. Therefore, DM patients with proteinuria (11.3%), ketonuria (4.5%) and leukocyturia (24.9%) are at risk of the above-mentioned complications. There is evidence that diabetes is associated with onset and severity of urologic disorders which result in complications, such as bladder, sexual dysfunction and urinary tract infections (23).

The results of this study revealed 11.3% (95% CI: 8.2-15) prevalence of proteinuria which is lower than microalbuminuria findings from the study conducted in India (36.3%) (24) and Saudi Arabia (37.4%) (25) and total albuminuria (macroalbuminuria plus microalbuminuria) prevalence in Tanzania (15.6%) (26). This variation may be due to ethnic variation (27), patient mix (11) and method of determination of urine protein. Studies supported that there are racial/ethnic differences in proteinuria due to DKD among patients with type 2 diabetes (28). Our study population comprised of both type 1 and type 2 DM patients but previous studies used only type 2 DM patients. A study conducted on young Japanese DM patients showed a higher incidence of nephropathy in type 2 than type 1 DM patients; more likely, type 2 DM is the major cause of DKD ([29]. The urine dipstick primarily measures albumin, but sensitivity and specificity are relatively lower than quantitative methods. In addition, most dipstick tests are sensitive to albumin but may not detect low concentrations of Bence Jones proteins and γ-globulins (30).

Proteinuria was significantly associated with elevated DBP and poor glycemic control in this study population. However, it was not significantly associated with age, sex, type of DM and other variables. In Saudi Arabia, microalbuminuria was positively related to BMI, hypertension, duration of DM and fasting plasma glucose. Similar with our study, no statistically significant correlation was found between microalbuminuria and age (25). In India, age, DBP, fasting plasma glucose and duration of DM were found to be associated with microalbuminuria (24). A study in the United Kingdom also identified that male gender and increased WC were the independent risk factors

![Figure 2: Alteration of urine microscopic parameters of DM patients attending at University of Gondar Hospital, Gondar, Northwest Ethiopia, 2017 (n= 353)](image-url)
of albuminuria (27). Study population diversity and sample size inconsistency may affect associated risk factors for proteinuria.

This study showed 13.6% hematuria and 3.9% abnormally increased number of RBCs. The source of this hematuria might be DKD. Moreover, hematuria can be caused by other kidney diseases, cystitis, pyelonephritis, urinary tract infection, kidney stone and cancer of the urinary tract, which are directly or indirectly related to DM (31). Hematuria was significantly associated with type 1 DM and female sex. In addition to urinary system disease, hematuria can happen physiologically in females who were at menstruation and sexual intercourse prior to the data collection time.

This study depicted 24.9% leukocyturia, 1.7% positive for nitrite, 17.8% bacteriuria and 12.5% abnormally increased WBCs. All of the above figures represent the presence of infection in the urinary system, which is probably considered as the complication of DM. A study conducted in the Netherlands has found that diabetic patients were more at risk for urinary tract infection (UTI) than hypertensive patients without diabetes (32). The prevalences of asymptomatic bacteriuria and incidence of UTIs are more frequent to happen in DM patients than individuals without diabetes (33). UTIs are familiar or serious and can lead to undesirable effects in type 2 DM patients (34). Our study demonstrated that leukocyturia was associated with male sex, and different studies found that UTIs commonly affect diabetic women more than non-diabetic women (35). The defect in urinary cytokine secretion and increased bacterial colonization of uroepithelial cells are the potential mechanisms of increased prevalence of UTIs in such patients (36).

In this study, poor glycemic control, younger age and hypertension were associated with glucosuria. If the blood glucose level is above the renal threshold, it will be excreted through urine. Young DM patients may have low awareness and practice regarding blood glucose control, and there is the probability of glucosuria in those patients. As various studies documented, hypertension is usually associated with nephropathy (37,38).

The prevalence of altered urine parameters of DM patients was found to be significant. These increased prevalences of altered urine parameters are potential indicators of diabetic kidney disease. Proteinuria was associated with diastolic blood pressure and poor glycemic control. Hematuria was higher among female participants compared to their counterparts. On the other hand, male participants were more likely to have leukocyturia than females. Urine parameters could help in the diagnosis of diabetic kidney disease. Moreover, the result of this study can be used as a baseline data for further longitudinal and multicenter studies in developing countries.

