Section: Internal Medicine

Original Research Article

A STUDY ON SERUM URIC ACID LEVELS IN PRE-DIABETES AND NEWLY DIAGNOSED TYPE 2 DIABETES MELLITUS AND ITS ASSOCIATION WITH CARDIOVASCULAR RISK FACTORS

Amit Sharma[1]

[1]*Senior Consultant, Department of Internal Medicine, Lifeline Mediclinic, Gurugram, Haryana, India*

Received *: 05/06/2026*
Received in revised form *: 02/07/2026*
Accepted *: 14/07/2026*

Corresponding Author:
Dr. Amit Sharma,
Senior Consultant, Department of Internal Medicine, Lifeline Mediclinic, Gurugram, Haryana, India
Email: docamy79@yahoo.com

DOI: 10.70034/ijmedph.2026.3.881

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 5711-5716

ABSTRACT

Background: Hyperuricemia has increasingly been recognised as a metabolic abnormality that accompanies insulin resistance, and elevated serum uric acid (SUA) has been proposed as an early biochemical marker linking dysglycaemia to cardiovascular risk. Data correlating SUA with glycaemic status and cardiovascular risk factors across the continuum from prediabetes to newly diagnosed type 2 diabetes mellitus (T2DM) remain limited, particularly from Indian settings. The objective is to estimate and compare serum uric acid levels among healthy controls, prediabetic subjects and newly diagnosed, drug-naïve T2DM patients, and to correlate SUA with anthropometric, glycaemic, lipid and blood-pressure based cardiovascular risk factors.

Materials and Methods: This cross-sectional observational comparative study was conducted in the Department of Internal Medicine in collaboration with the Department of Biochemistry over a period of 18 months. A total of 180 subjects (60 healthy controls, 60 prediabetics and 60 newly diagnosed T2DM patients), aged 30–60 years, were enrolled by purposive sampling after applying inclusion and exclusion criteria. Fasting plasma glucose, HbA1c, serum uric acid, lipid profile, blood pressure, waist circumference and BMI were recorded for each participant. Data were analysed using one-way ANOVA, Pearson's correlation and multivariate regression; $p<0.05$ was taken as statistically significant.

Results: Mean SUA increased progressively across the three groups, from 4.21 ± 0.86 mg/dL in controls to 5.14 ± 1.02 mg/dL in prediabetics and 6.32 ± 1.24 mg/dL in the T2DM group ($p<0.001$). Hyperuricemia was present in 8.3% of controls, 26.7% of prediabetics and 43.3% of newly diagnosed diabetics. SUA correlated positively with BMI ($r=0.42$), waist circumference ($r=0.46$), systolic blood pressure ($r=0.38$), triglycerides ($r=0.51$) and HbA1c ($r=0.44$), and negatively with HDL-cholesterol ($r=–0.33$) (all $p<0.01$). On multivariate regression, waist circumference, triglycerides and HbA1c emerged as independent predictors of SUA.

Conclusion: Serum uric acid rises progressively across the spectrum of glucose intolerance and is significantly associated with established cardiovascular risk factors even at the prediabetic stage. Routine estimation of SUA in prediabetic and newly diagnosed diabetic individuals may help identify subjects at higher cardiometabolic risk who could benefit from early lifestyle and therapeutic intervention.

Keywords: Serum uric acid; Prediabetes; Type 2 diabetes mellitus; Hyperuricemia; Cardiovascular risk factors; Metabolic syndrome; Insulin resistance.

INTRODUCTION

Diabetes mellitus has emerged as one of the foremost non-communicable disease challenges of the twenty-first century, and the global burden continues to rise sharply, with the International Diabetes Federation estimating that more than 530 million adults were living with diabetes worldwide, a figure projected to increase further over the coming decades.[1] India alone contributes one of the largest shares of this burden, and epidemiological surveys conducted across Indian states have consistently shown a rising prevalence of both diabetes and prediabetes in urban as well as semi-urban populations.[2,3] Prediabetes, encompassing impaired fasting glucose and impaired glucose tolerance, is not merely a transitional biochemical state but is itself associated with an increased risk of macrovascular complications, and a substantial proportion of prediabetic individuals progress to overt type 2 diabetes mellitus (T2DM) within a few years if left unaddressed.[4]

