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Evaluation of serum uric acid to creatinine ratio as a biomarker in type 2 diabetes mellitus in Sana'a city, Yemen

Hamid Moh. Al-gabr

han-chin@hotmail.com

Albaydha University

Ghadeer M. Al-shawsh

Azal university for human devlopment

Ahmed A. Razoom

Azal university for human devlopment

Sondus Y. Al-faqeeh

Azal university for human devlopment

Maha J. Mahsoon

Azal university for human devlopment

Aya A. Anam

Azal university for human devlopment

Nada S. Al-sharif

Azal university for human devlopment

Sumaia A. Al-yafeai

Azal university for human devlopment

Laila M. Al-qutaibi

Azal university for human devlopment

Afaf O. Al-hadad

Azal university for human devlopment

Aisha A. shaiea

Azal university for human devlopment

Marwah M. Al-Ezy

Azal university for human devlopment

Rowaida H. Al-Moqbeli

Azal university for human devlopment

Aseel H. Aboassaj

Azal university for human devlopment

Moneer M. Al-anss

Azal university for human devlopment

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Abstract

Purpose

This study has been conducted in Yemen on the evaluation of serum uric acid to creatinine ratio in diabetes patients. and aim’s is to evaluate serum uric acid to creatinine ratio as a biomarker in type 2 diabetes mellitus in Sana'a city, Yemen.

Method

This comparative cross-sectional study, conducted at Al-Thora Modern General Hospital and Republican Teaching Hospital (Yemen), investigated the relationship between serum uric acid, creatinine, and renal function in 100 Yemeni patients with T2DM compared to 25 healthy controls. Participants were stratified into five cohorts: four therapeutic groups (glycemic regulator monotherapy, hypoglycemic agent monotherapy, combination therapy, and insulin therapy) and one non-diabetic control group. Statistical analysis using (SPSS).

Results

most favorable outcomes: with significantly lower HbA1c (8.73%) and uric acid levels (6.27 mg/dL). Then monotherapy, Insulin therapy demonstrated poorest glycemic control (HbA1c: 9.7%) along with elevated uric acid (9.5 mg/dL), whereas dietary intervention achieved moderate HbA1c reduction (7.65%) but showed variable creatinine (0.7 mg/dL) and uric acid (6.49 mg/dL) concentrations.

Conclusion

Notably, the serum uric acid-to-creatinine ratio emerged as a sensitive predictor of early renal impairment in diabetic patients, particularly within the insulin-treated cohort across both genders.

Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by sustained hyperglycemia resulting from defects in insulin secretion, insulin action, or both. It affects over 537 million adults globally, with rising prevalence worldwide [1] Type 2 diabetes (T2DM) accounts for more than 90% of all diabetes cases [2] and is associated with more than one million deaths annually, making it the seventh leading cause of death globally [3]. Chronic hyperglycemia damages multiple organ systems including the cardiovascular, renal, nervous, and visual systems leading to progressive dysfunction and failure [4]. Several forms of diabetes exist, Type 1 results from autoimmune destruction of pancreatic beta-cells, causing absolute insulin deficiency. The most common form is Type 2, arises from insulin resistance coupled with relative insulin deficiency. Other variants include gestational diabetes, latent autoimmune

diabetes in adults (LADA), and maturity onset diabetes of the young (MODY), a rare inherited form. The etiology of diabetes is multifactorial involving genetic predisposition, sedentary lifestyle, obesity, physical inactivity, and environmental exposures [5, 6, 7]. Family history and specific gene mutations (e.g., MODY) increase risk [8]. Lifestyle factors such as poor diet, obesity, and lack of exercise promote insulin resistance [9]. Medical conditions like polycystic ovary syndrome (PCOS), Cushing's syndrome, and thyroid disorders also contribute, as do environmental factors including air pollution, pesticide exposure, and vitamin D deficiency [10, 11]. Obesity is the single most important modifiable risk factor for T2DM, with nearly 90% of patients being overweight or obese [12, 19]. T2DM reduces life expectancy by 8–10 years, is challenging to treat, and imposes a substantial economic burden. Its prevalence exceeds 600 million worldwide, with projections reaching 642 million by 2040 [13, 14, 15, 16]. Incidence varies geographically and across ethnic groups; Asian populations show higher rates than Caucasians [19, 20, 21, 22], while the highest risk is observed in Black/African American populations [23]. In the Middle East prevalence is notably high: Yemen 10.4%, Saudi Arabia 23.7%, and Jordan 17% [24]. Many cases remain undiagnosed for years because hyperglycemia develops gradually without noticeable symptoms [25, 26, 27].

Hyperuricemia has emerged as a potential risk factor for cardiovascular disease, chronic kidney disease (CKD), and metabolic syndrome [28, 29, 30, 31]. Conversely, diabetes, obesity, and fatty liver disease may promote hyperuricemia [32]. CKD affects approximately 850 million people worldwide, accounting for 1.2 million deaths in 2017 [33, 34]. Diabetic kidney disease (DKD) occurs in about one-third of T2DM patients, largely due to microvascular damage, hypertension, and age-related nephron loss. Elevated serum uric acid and creatinine are frequently observed in diabetic patients [35]. Recent evidence suggests that the serum uric acid-to-creatinine ratio (SUA/SCr) may predict CKD incidence more accurately than uric acid alone in T2DM patients [36] and could serve as a biomarker for metabolic syndrome [46]. However, data on SUA/SCr in hypertensive or diabetic populations remain limited. Therefore, this study aimed to evaluate the SUA/SCr ratio as a biomarker in T2DM patients in Sana'a city, Yemen

Materials and Methods

Study Design:

This is a comparative Cross-sectional study aimed at investigating the evaluation of serum uric acid to creatinine ratio as a biomarker in T2DM. The study was conducted in hospitals in Sana'a, Yemen, including the Republican Teaching Hospital Authority and Al-Thawra General Model Hospital during September- October 2024 at the outpatient diabetes clinics (OPD) for one time. These hospitals were chosen due to the high number of diabetic patients visiting for treatment. And the study population consisted of 125 participants the samples size was calculated using G-power software (version 3.1) based on an expected samples effect size (f = 0.3) for ANOVA, with an α error of 0.5 and power (1-β) of 80%, resulting in a required minimum sample of 125 participants participant were divided into five groups:

- Group 1: T2DM Patients using glycemic regulator monotherapy.
- Group 2: T2DM Patients on combined glycemic regulators and hypoglycemic agents.
- Group 3: T2DM Patients on insulin therapy.
- Group 4: T2DM Patients managing their condition through diet and lifestyle only.
- Group 5: Age- and sex matched healthy adults with no history of diabetes or kidney disease as a control group

Data were collected using a questionnaire which included both closed-ended (yes/no) which include 10 questions and open-ended which include 5 questions to capture quantitative and qualitative data. The questionnaire consisted of 15 core questions divided into three domains:

1. Lifestyle Factors:

- Smoking status (Q3: yes/No).
- Physical activity (Q4: yes/No).
- Dietary regimen (Q6: yes/No with open-ended follow-up: Describe your diet).

