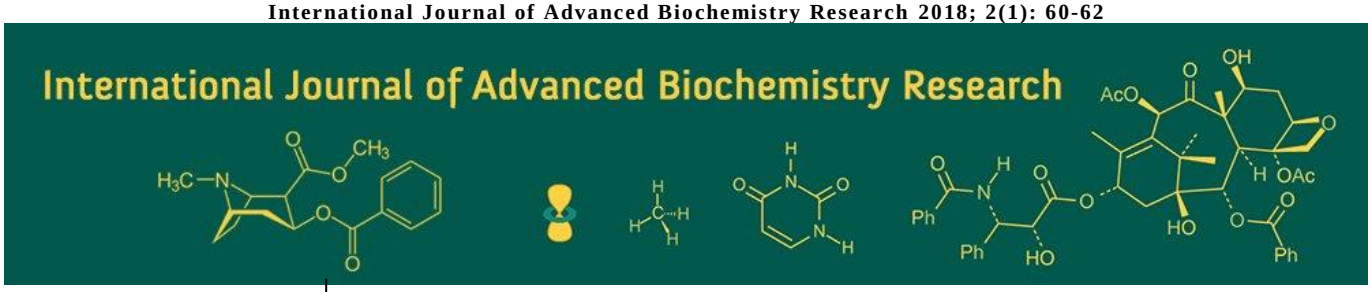

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A study on association of serum uric acid in hypertensive patients

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Abstract
Background: Uric acid is the end product of purine metabolism. Metabolic disorders of uric acid are associated with many disease states. Substantial evidence suggests the possible role of uric acid as a mediator of high blood pressure. Elevated uric acid is closely associated with new onset essential hypertension in adolescents and prehypertension; and urate-lowering agents can significantly improve these early stages of hypertension. Uric acid also influences salt sensitivity of blood pressure through two phases. Local renin-angiotensin-aldosterone system activation initiates renal damage, arteriolopathy, and endothelium dysfunction, which is followed by the dysregulation of sodium homeostasis, thereby leading to increased salt sensitivity. In this review we summarize the available evidence to contribute to a better understanding of the casual relationship between uric acid and early or intermediate stages of hypertension. We hope our review can contribute to the prevention of hypertension or provide new insights into a treatment that would slow the progression of hypertension.

Keywords: Serum uric acid, blood pressure, hypertension, hypertensive

Introduction
Uric acid is the end product of purine metabolism. Metabolic disorders of uric acid are associated with many disease states. Substantial evidence suggests the possible role of uric acid as a mediator of high blood pressure. Elevated uric acid is closely associated with new onset essential hypertension in adolescents and prehypertension; and urate-lowering agents can significantly improve these early stages of hypertension. Uric acid also influences salt sensitivity of blood pressure through two phases. Local renin-angiotensin-aldosterone system activation initiates renal damage, arteriolopathy, and endothelium dysfunction, which is followed by the dysregulation of sodium homeostasis, thereby leading to increased salt sensitivity. In this review we summarize the available evidence to contribute to a better understanding of the casual relationship between uric acid and early or intermediate stages of hypertension. We hope our review can contribute to the prevention of hypertension or provide new insights into a treatment that would slow the progression of hypertension.

Hypertension is an increasing important medical and public health issue. Hypertension markedly increases the risk for myocardial infarction, stroke, congestive heart failure, peripheral vascular disease and end stage renal disease. Various risk factors for development of hypertension, both modifiable and non-modifiable, have been identified to aid in its prevention and management. In recent years, various studies have shown serum uric acid (SUA) levels to be an independent predictor for developing hypertension [1].

Uric Acid (UA) is a heterocyclic compound whose concentration in the body depends upon the balance between purine breakdown and rate of urate excretion [1]. Plasma uric acid is a circulating marker of oxidative damage in a variety of pathological conditions such as ischemic liver injury, hyperlipidemia, chronic heart disease, atherosclerosis, ischemic reperfusion injury, and diabetes [2]. The serum uric level depends on gender, lifestyle, meals, and previous use of diuretics [3].

