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Prevalence and Determinants of Hyperuricemia Among Type 2 Diabetes Mellitus Patients at Selected Government Hospitals in Gurage Zone: A Cross- Sectional Study

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Abstract

Background: Hyperuricemia has a detrimental influence on glycemic regulation and contributes to unfavorable patient outcomes. Currently, elevated serum uric acid (SUA) is emerging as a potential marker for diabetes. This study aimed to explore the prevalence of hyperuricemia (HUA) and its determinants among patients with type 2 diabetes mellitus (T2DM).

Methodology: A cross-sectional study was conducted from March 1 to May 30, 2024, among T2DM patients at selected government hospitals in the Gurage Zone. SUA and several other metabolic and clinical parameters were examined. Multiple regression analysis was performed to identify risk factors independently associated with HUA.

Results: The prevalence of HUA in patients with DM was 36.9%. A notable incidence of HUA was evident among male individuals (57.1%). Significant associations (p<0.05) were noted between HUA and individuals aged between 40–50 years and those above 50 years, tobacco consumption, body mass index \geq 25 kg/m2, duration of diabetes between 5–10 years and over 10 years, poor glycemic control, decreased high-density lipoprotein levels, elevated triglycerides, and higher total cholesterol among patients diagnosed with T2DM.

Conclusion: The burden of HUA among T2DM patients is quite high, and HUA is associated with older age, longer duration of diabetes, poor glycemic control, high body mass index, cigarette smoking and dyslipidemia. This calls for regular screening of HUA in diabetic patients.

Keywords: Hyperuricemia, Type 2 Diabetes Mellitus and Determinants

Introduction

Diabetes mellitus is a metabolic disorder caused by genetic and environmental factors, leading to insulin insensitivity, insulin deficiency, and impaired biological functions [1]. Worldwide, an estimated 537 million adults between the ages of 20 and 79 will be affected by diabetes mellitus in 2021. However, 44.7% of this population remains unaware of their diabetes status [2]. According to a 2021 report from the International Diabetic Federation, the prevalence of undiagnosed diabetes in sub-Saharan Africa was 54%. This represents an increase of 4% compared with that in 2019, with the African region having the highest occurrence compared with other regions [2]. Ethiopia is estimated to constitute the fourth highest number of diabetes cases in the African region (1.3 million diabetes cases) [3]. SUA is the

final product of purine breakdown, originating from either endogenous metabolic processes or exogenous dietary intake [4]. Aberrant metabolism of uric acid (UA), compromised renal excretion, or a diet rich in purines can lead to HUA, resulting in elevated levels of UA in the bloodstream [5].

Currently, elevated SUA is emerging as a potential marker for diabetes [6]. Elevated SUA levels are associated with obesity, diabetes mellitus, hypertension, dyslipidemia, metabolic syndrome, liver dysfunction, cardiovascular disease, and chronic kidney disease. Insulin can increase renal reabsorption of uric acid, increasing SUA levels [7]. Furthermore, there is growing observational evidence that an elevated SUA level precedes the development of insulin resistance and diabetes [8]. Elevated SUA precedes insulin resistance and diabetes. High SUA levels increase all-cause and cardiovascular vascular disease mortality in individuals with diabetes [9]. Serum UA is correlated with body mass index (BMI), triglycerides, and the glomerular filtration rate in DM patients [10]. High intracellular SUA levels cause metabolic syndrome, hypertension, and DM; high extracellular SUA levels lead to gout and nephrolithiasis [11]. High UA increases diabetes risk. A 1-mg/dL increase in UA increases diabetes risk by 17% [12].

Emerging research also suggests that HUA may significantly impact lipid metabolism, potentially leading to dyslipidemia, which is characterized by an imbalance of lipids in the bloodstream [13]. Elevated UA levels are strongly linked to prediabetes on the basis of HbA1c criteria, highlighting the pivotal role of UA in disrupting glucose metabolism. A notable positive correlation was identified between dyslipidemia and serum UA levels [14]. Across different glycemic statuses and SUA quartiles, there is a notable increasing trend in mean low-density lipoprotein (LDL), total cholesterol (TC), and triglyceride (TG) levels, accompanied by a decreasing trend in high-density lipoprotein (HDL) levels [15]. SUA is not commonly assessed in our clinical setting; however, it serves as a crucial indicator of disease progression. HUA has a detrimental influence on glycemic regulation and contributes to unfavorable patient outcomes. There is currently a lack of established clinical protocols for managing asymptomatic HUA, both at local and global scales.

