

ORIGINAL ARTICLE**Evaluation of Serum Creatinine levels in Hypertensive Patients**¹Farhat Nadeem, ²Priya G Shinde¹Assistant Professor, Department of Physiology, Narayana Medical College, Chinthareddipalem, Nellore, Andhra Pradesh, India²Assistant Professor, Department of Physiology, RVM Institute of Medical Sciences, Hyderabad, Telangana, India**ABSTRACT:**

Background: The present study was planned for assessing Serum Creatinine levels in Hypertensive Patients. **Materials & methods:** A total of 100 participants were recruited for the study, comprising 50 individuals diagnosed with hypertension and 50 age- and sex-matched healthy controls. Venous blood samples were drawn from all participants under aseptic conditions. The samples were promptly processed, and serum creatinine concentrations were measured using a standardized biochemical assay. All the results were compared. **Results:** The comparison of serum creatinine levels between the hypertensive and control groups revealed a statistically significant elevation in the hypertensive cohort. The mean serum creatinine in hypertensive patients was 1.39 ± 0.25 mg/dL, notably higher than the 0.95 ± 0.12 mg/dL observed in the control group. **Conclusion:** Hypertension exerts a profound impact on renal function, making the renal profile a critical parameter in the evaluation and management of hypertensive patients. Chronic elevation of blood pressure leads to structural and functional alterations in the renal vasculature.

Key words: Hypertension, Creatinine, Renal**Corresponding author:** Priya G Shinde, Assistant Professor, Department of Physiology, RVM Institute of Medical Sciences, Hyderabad, Telangana, India**This article may be cited as:** Nadeem F, Shinde PG. Evaluation of Serum Creatinine levels in Hypertensive Patients. J Adv Med Dent Sci Res 2017;5(4):160-162.**INTRODUCTION**

Various classes of antihypertensive agents may reduce glomerular filtration rate (GFR) and elevate serum creatinine and potassium levels. Careful monitoring during antihypertensive therapy is essential to detect underlying renal dysfunction and prevent treatment-related complications.^{1,2}

Initial investigations by Bianchi et al, followed by more rigorous experimental work by Rettig et al,³⁻⁴ provided compelling evidence that blood pressure regulation is intrinsically linked to renal physiology. In these studies, kidney transplantation from genetically hypertensive donor rats—even those maintained normotensive via antihypertensive therapy—led to a progressive elevation in blood pressure in recipient animals, provided immune rejection was suppressed. Conversely, transplantation of kidneys from normotensive donors induced normotension in hypertensive recipients, albeit with a less pronounced effect, underscoring the kidney's central role in the pathogenesis and perpetuation of hypertension.⁵ Hence; the present study was planned for assessing Serum Creatinine levels in Hypertensive Patients.

MATERIALS & METHODS

A total of 100 participants were recruited for the study, comprising 50 individuals diagnosed with hypertension and 50 age- and sex-matched healthy controls. Comprehensive demographic and clinical data were systematically collected from all

hypertensive subjects, including variables such as age, sex, body mass index (BMI), duration of hypertension, medication history, and presence of comorbidities. Venous blood samples were drawn from all participants under aseptic conditions. The samples were promptly processed, and serum creatinine concentrations were measured using a standardized biochemical assay. These creatinine levels served as a surrogate marker of renal function, with comparisons made between hypertensive patients and healthy controls to assess the impact of hypertension on renal status. All laboratory and clinical data were systematically entered into a Microsoft Excel sheet, ensuring accuracy and traceability. The compiled dataset was subsequently exported to SPSS software (version 26.0) for statistical analysis. Descriptive statistics—including mean, standard deviation, and percentage distributions—were calculated. Inferential statistics, such as the independent samples t-test, were employed to determine whether the difference in serum creatinine levels.

