Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(1), 912-921 | ISSN:2251-6727

The Impact of Hypertension on Renal Function: Mechanisms and Therapeutic Approaches in the U.S. Population

[1]Mohammed Nadeem Ansari, [2]Kashif Latif, [3]Faisal H. Cheema, [4]Kent Stonking, [5]Ambreen Qamar,

[1]Director- Clinical Research Services, MD, MPH, MBA, Department of Endocrinology, AM Diabetes & Endocrinology Center, TN 38133, United States.

[2]Principal Investigator, MD, Department of Endocrinology, AM Diabetes & Endocrinology Center, TN 38133, United States.

[3]CEO Clinre, MD, Department of Internal Medicine, ClinRe, 3434 Washington Blvd, Arlington, Virginia- 22201

[4]Head Pharmacist, PharmD, CDCES, Department of Endocrinology, AM Diabetes & Endocrinology Center, TN 38133, United States.

[5]Research Scholar, MBBS, Mphil, Department of Sleep Medicine, ClinRe, 3434 Washington Blvd, Arlington, Virginia- 22201

Corresponding Author

Mohammed Nadeem Ansari, Director- Clinical Research Services, MD, MPH, MBA, Department of Endocrinology, AM Diabetes & Endocrinology Center, TN 38133, United States.

(Received: 16 September 2024 Revised: 11 October 2024 Accepted: 11 January 2025)

KEYWORDS
Kidneys, Hypertension, blood pressure and Renal Function

ABSTRACT:

Introduction: High blood pressure and kidney function have a complicated relationship, and hypertension is one of the main causes of chronic kidney disease (CKD). Chronic hypertension causes renal dysfunction by harming the kidneys through processes like glomerular hyperfiltration and vascular remodeling. Kidney disease, on the other hand, can make hypertension worse by upsetting the sodium and fluid balance. A key player in this relationship is the renin-angiotensin-aldosterone system (RAAS), which controls blood pressure and kidney function.

Aims: To investigate the relationship between hypertension (high blood pressure) and renal function, focusing on the mechanisms that link elevated blood pressure to kidney damage and deterioration of renal function.

Materials and method: The present study was a Cross-sectional study. This Study was conducted from 1 year. Total 100 patients were included in this study.

Result: The laboratory values across the five groups show relatively consistent results with slight variations. Total protein levels remain stable, ranging from 7.1 to 7.3 g/dL, while albumin levels are slightly higher in the 121-130 mmHg group (4.3 g/dL) but return to 4.2 g/dL in the lowest and highest groups. Hemoglobin levels range from 13.6 to 13.9 g/dL, with minimal variation.

Conclusion: In conclusion, hypertension has a significant impact on renal function, contributing to kidney damage through mechanisms such as increased glomerular pressure, endothelial dysfunction, and renal fibrosis. Over time, high blood pressure can lead to a decline in glomerular filtration rate (GFR), promoting the development of chronic kidney disease (CKD).

INTRODUCTION

Worldwide, hypertension is a major risk factor for cardiovascular disease and death from all causes. According to a Global Burden of Disease research, smoking, hyperglycemia, elevated body mass index, and small for gestational age birth weight were the next most common causes of cardiovascular disease-related mortality, accounting for 10.4 million deaths and 218 million disability-adjusted life years[1]. Hypertension and chronic kidney disease are closely intertwined conditions as hypertension can lead to deteriorating

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(1), 912-921 | ISSN:2251-6727

renal function and progressive chronic kidney disease can contribute to worsening hypertension [2,3]. A global health concern, chronic kidney disease (CKD) is estimated to affect 11–13% of people globally. Its prevalence is comparable in the Japanese population [4]. Furthermore, the prevalence of CKD has been steadily increasing in Japan and many other nations. Chronic kidney disease (CKD) is a known risk factor for cardiovascular disease (CVD), end-stage renal disease (ESRD), and all-cause mortality [5]. In light of these facts, one of the most important public health priority is preventing the advancement of CKD.About 80 to 85% of CKD patients have hypertension, which is significantly more common than in the general population. Furthermore, hypertension increases the risk of CVD, CKD development, and the incidence of ESRD. Blood pressure (BP) is also closely linked to renal function.

The prevalence of hypertension has been on the rise which causes a large global economic burden worldwide. Hypertension is a major independent risk factor for cardiovascular events, cerebrovascular events and renal disease. Hypertension-induced kidney damage is associated with increased cardiovascular morbidity and mortality. The coexistence of hypertension and obesity, hyperlipidemia [6] or diabetes results in worse renal dysfunction than isolated occurrence of either risk factor alone. Evidence from numerous clinical trials has demonstrated benefits of blood pressure (BP) control. The target of 140/90mmHg that has been established from observational data remains fully justifiable. However, it is unclear whether the available results could be extrapolated to elderly patients. It is unknown whether the target of 140/90mmHg is most optimal goal to protect elderly renal function. It is debatable about the BP targets when hypertension in the elderly is complicated by coexist with obesity, hyperlipidemia or diabetes. The lower target limit of BP for hypertension treatment is still not determined. On the other hand, Intensive hypertension treatment probably causes hypotension in elderly patients which is also an independent predictor of cardiovascular disease and of all-cause mortality [7].