This study was conducted after obtaining ethical approval from the Research and Ethics Review Committee of the School of Biomedical and Laboratory Sciences, College of Medicine and Health Sciences, University of Gondar. Informed written consent was taken from each of the study participant to participate in the study. The results of this study were based on only one urine sample because of budget limitation. This study was also a cross-sectional study, and it was not possible to assess the alteration of urine parameters repeatedly for a long period.

**ABBREVIATIONS**

BMI: Body Mass Index; BP: Blood Pressure; CI: Confidence Interval; DBP: Diastolic Blood Pressure; DKD: Diabetic Kidney Disease; DM: Diabetes Mellitus; FBG: Fasting Blood Glucose; RBC: Red Blood Cell; SOPs: Standard Operation Procedures; UoGH: University of Gondar Hospital; UTI: Urinary Tract Infection; WBC: White Blood Cell; WC: Waist Circumference.

**ACKNOWLEDGMENTS**

The authors appreciate the commitment of the study participants, data collectors and the University of Gondar Hospital laboratory staffs in the course of this study.

**REFERENCES**

DOI: http://dx.doi.org/10.4314/ejhs.v27i8.9
1. American Diabetes Association: Diagnosis and classification of diabetes mellitus. *Diabetes care.* 2004;27(suppl 1):s5-s10.

2. Tuttle KR, Bakris GL, Bilous RW, Chiang JL, De Boer IH, Goldstein-Fuchs J, Hirsch IB, Kalantar-Zadeh K, Narva AS, Navaneethan SD: Diabetic kidney disease: a report from an ADA Consensus Conference. *American journal of kidney diseases.* 2014;64(4):510-533.

3. Gueriguata L, Whiting D, Hambleton I, Beagley J, Linnenkamp U, Shaw J: Global estimates of diabetes prevalence for 2013 and projections for 2035. *Diabetes research and clinical practice.* 2014;103(2):137-149.

4. NCD Risk Factor Collaboration: Worldwide trends in diabetes since 1980: a pooled analysis of 751 population-based studies with 4·4 million participants. *The Lancet.* 2016;387(10027):1513-1530.

5. da Rocha Fernandes J, Ogurtsova K, Linnenkamp U, Seuring T, Zhang P, Cavan D, Makaroff LE: IDF Diabetes Atlas estimates of 2014 global health expenditures on diabetes. *Diabetes research and clinical practice.* 2016;117:48-54.

6. American Diabetes Association: Diagnosis and classification of diabetes mellitus. *Diabetes care.* 2014;37(Supplement 1):S81-S90.

7. Gavin III JR, Alberti K, Davidson MB, DeFronzo RA: Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes care.* 1997;20(7):1183-1197.

8. Forbes JM, Cooper ME: Mechanisms of diabetic complications. *Physiological reviews.* 2013;93(1):137-188.

9. American Diabetes Association: Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes care.* 2003;26(suppl 1):s5-s20.

10. Deshpande AD, Harris-Hayes M, Schootman M: Epidemiology of diabetes and diabetes-related complications. *Physical therapy.* 2008;88(11):1254-1264.

11. Schrijvers BF, De Vriese AS, Flyvbjerg A: From hyperglycemia to diabetic kidney disease: the role of metabolic, hemodynamic, intracellular factors and growth factors/cytokines. *Endocrine Reviews.* 2004;25(6):971-1010.

12. Reidy K, Kang HM, Hostetter T, Susztak K: Molecular mechanisms of diabetic kidney disease. *The Journal of clinical investigation.* 2014;124(6):2333-2340.

13. Adeosun OG, Anetor JI, Ogunlewe JO, Ikem RT, Kolawole BA, Arogundade FA, Oyediji SO: Evaluation of alterations in the urine biochemical profiles of type 2 diabetes mellitus patients in Southwest, Nigeria. *African Journal of Biotechnology.* 2014;13(1):175-180.

14. Jefferson J, Shankland S, Pichler R: Proteinuria in diabetic kidney disease: a mechanistic viewpoint. *Kidney international.* 2008;74(1):22-36.

15. Ghuhoyschi C, Ghuhoyschi G, Petrica L, Timar R, Velcioc S, Ionita I, Kaycsa A, Timar B: Urinary biomarkers in the assessment of early diabetic nephropathy. *Journal of diabetes research.* 2016;2016.

16. Mogensen C: Microalbuminuria and hypertension with focus on type 1 and type 2 diabetes. *Journal of internal medicine.* 2003;254:45-66.

