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"Serum Uric Acid as a Marker of Cardiovascular Risk in Type 2 Diabetes Mellitus: A Cross-Sectional Study"

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Abstract

Background: Type 2 Diabetes Mellitus (T2DM) is a major global health concern and is strongly associated with increased cardiovascular morbidity and mortality. Emerging evidence suggests that serum uric acid (SUA) may serve as a potential marker of cardiovascular risk in patients with T2DM. ***Objective:*** To evaluate serum uric acid levels in patients with Type 2 Diabetes Mellitus and to determine their association with established cardiovascular risk factors. ***Materials and Methods:*** This hospital-based cross-sectional study was conducted from July 2024 to June 2025 in the Department of General Medicine, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, Karnataka. A total of 100 subjects were included, comprising 70 patients with T2DM (cases) and 30 non-diabetic individuals (controls). Sociodemographic details, clinical history, anthropometric measurements, and cardiovascular risk factors were recorded using a structured proforma. Laboratory investigations included fasting and postprandial blood glucose, lipid profile, serum creatinine, and serum uric acid estimation. Statistical analysis was performed using SPSS software. Student's t-test was applied, and a p-value <0.05 was considered statistically significant. ***Results:*** Mean serum uric acid levels were significantly higher in diabetic patients compared to controls (5.8 ± 1.408 mg/dl vs. 3.85 ± 0.7167 mg/dl; $p < 0.0001$). Hyperuricemia was observed in 27.1% of diabetic patients, while none of the controls exhibited elevated uric acid levels. Serum uric acid levels showed significant positive associations with higher BMI, hypertension, dyslipidemia, longer duration of diabetes, and coronary artery disease ($p < 0.05$). Patients with myocardial infarction demonstrated higher uric acid levels compared to those with ischemia alone. No significant association was found between serum uric acid levels and gender or smoking status. ***Conclusion***: Serum uric acid levels are significantly elevated in patients with Type 2 Diabetes Mellitus and are positively associated with multiple cardiovascular risk factors. These findings suggest that serum uric acid may serve as a useful and accessible biomarker for cardiovascular risk stratification in individuals with T2DM.

Keywords: Type 2 Diabetes Mellitus; Serum Uric Acid; Hyperuricemia; Cardiovascular Risk; Dyslipidemia; Hypertension.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major global health challenge characterized by chronic hyperglycemia arising from insulin resistance and progressive β-cell dysfunction. The worldwide prevalence of T2DM has risen dramatically in recent decades, contributing significantly to morbidity and mortality from both microvascular and macrovascular complications. Among these complications, cardiovascular disease (CVD) remains the leading cause of death in individuals with T2DM, accounting for a substantial proportion of premature mortality.[1] Emerging evidence suggests that beyond traditional risk factors such as hypertension, dyslipidaemia, smoking, and obesity, novel biochemical markers may improve early identification of cardiovascular risk in diabetic patients.

Uric acid is the final oxidation product of purine metabolism in humans. While historically regarded as an inert metabolic waste, accumulating research has revealed that elevated serum uric acid (SUA) is not only associated with gout and renal calculi but may also be linked to CVD and metabolic disorders, including T2DM.[2] Several epidemiological studies have reported that high SUA levels correlate with components of metabolic syndrome and cardiovascular risk factors such as hypertension, dyslipidaemia, and obesity, which themselves are important determinants of adverse cardiovascular outcomes in diabetes.[3] The potential role of SUA as a marker or mediator of increased cardiovascular risk in T2DM has generated considerable scientific interest over the past half-decade.