Chronic kidney disease (CKD), characterized by persistent impairment of kidney function and structure, is emerging as a global health burden due to its rapidly increasing prevalence and associated high morbidity and mortality rates. Chronic conditions, such as diabetes mellitus, hypertension, and obesity, are major risk factors for CKD development. [8]Currently, the treatment of underlying conditions, such as diabetes mellitus and hypertension, is the primary approach to prevent CKD onset. However, a more comprehensive elucidation of the pathological mechanisms underlying CKD is urgently needed as this enhanced understanding would facilitate the identification of novel therapeutic targets for CKD prevention and treatment. Renal fibrosis is a pathological hallmark of CKD that is characterized by the dysregulated overproduction and accumulation of extracellular matrix (ECM) proteins, [9] especially fibronectin (FN) and collagen, which culminate in structural deformation and compromised functional integrity of the renal parenchyma. [10] Transforming growth factor-β (TGF-β) stands out as the most potent profibrogenic factor. TGF-β expression levels are upregulated in fibrotic pathologies affecting multiple organs, including the pulmonary, hepatic, and renal systems. Elevated levels of TGF-β directly upregulate ECM components, such as FN and collagen, via SMAD-dependent and SMAD-independent signaling cascades. Concomitantly, TGF-β exerts a suppressive effect on ECM degradation through inhibition of matrix metalloproteinases (MMPs). [11]Additionally, TGF-β serves as a key mediator in activating the trans-differentiation of myofibroblast precursor cells and promoting proliferation of glomerular mesangial cells, both of which processes contribute to the pathogenic overproduction of ECM components in the fibrotic renal milieu. In this context, inhibition or neutralization of TGF-β attenuates excessive ECM deposition and ameliorates renal fibrosis in animal models.

Blood pressure (BP) is one of the most important determinants of the cardiovascular and renal health of populations. This is why lowering the prevalence of hypertension, defined as a systolic BP >140 mm Hg or a diastolic BP >90 mm Hg, has been set as a major objective by the World Health Organization. [12] A pooled analysis of >1400 population-based studies, in which 19.2 million adults aged ≥18 years had their BP measured, has shown that both age-standardized systolic and diastolic BPs decreased substantially between 1975 and 2015,[13] suggesting an increased awareness and a global improvement in BP control. Yet, this amelioration occurred essentially in high-income and in some middle-income countries, whereas the prevalence of elevated BP rather increased in lower-income countries. Moreover, the worldwide number of

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(1), 912-921 | ISSN:2251-6727

individuals with hypertension continues to rise due mainly to the growth and the aging of populations. A rightward shift in the distribution of BP in low- and middle-income countries might also play a role in the increased prevalence of patients with hypertension.[14],[15] The most recent epidemiologic survey has analyzed the trajectory of BP in 200 countries and territories between 1990 and 2019.[16] This very large survey (>100 million participants included) has confirmed the ongoing improvement in the detection, treatment, and control of hypertension but with a large variability between countries; low-income countries are still lagging behind.

Chronic kidney disease (CKD) is defined as a persistent estimated glomerular filtration rate (eGFR) <60 mL/(min·1.73m2), albuminuria (albumin/creatinine ratio [UACR] ≥30 mg/g), or other markers of kidney damage for at least 3 months. Based on the levels of eGFR and albuminuria, CKD is classified into 5 stages according to the level of GFR (G1–G5) and three categories according to the absence or presence of albuminuria (A1, A2, and A3).[17] CKD affected about 9% of the World population in 2017, but with large variations between regions, the burden being greater in low-income regions where the CKD prevalence ranged between 8% and 16%.[18],[19] The prevalence of CKD is higher in females than in males and differs according to race. Of note, the prevalence of CKD is usually calculated based on the CKD-EPI formula with correction for gender and race and figures vary if other equations are used. [20] Recently, a new creatinine-based and cystatin C–based formula to estimate GFR without race has been proposed.[21] This new equation has generated many comments suggesting that it should be used only in the United States as other formulas based on local cohorts are probably more accurate in Europe and Africa.[22]

MATERIALS AND METHODS

Study Area:

This study was conducted at the ClinRe Research Trial Site in collaboration with the AM Diabetes & Endocrinology Center in Memphis, Tennessee. ClinRe, an advanced integrated network, employs data-driven methodologies and analytics to optimize the alignment of clinicians and patients with relevant clinical trials. Partnering with the AM Diabetes & Endocrinology Center, a leading institution in the management of diabetes and endocrine disorders, provided an ideal environment for conducting rigorous and high-quality research in the field of endocrinology.

