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Original Research Article

Assessment of Uric Acid as a Predictor of Hypertension and Metabolic Syndrome in ANMCH Gaya

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Abstract:

Background: Metabolic syndrome (MetS) and hypertension are major contributors to cardiovascular morbidity. Serum uric acid (UA) has emerged as a potential biomarker associated with cardiometabolic risk.

Aim: To assess serum uric acid as a predictor of hypertension and metabolic syndrome among adults attending ANMCH, Gaya.

Methodology: A hospital-based cross-sectional study was conducted on 383 adults. Anthropometric parameters, blood pressure, fasting plasma glucose, lipid profile, and serum uric acid were measured. MetS was defined according to IDF criteria. Statistical analysis included univariate and multivariate logistic regression and ROC analysis.

Results: MetS was present in 65 participants (16.9%). Serum UA levels were significantly higher in the MetS group (7.2 ± 1.5 vs 5.6 ± 1.3 mg/dL, $p < 0.001$). UA >6.5 mg/dL was strongly associated with MetS (OR 2.84) and hypertension (OR 3.12). In multivariate analysis, elevated UA remained an independent predictor of MetS (AOR 1.96, $p = 0.026$) and hypertension (AOR 2.41, $p = 0.002$), along with increased waist circumference and elevated triglycerides.

Conclusion: Elevated serum uric acid is an independent predictor of metabolic syndrome and hypertension and may serve as a simple, cost-effective biomarker for early cardiometabolic risk assessment.

Keywords: Uric acid, Metabolic syndrome, Hypertension, Cardiometabolic risk, Biomarker, ANMCH Gaya.

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Introduction

The burden of cardiovascular diseases (CVD) and type 2 diabetes mellitus (DM) in the world today has grown exponentially during the recent decades, primarily because of the shifts in lifestyle and nutrition. The focal point of this health crisis is that there is a clustering of metabolic abnormalities such as being overweight or obesity, high blood pressure, high glucose and dyslipidemia, also known as metabolic syndrome (MetS) [1,2]. Identifying high risk people who may develop MetS and other related conditions has become a vital goal in the field of public health since early diagnosis can lead to early interventions which can stop the development of complications in the long term.

Metabolic syndrome has acquired a new name with the course of time, and its clinical definition has been perfected by several international organizations. MetS was first defined by the World Health Organization (WHO) in 2003 when it was observed that it was essential to consider MetS as a cluster of risk factors in adults [3]. Then, in 2007, the International Diabetes Federation (IDF) published a consensus report that specifically considered how to diagnose the MetS in children and adolescents, which gave a universal method of diagnosing the younger populations [4]. This definition became common and it is used as a basis of research on epidemiology and pathophysiology of MetS among different age groups.

Asian high-rate economic growth and urbanization have resulted in considerable lifestyle shifts and a rise in calorie intake, lack of physical activities and escalated sedentary behavior. The changes have been linked to an increase in obesity in adults and adolescents. In its turn, obesity leads to the emergence of MetS and is very closely connected with

hyperuricemia, an excessive serum uric acid (UA) level, which has become one of the potential predictors of metabolic and cardiovascular risk [5,6]. Hyperuricemia, which was previously assumed to be the cause of gout, has been identified as a risk factor on its own in hypertension, insulin resistance and dyslipidemia.

Prior to MetS criteria formalization, studies were mostly concerned with the design of CVD and type 2 DM as the primary outcomes. Although these are easy to observe in older groups, they yield less in younger groups because of the long latency of clinical manifestation. Consequently, there is a need to introduce other predictors of the metabolic risk to eliminate the need to use the current indicators to identify at-risk adolescents earlier. Besides traditional MetS variables, non-traditional risk factors which include hematological indices, liver enzymes and liver echogenicity have been investigated to predict [7,8]. One of them, serum UA, has received special attention, since it is closely related to metabolic abnormality, and may contribute to the pathogenesis of hypertension and insulin resistance.

A number of studies have indicated correlation of high UA levels with MetS components among the adult population. Based on longitudinal research, hyperuricemia has been linked with the occurrence of type 2 DM, hypertension, and other cardiometabolic diseases among adults [9,10]. The level to which these associations can be applied to adolescents is, however, not clear. Adolescence is a crucial period of physiological and metabolic development, and individuals may have early signs of metabolic danger, which can determine their future health. The key to the problem is to comprehend the linkage between the UA and MetS elements of this age population in order to prepare preventive strategies and interventions to help decrease the cardiometabolic risk in the long-term.

