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PRANAYAM-A POTENT SUPPRESSOR AGAINST INFLAMMATION IN HYPERTENSIVE SUBJECTS

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ABSTRACT

Total 100 hypertensive subjects and 100 sex matched controls were enrolled in this study. Inclusion criteria was patients with blood pressure 140/90 mm of Hg, while patients with secondary hypertension, stroke, CAD, MI and diabetes mellitus were excluded. Serum hs-CRP was estimated by **Latex enhanced immunoturbidimetric assay**. Serum Uric acid was estimated by **Uricase / POD end point assay**. We estimated hs-CRP and Uric acid in hypertensives and controls, then we suggested hypertensives for 3 months Pranayam, again the same parameters were estimated and compared by using **Students t test** and **one - way ANOVA** to determine significant differences.

Before Pranayam, hs-CRP was increased significantly (<0.001) in hypertensive subjects compared to controls. Uric acid was also increased significantly ($p<0.001$) in hypertensives compared to controls. But after 3 months Pranayam, there was significant ($p<0.001$) decrease in hs-CRP and insignificant ($p>0.05$) decrease in uric acid levels in hypertensives compared to hypertensives before Pranayam.

It was concluded that Pranayam can significantly suppress inflammation and can reduce inflammatory markers (hs-CRP & Uric acid) in hypertensives. There is positive co-relation between hs-CRP and uric acid in hypertensive subjects, which are all documented as proinflammatory markers for hypertension.

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KEY WORDS : C- Reactive Protein, Highly sensitive, Pranayam, Uric acid.

Introduction

Hypertension is a major cause of morbidity in developing countries which are in a state of epidemiological transition. Hypertension has been shown to be a major risk factor not only for cerebrovascular morbidity and mortality but also for cognitive impairment and dementia.

Hypertension is responsible for 57% of stroke death and 24% of coronary heart disease death in India⁹. Hypertension will be largest cause of death and disability in India by 2020. Hypertension is emerging as a major health problem. The prevalence of hypertension has increased in urban communities as well as in rural people.² Mechanical stresses

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Fig. 1: Different Mudras of Pranayam in hypertension

associated with hypertension exaggerate the process of atherosclerosis. There is no question that this atherosclerotic vascular disease involves multiple inflammatory cells and elevation in hs-CRP and Uric acid levels are markers of this inflammatory process. C-reactive protein (CRP) was originally discovered as a substance in the serum of patients with pneumonia and was named for its ability to bind and precipitate the C-polysaccharide of pneumococcus¹¹. CRP is a 224-residue pentameric protein with a monomer mass of 25106 Da. CRP is synthesized in the liver and is normally present as a trace constituent of serum or plasma at levels less than 0.3 mg/dl. CRP is one of the acute-phase proteins, the serum or plasma levels of which rise during general nonspecific responses to a wide variety of diseases. Several prospective studies have demonstrated a direct correlation between hypertension and rise in CRP. It is a useful indicator of inflammatory processes⁸. Uric acid, a product of purine metabolism, is degraded in most mammals by the hepatic enzyme, urate oxidase (uricase), to allantoin, which is freely excreted in the urine. Uric

acid is also commonly associated with hypertension. It is present in 25% of untreated hypertensive subjects, in 50% of subjects taking diuretics, and in >75% of subjects with malignant hypertension. This increase in serum uric acid in hypertension may be due to the decrease in renal blood flow that accompanies the hypertensive state, since a low renal blood flow will stimulate urate reabsorption. This study aims at understanding the role of Pranayam in reducing inflammation in hypertension.

Methods

The study was carried out in the Department of Biochemistry, Gandhi Medical College, Bhopal (M.P.) in collaboration with the Department of Medicine, with the approval of protocol by the Ethics Committee of the institution. After obtaining informed consent from the subjects a hospital based cross-sectional study was carried out. A total of 362 adults in the age range of 20-60 years were screened, residing in the urban and rural surrounding of Bhopal which had representative mix of subjects from different communities. Based

TABLE -1: Demographic characteristics of study population.

S.No.	Variables	Hypertensive(n=100)	Controls (n=100)
1	Age (in yrs)	54.10 \pm 8.16	32.96 \pm 6.12
2	Sex (M/F)	58/42	55/45



Fig. 2 : hS-CRP & uric acid levels in hypertensive subjects before and after pranayam as compared to control

on the criteria scheduled in the Seventh Report of the Joint National Committee on Prevention Detection, Evaluation and Treatment of High Blood Pressure (JNC-7), 136 subjects identified as hypertensives and 94 as normotensive controls. Sample size was estimated on the basis of reported prevalence of hypertension in M.P. which is equal to 38%. Out of 136 hypertensive and 94 normotensives subjects. 100 subjects from hypertensive group were selected for study who fulfilled inclusion criteria [e^v 140/90 mm of Hg (JNC-7)] and they were compared with 100 age and sex matched healthy normotensive controls. Biochemical parameters (hs-CRP and Uric acid) were analyzed before and after 3 months of Pranayam. 126 Patients with secondary

hypertension, past history of stroke, diabetes mellitus, MI, CAD and patients below and above 20 & 60 years were excluded. Serum hs-CRP was estimated by Latex enhanced immunoturbidimetric assay^{3,13}. Serum Uric acid was estimated by Uricase / POD end point assay¹⁰.