Study Design:

A cross-sectional study design was employed to assess the prevalence and determinants of hypertension-related kidney dysfunction. This observational design collects data from a diverse group of individuals at a single time point, without manipulating variables, allowing for the identification of natural associations.

Cross-sectional studies are ideal for determining the prevalence of health conditions, analyzing associations, and providing a snapshot of health determinants. This design is efficient, cost-effective, and suitable for identifying risk factors and trends within the population. It offers valuable insights into hypertension and renal function, laying the foundation for future longitudinal research.

STUDY PERIOD: 12 Months

Inclusion Criteria:

- Adults aged 18 years or older.
- Diagnosed with hypertension (systolic BP ≥ 140 mmHg or diastolic BP ≥ 90 mmHg, per current guidelines).
- Normotensive individuals (BP < 120/80 mmHg) for comparison.
- Normal renal function (eGFR ≥ 60 mL/min/1.73 m²) or early-stage chronic kidney disease (eGFR < 60 mL/min/1.73 m²) not requiring dialysis.
- Ability to provide informed consent and comply with study protocols.

Exclusion Criteria:

- Secondary hypertension (e.g., renovascular disease, pheochromocytoma, Cushing's syndrome).
- Advanced chronic kidney disease (eGFR < 15 mL/min/1.73 m²) or those requiring dialysis.
- Active kidney disease (e.g., glomerulonephritis, nephrotic syndrome, AKI).

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(1), 912-921 | ISSN:2251-6727

- Major co-morbidities (e.g., uncontrolled diabetes, active infections) that could confound results.
- Medications significantly affecting BP or renal function (e.g., corticosteroids, immunosuppressants) unless stable for ≥6 weeks.
- Pregnant or breastfeeding women due to potential renal and hypertension complications.

SAMPLE SIZE and Source: : A total of 100 patients were included in this study, all recruited from the AM Diabetes & Endocrinology Center, located in Memphis, Tennessee, USA.

Statistical Analysis:

For statistical analysis, data were initially entered into a Microsoft Excel spreadsheet and then analyzed using SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism (version 5). Numerical variables were summarized using means and standard deviations, while categorical variables were described with counts and percentages. Two-sample t-tests, which compare the means of independent or unpaired samples, were used to assess differences between groups. Paired t-tests, which account for the correlation between paired observations, offer greater power than unpaired tests. Chi-square tests (χ^2 tests) were employed to evaluate hypotheses where the sampling distribution of the test statistic follows a chi-squared distribution under the null hypothesis; Pearson's chi-squared test is often referred to simply as the chi-squared test. For comparisons of unpaired proportions, either the chi-square test or Fisher's exact test was used, depending on the context. To perform t-tests, the relevant formulae for test statistics, which either exactly follow or closely approximate a t-distribution under the null hypothesis, were applied, with specific degrees of freedom indicated for each test. P-values were determined from Student's t-distribution tables. A p-value ≤ 0.05 was considered statistically significant, leading to the rejection of the null hypothesis in favour of the alternative hypothesis.

RESULT

Table 1: The association of BP levels at baseline with demographic Characteristics

Characteristics	≤ 120 n = 20	121–130 n = 10	131–140 n = 20	141–150 n = 25	151–160 n = 25
Male sex, %	72.9	74.3	69.5	69.7	73.5
Age, year	62.4 ± 8.1	61.9 ± 8.8	62.7 ± 8.2	62.3 ± 8.0	62.9 ± 7.7
BMI	25.4 ± 3.7	25.6 ± 3.5	25.5 ± 3.7	25.8 ± 4.2	26.1 ± 3.4
Abdominal girth	87.4 ± 10.7	89.0 ± 9.8	90.2 ± 9.7	91.2 ± 9.8	90.6 ± 9.1

Table 2: The association of BP levels at baseline and Laboratory values

		Baseline SBP				
		≤ 120 n = 20	121–130 n = 10	131–140 n = 20	141–150 n = 25	151–160 n = 25
Laboratory values	**Total protein, g/dL**	7.2 ± 0.5	7.2 ± 0.5	7.3 ± 0.5	7.3 ± 0.5	7.1 ± 0.5
	Albumin, g/dL	4.2 ± 0.3	4.3 ± 0.3	4.3 ± 0.4	4.3 ± 0.4	4.2 ± 0.4
	Hemoglobin, g/dL	13.6 ± 1.8	13.9 ± 1.8	13.8 ± 1.8	13.9 ± 1.7	13.8 ± 1.9
	BUN, mg/dL	20.2 ± 8.0	18.9 ± 7.2	19.5 ± 8.0	19.4 ± 8.1	19.5 ± 8.4
	Creatinine, mg/dL	1.10 ± 0.41	1.05 ± 0.48	1.07 ± 0.48	1.05 ± 0.49	1.10 ± 0.57
	eGFR, ml/ min/1.73 m^2	57.1 ± 21.2	60.3 ± 21.1	59.0 ± 22.1	55.8 ± 22.1	59.1 ± 21.7
	HbA1c, %	6.0 ± 1.1	6.1 ± 1.5	6.1 ± 1.1	6.2 ± 1.3	6.3 ± 1.2

