Correlation between SUA and Blood Pressure in Plateau Central

Abstract

Aim: To determine the Correlation between 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 cardiovascular[4] and chronic kidney disease in previous epidemiological studies.

Elevated SUA has been shown to predict development of high blood pressure and may contribute to increase 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 Method

200 subject of Panyam district of Mangu L. G. C. in Plateau central formed the study population with age range which was between 40 – 90 years age.

Both males and females were recruited into study. The intervention groups were 120 and control group was 80.

Blood samples was collected from the subjects into plain specimen tubes between the hours of

Hypertension was noted if systolic blood pressure 130mmHg, or upon self report of a medical diagnosis of hypertension or current treatment for hypertension with prescription medication.

Five mls of blood was aliquoted for assay uric acid was determined for each subject using the enzymatic uricase method for the enzymatic serum uric acid assay. Uricase method for uric acid assays

Results

The data obtained were coded into Stata Software for analysis. The data are presented as mean ± S.D. comparison was done by student's t-test for continuous variables. A BMI of >30kg/m^2 was found in 31 individuals giving a prevalence 15.5% with SUA prevalence of 41% with 32% being prevalence of hypertension.

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.

There is need for further health awareness on the control of the consumption the local alcoholic beverage and other lifestyle modification such as regular exercise and personal hygiene.

Further studies are needed to confirm these observations and to proof whether the relationship observed between SUA and incident hypertension at short-term follow-up is causal.

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 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 and 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 factor for peripheral arterial disease, insulin resistance and components of the metabolic syndrome[4]. Hyperuriceamia defined as 7mg in males and 6mg in females of common metabolic disorder occurring in 2.18% population varying among young to aged, 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 independent risk factor given its association with well recognized factors. At physiologic concentration, uric acid is antioxidant and beneficial, but at high level it becomes proxidant leading to pathological processes[6].

The result of pathophysiological processes of SUA increase such as impaired renal sodium handling but may also contribute to renal vascular damage particularly endothelial dysfunction. This may cause subtle endothelial damage that could lead stimulation of the renni-angiotensin-aldosteron 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 and in prehypertension associated with micro albuminuria[8]

The Framingham heart study showed that each increase in SUA by 1.3mg/dl was associated to the development of HTN with an odd ration of 1.17[9]

In normotensive men with SUA level greater than 7mg/dl there was an 80% increased risk for the development of HTN in a study the high SUA was observed in nearly 90% of adolescents with primary HTN and SUA level correlates with both systolic and diastolic HTN. The reduction in SUA to less than 5mg/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 *et al* and colleagues also found that serum acid was a powerful predictor of cardiovascular disease and all cause mortality[11].

It has not made known the prevalence of hyperuricaemia or blood pressure and the relationship between SUA and blood pressure in elderly rural farmers of plateau central.

It is well established that SUA predicts development of hypertension but the prevalence of hypertension and hyperuricaemia is not known in this setting. The association of SUA and hypertension is not known in this population.

Therefore, 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].

Materials and method

Research setting and Design

This is a cross sectional prospective study covering all neighbouring villages.

120 famers of Panyam district of Mangu L.G.C in Plateau central formed the study population with age range which was between 40 – 90 years age. Most of them were farmers or retired civil servants.

Both males and females were recruited into study. The intervention groups were 120 and control group was 80.

Sample and Sampling Method

This study was carried out at Panyam Primary Health Care.

Permission was obtained from district head. He mobilized his subjects to come to the PHC with overnight fast for the study.

A brief health education was given.

Blood samples was collected from the subjects into plain specimen tubes between the hours of 8:00am an 10:00am from the subject at the fasting state.

This blood was spun at 3000rpm for 5 minutes using gallenkamp bench-centrifugal, after clot retraction. The supernatant(serum) is extracted using Pasteur pipettes into storage tubes and stored frozen at -20^{o}C prior to analysis at Jos University Teaching Hospital Chemical Pathology Laboratory.

Data Collection and Analysis

Weight was measured using a portable weighing scale(standiometer) while a meter rule used in measurement of height. Both were taken in a standing position with shoes, and heavy clothing removed. The body mass index(BMI) was calculated using the formula. Weight (kg), divided by a square of the height(m). BMI was categorized using the WHO definitions: BMI>30kg/m^2 was defined as obesity while participants with BMI of 25.0 and 29.9 were considered overweight. underweight individuals were those with BMI<18.5 while normal weight individuals were those with BMI between 18.5 and 24.9. obesity was further sub-classified into class I (30-34.9kg/m2), class II(35-39.9kg/m2) and class III (>40kg/m2).

The aneroid sphygmomanometer was used in 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 130mmHg, or upon self report of a medical diagnosis of hypertension or current treatment for hypertension with prescription medication.

Five mls of blood was aliquoted for the assay. Blood glucose and uric acid was determined by glucose oxidase and uricase enzymatic method respectively.

Reference interval for glucose is 3.5-5.9 and for uric acid 120μmmol/L - 420μmmol/L.

