

A STUDY OF PREVALENCE OF METABOLIC SYNDROME AND HYPERURICEMIA IN TYPE 2 DIABETES MELLITUS

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Abstract

Background: The prevalence of Metabolic Syndrome (MetS) and hyperuricemia is on the rise globally, with significant implications for cardiovascular health. Understanding the intricate relationship between these conditions is crucial for developing targeted interventions. **Material and Methods:** A cross-sectional study was conducted on 150 T2DM patients in Navi Mumbai. Anthropometric measurements, biochemical analyses, and demographic information were collected. Prevalence rates were determined, and statistical analyses were employed to explore associations between hyperuricemia and MetS components. **Results:** The overall prevalence of MetS was 40%, with males exhibiting a higher prevalence (25.3%) compared to females (14.6%). Hyperuricemia was prevalent in 45% of the studied population, with a higher prevalence in males (24.6%) than females (20.6%). The co-occurrence of MetS and hyperuricemia was noted in 18% of cases. Serum triglyceride levels were significantly associated with hyperuricemia ($p < 0.04$), and serum uric acid exhibited a negative correlation with serum HDL-C ($p < 0.001$). **Conclusion:** This study provides valuable insights into the prevalence of MetS and hyperuricemia in a T2DM population. The observed associations between hyperuricemia and MetS components emphasize the need for comprehensive patient care. Understanding these relationships contributes to the development of targeted interventions, optimizing health risk management in individuals with T2DM.

INTRODUCTION

Diabetes mellitus, a chronic metabolic disorder characterized by elevated blood glucose levels, continues to pose a significant global health challenge.^[1] Type 2 diabetes mellitus (T2DM) accounts for most diabetes cases and its global burden are steadily increasing, with prevalence rates soaring across diverse populations. Lifestyle changes, urbanization, and an aging population contribute to the rising tide of diabetes.^[2] According to the International Diabetes Federation (IDF), an estimated 463 million adults were living with diabetes in 2019, and this number is projected to rise to 700 million by 2045.^[3] T2DM, characterized by insulin resistance and relative insulin deficiency, accounts for approximately 90% of these cases.^[4] The multifaceted nature of T2DM necessitates a comprehensive understanding of its complications to inform effective prevention and management strategies.

Metabolic syndrome (MetS) is a complex and interrelated set of metabolic risk factors that includes abdominal obesity, dyslipidemia,

hypertension, and insulin resistance.^[5] Individuals with T2DM often exhibit a heightened prevalence of MetS, thereby escalating their risk of cardiovascular diseases and other related complications.^[6] Central obesity, a hallmark of MetS, contributes to insulin resistance, creating a vicious cycle that exacerbates hyperglycemia in individuals with T2DM.^[7] Prevalence of MetS in individuals with T2DM is notably elevated, as indicated by several studies.^[8-10] Understanding the intricate relationships between the individual components of MetS and T2DM is crucial for developing targeted interventions to mitigate the cardiovascular risk associated with these conditions.

Among the numerous complications associated with T2DM, the clustering of metabolic abnormalities, and the elevated levels of uric acid, termed hyperuricemia, have emerged as critical factors influencing the disease trajectory.^[11] Uric acid, once considered a mere metabolic byproduct, is now recognized as a bioactive molecule with potential implications for insulin resistance and inflammation.^[12] Several studies have reported a higher prevalence of hyperuricemia in individuals

with T2DM compared to those without diabetes. Concurrently, hyperuricemia has been identified as a common comorbidity in T2DM.^[11,13-14] The intricate interplay between MetS, hyperuricemia, and T2DM presents a compelling avenue for exploration, shedding light on the intricate web of interconnected factors that contribute to the pathophysiology of the disease. Also, the complex interconnection between hyperuricemia and T2DM necessitates a closer examination to reveal the mechanisms linking these two entities and to explore whether interventions targeting uric acid levels could offer therapeutic benefits in the context of T2DM.