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(1), 912-921 | ISSN:2251-6727

	Uric acid, mg/ dL	6.2 ± 1.8	6.2 ± 1.2	6.1 ± 1.5	6.1 ± 1.6	6.0 ± 1.5
	TC, mg/dL	193 ± 34	195 ± 34	197 ± 37	201 ± 37	195 ± 357
	HDL-C, mg/dL	55 ± 16	53 ± 15	54 ± 15	56 ± 17	53 ± 14
	Non-HDL-C, mg/Dl	139 ± 34	145 ± 36	147 ± 37	149 ± 39	145 ± 36
	Triglyceride, mg/dL	151 ± 98	170 ± 131	172 ± 118	176 ± 171	176 ± 136

Figure: 1: HbA1c, %

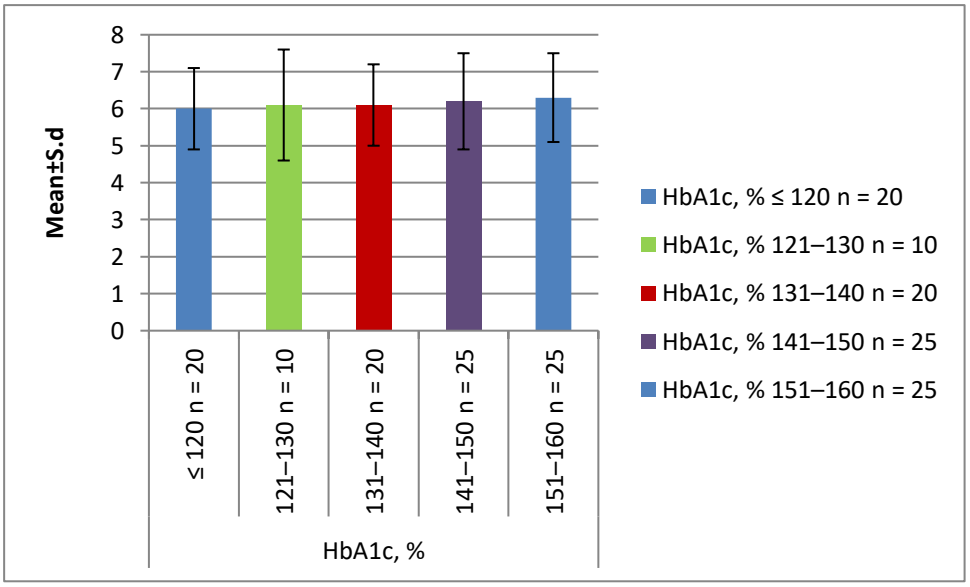
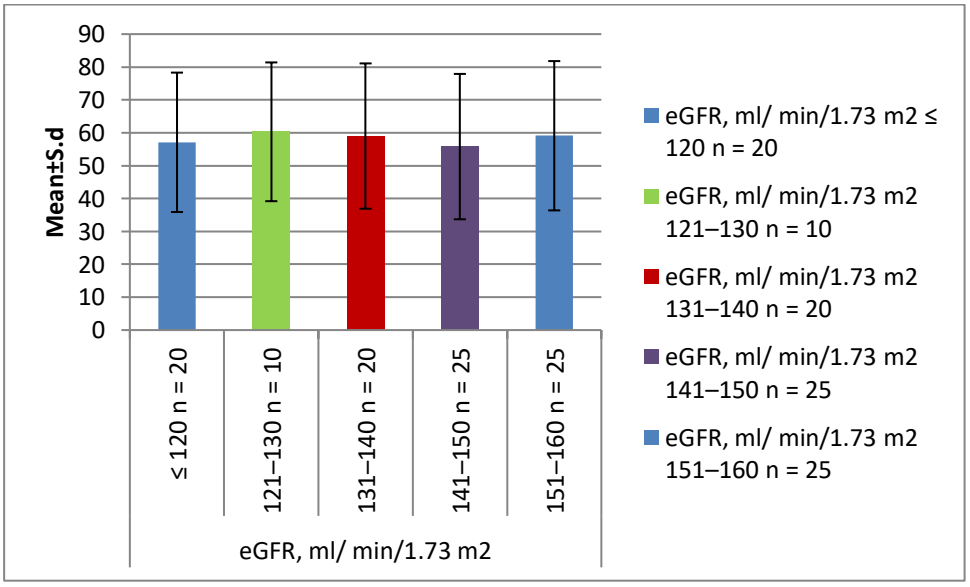


