ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

Examine The Relationship Between Renal Failure And The Clinical Profile And Side Effects Of Essential Hypertension

Dr. Ashok Kumar[1]*, Dr. Shivani Bansal[2], Dr. Ravi Kumar[3]

1. Professor, Department of General Medicine, Santosh Medical College & Hospital, Santosh Deemed to be University, Ghaziabad.
2. Professor, Department of General Medicine, Santosh Medical College & Hospital, Santosh Deemed to be University, Ghaziabad.
3. Assistant Professor, Department of General Medicine, Santosh Medical College & Hospital, Santosh Deemed to be University, Ghaziabad.

***Dr. Ashok Kumar – Corresponding Author**

ABSTRACT

Background: Adults with hypertension have a systolic pressure greater than 140 mmHg and a diastolic pressure greater than 90 mmHg (1). Secondary hypertension is the correct term for hypertension brought on by (secondary to) established disease processes, such as kidney disease and arteriosclerosis of the renal arteries (2). It is not reasonable to refer to hypertension as primary or essential when it results from complex and poorly understood processes.

Objective: To investigate the relationship between renal impairment and the clinical characteristics and side effects of essential hypertension

Materials & Methods: Between May 2014 and April 2015, patients with a diagnosis of hypertension who visited the Santosh Medical College and Hospital in Ghaziabad's department of medicine were assessed using inclusion and exclusion criteria (May 2014 to April 2015). At the Santosh Medical College and Hospital in Ghaziabad, Uttar Pradesh, a case-control research employing 100 patients as cases and 25 patients as controls was conducted on renal failure in critical hypertension.

Results: 36 patients (36.0%) had hypertension with a duration of less than or equal to one year, 44 patients (44.0%) with a duration of one to five years, 3 patients (3.0%) with a duration of six to ten years, and 17 patients (17.0%) with a duration of eleven to twenty years.

Conclusion: Based on demographic variables, assessment methods, and tools, renal impairment frequency differs between groups. Essential hypertension is characterized by increased LDL, hypertriglyceridemia, and hypercholesterolemia. In order to facilitate better categorization of absolute cardiovascular and renal risk factors, hypertensive subjects should have a more thorough evaluation of their renal function, especially in patients with hypertension that has been present for more than five years. The prevalence of hyperuricemia increases with patient age, hypertension duration, and severity.

Keywords: diastolic pressure, plasma concentration, creatinine, Glomerular, filtration, gastrointestinal

INTRODUCTION

Adults with hypertension have a systolic pressure greater than 140 mmHg and a diastolic pressure greater than 90 mmHg (1). Secondary hypertension is the correct term for hypertension brought on by (secondary to) established disease processes, such as kidney disease and arteriosclerosis of the renal arteries (2). It is not reasonable to consider the type of

ISSN PRINT 2319 1775 Online 2320 7876

Research paper **© 2012 IJFANS. All Rights Reserved,** **UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022**

hypertension that results from complex and poorly understood processes to be main or important (2,3).

Small amounts are lost through the epidermis and digestive (GI) tract, but the kidneys excrete about 75% of non-protein nitrogen as urea (10). Urea measurements are commonly available and are now recognized as a reliable indicator of kidney function (8). (10). Enzymatic reference intervals range from 15 to 40 mg/dl (2.5 to 6.6 mmol/l) (4). (8).

Clinically, measurements of plasma creatinine concentration are frequently employed as a measure of kidney function. About 1-2% of the entire muscle creatine pool is transformed each day to creatinine through the spontaneous, nonenzymatic loss of water (2),(10). Creatinine is a waste product of muscle creatine. An unexpected decrease in GFR results in a rise in plasma creatinine concentration because it is released into the blood at a steady rate and because its excretion closely matches the glomerular filtration rate (GFR).

So, a straightforward assessment of blood creatinine levels can reveal whether the GFR is normal and reveal details about the condition of the kidneys (2).

Men's and women's reference ranges for serum creatinine, as determined by the Jaffe method, are 0.9 and 1.3 mg/dl (80 and 115 mol/l) and 0.6 and 1.1 mg/dl (53 and 97 mol/l), respectively (8).

