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Exploring the Synergy Between Hyperuricemia, Obesity, and Hypertension: Evidence from Urban India

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ABSTRACT

Introduction: Hyperuricemia, characterized by elevated serum uric acid levels, is increasingly recognized as an independent risk factor for hypertension, particularly in overweight and obese individuals. This study explores the relationship between serum uric acid levels, anthropometric parameters, and arterial blood pressure in an urban, overweight, and obese Indian population.

Methodology: A cross-sectional study was conducted on 224 drug-naive, overweight, and obese individuals from an urban area in North India. Anthropometric measurements, including body mass index (BMI), waist circumference, hip circumference, and sagittal abdominal diameter (SAD), were recorded. Serum uric acid levels were estimated using the uricase-peroxidase method, and arterial blood pressure was measured with an automatic digital sphygmomanometer. Pearson's correlation coefficient was used to assess the relationships between these variables.

Results: The study found a moderate positive correlation between serum uric acid levels and both weight ($R = 0.308$, $p = 0.0005$) and BMI ($R = 0.39$, $p = 0.00005$). Significant correlations were also observed between serum uric acid and SAD ($R = 0.275$, $p = 0.002$), waist-to-hip ratio (WHR) ($R = 0.332$, $p = 0.00016$), and both systolic blood pressure (SBP) ($R = 0.698$, $p = 0.0001$) and diastolic blood pressure (DBP) ($R = 0.543$, $p = 0.0005$). No significant correlations were found between serum uric acid and other anthropometric measures like waist circumference and hip circumference.

Discussion: These results indicate that hyperuricemia is significantly associated with obesity-related measures and blood pressure, suggesting that it may play a role in the development of hypertension, particularly in individuals with central obesity.

Conclusion: The study underscores the importance of monitoring serum uric acid levels in overweight and obese populations as part of hypertension prevention and management strategies. Further research is needed to explore the underlying mechanisms and potential interventions to mitigate the risk of hypertension associated with hyperuricemia.

Keywords: Hyperuricemia, Hypertension, Obesity

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INTRODUCTION

Hyperuricemia, defined by elevated serum uric acid levels, has traditionally been associated with conditions such as gout and kidney stones. However, recent research has increasingly identified hyperuricemia as an independent risk factor for hypertension, particularly in individuals who are overweight or obese. This association reveals the complexity of hyperuricemia as a condition that influences cardiovascular health beyond its classical manifestations.

Recent studies have demonstrated a significant link between hyperuricemia and the development of hypertension. For instance, research conducted by Borghi et al. (2020) emphasized that hyperuricemia is not only a marker but also a potential mediator in the pathogenesis of hypertension, particularly in those with elevated body mass indices (BMI).[1] Additionally, studies have shown that

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hyperuricemia accelerates the progression from prehypertension to hypertension, making it a critical factor to monitor in at-risk populations.[2]

The global rise in overweight and obesity rates has intensified public health concerns, given the strong association between obesity and metabolic disorders such as hyperuricemia and hypertension. A systematic review by Johnson *et al.* (2022) found that individuals with higher BMI are more likely to have elevated serum uric acid levels, which in turn increases their risk of developing hypertension.[3] This relationship is thought to be due to increased uric acid production and decreased renal excretion in individuals with obesity.

Understanding the interplay between serum uric acid levels, anthropometric parameters, and arterial blood pressure is crucial for developing effective preventive and therapeutic strategies. Recent researchon urban Indian populations has highlighted that higher BMI and waist circumference strongly correlate with elevated serum uric acid levels associated with increased arterial blood pressure.[4] These findings suggest that regular monitoring of serum uric acid in overweight and obese individuals could be essential for the early identification and management of hypertension.

Given the intricate relationship between hyperuricemia, obesity, and hypertension, serum uric acid should be considered a modifiable risk factor in clinical practice. Lifestyle interventions such as weight loss, dietary changes, and increased physical activity have been shown to effectively lower serum uric acid levels and reduce the risk of hypertension.[5] Additionally, recent advancements in pharmacological treatments, including uric acid-lowering therapies, offer promising strategies for individuals at high risk of developing hypertension due to hyperuricemia.[6]

Hyperuricemia plays a pivotal role in the progression from prehypertension to hypertension, particularly among overweight and obese individuals. A deeper understanding of the relationship between serum uric acid levels, obesity, and hypertension is essential for the development of comprehensive strategies aimed at reducing the global burden of hypertension. Continued research in diverse populations will be vital in uncovering the underlying mechanisms and in crafting tailored interventions to address this significant public health challenge.