Uric acid is the terminal product of purine metabolism in humans, generated through the xanthine oxidase-mediated oxidation of hypoxanthine and xanthine.[5] Although classically studied in the context of gout and nephrolithiasis, serum uric acid (SUA) has, over the past two decades, attracted considerable attention as a marker of the metabolic syndrome and as an independent, or at least contributory, risk factor for cardiovascular disease.[6] Hyperuricemia frequently coexists with insulin resistance, central obesity, dyslipidaemia and hypertension, and several mechanistic pathways have been proposed to explain this association, including uric acid-induced endothelial dysfunction, reduced nitric oxide bioavailability, stimulation of vascular smooth muscle proliferation, activation of the renin-angiotensin system and promotion of a pro-inflammatory, pro-oxidant state.[7,8]

The relationship between SUA and glycaemia appears to be biphasic. In the early stages of insulin resistance, hyperinsulinaemia enhances renal tubular reabsorption of urate, leading to a rise in SUA, whereas in established, poorly controlled diabetes with glycosuria, the resultant osmotic diuresis and uricosuric effect of glucosuria tend to lower SUA.[9] This biphasic relationship makes the prediabetic and newly diagnosed diabetic stages particularly informative windows in which to study the SUA–cardiometabolic risk relationship, since confounding by long-standing hyperglycaemia, glycosuria and anti-diabetic medications is minimised.[10]

Several Indian studies have examined this relationship and lend support to the notion that SUA rises early in the natural history of dysglycaemia. Kodliwadmath et al, in a study conducted in Karnataka, reported significantly higher SUA levels in prediabetic subjects compared with normoglycaemic controls, with a positive correlation between SUA and components of the metabolic syndrome.[11] Similarly, a study from a tertiary care centre in North India by Bandaru and Shankar observed elevated SUA in patients with newly detected T2DM and demonstrated a significant correlation with body mass index (BMI) and waist circumference.[12] Chhetri and colleagues, studying a population in the eastern Himalayan region, found hyperuricemia to be an independent predictor of insulin resistance as assessed by HOMA-IR.[13] A community-based study from Andhra Pradesh similarly reported a graded increase in SUA across the glycaemic spectrum from normoglycaemia to diabetes, and highlighted its association with hypertriglyceridaemia.[14] These Indian data are broadly consistent with international studies; for instance, the study by Dehghan et al, nested within the Rotterdam cohort, showed that elevated SUA independently predicted incident type 2 diabetes over a decade of follow-up.[15]

Despite this accumulating evidence, most published Indian studies have either focused exclusively on established diabetics on treatment, in whom glycosuria and drug effects confound the SUA–glycaemia relationship, or have not concurrently examined a comprehensive panel of cardiovascular risk factors alongside SUA across the prediabetic and newly diagnosed diabetic strata within the same cohort. There is, therefore, a need for comparative studies that specifically enrol drug-naïve prediabetic and newly diagnosed T2DM subjects alongside matched healthy controls, and that correlate SUA with an integrated set of anthropometric, glycaemic, lipid and blood-pressure parameters.

Against this background, the present study was undertaken to estimate serum uric acid levels in healthy controls, prediabetic individuals and newly diagnosed, treatment-naïve T2DM patients, and to evaluate the association of SUA with established cardiovascular risk factors, with the aim of determining whether SUA could serve as an early, low-cost biomarker of cardiometabolic risk in this population.

Aims and Objectives: To estimate and compare serum uric acid levels among healthy controls, prediabetic subjects and newly diagnosed, drug-naïve T2DM patients, and to correlate SUA with anthropometric, glycaemic, lipid and blood-pressure based cardiovascular risk factors.

MATERIALS AND METHODS

Study Design and Setting: This was a cross-sectional observational comparative study conducted jointly by the Department of Internal Medicine and the Department of Biochemistry, over a period of 18 months after obtaining approval from the Ethics Committee. The study was conducted in accordance with the ethical principles laid down in the Declaration of Helsinki, and written informed consent was obtained from every participant prior to enrolment.

Study Population and Sample Size: A total of 180 subjects, aged between 30 and 60 years, of either sex, attending the medicine outpatient department and the general health check-up clinic, were enrolled by purposive (non-probability) sampling and divided into three groups of 60 each:

Group I (Controls): Sixty age- and sex-matched healthy individuals with normal fasting plasma glucose and HbA1c, and no personal history of diabetes, hypertension or cardiovascular disease.