2. Medical History:

- Diagnosis of type 2 diabetes (Q1).
- Family history of diabetes (Q2).
- History of kidney disease (Q5).
- Confirmation of HbA1c and creatinine/uric acid testing (Q7–Q8).

3. Treatment Adherence:

- Current diabetes medications (Q13, open-ended: "What type of medication do you use?").
- Use of uric acid/creatinine-lowering drugs (Q9–Q10).

Equipment and chemicals:

Centrifuge model CGOLDENWALL 80 – 2 Electric Lab Centrifuge (3500 rpm, 12x20 mL capacity, Spectrophotometer: V-1000 Spectrophotometer (AOE Instruments, Shanghai, China), Uric acid measurement: Enzymatic uricase method (Química Clínica Aplicada S.A., Spain), Creatinine measurement: Jaffe kinetic method (Química Clínica Aplicada S.A., Spain). All the chemicals and equipment were brushed from local market in Sana’a.

Sampling and Processing:

Sample volume 3 mL of blood was collected from each participant, but before starting the blood collection process, it is essential to clean the surface of hand and Blood Withdrawal is drawn at a moderate pace not too fast or too slow to avoid hemolysis (destruction of red blood cells), which can affect test results. After collection, blood samples were left at room temperature to clot. They were then centrifuged at 3500 rpm for 15 minutes to separate the serum. Serum-separating tubes (SSTs) with a gel barrier were used to prevent mixing and allow for temporary storage without affecting results. The separated serum was transferred to Eppendorf tubes and stored at 2–8°C until analysis for Uric Acid and Creatinine.

Measurement of Serum Uric Acid:

The method which used for measurement of uric acid follow the kits manual: 3 mL of blood was collected in SSTs and centrifuged after clotting at (3500 rpm) for 15 minutes. Serum was separated and stored in Eppendorf tubes at (2–8°C). the uricase buffer solution was prepared (1 mL of reagent per tube). And in a test tube, mix uricase buffer solution with (20 µL) of serum or standard uric acid solution to calculate the STD absorbance. Then Incubated at room temperature for 10 minutes then read the sample absorbance “End-point reaction”. And measure absorbance at 505 nm using a spectrophotometer.

$$\text{Using this formula: Concentration of sample } = \frac{Sample\ Absorbance}{Standard\ Absorbance} \times 5 = mg\ of\ uric\ acid/dl$$

Measurement of Serum Creatinine: :

The method which used for measurement of creatinine follow the kits manual:3 mL of blood was collected and centrifuged after clotting at (3500 rpm) for 15 minutes.

The serum was separated and stored in Eppendorf tubes. And the Picric acid solution (1.2% w/v) and alkaline buffer (0.75 N sodium hydroxide) were prepared. 1 mL alkaline buffer mix with (100 µL) of serum or standard creatinine solution, incubated at room temperature, and the first absorbance is measured at 20 seconds (initial reading). A second absorbance is taken after 80 seconds. The actual absorbance change (ΔA), representing the creatinine concentration, is calculated by subtracting the absorbance at 20 seconds from the absorbance at 80 seconds “kinetic reaction”: $\Delta A = A_{80\ sec} - A_{20\ sec}$. using Spectrophotometric absorbance at 510 nm, and calculation using :

$$\text{Concentration of sample} = \frac{\Delta\ Ab\ of\ Sample}{\Delta\ Ab\ of\ Standard} \times 2 = mg\ of\ creatinine/dl$$

Calculation of Serum Uric Acid to Creatinine Ratio (SUA/Cr)

The (SUA/Cr) has emerged as a potential biomarker for detecting early renal alterations in patients with (T2DM). Since both uric acid and creatinine are influenced by renal function, calculating their ratio may enhance the sensitivity of detecting subclinical kidney dysfunction. In this study, the SUA/Cr ratio was determined in diabetic patients receiving different therapeutic approaches, as well as in healthy controls, to evaluate its utility as an early indicator of renal impairment.

The SUA/Cr ratio was calculated using the following formula:

$$\mathrm{SUA/Cr} = \frac{Serum\ Uric\ acid\ (\frac{mg}{dL})}{Serum\ creatinine\ (\frac{mg}{dL})}$$

Statistical Analysis

Statistical analysis was performed using SPSS (IBM- SPSS Statistics Version 21). Data were summarized as mean count ± standard deviation (SD) for each study group. The comparison of means across the five groups were performed using one-way analysis of variance (ANOVA) for normally distributed data (glycemic regulator, combination therapy of glycemic regulator and hypoglycemic agent, Insulin, Diet, Control) followed by T-Test for pairwise comparisons. In addition to numerical summaries, graphical representations were also included to visualize group differences in serum uric acid, serum creatinine, and SUA/Cr ratio. As well as, Pearson's correlation coefficient was used to assess the correlation variables between the SUA/Cr ratio, and other continuous variables (HbA1c, FBG). ($p \leq 0.05$) was considered statistically significant.

Ethical Considerations

The study was approved by the institutional ethics committee of Azal University for human development (Ref. No. 24/113 – 10/2024) in accordance with institutional ethical guidelines, and informed consent was obtained from all participants before inclusion in the study. All methods were performed in accordance with the relevant guidelines and regulation of the Yemeni Ministry of Health and the institutional ethical standards.

Results and discussion

This comparative Cross-sectional study analyzed 125 participants: 100 individuals with T2DM sampled as randomly and 25 healthy controls. Participants were systematically stratified into five matched (n = 25 per group): Glycemic regulator (n = 25), Combination therapy of glycemic regulator and hypoglycemic agent (n = 25), Insulin (n = 25), Diet (n = 25), and Control (n = 25) groups. Baseline demographic characteristics, age distribution, dietary patterns, and key biochemical parameters are comprehensively presented in Table 1, providing essential context for subsequent comparative analyses.

Table (1): Baseline Characteristics and Primary Outcomes of Study Groups

Glucose (fasting blood glucose) level:

Table (2) showed the mean glucose level in glycemic regulator was 202 ± 58 mg/dL, with values ranging from 96 mg/dL to 297 mg/dL. This broad range reflects a considerable variation in glycemic stability among patients. Elevated glucose levels in some individuals may be associated with poor dietary habits, physical inactivity, or insufficient dosage of hypoglycemic agents. The insulin group revealed significant differences in glucose levels across the five groups 232 ± 70 mg/dL, with a range spanning from 131.24 mg/dL to 383.8 mg/dL. This considerable variability suggests that while some patients maintained acceptable glycemic levels, others experienced marked hyperglycemia. Such fluctuations may be attributed to inconsistencies in insulin dosing, timing of administration, dietary habits, or varying degrees of treatment compliance. As well as, some patients were smoking and effect of the levels of diabetic [6]. These results were disagreed with the studies of [37] **Čaušević et al, 2010** which the found the level of glucose was in men higher than women. While in this study the result found the men higher than women in glycemic regulator and diets group, but the others group showed the women higher than men.

Table (2) The level of glucose in samples (mg/dL)

HbA1c level:

The results in table (3) showed that HbA1c level in this group glycemic regulator was 8.47 ± 2.07%, indicating moderate glycemic control. However, the maximum recorded value reached 12.5%, which is well above the recommended target for diabetic patients (typically < 7%). This wide range suggests that while some patients maintained fair glycemic control, others experienced significant dysregulation. Possible contributing factors include inconsistent medication adherence or varying individual responses to treatment.