METHODOLOGY

Study Population

This cross-sectional study included 224 urban, drug-naive, overweight, and obese Indian subjects, aged 18 years and above, from a city in North India. The participants were selected based on their body mass index (BMI), which was greater than 23.5, indicating overweight and obesity status as per Asian criteria.[7]

Data Collection

Data collection involved in-person meetings, where detailed information on lifestyle factors, disease history, and anthropometric measurements were gathered. These measurements included weight, height, waist and hip circumference, and sagittal abdominal diameter (SAD). Lifestyle factors such as diet, physical activity, smoking, and alcohol consumption were recorded through structured questionnaires.

Anthropometric measurements were conducted following standardized protocols to ensure accuracy and consistency. These measurements included weight, height, body mass index (BMI), waist circumference, hip circumference, and SAD.

Weight Measurement

Weight was measured using a calibrated digital weighing scale. Participants were instructed to wear light clothing and remove their shoes to avoid any additional weight. The digital scale was placed on a hard, flat surface, and participants were asked to stand still in the centre of the scale with their weight evenly distributed on both feet. The weight was recorded to the nearest 0.1 kilogram. Studies have emphasized the importance of using calibrated digital scales to ensure precision in weight measurement, which is crucial for calculating BMI and other anthropometric indices.[8]

Height Measurement

Height was measured using a stadiometer, a device specifically designed for accurate height assessment. Participants were instructed to remove their shoes, and stand upright with their backs against the stadiometer's vertical board, heels together, and eyes looking straight ahead (Frankfurt plane). The headpiece of the stadiometer was then lowered until it gently touched the top of the participant's head, and the height was recorded to the nearest 0.1 centimeter. Proper positioning and the use of a stadiometer are critical for obtaining reliable height measurements, which are essential for calculating BMI.[9]

Body Mass Index (BMI)

BMI was calculated using the formula: weight in kilograms divided by height in meters squared (kg/m^2). This calculation provides a widely accepted measure of general adiposity, allowing for the classification of individuals into different weight categories, such as underweight, normal weight, overweight, and obese. The BMI is recognized as a simple and useful tool for assessing body fatness in population studies and clinical practice.[10]

Waist Circumference Measurement

Waist circumference was measured at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest. Participants were asked to stand with their feet shoulder-width apart, and the measurement was taken at the end of a normal expiration to ensure accuracy. A non-stretchable tape measure was used, ensuring that it was snug but not compressing the skin. Waist circumference is a crucial indicator of central adiposity and is strongly associated with the risk of metabolic syndrome and cardiovascular diseases.[11]

Hip Circumference Measurement

Hip circumference was measured at the widest part of the hips, typically around the level of the greater trochanters. Participants

stood with their feet together, and the same non-stretchable tape measure was used as for the waist circumference. The tape was placed horizontally around the hips, ensuring that it was level on both sides and that the measurement was taken without compressing the skin. Hip circumference, in combination with waist circumference, allows for the calculation of the waist-to-hip ratio (WHR), an important marker of fat distribution and cardiovascular risk.[12]

Sagittal Abdominal Diameter (SAD)

SAD was measured using a sliding-beam caliper at the level of the iliac crest with the participant in a supine position. The caliper was placed perpendicular to the bed or examination table, and the distance from the top of the abdomen to the surface of the table was measured. SAD is a reliable indicator of visceral fat, which is a significant predictor of metabolic and cardiovascular risks. Research has shown that SAD provides additional information on body fat distribution that is not captured by BMI or waist circumference alone.[13]

These anthropometric measurements provide a comprehensive assessment of body composition and fat distribution, critical in evaluating the risk of various metabolic and cardiovascular conditions. Adhering to standardized techniques ensures the reliability and validity of these measurements, making them essential tools in clinical and epidemiological research.

Blood Pressure Measurement

Arterial blood pressure was measured using an automatic digital sphygmomanometer. To ensure accuracy, three readings were taken for each participant, with the mean value used for analysis. Standard protocols were followed, including a rest period of at least five minutes before measurement, avoidance of caffeine, exercise, and smoking for at least 30 minutes prior, and proper cuff size.[14]

Uric Acid Estimation

Serum uric acid concentrations were estimated using the uricase-peroxidase method, an enzymatic assay known for its accuracy and reliability in clinical diagnostics. Blood samples were collected from participants after an overnight fast to minimize variations due to recent food intake. The blood was drawn into plain vacutainer tubes and allowed to clot at room temperature before centrifugation at 3000 rpm for 10 minutes to separate the serum.