There is a lack of data on HUA in individuals with type 2 diabetes in sub-Saharan Africa, including associated factors. Standard care does not include monitoring uric acid in DM patients, despite its known impact. DM patients face complications that affect morbidity and mortality. The high DM prevalence in the Gurage Zone suggests poor management. No study has investigated HUA prevalence and determinants in T2DM patients in the Gurage Zone. This study aimed to determine HUA prevalence and determinants in T2DM patients in the Gurage Zone.

Methods and Materials

Study Design, Setting and Period

A hospital-based cross-sectional study was conducted from March 1 to May 30, 2024, 2020, at hospitals in the Gurage zone of central Ethiopia, 158 km south of Addis Ababa. The Gurage zone has three primary hospitals and one general hospital, all with chronic follow-up clinics for diabetes and hypertension. This study took place at the diabetic clinics of Agena General Hospital (AGH) (200 beds), Wolkite University Comprehensive Specialized Hospital (WKUSH) (150 beds), and Attat Hospital (AH) (180 beds). These hospitals were selected due to their diverse patient populations from densely populated urban and rural areas, along with their available resources and support. During the study, the monthly male patients diagnosed with T2DM were highest at AGH, followed by WKUSH and AH [10, 15-19]. A total of 95 T2DM patients were recruited from AGH, 77 from WKUSH, and 50 from AH over the five months.

Study Participants

All T2DM patients > 18 years old who were willing to participate in this study were included. Pregnant women, severely ill individuals and patients on drugs known to have an effect on UA levels, except for anti-diabetic therapy, and patients taking lipid-lowering drugs were excluded from the study.

Sample Size and Sampling Technique

The sample size for this study was determined by using a single population proportion formula. Two previous studies conducted in Ethiopia utilized the prevalence reported in Sudan (prevalence of 15.3%), with a 5% margin of error and a standard Z score of 1.96, corresponding to a 95% CI [16]. Finally, the sample size was computed as: n= (Z) 2 p (1-p)/d²; n= [(1.96) 2* 0.153*(1-0.153)]/ (0.05)² = 200

With a 10% nonresponse rate, the minimum sample size is 222. The specified hospitals were chosen because they received a high number of patients with different ethnic backgrounds from highly populated regions that had both urban and rural areas. In addition, available resources, approvals, and cooperation at these sites allowed the smooth progression of the study. A consecutive sampling technique was used to select all diabetic male patients who fulfilled the inclusion criteria until the desired sample size was reached.

Variables

Dependent Variables: HUA (Yes/No).

Independent variables: Socio-demographic factors: Age, educational status, monthly income, occupation, marital status.

Behavioral and lifestyle factors: cigarette smoking, physical activity, alcohol consumption, khat chewing. Baseline clinical and physical characteristics: anthropometric measurement, glycemic control, duration of diabetes, and lipid profile.

Data Collection

Data were collected through face-to-face interviews using a questionnaire. Participants were informed about the study's objective, and verbal and written consent was obtained. Socio-demographic characteristics and clinical data were collected by trained nurses via a semi-structured questionnaire. In addition, trained laboratory technologists collect and analyze blood samples. Anthropometric measurements (weight and height) were taken according to the WHO stepwise approach guidelines. Height was measured to the nearest 0.5 cm using a standiometer, and weight was recorded to the nearest 0.1 kg with the patient wearing light clothes via a balance. BMI was calculated as weight divided by height squared (kg/m2) [20].

Blood pressure was measured by nurses via an analog sphygmomanometer. Five milliliters of fasting venous blood samples were collected via a serum separator test tube following aseptic blood collection procedures. Serum glucose levels, lipid profiles and UA levels were measured via a COBAS 600 chemistry analyzer.

Operational Definition

Participants were classified as nonsmokers or smokers on the basis of their smoking status. Alcohol consumption was classified as "nondrinker" or "drinker" depending on whether the participants reported consuming alcohol. Exercise status was classified as "yes" if the patient followed the recommended level of regular physical activity, including walking. Income was recorded in the local currency and converted to US dollars.

Glycemic control: The level of glycemic control was assessed by obtaining HbA1C values. Participants were grouped into normal glycemic control (i.e., HbA1C \leq 7) and poor control (i.e., HbA1C > 7) [17]. Alcoholic: The daily alcohol amount that respondents consume was calculated considering the average alcohol percent (%/ml) of each drink multiplied by the volume (ml) of the drink and volumetric mass density (which is 0.8 g/ml). Accordingly, participants were explained to be alcoholic if they drank more than 12g of ethanol of alcohol per day in the past six months of the survey [18]. Cigarette smoking: Those who smoke any tobacco products daily are considered tobacco users [17].

