Available online on 30.07.2022 at http://jddtonline.info

Journal of Drug Delivery and Therapeutics

Open Access to Pharmaceutical and Medical Research

Open Access Full Text Article

Research Article

Impact of Hypertension on Chronic Kidney Disease Patients

Trini Suryowati*, Fajar Lamhot Gultom, Ester Grace Nikti Muntu

Medical Faculty of Universitas Kristen Indonesia, Jakarta, Indonesia

Article Info:

Article History:

Received 09 June 2022
Reviewed 14 July 2022
Accepted 22 July 2022
Published 30 July 2022

Cite this article as:

Suryowati T, Gultom FL, Muntu EGN, Impact of Hypertension on Chronic Kidney Disease Patients, Journal of Drug Delivery and Therapeutics. 2022; 12(4):125-132

DOI: http://dx.doi.org/10.22270/jddt.v12i4.5466

*Address for Correspondence:

Trini Suryowati, Medical Faculty of Universitas Kristen Indonesia, Jakarta, Indonesia

Abstract

Hypertension is a progressive cardiovascular syndrome arising from complex and interrelated etiologies, characterized by an increase in systolic blood pressure higher than or equal to 140 mmHg or diastolic blood pressure higher than or equal to 90 mmHg. Hypertension is one of the main risk factors for chronic kidney disease. This research aims to know the impact of hypertension on chronic kidney disease patients in UKI General Hospital from 2019 – to 2020. The design of this research was retrospective. The sample used was 58 patients following inclusion criteria. The instruments used are medical records of hypertension patients in UKI General Hospital from 2019 – to 2020. The results showed that 23 (39.7%) patients with hypertension were male, 35 (60.3%) patients with hypertension were female, and 15 (25.9%) patients with hypertension aged 46 – 55 years and 56 – 65 years. The chi-square correlation test results between hypertension and chronic kidney disease obtained $p > 0.05$. It was concluded that in UKI General Hospital during 2019 – 2020, hypertension has no significant impact on chronic kidney disease.

Keywords: Cardiovascular Syndrome, Chronic Kidney Disease, Hypertension

INTRODUCTION

Hypertension is an increase in systolic blood pressure of more than 140 mmHg and diastolic blood pressure of more than 90 mmHg on two measurements with an interval of five minutes in a state of sufficient rest. Increased blood pressure in the long term is a significant factor in causing damage to the kidneys, heart, and brain if it is not detected early and gets adequate treatment.[1]

Hypertension is a disease with a relatively high prevalence in world health problems. The World Health Organization (WHO) 2015 stated that around 1.13 billion people worldwide suffer from hypertension, which means that every 1 in 3 people in the world is diagnosed with hypertension, and only 36.8% of them are taking medication. The number of people with hypertension in the world continues to increase every year; it is estimated that by 2025 there will be 1.5 billion people affected by hypertension, and every year, 9.4 million people die from hypertension and its complications. The Institute for Health Metrics and Evaluation (IHME) in 2017 stated that of the 53.3 million deaths in the world, 33.1% were caused by cardiovascular, 16.7% due to cancer, 6% due to diabetes, and endocrine disorders, and 4.8 % caused by lower respiratory tract infections. The Institute for Health Metrics and Evaluation also stated that from a total of 1.7 million deaths in Indonesia, the most risk factors that caused death were hypertension 23.7%, hyperglycemia 18.4%, smoking 12.7%, and obesity 7.7%. In Indonesia in 2016, the total death toll was 1.5 million, with the most common causes of death being cardiovascular disease 36.9%, cancer 9.7%, DM and endocrine 9.3%, and tuberculosis 5.9%. Data from the Health Social Security Administration (BPJS) states that the cost of hypertension services has increased yearly, in 2016 to 2.8 trillion rupiahs, in 2017 and 2018 to 3 trillion rupiahs.[2]