In Insulin group the mean HbA1c level was 9.7 ± 2.5%, reflecting moderate glycemic control across the group. However, the maximum recorded value of 15.0% highlights the presence of poorly controlled diabetes in some individuals. This could stem from factors such as severe insulin resistance, irregular insulin administration, or poor adherence to treatment protocols. The results agree with the study [38] (**Xing et al, 2022**) which they found stronger correlations were found in participants with a body mass and the level of (HbA1c) > 7% or female. As well as, [39] **Smith et al. (2020)** also found that insulin alone without concurrent lifestyle changes may be insufficient to achieve tight glycemic targets, consistent with our observations. The Diet group exhibited significantly elevated HbA1c which supports the conclusions of [40] **Minari et al. (2023)** in The Role of Diet in the Management of Type 2 Diabetes.

Table (3) The level of (HbA1c) in samples (mg/dL)

Creatinine level:

The average creatinine level in glycemic regulator was 0.92 ± 0.37 mg/dL, with the highest value recorded at 1.67 mg/dL. While the overall mean falls within the normal range, the elevated values in a

subset of patients raise concerns about possible renal impairment. Such elevations may be linked to long-term hyperglycemia and the onset of diabetic nephropathy.

The mean creatinine level in insulin group was 0.8 ± 0.30 mg/dL, with a peak value of 1.36 mg/dL. Although generally within the upper bounds of the normal range, the elevated readings in some cases may indicate early signs of renal impairment, possibly aggravated by the chronic metabolic burden of diabetes or long-term insulin use. The lowest mean creatinine levels were observed in the diet group, which may suggest improved renal function potentially linked to reduced protein consumption. However, this interpretation should be approached cautiously, given the presence of elevated creatinine values in some individuals. The drugs group had revealed significant differences levels across the five groups ($F = 2.84$, $p < 0.032$) Post-hoc pairwise comparisons using independent t-tests with Bonferroni correction showed that the drug group had significantly higher creatinine (1.04 ± 0.3 mg/dL) compared to the diet group (0.7 ± 0.31mg/dL), pointing to potential ongoing kidney stress despite improved glycemic control. While not directly addressed in the included references, these finding echoes [39] **Smith et al. (2020)** assertion that poor glycemic control even under insulin therapy can contribute to long-term renal complications.

Table (4) The level of Creatinine in samples (mg/dL)

Uric acid level:

In table (5) the results showed one patient exhibited an abnormally high uric acid level (38.6 mg/dL), which significantly exceeded the typical range for this group. Possible explanations include: Pre-analytical error, such as sample hemolysis or improper handling.

Undiagnosed metabolic disorder, such as Lesch-Nyhan syndrome or a similar purine metabolism defect. Concomitant use of diuretics, which can increase serum uric acid levels.

Due to the extreme nature of this outlier, the case was excluded from the calculation of group means to avoid skewing the data. However, clinical follow-up is strongly recommended, as this result may indicate a serious underlying condition.

One-way ANOVA revealed significant differences in serum uric acid levels across the five groups ($F = 8.42$, $p < 0.001$). Post-hoc pairwise comparisons using independent t-tests with Bonferroni correction showed that the insulin group had significantly higher uric acid levels (7.95 ± 5.03 mg/dL) compared to the control group (6.27 ± 2.44 mg/dL, $p < 0.001$) and the combination therapy group (7.82 ± 5.34 mg/dL, $p = 0.002$). No significant difference was observed between the diet and control groups ($p = 0.53$). Nevertheless, the outlier value of 38.6 mg/dL in one patient is markedly abnormal and indicative of severe hyperuricemia, warranting further investigation and possibly targeted pharmacological intervention. The diets and control group showed the lower level than the other group which showed (6.3, 6.49 mg/dL) respectively. As well as, the results showed between these two group diets and control no significant difference. The results indicated the group insulin, drugs, glycemic regulator the most group

effect of uric acid on kidney. In healthy normotensive individuals, serum uric acid level is an independent risk factor for decreased kidney function this finding agrees with [41, 42].

This result was agreed with the study of [42] **Bellomo et al, (2010)** which they found no significant difference between both women and men.

Table (5) The level of Uric acid in samples (mg/dL)

Ratio of uric acid to creatinine:

The table (6) showed that the results indicate the serum uric acid Upon analyzing the (SUA/Cr) across the five study groups, the highest ratio was found in the Insulin group (12.95), exceeding that of the Control group (7.64). This suggests a possible impact of insulin therapy on increasing uric acid levels or reducing its renal excretion, consistent with findings from [43]. In contrast, the Regulator and Reducer group showed the lowest ratio (6.35), indicating a strong therapeutic effect in lowering SUA/Cr and improving renal function. The Diet group exhibited a relatively high ratio (9.31), highlighting the limited effectiveness of diet alone in controlling renal biomarkers. The Regulator-only group presented a moderate effect (8.95), but was less effective compared to the combination treatment. These findings align with studies such as [36] **Chunlei et al. (2019)**, which emphasized the predictive value of SUA/Cr as an independent and non-invasive indicator of kidney function decline in type 2 diabetic patients, particularly in resource-limited settings like Yemen. As well as, to comparing the results between male and female, the results in figures (1,2,3) indicated the male were higher in all group than female except the diet group showed the female were higher than male. The studies of [44, 45] **Chen et al, (2022)** and **Mu et al., (2025)** founded UA/SCr is an independent risk factor for DKD in patients with T2DM and may be helpful for identifying normoalbuminuric DKD. In this study the results showed the ratio of SUA/Cr was high in man than women except in diet group.

Table (6): The ratio of uric acid to creatinine of the diabetic patients.

Figure (1): The ratio of uric acid to creatinine of the male diabetic patients.

Figure (2): The ratio of uric acid to creatinine of the female diabetic patients.

Figure (3): The ratio of uric acid to creatinine between male and female

Conclusion

This study provides novel data on the uric acid-to-creatinine ratio across distinct subgroups of type 2 diabetes mellitus (T2DM) patients, representing, to the authors' knowledge is the first the first such investigation. Its significance lies in establishing specific levels of uric acid and creatinine in different group comparing with control. A key finding was the uric acid to creatinine ratio was generally higher in male patients than in female. However, this pattern was reversed in the diet-managed group, where females exhibited a higher ratio. This divergence is likely attributable to differing dietary habits between

genders within this subgroup. Notably, the diet-managed group showed significant differences compared to other diabetic groups.

The (SUA/Cr) shows promise as a potential indicator may help detect early renal dysfunction or metabolic abnormalities before more significant damage occurs. However, it must be emphasized that dietary management should not be used as the sole treatment for T2DM unless under strict supervision by a clinical nutritionist or endocrinologist. The inclusion of a healthy, non-diabetic control group with biochemical markers (HbA1c, glucose, creatinine, and uric acid) within normal ranges provided a crucial reference point. The stark contrast between the control group and all diabetic groups strongly reinforces the metabolic and renal impairments associated with diabetes. These findings underscore the importance of individualized treatment strategies and suggest that the SUA/Cr ratio could be a useful adjunctive marker for monitoring renal risk in T2DM. Further prospective studies are needed to validate its predictive value and to explore the mechanisms underlying the observed differences between treatment groups.