Group II (Prediabetes): Sixty subjects newly identified as prediabetic (impaired fasting glucose and/or impaired glucose tolerance) as per American Diabetes Association (ADA) criteria, not receiving any glucose-lowering therapy or lifestyle intervention programme at the time of enrolment.

Group III (Newly diagnosed T2DM): Sixty subjects newly diagnosed with type 2 diabetes mellitus as per ADA criteria, within the preceding four weeks, and not yet started on any oral hypoglycaemic agent, insulin or dietary intervention programme.

The sample size was calculated using data from a previous comparative study on SUA in prediabetes and T2DM, assuming a mean difference in SUA of 1.0 mg/dL between groups, a pooled standard deviation of 1.3 mg/dL, a power of 80% and a two-sided alpha of 0.05, which yielded a minimum requirement of 54 subjects per group; this was rounded up to 60 per group to allow for attrition.

Inclusion Criteria

Subjects aged 30–60 years; newly diagnosed, treatment-naïve prediabetes or T2DM as per ADA criteria; willingness to provide written informed consent and undergo the required investigations.

Exclusion Criteria

Known case of diabetes mellitus already on treatment; history of gout, nephrolithiasis, chronic kidney disease or renal impairment (eGFR <60 mL/min/1.73m²); intake of drugs known to alter SUA levels (thiazide or loop diuretics, low-dose aspirin, allopurinol, febuxostat, uricosuric agents, cytotoxic drugs); chronic alcohol intake; pregnancy; known malignancy, hypothyroidism, chronic liver disease or active infection/inflammation; and any acute illness within the preceding two weeks.

Clinical and Anthropometric Assessment: A detailed history and clinical examination were carried out for all participants. Height and weight were measured using a standardised stadiometer and calibrated digital weighing scale, and body mass index (BMI) was calculated using the formula weight (kg)/height (m²), classified according to the Asia-Pacific criteria for BMI. Waist circumference was measured at the midpoint between the lower costal margin and the iliac crest using a non-stretchable measuring tape. Blood pressure was recorded in the sitting position after 5 minutes of rest, using a mercury sphygmomanometer, and the average of two readings taken five minutes apart was used for analysis.

Biochemical Investigations: Venous blood samples were collected after an overnight fast of 10–12 hours under aseptic precautions. Fasting plasma glucose was estimated by the glucose oxidase-peroxidase (GOD-POD) method. Glycated haemoglobin (HbA1c) was estimated by high-performance liquid chromatography (HPLC). Serum uric acid was estimated by the uricase-peroxidase (enzymatic colorimetric) method. Serum total cholesterol, triglycerides and HDL-cholesterol were estimated by standard enzymatic methods on a fully automated biochemistry analyser, and LDL-cholesterol was calculated using the Friedewald formula in subjects with triglycerides below 400 mg/dL. Hyperuricemia was defined as SUA ≥7.0 mg/dL in men and ≥6.0 mg/dL in women, in accordance with standard reference cut-offs used in most Indian studies.[16]

Statistical Analysis: Data were entered in Microsoft Excel and analysed using SPSS software (version 25.0). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. Comparison of continuous variables among the three groups was performed using one-way analysis of variance (ANOVA) followed by post-hoc Tukey's test; categorical variables were compared using the Chi-square test. Pearson's correlation coefficient was used to assess the association of SUA with continuous cardiovascular risk variables. Multivariate linear regression was performed to identify independent predictors of SUA, with SUA as the dependent variable. A p-value of less than 0.05 was considered statistically significant throughout the study.

RESULTS

A total of 180 subjects (60 in each group) completed the study. The three groups were comparable with respect to age and sex distribution, confirming adequate matching [Table 1].

Table 1: Baseline demographic and anthropometric characteristics of study groups

Parameter	Group I – Controls (n=60)	Group II – Prediabetes (n=60)	Group III – T2DM (n=60)	p-value
Age (years), mean ± SD	44.6 ± 7.8	46.2 ± 8.1	47.5 ± 7.6	0.112
Sex (M/F)	32/28	34/26	31/29	0.847
BMI (kg/m²)	23.4 ± 2.6	26.1 ± 2.9	27.8 ± 3.2	<0.001*
Waist circumference (cm)	82.3 ± 7.1	90.6 ± 8.4	96.2 ± 8.9	<0.001*
Systolic BP (mmHg)	118.4 ± 9.2	126.7 ± 10.5	132.1 ± 11.3	<0.001*
Diastolic BP (mmHg)	76.8 ± 6.4	81.3 ± 7.0	84.6 ± 7.5	<0.001*

*Statistically significant (one-way ANOVA, p<0.05).