Statistical Analysis

The statistical analysis was done by using the Statistical Pack-age for Social Sciences (SPSS 17). The results were expressed as Mean±Standard Deviation (SD). The differences between the groups were analyzed by using the Student's "t"-test and one way ANOVA. The p value <0.001 was considered as highly significant, p value <0.01 as moderate significant while p value >0.05 as insignificant.

TABLE -2 : Hemodynamics of study population

Blood Pressure	Mean±SD			P VALUE		
	Control(A)	Before Pranayam(B)	After Pranayam(C)	A - B	A - C	B - C
Systolic	118.7+4.89	164.5+13.6	152.42+9.38	<0.001	<0.001	<0.001
Diastolic	77.67+5.94	94.20+6.11	88.49+1.42	<0.001	<0.001	<0.001

TABLE -3 : hs-CRP & Uric acid levels in hypertensive subjects before and after pranayam as compared to control

S. No.	Parameter	Mean±SD			P VALUE		
		Control (A)	Before Pranayam (B)	After Pranayam (C)	A - B	A - C	B - C
1	hs-CRP(mg/L)	1.086±0.76	3.39±1.10	2.24±0.92	<0.001	<0.001	<0.001
2	Uric acid (mg/dl)	4.9±1.04	6.12±1.13	6.01±1.1	<0.001	<0.001	NS

hs-CRP value was found highly significant ($p < 0.001$) whereas uric acid level was found insignificant ($p > 0.05$) in cases before and after Pranayam.

Results

Demographic characteristics of hypertensives and controls have been discussed which includes age & sex as shown by Table-1.

Haemodynamics of study population before and after Pranayam as shown by Table-2.

Before Pranayam, hs-CRP and Uric acid levels were significantly ($p < 0.001$) elevated in hypertensives compared to controls. But when hypertensives were suggested for 3 months Pranayam, there was significant ($p < 0.001$) decrease in hs-CRP and insignificant ($p > 0.05$) decrease in Uric acid levels when compared to cases before Pranayam as shown by Table-3. These results indicate that Pranayam plays a potent role in suppression of inflammation in hypertensive subjects. Pranayam Mudras (Yogic breathing exercises) in hypertension is shown by Figure 1. Graphical presentation shown by Figure 2.

Discussion and Conclusions

Hypertension or High Blood Pressure is fast becoming a disease of the modern age. People are more and more becoming susceptible to this disease¹. Some of the major causes of high blood pressure could be blockages in the arteries, hardening of the arteries, excessive stress, high levels of cholesterol and triglycerides, wrong eating habits and obesity. Several researchers have reported that Pranayam techniques are beneficial in treating a range of stress related disorders, improving autonomic functions, relieving symptoms of asthma and reducing signs of inflammation. Practitioners reported that the practice of Pranayam develops a steady mind, strong will-power, and sound judgment and also claimed that sustained Pranayam practice extends life and enhances

perception¹². CRP is one of the acute-phase proteins, the serum or plasma levels of which rise during general nonspecific responses to a wide variety of diseases. Several prospective studies have demonstrated a direct correlation between hypertension and rise in CRP. It is a useful indicator of inflammatory processes⁸. Although the detection of elevated levels of CRP in the serum is not specific for any particular disease, it is a useful indicator of inflammatory processes⁷. CRP levels rise in serum or plasma within 24 to 48 hours following acute tissue damage, reach a peak during the acute stage and decrease with the resolution of inflammation or trauma¹¹. The increased level of CRP in human serum or plasma may last for several days before decreasing to normal levels⁴. Our study correlates with above studies and showed that hs-CRP level was significantly increased in hypertensives compared to controls, But when they were suggested for Pranayam hs-CRP level decreased significantly in hypertensives compared to hypertensives before Pranayam.