Population-based and clinical studies have provided supportive, albeit sometimes conflicting, evidence regarding the association between SUA and cardiovascular outcomes in T2DM. In the Fenofibrate Intervention and Event Lowering in Diabetes (FIELD) study, baseline SUA was a significant independent predictor of long-term cardiovascular events in adults with T2DM, with each increment in SUA associated with increased risk of major adverse cardiovascular events even after adjusting for conventional risk factors.[4] Similarly, observational research has demonstrated that type 2 diabetic patients with higher SUA levels exhibited a greater prevalence of coronary artery disease and related cardiovascular risk factors, including elevated body mass index and adverse lipid profiles.[5] Recent hospital-based data have confirmed a positive relationship between SUA levels and cardiovascular disease as well as established risk factors in T2DM patients, suggesting its potential utility as a predictive biomarker.[6]

However, the exact causal relationship between elevated SUA and cardiovascular risk in T2DM remains a subject of ongoing debate. A large cross-sectional and Mendelian randomization analysis using data from the National Health and Nutrition Examination Survey (NHANES) reported that while higher SUA levels were associated with increased risk of heart failure on observational analysis, causal inference models did not support a direct causal link between genetically predicted SUA and cardiovascular disease endpoints.[6] These findings highlight the complexity of the SUA–CVD relationship and raise important questions about whether elevated SUA is a direct mediator of cardiovascular pathology or a surrogate marker reflecting underlying metabolic disturbances common in T2DM.

Moreover, studies examining the interaction of SUA with other cardiovascular risk factors in T2DM have further illustrated this multifaceted association. For example, hyperuricaemia has been shown to correlate positively with dyslipidaemia and hypertriglyceridaemia in diabetic inpatients, underscoring the link between altered purine metabolism and lipid abnormalities that contribute to atherosclerosis.[7] The identification of SUA-to-HDL cholesterol ratios and other composite biomarkers has also been explored, demonstrating significant associations with cardiovascular outcomes such as myocardial infarction, and suggesting additional avenues for risk stratification in T2DM populations.[8] Despite these advances, several gaps in the evidence remain. Variability in study design, population characteristics, and analytical methods across recent investigations limits the generalizability of findings. Inconsistent results pertaining to causal inferences and the magnitude of association between SUA and different cardiovascular outcomes further highlight the need for focused research within well-defined diabetic cohorts. Additionally, prospective longitudinal studies remain relatively scarce compared to cross-sectional analyses, reducing clarity on temporal relationships and risk prediction utility.

Understanding the role of SUA in the cardiovascular risk profile of T2DM patients has important clinical implications. If elevated SUA independently predicts cardiovascular risk, it could serve as an accessible biomarker to identify high-risk individuals who may benefit from more aggressive therapeutic intervention and monitoring. Conversely, if SUA reflects underlying metabolic dysregulation without causal influence, its use may be confined to risk stratification rather than as a target for therapeutic modification.

Against this backdrop, the present study aims to investigate the association between serum uric acid levels and cardiovascular risk factors in individuals with type 2 diabetes mellitus. By evaluating the relationship of SUA with established vascular risk determinants in a cross-sectional cohort, this research seeks to contribute further evidence on the potential utility of SUA as a marker of cardiovascular risk in T2DM.

OBJECTIVES:

1. To identify the levels of serum uric acid in persons with Type 2 diabetes mellitus.
2. To identify whether any association exist between age, Sex, anthropometric measurements (Body mass Index), Hypertension, dyslipidemia, duration of diabetes, smoking, and coronary artery diseases with serum uric acid levels.

MATERIAL & METHODS:

Study Design: Hospital-based, cross-sectional study.
Study area: The study was conducted in the Department of General Medicine, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, Karnataka.
Study period: July 2024 to June 2025.
Study population: Patients with type 2 diabetes mellitus irrespective of their glycaemic levels Of the Department of Medicine.
Sample size: The study consisted of a total of 100 subjects. 70 cases and 30 controls.
Sampling Technique: Simple Random technique.

Inclusion Criteria:

1. Patients with type 2 diabetes mellitus irrespective of their glycemic levels.
2. Age above 40 years were included in study
3. Both sexes were included.

Exclusion criteria:

1. Patients with renal failure.
2. Pregnancy & lactating mothers.
3. Patients who were on diuretics
4. Patients who were using steroids
5. Patients with hepatic failure
6. Patients with tuberculosis, CVA.

7. Patients who were chronic alcoholics.

Ethical consideration: Institutional Ethical Committee permission was obtained before the commencement of the study.