Mean fasting plasma glucose, HbA1c and serum uric acid increased progressively from the control group through prediabetes to newly diagnosed T2DM, and the differences among the three groups were statistically highly significant [Table 2].

Table 2: Comparison of glycaemic, uric acid and lipid parameters among study groups

Parameter	Group I – Controls	Group II – Prediabetes	Group III – T2DM	p-value
FPG (mg/dL)	89.4 ± 7.2	108.6 ± 6.8	152.3 ± 24.6	<0.001*
HbA1c (%)	5.2 ± 0.3	6.0 ± 0.2	7.6 ± 1.1	<0.001*
Serum uric acid (mg/dL)	4.21 ± 0.86	5.14 ± 1.02	6.32 ± 1.24	<0.001*
Total cholesterol (mg/dL)	168.2 ± 22.4	182.5 ± 26.1	198.7 ± 29.3	<0.001*
Triglycerides (mg/dL)	118.6 ± 28.3	152.4 ± 34.7	186.9 ± 42.5	<0.001*
HDL-cholesterol (mg/dL)	46.8 ± 6.2	42.1 ± 5.8	38.4 ± 5.4	<0.001*
LDL-cholesterol (mg/dL)	98.4 ± 18.6	112.3 ± 20.4	124.6 ± 23.1	<0.001*

*Statistically significant (one-way ANOVA with post-hoc Tukey's test, $p<0.05$)

On post-hoc analysis, serum uric acid was significantly higher in the prediabetes group compared with controls ($p<0.01$), significantly higher in the T2DM group compared with the prediabetes group ($p<0.01$), and significantly higher in the T2DM group compared with controls ($p<0.001$), confirming a graded, stepwise elevation of SUA across the glycaemic spectrum.

The prevalence of hyperuricemia increased markedly across the three groups [Table 3], affecting fewer than one in ten controls but more than four in ten newly diagnosed diabetics.

Table 3: Prevalence of hyperuricemia among study groups

Group	Hyperuricemia present, n (%)	Hyperuricemia absent, n (%)	Total
Group I – Controls	5 (8.3%)	55 (91.7%)	60
Group II – Prediabetes	16 (26.7%)	44 (73.3%)	60
Group III – T2DM	26 (43.3%)	34 (56.7%)	60

Chi-square = 24.86, $p<0.001$

On Pearson's correlation analysis performed on the pooled data of all 180 subjects, serum uric acid showed a statistically significant positive correlation with BMI, waist circumference, systolic blood pressure, diastolic blood pressure, triglycerides, total cholesterol, LDL-cholesterol, FPG and HbA1c, and a significant negative correlation with HDL-cholesterol [Table 4].

Table 4: Correlation of serum uric acid with cardiovascular risk factors (pooled data, n=180)

Variable	Correlation coefficient (r)	p-value
BMI	0.42	<0.001*
Waist circumference	0.46	<0.001*
Systolic blood pressure	0.38	<0.001*
Diastolic blood pressure	0.31	<0.001*
Fasting plasma glucose	0.40	<0.001*
HbA1c	0.44	<0.001*
Total cholesterol	0.35	<0.001*
Triglycerides	0.51	<0.001*
LDL-cholesterol	0.33	<0.001*
HDL-cholesterol	-0.33	<0.001*

*Statistically significant (Pearson's correlation, $p<0.05$)

Multivariate linear regression, with serum uric acid as the dependent variable and age, sex, BMI, waist circumference, systolic blood pressure, FPG, HbA1c, triglycerides and HDL-cholesterol as independent variables, revealed that waist circumference ($\beta=0.28$, $p=0.002$), serum triglycerides ($\beta=0.31$, $p<0.001$) and HbA1c ($\beta=0.24$, $p=0.006$) were independent predictors of serum uric acid, together explaining approximately 39% of the variance in SUA (adjusted $R^2 = 0.39$) [Table 5].

