Oxidative Stress and Other Biochemical Parameters in Hypertension of Elderly Males

DRUTPAL SINGH BAGHEL¹, P.C. KOL² and BHASKAR REDDY³

¹Department of Biochemistry, ²Department of Pathology, ³Department of Anatomy, S.S. Medical College, Rewa, India.  
*Corresponding author E mail: drbaghel2011@gmail.com

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ABSTRACT

Hypertension is an epidemic in our societies and is the most common causes for cardiovascular diseases in the world. Prevalence of hypertension has increased by about 30 times among urban live in and by about 10 times among the rural in a relaxed and natural ways. Prevalence of hypertension rose with increasing age in every gender race group. The clinical materials for present study comprised 82 patients of male hypertensive patients admitted in G. M. and S. G. M. Hospital, S. S. Medical College, Rewa (M. P.), India. The age range was taken from 35 to 80 years. Oxidative stress can cause hypertension and hypertension can cause oxidative stress. Obtained data were analyzed statistically by using student “t” test. Extremely significantly (< 0.0001) decreased superoxide dismutase was found in male hypertensive patients (age 66 to 80 years).

Key words: Hypertension, oxidative stress.

INTRODUCTION

Hypertension is epidemic in our society and is the most common causes for cardiovascular disease in the world. The seventh report of the United State Joint National Committee for Detection, Evaluation, and Treatment of high blood pressure recommended a new classification system for hypertension¹. The Egyptian National Hypertension Survey reported on estimated prevalence of 26.3 percent (systolic blood pressure 140 mm Hg and diastolic blood pressure 90 mm Hg).²,³ India is a very large populous and typical developing country. Community surveys have documented that between three and six decade, prevalence of hypertension has increased by about 30 times among urban live and about 10 times among the rural in a relaxed and natural ways. The prevalence of hypertension has increased by about 30 times among the urban population over a period of 55 years and about 10 times among the rural population over a period of 36 years⁴.

The relationship between age and hypertension has been consistently demonstrated in cross-sectional survey as well as in longitudinal cohort studies conducted in western population⁵. Third National Health and Nutrition Examination survey (NHANES – III) which was conducted in a representative sample of 9901 non institutionalized United States civilians 18 years of age and older, the prevalence of hypertension raised with increasing age in every gender race group⁶. Men have a slightly higher prevalence of high blood pressure compared with women across all ethnic groups (24.7 versus 23.4 percent). Hypertension is more common in men than in women before menopause.⁷ The prevalence and incidence of hypertension also increase with age and the prevalence of isolated systolic hypertension raised from 5% at age 60 to greater than 10% at age 70 and 24% at age 80 year⁸,⁹.
MATERIALS AND METHODS

The clinical materials for present study comprised 82 male patients of hypertension admitted in G. M. and S. G. M. Hospital, S. S. Medical College, Rewa (M. P.), India. The age range was taken from 35 to 80 years. Blood samples were collected from the patients at the time of admission. Clinical investigations were performed in the Department of Biochemistry, S. S. Medical College, Rewa (M. P.), India. Serum glucose, protein (Total) and superoxide dismutase were estimated by GOD-POD\textsuperscript{10}, biuret\textsuperscript{11} and Mishra H P \textit{et al.,}\textsuperscript{12} methods respectively. Plasma malondialdehyde was estimated by Jean C D et al method (1983)\textsuperscript{13}. Serum electrolytes were estimated by electrolyte analyzer. Obtained data were analyzed statistically by using student “t” test.

RESULTS

1. Table: 1 showing comparison of male hypertensive patients between ages rang 35 – 50 and 51 – 65 years. We are observed, highly significant (p<0.001) increased biochemical values in the form of potassium, glucose, protein (Total), and malondialdehyde in age 51 to 65 years when compared to age 35 to 50 years of male hypertensive patients. Serum sodium was decreased highly significant (p<0.001). Significantly (< 0.05) increased

| Table 1: Significant test between age group (35 - 50 year) and (51 - 65 year) in male patients of hypertension |
|----------|---------------------------------|-----------------|-------|
| S. No.  | Parameters                     | Male hypertensive patients | t- test | P- value |
|         |                                 | 35 – 50 (22) years (19) | Mean ±SD | 51 – 65 (30) years (30) | Mean ±SD |       |
| 1       | Systolic blood pressure (mm Hg) | 145.91 ± 2.11    | 147.8 ± 3.66 | 2.169 | < 0.05 |
| 2       | Diastolic blood pressure (mm Hg)| 96 ± 2.47        | 97.4 ± 1.67 | 2.430 | < 0.05 |
| 3       | Serum Sodium ions (mEq / L)     | 130.98 ± 1.30    | 128.40 ± 2.04 | 5.201 | < 0.001 |
| 4       | Serum Potassium ions (mEq / L)  | 5.59 ± 0.21      | 6.02 ± 0.17 | 8.717 | < 0.001 |
| 5       | Serum Glucose (mg / dl)         | 116.42 ± 2.13    | 118.7 ± 2.02 | 3.930 | < 0.001 |
| 6       | S- Superoxide dismutase (EU / mg protein / ml) | 9.91 ± 0.26 | 9.71 ± 0.38 | 2.379 | < 0.05 |
| 7       | Plasma Malondialdehyde (nano mole / ml) | 5.20 ± 0.30 | 8.74 ± 0.45 | 3.008 | < 0.001 |