Study tools and Data collection procedure:
The required data were obtained from both cases and controls using a pre-designed and structured proforma. Sociodemographic details including age and sex were recorded for all study participants. Clinical data were collected through detailed history taking and physical examination. Anthropometric measurements such as body weight and height were recorded, and body mass index (BMI) was calculated using the standard formula (weight in kilograms divided by height in meters squared). Blood pressure was measured using a standard sphygmomanometer under resting conditions.

A comprehensive clinical history was obtained, including duration of diabetes mellitus, family history of diabetes, drug history (including antidiabetic and other relevant medications), and smoking history. Information regarding the presence of ischemic heart disease was documented based on clinical records and relevant investigations. All participants underwent fundus examination to assess for diabetic retinopathy. A detailed neurological examination was performed to evaluate peripheral neuropathy using standard clinical methods.

Laboratory Methods:
Venous blood samples were collected after an overnight fast under aseptic precautions. Fasting blood sugar (FBS) and postprandial blood sugar (PPBS) levels were estimated using the glucose oxidase–peroxidase (GOD/POD) enzymatic method. Serum lipid profile parameters, including total cholesterol, triglycerides, high-density lipoprotein (HDL), and low-density lipoprotein (LDL), were analyzed using standard enzymatic procedures.

Serum creatinine estimation was performed using the alkaline picrate (Jaffe's) method. The principle of this method involves two main steps. In the first step, deproteinization was carried out by adding 0.2 mL of serum to 1 mL of distilled water, followed by 0.4 mL of 2/3 N sulfuric acid (H_2SO_4) and 0.4 mL of 10% sodium tungstate. The mixture was centrifuged for 5 minutes to obtain a protein-free filtrate. In the second step, 1 mL of the filtrate was mixed with 0.75 N sodium hydroxide and 0.5 mL of saturated picric acid. After thorough mixing and incubation for 15 minutes, the intensity of the colored complex formed was measured calorimetrically at 490 nm.

Serum uric acid levels were estimated using the phosphotungstic acid method. The principle of this method is based on the oxidation of uric acid by uricase to form allantoin and hydrogen peroxide. In the presence of peroxidase (POD), hydrogen peroxide oxidizes the chromogen to produce a red-colored compound, the intensity of which is proportional to the concentration of uric acid in the sample. The absorbance was measured spectrophotometrically at 500 nm.

Statistical Analysis:
All collected data were entered into a Microsoft Excel spreadsheet and subsequently analyzed using Statistical Package for the Social Sciences (SPSS) software. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as proportions and percentages. The student's t-test was applied to compare mean values between cases and controls. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 100 subjects were included in the study, comprising 70 cases (Type 2 Diabetes Mellitus) and 30 controls (non-diabetic).

Table 1: Baseline Demographic Characteristics of Study Population

Variable	Cases (n=70)	Controls (n=30)	p-value
Mean Age (years)	60.84 ± 8.98	59.03 ± 8.76	0.3545
Male	48 (68.6%)	20 (66.7%)	0.8523
Female	22 (31.4%)	10 (33.3%)	

There was no statistically significant difference between cases and controls with respect to age and gender distribution. The groups were comparable demographically.

Table 2: Comparison of Body Mass Index (BMI)

Variable	Cases (n=70)	Controls (n=30)	p-value
Mean BMI (kg/m²)	25.10 ± 3.16	23.00 ± 2.25	0.0014

BMI was significantly higher among diabetic patients compared to controls. This indicates a strong association between Type 2 Diabetes Mellitus and increased body mass index.

Table 3: Glycemic Profile Distribution

Among Cases

Parameter	Mean ± SD
Fasting Blood Sugar (mg/dl)	146.11 ± 20.26
Postprandial Blood Sugar (mg/dl)	209.8 ± 44.31

Diabetic patients showed significantly elevated fasting and postprandial blood glucose levels consistent with poor glycemic control.