Figure: 2: eGFR, ml/ min/1.73 m^2



Systolic blood pressure (SBP) groups: ≤120, 121–130, 131–140, 141–150, and 151–160 mmHg. The percentage of male participants remained relatively stable across the groups, ranging from 69.5% to 74.3%. Age was also consistent, with an average of approximately 62 years in all groups. Body mass index (BMI) showed a gradual

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(1), 912-921 | ISSN:2251-6727

increase from 25.4 ± 3.7 kg/m² in the ≤120 group to 26.1 ± 3.4 kg/m² in the 151–160 group, though the differences were modest. Similarly, abdominal girth increased progressively from 87.4 ± 10.7 cm in the ≤120 group to 91.2 ± 9.8 cm in the 141–150 group, before slightly decreasing to 90.6 ± 9.1 cm in the 151–160 group. These findings suggest that while there are small variations in BMI and abdominal girth across the different BP categories, the differences are not large enough to indicate a strong relationship between these factors and blood pressure in this cohort.

Systolic blood pressure (SBP) groups (≤120, 121–130, 131–140, 141–150, and 151–160 mmHg) showed minimal variation. Total protein levels were stable across all groups, ranging from 7.1 ± 0.5 g/dL to 7.3 ± 0.5 g/dL, while albumin levels were similarly consistent at 4.2 ± 0.3 g/dL to 4.3 ± 0.4 g/dL. Hemoglobin levels showed little difference, with values ranging from 13.6 ± 1.8 g/dL to 13.9 ± 1.9 g/dL. Blood urea nitrogen (BUN) was comparable across groups, ranging from 18.9 ± 7.2 mg/dL to 20.2 ± 8.0 mg/dL. Creatinine levels were stable, with values between 1.05 ± 0.48 mg/dL and 1.10 ± 0.57 mg/dL. Estimated glomerular filtration rate (eGFR) was also similar, ranging from 55.8 ± 22.1 to 60.3 ± 21.1 ml/min/1.73 m². HbA1c levels ranged from 6.0 ± 1.1% to 6.3 ± 1.2%, suggesting no significant variation in glucose control across groups. Uric acid levels remained fairly constant, ranging from 6.0 ± 1.5 mg/dL to 6.2 ± 1.8 mg/dL. Lipid profiles, including total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), non-HDL-C, and triglycerides, showed only slight variations across the groups, with total cholesterol ranging from 193 ± 34 mg/dL to 201 ± 37 mg/dL and triglycerides between 151 ± 98 mg/dL and 176 ± 171 mg/dL.

DISCUSSION

The relationship between systolic blood pressure (SBP) and various health parameters, particularly kidney function, has garnered substantial interest in recent research. This analysis examines the trends and associations between SBP levels, demographic factors, body mass index (BMI), laboratory results, and indicators of kidney health in a sample of middle-aged individuals. The average age of participants was between 61.9 and 62.9 years, with no significant differences in age across the various SBP groups, suggesting that all participants were similarly predisposed to hypertension and its associated risks. The findings indicate that elevated SBP, particularly levels ≥130 mmHg, can serve as an independent risk factor for chronic kidney disease (CKD) and other comorbid conditions such as obesity and metabolic disorders.

Gender Distribution and Hypertension

In terms of gender, males were found to be the most prevalent demographic across all SBP categories, with a noticeable trend between 69.5% and 74.3%. This finding aligns with existing literature that reports men having a higher prevalence of hypertension compared to women, particularly as they age. Hypertension is known to be more common in males due to a combination of genetic, lifestyle, and hormonal factors. These findings underscore the importance of monitoring blood pressure levels in middle-aged males, as they are more susceptible to the cardiovascular and renal complications associated with sustained hypertension. Although gender differences in hypertension prevalence may vary across different age groups and populations, the consistent trend observed here is in line with previous studies that have demonstrated that men are more likely than women to develop hypertension and experience its long-term consequences.

Age and Hypertension

Georgianos PI et al [23] (2023) showed that hypertension is very common and remains often poorly controlled in patients with chronic kidney disease (CKD). Accurate blood pressure (BP) measurement is the essential first step in the diagnosis and management of hypertension.

Bouarich H et al [24] (2021) found that individuals over 65 years are the fastest expanding population throughout the world, due to the increase in human life expectancy. This growing geriatric population, with increasingly associated chronic diseases, has relevant medical, social, and economic impact. Aging is characterized by progressive structural and functional changes in the kidney and in the cardiovascular system, leading to decline in renal function and hypertension.

Carey RM et al [25] (2022) observed that hypertension, defined as persistent systolic blood pressure (SBP) at least 130 mm Hg or diastolic BP (DBP) at least 80 mm Hg, affects approximately 116 million adults in the US and more than 1 billion adults worldwide. Hypertension is associated with increased

risk of cardiovascular disease (CVD) events (coronary heart disease, heart failure, and stroke) and death.

