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ORIGINAL ARTICLE

Correlation Between Serum Uric Acid Levels and Metabolic Syndrome in Adults

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Abstract

Background: Metabolic syndrome is a group of metabolic abnormalities, and these abnormalities are associated with an increased risk of cardiovascular disease and DM. Due to surge in prevalence of obesity in young adults, the prevalence of metabolic syndrome will likely rise significantly in near future. The serum uric acid levels are significantly raised in metabolic syndrome and are associated with adverse cardiovascular mortality. **Aim:** This study aims to investigate correlation between serum uric acid levels and metabolic syndrome. **Discussion:** Existing studies showed positive correlation of hyperuricemia with metabolic syndrome in elderly patients. But there is limited research on uric acid levels in young population with obesity, hypertension and metabolic syndrome. Consequently, a cross-sectional observational study was conducted on metabolic syndrome patients and their correlation with serum uric acid levels. **Conclusion:** This study showed significant correlation with metabolic syndrome and serum uric acid levels in young adult patients. This higher serum uric acid levels in young adults of metabolic syndrome are rising a concern for cardiovascular adverse outcomes in these young population.

Keywords: Metabolic syndrome, Serum uric acid, Dyslipidaemia

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Abbreviations		
FBS	:	Fasting Blood Sugar
CVD	:	Cardiovascular Disease
LDL	:	Low Density Lipoprotein
SBP	:	Systolic Blood Pressure
DM	:	Diabetes Mellitus
TGs	:	Triglycerides
BMI	:	Body Mass Index
DBP	:	Diastolic Blood Pressure
HDL	:	High Density Lipoprotein



Introduction

Metabolic syndrome is a group of metabolic abnormalities, and these abnormalities are central obesity, raised blood pressure, insulin resistance, dyslipidaemia, proinflammatory and prothrombotic states [1].

Worldwide, the prevalence of metabolic syndrome (Metabolic syndrome) ranges from 10% to 84%. In India, adult

population overall had a 30% prevalence rate of metabolic syndrome [2].

Uric acid, a compound formed due to purine metabolism has been found to be associated and significantly correlated to CVDs. Additionally, impaired glucose tolerance further increases the risk of mortality and morbidity with adverse CVD outcomes [3]. Since the uric acid range is lower in the females it tends to get more positively correlated but at the same time males has been found to have higher association with risk factors like alcoholism. Also, hormonal factors like oestrogen have been found to be protective in females against metabolic syndrome but after menopause risk remains equal [4].

Pathophysiology

The unhealthy food habits and sedentary lifestyle along with genetic and epigenetic causes play a significant role in pathogenesis of metabolic syndrome. Since most of the metabolic syndrome pathways has been found to be triggered by central adiposity, high caloric intake can be attributed a causal role [5]. The chronic inflammatory state, neurohormonal activation and insulin resistance are crucial elements in the development of metabolic syndrome and its eventual transformation into Type 2 DM and CVDs. Initially normoglycaemia is maintained bv compensatory hyperplasia of beta cell mass and increased insulin secretory capacity. Eventually, beta cell dysfunction after a few years led to development of type 2 DM [6].

Obesity can lead to development of hyperuricemia by increasing synthesis of uric acid and decreasing its excretion through kidneys. On the other hand, hyperuricemia can cause obesity by increasing peripheral and hepatic lipogenesis. This obesity and hyperuricemia are substantial risk factors for development of DM [7].

It has been established that hyperuricemia results in loss of endothelial cell function. The lack of endothelial formed nitric oxide lowers blood flow to cells, which inhibits the action of insulin leading to hyperinsulinemia. This insulin resistance plays a vital role in Metabolic syndrome. Serum uric acid and triglycerides have a positive correlation with risk factors related to complications of DM [8].

Hyperuricemia and adverse CVD

Hyperuricemia is significantly with hypertension, associated dyslipidaemia, CVD and heart failure. In congestive heart failure, uric acid serves as an independent predictor of adverse outcomes in elderly hospitalized patients [9]. Epidemiological studies have showed significant increased cardiovascular risks among patients of gout, but there is still controversy that uric acid lowering therapy may benefit from adverse cardiovascular outcomes. Additional research is needed for uric acid lowering therapy in metabolic syndrome and whether to consider uric acid levels as a predictor or marker of metabolic syndrome [10].

Methodology

Study population: Patients older than 18 years of age coming to a tertiary hospital.

Sample size: 181 patients were recruited out of which 91 were males and 90 were females.

Inclusion criteria

- Age > 18 years
- Age < 60 years
- Informed consent
- Metabolic syndrome criteria fulfilled

Exclusion criteria

- Chronic renal failure
- Undergoing renal replacement therapy
- Acute or chronic gout
- Recent history of diuretics intake
- Malignancy

- Stroke
- History of CVD
- Drug history like antiepileptics steroid or recreational drugs
- Pregnancy
- Lactation
- Consent not given

Material and methods

3 ml of blood sample was taken for fasting blood sugar, lipid profile and uric acid. It was processed in fully automated biochemistry analyser XL 1000.

