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TO STUDY THE BIOCHEMICAL PARAMETER IN HYPERTENSION AND RENAL FAILURE

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Abstract

Background: In both economically developing and non-developing countries, hypertension is a major public health issue. India has been reported to have lower hypertension prevalence rates. The blood pressure of city dwellers is higher than that of rural dwellers. India is a populous and typical developing country with a vast population. Renal failure is defined as the retention of nitrogenous waste products due to impairment of renal function. Acute renal failure and chronic renal failure are the two types of renal failure. Acute renal failure is described as kidney failure that occurs in a matter of days or weeks. Acute renal failure is most common in older persons and is linked with considerable mortality and morbidity, with death rates ranging from 25 to more than 70% among hospitalized patients.

Aim: To establish the above biochemical parameters for diagnosis and management of hypertension and renal failure.

Material and Method: The clinical materials for present study entitled "To Study the Biochemical Parameter in Hypertension and Renal Failure" comprised 52 male hypertensive patients. 39 female hypertensive patients, 50 male chronic renal failure patients, 43 female chronic renal failure patients, 42 male chronic renal failure with hypertension, 40 female chronic renal failure with hypertension, 36 male acute renal failure, 48 female acute renal failure were admitted in Hospital.

Results: Results revealed that highly significant decreased level of protein (Total), creatinine, and P-MDA were observed respectively. Serum sodium was found to be significant. Significant increased potassium was observed. Highly significant level of glucose, glutathione reductase, glutathione peroxidase, and catalase were observed respectively. Focus on acute renal failure patients were analyzed in both male and female. Highly significant decreased level of protein (Total), superoxide dismutase and P-MDA were observed respectively, glutathione reductase, glutathione peroxidase, and catalase were observed respectively. Significant increased serum glucose was observed, and highly significant increased level of glutathione reductase, glutathione peroxidase, and catalase were observed respectively.

Conclusion: The study's key finding is that oxidative stress causes hypertension and can signal the onset of chronic renal failure. Oxidative stress in older people became more severe, especially if they had renal difficulties, especially in middle age.
Keywords: P-MDA, Glutathione reductase, glutathione peroxidase, SOD, DBP

Introduction

Hypertension is a pandemic in our communities and is the most common cause of cardiovascular disease in the world. <80 mmHg blood pressure is normal systolic and diastolic blood pressure, and varies between 130-139 mmHg and 80-84 mmHg used for normal systolic and diastolic blood pressure respectively. World Health Organization 1978 recommendation for hypertension systolic pressure above 160 mmHg or diastolic pressure above 95 mmHg (or both) for arterial hypertension and systolic pressure 140 to 160 mmHg or diastolic pressure 90 to 95 mmHg or both at the borderline. high blood pressure.[1,2]

High blood pressure is an important public health challenge in both economically developed and underdeveloped countries. Between 15 and 20 percent of Brazilian urban adults suffer from high blood pressure.[3] The Egypt National Hypertension Survey reported an estimated 26.3 percent prevalence. Low levels of hypertension have been reported in India.[4] Urban populations have higher blood pressure levels than rural people. India is a very large and densely populated and developing country.[5,6] Social studies have shown that between the ages of thirteen and sixteen, high blood pressure increases 30 times among urban dwellers and 10 among rural areas in a relaxed and natural environment. [7]

The kidneys regulate and govern many of the body's functions, as well as sophisticated mechanisms that keep homeostasis in check. As many as two-thirds of critically ill and injured people acquire clinical or biochemical signs of renal failure. Renal failure is defined as the retention of nitrogenous waste products due to impairment of renal function. Acute renal failure and chronic renal failure are the two types of renal failure.[8,9]