The age distribution of the study participants is another critical factor in understanding the observed relationships. With a mean age of around 62 years, the cohort is representative of the middle-aged and older adult population. This age range is particularly relevant since individuals over the age of 50 are more likely to experience a rise in blood pressure due to age-related changes in vascular stiffness and renal function. The pathophysiology of hypertension in older adults involves a complex interplay between changes in the renin-angiotensin-aldosterone system (RAAS), increased sympathetic nervous system activity, and the gradual loss of arterial elasticity. Additionally, as individuals age, kidney function tends to decline gradually, which may exacerbate the adverse effects of high blood pressure. In this study, the mean age of the participants correlates with an increased risk of hypertension-related kidney damage, which emphasizes the importance of early detection and management of high blood pressure to prevent renal impairment.

Blood Pressure, Body Mass Index, and Obesity

One of the most notable trends observed across the different SBP groups is the increase in body mass index (BMI) as systolic blood pressure rises. The ≤120 mmHg group had the lowest BMI at 25.4 ± 3.7, while the 151-160 mmHg group had the highest BMI at 26.1 ± 3.4. Although these changes in BMI are not statistically significant, they do suggest a potential association between increased SBP and higher BMI, which may reflect the underlying connection between obesity and hypertension. Obesity is a well-established risk factor for both hypertension and renal disease, and the gradual increase in BMI as SBP rises could be indicative of this relationship.

Obesity can increase the renal workload due to heightened systemic vascular resistance, activation of RAAS, and the inflammatory processes associated with excess adiposity. These factors contribute to kidney injury and are likely exacerbated in individuals with both obesity and high blood pressure. The renin-angiotensin-aldosterone system, in particular, plays a pivotal role in regulating blood pressure and sodium balance. In the context of obesity, dysregulation of this system contributes to increased sodium retention, vasoconstriction, and ultimately, elevated blood pressure, all of which place added stress on the kidneys. Furthermore, elevated levels of pro-inflammatory cytokines, such as TNF-α and IL-6, are often observed in obese individuals and contribute to glomerular injury and fibrosis, further increasing the risk for kidney disease.

Abdominal Obesity and Kidney Function

Georgianos PI et al [23] (2023) showed that hypertension is very common and remains often poorly controlled in patients with chronic kidney disease (CKD).

The study also found a gradual increase in abdominal girth as SBP increased, with values ranging from 87.4 cm in the ≤120 mmHg group to 91.2 cm in the 141-150 mmHg group. Abdominal obesity, characterized by an increased waist circumference and visceral fat accumulation, is a known risk factor for both metabolic and cardiovascular disorders, including hypertension. Central obesity, in particular, is associated with a heightened risk of developing insulin resistance, dyslipidemia, and hypertension, all of which are recognized risk factors for CKD. Visceral fat, unlike subcutaneous fat, is metabolically active and contributes to systemic inflammation, oxidative stress, and endothelial dysfunction, all of which play critical roles in the pathogenesis of renal disease.

The increase in abdominal girth observed in this cohort may reflect the growing prevalence of central obesity among individuals with elevated SBP, and this may serve as an early indicator of increased risk for kidney dysfunction. The association between abdominal obesity and kidney disease is well-documented, with studies showing that increased visceral fat correlates with a greater risk of glomerular injury and decline in renal function over time. Furthermore, abdominal obesity is also linked to increased levels of inflammatory markers such as C-reactive protein (CRP), which can further contribute to kidney damage and disease progression.

Laboratory Results and Renal Health

The data presented in the table above provides valuable insight into the laboratory values across varying baseline systolic blood pressure (SBP) groups in a cohort of individuals. The baseline SBP was categorized into five groups: ≤ 120 mmHg, 121-130

mmHg, 131-140 mmHg, 141-150 mmHg, and 151-160 mmHg, with the number of participants in each group varying (n = 20, 10, 20, 25, 25, respectively). Several laboratory parameters were assessed, including total protein, albumin, hemoglobin, BUN, creatinine, eGFR, HbA1c, uric acid, and lipid profiles (total cholesterol, HDL-C, non-HDL-C, and triglycerides).

Starting with the protein markers, total protein and albumin levels remained relatively consistent across the groups, with minor fluctuations. Total protein ranged from 7.1 to 7.3 g/dL, and albumin levels remained between 4.2 and 4.3 g/dL. Hemoglobin levels were also stable across the SBP groups, with slight variations but no clear trend, ranging from 13.6 g/dL to 13.9 g/dL. These results suggest that baseline SBP within the studied range does not significantly impact these specific protein and hematologic parameters.