While previous studies have individually investigated MetS or hyperuricemia in the context of T2DM, a comprehensive analysis that simultaneously considers both phenomena are lacking. This study aims to bridge this gap by providing a subtle understanding of the prevalence, interconnections, and clinical implications of MetS and hyperuricemia in individuals with T2DM.

MATERIALS AND METHODS

Study Design and Setting

This prospective study was conducted at a tertiary care hospital, employing a prospective research design. The study duration extended until the achievement of the predetermined sample size, ensuring a comprehensive exploration of the prevalence and interconnections between MetS and hyperuricemia in individuals with T2DM. Before initiating the study, approval was sought from the Institutional Ethics Committee (IEC Ref no-PDDYPMC/Ethics/PG Dissert/2015), ensuring that the research adhered to ethical standards and guidelines. Additionally, a written signed informed consent process preceded the enrolment of each participant. The study adhered to specific eligibility criteria for participant inclusion and exclusion. A sample size of 150 participants was determined, a number selected to provide statistically significant insights into the prevalence rates of MetS and hyperuricemia in the context of T2DM.

Inclusion Criteria

The inclusion criteria for this study encompassed consecutive patients diagnosed with T2DM, attending either the outpatient or inpatient departments of Medicine or Endocrinology within tertiary care unit. Specifically, participants were required to fall within an age group exceeding 40 years. A crucial facet of inclusion was the participants' willingness to provide informed consent, emphasizing the ethical imperative of ensuring that individuals voluntarily and knowingly agreed to participate in the study.

Exclusion Criteria

The study established specific exclusion criteria to ensure the integrity and feasibility of the investigation. Participants were excluded if the protocol for required investigations and assessments

was deemed impractical due to underlying reasons. Additionally, individuals unwilling to provide informed consent were excluded from participation. Pregnant individuals were excluded from the study to account for potential confounding variables associated with metabolic changes during pregnancy. Participants currently on thiazides, O.C pills, cytotoxic drugs, or anti Koch's therapy were excluded due to the potential influence of these medications on metabolic parameters. Individuals engaging in alcohol consumption or smoking were excluded. Also, patients diagnosed with lymphoma or leukemia, as well as those on xanthine oxidase inhibitors or uricosuric drugs, salicylates, were excluded to maintain a homogeneous study population. Conditions such as nephrotic syndrome and organ transplant were also considered exclusion criteria to avoid potential interference with the study's objectives.

Data Collection and Investigations

Demographic details of patients, encompassing their name, age, gender, address was recorded in a pre-designed case record form, ensuring a systematic approach to data collection. During the study, a comprehensive array of investigations was conducted to gain a thorough understanding of participant's metabolic and cardiovascular health. Fasting lipid profile measurements were taken to assess cholesterol and triglyceride levels in the fasting state. Fasting and post prandial blood sugar assessments were performed to gauge glycemic control, evaluating blood glucose levels in both fasting and postprandial states. Renal function tests, including the measurement of serum creatinine, blood urea, and total proteins, were carried out to provide insights into renal health. Serum uric acid levels were evaluated to discern any potential link with MetS and T2DM. A complete blood count was also performed for all the enrolled subjects. Anthropometric measurements, including weight, height, BMI, waist circumference, hip circumference, and thigh circumference, were meticulously recorded to provide data on body composition.

The study also involved the recording of the heart's electrical activity through Electrocardiogram (ECG) to assess cardiac health. Additionally, imaging procedures, such as Chest X-Ray and Ultrasonography of the abdomen, were conducted whenever required, contributing additional insights into the overall health status of the participants.