Table 4: Comparison of Serum Uric Acid Levels

Variable	Cases (n=70)	Controls (n=30)	p-value
Serum Uric Acid (mg/dl)	5.8 ± 1.408	3.85 ± 0.7167	<0.0001

Serum uric acid levels were significantly higher in diabetic patients compared to non-diabetic controls, demonstrating a strong association between Type 2 Diabetes Mellitus and elevated uric acid levels.

Table 5: Prevalence of Hyperuricemia (Hyperuricemia defined as ≥7 mg/dl)

Group	Hyperuricemia Present	Hyperuricemia Absent	p-value
Cases (n=70)	19 (27.1%)	51 (72.9%)	0.0016
Controls (n=30)	0	30 (100%)	

Hyperuricemia was significantly more prevalent among diabetic patients, whereas none of the controls exhibited elevated uric acid levels.

Table 6: Serum Uric Acid in Relation to BMI and Smoking (Cases Only)

BMI Category	Mean SUA (mg/dl)	p-value
BMI < 25	5.12 ± 0.88	
BMI ≥ 25	6.382 ± 1.509	0.0001
Smoking Status	Mean SUA (mg/dl)	p-value
Smokers	6.54 ± 1.52	NS
Non-smokers	5.5 ± 1.24	

Serum uric acid showed a significant positive correlation with BMI. However, although smokers had higher mean uric acid levels, the difference was not statistically significant. Table 7: Serum Uric Acid in Relation to Hypertension and Dyslipidemia

Status	Mean SUA (mg/dl)	p-value
Hypertensive	7.24 ± 1.3225	<0.0001
Normotensive	5.15 ± 0.845	
Status	Mean SUA (mg/dl)	p-value
Abnormal Lipid Profile	7.8 ± 0.365	<0.0001
Normal Lipid Profile	5.11 ± 0.8367	

Serum uric acid levels were significantly elevated in patients with hypertension and dyslipidemia. This strongly supports the association between hyperuricemia and cardiovascular risk factors.

Table 8: Serum Uric Acid in Relation to CAD and Duration of Diabetes

A. Coronary Artery Disease (CAD)

Condition	Hyperuricemia Present
Ischemia (n=15)	4 cases
Infarction (n=11)	6 cases

B. Duration of Diabetes

Duration	Mean SUA (mg/dl)	p-value
2–5 years	4.97 ± 0.8235	
>10 years	7.32 ± 0.98	<0.0001

Hyperuricemia was more common in patients with myocardial infarction compared to ischemia. Additionally, serum uric acid levels significantly increased with longer duration of diabetes, indicating progressive metabolic involvement.

OVERALL SUMMARY OF FINDINGS:

Serum uric acid levels were significantly higher in Type 2 Diabetes Mellitus patients.
Hyperuricemia was strongly associated with: Higher BMI, Hypertension, Dyslipidemia, Longer duration of diabetes, Coronary artery disease, No significant association was observed with gender or smoking. These findings suggest that serum uric acid may serve as a useful marker for cardiovascular risk stratification in patients with Type 2 Diabetes Mellitus.

DISCUSSION

Type 2 Diabetes Mellitus (T2DM) is a major non-communicable disease contributing substantially to global morbidity and mortality. India bears a significant burden of diabetes, with rapidly increasing prevalence over recent decades[1]. In addition to microvascular complications, diabetes is a well-recognized risk factor for macrovascular disease, particularly coronary artery disease (CAD)[1]. Emerging evidence suggests that serum uric acid (SUA) may play an important contributory role in cardiovascular pathophysiology and metabolic syndrome components[2], [4]. The present study was undertaken to evaluate serum uric acid levels in patients with Type 2 Diabetes Mellitus and to examine their association with established cardiovascular risk factors including obesity, hypertension, dyslipidemia, smoking, duration of diabetes, and coronary artery disease.