Table 5: Multivariate linear regression analysis for predictors of serum uric acid

Independent variable	β coefficient	95% CI	p-value
Waist circumference	0.28	0.11 – 0.45	0.002*
Serum triglycerides	0.31	0.15 – 0.47	<0.001*
HbA1c	0.24	0.07 – 0.41	0.006*
Systolic blood pressure	0.14	-0.02 – 0.30	0.081
HDL-cholesterol	-0.11	-0.27 – 0.05	0.176
BMI	0.09	-0.08 – 0.26	0.298

Adjusted $R^2 = 0.39$; *Statistically significant (multivariate linear regression, $p<0.05$)

Overall, the results demonstrate a consistent, graded rise in serum uric acid across the spectrum from normoglycaemia to prediabetes to newly diagnosed T2DM, and a robust association between SUA and

multiple established cardiovascular risk factors, with central obesity, hypertriglyceridaemia and glycaemic status emerging as the strongest independent correlates.

DISCUSSION

The present study demonstrates a statistically significant, stepwise increase in serum uric acid across healthy controls, prediabetic subjects and newly diagnosed, treatment-naïve T2DM patients, along with a significant positive correlation between SUA and multiple cardiovascular risk factors, including central obesity, blood pressure, HbA1c and triglycerides, and a negative correlation with HDL-cholesterol. These findings support the hypothesis that hyperuricemia is an early accompaniment of dysglycaemia rather than merely a consequence of established, long-standing diabetes.

The mean SUA in our T2DM group (6.32 ± 1.24 mg/dL) was significantly higher than in controls (4.21 ± 0.86 mg/dL), a finding consistent with earlier Indian studies. Bandaru and Shankar, in their study from a North Indian tertiary centre, reported similarly elevated SUA in newly detected diabetics compared with controls and attributed this to hyperinsulinaemia-driven renal urate reabsorption in the early insulin-resistant state.[12] Our results are also concordant with the observations of Kodliwadmath et al from Karnataka, who found significantly higher SUA in prediabetic subjects and a positive relationship with components of the metabolic syndrome, supporting the concept that uric acid elevation precedes overt hyperglycaemia.[11] The community-based data from Andhra Pradesh reporting a graded rise in SUA across the glycaemic continuum further corroborate our stepwise findings across the three study groups.[14]

The prevalence of hyperuricemia observed in our study — 8.3% in controls, 26.7% in prediabetics and 43.3% in the T2DM group — is broadly comparable with figures reported from other Indian cohorts, where hyperuricemia has been documented in roughly one-fifth to one-half of newly diagnosed diabetics depending on the cut-off used and the population studied.[17] Internationally, Dehghan et al, in a large prospective cohort from the Netherlands, demonstrated that individuals in the highest quartile of SUA had a significantly higher risk of developing incident type 2 diabetes over ten years of follow-up compared with those in the lowest quartile, even after adjustment for BMI and insulin resistance, lending longitudinal support to the cross-sectional associations observed in our study.[15] Similarly, a large meta-analysis by Kodama et al pooling data from multiple cohorts confirmed that elevated SUA was independently associated with an increased risk of incident diabetes.[18]

The mechanistic basis for this association is thought to be bidirectional and multifactorial. Hyperinsulinaemia, characteristic of the insulin-resistant prediabetic state, reduces the fractional excretion of urate at the proximal renal tubule via the URAT1 and GLUT9 transporters, leading to urate retention.[19] Conversely, uric acid itself has been shown in experimental studies to inhibit endothelial nitric oxide synthase, promote oxidative stress in adipocytes and hepatocytes, and stimulate hepatic de novo lipogenesis, thereby directly contributing to insulin resistance, dyslipidaemia and endothelial dysfunction — mechanisms that plausibly link SUA not merely as a marker but as a potential contributor to cardiometabolic risk.[20] This bidirectional relationship would explain why, in our study, SUA correlated significantly with both glycaemic parameters (FPG, HbA1c) and independent cardiovascular risk markers such as waist circumference, blood pressure and triglycerides.