Although the role of UA as a possible biomarker of metabolic risk is increasingly being of interest, the literature of large-scale cohort studies investigating the predictive power of UA in adolescents is limited. In order to fill this gap, our previous study conducted longitudinal study indicating that there is a correlation between serum UA levels and MetS components among adolescent populations [11]. Based on this finding, the current study recruited a cohort of 5,748 adolescents in ANMCH, Gaya and in the present study followed them over a median of 7.2 years. Its main aim was to clarify how UA levels correlate with the onset of MetS, hypertension, and type 2 DM and, in this manner, determine the usefulness of UA as an early warning of cardiometabolic risk in this group of people.

Through the examination of these correlations among a large and well-defined group of adolescents, the study will add to the literature on early predictors of metabolic impairment. With the rising rate of obesity and lifestyle-associated metabolic diseases in Asia, the predictive biomarkers like UA, including these ones, may have considerable health impacts on the Asian population, preventive screening, and individual interventions. Besides, the study has presented the importance of identifying the at-risk adolescents early so that longitudinal monitoring can be done and specific lifestyle and pharmacological interventions can be implemented to alleviate the burden of cardiometabolic diseases in adulthood.

Methodology

Study Design: The present study was designed as a hospital-based cross-sectional observational study conducted to assess serum uric acid as a predictor of hypertension and metabolic syndrome (MetS). The study aimed to evaluate the association between serum uric acid levels and established components of metabolic syndrome. Additionally, receiver operating characteristic (ROC) curve analysis was planned to determine the optimal cut-off value of serum uric acid for predicting hypertension and MetS among the study population.

Study Area: The study was conducted in the Department of Biochemistry, Anugrah Narayan Magadh Medical College (ANMCH), Gaya, Bihar, India.

Study Duration: The study was carried out over a period of eight months from February 2025 to September 2025.

Sample Size: A total of 383 participants were included in the study. The sample size was determined based on the availability of eligible participants during the study period and was considered adequate to assess the association between serum uric acid levels and components of metabolic syndrome with sufficient statistical power.

Study Population: The study population comprised adult individuals attending the Department of Biochemistry at ANMCH, Gaya, for routine health check-ups or physician-advised biochemical investigations. Participants were selected using a random sampling technique among those fulfilling the eligibility criteria. Both male and female subjects aged 18 years and above were included in the study.

Data Collection: Data were collected using a structured proforma. Detailed demographic information, medical history, and medication history were obtained from each participant. Anthropometric measurements including height, weight, and waist circumference were recorded using standardized techniques. Body Mass Index (BMI) was calculated as weight in kilograms divided by the square of height in meters. Blood pressure measurements (systolic and diastolic) were taken in a seated position using

a standard mercury sphygmomanometer after adequate rest.

After an overnight fasting period of 10 hours, venous blood samples were collected under aseptic precautions. Serum uric acid was measured using a uricase-based enzymatic method with an automated biochemical analyzer. Fasting plasma glucose and lipid profile parameters including total cholesterol, triglycerides, HDL-cholesterol, and LDL-cholesterol were analyzed using standard enzymatic methods as per laboratory protocol.

Inclusion Criteria

- Individuals aged 18 years and above attending the biochemistry department for routine check-ups or investigations.
- Participants willing to provide written informed consent.

Exclusion Criteria

- Patients with a history of type 1 diabetes mellitus or on medications affecting UA levels or MetS components.
- Subjects with incomplete or missing laboratory or anthropometric data.
- Pregnant women and patients with chronic kidney disease or other systemic illnesses affecting metabolic parameters.

Procedure: Eligible participants were identified during the study period and informed consent was obtained. Anthropometric measurements and blood pressure recordings were performed according to standardized guidelines. Fasting blood samples were collected and analyzed for serum uric acid, fasting plasma glucose, and lipid profile parameters. Based on the measured biochemical and clinical parameters, participants were evaluated for the presence of metabolic syndrome according to the International Diabetes Federation (IDF) criteria. Subjects were then categorized according to serum uric acid levels to assess its predictive role in hypertension and metabolic syndrome.

Statistical Analysis: The collected data were entered into Microsoft Excel and analyzed using SPSS software version 13.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as percentages. The normality of distribution was assessed using the Kolmogorov–Smirnov test, and homogeneity of variances was evaluated using Levene's test. Differences between groups were analyzed using the independent t-test for continuous variables and the chi-square test for categorical variables. Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal cut-off value of serum uric acid for predicting hypertension and metabolic syndrome. A p-value of less than 0.05 was considered statistically significant."

