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High Incidence of Hypertension Induced Epistaxis with Oxidative Stress in Older Female

Surendra Singh Moupachi	Department of Otorhinolaryngology, Shyam Shah Medical College, Rewa, Madhya Pradesh, India
P.C. Kol	Department of Pathology, Shyam Shah Medical College, Rewa, Madhya Pradesh, India
Adesh Patidar	Department of Pharmacology, Shyam Shah Medical College, Rewa, Madhya Pradesh, India
Drutpal Singh Baghel	Department of Biochemistry, Shyam Shah Medical College, Rewa, Madhya Pradesh, India

ABSTRACT

Essential hypertension can occur after middle age, although this is rise and should give rise to the suspicion of secondary hypertension. High blood pressure provides a new guideline for hypertension prevention and management. The reactive oxygen species has been proposed as a key mediator of the progression of essential hypertension. Biochemical parameters such as electrolytes, glucose, protein (T), creatinine, urea, superoxide dismutase (SOD), glutathione reductase (GSH-R), glutathione peroxidase (GSH-Px), catalase and plasma malondialdehyde (P-MDA) were varying significantly in female older hypertensive patients.

KEYWORDS Epistaxis, Hypertension, Oxidative stress

INTRODUCTION

Oxidative stress can causes hypertension and hypertension can cause oxidative stress. The upregulation of nicotinamide adenine dinucleotide phosphate (reduced form), oxidase and tubulointestitial accumulation of active T cells, macrophase and superoxide production cells are partly responsible for oxidative stress in several models of hypertension. Increasing evidence implicates reactive oxygen species in the pathogenesis of hypertension and its cardiovascular complication.[1] 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. Of the many factors implicated in hypertensive vascular disease, angiotensin II appears to be one of the most important.[2]

Essential hypertension can occur de novo after age of 60 year although this is rise and should give rise to the suspicion of secondary hypertension.[3] High blood pressure provides a new guideline for hypertension prevention and management; (1) In persons older than 50 years, systolic blood pressure 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 hypertension.[4]

Increased production of oxygen free radicals may play a role in much disease such as hypertension.[5] Among the defense system operating against the reactive oxygen species, superoxide dismutase, glutathione peroxidase and catalase are the most important antioxidant enzymes. The reactive oxygen species has been proposed as a key mediator of the progression of essential hypertension.[6]

Hypertension being the third commonest cause in this report shows epistaxis as evidence of poor blood pressure control. This collaborates with an earlier report also from Lagos in Nigeria of some patients who had epistaxis when their hypertension was not controlled due to ceasation of antihypertensive drug therapy.[7,8] The need for regular blood pressure check and compliance to antihypertensive medications must be emphasized. Varsney and Saxena in Dehradun India recorded hypertension as the second commonest cause of epistaxis after idiopathic causes while Chaiyasate et. al. reported hypertension to be the commonest cause of epistaxis followed by idiopathic causes in the Chiang Mai University Hospital Thailand.[7, 9]

It could be caused by a local disorder within the nose and paranasal sinuses or a result of a systemic disorder. Epistaxis could be unilateral or bilateral, mild, moderate, severe or torrential. Generally speaking, epistaxis could be as a result of congenital deficiencies in the clotting factor or vessel structure or acquired from trauma, infections etc. Although epistaxis may occur at any age or at any time and in any season, it is a common complaint in the pediatric age group and the winter months but has been shown to have bimodal age range presentation in reports from north America and Europe.[10–14]

The present studies were aimed to find out electrolyte imbalance, variation of biochemical parameters, antioxidant enzymes and oxidant product in older female patients of hypertension with epistaxis.

MATERIALS AND METHODS

The clinical material for present study comprised 79 older female patients with hypertension induced epistaxis, attended Otorhinolaryngology Department, G. M. Hospital, S. S. Medical College, Rewa (M.P.). Antihypertensive drugs administration effects were observed under supervision of Pharmacology Department, S. S. Medical College, Rewa (M.P.). The age range taken was from 20-80 years. Blood samples were collected from the patients at the time of admission. Clinical investigations were performed in the Clinical Biochemistry Section, Central Pathology Laboratory, Department Pathology, S.

S. Medical College, Rewa (M.P.) India. Serum glucose, protein (T), creatinine, urea, and superoxide dismutase were estimated by GOD-POD, biuret, jaffe's, diacetyl monoxime, and Misra H P et. al. methods respectively. Plasma malondialdehyde and haemolysate glutathione reductase, glutathione peroxidase, and catalase, were estimated by Jean C D et. al. method (1983), Horn H D (1963), Hafeman D G method (1974), and Asror K sinha method (1972) respectively. Serum electrolytes were estimated by electrolyte enalyzer. Obtained data were analyzed statistically by using student "t" test.

