



AN EFFECT OF SERUM URIC ACID LEVELS IN HYPERTENSION

Clinical Biochemistry

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ABSTRACT

BACKGROUND: Hypertension (HTN or HT), also known as high blood pressure (HBP), is a long-term medical condition in which the blood pressure in the arteries is persistently elevated. Worldwide, raised blood pressure is estimated to cause 7.5 million deaths, about 12.8% of the total of all deaths. Elevated levels of serum uric acid are strongly associated with development and progression of hypertension and renal diseases, but whether uric acid plays a causal role or whether it simply acts as an indicator in patients at risk for these conditions remains controversial. Many authorities do not consider an increased uric acid to be a true risk factor for cardiovascular diseases, because patients with hyperuricemia mostly have other well established risk factors for cardiovascular conditions like hypertension, renal disease, obesity, dyslipidaemias, and insulin resistance. This study was aimed to evaluate the levels of uric acid in hypertensive cases and normotensive controls. **MATERIAL AND METHOD:** This study was conducted on 60 individuals in which 30 considered as case and they having hypertension apart from any other non-communicable diseases. 30 healthy individuals also taken as controls. This study was conducted in a tertiary care centre. **RESULTS:** As we compared both of groups, we found a significant correlation between the uric acid and hypertension. Statistical analysis was done on SPSS by using Karl's Pearson's correlation. **CONCLUSION:** Analysed levels are shows that as systolic and diastolic blood pressure so that suggestion for future references is to maintain the blood pressure according to the guidelines as well as maintain the food restrictions and give preferences to physical exercise.

KEYWORDS

Hypertension, Uric Acid and Normotensive

INTRODUCTION

HTN is known as the "quiet executioner" since it normally has no notice signs or indications, and numerous individuals don't know they have it. A little measure of individuals may encounter manifestations, for example, dull cerebral pains, spewing, dazed spells, and more incessant nosebleeds. These side effects more often than not don't happen until the point when circulatory strain levels have come to a serious or dangerous stage. The best way to know for certain if a man has HTN is to have a doctor or other human services proficient measure blood pressure [1,2]. It has been evaluated that around 970 million individuals worldwide have hypertension (JNC8, 2015). By 2025 it is evaluated that 1.56 billion grown-ups will live with hypertension (HTN) [2-5]. Gigantic harm of veins caused by hypertension [6].

Hypertension is given this name due to a man not having any perceptible manifestations; a man can have hypertension for a considerable length of time without knowing it. Blood Pressure is the measure of power connected by the blood within the corridors as the blood is pumped all through the circulatory framework. Each time the heart muscle contracts, blood is squeezed against the dividers of the corridors and is estimated as systolic blood pressure (top number). At the point when the heart muscle unwinds between thumps, the weight on the conduit divider facilitates estimated as diastolic blood pressure. Hypertension is given this name due to a man not having any perceptible manifestations; a man can have hypertension for a considerable length of time without knowing it [7]. Uric acid at a certain blood pH value it works as predominantly urate ion but it is basically a weak acid and a heterocyclic compound. XOR (Xanthine oxidoreductase) catalyses the generation of urate from hypoxanthine and xanthine, and this is the end product of purine catabolism as in endogenous and exogenous pathways [8]. As xanthine oxidoreductase has two properties and it consist in two form one is oxidase and another is dehydrogenase, Oxidase term as more prevalent form [9]. During the Miocene epoch, humans lost the capacity to metabolize uric acid into allantoin due to nonsense mutations in the gene codifying the enzyme uricase [10]. However, a minimal amount of uric acid can be indirectly metabolized through its reaction with oxidants, lipid radicals, nitric oxide, and peroxynitrite [11]. Increased purine degradation (e.g., during DNA, RNA, and ATP breakdown) also leads to a rise in SUA [12]. In addition, increased activity of aldose reductase and XO, as occurring during ischemia, heat stress, and dehydration, have been associated with a rise in intracellular uric acid and serum uric acid. Uric acid-induced oxidative stress and inflammation may play a role, being responsible for the pathogenesis of endothelial dysfunction [12-15,23,24].