Physical inactivity: Respondents who did not achieve the WHO recommendations of total physical activity (level less than 600 MET-minute/week) or reported practicing no physical activities were classified as inactive. Those who meet this criterion (>600 MET-minutes/week) are classified as physically active [18]. Khat chewer: if the patient reported khat chewing (kata adulis forsik). Body mass index (BMI): a person's weight in kilograms (kg) divided by his or her height in meters squared (m²). On the basis of the WHO's 2004 BMI classification, participants who have a BMI (kg/ m^2) < 18.5, 18.5–24.9, 25–29.9 or > 30 kg/m² were classified as underweight, within the normal range, overweight, or obese, respectively [20].

HUA: Patients with serum uric acid levels greater than 7.2 mg/dl in males and greater than 6.0 mg/dl in females[19].

Data Processing, Analysis and Quality Assurance

The questionnaire and checklist were reviewed by experts and translated into Amharic, followed by back-translation for consistency. To ensure data quality, facilitators and data collectors were trained, and the tool was pretested on 10% of participants. Data collection done with daily supervision of data collectors by investigators. Data were collected using the Amharic version, checked for consistency, errors, completeness, accuracy, and clarity before being entered into Epidata version 3.1. The data were then exported to SPSS version 20 for recoding, cleaning, and analysis. All continuous independent variables were categorized.

The outcome variable was dichotomized and coded as '0' and '1', representing those who have no and have HUA, respectively. Continuous data were checked for normality with the Shapiro–Wilk test and were found not to be normally distributed. Descriptive statistics like frequency, percentage, and measure of central tendency with their corresponding measure of dispersion were used to describe demographic and other variables. Tables', graphs, and texts were used to present the findings.

Furthermore, the binary logistic regression analysis was applied to identify factors associated with ED. Those variables with a p-value ≤ 0.25 in the bi-variable analysis were entered into the multivariable logistic regression model to control the possible effects of confounder/s and to identify the significant factors. According to the Hosmer and Lemeshow test, the model was found to be adequate. Likewise, prior to identifying the significant factors, the presence of multicollinearity problem was examined using the Variance Inflation Factor (VIF), and no variable was found to have that problem.

Result

Baseline and Sociodemographic Characteristics of the Study Participants. A total of 222 participants were included in this study. The majority of the participants were male (57.2%), and 40.1% were younger than 40 years. The participants' ages ranged from 19--70 years (mean age: 51.07 ± 14.34 years). Among the total patients, 27.0% were

smokers. Among the enrolled patients, only 32.9% had a normal BMI, with 35.6% being overweight and 31.5% being obese. Among the enrolled patients, 66.2% had poorly controlled DM, and a total of 82 patients (36.9%) met the HUA definition. Other baseline and sociodemographic characteristics are described in Table 1.

| Variables | Category | Frequency (%) | |
|---------------------|---------------------|---------------|------|
| Age group | < 40 years | 89 | 40.1 |
| | 40-50 years | 83 | 37.4 |
| | >50 years | 50 | 22.5 |
| | Mean=51.07 ± 14.34 | | |
| Gender | Male | 127 | 57.2 |
| | Female | 95 | 42.8 |
| Educational Status | No Formal Education | 72 | 32.4 |
| | Primary | 51 | 23.0 |
| | Secondary | 36 | 16.2 |
| | Diploma | 32 | 14.4 |
| | Degree and above | 31 | 14.0 |
| Occupational Status | Farmer | 77 | 34.7 |
| | Merchant | 54 | 24.3 |
| | Government employee | 58 | 26.1 |
| | Not employed | 33 | 14.9 |
| Physical activity | Yes | 93 | 41.9 |
| | No | 129 | 58.1 |
| Alcohol drink | Yes | 106 | 47.7 |
| | No | 116 | 52.3 |
| Smoking | Yes | 60 | 27.0 |
| | No | 162 | 73.0 |
| Khat Chewing | Yes | 113 | 50.9 |
| | No | 109 | 49.1 |

Table 1 : Baseline and Sociodemographic Characteristics of Adult Type 2 DM Patients (n = 222) Baseline Clinical and Physical Characteristics of the Patients

Approximately 40.1% of the patients had coexisting hypertension. Among the enrolled patients, 53.2% had total cholesterol \geq 200 mg/dL, 36.5% had LDL cholesterol > 160 mg/dL, 48.2% had HDL cholesterol < 40 mg/dL in males and < 50 mg/dL in females, and 53.6% had triglycerides \geq 150 mg/dL (Table 2).

