Section: General Medicine

Original Research Article

A CASE CONTROL STUDY ON THE ASSOCIATION OF SERUM URIC ACID IN NEW AND RECENT ONSET OF PRIMARY HYPERTENSION AMONG THE PATIENTS ATTENDING MEDICINE OUT PATIENT DEPARTMENT OF A DISTRICT HOSPITAL

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ABSTRACT

Background: Aim: To study the association between levels of serum uric acid and in the development & pathogenesis of primary hypertension.
Materials and Methods: The prospective study conducted among diagnosed new & recent onset of hypertensive patients attending department of General Medicine, Imambara District Hospital, Hooghly, West Bengal from October 2017 to October 2018 after considering inclusion and exclusion criteria. 100 patients of new & recent onset of hypertensive patients are selected from OPD in a random fashion.
Results: In the present study, elevated level of Serum UA is significantly linked with Prehypertension and essential hypertension after controlling various confounding factors. The present study showed that the number of hyperuricaemic individuals and mean Serum UA level were significantly higher in newly diagnosed cases of hypertension as compared to prehypertensive and normotensive control. Serum UA was found to be positively and significantly associated with SBP in newly diagnosed cases of hypertension. In the present study, we found patient with PreHT and EHT often exhibited hyperuricaemia as co morbidity even if they were not taking medication.
Conclusion: In the present study, we found patient with PreHT and EHT often exhibited hyperuricaemia as co morbidity even if they were not taking medication.
Keywords: Hypertension, Uric Acid, Hyperuricaemia, Morbidity, EHT.

INTRODUCTION

Hypertension is an increasing important medical and public health issue. Hypertension markedly increases the risk for myocardial infarction (MI), stroke, congestive heart failure (CHF), peripheral vascular disease (PVD) and end stage renal disease (ESRD). Approximately 30% of adults are still unaware of their hypertension, more than 40% of individuals are not on treatment and two thirds of hypertensive patients are not being controlled to blood pressure levels less than 140/90 mmHg.[1]
Hyperuricemia is also common among adults with prehypertension, especially when microalbuminuria is present. The observation that hyperuricemia precedes the development of hypertension indicates that it is not simply a result of hypertension parse.[2]
Uric acid is a purine metabolite that in most mammals it is degraded by the hepatic enzymes uricase to allantoin. However, mutations in the uricase gene occurred during primate development; with the consequence that human have relatively higher levels of serum uric acid. An elevation in serum uric acid has been associated with an increased risk for the developmentof hypertension,[3,4] and 25% to 50% of hypertensive individuals are hyperuricemic.
Hyperuricemia also confers increased risk for cardiovascular mortality, especially in women.[5,6]
Despite incredible improvement in health since 1950, cardiovascular disease (CVD) remains among the

leading causes of death worldwide. Hypertension is the most common risk factor for cardiovascular morbidity and mortality. This positive relationship between blood pressure and cardiovascular risk has been shown to exist not only in those with higher blood pressure but also in individuals with high-normal blood pressure/prehypertension.[7] The complications resulting from hypertension are associated with the damage or failure ofvarious organs, leading to significant morbidity and mortality.[8] chronic kidney diseases.

(CKD) is one of the major complications associated with hypertension. Over the past decades, it has become increasingly evident that the prognosis, especially in terms of mortality and cardiovascular events of hypertensive patients is strongly affected by renal involvement. Therefore, the early detection and prevention of progressive CKD may not only alleviate the future burden of end-stage renal disease but also reduce the cardiovascular outcomes associated with hypertension.[9]

Hypertension is the most common chronic disease in developing and developed countries. It is diagnosed in 20–50% of the adult population and 10–30% of adults suffer from resistant hypertension.[10] The number of affected patients continue to rise known nonpharmacological methods and drugs registered/approved for treatment of arterial hypertension often do not achieve their intended therapeutic targets. Inadequate blood pressure control leads to life threatening complications such as stroke, heart diseases, and CKD.[10,11] These dismaying situations and the profound health impact of hypertension necessitate research efforts on early prevention and control of hypertension.

Serum uric acid was first noted to be associated with increased blood pressure by Frederick Mohamed in the 1870s.[12] Although the link was rediscovered periodically over the years; generally it was dismissed as a surrogate marker for decreased renal function that led to increased uric acid and increased risk for hypertension and cardiovascular disease. Recently, however, several lines of evidence suggest that increased serum uric acid may be a significant modifiable risk factor. Increased serum uric acid is associated with increased risk for future hypertension in several large longitudinal clinical trials as well as an independent risk factor for poor cardiovascular prognosis.