Hypertension is a significant risk factor for heart disease, congestive heart failure, stroke, visual impairment, and kidney disease. Kidney function to remove metabolic wastes and maintain fluid and electrolyte balance or body salt will be partially or completely disrupted when hypertension occurs in the long term.[3,4]

Systematic Review and Meta-Analysis – Global Prevalence of Chronic Kidney Disease in 2016 stated that the global prevalence of chronic kidney disease was on average 13.4%, the prevalence of chronic kidney disease with diabetes was 0.16%, the prevalence of chronic kidney disease and hypertension was 0.15%. Basic Health Research in 2018 states that in Indonesia, the prevalence of chronic kidney disease is on average 3.8‰, the prevalence of chronic kidney disease with diabetes is 8.5%, the prevalence of chronic kidney disease with hypertension is 34.1%, and the prevalence of kidney disease chronic with obesity by 21.8%. Based on these data, it can be concluded that hypertension is the second most common cause of chronic kidney disease after diabetes.[5,6]

The 2013 Basic Health Research stated that the prevalence of chronic kidney disease increases with age, a sharp increase in the 35-44 year age group compared to the 25-34 year age group. In Indonesia, kidney disease care is ranked as the second largest financing provider by the Social Security Administration (BPJS) for health after heart disease.[7]

It is essential to evaluate the impact of hypertension on patients with chronic kidney disease, hoping that the public can understand its impact on kidney health and that hospitals can provide full service for their patients. Based on the description above, the author conducted a study entitled "The Impact of Hypertension on Chronic Kidney Disease Patients at UKI General Hospital for 2019 - 2020."

Based on the description of the background above, a problem is formulated, namely: "What is the impact of hypertension on chronic kidney disease patients at UKI General Hospital for the period 2019 - 2020?" with the aim of the study, namely to determine the impact of hypertension on patients with chronic kidney disease at UKI General Hospital for the period 2019 – 2020.

LITERATURE REVIEW

Hypertension is a progressive cardiovascular syndrome arising from various complex and interrelated etiologies. Early syndrome markers often appear before blood pressure is persistently elevated; therefore, hypertension cannot be classified solely by blood pressure thresholds. Increased blood pressure is strongly associated with functional and structural abnormalities of the heart and blood vessels that damage the heart, kidneys, brain, blood vessels, and other organs that can cause early morbidity and death.[8]

Hypertension is an increase in systolic blood pressure of more than 140 mmHg or diastolic blood pressure of more than or equal to 90 mmHg in 2 measurements with a minimum examination distance of 10 minutes.[9] Hypertension is an increase in blood pressure in the arteries. On blood pressure examination, two numbers will be obtained, a higher number is obtained when the heart contracts or is called systolic, and a lower number is obtained when the heart relaxes or is called diastolic. Blood pressure is said to be high if, when sitting, the systolic pressure reaches 140 mmHg or more, the diastolic pressure reaches 90 mmHg or more, or there is an increase in both.[10]

The World Health Organization (WHO) 2015 stated that in the world, nearly 1 billion people have high blood pressure. By 2020, it is estimated that 1.56 billion adults will be living with hypertension. Every year hypertension kills nearly 8 billion people worldwide and 1.5 million people in the East-South Asia region. It is estimated that one-third of adults in South-East Asia suffer from hypertension.[11]

The American Heart Association (AHA) states that the American population aged over 20 years who suffer from hypertension is 74.5 million people, but about 90-95% of cases have no known cause. The Health Research and Development Agency (BalitBangKes), through Basic Health Research (Riskesdas) data in 2018, stated that hypertension sufferers in Indonesia were 34.1%. This number has increased compared to the previous figure in 2013, which was 25.8%.[12;13] The prevalence of hypertension increases with changes in people's lifestyles, such as smoking, obesity, physical activity, and psychosocial stress. Hypertension has become a public health problem and will become a bigger problem if it is not addressed early.[14] Classification of hypertension based on the etiology is divided into:

