Correlation between Serum Uric Acid and Blood Pressure in Plateau Central

Ayuba Affi¹*

¹Department of Chemical Pathology, University of Jos, Jos, Nigeria.

Author’s contribution

The sole author designed, analysed, interpreted and prepared the manuscript.

ABSTRACT

Aim: To determine the Correlation between Serum uric acid (SUA) and Blood Pressure in Plateau Central.

Background: Serum uric acid, an end product of purine metabolism, has been shown to be associated with an increased risk of hypertension cardiovascular⁴ and chronic kidney disease in previous epidemiological studies. Elevated SUA has been shown to predict the development of high blood pressure and may contribute to increasing BP by several mechanisms. Clinical trials in young patients have supported this mechanism but do not yet agree with pharmacologic reduction of SUA as first-line therapy for hypertension.

Materials and Methods: 200 (males and females) subject of Panyam district of Mangu L. G. C. in Plateau central formed the study population with aged 40 - 90 years. The intervention groups were 120 and control group was 80. Five ml of blood was aliquoted for assay uric acid for each subject using the enzymatic uricase method.

Results: The data obtained were coded into Stata Software for analysis. The data was presented as mean ± S.D. comparison was done by student’s t-test for continuous variables. A BMI of >30 kg/m² was found in 31 individuals giving a prevalence 15.5% with SUA prevalence of 41% with 32% being the prevalence of hypertension.

*Corresponding author: E-mail: ayubaaffi67@gmail.com;
**Conclusion:** The rural farmers were found to have high SUA and positively correlated to hypertension. With the high level of SUA and high BP, advocacy should be made on regular BP checks and possible provision of personal portable BP machine for self-monitoring.

Keywords: Blood pressure; Serum uric acid; hypertension; CVD.

**ABBREVIATIONS**

- **SUA** = Serum uric acid  
- **RAS** = Renni-angiotensin system  
- **HTN** = Hypertension  
- **SBP** = Systolic blood pressure  
- **DBP** = Diastolic blood pressures  
- **BMI** = Body mass index  
- **CVD** = Cardio-vascular diseases

**1. INTRODUCTION**

One of the worldwide public health challenges is hypertension and remains a leading cause of morbidity and mortality. It is the most common form of cardiovascular disease and its prevalence is growing higher with age and other risk factors like obesity, physical inactivity, diabetes mellitus and race [1,2]. The end product of purine metabolism is uric acid which has been shown to be associated with an increased risk of hypertension [3], cardiovascular and chronic kidney disease in previous epidemiological studies. Also, elevated levels of uric acid may progress to peripheral arterial disease, insulin resistance and components of the metabolic syndrome [4]. Hyperuricemia defined as 7 mg/dL in males and 6 mg/dL in females. It is a common metabolic disorder occurring in 2.18% population varying in age, sex and other factors like excessive alcohol [5]. SUA concentration could determine the outcome of hypertension and may also initiate hypertension, though it is less clear that hyperuricaemia can be regarded as an independent risk factor given its association with well-recognised factors. At physiologic concentration, uric acid is antioxidant and beneficial, but at a high level, it becomes a free radical leading to pathological processes [6]. The result of pathophysiological processes of SUA increases such as impaired renal sodium handling but may also contribute to renal vascular damage particularly endothelial dysfunction. This may cause subtle endothelial damage that may stimulate the renni-angiotensin-aldosterone system (RAAS) causing hypertension [7]. More than 20-40% of patients with untreated hypertension and 80% or more patients with malignant hypertension have high SUA levels. Hyperuricaemia common in primary HTN especially in patients with HTN of recent onset is associated with micro albuminuria [8].

The Framingham heart study showed that each increase in SUA by 1.3 mg/dl was associated with the development of HTN with an odd ration of 1.17 [9]. The reduction in SUA to less than 5 mg/dL with allupurinol was associated to the reversal of HTN in 86% of the patients [10]. A 12 years Italian study – (The PIUMA study) involving 1720 previously untreated hypertensive patients, Verdechia and colleagues also found that serum acid was a powerful predictor of cardiovascular disease and all-cause mortality [11]. Because of eminent cardiovascular disease morbidity, it has become pertinent to determine the prevalence of hyperuricaemia and hypertension and the association between SUA and the blood pressure [12-14].