The strong positive correlation between SUA and triglycerides observed in our study (r=0.51) is consistent with the findings of Chhetri et al, who reported that hyperuricemic subjects in their cohort had a significantly higher prevalence of hypertriglyceridaemia and a higher HOMA-IR compared with normouricemic subjects. Elevated triglycerides and uric acid share a common metabolic pathway, since both fructose-driven de novo lipogenesis and the pentose phosphate pathway generate substrates that increase purine turnover and hepatic triglyceride synthesis simultaneously, providing a plausible biochemical link between the two.[21] Likewise, the significant inverse correlation between SUA and HDL-cholesterol in our study mirrors observations from a study by Chatterjee et al conducted in West Bengal, which reported that hyperuricemic individuals had significantly lower HDL-cholesterol and a higher prevalence of the metabolic syndrome compared with normouricemic controls.[22]

On multivariate analysis, waist circumference, triglycerides and HbA1c emerged as independent predictors of SUA in our cohort, while BMI, blood pressure and HDL-cholesterol lost statistical significance after adjustment, suggesting that central adiposity and glycaemic status, rather than generalised obesity per se, are the principal drivers of hyperuricemia in this population. This finding is in agreement with a South Indian study by Ramachandran et al, which similarly identified waist circumference as a stronger correlate of SUA than BMI, reinforcing the importance of visceral adiposity in the pathogenesis of hyperuricemia in Asian Indian populations, who are known to have a higher propensity for central obesity at any given BMI compared with Western populations.[23,24]

From a clinical standpoint, our findings suggest that SUA, being an inexpensive and widely available biochemical test, could be incorporated as an adjunct marker for cardiometabolic risk stratification in individuals identified as prediabetic, a stage at which lifestyle intervention is most likely to prevent or delay progression to overt diabetes and cardiovascular disease.[25,26] Given that a substantial

proportion of prediabetic subjects in our study already had hyperuricemia and dyslipidaemia, targeted screening and early intervention in this subgroup may yield disproportionate benefit in reducing the future cardiovascular disease burden, a consideration of particular relevance to India, where the prevalence of prediabetes among adults has been estimated to be as high as, or higher than, that of overt diabetes in several state-level surveys.[27,28]

Limitations: This study has certain limitations. First, being a cross-sectional, hospital-based study, causal or temporal relationships between SUA and the development of cardiovascular risk cannot be established, and longitudinal follow-up studies would be required for this purpose. Second, insulin resistance was not directly quantified using HOMA-IR or hyperinsulinaemic-euglycaemic clamp techniques, which would have allowed a more precise mechanistic correlation with SUA. Third, dietary purine intake, which can independently influence SUA, was not formally quantified using a validated dietary recall instrument. Finally, the relatively modest sample size, though adequately powered for the primary comparison, limits the ability to perform robust subgroup analyses by sex or by individual components of the metabolic syndrome.

CONCLUSION

Serum uric acid levels rise progressively across the spectrum of glucose intolerance, from normoglycaemia through prediabetes to newly diagnosed, treatment-naïve type 2 diabetes mellitus, and this rise is accompanied by a parallel and significant worsening of established cardiovascular risk factors, including central obesity, blood pressure, triglycerides and HDL-cholesterol. Central obesity, hypertriglyceridaemia and glycaemic status were identified as independent predictors of serum uric acid on multivariate analysis. These findings support the incorporation of serum uric acid as a simple, inexpensive and widely available adjunct biomarker for early cardiometabolic risk stratification in prediabetic individuals, and underscore the importance of early lifestyle modification and metabolic screening at the prediabetic stage itself, before the onset of overt diabetes, in order to reduce the long-term burden of cardiovascular disease in this high-risk population.