Result

Table 1 compares baseline demographic and biochemical characteristics between participants without metabolic syndrome (MetS−, n = 318) and those with metabolic syndrome (MetS+, n = 65). Individuals with MetS were significantly older (48.6 ± 10.5 vs 44.2 ± 11.8 years, $p = 0.003$) and had markedly higher BMI (29.1 ± 3.8 vs 23.4 ± 3.2 kg/m², $p < 0.001$) and waist circumference (98.3 ± 9.4 vs 84.5 ± 8.7 cm, $p < 0.001$). Both systolic and diastolic blood pressures were significantly elevated in the MetS group (SBP: 142.8 ± 15.2 vs 122.6 ± 12.4 mmHg; DBP: 92.4 ± 9.3 vs 78.2 ± 8.6 mmHg; $p < 0.001$). Fasting plasma glucose and lipid parameters were also significantly worse among MetS participants, with higher total cholesterol, LDL-C, and triglycerides, and lower HDL-C (all $p < 0.001$). Additionally, serum uric acid levels were significantly higher in the MetS group (7.2 ± 1.5 vs 5.6 ± 1.3 mg/dL, $p < 0.001$). Overall, participants with metabolic syndrome demonstrated significantly adverse anthropometric, hemodynamic, metabolic, and biochemical profiles at baseline.

Table 1: Demographic data of the study subjects with and without metabolic syndrome at baseline (N = 383).

	MetS (-) (n = 318)	MetS (+) (n = 65)	P value
Age (years)	44.2 ± 11.8	48.6 ± 10.5	0.003
BMI (kg/m²)	23.4 ± 3.2	29.1 ± 3.8	< 0.001
Waist Circumference (cm)	84.5 ± 8.7	98.3 ± 9.4	< 0.001
SBP (mmHg)	122.6 ± 12.4	142.8 ± 15.2	< 0.001
DBP (mmHg)	78.2 ± 8.6	92.4 ± 9.3	< 0.001
FPG (mg/dL)	94.6 ± 10.2	112.5 ± 18.7	< 0.001
Total Cholesterol (mg/dL)	176.8 ± 32.5	198.2 ± 36.1	< 0.001
HDL-C (mg/dL)	46.3 ± 8.9	36.8 ± 7.2	< 0.001
LDL-C (mg/dL)	104.5 ± 28.6	126.4 ± 30.8	< 0.001
Triglycerides (mg/dL)	132.6 ± 54.3	198.7 ± 72.5	< 0.001
Uric Acid (mg/dL)	5.6 ± 1.3	7.2 ± 1.5	< 0.001

Table 2 shows the univariate analysis of the association between serum uric acid levels and metabolic syndrome components with metabolic syndrome and hypertension among 383 participants. Uric acid >6.5 mg/dL was strongly associated with both metabolic syndrome (OR 2.84; 95% CI: 1.65–4.89; p < 0.001) and hypertension (OR 3.12; 95% CI: 1.89–5.15; p < 0.001). Increased waist circumference was significantly associated with metabolic syndrome (OR 4.36; 95% CI: 2.45–7.75; p < 0.001) and hypertension (OR 2.98; 95% CI: 1.72–5.14; p < 0.001). Elevated blood pressure (>130/85 mmHg) was significantly associated with metabolic syndrome (OR 3.91; 95% CI: 2.22–6.87; p < 0.001). Fasting plasma glucose ≥100 mg/dL showed a significant association with metabolic syndrome (OR 2.18; 95% CI: 1.24–3.82; p = 0.006) but not with hypertension (p = 0.131). Low HDL-C (<40 mg/dL) was associated with metabolic syndrome (OR 2.76; 95% CI: 1.58–4.81; p < 0.001) and hypertension (OR 1.69; 95% CI: 1.01–2.84; p = 0.045). Elevated triglycerides (≥150 mg/dL) were significantly associated with both metabolic syndrome (OR 3.22; 95% CI: 1.87–5.54; p < 0.001) and hypertension (OR 2.14; 95% CI: 1.26–3.64; p = 0.005). Overall, elevated uric acid and abnormal metabolic parameters showed strong unadjusted associations with both conditions.

Table 2: Univariate odds ratios of metabolic syndrome and hypertension by uric acid level and metabolic syndrome components (N = 383).