RESULTS

In this study, a total of 150 participants were analyzed, comprising 82 males (54.7%) and 68 females (45.3%). The male group was further categorized by age, revealing that 37% of males were under 5 years old, 24% were between 6-10 years, 29% were aged 11-15 years, and 10% were above 16 years. Similarly, among females, 45%

were in the <5 years age group, 41% in the 6-10 years group, 10% in the 11-15 years group, and 10% were older than 16 years. Among 150 participants, 45% (n = 68) had diabetes for less than 5 years, 30% (n = 44) for 6-10 years, 13% (n = 20) for 11-15 years, and 12% (n = 18) for over 16 years (Table 1). The analysis of health conditions within the study cohort revealed notable distributions. 63% of participants exhibited hypertension, with 40% presenting MetS. Hyperuricemia was identified in 45% of cases. Furthermore, an intersection of MetS and hyperuricemia was observed in 18% of participants (Table 1). The lipid profile analysis of the study cohort highlighted distinct patterns. Most participants, constituting 57% (n = 85), exhibited elevated triglyceride levels, while 43% (n = 65) had triglyceride levels within the normal range. Regarding high-density lipoprotein cholesterol (HDL-C), 31% (n = 46) fell within the low-normal range, while 69% (n = 104) maintained normal HDL-C levels (Table 1). Examining the duration of diabetes in our cohort revealed varying associations with MetS. For individuals with diabetes for less than 5 years, 50% had MetS, compared to 23% in the 6-10 years group, 17% in the 11-15 years group, and 10% for over 16 years. [Table 1]

In our study, 40% were diagnosed with MetS, with a breakdown showing 46.3% of males and 32.4% of females affected. While statistical analysis hinted at an association between gender and MetS (Pearson Chi-Square = 3.031, p = 0.082), the odds ratio of 1.806 (95% CI: 0.926 - 3.523) suggested a moderate relationship. Further exploration into gender-specific odds ratios indicated non-significant trends for males (1.295, 95% CI: 0.973 - 1.724) and females (0.717, 95% CI: 0.486 - 1.059). The gender-specific distribution of health conditions among participants with diabetes reveals distinct patterns. Among males, 63% had MetS, while 54% had hyperuricemia. Notably, 52% of males had both MetS and hyperuricemia, highlighting a significant overlap. In contrast, among females, 37% had MetS, 46% had hyperuricemia, and 48% had both conditions (Table 2). Statistical analyses of hyperuricemia and gender distribution, including the chi-square test, revealed no significant association between gender and hyperuricemia (p = 0.954). The odds ratio for hyperuricemia (with/without) was 0.981 (95% CI: 0.515 - 1.872), indicating no substantial gender-related influence. Further examination of gender-specific odds ratios showed non-significant trends for both males (0.992, 95% CI: 0.740 - 1.329) and females (1.010, 95% CI: 0.710 - 1.438). These findings suggest a lack of significant gender-based impact on hyperuricemia in

the study population. The examination of the co-occurrence of MetS and hyperuricemia within the study's gender distribution, including the chi-square test, revealed no significant association between gender and the co-occurrence of MetS and hyperuricemia (Pearson Chi-Square = 0.105, p = 0.746). The odds ratio for the co-occurrence was 0.871 (95% CI: 0.378 - 2.006), suggesting no substantial association between gender and the likelihood of having both conditions. Further exploration of gender-specific odds ratios for males (0.938, 95% CI: 0.631 - 1.395) and females (1.077, 95% CI: 0.695 - 1.669) indicated no significant gender-specific associations. [Table 2]

The comparative analysis of clinical parameters among male (n=82) and female (n=68) participants with diabetes revealed several key insights. No significant differences were observed in age, height, weight, waist circumference (WC), body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), and duration of diabetes. However, fasting blood sugar (FBS) levels were higher in females (140.4 ± 25.0) compared to males (132.4 ± 26.6), demonstrating statistical significance (p<0.05). Males exhibited higher uric acid levels (5.9 ± 1.8) compared to females (5.1 ± 1.5), reaching statistical significance (p<0.01). Additionally, high-density lipoprotein cholesterol (HDL-C) was significantly higher in females (57.7 ± 15.8) compared to males (51.9 ± 19.5) (p<0.05). [Table 3]

