Available online at **www.ijmrhs.com**

ISSN No: 2319-5886

International Journal of Medical Research & Health Sciences, 2021, 10(12): 64-69

An Effect of Serum Uric Acid Levels in Hypertension

Mohmmad Amil Rahman[1*], Priyanka Thapa Manger[2] and Vivek Katiyar[3]

[1]Department of Biochemistry, Dr. Rajendra Prasad Govt. Medical College Kangra, Tanda, India

[2]Department of Biochemistry, Integral Institute of Medical Sciences and Research, Lucknow, India

[3]Department of Medicine, Integral Institute of Medical Sciences and Research, Lucknow, India

[*]Corresponding e-mail: kingamil007@gmail.com

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 the 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 whom 30 were considered as cases and they had hypertension apart from any other non-communicable diseases. 30 healthy individuals were also taken as controls. This study was conducted in a tertiary care center.* ***Results:*** *As we compared both groups, we found a significant correlation between uric acid and hypertension. Statistical analysis was done on SPSS by using Karl's Pearson's correlation.* ***Conclusion:*** *Analyzed levels are shown 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, Normotensive

INTRODUCTION

HTN is known as the "quiet executioner" since it normally has no noticeable 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 proficiently 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 works as predominantly urate ion but it is a weak acid and a heterocyclic compound. XOR (Xanthine Oxidoreductase) catalyzes 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 consists of two forms one is oxidase and another is dehydrogenase, Oxidase term a 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 [12-17]. Another possible cause of afferent arteriolopathy is the uric acid-mediated vascular smooth muscle cell proliferation [15,18-20]. Uric acid-mediated hypertension may also be caused by the up-regulation of thromboxane and endothelin-1. Both these vasoconstrictor molecules can be over-expressed in the presence of high uric acid concentrations, inducing the development of hypertension [21,22]. Uric acid can also mediate the development of hypertension through a crystal-dependent pathway [23,24]. Monosodium urate deposits have been found in the kidney medulla, cardiac valves, arteries, and within the atherosclerotic plaque [23,24]. These deposits could lead to kidney injury and arterial stiffness, increasing the risk for hypertension [23,24].

MATERIALS AND METHODS

Study Design and Study Subjects

This was an institutional-based case-control comparative study from January 2018 to June 2018. A total of 60 subjects (30 cases and 30 control) were enrolled in this study who were attending/admitted to the 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 before enrolling 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 be part of this study. For the second group, those who were healthy control and they did 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 by the institutional guidelines and regulations.

General Data Collection

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

Measurements of Blood Pressure Variables

Blood pressure was taken up by the professionals using a sphygmomanometer (Mercury Blood Pressure apparatus). Blood pressure is taken from a left arm in a 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 pressure (SBP/DBP). Hypertension patients' criteria according to JNCVIII the systolic blood pressure >140 mmHg and diastolic blood pressure >90 mmHg.

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 groups and collected in a plain (red top) vial. After getting the blood sample it undergoes the centrifugation process when serum is 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 covert into allantiol and hydrogen peroxide due to the presence of uricase after that hydrogen peroxide reacts with phenolic compound and 4-amino antipyrine by the catalytic action of peroxidase, due to this reaction red-colored quinoneimine dye complex. The intensity of the color is directly proportional to the uric acid concentration present in the sample.

Statistical Analysis

Numerical facts were analyzed by using IBM SPSS version 20. In tables, numeric data are summarized as mean ±

SD. The difference was done by the student's unpaired sample t-test (two-tailed). A p-value<0.05 was considered to be statistically significant.

RESULTS

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 and Figure 1).

Table 1 Subjects' variable settled in form of Mean ± SD; p-values are obtained from student's unpair t-test in comparison between the case and control groups

	Case (n=30)		Control (n=30)		p-Value
	Mean ± SD	Max	Mean ± 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					

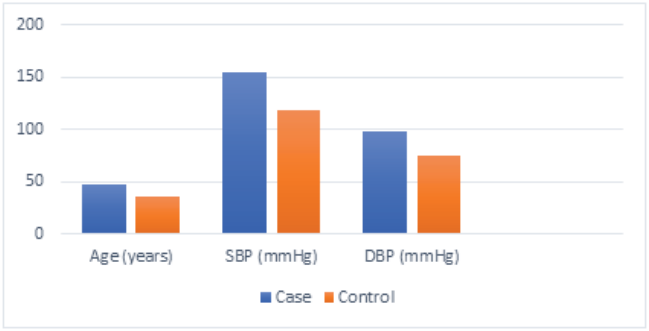

Figure 1 Represent the difference in both case and control groups

In the observation and evaluating of numerical found that the hoist level of uric acid was found in hypertensive cases when compared 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 ranges I (Table 2, Figure 2, and Figure 3).