Metabolic syndrome was defined on basis of National cholesterol education programme guidelines as shown in Figure 1 [11].

Glucose Oxidase-Peroxidase method was used to measure fasting blood sugar. In this method, Glucose oxidase breaks down glucose and in turn forms hydrogen peroxide which reacts with peroxidase causing the colour dye to change its colour [12].

For triglycerides, colorimetric enzymatic method with glycerophosphate oxidase was used. This technique follows enzymatic hydrolysis of TGs, and the byproduct gets oxidised by glycerol phosphate leading to generation of a colorimetric reaction. Since it's a quantitative method, colour intensity is directly proportional to the concentration of triglycerides and spectrophotometer is used to measure it [13].

HDL was measured by direct modified polyvinyl sulfonic acid and

polyethylene glycol methyl ether method. It is also an enzymatic method which specifically quantifies HDL and is hence an effective method to measure it without having a hindrance from other lipoproteins [14].

Uric acid is quantified with the help of uricase method in which the uricase enzyme forms allantoin by oxidation of uric acid and a colorimetric reaction is generated which can be measured with a spectrophotometer [15].

Results

This study was conducted among 181 patients who presented to GMSH, sector 16, Chandigarh. Among 181 patients 91 were males and 90 were females. Gender wise distribution of uric acid levels is shown in Table 1 and graphically represented in Figures 2 to 4. The normal uric acid is taken as ≤ 6.5 mg/dL for females and ≤ 7.5 mg/dL for males.

Among the 181 total participants, 76.8% (n=139) had uric acid levels within the normal range, while 23.2% (n=42) had elevated levels. A higher proportion of males (86.8%) had uric acid within the normal range compared to females (66.7%). Conversely, elevated uric acid levels were more common in females (33.3%) than in males (13.2%). The Chi-Square value of 10.306 and a p-value of 0.001 indicate a statistically significant difference between males and females in terms of uric acid levels, suggesting a potential gender-based variation in hyperuricemia prevalence.

Risk factors	Defining levels
Abdominal obesity	Waist circumference
Men	>102 CM (40 in)
Women	>88 CM (35 in)
Triglycerides	≥ 150 mg/dl
HDL cholesterol	
Men	< 40 mg/dl
Women	<50 mg/dl
Blood pressure	≥ 130/≥ 85 mmHg
Fasting glucose	≥ 110 mg/dL

Figure 1. Metabolic syndrome criteria

Uric Acid	Gender					Chi	р-	
	Μ	ale	I	Female	, r	Fotal	Square	value
≤6.5 F or ≤7.5 M	79	86.8%	60	66.7%	139	76.8%	10.306	.001
>6.5 F or >7.5 M	12	13.2%	30	33.3%	42	23.2%		
Total	91	100.0%	90	100.0%	181	100.0%		



Figure 2. Distribution of uric acid in males



Figure 3. Distribution of uric acid in females



Figure 4. Age wise distribution of patients with their uric acid levels

Parameters	Uric Acid					
	Pearson coefficient	p-value	No. of patients			
Waist circumference	.301	< 0.001	181			
SBP	.274	< 0.001	181			
DBP	.391	< 0.001	181			
FBS	.256	< 0.001	181			
Total Cholesterol	.405	< 0.001	181			
TGs	.273	< 0.001	181			
HDL	048	<0.518	181			

Table 2. Components of metabolic syndrome correlation with uric acid

Correlation of metabolic syndrome and its components with uric acid levels in 181 patients is shown in Table 2 and graphically represented in Figures 5 to 10.

Pearson coefficient indicate a positive correlation between uric acid levels and waist circumference [r=0.301, p<0.001], SBP [r=0.274, p<0.001], DBP [r=0.391, p<0.001], FBS [r=0.256, p<0.001], total cholesterol [r=0.405, p<0.001], and TGs [r=0.273, p<0.001] and all were statistically significant. These results suggest that higher uric acid is associated with worsening metabolic parameters. In contrast, HDL showed a negative correlation [r = -0.048, p = 0.518], although it was statistically insignificant.



Figure 5. Scatter plot of SBP vs Uric acid



Figure 6. Scatter plot of DBP vs Uric acid



Figure 7. Scatter plot of Waist circumference vs Uric acid



Figure 8. Scatter plot of FBS vs Uric acid



Figure 9. Scatter plot of TGs vs Uric acid



Figure 10. Scatter plot of HDL vs Uric acid

Conclusion

This study shows that elevated uric acid was correlated with metabolic syndrome, linking it to insulin resistance, obesity, hypertension, and dyslipidaemia. Regular monitoring of uric acid may help in early risk assessment, and further research is needed to explore its potential as a therapeutic target.

Future scope

Uric acid can be explored as a predictive biomarker for metabolic syndrome in future. Investigating its role in insulin resistance, hypertension, and dyslipidaemia may lead to targeted therapies. Further studies on genetic factors, lifestyle interventions, and AIbased predictive models could enhance risk assessment and treatment strategies.

Statements and Declarations

Conflicts of interest

The authors declare that they do not have conflict of interest.

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