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Kidney Function Impairment in Hypertensive and Non-Hypertensive Patients

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Abstract

Hypertension is recognized as one of the leading contributors to kidney damage. Additional factors such as diabetes, fluid deficiency, infections, inherited renal disorders, or certain environmental exposures can also cause kidney dysfunction in people who do not have high blood pressure. A reliable method for detecting reduced filtration capacity is to evaluate kidney performance through tests such as serum creatinine, blood urea, and estimated glomerular filtration rate (eGFR). The aim of this study was to investigate kidney dysfunction in hypertensive and non-hypertensive patients. This case control study was conducted at Mughal Labs Lahore, over the period of six months. A total of 80 patients aged from pediatric to geriatric years. Blood samples are collected and analyzed to check the kidney dysfunction (Blood Urea, Serum Creatinine and eGFR) using photometric method. Statistical analysis was performed using IBM SPSS 26, with continuous variables expressed as mean ± SD, and comparative analysis graphs. The study analyzed urea, creatinine, and eGFR using SPSS, comparing hypertensive and non-hypertensive groups with a t-test ($p < 0.05$). The mean kidney parameters were: blood urea (52±41mg/dL), serum creatinine (13±2mg/dL), eGFR (64±39mg/dL). 40 patient's patients had indicated impaired kidney function, indicated by high serum creatinine and low eGFR with hypertension. 20 patients had normal renal function which means that 5 were hypertensive (6.25%) and 15 were non hypertensive (18.75%). The remaining 20 patients were non hypertensive but still showed sign of kidney function impairment due to other causes. The study highlights high blood pressure as an important and preventable contributor to renal damage. Lowering the impact of chronic kidney disease and improving long-term health results require timely detection through regular kidney function assessments and proper management of blood pressure.

Key words: Hypertension, Non hypertension, Kidney dysfunction.

Introduction

The kidney performs several functions to maintain homeostasis in the body. They regulate the osmotic pressure by excreting either dilute or concentrated urine. They control the concentration of various ions in the blood, such as sodium, potassium, calcium and bicarbonate. They help maintain acid base balance by excreting hydrogen ions or bicarbonate. The kidneys also manage extracellular fluid volume by adjusting sodium and water excretion and contribute to blood pressure regulation through sodium balance and the production of hormones like renin. They also eliminate waste products, including urea, uric acid and creatinine.[1] Kidney function and blood pressure have a close bidirectional link. Kidney disease leads to high blood pressure, while elevated blood pressure can further damage the kidneys. In kidney disease, blood pressure

increases due to several factors. These include the buildup of salt in the body. The hyperactivity of the renin-angiotensin system and the sympathetic nervous system, and a reduced ability of blood vessels to relax properly. These changes make it harder for the body to control blood pressure, leading to hypertension.[2] The blood pressure regulation is linked to the renin-angiotensin system, which helps control blood pressure and fluid balance. This system involves a chain of reactions where the protein angiotensinogen is converted into angiotensin I and then into angiotensin II by the enzymes renin and angiotensin converting enzymes. Angiotensin II is a powerful substance that narrows blood vessels and rises blood pressure. Angiotensin II works by binding to angiotensin receptors. When the renin angiotensin become overactive, it can lead to high blood pressure and damage to organ like the heart, kidneys, and blood vessels.[3]

Blood pressure is controlled by the nervous system, hormones, and blood flow. These system work together to keep blood pressure stable. Blood pressure depends on how much blood the heart pumps and resistance it meets in the blood vessels. Sodium and water levels are affected by what we eat and how much the kidneys remove. Hormones like vasopressin help to maintain the water and salt balance. When this system performs well, when sodium level is change it will also cause to change the water level and keeps the things balance. Since modern diets are high in salt, the kidneys are mainly responsible for removing the excess. When sodium level will be change can affect the blood vessels and increase blood pressure. Since fluid volume is closely connected to blood volume, any variation in body fluids influences blood pressure. The body senses these shifts and communicates with the kidneys through nerves or hormones such as ANP to make the necessary corrections. When blood volume rises, the veins expand, allowing more blood to flow back to the heart, which increases blood pressure. As a result, the elevated pressure signals the kidneys to excrete additional salt and water. The kidneys play a crucial role in this mechanism, especially in conditions like high blood pressure (hypertension).[4]