RESULTS

A total of 50 participants were included in both the hypertension and control groups. In the hypertensive group, 30% (n = 15) were aged below 50 years, and 70% (n = 35) were 50 years or older. Similarly, the control group comprised 36% (n = 18) individuals younger than 50, and 64% (n = 32) aged 50 or above. The difference in age distribution between the two

groups was not statistically significant ($p = 0.128$). With regard to sex distribution, the hypertensive group consisted of 64% ($n = 32$) males and 36% ($n = 18$) females, while the control group included 58% ($n = 29$) males and 42% ($n = 21$) females. This difference also lacked statistical significance ($p = 0.339$), indicating that both groups were demographically comparable in terms of age and gender. The comparison of serum creatinine levels between the hypertensive and control groups revealed a statistically significant elevation in the hypertensive

cohort. The mean serum creatinine in hypertensive patients was 1.39 ± 0.25 mg/dL, notably higher than the 0.95 ± 0.12 mg/dL observed in the control group. This difference was found to be statistically significant with a p-value of 0.001, indicating a strong association between hypertension and renal function impairment. Elevated serum creatinine levels in hypertensive individuals reflect potential reduction in glomerular filtration rate (GFR), supporting the link between chronic hypertension and progressive renal dysfunction.

Table 1: Demographic data

Demographic data	Hypertension group		Control group		p-value
	Number	Percentage	Number	Percentage	
Age of less than 50 years	15	30	18	36	0.128
Age of more 50 years	35	70	32	64	
Males	32	64	29	58	0.339
Females	18	36	21	42	

Table 2: Comparison of serum creatinine levels

Serum creatinine levels (mg/dL)	Hypertension group	Control group
Mean	1.39	0.95
SD	0.25	0.12
p-value	0.001 (Significant)	

DISCUSSION

Growing evidence supports the role of the kidney in the pathogenesis of both cardiac and cerebrovascular complications. Recent studies highlight that, in addition to microalbuminuria, even mild elevations in serum creatinine serve as independent predictors of adverse outcomes. Notably, the prognostic value of serum creatinine is evident even within near-normal reference ranges. In hypertensive cohorts, a consistent association has been observed between serum creatinine levels and cardiovascular events; however, many of these investigations did not adequately control for treatment variables or involved populations at relatively low baseline risk.⁶⁻⁹ Hence; the present study was planned for assessing Serum Creatinine levels in Hypertensive Patients.

A total of 50 participants were included in both the hypertension and control groups. In the hypertensive group, 30% ($n = 15$) were aged below 50 years, and 70% ($n = 35$) were 50 years or older. The comparison of serum creatinine levels between the hypertensive and control groups revealed a statistically significant elevation in the hypertensive cohort. The mean serum creatinine in hypertensive patients was 1.39 ± 0.25 mg/dL, notably higher than the 0.95 ± 0.12 mg/dL observed in the control group. This difference was found to be statistically significant with a p-value of 0.001, indicating a strong association between hypertension and renal function impairment. Elevated serum creatinine levels in hypertensive individuals reflect potential reduction in glomerular filtration rate (GFR), supporting the link between chronic hypertension and progressive renal dysfunction. Peter W. de Leeuw conducted a study for assessing clinical

Significance of Renal Function in Hypertensive Patients at High Risk. The INSIGHT study was a double-blind, randomized, multicenter trial in patients with hypertension and at least 1 additional cardiovascular risk factor. Treatment consisted of nifedipine gastrointestinal therapeutic system, 30 mg/d, or hydrochlorothiazide-amiloride (25 mg/d of hydrochlorothiazide and 2.5 mg/d of amiloride hydrochloride). Primary outcome was a composite of cardiovascular death, myocardial infarction, heart failure, and stroke. Renal function was assessed by measuring creatinine clearance, serum creatinine level, and serum uric acid level and by the presence of proteinuria. Creatinine clearance fell more in nifedipine recipients than in hydrochlorothiazide-amiloride recipients. Renal insufficiency developed in 2% of nifedipine recipients and 5% of hydrochlorothiazide-amiloride recipients. Primary outcomes occurred in 15% of patients with increased serum creatinine levels and 6% of patients with normal levels (odds ratio [OR] 2.89; 95% confidence interval [CI], 1.92-4.36; $P < .001$). Primary outcomes were more likely in patients with low creatinine clearance (< 60 mL/min) than in those with higher clearances (9% vs 5%, respectively [OR, 1.51, 95%CI, 1.22-1.88; $P < .001$]). Renal function is an important predictor of risk in hypertensive patients at high risk. Antihypertensive treatment with a long-acting dihydropyridine calcium channel blocker may better preserve renal function than would treatment with diuretics.⁹ Adel A. Youssef et al examined the temporal relation between blood pressure and renal function reflected by serum creatinine in a biracial (black-white) community-based population enrolled