Primary Hypertension (Essential hypertension) is a combination of genetic and environmental factors that affect renal and vascular function. The cause of primary hypertension is a deficiency in the ability of the kidneys to excrete sodium, which increases extracellular fluid volume and cardiac output, resulting in increased blood flow to tissues. Increased blood flow to tissues causes arteriolar constriction and increases peripheral vascular resistance and blood pressure.[15]

Secondary Hypertension (Non-essential hypertension) is caused by disease in other organs resulting in increased peripheral vascular resistance and cardiac output. The focus on secondary hypertension is kidney disease or excess levels of the hormone aldosterone and cortisol. These two hormones stimulate sodium and water retention, which can increase blood volume and pressure.[15] The classification of hypertension according to the Joint National Committee VII (JNC VII) is as follows.

Table 1: Classification of Hypertension Degrees (Joint National Committee VII, 2003)[16]

Blood Pressure Classification	Systolic (mmHg)	Diastolic (mmHg)
Normal	< 120	< 80
Pre-hypertension	120-139	80-89
Stage 1 hypertension	140-159	90-99
Stage 2 hypertension	≥ 160	≥ 100

The risk factors for hypertension are genetics, age, gender, obesity, stress, lack of exercise, salt intake patterns, and smoking habits.[3] Hypertension usually does not cause typical symptoms. For this reason, regular blood pressure checks and additional examinations of the kidneys and blood vessels should be carried out. Severe or chronic and untreated hypertension can cause damage to the brain, eyes, heart, and kidneys, causing symptoms such as headache, fatigue, nausea, vomiting, shortness of breath, restlessness, and blurred vision. Patients with severe hypertension can also experience decreased consciousness and coma due to brain swelling and hypertensive encephalopathy, which requires immediate treatment. The symptoms of hypertension are hazardous if ignored.[10]

The mechanism of hypertension is through the formation of angiotensin II from angiotensin I by the angiotensin I converting enzyme (ACE). ACE plays an essential physiological role in regulating blood pressure. Blood contains angiotensinogen, which is produced in the liver. Renin produced by the kidneys will be converted into angiotensin I. ACE in the lungs converts angiotensin I to angiotensin II. Angiotensin II is essential in raising blood pressure through two main actions. The first action is to increase antidiuretic hormone secretion (ADH) and thirst. The second action stimulates aldosterone secretion from the adrenal cortex.[3]

Receptors that receive changes in blood pressure are baroreceptor reflexes found in the carotid sinus and aortic arch. With genetic disorders and environmental risk factors in hypertension, neurohormonal disorders occur, namely the central nervous system and the renin-angiotensin-aldosterone system, as well as inflammation and insulin resistance. Insulin resistance and neurohormonal disturbances cause systemic vasoconstriction and increased peripheral resistance. Inflammation causes kidney disorders accompanied by disorders of the renin-angiotensin-aldosterone (RAA) system, which causes kidney salt and water retention, increasing blood volume. Increased peripheral resistance and blood volume are the two leading causes of hypertension.[17]

Hypertension and kidney disease are closely related because hypertension is one of the main triggering factors for kidney disease, and conversely, when kidney function is impaired, blood pressure will increase and can cause hypertension. Damage to the kidneys in certain parts, especially the cortex or outer layer of the kidneys, will stimulate the production of the hormone renin, which will increase blood pressure, resulting in persistent hypertension. When the kidneys are damaged,

the excretion of water and salt will be disrupted and cause the contents of the blood vessel cavity to increase, resulting in hypertension. Uncontrolled hypertension will weaken and narrow the blood vessels that supply the kidneys and prevent the kidneys from functioning normally.[18]