Elevated blood pressure is a widespread chronic condition that tends to develop more frequently as people grow older. It can result in major heart and kidney complications. Raised blood pressure often occurs together with other cardiovascular risk factors. Nowadays, healthcare professionals mostly rely on automated devices to measure blood pressure. The main cause of essential hypertension is the kidneys reduced ability to remove salt from the body at normal blood pressure levels. Other parts of the body, like the brain, hormones, large arteries, and small blood vessels, also play a role. Healthy habits can help lower blood pressure, and medications can reduce the risk of related health problems.[5]

Essential hypertension is high blood pressure with no known cause. It raises the risk of problems in the brain, heart, and kidneys. In developed countries, more than 90% of people are likely to develop high blood pressure (above 140/90 mm Hg). It often occurs along with other health issues like aging, being overweight, insulin resistance, diabetes, and high cholesterol. Early damage to organs such as thickening of the heart, small amounts of protein in the urine, and memory problems can happen early in the disease. However, serious problems like stroke, heart attack, kidney failure, and dementia usually happen after many years of uncontrolled high blood pressure.[6]

Even the wealthiest nations will face a significant financial burden due to the high number of deaths caused by inadequate access to renal replacement therapy in

impoverished nations and the significant rise of ESKD patients in the future. Cost-effectiveness of preventive methods to lower the illness burden should be evaluated in relation to the local economic development and resource. Large-scale trials are still needed to assess strategies lowering the cardiovascular risk in CKD, particularly in individuals with end-stage or severe kidney disease.[7]

Uncontrolled hypertension produces target organ damage (TOD) in the heart, brain, eyes, arteries and kidneys, and the combination of TOD and hypertension raises the total cardiovascular risk. Patients with hypertension often have chronic kidney disease (CKD), which is either a cause or an effect of hypertension and significantly increases the risk of cardiovascular events associated with hypertension. In addition, hypertension is the second most common cause of end-stage renal disease, and it aggravates the age-related decrease of renal function if blood pressure (BP) is not well controlled.

Literature Review

E Ku et al. (2019) concluded that Chronic kidney disease (CKD) and hypertension are strongly related because high blood pressure can impair kidney function, and decreased kidney function can raise blood pressure even more. Reduced nephron mass, salt and fluid retention, sympathetic nervous system activation, hormonal alterations such elevated renin-angiotensin-aldosterone activity, and endothelial dysfunction all contribute to the development of hypertension in chronic kidney disease. A systolic blood pressure of less than 130mm Hg is the suggested treatment goal for CKD patients. Diuretics, ACE inhibitors, and angiotensin receptors blockers are some of the management techniques. Uncontrolled hypertension accelerates renal disease and raises cardiovascular risks. Strict blood pressure control reduces mortality and cardiovascular events, but it may not decrease the course of CKD. [8]

M Gorostidi et al. (2025) concluded that one significant risk factor for death from chronic kidney disease (CKD) is hypertension. Using data from the Spanish ABPM Registry, 34,006 hypertension patients with estimated glomerular filtration rate (eGFR) were monitored for a median of 9.7 years. 1,457 (33%) of them passed away during follow-up, and 4,408 (13%) had eGFR <60 mL/min/1.73 m². Higher 24-hour, daytime, and midnight systolic BP were connected to higher total mortality, while office BP indicated a U-shaped connection. Lower 24-hour systolic BP (<120 mmHg) reduced mortality only in patients with poor renal function. Ambulatory BP readings were more informative than clinic BP for predicting death, with lower ambulatory BP related with lower mortality in individuals with limited renal function.[9]

Pinho et al. (2015) concluded that chronic kidney disease (CKD) is a major global health problem linked to high cardiovascular morbidity and mortality. Patient safety, as defined by the WHO, involves reducing unnecessary harm in healthcare, and, managing disease-related risks directly affects is one of the most important risk factors for CKD, contributing both to its development and progression, while CKD itself is a major cause of secondary hypertension. Its prevalence ranges from 1.5% to 43.3% worldwide and is influenced by aging and socioeconomic conditions. In Brazil, hypertension accounts for 34% of dialysis cases. The study hypothesized is associated with additional cardiovascular and renal risk factors and compared hypertensive patients with and without CKD, including their anti-hypertensive treatments.[10]

JS Lawson et al. (2021) concluded that hypertension and CKD are frequently observed together, and they have an impact on one another. High blood pressure can harm organs including the eyes, brain, heart, and kidneys. Hypertension also causes proteinuria, which increases the risk of CKD progression and death. Regular blood pressure monitoring and effective treatment are necessary. Systolic blood pressure should be kept below 160 mmHg, according to guidelines. Telmisartan and amlodipine besylate are useful medications for controlling hypertension in with chronic kidney disease.[11]