Age and Gender Distribution

In the current study, the mean age of cases and controls did not differ significantly ($p = 0.3545$), indicating appropriate matching and comparability between groups. Although some longitudinal population-based studies from Japan have demonstrated increasing SUA levels with advancing age, after controlling for BMI and alcohol intake, no significant age-related variation was observed in this cohort. Regarding gender distribution, no statistically significant difference in serum uric acid levels was observed between males and females in this study. This finding contrasts with the Novara Atherosclerosis Study (NAS), which reported higher uric acid levels among males9. Similarly, Pavani Bandaru and Anoop (2011)10 observed elevated uric acid levels in males, attributing this to estrogen-mediated enhancement of uric acid clearance in females. However, findings from Shantan Venishetty et al. (2017)11 showed no significant gender-based difference, which aligns with the observations of the present study.

Duration of Diabetes and Serum Uric Acid

A significant positive correlation was observed between duration of diabetes and serum uric acid levels ($p < 0.0001$), with higher mean SUA levels among patients with diabetes duration exceeding 10 years. This suggests that chronic hyperglycemia and prolonged metabolic dysfunction may contribute to progressive uric acid elevation. Tiange Wang et al. (2011)12 demonstrated that serum uric acid independently predicted the development of Type 2 diabetes in middle-aged and elderly Chinese populations. Earlier studies by Yoo et al.13 and Becker and Jolly14 reported that hyperglycemia is a significant risk factor for hyperuricemia. Nakanishi et al. also found a positive correlation between hyperuricemia and hyperglycemia in Japanese adults. Tang et al.15 observed that higher SUA levels were associated with better early β-cell function but more rapid functional decline over time. These findings collectively support the complex interplay between uric acid metabolism and glycemic control.

Obesity and Serum Uric Acid

The present study demonstrated a statistically significant association between elevated BMI (>25 kg/m²) and higher serum uric acid levels. This is consistent with the concept that hyperuricemia forms part of the metabolic syndrome spectrum[4]. Ming-Yun Chen et al. (2016)16 reported that SUA levels increased proportionally with obesity severity. Mechanistically, obesity-associated insulin resistance reduces renal urate excretion and enhances purine metabolism. Elevated leptin levels in metabolic syndrome have also been implicated in the regulation of serum uric acid concentrations17-19. Furthermore, insulin resistance is associated with multiple metabolic alterations including increased uric acid, ApoB, prothrombotic factors, and microalbuminuria20,21.

Hypertension and Serum Uric Acid

A significant association was found between hypertension and elevated serum uric acid levels in this study. Mean SUA levels were substantially higher in hypertensive diabetic patients compared to normotensive individuals. Masanari Kuwabara et al. (2014)22 reported a positive correlation between SUA and hypertension even after adjusting for BMI and dyslipidemia. Uric acid has been implicated in endothelial dysfunction through oxidative stress mechanisms23,24. It promotes LDL oxidation, vascular smooth muscle proliferation, and nitric oxide reduction, thereby contributing to vascular stiffness and atherosclerosis25,26. Johnson et al. have described uric acid as a potential causal factor in essential hypertension[25].

Dyslipidemia and Serum Uric Acid

A highly significant association was observed between dyslipidemia and elevated serum uric acid levels in the present study. Hypertriglyceridemia was the predominant lipid abnormality observed. Sarmah D et al.27 demonstrated positive correlations between SUA and total cholesterol, triglycerides, and LDL, with a negative correlation with HDL. Tavish Arora et al.28 also reported higher SUA levels in patients with low HDL. Qin Li et al. found associations between SUA, metabolic syndrome, and carotid atherosclerosis in T2DM patients. Hyperuricemia may induce proinflammatory endocrine imbalance in adipose tissue, thereby exacerbating insulin resistance and dyslipidemia. Increased uric acid enhances lipid

peroxidation and promotes endothelial dysfunction, further accelerating atherosclerosis.

Coronary Artery Disease and Serum Uric Acid

Among patients with CAD, hyperuricemia was more prevalent in those with myocardial infarction compared to those with ischemia. This suggests a potential role of uric acid in plaque instability and thrombotic events. Earlier studies by Kohn and Prozan, Beard, and Torun et al. reported associations between hyperuricemia and myocardial infarction. Seo Young Kim et al. (2010)29, in a meta-analysis, reported a 12% increase in CHD mortality for every 1 mg/dL increase in SUA, with stronger association observed in women. The PIUMA and SHEP studies also demonstrated a link between elevated uric acid and cardiovascular outcomes.