Small amounts are lost through the epidermis and digestive (GI) tract, but the kidneys excrete about 75% of non-protein nitrogen as urea (10). Urea measurements are commonly available and are now recognized as a reliable indicator of kidney function (8). (10). Enzymatic reference intervals range from 15 to 40 mg/dl (2.5 to 6.6 mmol/l) (4). (8).

Clinically, measurements of plasma creatinine concentration are frequently employed as a measure of kidney function. About 1-2% of the entire muscle creatine pool is transformed each day to creatinine through the spontaneous, nonenzymatic loss of water (2),(10). Creatinine is a waste product of muscle creatine. The steady rate of release into the blood combined with the strong relationship between its excretion and glomerular filtration rate (GFR) make plasma creatinine content sensitive to aberrant GFR fluctuations (2),(11).

So, a straightforward assessment of blood creatinine levels can reveal whether the GFR is normal and reveal details about the condition of the kidneys (2).

Men's and women's reference ranges for serum creatinine, as determined by the Jaffe method, are 0.9 and 1.3 mg/dl (80 and 115 mol/l) and 0.6 and 1.1 mg/dl (53 and 97 mol/l), respectively (8). In India, adults have a 5- 15% incidence of hypertension, compared to 10-12% in the West. In 5–15% of patients, essential hypertension results in clinical proteinuria and a severe decline in renal function. The development of more sensitive techniques to measure urine albumin excretion (UAE) has shown that people with hypertension are more likely (by 25–100%) than the general population to have microalbuminuria. [9-11]

Target organ involvement in hypertension individuals has found to be connected with a multitude of cardiovascular, pulmonary, and neurological symptoms. Focused neurological impairments, dyspnea, chest pain, headaches, and vision loss are thought to be the most prevalent symptoms experienced by people who have acute target organ damage caused by hypertension (7). When a patient exhibits any of these symptoms in addition to a high blood pressure, the doctor should do a thorough evaluation to rule out target organ damage.

METHODS AND MATERIALS

Between May 2014 and April 2015, patients with a diagnosis of hypertension who visited the Santosh Medical College and Hospital in Ghaziabad's department of medicine were assessed using inclusion and exclusion criteria (May 2014 to April 2015). At the Santosh Medical

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

College and Hospital in Ghaziabad, Uttar Pradesh, a case-control research employing 100 patients as cases and 25 patients as controls was conducted on renal failure in critical hypertension.

Based on the blood pressure (BP) readings taken using a mercury sphygmomanometer in both arms, hypertension was determined to exist. Three recordings spaced a week apart were used for outpatient patients. With the patient lying down, back supported for five minutes, and arm supported at the level of the heart, the patient's blood pressure was measured. All readings above 140/90 mmHg were considered to be hypertensive, which was validated on numerous instances as previously described. For each patient, a thorough case record was created using a proforma that was particularly created. The following essential historical criteria were taken into account: the length of hypertension and its treatment; a history of smoking; symptoms relating to the nervous and cardiovascular systems that may have suggested harm to the target organ.

The SPSS application was used to analyze all of the parameters. The ANOVA test was used to compare all the variables. On independent samples, cases and controls were contrasted using the chi-square test and the t test, and the p value was determined. The mean Standard Mean Error is displayed for all data (SEM).

RESULTS

Table 1 : No. of male patients were 71 and female patients 29 in case group

VARIABLES	FEMALE (N= 29)		MALE (N= 71)		p- Value
	MEAN	SD	MEAN	SD	
Age	55.00	10.25	53.93	8.91	0.603
BMI	29.40	2.10	29.38	1.61	0.959
systolic BP	152.83	10.14	152.85	10.04	0.993
Diastolic BP	97.86	5.40	96.51	5.21	0.248
TC	217.41	54.69	216.52	55.35	0.942
TG	160.97	49.16	164.54	42.36	0.716
HDL	39.66	5.02	38.08	5.74	0.199
LDL	117.16	22.67	120.09	25.59	0.593
Uric Acid	7.84	3.00	7.05	2.51	0.181
Serum creatinine	1.16	0.40	1.08	0.44	0.399
Blood Urea	39.86	15.00	40.82	19.52	0.813
Seum K^+	4.22	0.52	4.28	0.66	0.663
Serum Na^+	139.47	5.95	139.87	4.21	0.705
Micro albuminuria	34.31	27.10	32.72	25.71	0.783