| Clinical variables | Category | Frequency (%) | |
|-------------------------|-----------------------|---------------|------|
| BMI | Normal | 73 | 32.9 |
| | Over Weight | 79 | 35.6 |
| | Obese | 70 | 31.5 |
| Duration of diabetes | <5yrs | 90 | 40.5 |
| | 5-10yrs | 93 | 41.9 |
| | >10yrs | 39 | 17.6 |
| Uric Acid Status | Hyperuricemia | 82 | 36.9 |
| | Normal | 140 | 63.1 |
| Coexisting hypertension | Yes | 89 | 40.1 |
| | No | 133 | 59.9 |
| Glycemic control | Good (Hba1C \leq 7) | 75 | 33.8 |
| | Poor (Hba1C > 7) | 147 | 66.2 |
| HDL | Normal (>40 Mg/DI) | 115 | 51.8 |
| | Abnormal (<40 Mg/DI) | 107 | 48.2 |
| LDL | Normal (≤100 Mg/DI) | 141 | 63.5 |
| | Abnormal (>100 Mg/Dl) | 81 | 36.5 |
| ТС | Normal (<200 Mg/DI) | 104 | 46.8 |
| | Abnormal (≥200 Mg/DI) | 118 | 53.2 |

| TG | Normal (<150 Mg/DI) | 103 | 46.4 |
|----|----------------------|-----|------|
| | Abnormal (≥150 Mg/DI | 119 | 53.6 |

Table 2: Baseline Clinical and Physical Characteristics of Adult Type 2 DM Patients (n = 222)

Note: BMI: body mass index, LDL: low-density lipoprotein, HDL: high-density lipoprotein, TC: total cholesterol, TG: triglycerides

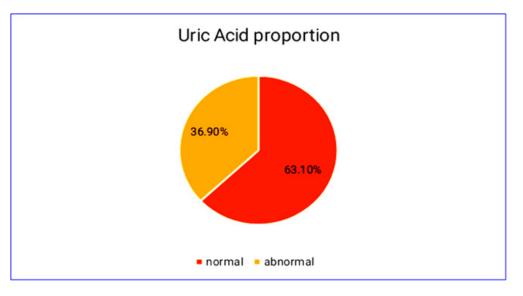


Figure 1: Serum Uric Acid Proportions of Adult T2DM Patients (n=222)

SUA Levels of the Study Participants According to Socio-Demographic Characteristics

Approximately 42.9% (60) of the study participants aged 40–50 years had HUA. There was a high prevalence of HUA among males (57.1%), with a BMI \geq 25 kg/m2 (81.4%). HUA was significantly associated with the age groups 40–50 years (P<0.001) and >50 years (p=.004), alcohol consumption (p=.002), cigarette smoking (p=.011), and a BMI greater than or equal to 25 kg/m2 (P<0.001). However, there were no significant associations between HUA and sex, occupation, educational status, physical activity, or khat chewing (S1Table 1).

SUA Levels of the Study Participants According to Lipid Profile

The prevalence rates of HUA according to the lipid profile are shown in Table 3 . Patients with hyper-triglyceridemia and reduced HDL cholesterol had a greater prevalence of HUA (32.5% vs 23.1%; p=0.008 and 33.7% vs. 24.3%; p=0.014, respectively).

| Variables | Category | HUA | | P-VALUE | |
|-----------|-----------------------|-----------|-----------|----------------|--|
| | | No n (%) | Yes n (%) | | |
| HDL | Normal (>40 mg/dl) | 61(74.4%) | 54(38.6%) | P<0.001 | |
| | Abnormal (<40 mg/dl) | 21(25.6%) | 86(61.4%) | | |
| LDL | Normal (<=100 mg/dl) | 67(81.7%) | 74(52.9%) | P<0.001 | |
| | Abnormal (>100 mg/dl) | 15(18.3%) | 66(47.1%) | | |
| ТС | normal (<200 mg/dl) | 66(80.5%) | 41(29.3%) | P<0.001 | |
| | abnormal (>200 mg/dl) | 16(19.5%) | 99(70.7%) | | |
| TG | normal (<150 mg/dl) | 54(65.9%) | 58(41.4%) | P<0.001 | |
| | abnormal (>150 mg/dl | 28(34.1%) | 82(58.6%) | | |

Table 3: Serum Uric Acid Levels of the Study Participants According to the Lipid Profile of Adult Type 2 DM Patients (n = 222)

Note: LDL: low-density lipoprotein; HDL: high-density lipoprotein; TC: total cholesterol; TG: triglycerides; HbA1c: hemoglobin A1c

