

Original Research

Microalbuminuria in patients with essential hypertension

¹Pradeep Kumar, ²Surendra Kumar Ghintala

¹Government Medical Officer, Department of General Medicine, District Hospital, Churu, Rajasthan, India;

²Senior resident, Department of General Medicine, PDU Medical College, Churu, Rajasthan, India

ABSTRACT:

Background: Hypertension is a disorder of circulatory regulation. Sustained hypertension causes accelerated atherosclerosis with consequent coronary heart disease (CHD), heart failure, and stroke and renal failure. The present study recorded prevalence of microalbuminuria in patients with essential hypertension. **Materials & Methods:** 94 patients of essential hypertension of both genders were included. Group I comprised of 94 cases of essential hypertension and group II comprised of 94 healthy subjects. Investigations such as blood urea and serum creatinine, plasma glucose – fasting and postprandial, serum electrolytes–sodium and potassium, serum uric acid, serum calcium and phosphate were carried out. Microalbuminuria was measured in all patients in a 24hours urinary sample. **Results:** out of 94 patients, males were 62 and females were 32. Out of 94 patients, microalbuminuria was present in 36 (38.2%). The mean blood urea (mg/dl) was 28.5 and 22.1, serum creatinine (mg/dl) was 0.89 and 0.72, uric acid (mg/dl) was 4.71 and 3.82, sodium (meq/L) was 142.3 and 138.4, potassium (meq/L) was 4.93 and 3.12, uric acid (mg/dl) was 4.78 and 3.76, serum calcium (meq/L) was 9.24 and 9.15 and serum phosphate (meq/L) was 3.85 and 3.32 in group I and group II respectively. **Conclusion:** Microalbuminuria was seen in a significant number of newly detected and untreated patients of essential hypertension.

Key words: Essential hypertension, Microalbuminuria, blood urea

Received: 18 February, 2019

Accepted: 22 March, 2019

Corresponding author: Surendra Kumar Ghintala, Senior resident, Department of General Medicine, PDU Medical College, Churu, Rajasthan, India

This article may be cited as: Kumar P, Ghintala SK. Microalbuminuria in patients with essential hypertension. J Adv Med Dent Res 2019;7(4):163-166.

INTRODUCTION

Hypertension is a disorder of circulatory regulation. Sustained hypertension causes accelerated atherosclerosis with consequent coronary heart disease (CHD), heart failure, and stroke and renal failure.¹ If untreated, approximately 50% of patients develop heart disease, 33% develop stroke, and 10%–15% develop renal failure.²

Experimental and clinical studies recognise two major causes for the increased UAE in essential hypertension haemodynamic changes leading to elevation in intraglomerular pressure; generalised angiopathy, perhaps related to endothelial dysfunction, characterised by renal and systemic transvascular albumin leakage.³ Among hypertensive patients the prevalence of microalbuminuria is about 25% and is higher than that observed in diabetic patients (20%).⁴

Hypertension (HT) is a growing public health problem and it is now being widely reported in many rural and urban parts as one of the commonest cause of morbidity and mortality.² The reasons for this

growing burden are multiple, ranging from socio-economic changes and genetic influence. At a genetic level, there is growing evidence showing an association between elevated diastolic BP and CaMK4 affecting endothelial functions like controlling vascular resistance hence increasing the risk of HT.⁵

The relationship between microalbuminuria and atherosclerotic processes seems very tight and increased UAE has been considered a marker of prevalent subclinical atherosclerosis. Clinical studies have clearly shown that among hypertensive patients with microalbuminuria an increased cardiovascular risk exists compared to normoalbuminuric patients with similar blood pressure levels.⁶ The present study recorded prevalence of microalbuminuria in patients with essential hypertension.

MATERIALS & METHODS

The present study was conducted among 94 patients of essential hypertension of both genders. All patients

were informed regarding the study and their written consent was obtained. Demographic profile of the patients was recorded. Group I comprised of 94 cases of essential hypertension and group II comprised of 94 healthy subjects. All underwent detailed history and physical examination. Investigations such as blood urea and serum creatinine, plasma glucose – fasting and

postprandial, serum electrolytes—sodium and potassium, serum uric acid, serum calcium and phosphate, lipid profile, x-ray chest and electrocardiography were carried out. Microalbuminuria was measured in all patients in a 24hours urinary sample. Results thus obtained were statistically assessed. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 94		
Gender	Male	Female
Number	62	32

Table I shows that out of 94 patients, males were 62 and females were 32.

