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Original Research Article

Evaluation of factors influencing hypertension among adults of South Karnataka - India

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ABSTRACT

Background: Studies have shown that BP is an independent risk factor for CVD. This relationship is independent as well as consistent and continuous. Observations involving more than 1 million individuals have shown that death from both CVD and stroke increases progressively and linearly from BP levels of as low as 115mm systolic and 75 mm diastolic upwards. The increased risks are present in all age groups ranging from 40 to 89 years old. Hence the present study was undertaken to evaluate the factors influencing hypertension among Adults.
Methods: In the present study 400 patients both out-patient and in-patients attending Adichuchanagiri Institute of Medical Sciences were evaluated for factors influencing Hypertension and other cardiovascular diseases. The 400 patients were divided into 200 cases who were again divided into categories based on severity of hypertension into stage 1 and stage 2 and duration of <5 years and ≥5 years.
Results: A positive correlation was found between the severity of hypertension, the patients who were found to be having stage 2 hypertension had an increase in SUA levels which was statistically significant when compared with those with stage 1 hypertension.
Conclusions: With the results based on the study carried out author concluded that there is direct relation between hyperuricemia and hypertension. Also, the study showed that the SUA levels were significantly increased in patients with Stage 2 hypertension as compared with those of stage 1 hypertension, showing that the severity of hypertension also related to the SUA levels.

Keywords: Blood pressure, Cardiovascular diseases, Hypertension, Hyperuricemia, Serum uric acid

INTRODUCTION

For every increment of 20 mm hg systolic or 10mm diastolic there was a doubling of mortality from both ischemic heart disease and stroke.[1] Evidence also warrants greater attention to the importance of SBP (Systolic Blood Pressure) as a major risk factor for CVD (Cardio- Vascular Diseases). The rise in SBP continues throughout life, in contrast to DBP (Diastolic Presssure), which rises until approximately 50 years age, tends to level off over the next decade, and may remain same or fall later in life. Clinical trials have demonstrated that control of isolated systolic hypertension reduces total mortality, CV(Cardio Vascular) mortality, and stroke and HF(Heart Failure) events.[2,3] The best operational definition for hypertension is "the level at which the benefits (minus the risks and costs) of action exceed the risks and costs (minus the benefits) of inaction".[4]

In contrast with the classification provided in the JNC(Joint National Committee) VI report, a new category designated prehypertension has been added and stages 2 and 3 have been combined.[5] Patients with prehypertension are at increased risk for progression to hypertension; those in the 130/80 to 139/89 mm hg BP range are at twice the risk to develop hypertension then

those with lower values.[6] Patients with arterial hypertension and no definable cause are said to have primary, essential, or idiopathic hypertension. Individuals in whom a specific structural organ or gene defect is responsible for hypertension are defined as having a secondary form of hypertension.[7] Essential hypertension is almost certainly a polygenic disorder, involving multiple genes, each having small effects on blood pressure.[8]

Both the rising SBP and falling DBP levels logically are associated with an increased risk for atherosclerotic vascular diseases. The resultant widening pulse pressures have been widely reported to be the best prognostic indicator of cardiovascular risk. However, an analysis of data from one million adults in 61 prospective studies found that, for predicting mortality from both stroke and coronary disease, the SBP is slightly more informative than DBP and that pulse pressure is much less informative.[9] Hence the present study was undertaken with the objective of evaluation of factors influencing hypertension among adults of south Karnataka.

METHODS

This was a hospital-based study carried among patients attending Adichuchanagiri Institute of Medical Sciences. 400 patients who attended the outpatient and in-patient at the department of Medicine were evaluated for factors contributing hypertension of which 200 were cases and 200 were controls. The study was conducted from April 2004 to September 2005.

Inclusion criteria

Adult male and female patients more than 18 years of age diagnosed as hypertensive according to JNC VII classification for hypertension.

Exclusion criteria

Patients were excluded if they had any of the following - Diabetes Mellitus, Ischaemic Heart Disease. All cases of secondary hypertension, Clinical Findings of gout or extra- articular manifestations of hyperuricemia, Obesity (body weight exceeding 25% of body weight), History alcohol abuse, Renal disease, pre-eclampsic toxemia. Controls were patients without hypertension who were matched for age and sex with that of the cases.

Ethical clearance was obtained from institutional ethical committee before the commencement of study and verbal informed consent was taken from all the study participants.