**2. MATERIALS AND METHODS**

**2.1 Research Setting and Design**

This is a prospective study covering all neighbouring villages. 200 farmers and retired civil servants of Panyam district of Mangu L.G.C in Plateau central formed the study population aged 40 - 90 years. Both males and females were recruited into the study. The intervention groups were 120 and control group was 80.

**2.2 Sample and Sampling Method**

This study was carried out at Panyam Primary Health Care.

Permission was obtained from the district head. He mobilised his subjects to have an overnight fast for the study. Blood samples were collected from the subjects into plain specimen tubes between 8:00 to 10:00 am. This blood was spun at 3000 rpm for 5 minutes using gallenkamp bench-centrifugal, after clot retraction. The supernatant (serum) extracted using Pasteur pipettes into storage tubes and stored frozen at -20°C before analysis at Jos University Teaching Hospital Chemical Pathology Laboratory.
2.3 Data Collection and Analysis
Weight was measured using a portable weighing scale (stadiometer) while a meter rule used in the measurement of height. Both were taken in a standing position with shoes, and heavy clothing removed. The body mass index (BMI) was calculated as follows:

$$BMI = \frac{Weight \ (kg)}{height \ (m^2)}.$$ 

BMI was categorised using the WHO definitions.

The aneroid sphygmomanometer was used in the measurement of blood pressure. Blood pressure was measured in the right arm after at least 15 min of rest and while participants were sitting down. The cuff was applied evenly and snugly around the bare arm, with the lower edge 2.5 cm above the antecubital fossa. The participants must not have eaten, smoked tobacco or taken alcoholic beverages for at least 30 minutes before the measurements. The first and fifth Korotkoff sounds were taken as the systolic blood pressure (SBP) and diastolic blood pressures (DBP) respectively. Hypertension was noted if systolic blood pressure 130 mmHg, or upon self-report of a medical diagnosis of hypertension or current treatment for hypertension with prescription medication.

Five ml of blood was aliquoted for the assay. Blood glucose and uric acid were determined by glucose oxidase and uricase enzymatic method respectively. The reference interval for glucose is 3.5–5.9 and for uric acid 120 µmmol/L – 420 µmmol/L.

2.4 Ethics
Individual consent was obtained from all recruited individuals. Ethical clearance was obtained from the research and ethical committee of Jos University Teaching Hospital.

2.5 Statistical Analysis
The stata software was employed for the analysis. Pearson chi-square were used for nominal and the independent samples –test for continuous variables. A value below 0.05 was considered significant.

3. RESULTS

3.1 Characteristics of Study Participants According to Their Quartiles
The analysis was conducted on 200 individuals who had both BMI, GLU, Uric acid and blood pressure assessment. The mean age of the individuals was 63.63 years and approximately 43% were men. SUA prevalence of 41% and 32% prevalence of hypertension was noted.

3.2 Associations
Partial Spearman correlation analysis demonstrated the strongest association between Uric acid and body mass index (BMI). It also signifies a low correlation between uric acid and blood pressure. SBP and DBP had a very strong Correlation from the data analysed.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>1st Quartile</th>
<th>2nd Quartile</th>
<th>3rd Quartile</th>
<th>4th Quartile</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
<td>47.5±7.5</td>
<td>59±3</td>
<td>85±5</td>
<td>110±10</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>BMI</td>
<td>19.58±2.64</td>
<td>23.40±1.05</td>
<td>26.52±2.06</td>
<td>33.73±4.84</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>GLU(F/R)</td>
<td>2.04 – 3.85</td>
<td>3.93 – 4.76</td>
<td>4.81 – 6.5</td>
<td>6.6 – 11.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>URIC ACID</td>
<td>59-246</td>
<td>265-436</td>
<td>450-750</td>
<td>793-1963</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>SBP</td>
<td>110±10</td>
<td>135±5</td>
<td>160±20</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>DBP</td>
<td>75±5</td>
<td>85±5</td>
<td>110±10</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
</tbody>
</table>