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

		Uric Acid	DBP	SBP
Uric Acid	Pearson Correlation	1	0.428*	0.483**
	Sig. (2-tailed)		0.018	0.007
	N	30	30	30
DBP	Pearson Correlation	0.428*	1	0.722**
	Sig. (2-tailed)	0.018		0
	N	30	30	30
SBP	Pearson Correlation	0.483**	0.722**	1
	Sig. (2-tailed)	0.007	0.000	
	N	30	30	30
*: Correlation is significant at the 0.05 level (2-tailed); **: Correlation is significant at the 0.01 level (2-tailed)				

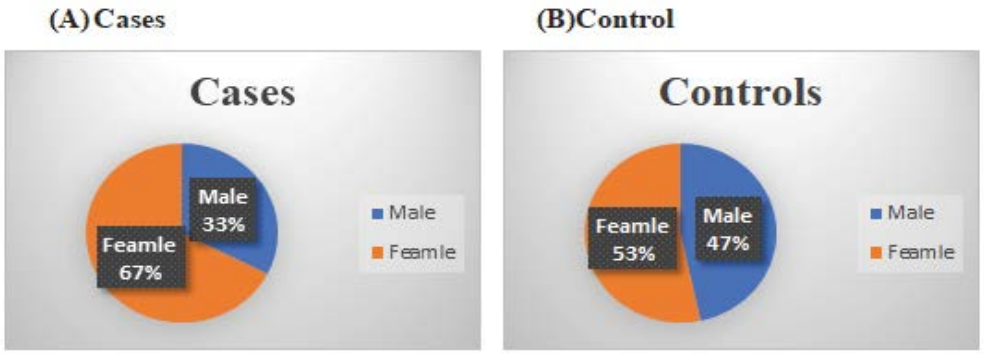

Figure 2 Incident of gender in both groups; A for cases and in this chart the incidence of female is 67%

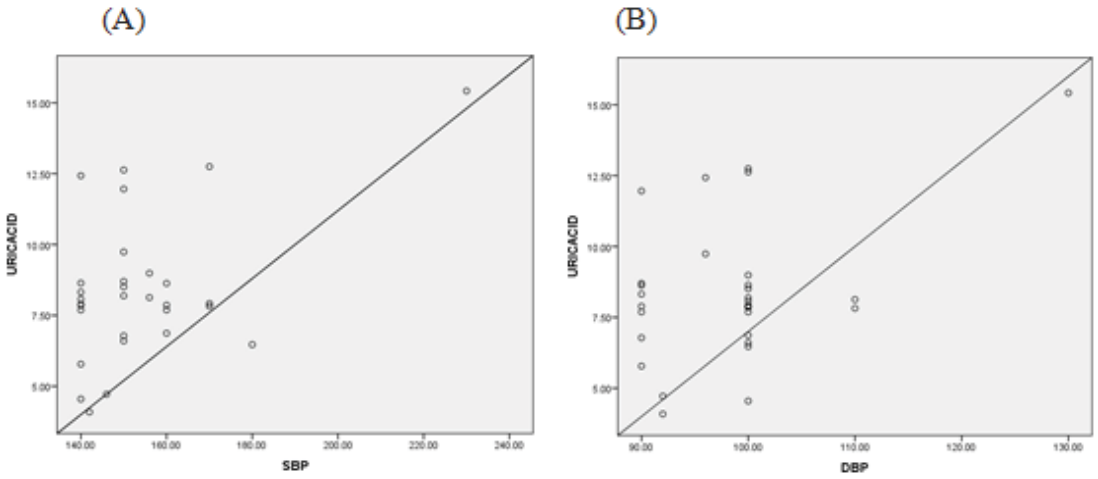

Figure 3 Scatter diagram showing correlation A (Uric acid vs. SBP) and B (Uric acid vs. DBP)

DISCUSSION

Discovered essentially hoisted levels of serum uric acid in both male cases and female cases as compared to their control gathering. Late epidemiological investigations have another conclusion that uric acid is a noteworthy and autonomous hazard factor for the improvement of the 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 using 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 in 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 a quiet executioner so it is hard to diagnose with very few symptoms but our study concludes that serum uric acid is elevated with systolic/diastolic blood pressure. We also found that the incidence of females in the case group is high.

DECLARATIONS

Conflict of Interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

REFERENCES

[1] Koda-Kimble, Mary Anne. "Koda-Kimble and Young's applied therapeutics: The clinical use of drugs." *Lippincott Williams & Wilkins*, 2012.

[2] "High blood pressure" Centers for Disease Control and Prevention. https://www.cdc.gov/bloodpressure/index.htm

[3] Heidenreich, Paul A., et al. "Forecasting the future of cardiovascular disease in the United States: A policy statement from the American Heart Association." *Circulation,* Vol. 123, No. 8, 2011, pp. 933-44.

[4] Mayo Clinic. "High blood pressure (hypertension)" Mayo Foundation for Medical Education and Research. https://www.mayoclinic.org/diseases-conditions/high-blood-pressure/diagnosis-treatment/drc-20373417

[5] "Hypertension" World Heart Federation. https://world-heart-federation.org/what-we-do/hypertension/

[6] Toratora, G. J., and B. Derickson. "Principles of anatomy and physiology. 11th edi." 2007.