Hypertension can trigger barotrauma stimulation of the glomerular capillaries and increase capillary pressure. If it lasts long, it can cause of chronic hypertension resulting in narrowing of preglomerular arteries with reduction in glomerular blood flow or glomerulosclerosis. This condition can cause chronic hypoxia. This hypoxic state increases the need for oxygen metabolism, thereby triggering the release of vasoactive substances such as endothelin, angiotensin, and norepinephrine in endothelial cells of local blood vessels, causing vasoconstriction. Activation of the renin-angiotensin system can also cause oxidative stress, increasing oxygen demand and exacerbating hypoxia, and causing vasoconstriction. Oxidative stress reduces the efficiency of sodium transport and damages DNA, lipids, and proteins, resulting in tubulointerstitial fibrosis and exacerbating kidney damage.[19]

The bean-shaped kidney is located retroperitoneally in the posterior abdominal region. The kidneys are located at approximately the level of the XII thoracic vertebra superiorly and the III lumbar vertebrae inferiorly. The right kidney is lower than the left kidney because of its position against the liver. The left kidney is more extended and slimmer than the right kidney and is closer to the body's midline. Each kidney is 6 – 7.5 cm long and 1.5 – 2.5 cm thick. The weight varies between 120 – 170 grams, or approximately 0.4% of body weight.[20]

The kidneys are enveloped and associated with fascia and a particular layer of fat. Outside the renal capsule is an accumulation of extraperitoneal fat, the corpus adipose perirenal, which surrounds the kidney. The medial portion of the anterior lamina of the renal fascia continues above the vasa at the renal hilum and joins the connective tissue connecting the abdominal aorta and inferior vena cava. The posterior lamina of the renal fascia passes medially between the kidney and the fascia lining the quadrates lumborum muscle to merge with the fascia lining the psoas major muscle. The inferior lamina of the anterior and posterior lamina of the renal fascia encloses the ureter.[21]

Renal blood flow comes from the renal artery, a branch of the abdominal aorta, while the renal vein is a branch of the inferior vena cava. The renal artery system is the end arteries or arteries that do not have anastomoses with other arterial branches so that if there is damage to one of the arterial branches, it will result in ischemia or necrosis. The renal innervation originates from the renal sympathetic plexus and is distributed along the branches of the renal artery and vein.[22]

Kidneys perform a vital function as a regulator of blood volume and chemical composition in the body by selectively excreting solutes and water. The kidney maintains the body's H2O balance, maintains plasma volume, maintains acid-base balance, and excretes metabolic waste products and foreign compounds.[23] Signs of kidney damage include Albuminuria (Albumin Excretion Rate or AER 30 mg/24 hours and Albumin-to-creatinine Ratio or ACR 30 mg/g), urinary sediment abnormalities, electrolyte abnormalities due to tubular disorders, histologically detected abnormalities, structural abnormalities detected by medical examination, and a history of previous kidney transplantation. Decreased Glomerular Filtration Rate (GFR) < 60 mL/min/1.73 m2.[24,25]

Chronic kidney disease is a pathophysiological process with various etiologies, can cause a progressive decline in kidney function and generally end with kidney failure. Kidney failure is an irreversible decline in kidney function until it cannot work and requires permanent kidney replacement therapy, such as dialysis or kidney transplantation. In conditions of kidney failure, the kidneys lose the ability to filter the body's electrolytes and maintain the balance of fluids and body chemicals such as sodium and potassium in the blood or urine production.[26]

Kidney International Supplements in 2013 classified chronic kidney disease based on the cause, Glomerular Filtration Rate (GFR) category, and the category of albuminuria. The cause of chronic kidney disease is based on the presence or absence of systemic disease, and the location within the kidney observed or presumed on anatomic pathological findings.[27]

Table 2: Categories of Glomerular Filtration Rate (GFR) (UMHS Chronic Kidney Disease Guideline, 2019)[28]

GFR Category	GFR (mL/min/1.73 m²)	Description
G1	> 90	Normal or high
G2	60 – 89	Light drop*
G3a	45 – 59	Mild to moderate decrease
G3b	30 – 44	Moderate to severe loss
G4	15 – 29	Weight loss
G5	< 15	Kidney failure

* Relative to young adulthood

If there is no kidney damage, GFR categories G1 or G2 do not meet the criteria for CKD.