Table II Prevalence of microalbuminuria

Total	Prevalence	Prevalence
94	36	38.2%

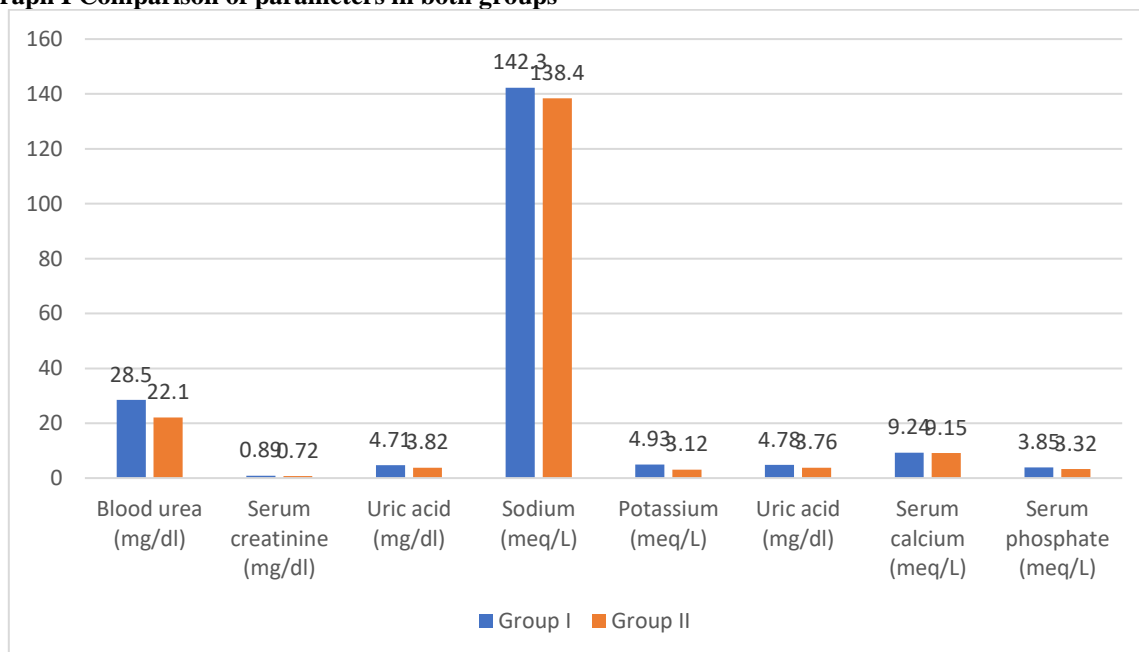
Table II shows that out of 94 patients, microalbuminuria was present in 36 (38.2%).

Table III Comparison of parameters in both groups

Parameters	Group I	Group II	P value
Blood urea (mg/dl)	28.5	22.1	0.14
Serum creatinine(mg/dl)	0.89	0.72	0.05
Uric acid(mg/dl)	4.71	3.82	0.03
Sodium (meq/L)	142.3	138.4	0.02
Potassium(meq/L)	4.93	3.12	0.04
Uric acid(mg/dl)	4.78	3.76	0.01
Serum calcium (meq/L)	9.24	9.15	0.14
Serum phosphate (meq/L)	3.85	3.32	0.05

Table III shows that mean blood urea (mg/dl) was 28.5 and 22.1, serum creatinine (mg/dl) was 0.89 and 0.72, uric acid (mg/dl) was 4.71 and 3.82, sodium (meq/L) was 142.3 and 138.4, potassium (meq/L) was 4.93 and 3.12, uric acid (mg/dl) was 4.78 and 3.76, serum calcium (meq/L) was 9.24 and 9.15 and serum phosphate (meq/L) was 3.85 and 3.32 in group I and group II respectively. The difference was significant (P< 0.05).

Graph I Comparison of parameters in both groups



DISCUSSION

Microalbuminuria and vascular dysfunctions are known to occur early in the course of essential hypertension.⁷ Microalbuminuria has been postulated to represent the renal manifestation of generalized, genetically conditioned vascular endothelial dysfunction that may underlie the link between an increased UAE and an elevated risk for cardiovascular disease.⁸ Endothelial dysfunction has been proposed to be a plausible pathophysiological mechanism of microalbuminuria. Hypertensive nephropathy is a common finding in patients with hypertension and is a common cause of chronic kidney disease.⁹ Progressive nephrosclerosis from vasculo-endothelial disease is the renal correlate of the same process that lead to coronary artery diseases, cerebrovascular diseases, hypertensive retinopathy and left ventricular dysfunction. It has been pointed out that cardiovascular risk progressively increases as renal function declines.¹⁰ The present study recorded prevalence of microalbuminuria in patients with essential hypertension.