Kidneys play a dual role in primary hypertension. On the one hand, it is the cause of high blood pressure, and on the other hand, it may suffer from the long-term effects of high blood pressure. High blood pressure is a common problem in

patients with chronic kidney failure and is considered one of the most important factors in determining the role of kidney failure, and high blood pressure itself accelerates kidney failure.[10] Systemic hypertension is often accompanied by chronic kidney failure. Sodium and the activity of the rennin-angiotensin-aldosterone system in patients with chronic kidney failure all cause high blood pressure. Hypertension is the result of increased fluid retention and stimulation of the rennin-angiotensin-aldosterone system. Hypertension can lead to the development of chronic renal failure. The seventh report of the National Joint Committee on Prevention, Diagnosis, Diagnosis, and Treatment of hypertension recommends targeted blood pressure below 130/80 mmHg in patients with chronic kidney failure. Smoking involves oxidative stress and is found to be a strong predictor of endothelial damage in patients with high blood pressure in patients with high blood pressure.[11,12]

As kidney function deteriorates, patients develop complications related to fluid over load, electrolyte imbalance, and the build-up of nitrogenous waste. Prevalence of hypertension, chronic renal failure, chronic renal failure with hypertension, and acute renal failure are significant for age, sex, smoking, alcohol addiction, electrolyte imbalance, biochemical abnormality, oxidative stress, and oxidant product respectively. 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 renal failure and renal system related complication. The present studies were aimed to find out electrolyte imbalance, variation of biochemical parameters, antioxidant enzymes and oxidant product in disease stage.

Material and Methods

The clinical materials for present study entitled "To Study the Biochemical Parameter in Hypertension and Renal Failure" comprised 52 male hypertensive patients. 39 female hypertensive patients, 50 male chronic renal failure patients, 43 female chronic renal failure patients, 42 male chronic renal failure with hypertension, 40 female chronic renal failure with hypertension, 36 male acute renal failure, 48 female acute renal failure were admitted in Hospital and 150 age and sex matched healthy individual control groups.

Age:

- The age ranges taken from 20 - 80 years for the hypertensive patients and for normal healthy controls
- The age range taken from 30 - 80 years for chronic renal failure, chronic renal failure with hypertension, acute renal failure and its healthy normal control groups

Inclusion criteria: inclusion criteria: Only hypertension, chronic renal failure, chronic renal failure with hypertension, acute renal failure subjects were considered without any ether complications.

Exclusion criteria: For Hypertension: Myocardial Infarction, cardiac arrest, stroke and diabetes mellitus etc. For Chronic Renal Failure: Diabetes mellitus, diabetic nephropathy, myocardial infarction, cardiovascular disease etc. For chronic renal failure with hypertension: Myocardial Infarction, cardiac arrest, stroke, diabetes mellitus etc.

Blood sample collection: 3 ml blood in plain vial. This blood used for separation of serum. Serum used for estimation of sodium ions, potassium ions, glucose, protein, creatinine, urea, and superoxide dismutase. 3 ml blood in 0.4 ml sodium citrate vial (3.8 mg % w/v). This blood used for separation of plasma and preparation of hemolysate. Plasma used for estimation of malondialdehyde. Hemolysate used for estimation of glutathione reductase, glutathione peroxidase, and catalase.

Separation of serum:

Blood samples were collected in simple vials and incubated for 45 minutes at 37°C. The clot was removed after incubation, and the remaining material was placed in a centrifuge test tube. This sample was centrifuged for 10 to 20 minutes at 3000rpm. The serum sodium ions, potassium ions, glucose, protein, creatinine, urea, and superoxide dismutase were all measured in the supernatant, which was collected in a clean and dry serum test tube.