Table 3: Albuminuria category (UMHS Chronic Kidney Disease Guideline, 2019)[29]

Albuminuria Category	*Albumin Excretion Rate* (AER) (mg/24 hours)	*Albumin-to-creatinine Ratio*(ACR) (Equivalent approx.)		Description
		(mg/mmol)	(mg/g)	
A1	< 30	< 3	< 30	Normal to mild improvement
A2	30 – 300	3 – 30	30 – 300	Moderate improvement *
A3	>300	> 30	> 300	Weight gain**

*Relatively young adults

**Includes nephrotic syndrome (Albumin excretion > 2,200 mg/24 hr [ACR > 2220 mg/g; > 220 mg/mmol]). Chronic kidney disease can be caused by several things, including: Glomerulonephritis, Chronic Pyelonephritis, kidney stones, Polycystic Kidney Disease, Diabetic Nephropathy, Hypertensive Nephrosclerosis, Diabetes Mellitus, Hypertension.[30,31]

The Association of Indonesian Internal Medicine Specialists in 2006 explained that the pathophysiology of chronic kidney disease initially depends on the underlying disease, but in subsequent developments, the process is more or less the same. Reduction of kidney mass results in structural and functional hypertrophy of the remaining nephrons (surviving nephrons) as a compensatory effort, which is mediated by vasoactive molecules such as cytokine and growth factors. It will result in hyperfiltration, increased capillary pressure, and glomerular blood flow. This adaptation process is brief, followed by a progressive decline in nephron function, even

though the underlying disease is no longer active. Increased intrarenal renin-angiotensin-aldosterone axis activity also contributes to hyperfiltration, sclerosis, and progression. Long-term activation of the renin-angiotensin-aldosterone axis is partly mediated by transforming growth factor (TGF-β). Other factors contributing to chronic kidney disease progression are albuminuria, hypertension, hyperglycemia, and dyslipidemia. Interindividual variability exists for glomerular and tubulointerstitial sclerosis and fibrosis.[32]

Depending on the disease's course, various causes such as infection, vascular, toxic substances, and urinary tract obstruction can cause nephron damage, resulting in a decrease in the glomerular filtration rate and chronic kidney disease. In this condition, the kidneys experience impaired excretory and non-excretory functions, decreased renal function, and kidney products. The end of protein metabolism accumulates in the blood, and uremia occurs, which can affect the body's system. In the early stages of chronic kidney disease, renal reserve power is lost, and the glomerular filtration rate is still average or increased. Then there is a progressive decline in renal nephron function, characterized by increased serum urea and creatinine levels. In the glomerular filtration rate of 60%, there are no complaints or asymptomatic symptoms, but there has been an increase in serum urea and creatinine levels. When the glomerular filtration rate is 30%, complaints such as nocturia, weakness, nausea, decreased appetite, and weight loss occurs. If the glomerular filtration rate is less than 30%, signs and symptoms of uremia will be seen, such as anemia, increased blood pressure, impaired phosphorus and calcium metabolism, pruritus, nausea, vomiting, and other symptoms. In this situation, it is easy for infections such as urinary tract infections, respiratory infections, and gastrointestinal infections to occur. In addition to the incidence of infection, water balance disorders such as hypovolemia or hypervolemia also occur, and disturbances in sodium and potassium electrolyte balance. In a condition with less than 15% glomerular filtration rate, more severe symptoms and complications occur, requiring renal replacement therapy, such as dialysis or kidney transplantation. This condition is referred to as the stage of renal failure.[33,34]

The prognosis of chronic kidney disease can be predicted based on the classification category, among others, by determining the cause, Glomerular Filtration Rate (GFR), and the category of albuminuria. Kidney Disease Improving Global Outcomes in 2012 stated the prognosis of chronic kidney disease based on the Glomerular Filtration Rate (GFR) and albuminuria categories.