An examination of clinical parameters in participants with diabetes revealed significant associations with hyperuricemia, MetS, and their combination (Table 4). Individuals without hyperuricemia, MetS, or their combination exhibited lower weight, BMI, DBP, SBP was lower in those without these conditions. FBS levels were significantly elevated in individuals with hyperuricemia and MetS, both independently and in combination. Uric acid levels were higher in those with hyperuricemia, MetS, or both, with the combination showing the most substantial increase. HDL-C levels were significantly higher in individuals without these conditions. Conversely, triglyceride levels were elevated in those with hyperuricemia, MetS, or their combination, with the combination exhibiting the highest levels. These findings underscore the distinct impact of Hyperuricemia, MetS, and their combination on various clinical parameters, highlighting the importance of considering these conditions in the comprehensive management of diabetes-related health outcomes. [Table 4]

Table 1: Demographic and Health Characteristics of Participants with Diabetes

Demographics				Diabetes (n =150)	
Age Groups	N (%)	Male (n=82)	Female (n=68)	Duration (years)	N (%)
41-50	58 (39)	30 (37)	28 (45)	<5	68 (45)
51-60	43 (28)	20 (24)	23 (41)	6-10	44 (30)
61-70	34 (23)	24 (29)	10 (34)	11-15	20 (13)

	71-80	15 (10)	8 (10)	7 (10)	>16	18 (12)
Distribution						
Cases	Hypertension N (%)	MetS N (%)	Hyperuricemia N (%)	MetS & Hyperuricemia N (%)		
Yes	95 (63)	60 (40)	68 (45)	27 (18)		
No	55 (37)	90 (60)	82 (55)	123 (82)		
High Normal	Triglyceride N (%)	Low Normal	HDL-C N (%)			
	85 (57) 65 (43)		46 (31) 104 (69)			
Duration of DM (years)	<5	6-10	11-15	>16		
With Met S N (%)	30 (50)	14 (23)	10 (17)	6 (10)		
Without MetS N (%)	38 (42)	30 (33)	10 (11)	12 (14)		

Table 2: Gender-Based Classification of MetS, Hyperuricemia, and their Combination in Participants with Diabetes

Sex	MetS		Hyperuricemia		MetS + Hyperuricemia	
	With	Without	With	Without	With	Without
Male	38 (63)	44 (49)	37 (54)	45 (55)	14 (52)	68 (55)
Female	22 (37)	46 (51)	31 (46)	37 (45)	13 (48)	55 (45)

Table 3: Gender-Based Comparison of Clinical Parameters in Participants with Diabetes

Parameters	Males (n=82)	Females (n=68)
Age (years)	57.3 ± 10.4	55.1 ± 9.4 ^{NS}
Height (cms)	170.0 ± 6.5	169.1 ± 6.7 ^{NS}
Weight (kgs)	65.3 ± 11.2	66.4 ± 13.2 ^{NS}
WC	79.7 ± 8.7	81.4 ± 10.7 ^{NS}
BMI	22.6 ± 3.4	23.1 ± 4.0 ^{NS}
SBP	136.2 ± 17.6	136.2 ± 16.8 ^{NS}
DBP	88.9 ± 14.8	88.4 ± 12.5 ^{NS}
Duration	7.6 ± 6.2	7.3 ± 5.8 ^{NS}
FBS	132.4 ± 26.6	140.4 ± 25.0*
Uric Acid	5.9 ± 1.8	5.1 ± 1.5**
HDL-C	51.9 ± 19.5	57.7 ± 15.8*
Triglyceride	148.1 ± 27.1	138.8 ± 31.8 ^{NS}

Data represents Mean ± SD. Symbols represents statistical significance, *p<0.05, **p<0.01 and NS- Non-significance. Abbreviations: MetS- Metabolic Syndrome; WC- Waist circumference; SBP-Systolic blood pressure; DBP- Diastolic blood pressure

