

Study and Classify of Renal Dysfunction in Patients with Essential Hypertension

Dr. Shivani Bansal^{1*}, Dr. Ashok Kumar², Dr. Ranjum Chaudhary³

^{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.

Corresponding Author: ^{1*}Dr. Shivani Bansal

ABSTRACT

Background: The adverse effects of hypertension principally involve the blood vessels, the central nervous system, the retina, the heart and the kidney, and can often be detected by simple clinical means(4). The hypertension is the most important modifiable risk factor for coronary heart disease, stroke, congestive heart failure, peripheral vascular disease and end-stage renal diseases.

Aims & Objectives: To study and classify of Renal dysfunction in patients with essential hypertension

Methods & Materials: The patients who visited to Medicine Department, Santosh Medical College and Hospital, Ghaziabad, with a diagnosis of Hypertension were studied considering the inclusion and exclusion criteria during a period starting from May 2014 to April 2015 from one year (May 2014 to April 2015). A Case Control study with 100 patients taken as case and 25 patients as control, were studied for the renal dysfunction in essential hypertension was undertaken at Santosh Medical College and Hospital, Ghaziabad, Uttar Pradesh.

Results: 36 patients (36.0%) had hypertension less than or equal to 1 year, 44 patients (44.0%) had hypertension between 1-5 years, 3 patients (3.0%) had hypertension between 6-11 years, and 17 patients (17.0%) had hypertension between 11-22 years.

Conclusion: The prevalence of renal dysfunction varies in different population, based on the characteristics of the population and techniques and protocols used for evaluation. Microalbuminuria reflects a state of increased renal endothelial permeability and hence considered an early marker of diffuse endothelial dysfunction. Hypercholesterolemia, hypertriglyceridemia, increased LDL all are seen in essential hypertension

Keywords: Hypertension, arteriosclerosis, renal arteries, adenosin, guanosine, purine nucleosides

1. INTRODUCTION

Hypertension in adults is defined by systolic pressure greater than 140 mmHg and diastolic pressure greater than 90 mmHg(1). Hypertension that is a result of (secondary to) known disease processes as a disease of the kidneys and arteriosclerosis of the renal arteries, is

logically called secondary hypertension because of high blood volume (2). Hypertension that is the result of complex and poorly understood processes is not so logically called primary or essential hypertension (2),(3).

The adverse effects of hypertension principally involve the blood vessels, the central nervous system, the retina, the heart and the kidney, and can often be detected by simple clinical means(4). The hypertension is the most important modifiable risk factor for coronary heart disease, stroke, congestive heart failure, peripheral vascular disease and end-stage renal disease (5),(6).

The clinical investigations of renal function such as uric acid, urea and creatinine are important to identify renal dysfunction, to diagnosis renal disease, to monitor disease progress and to monitor response to treatment(7). Uric acid is the major product of the catabolism of the purine nucleosides, adenosine and guanosine. Using enzymatic method, the reference intervals for serum uric acid has been reported to be 3.5 – 7.2 mg/ dl (208-428 $\mu\text{mol/l}$) for males and 2.6-6 mg / dl (155-357 $\mu\text{mol/l}$) for females(8). Urea is formed in the liver from ammonia released by deamination of amino acids (9).

Raised serum uric acid has been reported to be associated with increased risk of coronary heart disease and is commonly encountered with essential hypertension, even untreated hypertension and type-2 diabetes, which are in turn associated with coronary heart disease. It is not known whether raise serum uric acid increases the risk of hypertension and type-2 diabetes independently of known risk factors such as age, obesity, alcohol consumption and physical activity.(10-12)

According to National Kidney Foundation Microalbuminuria is defined as a Urine Albumin Excretion Rate (UAER) of approximately 30-300 mg/day in at least two of three consecutive samples of non-ketotic sterile urine.[13] The association between microalbuminuria and hypertension was described long time ago.[14-16] In 1976, parving et al highlighted the relation between microalbuminuria and the severity of hypertension.

The incidence of hypertension in India is 5 –15% in the adult population against 10–12% in the West. Essential hypertension produces clinical proteinuria and a significant reduction in renal function in 5 – 15% of patients.[17-19] The advent of more sensitive methods to quantitate the urinary albumin excretion (UAE) has revealed higher frequency (25-100%) of microalbuminuria in patients with hypertension than normotensive population. Incidence of hypertension in India (16) 3.80 to 15.63% in men and 2 to 15.38% in women in the urban areas and from 1.57 to 6.93% in men and 2.38 to 8.81% in women in rural areas(16). According to the sex incidence both the sexes are equally affected, but few books mention that males are affected more up to the age of 50 yrs, and females are affected more after age of 50 years.

2. MATERIALS AND METHODS

The patients who visited to Medicine Department, Santosh Medical College and Hospital, Ghaziabad, with a diagnosis of Hypertension were studied considering the inclusion and exclusion criteria during a period starting from May 2014 to April 2015 from one year (May 2014 to April 2015). A Case Control study with 100 patients taken as case and 25 patients as

control, were studied for the renal dysfunction in essential hypertension was undertaken at Santosh Medical College and Hospital, Ghaziabad, Uttar Pradesh.

