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TO ESTIMATE SERUM URIC ACID IN ESSENTIAL HYPERTENSIVE MEN AT INDEX MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, INDORE

Biochemistry

Kapil Shrivastav Asst. Prof., Dept. of Biochemistry, Gajara Raja Medical College, Gwalior, M.P.

Ramesh Chandra Thanna* Dept. of Biochemistry, Amaltas Institute of Medical Science, Dewas, M.P. *Corresponding Author

ABSTRACT

Background: Hypertension is a major health burden and leading cause of death in the world. Although it is common in economically developed countries, than in developing countries, it is a greater population burden in the latter because of much larger population. Lower socio economics group have higher rate of hypertension, due to ineffective treatment.

Hypertriglyceridemia, raised LDL and raised VLDL are seen in Essential hypertensive. HDL is less in Essential hypertensive. Hyperuricemia is seen in essential hypertensive. Both dyslipidemia and hyperuricemia increase as age increase in essential hypertensive. In normotensives, few have hypertriglyceridemia. Though hypertriglyceridemia with increase as age increase, it is not associated hyperuricemia. This concludes that dyslipidemia is correlated to hyperuricemia as essential hypertensive in essential hypertensive and not in normotensives.

Study Design: Case Control Study

KEYWORDS

Serum Uric Acid & Hypertensive Men.

INTRODUCTION

Hypertension is a major health burden and leading cause of death in the world. Although it is common in economically developed countries, than in developing countries, it is a greater population burden in the latter because of much larger population. Lower socio economics group have higher rate of hypertension, due to ineffective treatment [1].

Hypertension is the third leading killer disease in the world and is responsible for 1 in every 8 deaths. About 1 billion people are affected by hypertension worldwide[2]. The prevalence of hypertension is known to increase with age. Over 50% of individuals aged 60 to 69 and over 75% of those aged 70 years and older are affected. Recent Framingham Heart Study reported that lifetime risk of developing hypertension is approximately 90% for men and women who are normotensive at 55-65 years old and survived to the age of 80-85 years[3].

Uric acid associated with metabolic syndrome to such an extent that it is been proposed as an active participant in the syndrome. Uric acid also correlates with triglyceride and HDL[4]. Uric acid is powerful risk marker seen in untreated hypertensive. It is independent predictor of mortality in patients with coronary heart disease [4-7].

AIMS AND OBJECTIVES

- To estimate serum uric Acid in essential hypertensive men.

MATERIAL AND METHODS

Place and Duration: the present study was undertaken in Place and Duration Index Medical College Hospital & Research Centre, Indore from outpatient department patient database from (September 2011 to Feb 2014).

INCLUSION CRITERIA:

Groups A consist of 30 healthy men aged between 35-65 yrs. Their BP was recorded<140/90.

EXCLUSION CRITERIA

Diabetes mellitus
Renal disease
History and presence of jaundice
Familial Hypelipidemia
Chronic liver disease
Patients on lipid lowering drugs Smoking
Alcoholics
Obese BMI <25
Gout
Female

Data Collection: all these subjects in Group A and Group B were subjected selection to medical examination and a fixed questionnaire. Physical examination include following anthropometric measurements. Height was measured by using a vertical board with attached metric scale. The individual was made to stand bare foot on a flat surface with weight evenly distributed on both the feet together and head positioned so that perpendicular to body. Head, Back, Buttocks & heels are in contact with vertical board. The headboard was brought in contact with the head to compress the hair the reading recorded to the nearest 0.1cm.

Weight: Weight was recorded by making the patient stand on a dial type weighing machine with body weight distributed between both feet.

1. Body Mass Index was calculated using Weight in kg. / (Height in meter)[8] -

Underweight: 18
Normal: 18-24.9
Grade 1 (over weight): 25-29.9
Grade II (obese): 30-39.9
Grade III (very obese): >40.0

2. Method of recording Blood pressure

Instrument used is sphygmomanometer. It is kept level of art and cuff tied around upper arm. Pressure is raised to 200 mmHg and gradually released. Variation of sound was heard with stethoscope placing its chest piece on brachial artery, a little below the cuff. The sounds are heard due to occurrence of turbulence in flow of blood through the narrow blood vessel, when the manometric pressure just coincides with systolic blood pressure. When pressure from cuff is released, normal stream line flow sets in & sound is no longer heard.

This manometric pressure coincides with diastolic Blood pressure. As pressure is released, various sounds are heard known as Korokoff sounds.

Phase I-sudden appearance of tapping sound persisting for 15mmHg. This indicates systolic pressure.

Phase II- murmur persists for another 15mm Hg.

Phase III- clear loud gong sound for 20mmHg.

Phase IV- muffled & fading sound. This indicate diastolic pressure BP is recorded 3 times at 30 min interval mean BP is taken.

Biochemical tests:

Morning sample blood was drawn after 12hr fasting. The samples of blood were allowed to stand to clot. Precaution were taken so that the blood did not hemolysed, serum was separated by centrifugation and analysis by the following methods.

1. Uric acid by phosphotungstic acid method-method of caraway

Principle:
The procedure is based on oxidation of uric acid by acid Phosphotungstic reagent in alkaline medium. Phosphotungstic acid itself gets reduced to tungsten blue. Sodium bi carbonate is used as alkali. The amount of tungsten blue formed is estimated at wavelength from (690-710onm) 660 nm.