Table 4: Comparison of Clinical Parameters among T2DM Patients with Hyperuricemia, MetS, and Hyperuricemia + MetS

Parameters	Hyperuricemia		MetS		Hyperuricemia + MetS	
	With (n=68)	Without (n=82)	With (n=60)	Without (n=90)	With (n=27)	Without (n=123)
Age	57.3 ± 10.4	55.1 ± 9.4 ^{NS}	56.2 ± 9.8	56.4 ± 10.2 ^{NS}	54.6 ± 10.8	56.7 ± 9.8 ^{NS}
Height	170.0 ± 6.5	169.1 ± 6.7 ^{NS}	169.0 ± 5.7	170.0 ± 7.1 ^{NS}	167.4 ± 5.7	170.1 ± 6.7 ^{NS}
Weight	65.3 ± 11.2	66.4 ± 13.2 ^{NS}	62.5 ± 11.6	68.0 ± 12.0**	59.5 ± 9.1	67.2 ± 12.3**
WC	79.7 ± 8.7	81.4 ± 10.7 ^{NS}	78.1 ± 8.8	82.0 ± 10.0**	76.0 ± 7.1	81.4 ± 9.9**
BMI	22.6 ± 3.4	23.1 ± 4.0 ^{NS}	21.8 ± 3.5	23.5 ± 3.7**	21.2 ± 3.1	23.2 ± 3.8*
SBP	136.2 ± 17.6	136.2 ± 16.8 ^{NS}	140.7 ± 16.7	133.2 ± 16.9**	141.3 ± 15.9	135.1 ± 17.3 ^{NS}
Diastolic BP	88.9 ± 14.8	88.4 ± 12.5 ^{NS}	91.7 ± 15.1	86.6 ± 12.5*	91.0 ± 14.5	88.2 ± 13.6 ^{NS}
Duration	7.6 ± 6.2	7.3 ± 5.8 ^{NS}	7.0 ± 5.9	7.7 ± 6.1 ^{NS}	5.9 ± 5.2	7.8 ± 6.2 ^{NS}
FBS	132.4 ± 26.6	140.4 ± 25.0**	129.4 ± 28.4	140.4 ± 23.6*	138.3 ± 29.2	135.5 ± 25.5 ^{NS}
Uric Acid	5.9 ± 1.8	5.1 ± 1.5**	5.5 ± 1.8	5.5 ± 1.8 ^{NS}	7.2 ± 0.6	5.2 ± 1.7**
HDL-C	51.9 ± 19.5	57.7 ± 15.8*	45.9 ± 19.2	60.3 ± 14.8***	53.5 ± 16.5	54.8 ± 18.5*
Triglyceride	148.1 ± 27.1	138.8 ± 31.8*	161.9 ± 15.0	131.9 ± 30.8***	160.9 ± 14.9	140.1 ± 30.7**

Data represents Mean ± SD. Symbols represents statistical significance, *p<0.05, **p<0.01, ***p<0.001 and NS- Non-significance. Abbreviations: MetS- Metabolic Syndrome; WC- Waist circumference; SBP-Systolic blood pressure; DBP- Diastolic blood pressure.

DISCUSSION

This study focused on individuals with T2DM to assess the prevalence of hyperuricemia and MetS). Patients with MetS face a higher risk of

cardiovascular disease compared to those associated with individual components of MetS alone.

In the current investigation, the prevalence of patients experiencing MetS is identified as 40%. This observed prevalence stands in contrast to

previous studies, with Eliaesson et al reporting a significantly higher prevalence of 77%,^[15] Mundhe et al,^[16] noting a prevalence of 43.5%, Patel et al documenting 85%,^[17] and Ogbera et al finding a prevalence of 60%.^[18] A higher prevalence of MetS among males was also observed accounting for 25.3%, in contrast to females with a prevalence of 14.6%. This finding aligns with the research of Marques Vidal et al,^[19] which reported a prevalence of 23% in males compared to 12% in females, and Patel et al, who documented a prevalence of 50% in males versus 38% in females.^[17] Such variations in prevalence across studies may arise from differences in sample demographics, geographic locations, and the specific criteria used to define MetS. It is essential to recognize the impact of factors such as population characteristics, lifestyle patterns, and genetic predispositions on the variability in MetS prevalence.