| Table 2: Significant test between age groups 51-65 and 66-80 years in male hypertensive patients |
|----------|---------------------------------|-----------------|-------|
| S. No.  | Parameters                     | Male hypertensive patients | t- test | P- value |
|         |                                 | 51-65 (19) years (19) | Mean ±SD | 66-80 (30) years (30) | Mean ±SD |       |
| 1       | Systolic blood pressure (mm Hg) | 146.8 ± 3.66    | 148.76 ± 3.92 | 2.002 | < 0.05 |
| 2       | Diastolic blood pressure (mm Hg)| 97.4 ± 1.67     | 99.47 ± 3.67 | 2.812 | < 0.001 |
| 3       | Serum Sodium ions (mEq / L)     | 128.40 ± 2.04   | 127.32 ± 2.09 | 2.025 | < 0.05 |
| 4       | Serum Potassium ions (mEq / L)  | 6.02 ± 0.17     | 6.50 ± 0.22 | 9.456 | < 0.001 |
| 5       | Serum Glucose (mg / dl)         | 118.7 ± 2.02    | 127.27 ± 4.17 | 10.131 | < 0.001 |
| 6       | S- Superoxide dismutase (EU / mg protein / ml) | 9.71 ± 0.38 | 9.08 ± 0.50 | 5.494 | < 0.0001 |
| 7       | Plasma Malondialdehyde (nano mole / ml) | 8.74 ± 0.45 | 8.96 ± 0.37 | 2.068 | < 0.05 |
systolic and diastolic blood pressure and decreased serum superoxide dismutase were found in age 51 – 65 years when compared to age 35 to 50 years of male hypertensive patients.

2. Table: 2 showing comparison of male hypertensive patients between ages range 51 – 65 and 66 – 80 years. Significantly (< 0.05) increased systolic blood pressure and plasma malondialdehyde and decreased sodium and protein (Total) were found in age 66 – 80 years of male hypertensive patients when compared to age 51 to 65 years of male hypertensive patients. Highly significant (p<0.001) increased diastolic blood pressure, potassium and glucose were found in male hypertensive patients (age 66 to 80 years). Extremely significantly (< 0.0001) decreased superoxide dismutase was found in male hypertensive patients (age 66 to 80 years).

DISCUSSION

Amongst several diseases, that affects the human these days, hypertension is considered the most dreaded. Age – related increase in hypertension prevalence have also been reported in numerous other national surveys conducted in different countries at various stages of economic development14. Emerging evidence indicated that hypertension is a vascular disease associated with inflammation, induced through redox-sensitive mechanisms that are regulated by angiotensin-II. High blood pressure is linked to vascular damage, oxidative stress and inflammation. Out of the many factors implicated in hypertensive vascular disease, angiotensin-II appears to be one of the most important15 and hyperglycemia also significant in hypertension16. Hypertension with hyperglycemia responsible for cardiovascular events. Angiotensin II has direct and indirect effects on insulin and its signaling pathways, providing support for the biologic mechanism underlying the benefits of renin–angiotensin system, which causes hyperglycemia17. Hypertension and electrolyte abnormalities are common in alcoholics18. Nicotine can be a potent stimulus to ADH release in humans, and this may have contributed to the development of hyponatremia. Its likely, therefore, that smoking was main reason of pathophysiology of the hyponatremic-hypertensive syndrome in many patients19. Some other studies have been found similar results20.

Hypertension occur de novo after 60 year of age. Although this increase and should give rise to the suspicion of secondary hypertension21. High Blood Pressure2 provides a new guideline for hypertension prevention and management; (1) In the persons older than 50 years, systolic blood pressure of more than 140 mm Hg is a much more important cardiovascular disease (CVD) risk factor than diastolic blood pressure; (2) The risk of CVD, beginning at 115 / 75 mm Hg, doubles with each increment of 20 / 10 mm Hg; individuals who are normotensive at 55 years of age have a 90% lifetime risk for developing hypertension22. Familial hyperkalemia and hypertension is an autosomal dominant disorder characterized by hyperkalemia, hypertension and low renin23. Abnormality in membrane Na⁺-K⁺-2Cl⁻ cotransport could be responsible for the hyperkalemia. Hyponatremic hypertensive syndrome is a rare condition of renovascular hypertension characterized by electrolyte abnormality and high rennin activity24. A marked increase in urinary protein excretion showed serum protein level was decreased25. The hyponatremic hypertensive syndrome is a rare but serious complication of renovascular disease. The syndrome is characterized by hypertension and profound natriuresis, leading to body sodium and water depletion26. These results have supported by some other workers27.

There are growing evidence that increased oxidative stress and associated oxidative damage are mediators of vascular injury in cardiovascular disease. Stress is intensified with the process of aging and in the elderly, this is accompanied by a more common accuracy of primary hypertension28. Malondialdehyde is the most abundant among the reactive aldehyde derived from lipid peroxidation. These aldehyde have been implicated as the causative agents in cytotoxic processes, and it is possible that aldehydes released from the cell membrane may diffuse, interact and induce oxidative modifications in other cells and in low-density lipid molecules, thereby increasing the risk of cardiovascular damage29. The upregulation of nicotinamide adenine dinucleotide phosphate (reduced NADPH) and the tubule interstitial
accumulation of active T cells, macrophage, and superoxide-production cells are partly responsible for oxidative stress in several models of hypertension\textsuperscript{30}. It can be concluded that oxidative stress can cause hypertension and vice versa. Superoxide dismutase and malondialdehyde markers are most important for evaluation of hypertension.

REFERENCES