Ethics

Individual consent was obtained from all recruited individuals

Ethical clearance was obtained from research and ethical committee of Jos University Teaching Hospital

Statistical Analysis

The stata software was employed for the analysis.

Pearson chi square will be used for nominal and the independent samples –test for continuous variables. A value below 0.05 was considered significant

Results

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. Prevalence 15.5% with SUA prevalence of 41% with 32% being prevalence of hypertension.

157 Table 1

158 **Characteristics of study participants according to their quartiles**

Characteristics	1ST QUARTILE	2ND QUARTILE	3RD QUARTILE	4TH QUARTILE	P-VALUE
	≤0.2	**0.2-0.24**	**0.25-0.29**	**≥0.30**	
AGE	47.5±7.5	59±3	85±5	110±10	<0.0001
BMI	19.58±2.64	23.40±1.05	26.52±2.06	33.73±4.84	<0.0001
GLU(F/R)	2.04 – 3.85	3.93 – 4.76	4.81 – 6.5	6.6 – 11.6	<0.0001
URIC ACID	59-246	265-436	450-750	793-1963	<0.0001
SBP	110±10		135±5	160±20	<0.0001
DBP	75±5		85±5	110±10	<0.0001

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160 **Associations**

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

165 Table 2.

166 **Partial Spearman correlation coefficients among Uric acid, blood pressure,**
167 **Body mass index and**

	URIC ACID	BMI	SBP	DBP
BMI	0.1453			
SBP	-0.0394	-0.0740		
DBP	-0.0758	-0.0767	0.7561	
GLU(F/R)	0.1265	0.0108	0.0972	0.0250

168 **Graphical Representation**

169 **Histograms**

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Fig1

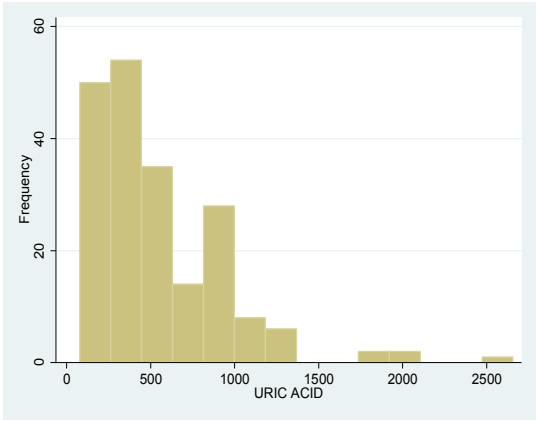

Fig 1 shows uric acid levels and percentage population

Fig2

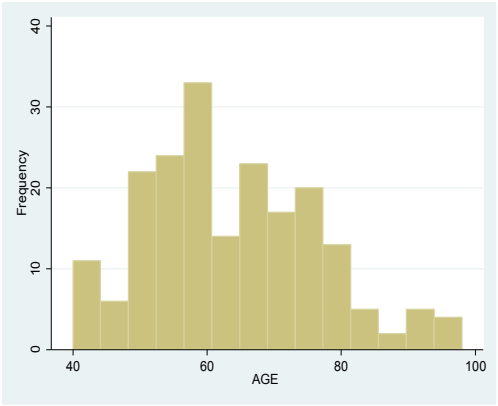

Fig2 shows age and percentage

Population

Fig 3

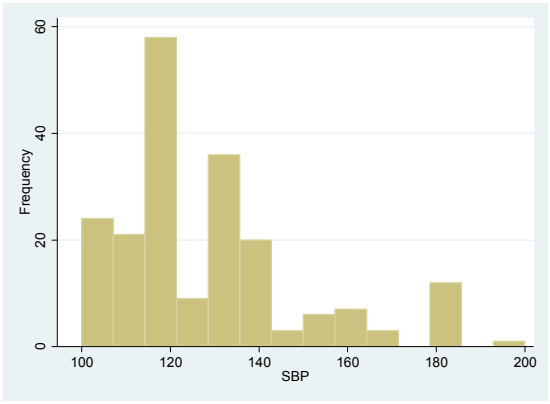

Fig 3 shows SBP and percentage population

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 Africa 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 120/80mmHg. The SBP was obviously higher in elderly intervensive than the control. Clearly, 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 569µmol/L compared with the control 484µmol/L from studies[17], it showed that development of HTN have generally been consistent. The SUA was found to be significant, higher in the intertensives 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 consistence with the Framingham Heart Study[18-19] that reported SUA was not casual risk factors for CVS 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 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 hypeuricaemia and hypertension is African population is known that blood pressure was more pronounce in the African men[24].

The adoption of more western seed diet, because of industrialization had a great effect on the latter mentioned. Due to 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 report. 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[26].

Conclusion and recommendation

In conclusion, rural farmers were found to have high SUA and positively associated 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 need for further health awareness on the control of the consumption the local alcoholic beverage and other lifestyle modification such as regular exercise and personal hygiene.

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