AEG Raine et al. (1994) concluded that there is a tight relationship between renal function and hypertension. By modifying renal haemodynamics and salt excretion, the kidney controls blood pressure; however, these processes are disrupted in established hypertension. Although hypertensive nephropathy significantly more common in Black people, the relationship between benign hypertension and renal injury is full unclear, even if malignant hypertension is obviously the cause of renal failure. Renal disease frequently results in secondary hypertension, and their combination speeds up the deterioration of the kidneys. The necessity of controlling hypertension to preserve kidney function is demonstrated by the fact that effective blood pressure control, particularly with ACE inhibitors, can halt renal degeneration, especially in diabetic nephropathy.[12]

F Viazzi et al. (2017) concluded that over the course of a four-year follow-up, the records of 17,160 patients of hypertension who all had normal baseline renal function (eGFR $\geq$ 60 mL/min/1.73/m²) were examined. 23% had albuminuria at baseline. Twenty percent experienced renal impairement throughout the follow-up period, of which 28% had albuminuria and 17% did not. The likelihood of developing stage 3 chronic kidney disease (CKD) was 1.8 higher in individuals with baseline albuminuria. Every 5 mL/min drop in baseline eGFR below 90 mL/min increased the risk of CKD by 1.54 times ($p < 0.001$) in individuals without albuminuria. Although renal deterioration is more common in patients without albuminuria, the results demonstrate that both albuminuria and decreased eGFR independently predict renal decline, suggesting a common non-albuminuric phenotype with hypertension.[13]

AM De Bhailis et al. (2022) concluded that chronic kidney disease (CKD) and hypertension are connected conditions that exacerbate each other. While CKD causes high blood pressure through a variety of processes, hypertension contributes to the deterioration of renal function. Reduced nephron count, sodium retension, volime expansion, sympathetic nervous system activation, hormonal balance via the renin-angioensin-aldosterone system (RAAS), and endothelilal dysfunction are the causes of hypertension in chronic kidney disease (CKD). The development of end stage renal disease is accelerated by poorly managed hypertension. This review emphasis the involvement of sympathetic nervous system, RAAS activation, and sodium balance in the intricate pathophysiology of hypertension in chronic kidney disease. In order to manage hypertension linked to chronic kidney disease (CKD) and prevent additional renal consequences, it also addresses renovascular disease as a cause and treatment target.[14]

VA Luyckx et al. (2013) concluded that Adverse in-utero experiences might hinder kidney development and decreased the number of nephrons, increasing the lifetime risk of hypertension and chronic kidney disease, according to developmental programming of non-communicable diseases. Prematurity, low birthweight, and rapid weight gain

during childhood are important makers of low nephron number and are associated with kidney illness, proteinuria, and hypertension in later life.[15]

Material and Methods

This study will be conducted as a case–control design at Mughal Labs over a period of three to four months after synopsis approval. A total of 80 patients will be included, with the sample size calculated using Cochran's formula ($N_0 = Z^2 \times p \times [1 - p] / e^2$). Participants of both genders from pediatric to geriatric age groups will be enrolled based on informed consent and availability of renal function test reports, including serum urea, serum creatinine, and eGFR. Patients with diabetes mellitus, pregnant women, those with incomplete data, and individuals with acute severe illness will be excluded. Standard laboratory equipment, including a biochemical analyzer and routine sampling accessories, will be used for analysis.

Results:

In this study, statistical analysis was undertaken to analyse the behaviour of dependent variables related to renal function tests, including blood creatinine, urea, and eGFR. All data were first examined for completeness and accuracy, followed by assessment of normalcy. For normally distributed data, continuous variables were expressed as mean ± standard deviation (SD); for non-normally distributed data, they were written as median with inter-quartile range (IQR). Comparisons between hypertension and non-hypertensive groups were performed using the independent samples paired t-test for normally distributed variables. A p-value below 0.05 was considered statistically meaningful. All data analyses were performed using SPSS software. This approach assisted in identifying the association and variation in kidney function indicators between the study groups.