17. Basi S, Fesler P, Mimran A, Lewis JB: Microalbuminuria in type 2 diabetes and hypertension. *Diabetes care.* 2008;31(Supplement 2):S194-S201.

18. Weir MR: Microalbuminuria in type 2 diabetics: an important, overlooked cardiovascular risk factor. *The Journal of Clinical Hypertension.* 2004;6(3):134-143.

19. American Diabetes Association: Standards of Medical Care In Diabetes-2017. *Diabetes Care.* 2017;40 (Supplement 1):S1-S87.

20. De Boer IH, Rue TC, Cleary PA, Lachin JM, Molitch ME, Steffes MW, Sun W, Zinman B, Brunzell JD: Long-term renal outcomes of patients with type 1 diabetes mellitus and microalbuminuria: an analysis of the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications cohort. *Archives of internal medicine.* 2011;171(5):412-420.

DOI: http://dx.doi.org/10.4314/ejhs.v29i1.9
21. Satirapoj B, Adler SG: Comprehensive approach to diabetic nephropathy. *Kidney research and clinical practice*. 2014;33(3):121-131.

22. National Institutes of Health: Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *Circulation*. 2002;106:3143-3421.

23. Brown JS, Wessells H, Chancellor MB, Howards SS, Stapleton AE, Steers WD, Van Den Eeden SK, McVary KT: Urologic complications of diabetes. *Diabetes care*. 2005;28(1):177-185.

24. Varghese A, Deepa R, Rema M, Mohan V: Prevalence of microalbuminuria in type 2 diabetes mellitus at a diabetes centre in southern India. *Postgraduate medical journal*. 2001;77:399-402.

25. AlFehaid AA: Prevalence of microalbuminuria and its correlates among diabetic patients attending diabetic clinic at National Guard Hospital in Alhasa. *Journal of family & community medicine*. 2017;24(1):1.

26. Lutale JJK, Thordarson H, Abbas ZG, Vetvik K: Microalbuminuria among type 1 and type 2 diabetic patients of African origin in Dar Es Salaam, Tanzania. *BMC nephrology*. 2007;8:2.

27. Retnakaran R, Cull CA, Thorne KI, Adler AI, Holman RR: Risk Factors for Renal Dysfunction in Type 2 Diabetes. *UK Prospective Diabetes Study 74*. 2006;55:1832-1839.

28. Bhalla V, Zhao B, Azar KM, Wang EJ, Choi S, Wong EC, Fortmann SP, Palaniappan LP: Racial/ethnic differences in the prevalence of proteinuric and nonproteinuric diabetic kidney disease. *Diabetes care*. 2013;36(5):1215-1221.

29. Yokoyama H, Okudaira M, Otani T, Sato A, Miura J, Takaike H, Yamada H, Muto K, Uchigata Y, Ohashi Y: Higher incidence of diabetic nephropathy in type 2 than in type 1 diabetes in early-onset diabetes in Japan. *Kidney international*. 2000;58(1):302-311.

30. Simerville JA, Maxted WC, Pahira JJ: Urinalysis: a comprehensive review. *American Family Physician*. 2005;71(6):1153-1162.

31. Matulewicz RS, Meeks JJ: Blood in the Urine (Hematuria). *JAMA*. 2016;316(14):1508.

32. Muller L, Gorter K, Hak E, Goudzwaard W, Schellevis F, Hoepelman A, Rutten G: Increased risk of common infections in patients with type 1 and type 2 diabetes mellitus. *Clinical infectious diseases*. 2005;41:281-288.

33. Geerlings SE: Urinary tract infections in patients with diabetes mellitus: epidemiology, pathogenesis and treatment. *International journal of antimicrobial agents*. 2008;31S:S54-S57.

34. 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;8:129-136.

35. Stapleton A: Urinary tract infections in patients with diabetes. *The American journal of medicine*. 2002;113(1A):80S-84S.

36. Hoepelman AI, Meiland R, Geerlings SE: Pathogenesis and management of bacterial urinary tract infections in adult patients with diabetes mellitus. *International journal of antimicrobial agents*. 2003;22:S35-S43.

37. Couser WG, Remuzzi G, Mendis S, Tonelli M: The contribution of chronic kidney disease to the global burden of major noncommunicable diseases. *Kidney International*. 2011;80(12):1258-1270.

38. Barsoum RS: Chronic kidney disease in the developing world. *The New England journal of medicine*. 2006;354(10):997-999.

DOI: http://dx.doi.org/10.4314/ejhs.v27i8.9