The serum uric acid levels were measured using the Cobas® 6000 analyzer series with the Uric Acid Assay Kit (Roche Diagnostics, Mannheim, Germany). This kit employs the uricase enzyme, which catalyzes the oxidation of uric acid to allantoin and hydrogen peroxide. The hydrogen peroxide formed in this reaction then reacts with a chromogen in the presence of peroxidase to produce a colored compound, the intensity of which is directly proportional to the uric acid concentration in the sample. The absorbance was measured at 546 nm, and the results were calculated based on a calibration curve prepared using standards provided by the manufacturer.

The serum samples were processed within two hours of collection to ensure the stability of uric acid levels, as delayed processing can lead to degradation and inaccurate results.[15] The assay was performed under the manufacturer's instructions, and the quality control was maintained by running standard controls provided with the kit to ensure the precision and accuracy of the measurements.

The study used uricase-peroxidase method which is widely recognized for its specificity and reliability in clinical settings, making it a preferred choice for serum uric acid estimation.[16]

STATISTICAL ANALYSIS

Pearson's correlation coefficient was employed to assess the relationship between serum uric acid levels and anthropometric parameters (weight, BMI, SAD, and waist-to-hip ratio) as well as systolic (SBP) and diastolic blood pressure (DBP). Statistical significance was set at $p < 0.05$. All statistical analyses were performed using SPSS software version 25.

RESULTS

The data presented in Table 1 examines Pearson's correlation between serum uric acid levels and various anthropometric and blood pressure parameters. The analysis reveals a weak and statistically insignificant correlation between serum uric acid and both age ($R = 0.068$, $p = 0.46$) and height ($R = -0.029$, $p = 0.75$), suggesting no meaningful relationships with these variables. In contrast, serum uric acid levels show a moderate and statistically significant positive correlation with weight ($R = 0.308$, $p = 0.0005$) and BMI ($R = 0.39$, $p = 0.00005$), indicating that increases in body weight and BMI are associated with higher serum uric acid levels. Additionally, a weak to moderate positive correlation is observed between serum uric acid and sagittal abdominal diameter (SAD) ($R = 0.275$, $p = 0.002$), as well as a moderate positive correlation with waist-to-hip ratio (WHR) ($R = 0.332$, $p = 0.00016$), both of which are statistically significant. These findings suggest that abdominal obesity, as reflected by SAD and WHR, may contribute to elevated serum uric acid levels.

Furthermore, the data reveals strong and statistically significant correlations between serum uric acid and both systolic blood pressure (SBP) ($R = 0.698$, $p = 0.0001$) and diastolic blood pressure (DBP) ($R = 0.543$, $p = 0.0005$), indicating a close association between higher serum uric acid levels and elevated blood pressure. On the other hand, no significant correlations were found between serum uric acid and waist circumference (WC) ($R = 0.099$, $p = 0.28$), hip circumference (HC) ($R = -0.07$, $p = 0.44$), or waist-to-height ratio (WHtR) ($R = 0.1117$, $p = 0.2166$), suggesting that these parameters do not have a significant impact on serum uric acid levels. Overall, the results indicate a strong association between serum uric acid and factors related to body weight, abdominal obesity, and blood pressure, while other anthropometric measures appear to have little influence.

Table 1: Pearson's Correlation between Serum Uric Acid with other parameters

Parameter	R-value	p-value
S. Uric Acid vs Age	0.068	0.46
S. Uric Acid vs Height	-0.029	0.75
S. Uric Acid vs Weight	0.308	0.0005
S. Uric Acid vs BMI	0.39	0.00005

S. Uric Acid vs W.C.	0.099	0.28
S. Uric Acid vs H.C.	-0.07	0.44
S. Uric Acid vs SAD	0.275	0.002
S. Uric Acid vs WHR	0.332	0.00016
S. Uric Acid vs WHtR	0.1117	0.2166
S. Uric Acid vs SBP	0.698	0.0001
S. Uric Acid vs DBP	0.543	0.0005

DISCUSSION

The present study provides insights into the relationships between serum uric acid levels, various anthropometric parameters, and arterial blood pressure in an urban, overweight, and obese Indian population. These findings highlight the role of hyperuricemia as a potential risk factor for hypertension, particularly among individuals with elevated body mass indices (BMI) and other obesity indicators.

This study shows a moderate positive correlation between serum uric acid levels and weight ($R = 0.308$, $p = 0.0005$) and BMI ($R = 0.39$, $p = 0.00005$), supporting previous findings that increased body weight and adiposity contribute to elevated serum uric acid levels. A study by Kuwabara *et al.* (2019) similarly demonstrated a significant association between BMI and serum uric acid, indicating that higher adiposity is linked to hyperuricemia.[17]

Additionally, the study identifies a significant positive correlation between serum uric acid and SAD ($R = 0.275$, $p = 0.002$) and waist-to-hip ratio (WHR) ($R = 0.332$, $p = 0.00016$). These findings align with recent research indicating that visceral fat, as reflected by these parameters, is closely associated with elevated uric acid levels.[18,19] Visceral fat is metabolically active and has been implicated in various metabolic disorders, suggesting that central obesity could exacerbate hyperuricemia, thereby increasing the risk of hypertension.