Table No.01: Descriptive statistics for dependent variables

Parameters	Maximum	Minimum	Mean	Std. Deviation
Urea	232	14	52	41
creatinine	13	.53	2	2
eGFR	171	4	64	39

The table presents the descriptive statistics for the renal function indicators used as dependent variables in this research. For urea, the highest observed value is 232 mg/dL, while the lowest is 14 mg/dL. The average urea level is 52 mg/dL, with a standard deviation of 41, indicating substantial variation among the participants. Creatinine levels range from 0.53 mg/dL to 13 mg/dL, with a mean of 2 mg/dL and a standard deviation of 2, showing moderate differences across individuals. The eGFR values also demonstrate wide dispersion, spanning from 4 mL/min/1.73m² to 171 mL/min/1.73m². The mean eGFR is 64, with a standard deviation of 39, suggesting considerable decline and variability in renal function among the study subjects.

Graphical Description of Study:

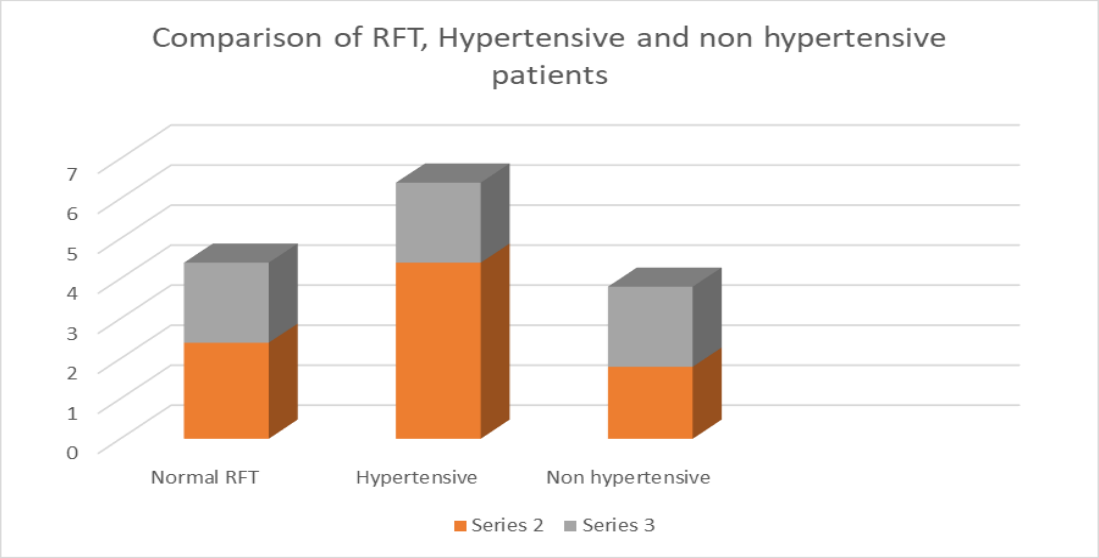

Figure No. 02: Comparison of Creatinine and eGFR

Out of the 80 patients whose kidney function was assessed, twenty individuals (25%) displayed normal renal function tests (RFTs). Among those with compromised kidney function, forty patients (50%) were hypertensive, indicating that kidney impairment is more prevalent in individuals with hypertension. Additionally, twenty participants (25%) showed reduced renal function despite not having hypertension. This distribution illustrates both the significant impact of hypertension on kidney health and the potential for renal dysfunction even in the absence of elevated blood pressure.

Distribution of Sample According to Age:

This graph displays the distribution pf renal function indicators, including blood urea, serum creatinine and eGFR.