Methods:

1. Serum sodium estimation by end-point kit method
2. Serum potassium estimation by end-point kit method
3. Serum glucose estimation by end point GOD-POD method
4. Serum protein (Total) estimation by biuret method (endpoint method)
5. Serum creatinine estimation by Jaffe's reaction method
6. Serum urea estimation by DAM (diacetyl monoxime) method
7. Serum superoxide dismutase estimation by Mishra H. P. and Fridovich I (1972)[15]
8. Glutathione Reductase (GSH- R) By Bergmeyer H. U. ed. Method in Enzymatic Analysis.[16]
9. Glutathione Peroxidase (GSH- Px) By Hafeman D.G. Method (1974)[17]
10. Catalase by Asror K. Sinha (Colorimetric Method)-1972[18]
11. Plasma Malondialdehyde (P-MDA) estimation by Jean C. D. etal (1983)[19]
12. Estimation of Haemoglobin (Sahli's method)

Statistical Analysis: The data was analyzed by using Statistical Package for the Social Sciences (SPSS) Version 16.0. Significance testing of difference for mean ± SD of three groups was done by Analysis of variance test (ANOVA).

Result:

Table 1: Studies of different parameters in subjects of different study groups

Particulars	Hypertensive Patients	Chronic Renal Failure	Acute Renal Failure	Chronic Renal Failure with Hypertension
Electrolyte				
Serum sodium	129.44±3.08	126.33±3.67	127.94±2.85	122.21±3.11
potassium	6.38±0.20	7.04±0.32	6.74±0.23	7.86±0.36
Biochemical Parameters				
Serum glucose	119.9±2.14	128.09±5.05	120.55±4.27	132.69±4.46
Serum protein (Total)	6.65±0.21	5.88±0.32	6.75±0.28	6.89±0.40
Serum creatinine	2.96±0.22	4.28±0.57	4.53±0.62	5.17±0.28
Serum urea	48.7±2.39	52±3.27	48.73±2.87	55.90±3.87
Antioxidant / Oxidant product				
Superoxide dismutase	10.75±0.41	10.42±0.50	10.88±0.17	9.86±0.25
Glutathione reductase	18.94±0.22	19.28±0.32	19.65±0.16	17.63±0.10
Glutathione peroxidase	8.11±0.16	6.90±0.18	9.5±0.07	8.9±0.07
Catalase	5.6±0.09	5.46±0.10	5.33±0.15	4.7±0.27
Malondialdehyde	10.78±0.47	12.83±0.56	11.5±0.74	12.88±0.69

Table 2: Studies of different parameters in subjects of different control groups

Particulars	Hypertensive Patients	Chronic Renal Failure	Acute Renal Failure	Chronic Renal Failure with Hypertension
Electrolyte:				
Serum sodium	138.72±2.19	141.39±2.33	143.64±2.53	146.73±2.84
Serum potassium	5.07±0.31	5.55±0.63	6.61±0.52	6.16±0.50
Biochemical Parameters:				
Serum glucose	80.45±5.73	90.66±3.18	101.22±4.72	107.55±5.44
Serum protein (Total)	6.86±0.36	7.20±0.35	7.58±0.45	7.88±0.61
Serum creatinine	0.91±0.11	0.95±0.13	0.98±0.16	1.04±0.09
Serum urea	25.41±3.07	28.82±3.93	37.46±5.20	40.24±5.11
Antioxidant / Oxidant product:				
Superoxide dismutase	13.38±1.94	14.59±1.15	13.65±1.80	15.78±1.51
Glutathione reductase	21.56±0.18	20.98±0.19	20.31±0.16	22.91±0.25
Glutathione peroxidase	12.08±0.63	10.88±0.17	10.28±0.10	13.77±0.49
Catalase	7.40±0.14	6.87±0.18	6.29±0.16	6.99±0.35
Malondialdehyde	3.96±0.52	4.51±0.50	4.71±0.30	4.86±0.69