We found increased uric acid levels in hypertensives compared to controls, after Pranayam it gradually decreased in hypertensives but difference was found insignificant when hypertensives after Pranayam compared with hypertensives before Pranayam. The increase in serum uric acid in hypertension may be due to the decrease in renal blood flow that accompanies the hypertensive state, since a low renal blood flow will stimulate urate reabsorption. Hypertension also results in micro vascular disease and this can lead to local tissue ischemia. In addition to the release of lactate that blocks urate secretion in the proximal tubule, ischemia also results in increased uric acid synthesis. With ischemia, ATP is degraded to

adenine and xanthine and there is also increased generation of xanthine oxidase. The increased availability of substrate (xanthine) and enzyme (xanthine oxidase) results in increased uric acid generation as well as oxidant (O₂[•]) formation. Other factors may also contribute that uric acid is associated with hypertension, including alcohol abuse, lead intoxication, obesity and insulin resistance, and diuretic use. A worker⁶ studied plasma uric acid level and risk for incident hypertension. According to their study, uric acid level and risk for incident hypertension was examined respectively among old men who participated in the health professions. In conclusion, no independent association between uric acid level and risk for incident hypertension was found among older men. Another worker⁵ studied independent impact of hyperuricemia on the future risk of hypertension. A novel rodent model and a recent randomized trial of hyperuricemic adolescents with hypertension suggest a pathogenetic role of uric acid in hypertension, but it remains unknown whether these findings apply to adult populations where the larger burden exists. In conclusion, our study has documented increased inflammatory markers (hs-CRP & Uric acid) in hypertensive

subjects and has pointed to the significance of Pranayam in controlling inflammation in hypertension by change in lifestyle. Changes in lifestyle may involve dietary intervention, increase in physical exercise for controlling blood pressure, avoidance of smoking and control of overweight and obesity. By proper education regarding meditation and regular yogic exercises in the form of Pranayam can make miracle in the life of hypertensive subjects and we can check harmful effects of high blood pressure in the beginning of disease *i.e.* in early stage of hypertension by reducing inflammation. Because **“Sound body keeps sound mind”**.

LIMITATIONS OF THE STUDY

Patients included in the present study attended O.P.D of Medicine department. This study was subjected to 100 hypertensive cases within 20-60 years of age. The laboratory of biochemistry was well equipped with semi autoanalyser, colorimeter and spectrophotometer. Hence, all investigation were carried out on auto analyser. Duration of Pranayam could be increased upto 6 months so that prolonged effects of Pranayam could be assessed. All the prehypertensives were excluded from our study.

References

1. ACHARYA, BALKRISHNA (2007) *Yog-In Synergy with Medical Science*. Haridwar : Divya Prakashan; 2007-6, pp. 65-71.
2. CHOBANIAN, A.V., BAKRIS, G.L., BLACK, H.R., CUSHMAN, W.C., GREEN, L.A. AND IZZO, J.L. J.R., *ET AL.* (2000) The Seventh Report of the Joint National Rodgers A, Lawes C, MacMahon S. Reducing the global burden of blood pressure related cardiovascular disease. *J Hypertens*; **18**:S3-6.
3. CLAUS D.R., OSMAND, A.P. AND GEWURZ, H. (1976) Radioimmunoassay of human C-reactive protein and levels in normal sera. *J. Lab. Clin Med.* **87** : 120-128.
4. DIXON, ARMOS AND M.C. CONKEY (1972) *Pharmacological methods in control of Inflammation*, pg. No. 109.
5. FETER, C. GRAYEON, SEO YOUNG, KIM, MICHAEL P.L. AND VALLEY, HYON K. CHOI (2010) Independent impact of Hyperuricemia on the future risk of hypertension: A systemic review and meta – analysis, <http://www.rheumatology.org>.
6. JOHN, P. FORMAN, HYON, CHYOI AND CARY, C. GURHAN (2007) Plasma uric acid level and risk for incident Hypertension among men. *Journal of the American society of Nephrology*, **18** (1) 287-292.
7. MORLEY, J.J. AND KUSHNER, I. (1982) Serum C-reactive protein events. *Annals of N.Y. Acad Sci.* **389** : 406-417.
8. PIETILA, K., HARMOINEN, A., HERMENS, W., SIMONS, M.L., VAN DE WERF AND VERSTRAETE, M., *ET AL.* (1993) Serum C reactive protein and infarct size in Myocardial infarct patients with a closed versus an open infarct related coronary artery after thrombolytic therapy. *Eur Heart J.* **14**(7): 915-9.

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9. R. (2004) Trends in hypertension epidemiology in India. *Journal of Human Hypertension*; **18**:73–78.
10. SCHULTZ, A.L. (1993) Non protein compounds in clinical chemistry, theory analysis and co-relation, Kaplan LA, Pesce AJ *et al.* 1230-1261.
11. SCHULTZ, D.R. AND ARNOLD P.I. (1990) “Properties of four acute phase proteins : C-reactive protein, serum amyloid A protein, glycoprotein and fibrinogen” *Seminars in arthritis and Rheumatism* **20** : 129-147.
12. SWAMI, RAMDEV (2005) Pranayama Rahasya. Haridwar : Divya Prakashan; **3**.
13. WASUNNA, A., WHITELAW, A., GALLIMORE, R. AND HAWKIN, P.N. (1990) Peps M.B.C.-reactive protein and bacterial infection in preterm infection in preterm infants *Eur J. Pediat* ; **149** : 424-427.