Uric acid exerts a pro-inflammatory effect on endothelial cells which may be associated with MetS risk factors such as elevated triglyceride (TG) levels, hypertension and insulin resistance [4]. Moreover, in recent years, elevated SUA levels in adults have been suggested as CVD risk factors in some studies [5, 6]. Present study was aimed to study of association of serum uric acid and blood pressure in hypertensive patients at a tertiary hospital.

Material and methods

Present study was prospective, comparative, observational study, conducted under department of Biochemistry, at Kanachur Institute of Medical Sciences, Mangalore. Study duration was of 1 year (July 2016 to June 2017).

Inclusion criteria

- Cases – Subjects of either gender, age >18 years, diagnosed as hypertensive (first time) were enrolled in this study during a regular routine health check-up at general medicine OPDs.
- Controls - Age & gender matched normotensives subjects at general medicine OPDs.

Exclusion criteria

- Individuals having pre-existing hypertension
- Individuals having a known history of gout and cardiac or severe renal diseases
- Subjects who were already under medication for anti-hyperuricemic
- Subjects who were not willing to participate

Study protocol was informed to all subjects and written informed consent was obtained from them prior to enrolment in the study. General information such as name, age, gender, previous medical history was noted in CRF. General & systemic examination findings along with Body mass index (BMI) [body weight in kgs divided by (height in m)^2] was noted in CRF.

Blood pressure (BP) was measured by trained professionals using a digital BP machine (Philips) on the left arm in a sitting position after at least 10 minutes of rest. Three recordings of blood pressure as systolic and diastolic blood pressure (SBP and DBP) has been taken after a minimum of 5 minutes of rest to avoid any possible effects of anxiety and with an interval of 5 minutes.

Hypertension was defined as per JNC -7 classification of hypertension [7].

The venous blood samples were obtained after an overnight fasting (≥12hrs) for estimation of complete blood counts, renal function tests (Serum Urea, Serum Creatinine & Serum uric acid), Thyroid-stimulating hormone (TSH), random blood sugar and serum lipids: total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high- density lipoprotein cholesterol (HDL-C), The values were determined calorimetrically using commercially available diagnostic kits using an auto-analyser. Hyperuricemia was defined as SUA levels >416.4 _mol/L (7.0 mg/dL) in men and >356.9 _mol/L (6.0 mg/dL) in women.

Data was collected and compiled using Microsoft Excel, analysed using SPSS 23.0 version. Frequency, percentage, means and standard deviations (SD) was calculated for the continuous variables, while ratios and proportions were calculated for the categorical variables. Pearson's correlation coefficient test was performed to assess the interrelationships between baseline variables and SUA concentrations. Difference of proportions between qualitative variables were tested using chi- square test or Fisher exact test as applicable. P value less than 0.5 was considered as statistically significant.

Results

Table 1: General characteristics

Variables	Hypertensive (n=100) [mean ± SD/ n (%)]	Normotensive (n=100) [mean ± SD/ n (%)]
Age (years)	53.75 ± 9.74	51.38 ± 11.23
Gender		
Male	69 (69 %)	71 (71 %)
Female	31 (31 %)	29 (29 %)
BMI (kg/m2)	22.51 ± 2.56	22.94 ± 2.16
Co-morbidity		
Smoking,	19 (19 %)	15 (15 %)
Alcohol,	19 (31 %)	16 (16 %)
Tobacco chewing,	16 (16 %)	15 (15 %)

Table 2: Examination findings

Variables	Hypertensive (mean ± SD)	Normotensive (mean ± SD)	P value
Pulse rate (/min)	81.35 ± 11.54	80.61 ± 10.97	0.089
Respiratory rate (/min)	18.96 ± 3.01	19.09 ± 2.91	0.086
SBP (mm Hg)	155.35 ± 18.51	131.38 ± 14.91	<0.0001
DBP (mm Hg)	96.12 ± 7.11	83.92 ± 6.01	<0.0001