Factors Associated With HUA

Independent variables with P values less than 0.25 in the binary logistic regression model were entered into the multivariate analysis model to identify independent predictor variables. According to this statistical analysis, T2DM patients aged 40--50 years were approximately 6 times (AOR=6.095, 95% CI: 1.572--23.638), p=0.009), and those aged above 50 years were approximately 71.4 times (AOR=71.385, 95% CI: 9.179--555.174, P<0.001) more likely to

develop HUA than those aged less than 40 years. T2DM patients who smoked cigarettes were 1.88 times (AOR=14.888, 95% CI: 1.285--18.588, p=0.020) more likely to develop HUA than those who did not smoke cigarettes. Those study subjects with a BMI \geq 25 kg/m2 during the study period were approximately 7.9 times (AOR=7.924, 95% CI: 2.005–31.316, p=0.003) more likely to develop HUA than those with a BMI <25 kg/m2. Moreover, lower high-density lipoprotein (p=0.010), higher triglyceride (p=0.013) and higher total cholesterol (p=0.014) levels were independently associated with HUA (Table 4).

| Variables | Category | COR (Lower-Upper) | AOR (Lower-Upper) | P Value |
|-------------------------|-----------------------|-------------------|---------------------|---------|
| Age group | < 40 years | 1 | 1 | 1 |
| | 40-50 years | 0.02(0.005103) | 6.10(1.572-23.638) | 0.009 |
| | >50 years | 0.11(0.024484) | 6.9(1.786-11.982) | P<0.001 |
| Coexisting hypertension | Yes | 1.62(0.917-2.859) | 1.70(0.596-4.868) | 0.320 |
| | No | 1 | 1 | 1 |
| Alcohol drink | Yes | 2.43(1.379-4.270) | 2.37(0.710-7.923) | 0.160 |
| | No | 1 | 1 | 1 |
| Smoking | Yes | 2.38(1.211-4.667) | 4.89(1.285-18.588) | 0.020 |
| | No | 1 | 1 | 1 |
| Khat Chewing | Yes | 1.56(0.902-2.703) | 1.44(0.511-4.025) | 0.493 |
| | No | 1 | 1 | 1 |
| BMI | <25 kg/m ² | 1 | 1 | 1 |
| | ≥25 kg/m ² | 0.25(0.126508) | 7.92(2.005-31.316) | 0.003 |
| Duration of diabetes | <5yrs | 1 | 1 | 1 |
| | 5-10yrs | 0.05(0.015185) | 8.14(2.624-25.270) | P<0.001 |
| | >10yrs | 0.24(0.068850) | 35.08(2.897-424.77) | 0.005 |
| Glycemic control | Good (HbA1c \leq 7) | 1 | 1 | 1 |
| | Poor (HbA1c > 7) | 195(0.108352) | 3.53(1.088-11.463) | .036 |
| HDL | Normal (>40 mg/dl) | 1 | 1 | 1 |
| | Abnormal (≥40 mg/dl) | 0.22(0.118394) | 5.21(1.483-18.282) | 0.010 |
| LDL | Normal (≤100 mg/dl) | 1 | 1 | 1 |
| | Abnormal (>100 mg/dl) | 0.25(0.131481) | 2.70(0.813-8.960) | 0.105 |
| тс | Normal (<200 mg/dl) | 1 | 1 | 1 |
| | Abnormal (≥200 mg/dl) | 0.10(0.052194) | 4.52(1.360-14.984) | 0.014 |
| TG | Normal (<150 mg/dl) | 1 | 1 | 1 |
| | Abnormal (≥150mg/dl) | 0.37(0.208647) | 4.32(1.354-13.776) | 0.013 |

Table 4: Multivariable Binary Logistic Regression for Hyperuricemia in Adult Type 2 DM Patients (N = 222)

Note: BMI: body mass index, LDL: low-density lipoprotein, HDL: high-density lipoprotein, TC: total cholesterol, TG: triglycerides, HbA1c: hemoglobin A1c.

Discussion

The prevalence of HUA in our study sample was 36.9%, which was comparable to that reported in studies performed in Iraq (39.47%) and Uganda (38.57%) [20, 21]. In contrast to the current findings, a lower prevalence of HUA was reported by Arersa et al in Ethiopia, and a higher prevalence of HUA was reported by Abdel et al in Tanzania (40%) [22, 23]. The difference in magnitude may be attributed to many factors, including diet and exercise, genetics, geographical location, and weather, as well as different cutoff values for SUA and exclusion criteria in different studies.