Comparative study between hypertensive patients and healthy controls were revealed that hypertensions were found in patients highly significant decreased level of sodium, protein, glutathione reductase, glutathione peroxidase, catalase, and superoxide dismutase were observed respectively. Highly significant increased level of potassium, glucose, creatinine, urea, and malondialdehyde were observed respectively. All biochemical markers were analyzed in both male and female chronic renal failure associated with hypertension. Highly significant decreased level of sodium, protein, glutathione reductase, glutathione peroxidase, catalase, and superoxide dismutase were observed respectively. Highly significant increased level of potassium, glucose, creatinine, urea, and malondialdehyde were observed respectively. Acute renal failure was also investigated in both male and female patients. Highly significant decreased level of sodium, protein, glutathione reductase, glutathione peroxidase, catalase, and superoxide dismutase were observed respectively. Highly significant increased level of potassium, glucose, creatinine, urea, and malondialdehyde were observed respectively.

Discussion

Amongst several diseases, that affects the human these days, hypertension is considered the most dreaded. There is discussion about diseases condition in three ways i.e. comparative study between individual's healthy controls and disease.

Hollenberg N K and colleague (1984)[20] suggested that hypertension subject's exhibit increased renal vascular resistance. Hypertension in patients with renal illness may be caused in part to lower renal blood flow and glomerular filtration rate, according to **Muhlhauser I and coworkers (1996).**[21] Hypertension is a major risk factor for chronic kidney disease in and of itself, and it is almost always present in patients with renal failure. The most important mechanism involved in the rise of blood pressure in people with kidney illness has been thought to be sodium retention and activation of the rennin-angiotensin system. [22]

Hyponatremia, defined as blood sodium 130 mEq/L, was shown to be prevalent in 19.69 percent of all renal failure patients, according to **Thomas A Vurgese et al (2006).**[23] Hyperglycemia can lead to hyponatremia. Chronic renal failure is an oxidative state, and the degree of intracellular and extracellular oxidative stress is proportional to the severity of renal failure. Because the kidneys are unable to eliminate potassium due to a lower glomerular filtration rate, elevated potassium levels are frequently linked to chronic renal disease. **Metheny N, (2000)**[24]

Julian Sequra et al (2002)[25] have observed similar significant higher values. According to Peralta **Carmen A and colleagues (2006)**[26], the metabolic syndrome (hypertension, hyperglycemia, hyperlipidemia, and obesity) was linked to chronic kidney disease in a large population-based cross-sectional analysis (the National Health and Nutrition Evaluation Survey III). Hyperglycemic disorders, according to **Antonios H Tzamaloukas and associates (2007)**[27], disrupt the tonicity of body fluids, the distribution of body water between major body fluid compartments, and the external balance of body solute and water.

Therefore, from above study we are concluding that hypertension, chrome renal failure, chronic renal failure with hypertension, and acute renal failure are associated with increased serum potassium, creatinine, urea and plasma malondialdehyde and hyponatremia and decreased antioxidant enzymes are also associated with the disease.

Conclusion:

From the analysis of data in the study, the conclusion is the incidence of kidney disease noted as common as hypertension, because of the high population density and lack of adequate health care facilities, especially in Central India, and also long term treatment of kidney disease is very expensive. The main novel finding of this study is that oxidative stress leads to hypertension and can precede development of chronic renal failure. Oxidative stress in elderly patients intensified especially if the patients have associated with renal complications especially in middle age.

References:

1. World Health Organization. Arterial hypertension. WHO Tech Rep Ser. 1978; 1; 628.
2. Ibrahim MM, Rizk H, Appel L J, et al. Hypertension, prevalence, treatment, and control in Egypt; result from the Egyptian National Hypertension Project. Hypertension. 1995; 26(Pt 1) 886 - 890.
3. Ashour Z, et al. The Eygyption National Hypertension Project (NHP). Design and rational. The NHP investigation team. Hypertension. 1995; 26 (6 pt 1): 880 -885.
4. Reddy K S. Cardiovascular disease in India. World Health Statistics Quartely. 1993; 46 (2): 101 - 107.
5. Gopinath N, et al. A 5 year follows - up study of hypotension in renal community. Indian Heart Journal. 1995; 47:129- 133.
6. Gupta R, et al. Hypertension epidemiology in India: Meta-analysis of 50- year prevalence rate and blood pressure trends. Journal of Human Hypertension. 1996; 10 (7): 465 – 472
7. Kumar K V, Das U N. Are free radicals involved in the pathobiology of human essential hypertension? Free Radical Res Commun. 1993; 19 (1): 159 -166
8. Hoste E, demon G, Kersten A, et al. Clinical evaluation of the new RIFLE criteria for acute renal failure. Crit Care. 2004; 8: 81.
9. Jonthon Costa, Robert S, Crausman, Marc S Weinberg. Acute and chronic renal failure. J Am Podiatr Med Assoc. 2004; 94 (2): 168 - 176.
10. Wall S J. Relationship between proteinuria and progressive disease. Am J Kidney Dis. 2001; 37 (1): 13 -16
11. Campese V M. Neurogenic factors and hypertension in renal disease. Kidney Int Suppl. 2000; 70; 2 - S6
12. Arum V Chobanian, Geoge L Bakris, Hemy R Bloak, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. JAMA. 2003; 289: 2560 - 2571.
13. Martin Tepel. Oxidative stress: Does it play a role in the genesis of essential hypertension and hypertension of uraemia? Nephrol Dial Transplant. 2003; 18; 1439-1442.
14. Shipak MG, Fried L F, Crurrp C, et al. Elevations of inflammatory and procoagulant biomarkers in elderly person with renal insufficiency. Circulation. 2003; 107: 87-92.
15. Mishra H P, fridovich I. The role of superoxide anion in the autoxidation of epinephrine and simple assay for superoxide dismutase. J Bio Chem. 1972; 247: 3170-3175.

16. Bergmayer H V. 'Methods of Enzymatic Analysis', A. P., N. Y.1974: 1-196
17. Hafeman D G, Sunde R A, Hoekstra W G. Effect of selenium on erythrocyte and liver glutathione peroxidase in rat. J A/ufr.1974; 104: 580 – 587
18. Sinha K A. Colorimetric assay of catalase. Analytical Biochemistry. 1972; 47: 389- 394.
19. Jean C D, Maryse T, Marie J F. Plasma malondialdehyde levels during myocardial infarction. Clinica Chimica Acta. 1983; 129: 319 - 22.
20. Hollenberg N K, Sandor T. Vasomotion of renal blood flow in essential hypertension; Oscillation in xenon transit. Hypetension 1984; 6: 579
21. Muhlhauser i, Prange K, Sawieki P T, et al. Effects of dietary sodium on blood pressure in IDDM patients with nephropathy. Diabetologia. 1996; 39: 212.
22. Guyton A C, Gleman T G, Wicox C S. Quantitative analysis of the pathophysiology of hypertension. J Am Soc Nephrol. 1999; 11: 2248 – 2159
23. Thomos Abraham Vurgese, S B Radhakishan. Frequency and etiology of hyponatremia in adult hospitalized patients in medical ward of a general hospital in Kuwait. Kuwait Med J. 2006; 38 (3); 211 - 213.
24. Metheyny N. Fluid and electrolyte balance. 4th ed. Philadelphia, Pa: Lippincott Williams & Wilkins, 2000.
25. Julian Sequra, Carlos Campo, Luis M Ruilope. How relevant and frequent is the presence of mild renal insufficiency in essential hypertension? J Clin Hypertens. 2002; 4 (5): 332 - 336.
26. Perneger T V, Whelton P K, Puddey I B, Klag M J. Risk of end-stage renal disease associated with alcohol consumption. Am J Epidemiology. 1999; 150(12): 1275-1281.
27. Antonios H Tzamaloukas, Alan R Levinstone, Kanneth D Gardner Jr. Hyperglycemia in advanced renal failure: Sodium and water metabolism. Nephron. 1982; 31: 40-44