Abbreviations

DM:Diabetes mellitus

T2DM: Type 2 Diabetes mellitus

MODY: Maturity Onset Diabetes of the Young

PCOS: polycystic ovary syndrome

ASCVD: atherosclerotic cardiovascular disease

AH: arterial hypertension

CKD: Chronic Kidney Disease

DKD: Diabetic kidney disease

Declarations

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J. Mahsoon**;** Aya A. Anam; Nada S. Al- sharif; Sumaia A. Al- yafeai; Laila M. Al-qutaibi ; Afaf O. Al-hadad ; Aisha A. shaiea; Marwah M. Al- Ezy; Rowaida H. Al- Moqbeli; Aseel H. Aboassaj; Moneer M. Al-anss,collected the samples, conducted the practical experiments, and also contributed to reviewed the manuscript.

Declarations Conflict of interest: The authors declare no competing interests.

Data availability: the data that support the findings of this study are available from the corresponding author upon reasonable request. Due to the ethical restrictions and the fact that patient consent for public data sharing was not obtained, the raw data are not publicly available. Any request for data sharing will be considered on a case-by-case basis, and data will be provided in de-identified form to protect patient privacy.

References

1. Saeedi, P., Petersohn, I., Salpea, P., Malanda, B., Karuranga, S., Unwin, N., Colagiuri, S., Guariguata, L., Motala, A. A., Ogurtsova, K. J. D. R. & Practice, C. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas. 157**,** 107843 (2019).
2. Sun, H., Saeedi, P., Karuranga, S., Pinkepank, M., Ogurtsova, K., Duncan, B. B., Stein, C., Basit, A., Chan, J. C., Mbanya, J. C. J. D. R. & Practice, C. IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. 183**,** 109119 (2022).
3. Khan, M.A. B., & Hashim, M. J. et al., Epidemiology of Type 2 Diabetes–Global Burden of Disease and Forecasted Trends. Journal of Epidemiology and Global Health, 10(1), 107-111 (2020)
4. Siddiqui, I., Mirza, M B., Nabeel, A K., Environmental and Lifestyle Determinants of Type 2 Diabetes Mellitus. Research square. 2024 (1) 1-36. https://doi.org/10.21203/rs.3.rs-4738489/v1 (2024).
5. Madhuchhanda Maity and Dr. Abha Kumari. Understanding Diabetes Mellitus: A Descriptive Study of Its Types, Causes and Health Impacts. Bharati International Journal Of Multidisciplinary Research & Development.-(3):6. https://doi.org/10.70798/Bijmrd/03060005 (2025).
6. Khan M, Jaiswal A, Wandile B. A Comprehensive Review of Modifiable Cardiovascular Risk Factors and Genetic Influences in Dementia Prevention. Cureus 15(11): e48430. https://DOI.10.7759/cureus.48430 (2023)
7. Fang L, Sheng H, Tan Y and Zhang Q.. Prevalence of diabetes in the USA from the perspective of demographic characteristics, physical indicators and living habits based on NHANES 2009-2018. Front. Endocrinol. 14:1088882. doi:10.3389/fendo.2023.1088882 (2023)
8. Alam,U.; Asghar, O.; Azmi, S.; Malik, R.A. Chapter 15—General aspects of diabetes mellitus. In Handbook of Clinical Neurology; Zochodne, D.W., Malik, R.A., Eds.; Diabetes and the Nervous System; Elsevier: Amsterdam, The Netherlands, 2014; Volume 126, pp. 211–222. Available online: https://www.sciencedirect.com/science/article/pii/B9780444534804000151 (2014).

9. Lin, X.; Li, H. 2021. Obesity: Epidemiology, Pathophysiology, and Therapeutics. Front. Endocrinol. 2021, 12, 706978. [7] Fang L, Sheng H, Tan Y and Zhang Q. Prevalence of diabetes in the USA from the perspective of demographic characteristics, physical indicators and living habits based on NHANES 2009-2018. Front. Endocrinol. 14:1088882. doi:10.3389/fendo.2023.1088882 (2023).
10. Beulens, Joline W. J., MariaG.M.Pinho, TaymaraC.Abreu, Nicole R. den Braver, et al. Environmental risk factors of type 2 diabetes an exposome approach. Diabetologia (2022) 65:263–274. https://doi.org/10.1007/s00125-021-05618-w .(2022)
11. [11] Khalil, W.J.; Akeblersane, M.; Khan, A.S.; Moin, A.S.M.; Butler, A.E. Environmental Pollution and the Risk of Developing Metabolic Disorders: Obesity and Diabetes. Int. J. Mol. Sci. 2023, 24, 8870. https://doi.org/10.3390/ijms24108870 (2023).
12. Mohajan, D., and Mohajan, H K. Obesity and Its Related Diseases: A New Escalating Alarming in Global Health. journal of Innovations in Medical Research. .2023(2), 3. https://doi:10.56397/JIMR/2023.03.04 (2023).
13. Joline W. J. B., MariaG.M.P., TaymaraC.A., et al., Environmental risk factors of type 2 diabetes an exposome approach. Diabetologia 65:263–274. https://doi.org/10.1007/s00125-021-05618-w (2022).
14. IDF. IDF Diabetes Atlas, 10th Edition Committee. IDF Diabetes Atlas (International Diabetes Federation, (2021).
15. Misra Shivani , Naomi Holman, Emma Barron, Peter Knighton, Justin Warner, Partha Kar, Bob Young, Jonathan Valabhji. Characteristics and care of young people with type 2 diabetes included in the national diabetes audit datasets for England. Diabetic Medicine. 2023;40:e14940. https://doi.org/10.1111/dme.14940 (2023).
16. Zakir M, Ahuja N, Surksha M, et al.. Cardiovascular Complications of Diabetes: From Microvascular to Macrovascular Pathways. Cureus 15(9): e45835. https://DOI 10.7759/cureus.45835 (2023).
17. Pardhan, S. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021 (2023).
18. Yahr, J., Thomas, G., Calle, J., & Taliercio, J. J. Resistant Hypertension: A Stepwise Approach. Cleveland Clinic Journal of Medicine, 90(2), 115-125 (2023)
19. WHO. Diabetes Atlas, 10th ed. Geneva: World Health Organization, (2021).
20. Yao X, Zhang J, Zhang X, Jiang T, Zhang Y, Dai F, Hu H and Zhang Q. Age at diagnosis, diabetes duration and the risk of cardiovascular disease in patients with diabetes mellitus: a cross-sectional study. Front. Endocrinol. 14:1131395. https://doi:10.3389/fendo.2023.1131395 (2023)
21. Calvin, K. , Venkat Narayan, K.M., Juliana, C. N., Chan,Prabhat Jha and Baiju R. Shah**.** Pathophysiology, phenotypes and management of type 2 diabetes mellitus in Indian and Chinese populations. Nature Reviews- Endocrinology. V:18, 413-432. https://doi.org/10.1038/s41574-022-00669-4 (2022).
22. Goff LM, Ladwa M, Hakim O, Bello O. Ethnic distinctions in the pathophysiology of type 2 diabetes: a focus on black African-Caribbean populations. Proc Nutr Soc; 79(2): 184-93 (2020).