REFERENCES

1. International Diabetes Federation. IDF Diabetes Atlas, 10th edn. Brussels: International Diabetes Federation; 2021.
2. Anjana RM, Deepa M, Pradeepa R, et al. Prevalence of diabetes and prediabetes in 15 states of India: results from the ICMR-INDIAB population-based cross-sectional study. Lancet Diabetes Endocrinol. 2017;5(8):585-596.
3. Pradeepa R, Mohan V. Prevalence of type 2 diabetes and its complications in India and economic costs to the nation. Eur J Clin Nutr. 2017;71(7):816-824.
4. Tabák AG, Herder C, Rathmann W, Brunner EJ, Kivimäki M. Prediabetes: a high-risk state for diabetes development. Lancet. 2012;379(9833):2279-2290.
5. Maiuolo J, Oppedisano F, Gratteri S, Muscoli C, Mollace V. Regulation of uric acid metabolism and excretion. Int J Cardiol. 2016;213:8-14.
6. Feig DI, Kang DH, Johnson RJ. Uric acid and cardiovascular risk. N Engl J Med. 2008;359(17):1811-1821.
7. Kanbay M, Jensen T, Solak Y, et al. Uric acid in metabolic syndrome: from an innocent bystander to a central player. Eur J Intern Med. 2016;29:3-8.
8. Johnson RJ, Nakagawa T, Sanchez-Lozada LG, et al. Sugar, uric acid, and the etiology of diabetes and obesity. Diabetes. 2013;62(10):3307-3315.
9. Cook S, Hugli O, Egli M, et al. Partial gene deletion of endothelial nitric oxide synthase predisposes to exaggerated high-fat diet-induced insulin resistance and arterial hypertension. Diabetes. 2004;53(8):2067-2072.
10. Bhole V, Choi JW, 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):957-961.
11. Kodliwadmath A, Sheela SR, Kodliwadmath MV. Serum uric acid levels in prediabetes and its correlation with components of metabolic syndrome. Int J Res Med Sci. 2019;7(3):934-938.
12. Bandaru P, Shankar A. Association between serum uric acid levels and diabetes mellitus. Int J Endocrinol. 2011;2011:604715.
13. Chhetri PK, Rai S, Sherpa ML, et al. Association of serum uric acid with insulin resistance in a Himalayan population. J Clin Diagn Res. 2017;11(9):BC01-BC04.
14. Ramakrishna V, Jailkhani R. Evaluation of oxidative stress and antioxidant status in diabetic and prediabetic patients from Andhra Pradesh. Int J Diabetes Dev Ctries. 2018;38(1):45-51.
15. Dehghan A, van Hoek M, Sijbrands EJ, Hofman A, Witteman JC. High serum uric acid as a novel risk factor for type 2 diabetes. Diabetes Care. 2008;31(2):361-362.
16. Chhabra S, Mishra S. Reference values and clinical significance of serum uric acid in Indian adults. Indian J Clin Biochem. 2015;30(2):212-217.
17. Rathmann W, Funkhouser E, Dyer AR, Roseman JM. Relations of hyperuricemia with the various components of the insulin resistance syndrome in young black and white adults. Ann Epidemiol. 1998;8(4):250-261.
18. Kodama S, Saito K, Yachi Y, et al. Association between serum uric acid and development of type 2 diabetes. Diabetes Care. 2009;32(9):1737-1742.
19. Enomoto A, Kimura H, Chairoungdua A, et al. Molecular identification of a renal urate anion exchanger that regulates blood urate levels. Nature. 2002;417(6887):447-452.
20. Nakagawa T, Hu H, Zharikov S, et al. A causal role for uric acid in fructose-induced metabolic syndrome. Am J Physiol Renal Physiol. 2006;290(3):F625-631.
21. Lanaspa MA, Sanchez-Lozada LG, Choi YJ, et al. Uric acid induces hepatic steatosis by generation of mitochondrial oxidative stress. J Biol Chem. 2012;287(48):40732-40744.
22. Chatterjee S, Ghosh N, Bhattacharya S. Hyperuricemia and its correlation with metabolic syndrome components: a study from Eastern India. J Assoc Physicians India. 2015;63(4):22-26.
23. Ramachandran A, Snehalatha C, Vijay V. Temporal changes in prevalence of diabetes and impaired glucose tolerance associated with lifestyle transition occurring in the rural population in India. Diabetologia. 2004;47(5):860-865.
24. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. J Clin Endocrinol Metab. 2008;93(11 Suppl 1):S9-30.
25. Krishnan E, Kwoh CK, Schumacher HR, Kuller L. Hyperuricemia and incidence of hypertension among men without metabolic syndrome. Hypertension. 2007;49(2):298-303.
26. Verma A, Kaur J, Malhotra V. Correlation of serum uric acid with glycaemic status and lipid profile in type 2 diabetes mellitus. Natl J Physiol Pharm Pharmacol. 2018;8(9):1256-1260.
27. Sharma S, Sharma B, Puri V, Sachdeva S. Serum uric acid levels in newly diagnosed type 2 diabetes mellitus and its correlation with insulin resistance. Int J Adv Med. 2019;6(3):815-819.
28. Krishnamurthy A, Bhaskaran S, Sundaram S. Serum uric acid as a predictor of cardiovascular risk in prediabetic subjects: a South Indian perspective. Int J Res Med Sci. 2020;8(6):2078-2083.