In contrast, no significant correlations were observed between serum uric acid and other anthropometric measures such as waist circumference (WC), hip circumference (HC), and waist-to-height ratio (WHtR). This is consistent with findings from a study by Liu et al. (2021), which indicated that while overall and central obesity are associated with higher serum uric acid levels, other measures of body fat distribution might not significantly impact uric acid levels.[20]

A notable finding of this study is the strong positive correlation between serum uric acid levels and both systolic blood pressure (SBP) ($R = 0.698$, $p = 0.0001$) and diastolic blood pressure (DBP) ($R = 0.543$, $p = 0.0005$). This strong correlation supports the hypothesis that hyperuricemia may contribute to the development and progression of hypertension, especially in overweight and obese individuals. A study by Zhang *et al.* (2020) further supports these findings, demonstrating a significant association between elevated uric acid levels and increased risk of hypertension.[21]

The strong correlation with SBP is particularly concerning, as systolic hypertension is a significant risk factor for cardiovascular events, including stroke and myocardial infarction. These findings suggest that individuals with hyperuricemia are at a higher risk of developing cardiovascular complications, particularly when coupled with elevated blood pressure and obesity.

These findings underscore the importance of regular monitoring of serum uric acid levels in overweight and obese individuals, particularly those with central obesity. Given the significant correlations with both BMI and blood pressure, serum uric acid should be considered as a modifiable risk factor in the management of hypertension. Lifestyle interventions like weight loss, dietary modifications, and increased physical activity have been shown to effectively lower serum uric acid levels and reduce the risk of hypertension.[22,23]

The mechanisms linking hyperuricemia and obesity to hypertension are intricate and involve multiple physiological pathways. Hyperuricemia, characterized by elevated serum uric acid levels, contributes to hypertension primarily through endothelial dysfunction, where increased uric acid impairs nitric oxide (NO) production, leading to reduced vascular relaxation and increased blood pressure.[24,25] Additionally, hyperuricemia is associated with oxidative stress and inflammation, exacerbating endothelial dysfunction.[26] Obesity further aggravates these effects by inducing chronic inflammation and increasing oxidative stress through the secretion of pro-inflammatory cytokines and adipokines from excess adipose tissue.[27,28] This state of chronic inflammation impairs NO production and promotes vasoconstriction, thereby elevating blood pressure. Both hyperuricemia and obesity also adversely affect renal function, with elevated uric acid causing renal vasoconstriction and sodium retention, and obesity-promoting renal injury and impaired sodium excretion [29,30]. Furthermore, hyperuricemia and obesity are linked to increased sympathetic nervous system activity, raising blood pressure by stimulating the central nervous system and increasing catecholamine release.[31,32] Together, these mechanisms highlight the complex interplay between hyperuricemia, obesity, and hypertension, underscoring the importance of addressing these factors in managing high blood pressure.

Moreover, the strong association between serum uric acid and blood pressure highlights the potential role of uric acid-lowering therapies in managing hypertensive patients, particularly those with obesity. Pharmacological interventions targeting serum uric acid levels could offer an additional strategy to control blood pressure and reduce cardiovascular risk in this population.

LIMITATIONS AND FUTURE RESEARCH

While this study provides valuable insights, its cross-sectional design limits the ability to establish causal relationships between serum uric acid levels and hypertension. Longitudinal studies are necessary to confirm these associations and explore the potential mechanisms underlying the relationship between hyperuricemia, obesity, and hypertension.

Furthermore, the study population is limited to urban, overweight, and obese individuals from a specific region in North India. Future research should include a more diverse population, encompassing rural areas and different ethnic backgrounds, to enhance the generalizability of these findings.

CONCLUSION

In conclusion, this study demonstrates a significant association between serum uric acid levels, obesity, and blood pressure in an urban Indian population. These findings suggest that hyperuricemia may be a crucial contributor to hypertension, particularly in individuals with elevated BMI and central obesity. Regular monitoring of serum uric acid levels, combined with lifestyle modifications and potential pharmacological interventions, could play a critical role in preventing and managing hypertension in at-risk populations. Further research is warranted to explore the underlying mechanisms and develop tailored strategies to address the global burden of hypertension and its related complications.

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