Elevated serum uric acid levels have been associated with an increased risk for CVD. The potential mechanisms by which serum uric acid may directly affect cardiovascular risk include enhanced platelet aggregation and inflammatory activation of the endothelium. Uric acid is the end product of purine metabolism; Its transformation to allantoin is catalyzed by urate oxidase in most mammals and it is predominantly cleared by the kidneys. However, mutations in the uricase gene occurred during human evolution and the levels of serum uric acid in humans are higher than those in other mammals. Several studies have found that an elevated uric acid level is an independent risk factor for cardiovascular disease after controlling for the contribution of established risk factors by multivariate analyses. The lack of a mechanism by which uric acid can cause cardiovascular disease, coupled with the inconclusive clinical and epidemiological data has left the issue unsolved.

Aim & Objectives

Aim

To study the association between levels of serum uric acid and in the development & pathogenesis of primary hypertension.

Objectives

1. To find the association of hyperuricemia in recent onset of primary hypertensive patients.
2. To find the association of hyperuricemia in hypertensive patients with reference to gender, smoking and BMI.

MATERIALS AND METHODS

The study area will be conducted in outpatient department (OPD) of department of General Medicine, Imambara District Hospital, Hooghly, West Bengal.

Study Population

Case

Patients who are coming for screening of blood pressure in the OPD of department of General Medicine, Imambara District Hospital, Hooghly, West Bengal apart from recent onset hypertensive patients were selected for the study and they were studied for serum uric acid levels.

Control

Normotensive controls were selected for the study and evaluated for clinical and laboratory data.

Inclusion Criteria for The Cases:

1. Hypertensive patients, whom are of new-onset and recent onset (<1 yr).
2. Age group between 20 to 50 years.
3. Both sexes were included.
4. Stage 1 and stage 2 hypertension according to JNC-VII.[1]

Exclusion Criteria for The Cases:

1. Hypertensive heart disease as evidenced by LVH- ECG-voltage criteria.
2. Diabetes mellitus – Type1 and Type 2
3. Patients with CKD.
4. Hypertensive patients with known Cerebro vascular disease.
5. Hypertensive patients with CAD- Myocardial Ischemia or Infarction.
6. Patients with long term drug intake like steroids, Anti-Tuberculosis Treatment (ATT), diuretics, anti metabolite or chemotherapy drugs.
7. Patients in whom BMI>30.71
8. Pregnancy women

Inclusion Criteria for The Controls:

1. Normotensive volunteers.
2. Age group between 20-50 year.
3. Both sexes were included.

Exclusion Criteria for The Controls:
Patients unwilling to participate in the study.

Study Tools
A well designed proforma containing various parameters under study were used for data collection. The data were maintained on computer. Blood and urine sample collected for biochemical analysis of test.

Study Technique
Face to face interview to obtain informed consent from the participant patients. Then measure their biochemical parameters if they consented. History, physical examination laboratory reports, old hospital records were reviewed.

Study Variables
Age, gender treatment by antihypertensive biochemical parameters, serum urea, fasting blood sugar
(FBS), post prandial blood sugar (PPBS).

Study Design
This study is observational analytical case-control study.

Study Duration
One year, from October 2017 to October 2018.

Methodology
Study was carried out after obtaining the approval from institutional scientific committee and ethical committee at R.G.Kar Medical College & Hospital, Kolkata. The study population included was new & recent onset of hypertensive patients who presented to Imambara District Hospital, Hooghly, West Bengal.

The prospective study conducted among diagnosed new & recent onset of hypertensive patients attending department of General Medicine, Imambara District Hospital, Hooghly, West Bengal from October 2017 to October 2018 after considering inclusion and exclusion criteria. 100 patients of new & recent onset of hypertensive patients are selected from OPD in a random fashion.

Written and informed consent was obtained from all the participants prior to the study. Data collected by using structured schedule. A detailed history was taken and examination done as per the proforma. All patients in addition to hematological and routine urine work up done.

Defination of Study Subject
Patients with blood pressure greater than 140 mm Hg systolic and 90 mm Hg diastolic with no definable cause are said to have primary, essential or idiopathic hypertension during study period and within the study area are considered as study subject.1

Operational Definitions

Hypertension
Hypertension is defined as systolic blood pressure of ≥140mmHg or diastolic blood pressure
≥90 mmHg.[1]

Table 1: Blood pressure classification: By JNC VII,[1]

Normal	Systolic, mm Hg	Diastolic, mm Hg
Pre hypertension	120-139	or 80-89
Stage 1	140-159	or 90 – 99
Stage 2	≥160	or ≥100
Isolated systolic hypertension	>140	and < 90

Hyperuricemia
Hyperuricemia is defined as serum uric acid level more than > 7.0 mg/dl in Indian men and
> 6.0 mg/dl in Indian women.[2]
Body Mass Index

It is calculated by using the following formula = $\frac{\text{weight(kg)}}{\text{height(meter)2}}$

Results and Analysis
Statistical Analysis
Statistical Analysis was performed with help of Epi Info (TM) 3.5.3. EPI INFO which is a trademark of the Centers for Disease Control and Prevention (CDC). Test will be used to test the association of different study variables. T-test will used to compare the means. Significance level will set at p≤ 0.05.