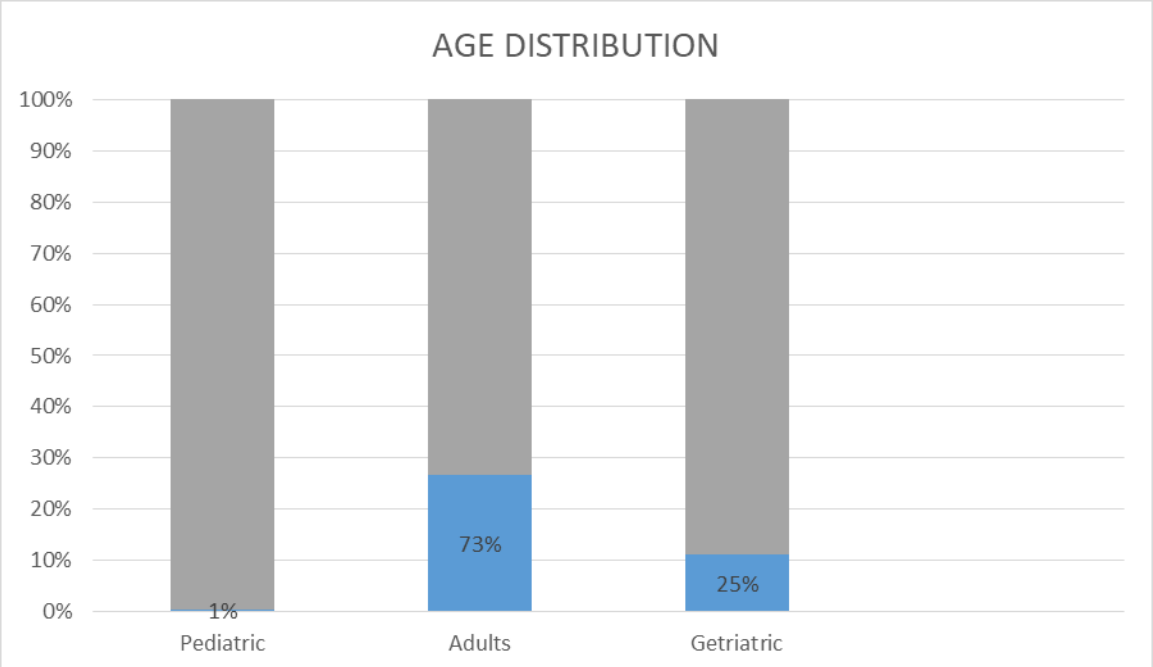

Figure No. 03: Bar graph showing kidney dysfunction among age groups

The study included 80 participants to investigate renal function impairment in hypertensive and non-hypertensive individuals. The age distribution revealed that 1% were pediatric patients, 73% were adults, and 25% were elderly. This pattern indicates that adults constituted the majority of the sample, which aligns with the higher prevalence of hypertension and kidney dysfunction in this age group. Although the inclusion of both pediatric and geriatric subjects provides a more comprehensive understanding of renal function across different life stages, these groups are relatively smaller compared to adults. Overall, the sample size offers a representative picture of kidney function variations in both hypertensive and non-hypertensive individuals across a range of age categories.

Distribution of Sample According to Gender:

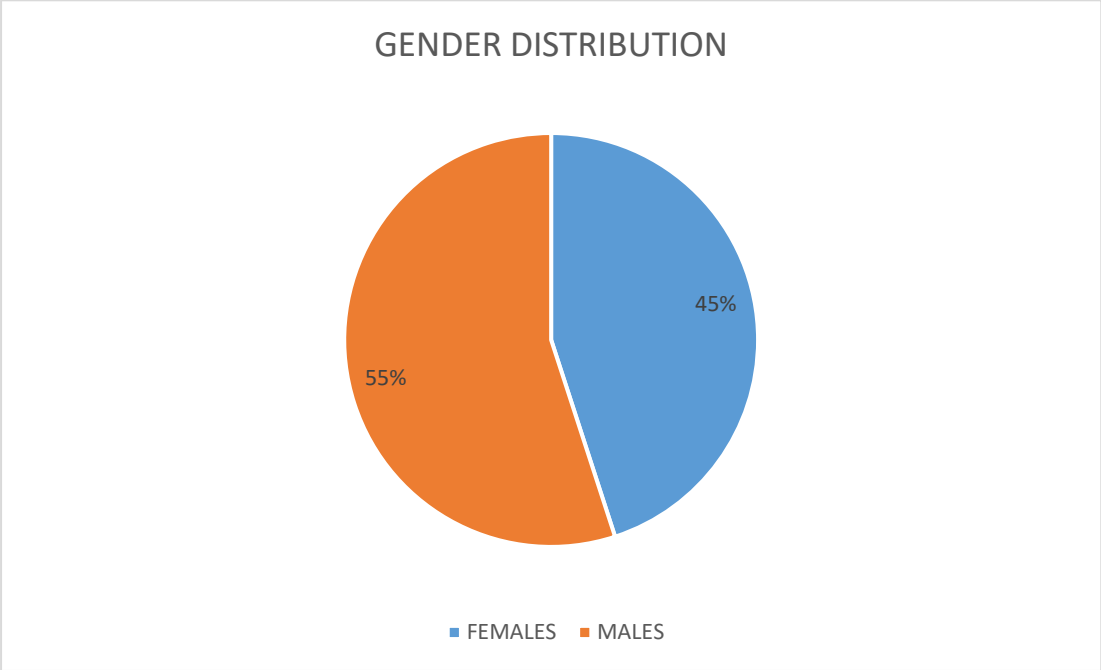

Figure No. 04 Pie graph illustrating illness distribution by sex

To assess gender patterns and their potential influence on renal outcomes, a total of 80 samples were evaluated in this study on kidney function impairment among hypertensive and non-hypertensive individuals. The sample showed a slightly higher proportion of males, with 45% females and 55% males. This gender breakdown helps determine whether sex-related differences contribute to variations in kidney function indicators across both hypertensive and non-hypertensive groups. The reasonably balanced yet diverse sample allows for more reliable comparisons and strengthens the overall credibility of the study results.

Normality Test:

Distribution normality of continuous variables (urea, creatinine, eGFR) was assessed using Shapiro-Wilk tests. Urea and creatinine values demonstrated significant deviation from normality in the hypertensive group ($p<0.001$), whereas eGFR followed normal distribution in both groups ($p>0.05$). Consequently, non-parametric Mann-Whitney U tests were employed for urea and creatinine comparisons, while parametric independent t-tests were utilized for eGFR analysis

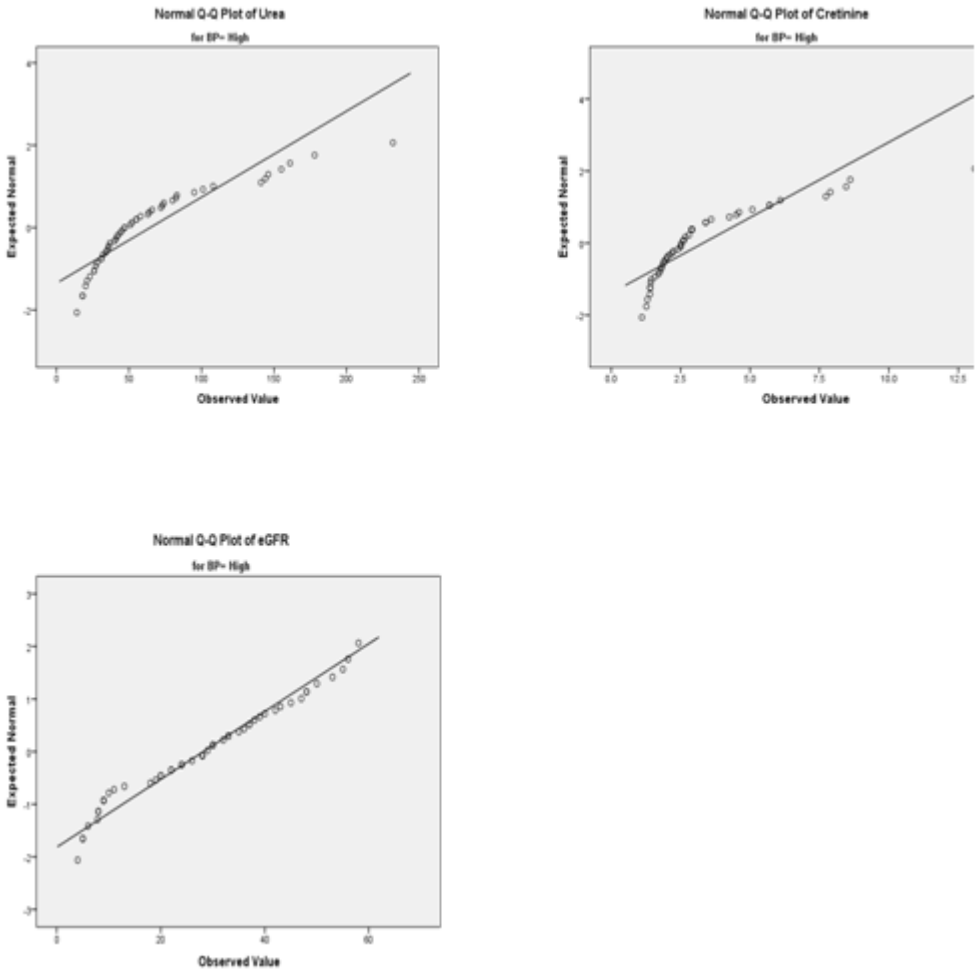

Figure1: Normal QQ Plot of urea, creatinine and Egfr FOR Hypotensive Group

Table1: Non-Parametric Analysis of Urea and Creatinine

Parameter	Hypertensive (n=51) Median (IQR)	Normotensive (n=29) Median (IQR)	Mann-Whitney U	Z-Score	P-Value
Urea (mg/dl)	47 (28-76)	29 (26-33)	325	-4	< 0.001
Creatinine (mg/dl)	2 (1-3)	1 (0-1)	32	-7	< 0.001