Another possible cause of afferent arteriopathy is the uric acid mediated vascular smooth muscle cell proliferation [15,16,17,18]. Uric acid mediated hypertension may also be caused by the up-regulation of thromboxane and endothelin-1. In fact, both these vasoconstrictor molecules can be over-expressed in the presence of high uric acid concentrations, inducing the development of hypertension [19,20]. Uric acid can also mediate the development of hypertension through a crystal-dependent pathway [21,22]. In fact, monosodium urate deposits have been found in the kidney medulla, cardiac valves, arteries, and within the atherosclerotic plaque [21,22]. These deposits could lead to kidney injury and arterial stiffness, increasing the risk for hypertension [21,22].

MATERIAL & METHODS:

Study design and study subjects: This was an institutional based case control comparative study between January 2018 to June 2018. A total of 60 subjects (30 cases and 30 control) were enrolled in this study who were attending/admitted in department of medicine of Integral Institute of Medical Sciences and Research, Lucknow. All study subjects were informed about the study aims and written informed consent was obtained from them prior to enroll in the study. A detailed clinical history including age, sex, occupation, duration of illness was collected from the patients. Exclusion criteria for case Diabetes Mellitus, Gout, History of cardiovascular, Kidney disease, Pregnant females and those who refuse to being part of this study. For the second group those who were healthy control and they not suffer from any disorder. The Ethics Committee at the Integral Institute of Medical Sciences and Research, Lucknow approved the study. All the steps of the methods section were conducted in accordance with the institutional guidelines and regulations.

General data collection. General data age, sex and other demography data were taken from the subject who attend outdoor as well indoor in department of medicine of Integral Institute of Medical Sciences and Research, Lucknow.

Measurements of blood pressure variables. Blood pressure were taken up by the professionals using sphygmomanometer (Mercury Blood Pressure apparatus). Blood pressure taken from left arm on sitting position with ten minutes of resting phase. Local agents (Tea, Coffee, smoking) which can increase blood pressure should be prohibited by the patient before taking their blood pressures (SBP/DBP). Hypertension patients' criteria according to JNCVIII the

systolic blood pressure >140mmHg and diastolic blood pressure >90mmHg.

Blood samples and Principle.

Two groups were taken for the study at different selection criteria for the cases age group 18 – 65 years. The cases will be selected as per the proforma enclosed. 2 ml of blood were collected from both of groups and collected in plain (red top) vial. After getting the blood sample it undergoes with the centrifugation process when serum separated. Select the literature for analysis for this estimation uric acid the selected method was uricase/PAP method. Uric acid containing samples after mixing with reagent it convert into allantoin and hydrogen peroxide due to the presence of uricase after that hydrogen peroxide react phenolic compound and 4-aminoantipyrine by the catalytic action of peroxidase, due this reaction red coloured quinoneimine dye complex. Intensity of colour is directly proportional to the uric acid concentration present in the sample.

Statistical Analysis:

Numerical facts analyzed by using IBM SPSS version 20. In tables, numeric data are summarized as mean $\pm$ SD. The difference between was done by student's unpaired sample t-test (two-tailed). A p-value < 0.05 was considered to be statistically significant.

RESULT:

Mean age in years in cases we found 47.3 ± 12.99 and for the control it was 35.26 ± 12.14 and these numerical facts clearly shows that as age factor increases the level of serum uric acid also hoisted with hypertension secondly, we found significant correlation $p < 0.0005$.

Table 1. Subjects' variable settled in form of Mean $\pm$ SD. P-values are obtained from student's unpaired t-test in comparison between the case and control groups.