Hyperuricemia, characterized by elevated levels of uric acid, is becoming increasingly prevalent globally. Existing literature, as described by Nakagawa et al,^[20] Conen et al,^[21] and Schachter et al,^[22] has established a strong association between hyperuricemia and key components of MetS, including obesity, dyslipidemia, hyperglycemia, and hypertension. Our investigation revealed an overall prevalence of hyperuricemia at 45%, aligning with findings from Ogbera et al. (25%),^[18] Mundhe et al. (25.3%),^[16] and Chen et al. (13.1%).^[23] The total number of patients with hyperuricemia in our study was 68, comprising 37 males and 31 females. The prevalence of hyperuricemia was higher in males (24.6%) than in females (20.6%), consistent with the observations by Conen et al. (35.1% vs. 8.7%),^[21] and Chen et al. (19.07% vs. 3.42%).^[23]

Furthermore, our study's overall prevalence of hyperuricemia in 150 patients showed a distribution of 54% in males and 46% in females, diverging from the pattern observed in Ogbera et al. (41% in males and 59% in females).^[18] This discrepancy may be attributed to hormonal influences, with estrogen in females potentially promoting uric acid excretion and may be more important for men to prevent hyperuricemia.^[24]

In our study, the prevalence of hyperuricemia in individuals with MetS was 18%, while it was 82% in those without MetS. These findings align with observations from Mundhe et al. (21.3% vs. 78.7%) and Cheng et al. (24.7%).^[16,23] Interestingly, the prevalence of hyperuricemia showed a near-equal distribution between males and females, each at 9%, consistent with the findings of Tuomilehto et al. (22% vs. 11%),^[25] and Mundhe et al.^[16] An intriguing aspect revealed in our study is the marked association between hyperuricemia and serum triglyceride levels ($p < 0.04$), reinforcing similar findings in studies by Conen et al., Mundhe et al., and Schachter et al.^[16,21,22] This association reflects the intricate relationship between hyperuricemia and dyslipidemia, a key component of MetS.

Furthermore, our data indicated a negative correlation between serum uric acid and serum HDL-C ($p < 0.001$). This observation echoes with the findings of the previous studies.^[16,26,27] Hyperuricemia and Hypertriglyceridemia are suggested to be associated with MetS [28–30]. Many investigators are studying the mechanism of emergence of this syndrome. The mechanism of negatively correlated uric acid and HDL-C may be due to the relationship between decreased HDL-C levels and MetS.^[27]

Acknowledging the limitations of our study is crucial for a proper interpretation of the findings. Our modest sample size may not comprehensively capture the diverse demographics of Navi Mumbai, thus limiting the generalizability of the results. The focus on Navi Mumbai as a single center and potential unaccounted confounders further impact the study's broader applicability. Additionally, constraints related to healthcare access warrant consideration in understanding results. However, despite these limitations, our study provides valuable insights into health dynamics, shedding light on areas for refinement in future research endeavours.

CONCLUSION

The in-depth examination of a diverse array of health parameters within the Navi Mumbai population, particularly focusing on the prevalence and interaction of MetS and hyperuricemia in individuals with T2DM, has yielded valuable insights. Within the 150 patients studied, a substantial 18% exhibited the co-occurrence of MetS and hyperuricemia, emphasizing a critical intersection between metabolic and uric acid-related disorders. This co-occurrence was more prevalent in males (52%) than females (48%), emphasizing the importance of gender-specific considerations in addressing these health conditions. Significant associations were uncovered between the co-occurrence of MetS and hyperuricemia and key variables, including BMI, SUA, HDL-C, and serum triglycerides. Significance of the study lies in its potential implications for clinical practice and public health interventions, particularly within the context of T2DM. A deeper understanding of the intricate relationship between MetS and hyperuricemia can inform tailored and effective approaches to patient care. Furthermore, this research serves as a catalyst for future investigations, encouraging exploration into the mechanisms driving this co-occurrence and its broader ramifications for long-term health outcomes in T2DM.