23. Lu, Brian, Peng Li, Andrew B. Crouse, Tiffany Grimes, Matthew Might, Fernando Ovalle, and Anath Shalev. Data-driven Cluster Analysis Reveals Increased Risk for Severe Insulin-deficient Diabetes in Black/African Americans. The Journal of Clinical Endocrinology & Metabolism, 110, 387–395. https://doi.org/10.1210/clinem/dgae516 (2025).
24. Saghir, Sultan AM, Abdullah, EA Alhariri, Sameer A Alkubati, Amer A Almiamn, Saleem H Aladaileh, Naelah A Alyousefi. Factors associated with poor glycemic control among type-2 diabetes mellitus patients in Yemen. Tropical Journal of Pharmaceutical Research July 2019; 18 (7): 1539-1546. http://dx.doi.org/10.4314/tjpr.v18i7.26 (2019).
25. Yoshino, M., Kayser, B. D., Yoshino, J., Stein, R. I., Reeds, D., Eagon, J. C., Eckhouse, S. R., Watrous, J. D., Jain, M., Knight, R., Schechtman, K., Patterson, B. W. & Klein, S. Effects of Diet versus Gastric Bypass on Metabolic Function in Diabetes. *New England Journal of Medicine,* 383**,** 721-732 (2020).
26. Magliano, D. J., Sacre, J. W., Harding, J. L., Gregg, E. W., Zimmet, P. Z. & Shaw, J. E. J. N. R. E. 2020. Young-onset type 2 diabetes mellitus—implications for morbidity and mortality. 16 321-331. (2020).
27. Chung, S., Azar, K. M. J., Baek, M., Lauderdale, D. S. & Palaniappan, L. P. Reconsidering the Age Thresholds for Type II Diabetes Screening in the U.S. *American Journal of Preventive Medicine,* 47**,** 375-381(2014)
28. Schellenberg, E. S., Dryden, D. M., Vandermeer, B., Ha, C. & Korownyk, C. Lifestyle interventions for patients with and at risk for type 2 diabetes: a systematic review and meta-analysis. *Annals of internal medicine,* 159**,** 543-551.(2013)
29. Bellou, V., Belbasis, L., Tzoulaki, I. & Evangelou, E. Risk factors for type 2 diabetes mellitus: an exposure-wide umbrella review of meta-analyses. *PloS one,* 13**,** e0194127 (2018)
30. Yanai, H., Adachi, H., Hakoshima, M. & Katsuyama, H. Molecular Biological and Clinical Understanding of the Pathophysiology and Treatments of Hyperuricemia and Its Association with Metabolic Syndrome, Cardiovascular Diseases and Chronic Kidney Disease. *International Journal of Molecular Sciences,* 22**,** 9221 (2021)
31. Nakayama, A., Kurajoh, M., Toyoda, Y., Takada, T., Ichida, K. & Matsuo, H. Dysuricemia. *Biomedicines,* 11**,** 3169 (2023)
32. Wen, S., Arakawa, H. & Tamai, I. Uric acid in health and disease: From physiological functions to pathogenic mechanisms. *Pharmacology & Therapeutics,* 256**,** 108615 (2024)
33. Jager, K. J., Kovesdy, C., Langham, R., Rosenberg, M., Jha, V. & Zoccali, C. A single number for advocacy and communication—worldwide more than 850 million individuals have kidney diseases. Oxford University Press (2019)
34. Bikbov, B., Purcell, C. A., Levey, A. S., Smith, M., Abdoli, A., Abebe, M., Adebayo, O. M., Afarideh, M., Agarwal, S. K. & Agudelo-Botero, M. Global, regional, and national burden of chronic kidney disease, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *The lancet,* 395**,** 709-733 (2020)
35. Delanaye, P., Cavalier, E. & Pottel, H. 2017. Serum Creatinine: Not So Simple! *Nephron,* 136**,** 302-308 (2017).

36. Chunlei, Y., Liubao, G., Tao, W. & Changying, X. The association between serum uric acid to creatinine ratio and renal disease progression in type 2 diabetic patients in Chinese communities. *Journal of diabetesand its complications,* 33**,** 473-476 (2019).
37. Čaušević1 Adlija *, Sabina Semiz1 , Amra Macić-Džanković2 , Bakira Cico3 , Tanja Dujić1 , Maja Malenica1 , Tamer Bego1. Relevance Of Uric Acid In Progression Of Type 2 Diabetes Mellitus. *Bosnian Journal Of Basic Medical Sciences* 2010; 10 (1): 54-59 (2010).
38. Xing Yuling , Jinhu Chen , Jing Liu , Guangyao Song , Huijuan Ma. Relationship Between Serum Uric Acid-to-Creatinine Ratio and the Risk of Metabolic-Associated Fatty Liver Disease in Patients with Type 2 Diabetes Mellitus. Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy 15 (2022).
39. Smith, TA., Alexandra, AM., Bruce, RK., Carmel, ES. Insulin strategies for and protein in typ 1 diabetes: A systematic review. Diabetic Medicine (38):11. 14641. https://doi.org/10.1111/dme.14641 (2020).
40. Minari, T.P.; Tácito, L.H.B.; Yugar, L.B.T.; Ferreira-Melo, S.E.; Manzano, C.F.; Pires, A.C.; Moreno, H.; Vilela-Martin, J.F.; Cosenso-Martin, L.N.; Yugar-Toledo, J.C. Nutritional Strategies for the Management of Type 2 Diabetes Mellitus: A Narrative Review. Nutrients 2023, 15, 5096. https://doi.org/10.3390/nu15245096 (2023).
41. Luo, Yingxian ; Qiong, Wu; Runtang, Meng; Fuzhi, Lian; Chen, Jiang;Meiyu Hu; Yaxin Wang; Haiyan Ma.. Serum Uric Acid Levels and Their Association with Renal Function Decline and Kidney Disease Progression in Middle-Aged and Elderly Populations: A Retrospective Cohort Study. 16. https://doi.org/10.2147/JMDH.S435537 (2024).
42. Bellomo Gianni, Sandro Venanzi, Claudio Verdura, Paolo Saronio, Antonella Esposito, and Mario Timio. Association of Uric Acid With Change in Kidney Function in Healthy Normotensive Individuals. *American Journal of Kidney Diseases*., Vol xx, No x (2010).
43. Gu, Liubao, Liji Huang, Haidi Wu, Qinglin Lou, and Rongwen Bian. Serum uric acid to creatinine ratio: A predictor of incident chronic kidney disease in type 2 diabetes mellitus patients with preserved kidney function. *Diabetes & Vascular Disease Research*, Vol. 14(3) 221–225 (2017).
44. Chen,li, Zhangxiang, Zhu., Shandong, Ye., Mao, Zheng., The Serum Uric Acid to Serum Creatinine Ratio is an Independent Risk Factor for Diabetic Kidney Disease. *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy* 15 3693–3703 (2022).
45. Mu H, Zhang Q, Huang W, Pan Q, Zhang Y, Lu Y, Zhu Z, Jiang X, Wang G, Zheng M and Chen L The serum uric acid to creatinine ratio as a diagnostic biomarker for normoalbuminuric diabetic kidney disease. Front. Med. 12:1584049. doi: 10.3389/fmed.2025.1584049 (2025).
46. Tao, J., Shen, X., Li, J., Cha, E., Gu, P.-P., Liu, J., Zhu, W., He, L.-L., Li, G.-Q. & Wang, Z. Serum uric acid to creatinine ratio and metabolic syndrome in postmenopausal Chinese women. *Medicine,* 99**,** e19959 (2020).