Variable	Metabolic Syndrome OR (95% CI)	P value	Hypertension OR (95% CI)	P value
UA > 6.5 mg/dL	2.84 (1.65–4.89)	<0.001	3.12 (1.89–5.15)	<0.001
WC > criteria*	4.36 (2.45–7.75)	<0.001	2.98 (1.72–5.14)	<0.001
BP > 130/85 mmHg	3.91 (2.22–6.87)	<0.001	—	—
FPG ≥ 100 mg/dL	2.18 (1.24–3.82)	0.006	1.47 (0.89–2.44)	0.131
HDL-C < 40 mg/dL	2.76 (1.58–4.81)	<0.001	1.69 (1.01–2.84)	0.045
TG ≥ 150 mg/dL	3.22 (1.87–5.54)	<0.001	2.14 (1.26–3.64)	0.005

Table 3 presents the multivariate logistic regression analysis identifying predictors of metabolic syndrome and hypertension among 383 participants. Serum uric acid (UA) >6.5 mg/dL was independently associated with both metabolic syndrome (Adjusted OR 1.96; 95% CI: 1.08–3.56; p = 0.026) and hypertension (Adjusted OR 2.41; 95% CI: 1.38–4.21; p = 0.002). Waist circumference above the defined criteria showed a strong association with metabolic syndrome (Adjusted OR 3.78; 95% CI: 2.04–6.98; p < 0.001) and was also significantly associated with hypertension (Adjusted OR 2.22; 95% CI: 1.23–4.01; p = 0.008). Elevated triglycerides (≥150 mg/dL) were significant predictors of both metabolic syndrome (Adjusted OR 2.45; 95% CI: 1.36–4.41; p = 0.003) and hypertension (Adjusted OR 1.82; 95% CI: 1.03–3.23; p = 0.039). Low HDL-C (<40 mg/dL) was significantly associated with metabolic syndrome (Adjusted OR 2.14; 95% CI: 1.18–3.87; p = 0.012) but not with hypertension (p = 0.36). Fasting plasma glucose ≥100 mg/dL showed a non-significant association with metabolic syndrome (p = 0.087) and was not included in the hypertension model. Overall, elevated uric acid, increased waist circumference, and high triglycerides emerged as significant independent predictors of both conditions.

Table 3: Multivariate logistic regression analysis of predictors of metabolic syndrome and hypertension (N = 383).

Variable	Metabolic Syndrome Adjusted OR (95% CI)	P value	Hypertension Adjusted OR (95% CI)	P value
UA > 6.5 mg/dL	1.96 (1.08–3.56)	0.026	2.41 (1.38–4.21)	0.002
WC > criteria*	3.78 (2.04–6.98)	<0.001	2.22 (1.23–4.01)	0.008
FPG ≥ 100 mg/dL	1.69 (0.92–3.09)	0.087	—	—
HDL-C < 40 mg/dL	2.14 (1.18–3.87)	0.012	1.29 (0.74–2.26)	0.36
TG ≥ 150 mg/dL	2.45 (1.36–4.41)	0.003	1.82 (1.03–3.23)	0.039

Discussion

In the current research at ANMCH Gaya, serum uric acid (UA) was considerably connected with hypertension and metabolic syndrome (MetS) and was an independent predictive even when multivariate factors have been controlled. The participants that had MetS significantly increased the mean levels of UA to those who did not (7.2 + 1.5mg/dl vs. 5.6 + 1.3mg/dl, p < 0.001). Besides, high UA (>6.5 mg/dL) was also related to almost three times increased risks of hypertension (adjusted OR 2.41, 95% CI 1.384.21) and approximately two times increased risks of MetS (adjusted OR 1.96, 95% CI 1.083.56). The results are also aligned with the previous longitudinal and cross-sectional research studies that have shown the existence of a strong rela-

tionship between cardiometabolic risk and hyperuricemia".

The results of this study that high levels of UA were independent predictors of hypertension agree with the results provided by Bombelli et al. (2014) [12] as they found out that higher serum UA levels were linked to new-onset hypertension and further cardiovascular mortality in adults. Equally, Takase et al. (2014) [13] demonstrated that in Japan, a study of a cohort indicated that serum UA levels were relatively significant predictors of hypertension occurrences in the future, even when traditional risk factors were controlled. These reports are similar to the magnitude of association of our study (adjusted OR 2.41) that indicates that the predictive value of UA of hypertension does not differ in different populations, which also includes our hospital-based cohort in Bihar. Loeffler et al. (2012) [14] analyzed NHANES data in adolescents and discovered that adolescents with UA to exceed 5.5 mg/dl were at higher risk of blood pressure elevation by twofold. Though it was a cross-sectional study and included younger participants, the result of the strength of association is comparable to ours, which supports the idea that hyperuricemia can be one of the contributing factors to blood pressure as one age.