In terms of renal function, BUN and creatinine levels were consistent across all SBP groups, with BUN ranging from 18.9 to 20.2 mg/dL and creatinine levels from 1.05 to 1.10 mg/dL. Additionally, estimated glomerular filtration rate (eGFR), which is a crucial marker of kidney function, was also relatively stable across SBP groups, fluctuating between 55.8 and 60.3 ml/min/1.73 m^2. This indicates that baseline SBP did not appear to have a significant impact on kidney function in this cohort.

For glycemic control, HbA1c levels slightly increased with rising SBP, from 6.0% in the lowest SBP group (≤ 120 mmHg) to 6.3% in the highest SBP group (151-160 mmHg). While the increase is modest, it could suggest a potential association between higher SBP and slightly worsened glycemic control, although further investigation would be needed to establish a clear cause-and-effect relationship.

Uric acid levels showed only minimal variation across the groups, with levels ranging from 6.0 to 6.2 mg/dL, indicating that SBP does not significantly affect uric acid levels in this sample.

Regarding lipid profile, total cholesterol (TC) levels showed a gradual increase from 193 mg/dL in the lowest SBP group to 201 mg/dL in the highest group, though the differences were modest. HDL-C levels were consistent across all groups, fluctuating between 53 and 56 mg/dL, which suggests that higher SBP does not notably alter HDL-C concentrations. Non-HDL-C, however, showed a gradual increase from 139 mg/dL in the lowest SBP group to 149 mg/dL in the highest group, potentially indicating a trend towards higher levels of atherogenic cholesterol in individuals with higher SBP. Triglyceride levels were somewhat elevated across all groups, with values ranging from 151 mg/dL to 176 mg/dL, though no clear relationship to SBP was observed.

In summary, while several laboratory parameters such as total protein, albumin, and hemoglobin were stable across baseline SBP groups, subtle trends were observed in markers of kidney function, glycemic control, and lipid profiles, with slight increases in BUN, creatinine, HbA1c, and non-HDL-C as SBP increased. These observations may suggest that higher SBP is associated with modest changes in metabolic and renal parameters, though more extensive research would be necessary to determine the clinical significance of these findings. Additionally, the lack of major variations in key laboratory parameters may indicate that the cohort's baseline SBP, within the studied range, is not a strong determinant of these particular health markers. Further investigation, particularly with larger sample sizes and longitudinal data, would be needed to explore the underlying relationships between blood pressure and these laboratory measures.

CONCLUSION

In conclusion, renal fibrosis, endothelial dysfunction, and elevated glomerular pressure are some of the ways that hypertension damages the kidneys and has a substantial effect on renal function. Chronic kidney disease (CKD) can develop as a result of high blood pressure's gradual reduction of glomerular filtration rate (GFR). In order to stop or reduce renal development, early detection and efficient care of hypertension are essential. Protecting kidney function requires a variety of treatment strategies, such as changing one's lifestyle, using antihypertensive drugs (ACE inhibitors, ARBs, and diuretics), and managing other risk factors including diabetes. To maximize results for patients with kidney disease linked to hypertension, a multidisciplinary strategy combining cardiologists and nephrologists is crucial.

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(1), 912-921 | ISSN:2251-6727