In this study, the odds of having HUA were approximately 6 and 7 times greater among participants aged 40--50 and >50 years, respectively, than among younger participants. Consistent findings were reported in a previous study by Woyesa et al [24]. One possible explanation for the link between older age and HUA is likely multifactorial and could involve a combination of biological, lifestyle, and disease-related factors. The probability of having HUA was 3.5 times greater in men with poor blood sugar than in those with well-controlled blood sugar. This result was also noted by Rashad et al [20]. The possible mechanism linking HUA to poorly controlled blood sugar in people with diabetes may be related to elevated blood sugar interfering with the reabsorption of uric acid in the kidneys, specifically in the proximal tubule portion of the nephron [25].

The present study revealed that patients with a duration of DM of 5–10 years were 8 times more likely to develop HUA than those with a duration of DM of \geq 10 years were 35 times more likely to develop HUA. This finding is supported by

a study conducted in Japan, which reported that patients with a duration of DM of ≥ 10 years were 3.96 times more likely to develop HUA than those with a duration of DM of < 10 years [22]. This may be because, during the later stages of DM, elevated UA acts as a pro-oxidant that results in chronic complications such as atherosclerosis [26]. The study participants who smoked independently were approximately 5 times more likely to develop HUA than those who did not smoke. Consistent findings were reported by a previous study conducted at the Ethiopian Public Health Institute, which demonstrated that smokers were 2.05 times more likely to develop HUA (AOR=2.05, 95% CI 1.01–4.19) [27]. The link between smoking and HUA in individuals with T2DM may result from smoking-induced oxidative stress, inflammation, and increased insulin resistance, which impair renal function and reduce uric acid clearance. These factors highlight the importance of smoking cessation in diabetes management.

This study revealed that participants with a higher BMI (\geq 25 kg/m²) had a 7.9-fold increased risk of HUA compared with those with a normal BMI, which aligns with findings from Tanzania and Ethiopia, where obesity was also linked to HUA in T2DM patients [22, 23]. This association likely stems from metabolic disturbances such as insulin resistance, which impair uric acid excretion, leading to uric acid accumulation and increased oxidative stress [28]. This combination increases the risk of renal impairment in T2DM patients [29]. In contrast, a Kenyan study revealed no significant BMI and HUA link, despite the prevalence of overweight and obesity [30].

Our study revealed a significant link between serum uric acid and lipid abnormalities, specifically high TC, low HDL (p=0.010), and elevated TG, in line with findings from Morocco [31]. Participants with high TG levels had a -fold increased risk of HUA. HUA and hypertriglyceridemia are suggested to be associated with insulin resistance syndrome [32]. The associations among insulin resistance, HUA, and hypertriglyceridemia are complicated. This might be expected because UA production is linked to glycolysis and because glycolysis is controlled by insulin [33]. UA was negatively correlated with serum HDL-C (p<0.05). This finding is consistent with the findings of Rho et al [34]. The mechanisms of this condition may be related to the relationship between decreased HDL-C levels and insulin resistance syndrome [35]. The limitations of this study were that, since this was a cross-sectional study, a causal-effect relationship could not be established. The questionnaire for this study was not validated or pilot tested. Data concerning patient khat chewing, alcohol use, and smoking habits were obtained from the patients themselves, which may have resulted in recall bias.

Conclusion

The burden of HUA among patients with T2DM is high, and it is associated with aging, cigarette smoking, high BMI, elevated TC, elevated TG, and lower HDL. This calls for regular screening of HUA in this population, as well as more studies dedicated to establishing the outcomes associated with HUA to develop guidelines on approaches to treatment. The implementation of strategies to cease cigarette smoking and improve BMI among patients is advised, given that most patients are overweight or obese.

Declarations

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Author Contributions

The authors made significant contributions to the manuscript. The authors have read and given their final approval of the version of the manuscript submitted for publication.

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Reference

- 1. American Diabetes Association. (2013). Diagnosis and classification of diabetes mellitus. *Diabetes care*, *36*(Supplement_1), S67-S74.
- Ogurtsova, K., Guariguata, L., Barengo, N. C., Ruiz, P. L. D., Sacre, J. W., Karuranga, S., ... & Magliano, D. J. (2022). IDF diabetes Atlas: Global estimates of undiagnosed diabetes in adults for 2021. *Diabetes research and clinical practice*, *183*, 109118.
- 3. Atlas, D. (2015). International diabetes federation. IDF Diabetes Atlas, 7th edn. Brussels, Belgium: *International Diabetes Federation*, 33(2).