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

Table 2 : Gender Distribution of patients

SEX	CONTROL		CASE	
	No.	PERCENT	No.	PERCENT
MALE	16	64.0%	71	71.0%
FEMALE	9	36.0%	29	29.0%
TOTAL	**25**	**100.0%**	**100**	**100.0%**

DISCUSSION

A 46% incidence of hyperuricemia was found among hypertensives in Kinsey's research of 400 hypertensive patients. In his study of 46 hypertension individuals, Kolbe discovered that 26 of them had elevated SUA levels (56%). [11]

Breckenridge found that 90 of the 333 patients (or 27%) who were enrolled in the clinic at the time had hyperuricemia, and 274 of the 470 patients receiving antihypertensive medication (or 58%) had elevated SUA levels.

[15-18] It is likely that uric acid causes reduced renal blood flow earlier and more sensitively than serum creatinine. It has recently been proposed that the higher risk of hypertension in individuals with raised blood uric acid levels may be related to this increased creation of free radicals, as uric acid may play a role in the formation of free radicals and oxidative stress. In our study, 21% of participants had microalbuminuria (21 out of 100 patients). 40 (26.6%) of the 150 cases of essential hypertension investigated by Hitha B et al in 2008 had microalbuminuria.

Worldwide, hypertension is a serious issue for public health. In India, the prevalence of hypertension ranges from 5 to 15% compared to 10 to 12% in the West. A degenerative process called hypertension affects the blood flow to organs like the heart, kidneys, and liver. These organs' damage is referred to as "target organ damage." The goal of the current investigation is to determine whether renal impairment is common in people with essential hypertension and to determine whether it is related to the clinical profile and complications of essential hypertension. [19-21]

Microalbuminuria was present in a significantly reduced percentage of those in the treated group. Although many hypertensive patients are receiving medication therapy, appropriate control was not always attained in many of them. This observation may be caused by the bias in classification of hypertensive patients into treated and untreated groups based on the treatment history. [22] In this investigation, total cholesterol was observed to increase in 24 (24%) patients over the normal value and LDL in 26 (26%) patients above the normal value. Serum concentrations of TC, TG, and LDL were also considerably higher in hypertension patients than in normotensive subjects. Untreated hypertensives are more likely than normotensives to have dyslipidemia, and lipid levels rise as blood pressure rises. Although no clear pattern of dyslipidemia has been observed in hypertensive people, numerous studies have revealed that aberrant levels of total cholesterol (TC), triglycerides (TG), and nearly all lipoprotein fractions are more common in people with hypertension than in the general population. It is well established that anomalies in blood lipid and lipoprotein levels result from changes in lipid metabolism, which are linked to hypertension. Additionally, it is known that hyperlipidaemia significantly affects the prognosis for hypertension individuals.

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

Patients with low HDL cholesterol and high TG levels have more widespread coronary atheromas than those with an isolated elevation of LDL cholesterol, according to research by Hausmann et al. using intravascular ultrasonography. Additionally, Halperin et al. has demonstrated that dyslipidemia in those who appear to be in good health causes hypertension. [23-25] Lepira et al. and Kesteloot et al. observed that although newly diagnosed hypertension patients tended to have a higher level of LDL, TG, and TC, their TC, TG, and LDL-C did not substantially differ from those of control participants. As a result, the study's findings indicate a significant link between renal function testing and essential hypertension.

CONCLUSION

Depending on demographic traits, examination methods, and tools, the prevalence of renal impairment varies amongst populations. Essential hypertension is characterized by elevated LDL, hypertriglycemia, and hypercholesterolemia. Age of the patients and the length and severity of hypertension all raise the prevalence of hyperuricemia. To enable better categorization of absolute cardiovascular and renal risk factors, hypertensive persons should undergo more thorough screening for renal function, especially in those with hypertension that has been present for longer than five years. Renal function testing is a useful method for identifying a subgroup of patients with greater renal problems when compared to people with mild hypertension in patients with moderate to severe hypertension.