Information was obtained regarding socio demographic profile. The clinical examination consisted of a medical history, a physical examination, blood pressure measurement, anthropometric measurements, measurement of fasting serum uric acid levels and other parameters like Blood haemogram, Renal function tests (blood urea, serum creatinine), Electrocardiogram, Chest X-ray, Lipid profile (Total cholesterol, triglycerides, HDL- cholesterol, LDL- cholesterol), urine for protein and sugar. The patients were asked to fast for 12 hours and to avoid smoking and heavy physical Exercise for more than 2 hours before the examinations. After a 5 min rest in a quiet room, systolic and diastolic blood pressures were measured in the sitting position twice at an interval of a few minutes on the right arm with a standard mercury sphygmomanometer.[10] Anthropometric measurements included height and body weight, which were measured while the subject was wearing light clothing without shoes. The body mass index was calculated using the formula kg/m^2.

Hypertension was defined according to the JNC VII classification of hypertension, those with SBP 140-159 mm hg or DBP of 90-99 mm hg was labeled as having Stage 1 hypertension, and those with SBP ≥160 or DBP ≥100 mm hg were labeled as Stage 2 hypertension.

Statistical analysis

The data was entered in Microsoft excel worksheet later data was cleaned and transferred to SPSS version 20. Results are presented in univariate analysis using percentage and mean. Were as bivariate and multivariate analysis such as chi square test, t value and P value were used to check the association. P value less than 0.05 was taken as statistically significant.

RESULTS

During the 18 months study period from April 2004 to September 2005 a total of 400 patients were studied of which 200 patients were cases that were categorized into Stage 1 or Stage 2 hypertension (base on JNC VII classification) and 200 were controls who were patients without hypertension. The total number of male cases was 145 and the total no of female cases 55. The age group ranged from 20 years to 90 years. The total number of male controls was 145 and the total no of female controls were 55. The age group ranged from 20 years to 90 years. The controls were adjusted with the cases for age and sex, the 400 patients were divided into 200 cases who were again divided again into categories based on severity of hypertension into stage 1 and stage 2 and duration of <5 years and ≥5 years. The socio demographic factors influencing hypertension were increased age group (>50 years), male gender and familial history. The study showed a rise in SUA levels in hypertensive to highly significant p=0.004 when compared to that of normotensive. The incidence of hyperuricemia among hypertensives and normotensives were 37% and 17% respectively. A positive correlation was found between the severity of hypertension, the patients who were found to be having stage 2 hypertension had an increase in SUA levels which was highly significant p= 0.000 when compared with those

with stage 1 hypertension. The mean SUA in patients with stage 1 hypertension was 5.0312±0.77 and those with stage 2 hypertension was 6.4421±1.615.

Author also found a positive correlation between SUA levels and the duration of hypertension patients with a duration of hypertension ≥5 years had a significant increase in the SUA levels p=0.000 than those patients with hypertension for a duration of <5 years. As it has been shown that Hyperuricemia is observed in untreated hypertension may be due to a decrease in the renal blood flow and early nephrosclerosis, unexplained rise in SUA levels in Essential Hypertension can be used as a simple biochemical marker in determining the severity and duration of hypertension. SUA and risk for severity of hypertension:

The severity of hypertension was divided into stage 1 and stage 2 based on the JNC VII classification of hypertension. In the study done at this hospital the total number of patients assessed to have stage 1 hypertension was 48 patients (both male and female patients), the total number of patients having stage 2 hypertension was 152(both male and female patients). The data analysis for SUA levels in the stages of hypertension showed a mean serum uric acid level in stage 1 hypertension of 5.0312 with a standard deviation of ±0.77.

The mean serum uric acid levels in stage 2 hypertensive patient were 6.4421 with a standard deviation of 1.615. The t-value was 8.213 and a p-value of 0.000 which was significant. The data analysed showed that there was a significant rise in hypertension in patients who were having stage 2 hypertension that is those with an SBP ≥160 and a DBP ≥100 than those with stage 1 hypertension (SBP 140- 159 and DBP 90 - 99) (Table 1). The duration of hypertension was divided into 2 categories - those with hypertension for duration of hypertension <5 years and those with a duration of hypertension ≥5 years. The total number of patients with hypertension for duration of <5 years was 96, and the total number of patients with duration of hypertension ≥5 years was 104.

Table 1: The causes of death.