OBSERVATIONS

Table 1: Mean±SD value and significant test between age group (20–30 year) and age group (31–50 year) in female hypertensive patients

S. No.	Particulars	Mean±SD (20–30 Years) (n=19)	Mean±SD (31–50 Years) (n=30)	t-test	P-value
Electrolyte:					
1	Serum Sodium ions (mEq/L)	131.38 ± 2.85	127.97 ± 2.70	4.216	P<0.001
2	Serum Potassium ions (mEq/L)	5.88 ± 0.15	6.07 ± 0.27	2.799	P<0.0001
Biochemical Parameters:					
3	Serum Glucose (mg/dl)	115.61 ± 2.03	117.37 ± 3.35	2.059	P<0.05
4	Serum Protein (Total) (gm/dl)	6.58 ± 0.19	6.36 ± 0.17	4.217	P<0.001
5	Serum Creatinine (mg/dl)	0.72 ± 0.06	1.69 ± 0.19	21.511	P<0.001
6	Serum Urea (mg/dl)	36.45 ± 4.12	45.57 ± 1.38	11.227	P<0.001
Antioxidant / Oxidant product:					
7	Superoxide dismutase (EU/mg protein/ml)	9.76 ± 0.30	9.75 ± 0.22	0.134	P=0.894
8	Glutathione reductase (EU/gm protein)	17.94 ± 0.12	17.31 ± 0.12	17.906	P<0.001
9	Glutathione peroxidase (EU/mg % Hb)	7.45 ± 0.16	6.76 ± 0.15	15.291	P<0.001
10	Catalase (EU/mg protein/ml)	4.79 ± 0.13	4.17 ± 0.04	24.484	P<0.001
11	Plasma Malondialdehyde (nano mol/ml)	4.99 ± 0.17	8.66 ± 0.32	45.936	P<0.001
Blood pressure:					
12	Systolic blood pressure (mm Hg)	145.89 ± 1.94	149.2 ± 4.50	3.024	P<0.0001
13	Diastolic blood pressure (mm Hg)	99.37 ± 3.65	96.8 ± 1.34	3.517	P< 0.001

Table 2: Mean±SD value and significant test between age group (31–50 year) and age group (51–80 year) in female hypertensive patients

S. No.	Particulars	Mean±SD (31–50 years) (n=30)	Mean±SD (51–80 years) (n=30)	t-test	P-value
Electrolyte:					
1	Serum Sodium ions (mEq/L)	127.97 ± 2.70	125.38 ± 2.63	3.764	P<0.001
2	Serum Potassium ions (mEq/L)	6.07 ± 0.27	6.74 ± 0.14	12.066	P<0.001
Biochemical Parameters:					
3	Serum Glucose (mg/dl)	117.37 ± 3.35	126.23 ± 3.82	9.551	P<0.001
4	Serum Protein (Total) (gm/dl)	6.36 ± 0.17	6.10 ± 0.19	5.586	P<0.001
5	Serum Creatinine (mg/dl)	1.69 ± 0.19	2.01 ± 0.28	5.180	P<0.001
6	Serum Urea (mg/dl)	45.57 ± 1.38	50.44 ± 2.03	10.867	P<0.001
Antioxidant / Oxidant product:					
7	Superoxide dismutase (EU/mg protein/ml)	9.75 ± 0.22	9.43 ± 0.30	4.711	P<0.001
8	Glutathione reductase (EU/gm protein)	17.31 ± 0.12	16.59 ± 0.14	21.387	P<0.001
9	Glutathione peroxidase (EU/mg % Hb)	6.76 ± 0.15	6.26 ± 0.03	17.903	P<0.001
10	Catalase (EU/mg protein/ml)	4.17 ± 0.04	3.81 ± 0.06	27.344	P<0.001
11	Plasma Malondialdehyde (nano mol/ml)	8.66 ± 0.32	8.91 ± 0.32	3.026	P<0.0001
Blood pressure:					
12	Systolic blood pressure (mm Hg)	149.2 ± 4.50	148.93 ± 4.83	0.224	P=0.824
13	Diastolic blood pressure (mm Hg)	96.8 ± 1.34	97.2 ± 2.26	0.834	P=0.408

RESULTS

Table 1: Results of this table revealed that systolic blood pressure was increased significantly (P<0.001) in the age group of 30–50 years of female hypertension with epistaxis patients. Serum glucose was increased significantly (P<0.05) in the age group of 30–50 years of female hypertensive patients. Serum potassium ions, creatinine, urea and P-MDA were increased significantly (P<0.001) in the age group of 30–50 years of female hypertensive patients. Serum sodium ions, protein (T), GSH-R, GSH-Px and catalase were decreased significantly (P<0.001) in the age group of 30–50 years of female hypertensive patients.