Conflict of Interest

Authors Declare No Conflict of Interest

REFERENCES

1. Bhuri M, Rastogi V, Tungare K, Marar T. A review on interplay between obesity, lipoprotein profile and nutrigenetics with selected candidate marker genes of type 2 diabetes mellitus. *Mol Biol Rep.* 2022;49(1):687–703.
2. Bhuri M, Tungare K, More S, Sukumaran S, Vidhate D, Gharat A, et al. Association Study between T2DM and CAPN10 SNP-19 (rs3842570) Polymorphism in Navi Mumbai Population. *Asian Pac J Health Sci.* 2022;9(2):178–82.
3. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, et al. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. *Diabetes Res Clin Pract.* 2019 Sep 1; 157:107843.
4. Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, et al. Pathophysiology of Type 2 Diabetes Mellitus. *Int J Mol Sci.* 2020;21(17):6275.
5. Huang PL. A comprehensive definition for metabolic syndrome. *Dis Model Mech.* 2009;2(5–6):231–7.
6. Bhalwar R. Metabolic syndrome: The Indian public health perspective. *Med J Armed Forces India.* 2020;76(1):8–16.
7. Wondmkun YT. Obesity, Insulin Resistance, and Type 2 Diabetes: Associations and Therapeutic Implications. *Diabetes Metab Syndr Obes Targets Ther.* 2020; 13:3611–6.
8. Abagre TA, Bando DA, Addo-Lartey AA. Determinants of metabolic syndrome among patients attending diabetes clinics in two sub-urban hospitals: Bono Region, Ghana. *BMC Cardiovasc Disord.* 2022;22(1):366.
9. Ginsberg HN, MacCallum PR. The Obesity, Metabolic Syndrome, and Type 2 Diabetes Mellitus Pandemic: Part I. Increased Cardiovascular Disease Risk and the Importance of Atherogenic Dyslipidemia in Persons with the Metabolic Syndrome and Type 2 Diabetes Mellitus. *J Cardiometab Syndr.* 2009;4(2):113–9.
10. Charkos TG, Getnet M. Metabolic syndrome in patients with type 2 diabetes mellitus at Adama Hospital Medical College, Ethiopia: a hospital-based cross-sectional study. *Front Clin Diabetes Healthc.* 2023;4.
11. Xiong Q, Liu J, Xu Y. Effects of Uric Acid on Diabetes Mellitus and Its Chronic Complications. *Int J Endocrinol.* 2019; e9691345.
12. Han R, Zhang Y, Jiang X. Relationship Between Four Non-Insulin-Based Indexes of Insulin Resistance and Serum Uric Acid in Patients with Type 2 Diabetes: A Cross-Sectional Study. *Diabetes Metab Syndr Obes.* 2022; 15:1461–71.
13. Bhole V, Choi JWJ, Kim SW, de Vera M, Choi H. Serum Uric Acid Levels and the Risk of Type 2 Diabetes: A Prospective Study. *Am J Med.* 2010;123(10):957–61.
14. Čaušević A, Semiz S, Macić-Džanković A, Cico B, Dujić T, Malenica M, et al. Relevance of uric acid in progression of type 2 diabetes mellitus. *Bosn J Basic Med Sci.* 2010;10(1):54–9.
15. Eliasson B, Cederholm J, Nilsson P, Gudbjörnsdóttir S, Steering Committee of the Swedish National Diabetes Register. The gap between guidelines and reality: Type 2 diabetes in a National Diabetes Register 1996–2003. *Diabet Med J Br Diabet Assoc.* 2005;22(10):1420–6.