Stage of hypertension	Number	SUA (Mean± SD)	T value and p value
Stage 1	48	5.0312±0.77	t = 8.213, p = 0.000
Stage 2	152	6.4421±1.615	
<5 years	96	5.163±1.255	t- value = 9.891, p = 0.000
≥5 years	104	6.972±1.326	

The mean SUA level in patients with hypertension<5 years was 5.163 with a standard deviation of 1.255. The mean SUA level in patients with hypertension ≥5 years was 6.972 with a standard deviation of 1.326. The analyzed data showed a t-value of 9.891 and a p-value=0.000 which showed that there is significant increase in SUA levels in patients with hypertension ≥5 years than those with a duration of <5 years (Table 1).

DISCUSSION

Elevated serum uric acid is correlated with several risk factors including renal dysfunction, hypertension, insulin resistance, hyper-homocystenemia and hyperlipidemia, it is debated whether SUA is an independent cardiovascular risk factor. Goldstein and Manowitz showed in an adolescent population that, with age, weight, height and sexual maturity controlled, SUA significantly predicted blood pressure even in adolescents.[9] Three possible conclusions can be drawn from the association of hypertension with raised SUA levels. Hypertension may arise as a result of hyperuricemia, hypertension can cause hyperuricemia and the duration and severity of hypertension is related directly to the SUA levels. As to the possibility that Hypertension can cause hyperuricemia, it is thought that hyperuricemia can result from either overproduction of uric acid or from under excretion of uric acid. Overproduction of uric acid can be measured by the rate of incorporation of acid precursors such as Glycine labeled N 15, into the uric acid pool. Such a study carried out in 4 hypertensive patients with raised SUA levels did not show any overproduction of uric acid.

In the study of Breckenridge excretion of uric acid and uric acid clearance were lower in all hypertensive patients than in the normal group. When the uric acid clearance was expressed per 100ml of glomerular filtrate, there was no significant difference between normal subjects and hypertensive patients who had normal SUA levels, but the difference between those 2 groups and the hyperuricemic hypertensives was significant and they suggested a renal tubular abnormality in the handling of uric acid, the nature of the abnormality was not clear. Later Messerli et al, showed that hyperuricemia in hypertensive is due to early renal vascular involvement, namely, Nephrosclerosis. SUA rises because of impaired renal tubular function, which is the main site of regulation of SUA due to nephrosclerosis. Tykarski in his study showed that SUA levels in hypertensives are due to impaired tubular secretion of urate.

In the present study incidence and severity of hyperuricemia between cases and controls correlated significantly with the severity of hypertension. This correlated with both the Kinsey and Breckenridge studies, but according to Cannon et al, severity of hypertension had no relation to SUA level.[11] This study agrees with the study of Tykarski et al, in that there is a positive correlation between SUA and severity of hypertension.[12]

In this study the incidence of Hyperuricemia in cases with stage 1 hypertension was 4.2 % and those with stage 2 hypertension was 42.11 As to the possibility as to whether SUA levels was related to the severity and duration of hypertension, Breckenridge in his study

showed an increasing incidence of hyperuricemia as the diastolic BP increased in his study, but there was no tendency for hyperuricemia to occur, only with patients with more severe hypertension.

Kinskey also found that hyperuricemia was common in patients with more severe grades of hypertension. Comparison showed that SUA increased significantly with duration of hypertension in our study. This was similar to the finding of Tykarski et al, who encountered positive correlation between duration of hypertension and SUA in their study.

The PIUMA study demonstrates a strong independent association between SUA and CV risk in initially untreated and asymptomatic adult subjects with essential hypertension, but it is unable to answer the question of whether SUA exerts direct toxic effects. As extensively reviewed by Puig and Ruilope, both uric acid and superoxide radicals are produced for the effect of xanthine oxidase in the late phase of purine metabolism.[13] Superoxide radicals, which may cause tissue and vascular damage, are increased in subjects with essential hypertension It would be important to clarify whether such increase is due, at least in part, to enhanced xanthine oxidase activity and whether inhibition of this enzyme by allopurinol may reduce CV risk.[14-16]

In this study author found that there is definite relation in SUA levels between hypertensive patients and normotensive patients and there is a directly proportional relation in the levels of SUA in relation to the duration and severity of hypertension. Hence the possibility of serum uric acid acting by the production of free radicals and causing oxidative stress leading to hypertension and whether the duration and severity of hypertension lead to renal dysfunction in the form of nephrosclerosis leading to higher levels of serum uric acid has to be considered as various other studies have also show to have a positive relation in the SUA levels and hypertension.

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