In present study, out of 94 patients, males were 62 and females were 32. Aggarwal et al¹¹ in their study hundred patients of essential hypertension (group A) in the age group of 18-65 years were included. A control group (group B) consisting of hundred healthy normotensive, age and sex matched volunteers were also entered into the study. Arterial blood pressure was measured by digital sphygmomanometer after five minutes of rest; the values reported represented the average of three consecutive measurements taken over a 15-minute period. Urine albumin excretion (UAE) was estimated by an immunoturbidometry method. Microalbuminuria was defined as UAE between 30 and 300 mg/24 hours. In this study it was observed that prevalence of microalbuminuria in essential hypertension was 47%. Risk factors for microalbuminuria included higher age, SBP and MAP. Microalbuminuria was associated with dyslipidemia, deranged renal parameters and end organ damage in form of LVH, ischemic changes, hypertensive retinopathy and renal dysfunction. In conclusion, this study confirmed that increased urinary albumin excretion is associated with a worse pattern of cardiovascular risk factors and is a marker of concomitant cardiovascular damage in essential hypertension.

We found that out of 94 patients, microalbuminuria was present in 36 (38.2%). We observed that mean blood urea (mg/dl) was 28.5 and 22.1, serum creatinine (mg/dl) was 0.89 and 0.72, uric acid (mg/dl) was 4.71 and 3.82, sodium (meq/L) was 142.3 and 138.4, potassium (meq/L) was 4.93 and 3.12, uric acid (mg/dl) was 4.78 and 3.76, serum calcium (meq/L) was 9.24 and 9.15 and serum phosphate (meq/L) was 3.85 and 3.32 in group I and group II respectively. Nappaale et al¹² assessed the prevalence of microalbuminuria, LVH in patients with microalbuminuria and the correlation between

microalbuminuria and LVH among newly diagnosed black adult hypertensive patients attending a large outpatient hypertension. The mean age/standard deviation of the study participants was 54.3 ± 6.2 years with a female predominance (162, 63.3 %). The prevalence of microalbuminuria among newly diagnosed hypertensive patients was 39.5 %. The prevalence of LVH among patients with microalbuminuria was found to be 17 %. There was a positive correlation between microalbuminuria and left ventricular hypertrophy among the newly diagnosed adult hypertensive patients. This study demonstrates that microalbuminuria is highly prevalent among newly diagnosed black hypertensive patients and in the presence of LVH. There is also a positive correlation between microalbuminuria and LVH among newly diagnosed hypertensive patients.

CONCLUSION

Authors found that the microalbuminuria was seen in a significant number of newly detected and untreated patients of essential hypertension.

REFERENCES

1. Mogensen CE. Microalbuminuria predicts clinical proteinuria and early mortality in maturity onset diabetes. *N Engl J Med* 1984; 310: 356–360.
2. Damsgaard EM et al. Microalbuminuria is a predictor of increased mortality in elderly people. *Br Med J* 1990; 300: 297–300.
3. Yudkin JS, Jackson CA. Microalbuminuria as predictor of vascular disease in non-diabetic subjects. *Lancet* 1988; 530–533.
4. Jensen JS et al. Arterial hypertension, microalbuminuria, and risk of ischemic heart disease. *Hypertension* 2000; 35: 898–903.
5. Parving HH. Microalbuminuria in essential hypertension and diabetes mellitus. *J Hypertens* 1996; 14 (Suppl 2): S89-S93.
6. Marre M, Bouhanick B, Berrut G. Microalbuminuria. In: Brenner D (ed). *Current Opinion in Nephrology and Hypertension*. Current Science: Philadelphia, 1994; 558–5638.
7. Homer D, Fliser D, Klimm HP, Ritz E. Albuminuria in normotensive and hypertensive individuals attending office of general practitioners. *J Hypertens* 1996; 14: 655–660.
8. Redon J et al. Factors related to the presence of microalbuminuria in essential hypertension. *Am J Hypertens* 1994; 7: 801–807.
9. Cirillo M et al. Pulse pressure and isolated systolic hypertension: association with albuminuria. *Kidney Int* 2000; 58: 1211–12.
10. Aggarwal HK, Jain D, Mor S, Yadav RK, Jain P. Prevalence and clinical correlates of microalbuminuria in patients with essential hypertension—a tertiary care center cross sectional study. *J Assoc Physicians India*. 2018 May 1;66(5):30-4.
11. Levy D, Savage DD, Garrison RJ, Anderson KM, Kannel WB, Castelli WP. Echocardiographic criteria for left ventricular hypertrophy: The Framingham Heart Study. *Am J Cardiol* 1987; 59: 956-60.
12. Juliet Nappaale, Davis Kibirige, Emmanuel Sekasanvu, Elias S Sebatta, James

Kayima, Peter Lwabi, Robert Kalyesubula.
Microalbuminuria and left ventricular hypertrophy
among newly diagnosed black African hypertensive

patients: a cross sectional study from a tertiary hospital
Uganda. BMC Research Notes 2015; 8: 198.