Table 2: Results of this table revealed that serum potassium ions, glucose, creatinine, urea and P-MDA were increased significantly (P<0.001) in the age group 51–80 years of female hypertension with epistaxis patients. Serum sodium ions, protein (T), SOD, GSH-R, GSH-Px and catalase were decreased significantly (P<0.001) in the age group 51–80 years of female hypertensive patients.

DISCUSSION AND CONCLUSION

Systolic blood pressure and concentration of serum glucose, potassium ions, creatinine, urea, and plasma MDA are increased significantly in hypertensive patients, shows by some others (Table 1).[15–17] Potassium regulation is maintained by a system which affects the rate of renal excretion of the ions and its distribution between the intra-extra-cellular spaces. Long-term regulation is accomplished by the interactions of several component of the control system. The direct effect of changes in plasma potassium concentration on potassium secretion by the cells of the distal nephron is the most powerful regulator of potassium excretion.[18] Some other associates correlated complication of hypertension with hyperglycemia. [19] Similar decreased values of sodium ions, protein (T), GSH-R, GSH-Px, and catalase were found significantly (P<0.001) in female hypertensive patients by other associates.[20] In addition to otolaryngological and general physical examination, including recording of blood pressure.70.45% patients of hypertensive epistaxis were treated success by nonsurgical / non interventional approaches.[21] Bleeding from the nose and nasopharynx is a common symptom of diverse conditions which may present as mild recurrent bleeds or severe life threatening rhinological emergency and may pose a challenge to even a skilled otolaryngologist.[22]

Globally, the true incidence remained unknown, but it is estimated that 60% of the population will at least have an episode of epistaxis in their life time and 6% of them will seek medical attention.[21] Epistaxis is a reasonably common symptom encountered in our Otolaryngological experience. Epistaxis is essential problem of elderly population and that cardiovascular disorders apparently play a considerable role as a causative factor.[23–25] 8 Patients had epistaxis secondary to trauma and 1 patient due to blood dyscrasias also had hypertension. As expected, the female hypertension epistaxis pa-

tients (51–80 years) showed a significant (P<0.001) elevation in serum potassium ions, glucose, creatinine, urea, and P-MDA compared with that found in the same operated female hypertension epistaxis patients (30–50 years). Similar results have been explained by other workers (Table-2).[26–29] Hyperkalemia is 3 to 5 fold higher in more than 60 years of age.[30] Essential hypertension exhibit several red blood cell (RBC) ion transport abnormalities, insulin resistance which causes hyperglycemia. Extra cellular hyperosmolarity secondary to hyperglycemia causes a shift of water and potassium from the normal or elevated serum potassium concentration.[31] Prevalence of isolated systolic hypertension raised from 5% at age 60 to greater than 10% at age 70 and 24% at age 80 year.[32,19] Elevated serum creatinine levels are common in the community and are strongly associated with older age, treatment for hypertension.[33]

Similar reduced levels of sodium ions, protein (T), SOD, GSH-R, GSH-Px, and catalase were observed significantly (P<0.001) in female hypertensive patients by other workers.[26,28] The deficit in sodium ion transport across cell membranes (demonstrated in RBCs) in patients with essential hypertension or in normotensive subjects with a family history of hypertension; may alter the calcium / sodium ion exchange across vascular smooth muscle membrane.[34] Hyponatremia is seen in all age group, but there are important differences in elderly.[35] Hypertension in the elderly is associated with greater than normal levels of lipids oxidation and causes oxidative stress.[36] The concentrations of antioxidants such as superoxide dimutase, glutathione peroxidase, and glutathione reductase were fond to be decreased in patients with uncontrolled hypertension. These results suggest that an increased in free radicals generation in essential hypertension.[37] Reduced molecular synthesis or enzyme activity of superoxide dismutase, catalase, glutathione peroxidase in the rostral Ventrolateral medulla (RVLM), where sympathetic premotor neurons that generate tonic vasomotor tone are located, contribute to the pathogenesis of hypertension.[38] Hypertension caused, increases oxidation process which emphasized on importance and usefulness of antioxidants, because SOD is the first enzymatic antioxidant defense, its low level in hypertensive patients may be due to excess of oxidative stress.[39,40] The main novel conclusions of this study are that hypertension affects almost every metabolism of body as results of disturbed biochemicals. Hypertension ultimately leads to associated complication same way these condition results in to oxidative stress.

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