[7] Kowalski, Robert E. "Take the Presssure Off Your Heart." New Holland Publishers, 2007.

[8] Piani, Federica, Arrigo FG Cicero, and Claudio Borghi. "Uric acid and hypertension: Prognostic role and guide for treatment." *Journal of Clinical Medicine,* Vol. 10, No. 3, 2021, p. 448.

[9] Harrison, Roger. "Structure and function of xanthine oxidoreductase: Where are we now?" *Free Radical Biology and Medicine,* Vol. 33, No. 6, 2002, pp. 774-97.

[10] Alvarez-Lario, Bonifacio, and Jesus Macarron-Vicente. "Uric acid and evolution." *Rheumatology,* Vol. 49, No. 11, 2010, pp. 2010-15.

[11] Mandal, Asim K., and David B. Mount. "The molecular physiology of uric acid homeostasis." *Annual Review of Physiology,* Vol. 77, 2015, pp. 323-45.

[12] Sanchez-Lozada, Laura G., et al. "Uric acid and hypertension: An update with recommendations." *American Journal of Hypertension,* Vol. 33, No. 7, 2020, pp. 583-94.

[13] Zhang, Junxia, et al. "TLR2 and TLR4 mediate an activation of adipose tissue renin-angiotensin system induced by uric acid." *Biochimie,* Vol. 162, 2019, pp. 125-33.

[14] Corry, Dalila B., et al. "Uric acid stimulates vascular smooth muscle cell proliferation and oxidative stress via the vascular renin-angiotensin system." *Journal of Hypertension,* Vol. 26, No. 2, 2008, pp. 269-75.

[15] Yu, Min-A., et al. "Oxidative stress with an activation of the renin-angiotensin system in human vascular endothelial cells as a novel mechanism of uric acid-induced endothelial dysfunction." *Journal of Hypertension,* Vol. 28, No. 6, 2010, pp. 1234-42.

[16] Milanesi, Samantha, et al. "Uric acid and angiotensin II additively promote inflammation and oxidative stress in human proximal tubule cells by activation of toll-like receptor 4." *Journal of Cellular Physiology,* Vol. 234, No. 7, 2019, pp. 10868-76.

[17] Konukoglu, Dildar, and Hafize Uzun. "Endothelial dysfunction and hypertension." *Hypertension: From Basic Research to Clinical Practice,* 2016, pp. 511-40.

[18] Kanellis, John, et al. "Uric acid stimulates monocyte chemoattractant protein-1 production in vascular smooth muscle cells via mitogen-activated protein kinase and cyclooxygenase-2." *Hypertension,* Vol. 41, No. 6, 2003, pp. 1287-93.

[19] Kirca, M., et al. "Uric acid stimulates proliferative pathways in vascular smooth muscle cells through the activation of p38 MAPK, p44/42 MAPK and PDGFRβ." *Journal of Receptors and Signal Transduction,* Vol. 37,

No. 2, 2017, pp. 167-73.

[20] Kang, Duk-Hee, et al. "Uric acid causes vascular smooth muscle cell proliferation by entering cells *via* a functional urate transporter." *American Journal of Nephrology,* Vol. 25, No. 5, 2005, pp. 425-33.

[21] Liu, Huagang, et al. "The role of the gut microbiota in coronary heart disease." *Current Atherosclerosis Reports,* Vol. 22, No. 12, 2020, pp. 1-12.

[22] Romi, Muhammad Mansyur, et al. "Uric acid causes kidney injury through inducing fibroblast expansion, Endothelin-1 expression, and inflammation." *BMC Nephrology,* Vol. 18, No. 1, 2017, pp. 1-8.

[23] Piani, Federica, and Richard J. Johnson. "Does gouty nephropathy exist, and is it more common than we think?" *Kidney International,* Vol. 99, No. 1, 2021, pp. 31-33.

[24] Khanna, Puja, et al. "Systemic urate deposition: An unrecognized complication of gout?" *Journal of Clinical Medicine,* Vol. 9, No. 10, 2020, p. 3204.

[25] Bengtsson, Calle, et al. "Hyperuricaemia and risk of cardiovascular disease and overall death: A 12-Year follow-up of participants in the population study of women in Gothenburg, Sweden." *Acta Medica Scandinavica,* Vol. 224, No. 6, 1988, pp. 549-55.

[26] Freedman, David S., et al. "Relation of serum uric acid to mortality and ischemic heart disease: The NHANES I Epidemiologic Follow-up Study." *American Journal of Epidemiology,* Vol. 141, No. 7, 1995, pp. 637-44.

[27] Johnson, Richard J., et al. "Is there a pathogenetic role for uric acid in hypertension and cardiovascular and renal disease?" *Hypertension,* Vol. 41, No. 6, 2003, pp. 1183-90.

[28] Hwu, Chii-Min, and Kuan-Hung Lin. "Uric acid and the development of hypertension." *Medical Science Monitor,* Vol. 16, No. 10, 2010, pp. RA224-30.