Tables

Table (1): Baseline Characteristics and Primary Outcomes of Study Groups

Group	Age Distribution % (year)	Dietary Habits	Key Health Indicators
Glycemic regulator	24%: <40y 56%: 40-60y 20%: >60y	Irregular diet (60%)	HbA1c: 8.73% Uric acid: 8.25 mg/dl Creatinine:0.92 mg/dl
Combination therapy of glycemic regulator and hypoglycemic agent (drug)	24%: <40y 52%: 40-60y 24%: >60y	Partial diet control (45%)	HbA1c: 9.02% Uric acid: 7.82 mg/dl Creatinine:1.23 mg/dl
Insulin	28%: <40y 44%: 40-60y 28%: >60y	Therapy-compliant diet (30%)	HbA1c: 9.7% Uric acid: 9.5 mg/dl Creatinine:0.8 mg/dl
Diet	48%: <40y 40%: 40-60y 12%: >60y	Unstructured attempts (100%)	HbA1c: 7.65% Uric acid: 6.49 mg/dl Creatinine:0.7 mg/dl
Control	72% : <40y 24%: 40-60y 4% : >60y	Healthy diet (80%)	HbA1c: 5.4% Uric acid: 6.27 mg/dl Creatinine: 0.82 mg/dl

Note: the sum for combination therapy age groups is 100%.

Table (2) The level of glucose in samples (mg/dL)

Group	Mean ±SD (mg/dL)	Male(mg/dL)	Female (mg/dL)	Largest (mg/dL)	Lowest (mg/dL)
glycemic regulator	202±58 **b**	212	188	297	96
Drugs	214±62 **b**	182	248	355	134
Diets	173±74 **b**	193	154	383	99
Control	103±36 **a**	102	117	191	65
Insulin	232±70 **c**	228	236	383	131

Different superscript letters (a,b,c) **P- value ≤0.05, t-test) a=** control significantly lower than all diabetic group, **c**= insulin group had significantly higher than diet group (p=0.003), **b**= intermediate groups not significantly different from each other.

Table (3) The level of (HbA1c) in samples (mg/dL)

group	Mean ±SD	Male	Female	Largest	Lowest
glycemic regulator	8.73±1.95 **b**	9.1	8.25	12.5	5.9
drugs	9.02±2.2 **b,c**	8	10.12	14	6.3
diets	7.65±2.6**b**	8.37	6.99	15	5.1
control	5.4±0 **a**	5.4	5.5	5.5	5.4
insulin	9.7±2.5 **c**	9.58	9.86	15	6.2

Different superscript letters indicated significant difference (**P- value ≤0.05)** showed **a**= all diabetic groups differed significantly from control ($p < 0.05$). **b,c**= insulin and drugs groups are not significantly different from each other ($p = 0.42$), hence they share the letter (c) but drugs also shares (b) with diet and glycemic regulator.

Table (4) The level of Creatinine in samples (mg/dL)

group	Mean ±SD	Male	Female	Largest	Lowest
glycemic regulator	**a,b**0.92±0.37	0.98	0.84	1.67	0.24
drugs	1.23±1.7 **b**	0.87	1.62	9.09	0.40
diets	0.7±0.31 **a**	0.72	0.68	1.36	0.01
control	0.82±0.26 **a,b**	0.85	0.74	1.27	0.1
insulin	0.8±0.30 **a,b**	0.77	0.81	1.36	0.33

Different superscript letters indicated significant differences (**P- value ≤0.05,** t-test with Bonferroni) **a**= diet group significantly lower than drugs groups (p = 0.018). **b**= drugs group significantly higher than diet group. Groups with (a,b) are not significance different from either diet or drugs.

Table (5) The level of Uric acid in samples (mg/dL)

group	Mean ±SD	Male	Female	Largest	Lowest
glycemic regulator	8.25±4.33 b	8.92	7.39	19.77	2.86
drugs	7.82±5.34 a,b	7.04	8.66	28.21	2.35
diets	6.49±4.68 a	6.3	6.67	17.61	0.84
control	6.27±2.44 a	6.95	5.15	11.19	1.97
insulin	7.95±5.03 b	10.22	8.83	23.37	0.76

Different superscript letters (a,b) indicated significant differences (p- value ≤0.05 Post-hoc tests) **a**= control and diet not different from each other. **b**= insulin and **glycemic regulator** have significantly higher uric acid than diet (p = 0.007) and control (p = 0.002). drugs group (a,b) is intermediate and not significantly different from other.

Table (6): The ratio of uric acid to creatinine of the diabetic patients.

Group	Creatinine (mg/dL)	Uric acid (mg/dL)	SUA/Cr Mg/dL
Glycemic regulator	0.9216	8.248	8.9497 a,b
Drug	1.2308	7.8176	6.35164 a
Insulin	0.7948	9.4952	12.9467 c
Diet	0.6965	6.4888	9.31603 b
Control	0.8192	6.2668	7.6499 a,b

Different superscript letters (a,b,c) indicate significant differences between groups (t-test with Bonferroni, $p < 0.05$). **c**= Insulin group has the highest SUA/Cr ratio, significantly different from all other groups except glycemic regulator. **a**= Drugs group has the lowest ratio, significantly different from insulin and diet groups. Groups sharing (a,b) are not significantly different from each other.