Over All Analysis
The total number of subjects included in this study was 100. Of these 50 were study cases (Hypertensive new and recent onset) and another 50 were controls (Non – Hypertensive). Both the cases and controls selected were adjusted for age distribution, Sex, selected cardio vascular risk factors like BMI, smoking, family history. Many studies previously published had indicated that hyperuricemia is also associated with metabolic syndrome, as other conditions like CAD, stroke, preeclampsia, malignant hypertension and CKD. Thus, subjects selected for the study after excluding were: Cases–50; Controls–50.

RESULTS

Table 2: Distribution of Socio-demographic and clinical profile in cases and controls

	Cases	Controls
Total	50	50
Gender	M= 30; F= 20	M= 40; F= 10
Mean Age	37.34	37.21
BMI	20.96to30.00	20.59to29.38
Mean BMI	25.24	24.39

Blood Pressure		
Mean SBP (mmHg)	153.80	112.80
Mean DBP (mmHg)	92.08	73.16
Uric Acid(mg / dl)	3.0– 8.2	3.0– 7.2
Mean Uric Acid	5.375	3.870

Table 3: Distribution of Mean BMI in Two Groups

		Number	Mean	SD	Minimum	Maximum	Median	p-value
BMI	Case	50	25.2406	2.4664	20.9600	30.0000	25.2800	0.0463
	Control	50	24.3902	1.6717	21.0100	28.8800	24.5150	

In case, the mean BMI (mean± s.d.) of patients was 25.2406 ± 2.4664 kg/m2. In control, the mean BMI (mean± s.d.) of patients was 24.3902 ± 1.6717 kg/m2. Difference of mean BMI in two groups was statistically significant (p=0.0463).

Table 4: Distribution of Mean SBP in Two Groups

		Number	Mean	SD	Minimum	Maximum	Median	p-value
SB P	Case	50	153.8000	16.6464	70.0000	180.0000	150.0000	<0.0001
	Control	50	112.8000	4.5356	110.0000	120.0000	110.0000	

In case, the mean SBP (mean± s.d.) of patients was 153.8000 ± 16.6464 mmHg.
In control, the mean SBP (mean± s.d.) of patients was 112.8000 ± 4.5356 mmHg. Difference of mean SBP in two groups was statistically significant (p<0.0001).

Table 5: Distribution of Mean DBP in Two Groups

		Number	Mean	SD	Minimum	Maximum	Median	p-value
DBP	Case	50	92.0800	5.1104	80.0000	110.0000	90.0000	<0.0001
	Control	50	73.1600	4.5011	70.0000	82.0000	70.0000	

In case, the mean DBP (mean± s.d.) of patients was 92.0800 ± 5.1104 mmHg.
In control, the mean DBP (mean± s.d.) of patients was 73.1600 ± 4.5011 mmHg. Difference of mean DBP in two groups was statistically significant (p<0.0001).

Table 6: Distribution of Mean Smoking in Two Groups

Smoking	Case	Control	Total
No	38	32	70
Row%	54.3	45.7	100.0
Col%	76.0	64.0	70.0
Yes	12	18	30
Row%	40.0	60.0	100.0
Col%	24.0	36.0	30.0
Total	50	50	100
Row%	50.0	50.0	100.0
Col%	100.0	100.0	100.0

Chi-square value: 1.7143; p-value: 0.1904

In case, 12(24.0%) patients had smoking. In control, 18(36.0%) patients had smoking. Association of smoking in two groups was not statistically significant (p=0.1904).

Table 7: Distribution of Mean Uric Acid in Two Groups

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Uric Acid	Case	50	5.3500	1.2245	3.0000	8.2000	5.3000	<0.0001
	Control	50	3.8700	.6867	3.1000	6.2000	3.7000	

In case, the mean uric acid (mean± s.d.) of patients was 5.3500 ± 1.2245 mg/dl. In control, the mean uric acid (mean± s.d.) of patients was 3.8700 ± .6867 mg/dl. Difference of mean uric acid in two groups was statistically significant (p<0.0001).

Table 8: Elevation levels

Elevation	Case	Control	Total
No	39	48	87
Row%	44.8	55.2	100.0
Col%	78.0	96.0	87.0
Yes	11	2	13
Row%	84.6	15.4	100.0
Col%	22.0	4.0	13.0
Total	50	50	100
Row%	50.0	50.0	100.0
Col%	100.0	100.0	100.0

Chi-square value: 7.1618; p-value: 0.0074

In case, 11(22.0%) patients had elevation. In control, 2(4.0%) patients had elevation. Association of elevation in two groups was statistically significant (p=0.0074).