16. Mundhe SA, Mhasde DR. The study of prevalence of hyperuricemia and metabolic syndrome in type 2 diabetes mellitus. *Int J Adv Med.* 2016;3(2):241–9.
17. Patel JL, Suthar AM, Dalsaniya VB, Parikh AP, Suthar NN, Patel KL. A Study of Metabolic Syndrome and its Components in Type 2 Diabetes Mellitus Subjects and their Asymptomatic First-degree Relatives. 2013.
18. Ogbera AO, Azenabor AO. Hyperuricaemia and the metabolic syndrome in type 2 DM. *Diabetol Metab Syndr.* 2010 Apr 20; 2:24.
19. Marques-Vidal P, Mazoyer E, Bongard V, Gourdy P, Ruidavets JB, Drouet L, et al. Prevalence of insulin resistance syndrome in southwestern France and its relationship with inflammatory and hemostatic markers. *Diabetes Care.* 2002;25(8):1371–7.
20. Nakagawa T, Hu H, Zharikov S, Tuttle KR, Short RA, Glushakova O, et al. A causal role for uric acid in fructose-induced metabolic syndrome. *Am J Physiol Renal Physiol.* 2006 Mar;290(3): F625–631.
21. Conen D, Wietlisbach V, Bovet P, Shamlaye C, Riesen W, Paccaud F, et al. Prevalence of hyperuricemia and relation of serum uric acid with cardiovascular risk factors in a developing country. *BMC Public Health.* 2004; 4:9.
22. Schachter M. Uric acid and hypertension. *Curr Pharm Des.* 2005;11(32):4139–43.
23. Chen L ying, Zhu W hua, Chen Z wen, Dai H lei, Ren J jing, Chen J hua, et al. Relationship between hyperuricemia and metabolic syndrome. *J Zhejiang Univ Sci B.* 2007;8(8):593–8.
24. Sumino H, Ichikawa S, Kanda T, Nakamura T, Sakamaki T. Reduction of serum uric acid by hormone replacement therapy in postmenopausal women with hyperuricaemia. *Lancet Lond Engl.* 1999;21;354(9179):650.
25. Tuomilehto J, Zimmet P, Wolf E, Taylor R, Ram P, King H. Plasma uric acid level and its association with diabetes mellitus and some biologic parameters in a biracial population of Fiji. *Am J Epidemiol.* 1988;127(2):321–36.
26. Rho YH, Choi SJ, Lee YH, Ji JD, Choi KM, Baik SH, et al. The Prevalence of Metabolic Syndrome in Patients with Gout: A Multicenter Study. *J Korean Med Sci.* 2005;20(6):1029–33.
27. Schmidt MI, Watson RL, Duncan BB, Metcalf P, Brancati FL, Sharrett AR, et al. Clustering of dyslipidemia, hyperuricemia, diabetes, and hypertension and its association with fasting insulin and central and overall obesity in a general population. *Atherosclerosis Risk in Communities Study Investigators. Metabolism.* 1996;45(6):699–706.
28. Tai ES, Emmanuel SC, Chew SK, Tan BY, Tan CE. Isolated low HDL cholesterol: an insulin-resistant state only in the presence of fasting hypertriglyceridemia. *Diabetes.* 1999;48(5):1088–92.
29. Bo S, Cavallo-Perin P, Gentile L, Repetti E, Pagano G. Low HDL-cholesterol: a component of the metabolic syndrome only in the presence of fasting hypertriglyceridemia in type 2 diabetic patients. *Diabetes Metab.* 2001;27(1):31–5.
30. Bosello O, Zamboni M. Visceral obesity and metabolic syndrome. *Obes Rev Off J Int Assoc Study Obes.* 2000 May;1(1):47–56.