Figures

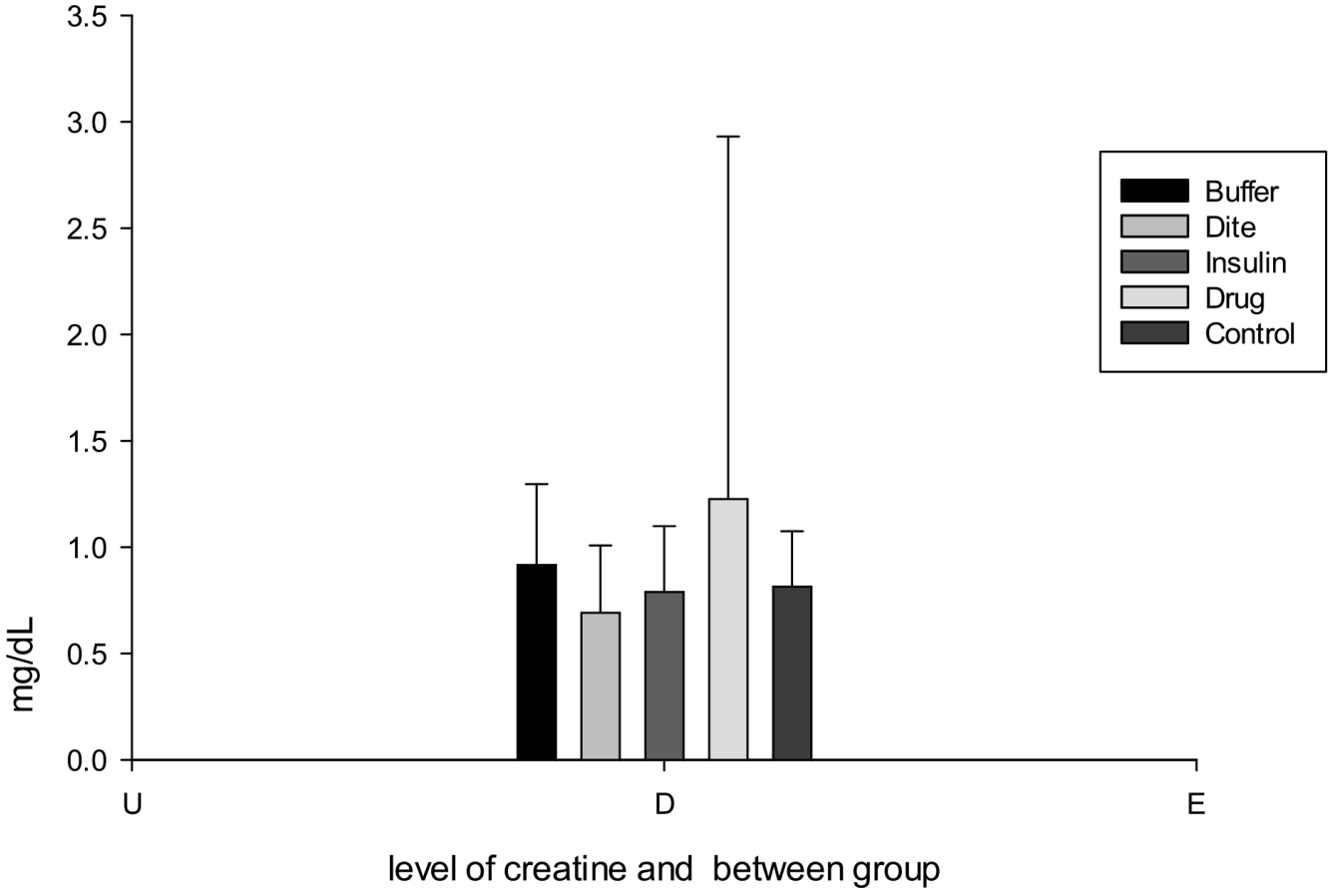


Figure 1

The ratio of uric acid to creatinine of the male diabetic patients.

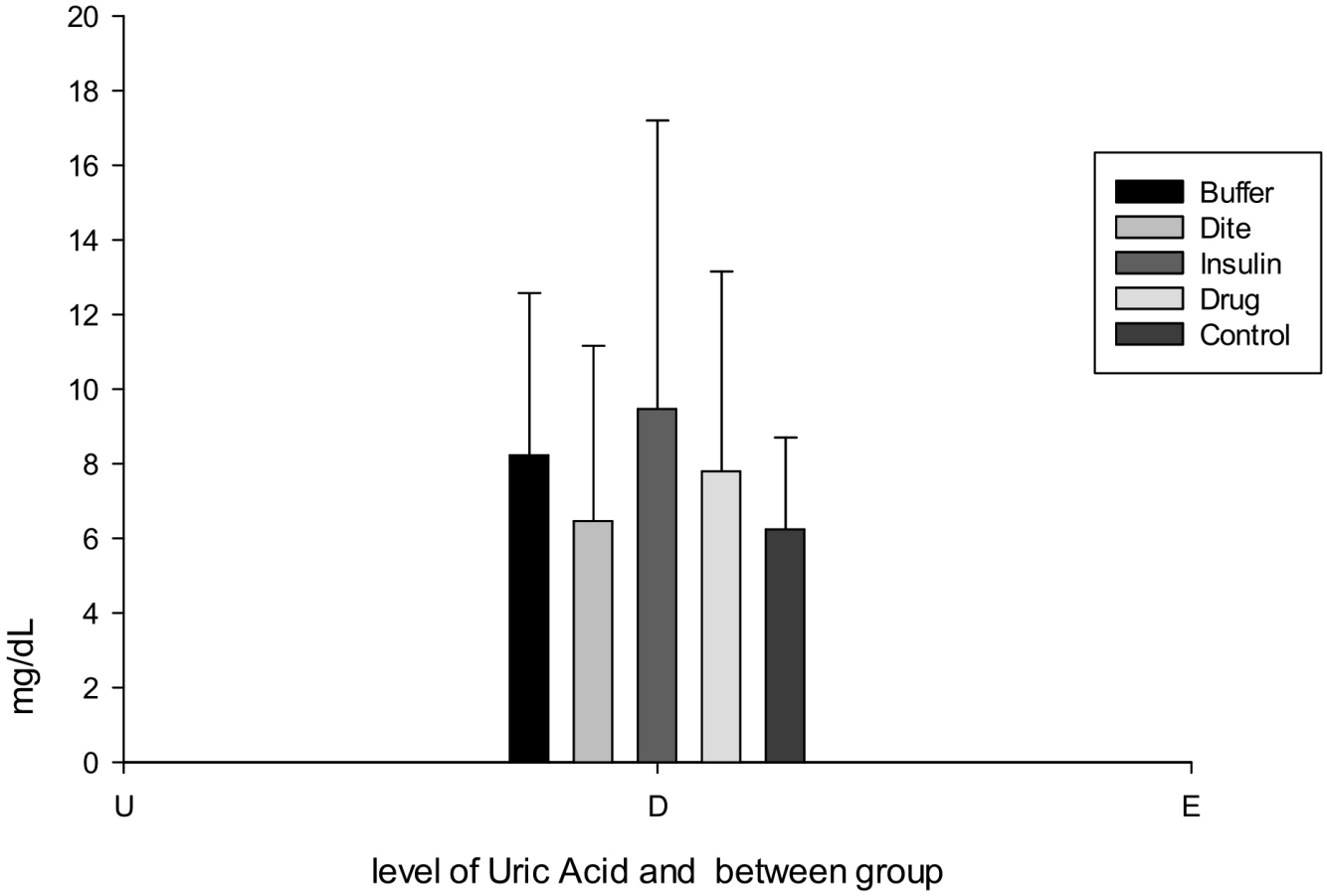


Figure 2

The ratio of uric acid to creatinine of the female diabetic patients.