- 4. McKeigue, P. M., Shah, B., & Marmot, M. G. (1991). Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *The Lancet*, *337*(8738), 382-386.
- 5. Tsushima, Y., Nishizawa, H., Tochino, Y., Nakatsuji, H., Sekimoto, R., Nagao, H., ... & Shimomura, I. (2013). Uric acid secretion from adipose tissue and its increase in obesity* Yu Tsushima1, 2, Hitoshi Nishizawa1, Yoshihiro Tochino1, Hideaki Nakatsuji1, Ryohei Sekimoto1, Hirofumi Nagao1, Takashi Shirakura2, Kenta Kato2, Keiichiro Imaizumi3, Hiroyuki Takahashi2, Mizuho Tamura2, Norikazu Maeda1, Tohru Funahashi4, and Iichiro Shimomura1.
- 6. Katsiki, N., Dimitriadis, G. D., & Mikhailidis, D. P. (2021). Serum uric acid and diabetes: from pathophysiology to cardiovascular disease. *Current pharmaceutical design*, *27*(16), 1941-1951.
- 7. Juraschek, S. P., McAdams-Demarco, M., Miller, E. R., Gelber, A. C., Maynard, J. W., Pankow, J. S., ... & Selvin, E. (2014). Temporal relationship between uric acid concentration and risk of diabetes in a community-based study population. *American journal of epidemiology*, *179*(6), 684-691.
- 8. Viazzi, F., Leoncini, G., Vercelli, M., Deferrari, G., & Pontremoli, R. (2011). Serum uric acid levels predict new-onset type 2 diabetes in hospitalized patients with primary hypertension: the MAGIC study. Diabetes care, 34(1), 126-128.
- 9. Li, B., Chen, L., Hu, X., Tan, T., Yang, J., Bao, W., & Rong, S. (2023). Association of serum uric acid with all-cause and cardiovascular mortality in diabetes. *Diabetes care*, *46*(2), 425-433.
- 10. Gaita, L., Timar, R., Lupascu, N., Roman, D., Albai, A., Potre, O., & Timar, B. (2019). The impact of hyperuricemia on cardiometabolic risk factors in patients with diabetes mellitus: a cross-sectional study. Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy, 2003-2010.
- 11. Singh, S. K., Singh, R., Singh, S. K., Iquebal, M. A., Jaiswal, S., & Rai, P. K. (2023). Uric acid and diabetes mellitus: an update. *Postgraduate medical journal*, *99*(1178), 1220-1225.
- 12. Kodama, S., Saito, K., Yachi, Y., Asumi, M., Sugawara, A., Totsuka, K., ... & Sone, H. (2009). Association between serum uric acid and development of type 2 diabetes. *Diabetes care*, *32*(9), 1737-1742.
- 13. Liu, F., Du, G. L., Song, N., Ma, Y. T., Li, X. M., Gao, X. M., & Yang, Y. N. (2020). Hyperuricemia and its association with adiposity and dyslipidemia in Northwest China: results from cardiovascular risk survey in Xinjiang (CRS 2008–2012). *Lipids in Health and Disease, 19,* 1-12.
- 14. Alqahtani, S. A. M., Awan, Z. A., Alasmary, M. Y., & Al Amoudi, S. M. (2022). Association between serum uric acid with diabetes and other biochemical markers. Journal of family medicine and primary care, 11(4), 1401-1409.
- 15. Shi, H., Liu, Y., Yang, D., Liang, P., Chen, C., Luan, H., & Shi, C. (2024). Inverted U-shaped associations between serum uric acid and fasting-plasma glucose level in non-diabetic, pre-diabetic, and diabetic adults: A population-based study in China. *Journal of Diabetes Investigation*, *15*(4), 483-490.
- 16. Mirghani, H. O. (2018). Hypertriglyceridemia, hyperuricemia, and anemia among sudanese patients with type 2 diabetes mellitus. *Indian J Basic Appl Med Res, 7*(3), 493-501.
- 17. Association GoAD. Standards of medical care in diabetes–2015: summary of revisions. Diabetes Care. 2015;38: S4.
- 18. Muturi, N. (2014). Alcohol consumption and reproductive health risks in rural Central Kenya. *Sexual & Reproductive Healthcare*, *5*(2), 41-46.
- 19. Sui, X., Church, T. S., Meriwether, R. A., Lobelo, F., & Blair, S. N. (2008). Uric acid and the development of metabolic syndrome in women and men. *Metabolism*, *57*(6), 845-852.
- 20. Rashad, B. H. (2024). Relationship Between Diabetes and Hyperuricemia in Zakho City–Kurdistan Region of Iraq: A Retrospective Cross-Sectional Study. *J Contemp Med Sci*/*Vo*1, *10*(3), 250-253.
- 21. Onchoke, V. B., Banturaki, A., Onyanga, N., Nganda, P., Munyambalu, D. K., Lagoro, C. A., ... & Muhumuza, J. (2023). Prevalence of hyperuricemia, associated factors and its effect on risk of coronary artery disease among out-patients with Diabetes Mellitus in Uganda.
- 22. Arersa, K. K., Wondimnew, T., Welde, M., & Husen, T. M. (2020). Prevalence and determinants of hyperuricemia in type 2 diabetes mellitus patients attending Jimma Medical Center, Southwestern Ethiopia, 2019. Diabetes, Metabolic Syndrome and Obesity, 2059-2067.
- 23. Abdel, K. A., Kalluvya, S. E., Sadiq, A. M., Ashir, A., & Masikini, P. I. (2024). Prevalence of Hyperuricemia and Associated Factors Among Patients with Type 2 Diabetes Mellitus in Northwestern Tanzania: A Cross-Sectional Study. Clinical Medicine Insights: *Endocrinology and Diabetes*, *17*, 11795514241274694.
- 24. Woyesa, S. B., Hirigo, A. T., & Wube, T. B. (2017). Hyperuricemia and metabolic syndrome in type 2 diabetes mellitus patients at Hawassa university comprehensive specialized hospital, South West Ethiopia. *BMC endocrine disorders*, 17, 1-8.
- 25. Tuomilehto, J., Zimmet, P., Wolf, E., Taylor, R., Ram, P., & King, H. (1988). Plasma uric acid level and its association with diabetes mellitus and some biologic parameters in a biracial population of Fiji. *American journal of epidemiology*, *127*(2), 321-336.
- 26. So, A., & Thorens, B. (2010). Uric acid transport and disease. *The Journal of clinical investigation*, *120*(6), 1791-1799.
- 27. Molla, M. D., Bekele, A., Melka, D. S., Teklemariam, M. D., Challa, F., Ayelign, B., ... & Geto, Z. (2021). Hyperuricemia and its associated factors among adult staff members of the Ethiopian Public Health Institute, Ethiopia. *International Journal of General Medicine*, 1437-1447.
- 28. Han, T., Meng, X., Shan, R., Zi, T., Li, Y., Ma, H., ... & Sun, C. (2018). Temporal relationship between hyperuricemia and obesity, and its association with future risk of type 2 diabetes. *International journal of obesity*, *42*(7), 1336-1344.
- 29. Li, Y., Fan, X., Li, C., Zhi, X., Peng, L., Han, H., & Sun, B. (2018). The relationships among hyperuricemia, body mass index and impaired renal function in type 2 diabetic patients. *Endocrine journal*, *65*(3), 281-290.