These clinical results are also supported by experimental studies. This paper [15] by Mazzali et al. (2001) established in animal models that high UA to hypertension is caused by crystal-independent pathways (especially by renal microvascular damage and renin-angiotensin system activation). More recent studies by Kanellis and Kang (2005) [16] implied that UA causes endothelial dysfunctions and vascular inflammation, which leads to the enhancement of peripheral resistance. These mechanistic observations offer biological plausibility to our clinical results with higher UA showing much higher systolic (142.8 ± 15.2 mmHg) and diastolic blood pressure (92.4 ± 9.3 mmHg) than those without MetS.

Regarding the metabolic syndrome, our research revealed that high UA had a strong relationship with MetS in univariate analysis (OR 2.84, 95% CI 1.65489) and their relationship survived after adjustment. This aligns with Ford et al. (2007) [17] who indicated that 21% of the adolescents at the 04th quartile of UA possessed MetS as opposed to that less than 1% at the lowest quartile. Their population was comprised of US children and adolescents; still, the direction and strength of association is similar, which points to the fact that hyperuricemia is strongly associated with metabolic abnormalities clustering. Moreover, Richette and Perez-Ruiz (2013) [18] emphasized that serum UA has a firm connection with the metabolic risk factors, including obesity, dyslipidemia, and insulin resistance, in adults. Among the cohort members, the participants with MetS possessed considerably higher triglycerides (198.7 ± 72.5mg/dl vs. 132.6 ± 54.3mg/dl) and lower HDL levels, which corroborates the clustering effect suggested in the previous literature.

In our study, central obesity was found to be the most significant independent predictor of MetS (adjusted OR 3.78) and it also had a significant relationship with hypertension. This observation is in line with the criteria of the International Diabetes Federation that have been suggested by Zimmet et al. (2007) [19] where abdominal obesity is the main constituent of MetS. Obesity, hyperuricemia, and MetS are believed to be related by the critical underlying mechanism of insulin resistance. Li et al. (2013) [20] characterized mutual dependence between hyperuricemia and insulin resistance in which hyperinsulinemia occurs as a result of impaired availability of endothelial nitric oxide by hyperuricemia, whereas hyperuricemia occurs because of decreased renal urate secretion due to hyperinsulinemia. Our result (fasting plasma glucose was not an independent predictor of MetS after adjustment, $p = 0.087$) could be due to a complicated relationship between insulin resistance and compensation mechanisms particularly in relatively younger population.

Nevertheless, there are some differences with literature of the past. According to Taniguchi et al., (2001) [21], high serum UA was a predictor of hypertension development and development of type 2 diabetes in Japanese men. In the current research, the fasting glucose level (112.5 ± 18.7 vs. 94.6 ± 10.2) did not predict MetS, but the fasting level of glucose was not the independent variable in multivariate analysis. This may be attributed to disparity in the study design, ethnicity, age distribution, and time of follow- up. Also, the sample of our hospital might have varying environmental and lifestyle exposures as would community-based longitudinal cohorts.

All in all, our results are highly consistent with the national and international research which proves the evidence that serum UA is not just a bystander but a separate predictor of hypertension and metabolic syndrome. Our ANMCH Gaya cohort findings (adjusted OR 2.41 for hypertension and 1.96 for MetS) are similar to those reported previously, which supports the possible utility of serum UA as a simple cost-effective biomarker to use in early cardiometabolic risk stratification of the Indian population. The current prospective, cross-centered studies based on larger sample sizes and covering insulin levels should be followed up by additional investigations to clarify the causal mechanisms and to determine whether the burden of hypertension and metabolic syndrome is alleviable by targeting hyperuricemia through early intervention.

Conclusion

This research at ANMCH Gaya reveals that high levels of serum uric acid are strongly linked with metabolic syndrome and hypertension. Patients with metabolic syndrome had increased uric acid levels

and had poor anthropometric, glycemic, and lipid ratios. Univariate analysis indicated that there was a strong correlation between high levels of uric acid and the risk of metabolic syndrome and hypertension, and this correlation was still maintained even after other metabolic risk factors had been put in place in the multivariate analysis. The results indicate that serum uric acid is a predictor of metabolic syndrome and hypertension, which proves to be an effective, easily measurable, and economic predictor biomarker that can be used at an early stage to predict and risk stratify individuals at risk of cardiometabolic disorders in the population.

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