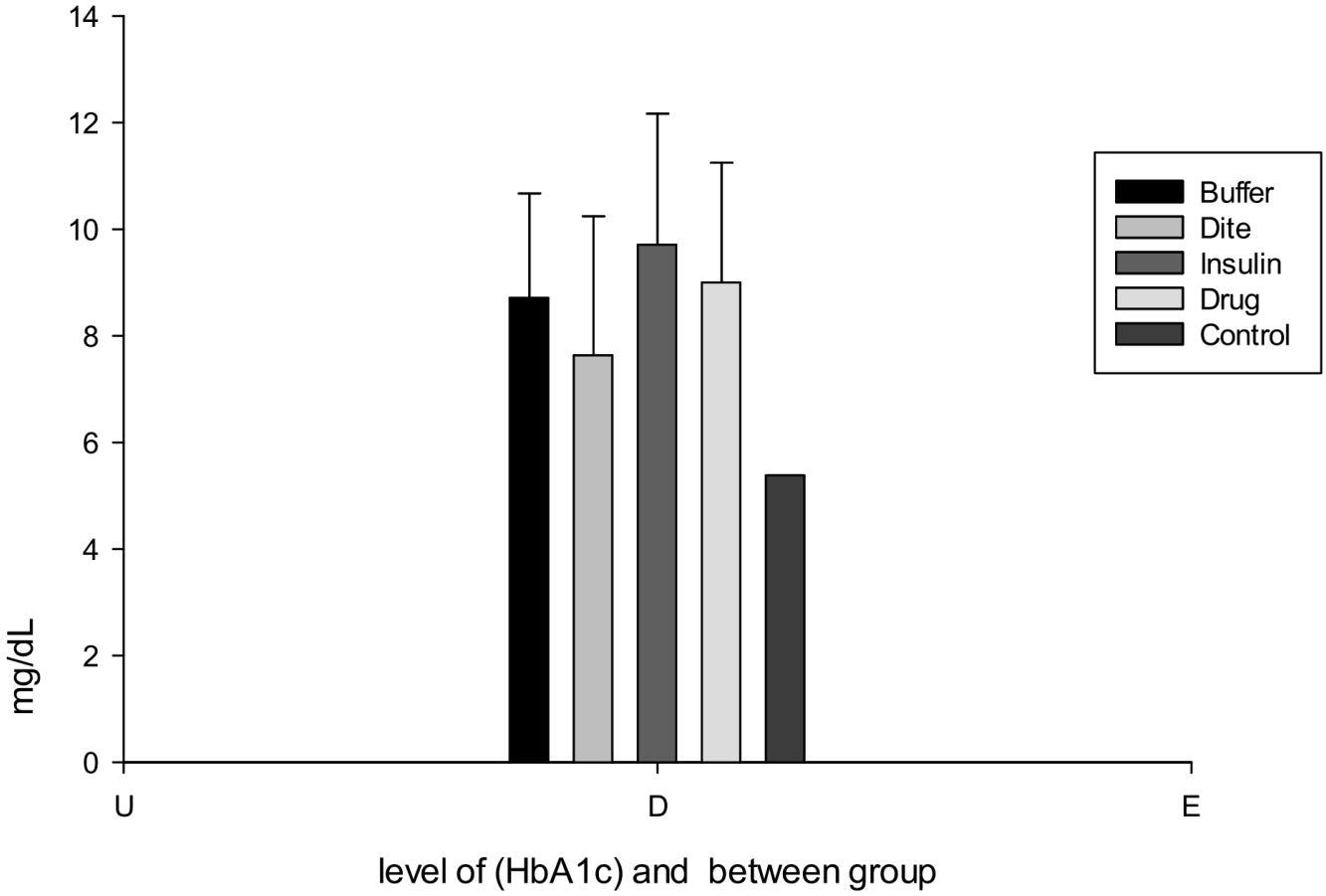


Figure 3

The ratio of uric acid to creatinine between male and female

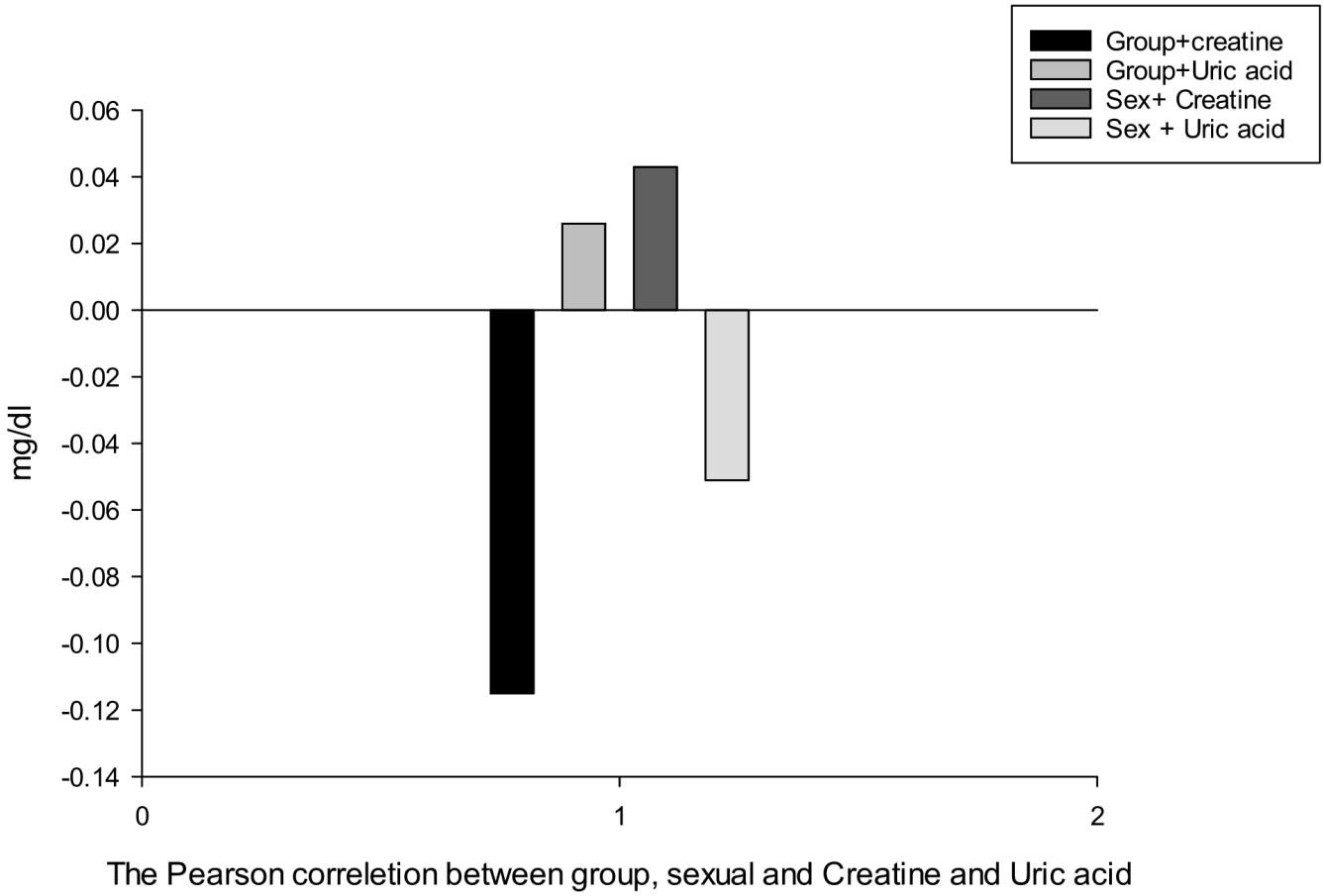


Figure 4

Legend not included with this version.