All the parameter were analysed by using software SPSS. All the parameters were compared using ANOVA test. Case and control were compared using by chi square test and independent sample t test and p value was calculated. All the results are presented as mean \pm Standard Mean Error (SEM).

3. RESULTS

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

S.No.	Variable	CONTROL		CASE		P- Value
		MEAN	SD	MEAN	SD	
1	Age	40.36	9.56	56.31	10.47	< 0.001
2	Systolic BP	120.48	7.17	148.66	7.54	< 0.001
3	Diastolic BP	79.2	3.10	94.26	2.66	< 0.001
4	TC	178.23	21.23	248.41	64.05	< 0.001
5	TG	127.56	14.44	175.2	54.11	< 0.001
6	HDL	45.28	6.05	32.66	1.154	< 0.001
7	LDL	112.04	13.89	122.14	19.32	0.015
8	Uric acid	6.144	2.53	7.28	2.66	0.056
9	Serum creatinine	0.968	0.213	1.43	0.58	0.0002
10	Blood urea	29.92	5.41	65.37	15.36	< 0.001
11	Serum K ⁺	4.292	0.439	4.26	0.621	0.809
12	Serum Na ⁺	141.08	3.76	139.75	4.75	0.196
13	Microalbuminuria	21.44	5.47	33.18	25.99	0.027
14	Creatinine clearance	83.59	25.98	71.63	36.64	<0.001

Table 2: Age distribution of patients

AGE (in Years)	CONTROL		CASE	
	No.	PERCENT	No.	PERCENT
30-40	16	64.0%	10	10.0%
41-50	5	20.0%	26	26.0%
51-60	2	8.0%	40	40.0%
61-70	2	8.0%	24	24.0%

Total	25	100.0%	100	100.0%
Mean ± SD	40.36 ±9.56		56.31 ±10.47	

The table 2 shows 40.0% cases in the 5th decade of life where as 26.0% of cases in their 4th decade of life and maximum number of controls are present in their 3rd decade of life (64.0%).

Table 3: Duration of hypertension

DURATION OF HYPERTENSION (in yrs)	CONTROL		CASE	
	No.	PERCENT	No.	PERCENT
Nil	25	100.0%	0	0.0%
≤ 1	0	0.0%	36	36.0%
2-5	0	0.0%	44	44.0%
6-10	0	0.0%	3	3.0%
11-22	0	0.0%	17	17.0%
TOTAL	25	100.0%	100	100.0%

The table 3 shows that in cases 36 (36.0%) patients had hypertension below or equal to 1 year, 44 (44.0%) had hypertension in between 1-5 years, 3 (3.0%) patients between 6-1 years and 17 (17.0%) patients had hypertension between 11-22 years.

4. DISCUSSION

Hypertension is a major public health problem all over the world. The incidence of hypertension in India is 5-15% as compared to 10-12% in the West. Hypertension is a degenerative process, taking place in blood vessels affecting blood supply to target organ like heart, kidney and liver. Damage of these organs are called Target organ damage.

The present study is regarding the prevalence of renal dysfunction in essential hypertension and correlation of renal dysfunction with clinical profile and complication of essential hypertension. In our study the incidence of hyperuricemia in controls was 16% and the incidence of hyperuricemia in cases was 30 %.

Elevated SUA levels have been associated with an increased risk for cardiovascular disease. The potential mechanisms by which SUA may directly affect cardiovascular risk include enhanced platelet aggregation and inflammatory activation of the endothelium. In few studies, the association of SUA with cardiovascular disease was uncertain after multivariate adjustment as in the Framingham Heart Study (1985) [23] and the ARIC study (1996), but in others the association remained certain and significant.

Sharma V K et al study showed a prevalence of 24% (12 out of 50 patients). Sabharwal R K et al showed a prevalence of 33.3% (58 out of 174 cases). In 1991 Stefano Bianchi et al

published the first large study on the prevalence of microalbuminuria in hypertensives; it was found to be 35%. [24] Palatini et al in HARVEST study and PREVEND-IT (Prevention of Renal and Vascular End Stage Disease- study performed in Dutch city of Groningen) showed a prevalence of 8-15%.

5. CONCLUSION

Hypertension is a major health problem all over the world. It is one of the most common risk factor for cardiovascular and renal disorders. Hence a thorough assessment is a prerequisite to correctly identify the patients who are at risk. The prevalence of renal dysfunction varies in different population, based on the characteristics of the population and techniques and protocols used for evaluation. Microalbuminuria reflects a state of increased renal endothelial permeability and hence considered an early marker of diffuse endothelial dysfunction. Hypercholesterolemia, hypertriglycemia, increased LDL all are seen in essential hypertension.