DISCUSSION

We found that in case, the mean age (mean± s.d.) of patients was 39.5600 ± 6.6337 years. In control, the mean age (mean± s.d.) of patients was 37.6200 ± 7.3175 years. Difference of mean age in two groups was not statistically significant (p=0.1680). Thus the age was matched in this study.

We found that in In cases, 7(14.0%) patients had 20-30 years of age, 18(36.0%) patients had 31-40 years of age and 25 (50.0%) patients had 41-50 years of age. Mean ± SD was 39.5600±6.6337. In controls, 13(26.0%) patients had 20-30 years of age, 19(38.0%) patients had 31-40 years of age and 18(36.0%) patients had 41-50 years of age. Mean ± SD was 37.6200±7.3175. Association of cases and control in relation to age was not statistically significant (p=0.2269).

Present study found that case, 20(40.0%) patients had female and 30(60.0%) patients had male. In control, 10(20.0%) patients had female and 40(80.0%) patients had male. Association of sex in two groups was not statistically significant (p=0.0290).

We found that case, the mean height (mean± s.d.) of patients was 164.9400 ± 7.2151 cm. In control, the mean height (mean± s.d.) of patients was 166.7200 ±7.6746 cm. Difference of mean height in two groups was not statistically significant (p=0.2350).

We found that case, the mean weight (mean± s.d.) of patients was 68.6000 ± 5.6677 kg. In control, the mean weight (mean± s.d.) of patients was 67.9800 ± 6.1195 kg. Difference of mean weight in two groups was not statistically significant (p=0.6003).

We found that in case, the mean BMI (mean± s.d.) of patients was 25.2406 ± 2.4664 kg/m2. In control, the mean BMI (mean± s.d.) of patients was 24.3902 ± 1.6717 kg/m2. Difference of mean BMI in two groups was statistically significant (p=0.0463).

We found that in case, the mean SBP (mean± s.d.) of patients was 153.8000 ± 16.6464 mmHg. In control, the mean SBP (mean± s.d.) of patients was 112.8000± 4.5356 mmHg. Difference of mean SBP in two groups was statistically significant (p<0.0001)

We found that in case, the mean DBP (mean± s.d.) of patients was 92.0800 ± 5.1104 mmHg. In control, the mean DBP (mean± s.d.) of patients was 73.1600 ± 4.5011 mmHg. Difference of mean DBP in two groups was statistically significant (p<0.0001). We found that in case, 12(24.0%) patients had smoking. In control, 18(36.0%) patients had smoking. Association of smoking in two groups was not statistically significant (p=0.1904).

In case, 33(66.0%) patients had family history. In control, 25(50.0%) patients had family history. Association of family history in two groups was not statistically significant (p=0.1050). In case, the mean uric acid (mean± s.d.) of patients was 5.3500 ± 1.2245 mg/dl. In control, the mean uric acid (mean± s.d.) of patients was 3.8700 ± .6867 mg/dl. Difference of mean uric acid in two groups was statistically significant (p<0.0001). n case, 11(22.0%) patients had elevation. In control, 2(4.0%) patients had elevation. Association of elevation in two groups was statistically significant (p=0.0074).

CONCLUSION

There is a future perspective that hypertension can be treated by lowering SUA levelsparticularly in new and recent onset primary hypertension. Thus it is important to monitor Serum UA level among prehypertensive and newly diagnosed essential hypertensive patients. Serum UA is strongly associated with BP in new and recent onset primary hypertension. Anelevated or high–normal serum uric acid value >5.5mg/dl (mean uric acid-5.7 in this study) in an adult being evaluated for hypertension strongly supports the presence of primary hypertension.The remarkable association of UA with BP in adults is consistent with recent animal model data and the hypothesis that UA might have a pathogenic role in the development of hypertension.

Limitations

The present study was a single center case control study done at a District Hospital located in Hooghly, West Bengal. This sample population may not representative of the community as a whole because of various strata like education, economic status environment, access to health care etc. The study was based on 100 patients due to limited study period. Thus to get a better result a study may be conducted based on considerably large number of patients. To draw any cause and effect relationship and generalize the results, prospective study with large number of cases is recommended.

Recommendation

Prevalence of hypertension is higher in younger patients of West Bengal so they need to be screen for blood pressure monitoring. This shows that blood pressure monitoring is preferred screening test. The above study has several limitations. The design of the study was case control and study population included only the outdoor hypertensive patients diagnosed new and recent onset of a single hospital. To draw any cause and effect relationship and generalize the results, prospective study with large number of cases is recommend.

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