in the Bogalusa Heart Study. The study included 662 young adults aged 19 to 32 years, (white men, n = 188; white women, n = 289; black men, n = 67; and black women, n = 118) who were followed for an average of 7.4 years. In black men, partial correlation adjusted for age, body mass index, serum glucose, uric acid, and cigarette smoking showed that baseline systolic and diastolic blood pressure are not significantly related to baseline serum creatinine, but significantly related to serum creatinine at follow-up ($r = 0.38$, $P = .008$ and $r = 0.42$, $P = .003$, respectively). Multivariate regression analysis further showed a significant prediction of serum creatinine at follow-up by baseline systolic and diastolic blood pressure (0.031 mg/dL and 0.037 mg/dL rise in follow-up serum creatinine for every 10 mm Hg increase in systolic ($P = .000$) and diastolic ($P = .001$) blood pressure at baseline, but not the other way around. Other race and sex groups did not show such significant temporal relations. We conclude that in young black men, higher blood pressure levels within normal range precede and explain part of the increase in serum creatinine, a measure of decline in renal function. Thus, our results underscore the beneficial effect of maintaining blood pressure levels lower than what is considered as the upper normal limit, particularly in black men.¹⁰

CONCLUSION

Hypertension exerts a profound impact on renal function, making the renal profile a critical parameter in the evaluation and management of hypertensive patients. Chronic elevation of blood pressure leads to structural and functional alterations in the renal vasculature.

REFERENCES

1. G. Bianchi, U. Fox, G.F. DiFrancesco. Blood pressure changes produced by kidney cross-transplantation between spontaneously hypertensive and normotensive rats. *ClinSciMol Med*, 47 (1974), pp. 435-438
2. R. Rettig, C. Folberth, H. Stauss. Role of the kidney in primary hypertension: a renal transplantation study in rats. *Am J Physiol*, 258 (1990), pp. F606-F611
3. O. Patschan, B. Kuttler, U. Heeman. Kidneys from normotensive donors lower blood pressure in young transplanted spontaneously hypertensive rats. *Am J Physiol*, 273 (1997), pp. R175-R180
4. E. Guidi, G. Bianchi, E. Rivolta. Hypertension in man with a kidney transplant: Role of familial versus other factors. *Nephron*, 41 (1985), pp. 14-21
5. Williams B, Poulter NR, Brown MJ, Davis M, McInnes GT, Potter JF, et al. Guidelines for management of hypertension: Report of the fourth working party of the British Hypertension Society, 2004—BHSIV. *J Hum Hypertens* 2004;18: 139-85.
6. Palmer BF. Managing hyperkalemia caused by inhibitors of the renin-angiotensin-aldosterone system. *N Engl J Med* 2004;351: 585-92.
7. McMurray JJV, O'Meara E. Treatment of heart failure with spironolactone—trial and tribulations. *N Engl J Med* 2004;351: 526-8.
8. Wang JG, Staessen J, Fagard RH et al. Prognostic significance of serum creatinine and uric acid in older Chinese patients with isolated systolic hypertension. *Hypertension* 2001;37:1069- 1074
9. Peter W. de Leeuw, M. Clinical Significance of Renal Function in Hypertensive Patients at High Risk. Results From the INSIGHT Trial. *Arch Intern Med* 2004;164;(22):2459-2464
10. Adel A. Youssef et al. Temporal relation between blood pressure and serum creatinine in young adults from a biracial community Get access Arrow. *Am J Hypertens* 2000;13:770–775