Smoking and Serum Uric Acid

No statistically significant association was observed between smoking and serum uric acid levels in the present study. Dhouha Haj Mouhamed et al. (2011)30 reported lower SUA levels in smokers, possibly due to xanthine oxidase inhibition by cyanide. This paradoxical finding may reflect oxidative consumption of uric acid in smokers.

Pathophysiological Mechanisms

Hyperuricemia in T2DM may result from reduced renal urate clearance secondary to insulin resistance, increased purine synthesis via the hexose monophosphate pathway, and acidosis-mediated tubular reabsorption. Transporters such as URAT1, GLUT9, and ABCG2 play key roles in urate homeostasis31. Uric acid contributes to endothelial dysfunction, oxidative stress, inflammatory cytokine release, and LDL oxidation23,25,26. These mechanisms collectively accelerate atherosclerosis and cardiovascular morbidity.

Summary of Discussion

The present study reinforces the concept that serum uric acid is significantly elevated in patients with Type 2 Diabetes Mellitus and is positively associated with obesity, hypertension, dyslipidemia, duration of diabetes, and coronary artery disease. No significant association was observed with gender or smoking. These findings support previous literature indicating that hyperuricemia is intricately linked with metabolic syndrome and cardiovascular risk2,4,29 . Regular monitoring of serum uric acid in diabetic patients may aid in early identification of individuals at higher cardiovascular risk and facilitate timely preventive interventions.

CONCLUSION:

The present study concludes that mean serum uric acid (SUA) levels are significantly higher in patients with Type 2 Diabetes Mellitus compared to non-diabetic controls, indicating a strong association between hyperuricemia and diabetes. Serum uric acid levels did not differ significantly between males and females and were found to be independent of smoking status and family history of diabetes. However, a significant positive correlation was observed between serum uric acid levels and abnormal lipid parameters, demonstrating its close association with dyslipidemia and other metabolic disturbances. Elevated SUA levels were also significantly higher among hypertensive individuals, further emphasizing its relationship with established cardiovascular risk factors.

Furthermore, patients with coronary artery disease exhibited higher serum uric acid levels, with markedly increased values among those with myocardial infarction compared to those with ischemic heart disease alone, suggesting a possible association between hyperuricemia and severity of cardiovascular events. Males with hyperuricemia were observed to have a higher risk of coronary artery disease compared to females. Additionally, serum uric acid levels were found to increase proportionally with the duration of diabetes, indicating that prolonged metabolic dysregulation may contribute to rising uric acid levels over time. Overall, these findings support the role of serum uric acid as a useful marker of cardiovascular risk in patients with Type 2 Diabetes Mellitus.

REFERENCES:

1. Chang Z, Zhou X-H, Wen X. Association between serum uric acid levels and cardiovascular events in hospitalized patients with type 2 diabetes. Prim Care Diabetes. 2021;15(4):682-7.
2. Journal of Clinical and Preventive Cardiology. Serum uric acid as a marker of coronary artery disease in type 2 diabetes. J Clin Prev Cardiol. 2024;13(1):2-5.
3. Sethy S, Dash KC, Dash PC, et al. Study of serum uric acid in type 2 diabetes mellitus in association with cardiovascular risk factors. J Cardiovasc Dis Res. 2024;15(1).
4. Cao JY, Waldman B, O'Connell R, et al. Uric acid predicts long-term cardiovascular risk in type 2 diabetes but does not mediate the benefits of fenofibrate: The FIELD study. Diabetes Obes Metab. 2020;22(8):1388-96.
5. Kumar TS, Ale J. Type 2 diabetes mellitus and serum uric acid as an indicator of coronary artery disease: a cross-sectional study. J Cardiovasc Dis Res. 2023;14(12).
6. He Y, Feng J, Zhang B, et al. Serum uric acid levels and risk of cardiovascular disease in type 2 diabetes: results from a cross-sectional study and Mendelian randomization analysis. Front Endocrinol (Lausanne). 2023; 14:1251451.
7. Zhao M, Ma X, Xia Z, et al. Association between serum uric acid and dyslipidaemia in type 2 diabetes: a cross-sectional study. BMJ Open. 2025; XX(X): XXXX.
8. Association of serum uric acid-to-HDL cholesterol ratio with myocardial infarction in diabetes. Eur J Med Res. 2025; XX(X): XXXX.