- 30. Shokat, M. (2018). Serum uric acid levels in patients with type 2 diabetes at Kenyatta national hospital (Doctoral dissertation, University of Nairobi).
- 31. Fennoun, H., Haraj, N. E., El Aziz, S., Bensbaa, S., & Chadli, A. (2020). Risk factors associated with hyperuricemia in patients with diabetes type 2: about 190 cases. *Diabetes Research: Open Access, 2020*(1), 12.
- 32. Bo, S., Cavallo-Perin, P., Gentile, L., Repetti, E., & Pagano, G. (2001). Low HDL-cholesterol: a component of the metabolic syndrome only in the presence of fasting hypertriglyceridemia in type 2 diabetic patients. *DIABETES AND METABOLISM*, 27(1), 31-35.
- 33. Mundhe, S. A., & Mhasde, D. R. (2016). The study of prevalence of hyperuricemia and metabolic syndrome in type 2 diabetes mellitus. *Int J Adv Med*, *3*(2), 241-249.
- 34. Rho, Y. H., Choi, S. J., Lee, Y. H., Ji, J. D., Choi, K. M., Baik, S. H., ... & Song, G. G. (2005). The prevalence of metabolic syndrome in patients with gout: a multicenter study. *Journal of Korean medical science*, 20(6), 1029.
- 35. Schmidt, M. I., Watson, R. L., Duncan, B. B., Metcalf, P., Brancati, F. L., Sharrett, A. R., ... & Atherosclerosis Risk in Communities Study Investigators. (1996). Clustering of dyslipidemia, hyperuricemia, diabetes, and hypertension and its association with fasting insulin and central and overall obesity in a general population. *Metabolism*, 45(6), 699-706.