	Case (n=30)		Control (n=30)		p-Value
	Mean $\pm$ SD	Max	Mean $\pm$ SD	Max	
Age (years)	47.3 ± 12.99	65	35.26 ± 12.14	65	<0.0005*
SBP (mmHg)	154 ± 17.7	180	118.8 ± 4.46	120	<0.0001*
DBP (mmHg)	98.2 ± 8.08	96	74.86 ± 7.13	80	<0.0001*
Uric Acid (mg/dl)	8.39 ± 2.48	13	4.40 ± 1.34	7	<0.0001*

*Statistically Significant

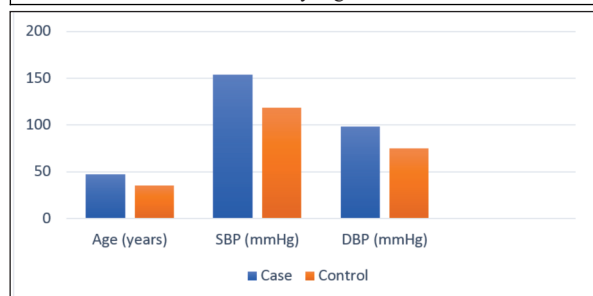


Figure No.1: Represent the difference in both case and control groups.

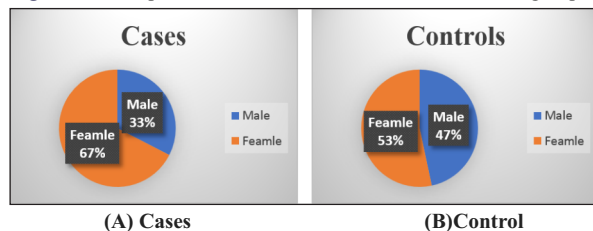


Figure No.2: Incident of gender in both groups. A for cases and in this chart the incidence of female is 67%.

		URICACID	DBP	SBP
	Pearson Correlation	1	.428*	.483**
	Sig. (2-tailed)		.018	.007
DBP	N	30	30	30
	Pearson Correlation	.428*	1	.722**
	Sig. (2-tailed)	.018		.000
SBP	N	30	30	30
	Pearson Correlation	.483**	.722**	1
	Sig. (2-tailed)	.007	.000	
	N	30	30	30

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 2. Karl Pearson's Correlation in serum uric acid with systolic and diastolic blood pressure

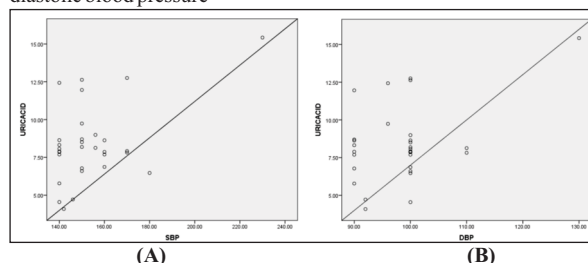


Figure No.3: Scatter diagram showing correlation A (Uric acid vs SBP) and B (Uric acid vs DBP). In the observation and evaluating of numerical found that the hoist level of uric acid found in hypertensive cases when compare with the controls. Mean blood pressure for the hypertensive group after calculating 154 ± 17.7 mmHg for systolic and diastolic mean is 118.8 ± 4.46 . As per JNC-VIII the grade of evaluating group under hypertension range I.

DISCUSSION:

discovered essentially hoisted levels of serum uric acid in both male cases and female cases as compared to their individual control gathering. Late epidemiological investigations have other conclusion that uric acid is a noteworthy and autonomous hazard factor for the improvement of cardiovascular issue and assume a critical job being developed of hypertension and renal disease [25, 26].

Test contemplates have exhibited that expanded uric acid instigates fundamental hypertension and renal damage by means of actuation of the renin angiotensin framework. Uric acid enters specifically into both endothelial and vascular smooth muscle cells and causes nearby hindrance of endothelial nitric oxide levels. It is in charge of incitement of vascular smooth muscle cell multiplication, and incitement of vasoactive and incendiary middle people, bringing about choking of vessels and hypertension [27, 28].

This builds purine oxidation, which prompts expanded receptive oxygen species, resulting vascular damage, decreased nitric oxide level taken after by vasoconstriction and lifted circulatory strain. In the present examination it is demonstrated that serum uric acid levels are expanded relatively when the systolic and diastolic pulse is expanded.

CONCLUSION:

As we mentioned hypertension is quiet executioner so it hard to diagnose with very few symptoms but as our study conclude that serum uric acid elevated with systolic/diastolic blood pressure. We also found that incidence of female in case group is high.

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