IQR = Interquartile Range; Z-score = standardized test statisti

Independent Sample T-Test for eGFR:

Statistical analysis confirms hypertensive patients have significantly lower eGFR than normotensive individuals (mean difference: -65.24 mL/min, t (78) = -18.34, $p < 0.001$). The assumption of equal variances is validated (Levene's test $p = 0.639$), and the 95% confidence interval (-72.32 to -58.15 mL/min) provides precise estimation of this substantial difference. These findings demonstrate severe renal filtration impairment associated with hypertension, with both statistical and clinical significance.

Table 2: Independent Sample T-Test for eGFR

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	T	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
eGFR	Equal Variance Assumed	.222	.639	-18	78	.000	-65	3	-72	-58
	Equal Variance not Assumed			-18	58	.000	-65	3	-72	-58

Discussion

Impaired kidney function is closely linked to hypertension and is a significant public health concern. Since the kidneys are essential for preserving fluid balance and blood pressure, long-term high blood pressure can seriously harm renal tissues. Chronic high blood pressure in hypertensive patients results in prolonged stress on the blood arteries in the kidneys, which produces glomerular sclerosis, decreased renal perfusion, and a gradual decrease in the GFR. Elevated levels of blood urea and serum creatinine, two important biochemical indicators of compromised kidney function, are a consequence of these pathological alterations. Additionally, proteinuria—an early sign of renal damage—is accelerated by hypertension, which also advances chronic kidney disease (CKD). The results of elevated renal biomarkers in hypertensive people corroborate the documented link between renal impairment and uncontrolled blood pressure.

Non-hypertensive patients, on the other hand, typically exhibit superior kidney function measures, with blood creatinine, urea, and estimated GFR levels that are normal or almost normal. However, other conditions including diabetes mellitus, dehydration, infections, nephrotoxic medications, or genetic predisposition may still cause kidney impairment in these patients. These factors can independently impact renal function and should not be disregarded, even though the risk is lower than in hypertensive individuals.[16]

Because of this, patients with hypertension often have lower glomerular filtration rates (GFR) and higher blood urea and creatinine levels. These results show a reduction in the kidneys' capacity to effectively filter waste. Microalbuminuria, often known as proteinuria, is seen in many hypertensive people and is an early indicator of endothelial dysfunction and glomerular injury. The following factors frequently affect how severe renal damage is in hypertension patients: The length of hypertension Inadequate management of blood pressure Comorbid conditions like dyslipidaemia and diabetic mellitus. Chronic kidney disease (CKD) and end-stage renal disease (ESRD) can result from long-term uncontrolled hypertension, which speeds up the development of mild renal impairment. These results are in line with earlier research showing that hypertensive populations are more likely than normotensive people to have impaired renal function.[17]

Serum creatinine, urea, and estimated GFR are examples of kidney function parameters that are typically normal or nearly normal in non-hypertensive patients. Glomerular integrity and filtration capacity are preserved when there is no prolonged high blood pressure because this lessens the mechanical strain on the renal vasculature.[18]

The greater proportion of subjects, pharmacologically treated for high BP, we found in the group with stage 1 and stage 2 CKD when compared to the group without renal dysfunction is probably due to the longer duration of hypertension, the higher BP values and the greater BMI observed in the former in comparison to the latter. It is likely that all these characteristics prompted the general practitioners to treat these patients with anti hypertensive drugs more frequently than those without CKD.[19]

Clinically, our results highlight the necessity of early identification, rigorous blood pressure control for hypertensive patients, and routine monitoring for all patients to stop the development of chronic kidney disease. These findings are supported by comparisons with other research, which show that hypertension people had a higher risk of renal impairment, although the study's.

The findings corroborate the theory that whereas non-hypertensive people have superior renal reserve, continuous blood pressure increase leads to progressive nephron loss. Additionally, the data highlight that renal impairment may be transient or owing to other reversible causes in non-hypertension patients, but it is frequently persistent and progressive in hypertensive patients.