REFERENCE

1. Stanaway JD et al. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990–2017: A systematic analysis for the Global Burden of Disease Stu. Lancet 392, 1923–1994 (2018).
2. Hill, N. R. et al. Global prevalence of chronic kidney disease—A systematic review and meta-analysis. PLoS ONE 11, e0158765 (2016).
3. Go, A. S., Chertow, G. M., Fan, D., McCulloch, C. E. & Hsu, C.-Y. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. N. Engl. J. Med. 351, 1296–1305
4. Kostapanos MS, Milionis HJ, Elisaf MS. Current role of statins in the treatment of essential hypertension. Expert Opin Pharmacother. 2010;11:2635–2650. doi: 10.1517/14656566.2010.491512.
5. Fenech G, Safar M, Blacher J. Orthostatic hypotension: marker of severity and management of antihypertensive treatment. Presse Med. 2012;41:1116–1121. doi: 10.1016/j.lpm.2012.02.008
6. Liu RM, Vayalil PK, Ballinger C, Dickinson DA, Huang WT, Wang S, Kavanagh TJ, Matthews QL, Postlethwait EM. Transforming growth factor β suppresses glutamate–cysteine ligase gene expression and induces oxidative stress in a lung fibrosis model. Free Radical Biology and Medicine. 2012 Aug 1;53(3):554-63.
7. Hayes JD, Dinkova-Kostova AT. The Nrf2 regulatory network provides an interface between redox and intermediary metabolism. Trends in biochemical sciences. 2014 Apr 1;39(4):199-218.
8. S.K. Kim et al. Negative correlation of urinary miR-199a-3p level with ameliorating effects of sarpogrelate and cilostazol in hypertensive diabetic nephropathy Biochem. Pharmacol. (2021)
9. Kim HJ, Sato T, Rodríguez-Iturbe B, Vaziri ND. Role of intrarenal angiotensin system activation, oxidative stress, inflammation, and impaired nuclear factor-erythroid-2-related factor 2 activity in the progression of focal glomerulosclerosis. Journal of Pharmacology and Experimental Therapeutics. 2011 Jun 1;337(3):583-90.
10. Hassanein EH, Ibrahim IM, Abd-Alhameed EK, Sharawi ZW, Jaber FA, Althagafy HS. Nrf2/HO-1 as a therapeutic target in renal fibrosis. Life Sciences. 2023 Dec 1;334:122209.
11. Liu Y, Uruno A, Saito R, Matsukawa N, Hishinuma E, Saigusa D, Liu H, Yamamoto M. Nrf2 deficiency deteriorates diabetic kidney disease in Akita model mice. Redox Biology. 2022 Dec 1;58:102525.
12. Fuchs FD, Whelton PK. High blood pressure and cardiovascular disease. Hypertension. 2020;75:285–292. doi: 10.1161/HYPERTENSIONAHA.119.14240
13. Zhou B, Carrillo-Larco RM, Danaei G, Riley LM, Paciorek CJ, Stevens GA, Gregg EW, Bennett JE, Solomon B, Singleton RK, Sophiea MK. Worldwide trends in hypertension prevalence and progress in treatment and control from 1990 to 2019: a pooled analysis of 1201 population-representative studies with 104 million participants. The Lancet. 2021 Sep 11;398(10304):957-80.
14. Cheung AK, Chang TI, Cushman WC, Furth SL, Hou FF, Ix JH, Knoll GA, Muntner P, Pecoits-Filho R, Sarnak MJ, Tobe SW. KDIGO 2021 clinical practice guideline for the management of blood pressure in chronic kidney disease. Kidney International. 2021 Mar 1;99(3):S1-87.
15. Inker LA, Eneanya ND, Coresh J, Tighiouart H, Wang D, Sang Y, Crews DC, Doria A, Estrella MM, Froissart M, Grams ME. New creatinine-and cystatin C–based equations to estimate GFR without race. New England Journal of Medicine. 2021 Nov 4;385(19):1737-49.
16. Delanaye P, Schaeffner E, Cozzolino M, Langlois M, Plebani M, Ozben T, Cavalier E. The new, race-free, Chronic Kidney Disease Epidemiology Consortium (CKD-EPI) equation to estimate glomerular filtration rate: is it applicable in Europe? A position statement by the European Federation of Clinical Chemistry and Laboratory Medicine (EFLM).

Clinical Chemistry and Laboratory Medicine (CCLM). 2023 Jan 27;61(1):44-7.

17. Kovesdy CP. Epidemiology of chronic kidney disease: an update 2022. Kidney international supplements. 2022 Apr 1;12(1):7-11.
18. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. Nature Reviews Nephrology. 2020 Apr;16(4):223-37.
19. Tsao CW, Aday AW, Almarzooq ZI, Anderson CA, Arora P, Avery CL, Baker-Smith CM, Beaton AZ, Boehme AK, Buxton AE, Commodore-Mensah Y. Heart disease and stroke statistics—2023 update: a report from the American Heart Association. Circulation. 2023 Feb 21;147(8):e93-621.
20. Stergiou GS, Palatini P, Parati G, O'Brien E, Januszewicz A, Lurbe E, Persu A, Mancia G, Kreutz R. 2021 European Society of Hypertension practice guidelines for office and out-of-office blood pressure measurement. Journal of hypertension. 2021 Jul 1;39(7):1293-302.
21. Lee H, Kwon SH, Jeon JS, Noh H, Han DC, Kim H. Association between blood pressure and the risk of chronic kidney disease in treatment-naïve hypertensive patients. Kidney research and clinical practice. 2021 Nov 17;41(1):31.
22. Pisano A, Mallamaci F, D'arrigo G, Bolignano D, Wuerzner G, Ortiz A, Burnier M, Kanaan N, Sarafidis P, Persu A, Ferro CJ. Assessment of hypertension in kidney transplantation by ambulatory blood pressure monitoring: a systematic review and meta-analysis. Clinical Kidney Journal. 2022 Jan;15(1):31-42.
23. Georgianos PI, Agarwal R. Hypertension in chronic kidney disease—treatment standard 2023. Nephrology Dialysis Transplantation. 2023 Dec;38(12):2694-703.
24. Bouarich H, Guillen AC, Puyol DR. Kidney and hypertension in older adults. Medicina Clínica (English Edition). 2021 Aug 27;157(4):178-84.
25. Carey RM, Moran AE, Whelton PK. Treatment of hypertension: a review. Jama. 2022 Nov 8;328(18):1849-61.