Procedure:
Deproteinisation: Add while shaking 5.4ml of dilute tungstic acid to 0.6mi serum and centrifuge. Out of this take 3ml of supernatant and add 0.6ml of sodium carbonate and 06.ml of phosphotungistic acid. Mix and take OD after 30 min at 700nm or red filter. Similarly take 3ml of standard. Add 0.6ml of sodium carbonate & 0.6ml of phophotungstic acid. Mix & take OD after 30min at 700mm. Volume of test is 0.3

RESULTS & OBSERVATION

- Descriptive statistical analysis has been carried out in the present. Results on continuous measurements are presented on Mean + SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5% level of significance. Student t test (two tailed, independent) has been used to find the significance of study parameters between control and cases. Chi-square test and Fisher exact test has been used to find the significance of raised levels of lipid parameters and uric acid between controls and cases. Odds Ratio has been used to find the strength of relationship of raised lipid parameters in cases when compared to controls. Effect size has been used to find the effect when compared to controls.

- **Significant figures:** P value
- Suggestive significance 0.05<P<0.10
- Moderately Significant 0.1<P<0.05
- Strongly significant P<0.01

Table 1: Comparison of BP between two groups

BP	Control No.	%	Cases No.	%
Normal value (<140/90)	30	100	0	0.0
Stage 1(140150/90-99)	0	0	04	13.3
Stage2 (≥160/100)	0	0	26	86.7
Total	30	100	30	100
All controls have BP<140/90				

Among Cases 86.7% we Stage l hypertension. s 86.7% were in stage || hypertension and13.3%.

Study Design: A case-control study consisting of 30 controls and 30 cases is undertaken to study the relationship between serum uric acid and lipid parameters.

Fig 1: Comparison of BP between two groups

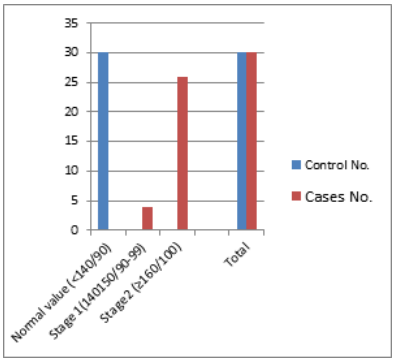

Table 2: Comparison of age in years between two groups

Age in Years.	Control No.	%	Cases No.	%
35-44	09	30	11	36.7
45-54	09	30	07	23.3
55-65	12	40	12	40.0
Total	30	100	30	100
All controls have BP<140/90	50.90±09.56		50.17±09.50	
Samples are age matched with p=0.767 30.0				

Fig 2: Comparison of age in years between two groups

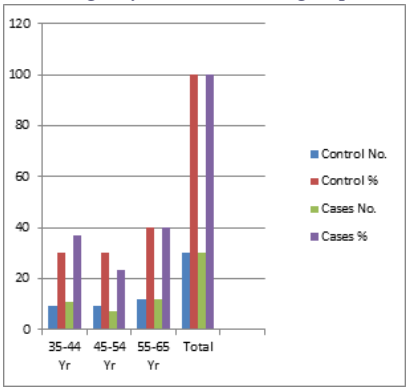

Table 3: Level of Blood Pressure according to age in cases and controls.

BP	Group	Age in years			Total
		35-44	45-54	55-65	
Normal value (<140/90)	Control	09	09	12	30
	Case	00	00	00	00
Stage 1(140150/90-99)	Control	00	00	00	00
	Case	04	04	00	04
Stage2 (≥160/100)	Control	00	00	00	00
	Case	07	07	12	26

As age increase BP also increase
Increase in predominantly seen in cases than in controls

DISCUSSION

Table 1 shows equal distribution of cases & control among all age groups.

Table 2 shows that all controls have BP < 140/90 and cases have > 140/90and, majority of them are in stage II, according to INC-7th report classification for hypertension.

Table 3 shows number of cases and control in different age groups. It shows that as age increase BP also increase and hypertensive are more in 55-65 yrs. of age group.

Hypertension is a degenerative process, taking place blood vessels affecting blood supply to target organs like heart, Kidney and Liver. Damage to these organs is called Target Organ Damage. These degenerative process increases Purine metabolism also, rising uric acid levels.

In hypertension, there is enhanced proximal tubular reabsorption and depressed tubular secretion of uric acid causing hyperuricemia. Diuretic treatment of hypertension will also cause hyperuricemia. Hyperuricemia is present in % cases of hypertension and increased in thiazide treatment.9&10

Uric acid is independent risk factor for atherosclerosis. Uric acid exertion is affected by kidney due to decreased renal perfusion in hypertension.

Hypertension complication like CCF, Heart failure has endothelial dysfunction due to dyslipidemia and raised uric acid. So in all hypertensive, dyslipidemia and serum uric acid is correlation at early stage will prevent complications of hypertension.

CONCLUSION:

Hypertriglyceridemia, raised LDL and raised VLDL are seen in Essential hypertensive. HDL is less in Essential hypertensive. Hyperuricemia is seen in essential hypertensive. Both dyslipidemia and hyperuricemia increase as age increase in essential hypertensive. In normotensives, few have hypertriglyceridemia. Though hypertriglyceridemia with increase as age increase, it is not associated hyperuricemia. This concludes that dyslipidemia is correlated to hyperuricemia as essential hypertensive in essential hypertensive and not in normotensives.

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