Conclusion

Kidney function impairment is closely linked to hypertension, as sustained high blood pressure damages renal vessels, resulting in increased serum creatinine and reduced eGFR. In this study of 80 patients, 75% showed impaired renal function, while 25% had normal results. Among the 60 patients with renal impairment, hypertension was present in 40 cases (66.7%), indicating a higher burden of kidney dysfunction in hypertensive individuals compared to non-hypertensive patients. A slight male predominance was observed among impaired cases. Overall, the findings emphasize hypertension as a key factor in renal decline and highlight the need for early screening and effective blood pressure control to prevent further kidney damage.

Recommendations

1. Regular RFT screening (creatinine, eGFR, urine albumin) for both hypertensive and at-risk non-hypertensive patients.
2. Maintain rigorous blood pressure control to prevent further renal injury.
3. Promote lifestyle modifications, such as a low-salt diet, adequate hydration, weight management, and consistent exercise.

Refrences

[1] Tanner, G.A., *Kidney function.* Medical Physiology-Principles for Clinical Medicine. 3rd ed. Philadelphia: Lippincott Willams and Wilkins, 2009: p. 391-418.

[2] Adamczak, M., et al., *Kidney and hypertension.* Kidney International, 2002. **61**: p. S62-S67.

[3] Coffman, T.M. and S.D. Crowley, *Kidney in hypertension: guyton redux.* Hypertension, 2008. **51**(4): p. 811-816.

[4] Navar, L.G., *The role of the kidneys in hypertension.* The journal of Clinical hypertension, 2005. **7**(9): p. 542-549.

[5] Staessen, J.A., et al., *Essential hypertension.* The Lancet, 2003. **361**(9369): p. 1629-1641.

[6] Messerli, F.H., B. Williams, and E. Ritz, *Essential hypertension.* The Lancet, 2007. **370**(9587): p. 591-603.

[7] Anyaegbu, E. and V. Dharnidharka, *Hypertension in the teenager.* Pediatric Clinics of North America, 2013. **61**(1): p. 131.

[8] Nwankwo, E., B. Nwankwo, and B. Mubi, *Prevalence of impaired kidney function in hospitalized hypertensive patients in Maiduguri, Nigeria.* The internet journal of internal medicine, 2006. **6**(1).
[9] Taddei, S., et al., *Hypertension, left ventricular hypertrophy and chronic kidney disease.* Heart failure reviews, 2011. **16**(6): p. 615-620.
[10] Segura, J. and L.M. Ruilope, *Hypertension in moderate-to-severe nondiabetic CKD patients.* Advances in chronic kidney disease, 2011. **18**(1): p. 23-27.
[11] McClellan, W.M. and W.D. Flanders, *Risk factors for progressive chronic kidney disease.* Journal of the American Society of Nephrology, 2003. **14**(suppl_2): p. S65-S70.
[12] Tsai, Y.-H., et al., *Association of chronic kidney disease with small vessel disease in patients with hypertensive intracerebral hemorrhage.* Frontiers in neurology, 2018. **9**: p. 284.
[13] Pirkle, J.L. and B.I. Freedman, *Hypertension and chronic kidney disease: controversies in pathogenesis and treatment.* Minerva urologica e nefrologica= The Italian journal of urology and nephrology, 2013. **65**(1): p. 37.
[14] Lv, J.-C. and L.-X. Zhang, *Prevalence and disease burden of chronic kidney disease.* Renal fibrosis: mechanisms and therapies, 2019: p. 3-15.
[15] Cha, R.-h., et al., *Association between blood pressure and target organ damage in patients with chronic kidney disease and hypertension: results of the APrODiTe study.* Hypertension Research, 2014. **37**(2): p. 172-178.
[16] Ku, E., et al., *Hypertension in CKD: core curriculum 2019.* American journal of kidney diseases, 2019. **74**(1): p. 120-131.
[17] Gorostidi, M., et al., *Mortality according to ambulatory blood pressure in hypertensive patients with normal or impaired kidney function.* Hypertension Research, 2025: p. 1-10.
[18] Pinho, N.A.d., R.d.C.B.d. Oliveira, and A.M.G. Pierin, *Hypertensive patients with and without kidney disease: assessment of risk factors.* Revista da Escola de Enfermagem da USP, 2015. **49**: p. 101-108.
[19] de Leeuw, P.W., et al., *Clinical significance of renal function in hypertensive patients at high risk: results from the INSIGHT trial.* Archives of internal medicine, 2004. **164**(22): p. 2459-2464.50.`