Table 1. Characteristics of study participants according to their quartiles

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>1st Quartile</th>
<th>2nd Quartile</th>
<th>3rd Quartile</th>
<th>4th Quartile</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uric acid</td>
<td>0.1453</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>-0.0394</td>
<td>-0.0740</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>-0.0758</td>
<td>-0.0767</td>
<td>0.7561</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBP</td>
<td>0.1265</td>
<td>0.0108</td>
<td>0.0972</td>
<td>0.0250</td>
<td></td>
</tr>
<tr>
<td>GLU(F/R)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Partial spearman correlation coefficients among uric acid, blood pressure, body mass index
4. DISCUSSION

This study was set-put to examine the prevalence of hypertension and hyperuricaemia among middle age and elderly rural farmers. The second aim was to determine any association between SUA levels and hypertension.
A high-risk group for the development of hypertension is the African population and identifying risk factors is therefore important for preventive actions against cardiovascular diseases [15].

Based on the seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of high blood pressure [16], normal BP is defined as less than 120/80 mmHg. The SBP was obviously higher in elderly intensive than the control. It is known that BP increases with age so it is consistent with previous work done [16] in HTN association with age; this only further proves that point. From this study, it was found that a mean SUA of 569 µmol/L compared with the control 484 µmol/L from studies [17], it showed that the development of HTN has generally been consistent. The SUA was found to be significant, higher in the intensives than the control. Previous work revealed SUA to be higher and cause HTN in the young. We discovered in this elderly population that SUA was significantly higher than the control for the reason of advanced age and the elevated BP. This work was in consistency with the Framingham Heart Study [18-19] that reported SUA was not casual risk factors for CVD events because uric acid was not independent of HTN. Logical data on the independent prognostic role of SUA might be accounted for the complex interrelations between SUA and a variety or risk markers for cardiovascular diseases including male gender epidemiological cardiovascular events.

This work showed a consistency with systolic HTN in the elderly program and Chicago studies in several individuals [20].

In our study, the relationship of SUA to CV HTN is and also apparent from inspection of studies by Alderman et al. [21] in subjects with hypertension. Longitudinal studies are needed to clarify the potential value of SUA to reflect and predict the vicious cycle leading to progressive renal damage and elevated blood pressure. Increased activity of the sympathetic nervous system has also been associated with reduced renal excretion of uric acid but the basic mechanisms are unknown [22].

The present study demonstrates a strong independent association between SUA and HTN, initially untreated and asymptomatic adult subjects with essential hypertension, but it is unable to answer the question of whether SUA exerts direct toxic effects.

Under most conditions, an elevated SUA is in equilibrium with intracellular level [23]. However, one confounding aspect is that SUA levels are known to fall in diabetic subjects, as glycosuria can lead to proximal tubular dysfunction and uricosuria. SUA levels have been also reported to be higher in those with better diabetes control.

A possible explanation for high-level SUA in the population is the high indulgence in local alcohol beverage which is known to cause hyperuricaemia. Another reason for high

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**Fig. 3. SBP and percentage population**

![Bar chart showing SBP and percentage population](image)
hyperuricemia and hypertension is African population is known that blood pressure was more pronounced in the African men [24].

The adoption of more western seed diet, because industrialisation had a great effect on the latter mentioned. Due to the high content of sugar (fructose) in western seed diet, the prevalence of obesity and diabetes increased resulting in concomitant increases in SUA levels. A confounding factor such as BMI could explain this association [25].

The finding this work relating SUA to hypertension incidence confirmed several previous reports. The strength of the association was modest in our study compared to other reports, for example, in the Olivet Study a high increment in SUA was associated an OR of 1.23 for hypertension incidence during 12 years follow up [13].

5. CONCLUSION

It is concluded that rural farmers were found to have high SUA and significantly correlated with hypertension. With the high level of SUA and high BP, advocacy should be made on regular BP checks and possible provision of personal portable BP machine for self-monitoring. There is a need for further health awareness on the control in the consumption of local alcoholic beverage and other lifestyle modification such as regular exercise and personal hygiene.

CONSENT AND ETHICAL APPROVAL

Individual consent was obtained from all recruited individuals. Ethical clearance was obtained from the research and ethical committee of Jos University Teaching Hospital.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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