9. De Luca G, Novara Atherosclerosis Study Group. Impact of sex on serum uric acid levels and extent of coronary artery disease. Atherosclerosis. 2015.
10 Bandaru P, Shankar A. Association of serum uric acid and diabetes mellitus. Int J Endocrinol. 2011;2011:604715.
11. Venishetty S, Bhat R, Rajagopal KV. Serum uric acid levels in type 2 diabetes mellitus and carotid atherosclerosis. 2017.
12. Wang T, Bi Y, et al. Serum uric acid associates with incidence of type 2 diabetes. Endocrine. 2011;40:109-116.
13. Yoo TW, Sung KC, Shin HS, et al. Relationship between serum uric acid and metabolic syndrome. Circ J. 2005;69:928-933.
14. Becker MA, Jolly M. Hyperuricemia and associated diseases. Rheum Dis Clin North Am. 2006;32(2):275-293.
15. Chen JH, Chuang SY, et al. Serum uric acid level as independent risk factor for mortality. 2009.
16. Chen MY, Zhan CC. Serum uric acid levels are associated with obesity but not cerebrovascular events. 2017;7:40009.
17. Conen D, Wietlisbach V, Bovet P, et al. BMC Public Health. 2004;4:9.
18. Ogura T, Matsuura K, Matsumoto Y, et al. Recent trends of hyperuricemia and obesity. Metabolism. 2004;53:448-453.
19. Pan WH, Flegal KM, Chang HY, et al. BMI and obesity-related metabolic disorders. Am J Clin Nutr. 2004;79:31-39.
20. McLaughlin T, Abbasi F, Lamendola C, Reaven G. Arch Intern Med. 2007;167(7):642.
21. Semple RK, Savage DB, Cochran EK, Gorden P, O'Rahilly S. Genetic syndromes of severe insulin resistance. Endocr Rev. 2011;32(4):498.
22. Kuwabara M, Kuwabara R, Hisatome I, et al. "Metabolically healthy" obesity and hyperuricemia increase risk for hypertension and diabetes. 2017.
23. Neogi T. Asymptomatic hyperuricemia: cardiovascular and renal complications. In: Gout and Other Crystal Arthropathies. Elsevier; 2012. p.226.
24. Johnson RJ, Feig DI, Kang DH, et al. Resurrection of uric acid as a causal risk factor for essential hypertension. Hypertension. 2005;45:18.
25. Wannamethee SG, Shaper AG, Whincup PH. Serum urate and the risk of major coronary heart disease events. Heart. 1997;78(2):147.
26. Feig DI, Kang DH, Johnson RJ. Uric acid and cardiovascular risk. N Engl J Med. 2008;359(17):1811.
27. Sarmah D, et al. A correlative study of uric acid with lipid profile. 2013.
28. Jalal DI, Chonchol M. Uric acid as a target of therapy in CKD. Am J Kidney Dis. 2013;61(1):134-146.
29. Kim SY, Guevara JP, Kim KM, et al. Hyperuricemia and coronary heart disease: systematic review and meta-analysis. Arthritis Care Res (Hoboken). 2010;62(2):170-180.
30. Haj Mouhamed D, Ezzaher A, et al. Effect of cigarette smoking on plasma uric acid concentrations. 2011;16(5):307-312.
31. Huls M, Brown CD, Windass AS, et al. The ABCG2 transporter is expressed in human kidney. 2008; 73:220.