REFERENCES

1. World Health Report 2002: Reducing risks, promoting healthy life. Geneva, Switzerland: World Health Organization, 2002

2. Norman M Kaplan, Braunwald's textbook of Cardiovascular Medicine 7th edition, "Systemic Hypertension: Mechanism and Diagnosis"; Elsevier Saunders 37:962.

3. Burt VL, Whelton P, Roccella EJ, Brown C, Cutler JA, Higgins M, et al. Prevalence of hypertension in the US adult population. Results from the Third National Health and Nutrition Examination Survey, 1988-1991. Hypertension.2005; 25: 305-313.

4. Bechung, Ooi Boon Seng, Chen, B.T.M. Toh, C.C.S. and KHOO, Causes Of hypertension in the young. Brit Med Jr, II, 744, 1970.

5. API Textbook of Medicine, 7th edition page, vol I, 531-534.

6. Aram V. C., George L. B., Henry R. B., William C.C., Lee A.G., Joseph L.I.et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation& Treatment of High Blood Pressure; JAMA: 2003; 289:2560- 2571.

7. David J.K., JacobW.U., RichardA.H., DavidA.W., Maged.S.B., Robert M.M. Lack of relationship between hypertension associated symptoms and blood pressure in hypertensive emergency department patients. American journal of emergency Medicine: 2005; (23)2: 106-110. .

8. Nelson's Textbook of Paediatrics, 18th edition, vol II, page no.1989-1995.

9. Kaplan N: Systemic hypertension: mechanisms and diagnosis, in Brauwald's Heart Disease, 7th ed, D Zipes et al (eds). Philadelphia, Saunders, 2005

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

10. Carl Erik Mogenson. Clinician's manual on microalbuminuria; Science Press Inc.2002

11. The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation& Treatment of High Blood Pressure; ArchIntern Med. 1997;157(21):2413-2446.

12. Hurst's Textbook of Cardiology. 12th edition, Page no. 1551-1560.

13.Prospective Studies Collaboration: "Age specific relevance of usual blood pressure to vascular mortality: A Meta-analysis of individual data for 1 million adults in prospective studies" Lancet 2002, 360; 1903.

14. Wilson PWF, Sullivan L, et al "Overweight and obesity as determinants of cardiovascular risk, The Framingham experience" Archives of Internal Medicine 2002; 162: 1867.

15. Desouza CA, Shaprio LF et al "Regular aerobic Exercise prevents and restores agerelated decline in endothelium dependent vasodilation in healthy men" Circulation 2000; 102: 1351.

16. Kaplan NM "Anxiety induced hyperventilation: A common cause of symptoms in patients with hypertension" Archives of Internal Medicine 1997: 157; 945.

17. Naomi DL Fischer, Gordon H. Williams " Hypertensive Vascular Diseases", Harrison's Textbook of Internal Medicine 16th edition,Vol.2; Mc-Graw Hill 1468- 1469.

18. Norman M Kaplan, Braunwald's textbook of Cardiovascular Medicine 7th edition "Systemic Hypertension: Mechanism and Diagnosis." Elsevier Saunders 37:973.

19. Haider AW, Larson MG, Franklin SS, and Levy D: "Systolic blood pressure, Diastolic blood pressure, and pulse pressure as predictors of risk for congestive heart failure in the Framingham Heart Study". Annuls of Internal Medicine 2003: 138; 10.

20. Boon D, Piek JJ, Van Montfrans GA: "Silent ischaemia and Hypertension" Journal of hypertension 2000:18; 1355.

21. Malhotra A., White DP "Obstructive Sleep apnea" Lancet 2002; 360: 237.

22. Schunkert H, Koenig W.,et al "Hematocrit profoundly affects left ventricular diastolic filling assessed by Doppler echocardiography" Journal of Hypertension 2000:18:1483-1484.

23. Ferrier KE, Bagnet JP, et al "Intensive cholesterol reduction lowers blood pressure and large artery stiffness in isolated systolic hypertension" Journal of American college of cardiology 2002:39;1020.

24.Kozokova M, de Simone C, Morizzo, Palombo C: "Coronary vasodilator capacity and hypertension induced increase in left ventricular mass" Hypertension 2003: 41; 224.

25. Shepard RJ, Zacharia PK, Schub C: "Hypertension and Left Ventricular diastolic dysfunction" Mayo Clinic Proc1989; 64:1521.