6. REFERENCES

1. Guyton A.C., and Hall J. E. Dominate Role of the Kidney in Long- Term Regulation of Arterial Pressure and in Hypertension: the integrated system for Pressure Control. In: Textbook of Medical Physiological. 11th ed. Elsevier Saunders: 2006; Vol.1: 219-231.
2. Fox S. I. Cardiac Output, Blood Flow, and Blood Pressure. In: Human Physiology. 8th ed. McGraw Hill: 2004; 406-439.
3. Widmaier E. P., Raff H, and Strang K. T. Cardiovascular Physiology. In: Vander's Human Physiology the Mechanism of Body Function. 10th ed. McGraw- Hill: 2006; 452-454.
4. Haslett C., Chilvers E. R., Boon N. A., and Colledge N. R. Cardiovascular Disease. In: Davidson's Principles and Practice of Medicine. 19th ed. Chirchil Living stone: 2002; 357-480.
5. Casey P. E., Philips A. C., Shapiro S., and Nguyen P. Controlling High Blood Pressure. The Permanent Journal: 2006 ;10(2): 13-16.
6. Berkin K. E., and Ball S. G. Essential Hypertension: The Heart and Hypertension. Heart: 2001; 86: 467-475.
7. Cortbett J. V. Renal Function Tests In: Laboratory Tests and Diagnostic Procedures. 5th ed. Prentice Hall Health: 2000; 90- 107.
8. Burits C. A. and Ashwood E. R. Renal Function and Nitrogen Metabolism. In: Tietz Fundamental Clinical Chemistry. 5th ed. McGraw Hill: 2001; 414-426.
9. Murray R. K., Granner D. K., Mayes P. A., and Rodwell V. W. Overview of Metabolism. In: Harper's Illustrated Biochemistry. 26th ed. McGraw-Hill: 2003;122-129.
10. Smith A. F., Beckett G. H., Walker S. W., and Rae P. W. Renal Disease. In: Lecture Notes on Clinical Biochemistry. 6th ed. Blackwell Science: 2000; 51-68.
11. Ganong W. F. Renal Function and Micturation. In: Review of Medical Physiology. 20th ed. Mc Graw-Hill Companies: 2005; 675-700.
12. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 Report. JAMA. 2003; 289: 2560

13. Liese AD, Hense HW, Lowel H, Doring A, Tietze M, Keil U: Association of serum uric acid with all-cause and cardiovascular disease mortality and incident myocardial infarction in the MONICA Augsburg cohort. *Epidemiology* 1999, 10:391-397.
14. Brand FN, McGee DL, Kannel WB, Stokes J III, Castelli WP: Hyperuricemia as a risk factor of coronary heart disease: the Framingham study. *Am J Epidemiol* 1985, 121:11-18.
15. Wannamethee SG, Shaper AG, Whincup PH: Serum urate and the risk of major coronary heart disease events. *Heart* 1997, 78(2):147-153.
16. Franse LV, Pahor M, Di Bari M, Shorr RI, Wan JY, Somes GW, Applegate WB: Serum uric acid, diuretic treatment and risk of cardiovascular events in the Systolic Hypertension in the Elderly Program (SHEP). *J Hypertens* 2000, 18(8):1149-1154.
17. Johnson RJ, Kang DH, Feig D, Kivlighn S, Kanellis J, Watanabe S, Tuttle KR, Rodriguez-Iturbe B, Herrera-Acosta J, Mazzali M: Is there a pathological role for uric acid in hypertension and cardiovascular and renal disease? *Hypertension* 2013, 41(6):1183-1190.
18. Alderman M, Aiyer KJ: Uric acid: role in cardiovascular disease and effects of losatan. *Curr Med Res Opin* 2014, 20(3): 369-379.
19. Wang JG, Staessen JA, Fagard RH, Birkenhager WH, Gong L, Liu L: Prognostic significance of serum creatinine and uric acid on older Chinese patients with isolated systolic hypertension. *Hypertension* 2011, 37(4):1069-1074.
20. Freedman DS, Williamson DF, Gunter EW, Byers T: Relation of serum uric to mortality and ischemic heart disease. The NHANES I Epidemiology Follow-up study. *Am J Epidemiol* 1995, 141:637-644.
21. Scheele KW. *Examen Chemicum Calculi Urinari*, Opuscula 1, P73, 1776. Cited from Levene P.A and Bass, L.W: *Nucleic acids*. New York, Chemical catalog company, 1931.
22. Mathialahan, T; Maclennan, KA; Sandle, LN; Verbeke, C; Sandle, GI. "Enhanced large intestinal potassium permeability in end-stage renal disease". *Journal of Pathology*; 2005; 206 (1): 46–51.
23. Desai, A. "Hyperkalemia associated with inhibitors of the renin-angiotensin-aldosterone system: balancing risk and benefit." *Circulation*; 2008; 118 (16): 1609–11.
24. Lindinger MI. "Potassium regulation during exercise and recovery in humans: implications for skeletal and cardiac muscle". *J. Mol. Cell. Cardiol*; 1995; 27 (4): 1011–1022.