Table 3: Hematological characteristics

Variables	Hypertensive (mean ± SD)	Normotensive (mean ± SD)	P value
Hemoglobin (g%)	11.64 ± 2.49	11.17 ± 2.47	0.082
Total Count (/mm^3)	7757.1 ± 3563.8	7855.5 ± 3921	0.069
Platelet Count (/mm^3)	218.06 ± 85.76	230.47 ± 91.95	0.081
Serum Urea (mg/dL)	27.81 ± 8.97	26.19 ± 9.01	0.055
Serum Creatinine (mg/dL)	0.81 ± 0.41	0.69 ± 0.24	0.068
Serum uric acid (mg/dL)	6.43 ± 2.11	6.01 ± 1.93	<0.0001
Fasting Blood Sugar (mg/dL)	109.69 ± 17.89	102.61 ± 23.13	0.074
TSH (mIU/mL)	2.24 ± 0.82	2.19 ± 0.78	0.075
Total Cholesterol (mg/dL)	213.78 ± 38.74	213.19 ± 42.29	0.064
LDL (mg/dL)	109.46 ± 22.98	109.7 ± 24.86	0.056
HDL (mg/dL)	52.28 ± 15.78	43.34 ± 15.2	0.045
TG (mg/dL)	103.71 ± 39.74	139.63 ± 43.72	0. 041

Discussion

In study by Chanchal S *et al.* [7] mean serum uric acid level in group A (Essential Hypertension) was significantly higher than group B (normotensive cases) (6.56 ± 0.76, 4.91 ± 0.97 mg/dl, $p<0.001$ respectively). 37.33% of patients had hyperuricaemia in group A as compared to 14% in group B ($p<0.01$, OR=3.66) indicating that a hyperuricaemic individual has 3.66 times more risk of developing Essential Hypertension as compared to the one with lower value of serum uric acid. Serum uric acid could be useful as a potential indicator for early risk detection of development of EHT.

Sujeet Raina *et al.* [8] studied 50 newly diagnosed cases of essential hypertension and 50 age and sex matched normotensive healthy volunteer. Prevalence of hyperuricemia was 24% among the cases and 6% among the controls ($p<0.05$). Odds ratio was 4.9 (CI=1.3 to 18.8). The mean SUA was significantly higher in the cases (5.5±1.7 mg/dl) than in the controls (4.9±1.1 mg/dl; $p<0.05$). Odds ratio in male hyperuricemic hypertensive versus hyperuricemic normotensive was 6 (CI=1.0 to 33.2) and 4.46 (CI=0.4 to 42.5) among female hyperuricemic hypertensive versus hyperuricemic normotensives.

Despite the clinical and epidemiological evidence, many authorities do not consider an elevated uric acid to be a true cardiovascular risk factor, because patients with hyperuricemia often have other well-established risk factors for cardiovascular disease, such as hypertension, renal disease, obesity, dyslipidemia, and insulin resistance.

At the tissue level, chronic exposure to increased UA promotes vascular changes leading to renal ischemia and stimulation of renin-angiotensin system and development of insulin resistance, hypertriglyceridemia, and hepatic steatosis through pro-oxidative mechanisms. Therefore, early screening of UA levels is advisable to prevent and manage complications of elevated levels of SUA [9].

Present study limitations were a cross-sectional study, did not permit us to make any inference on the causal relationship between uric acid and hypertension. Secondly, the limited sample size also limited the power of the analysis. A further study designed as a prospective randomized follow up study with a larger sample size would be required to substantiate the results of the present study.

Conclusion

Estimation of SUA levels is advisable at the diagnosis of hypertension in order to diagnose, prevent and manage complications of elevated levels of hypertension

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