Table 4: Prognosis of CKD (Kidney Disease Improving Global Outcomes, 2012)[35]

CKD prognosis is based on the category of Glomerular Filtration Rate (GFR) and albuminuria (KDIGO, 2012)		Description and range of persistent albuminuria		
		A1	A2	A3
Description and range of GFR figures (mL/min/1.73m^2)	G1			
	G2			
	G3a			
	G3b			
	G4			
	G5			

Information:

Green: low risk (If there are no other markers of kidney disease, not CKD)
Yellow color: moderately increased risk Orange color: high risk Red color: very high risk

RESEARCH METHOD

The research design used is a retrospective study to see the impact of hypertension on patients with chronic kidney disease at UKI General Hospital for 2019 – 2020. The study was carried out at UKI General Hospital, East Jakarta. Data collection and processing were carried out from June to August 2021. The research instruments used were notebooks, stationery, computers, and medical records that recorded the impact of hypertension on patients with chronic kidney disease at UKI General Hospital for 2019 – 2020. The population was all hypertensive patients at UKI General Hospital from 2019 – to 2020. Based on data from UKI Hospital, the number of patients was 58. Data processing in this study was carried out in several stages: editing, coding, tabulating and cleaning. Data analysis in this study used a computerized program, Statistical Package for the Social Sciences (SPSS), version 25 for windows and Minitab. Data analysis was carried out in stages with univariate analysis and bivariate analysis. Univariate analysis was used to determine the frequency distribution of each research variable. Bivariate analysis was used to determine the relationship between the independent variable and the dependent variable by using the chi-square correlation statistical test.

RESULT AND DISCUSSION

The research data collection was carried out at the Medical Records section of UKI General Hospital from June 23, 2021, to June 26, 2021. The data was obtained from the medical records of hypertensive patients at the UKI General Hospital for 2019 – 2020.

Table 5: Frequency Distribution by Gender

Gender	Number	%
Male	23	39.7
Female	35	60.3
Total	58	100

Based on table 5, the results are the frequency of hypertensive patients with male sex as many as 23 patients (39.7%) and female sex as many as 35 patients (60.3%). The frequency of hypertensive patients in the female gender is more than in the male gender.

Table 6: Frequency Distribution by Age Group

Age Group	Number	%
25 – 35	5	8.6
36 – 45	10	17.2
46 – 55	15	25.9
56 – 65	15	25.9
> 65	13	22.4
Total	58	100

Based on table 6, the results obtained are the highest frequency in the age group 46-55 years and 56-65 years with a total of 15 patients (25.9%), while the lowest frequency is in the age group 25-35 years with a total of 5 patients (8, 6%).

Table 7: Frequency Distribution by BMI

BMI	Number	%
Normal	11	19
Overweight	13	22.4
Obesity I	24	41.4
Obesity II	10	17.2
Total	58	100

Based on table 7, the results obtained are the highest frequency in obese BMI I (BMI 25.0 – 29.9) with a total of 24 patients (41.4%), while the lowest frequency is in obese BMI II (BMI > 30.0) with a total of 10 patients (17.2%).

Table 8: Frequency Distribution Based on Clinical Manifestations

Clinical Manifestations	Number	%
Out of breath	15	7
Headache	53	23
Nausea and vomiting	67	29
Blurred vision	2	1
Weak	25	11
Indigestion	3	1
Other	64	28
Total	229	100

Based on table 8, the results obtained are the highest frequency in clinical manifestations of nausea and vomiting with a total of 67 patients (29%), while the lowest frequency in clinical manifestations of blurred vision with a total of 2 patients (1%). Other clinical manifestations include chest pain, cough, fever, and cold sweats.

Table 9: Frequency Distribution by Degree of Blood Pressure

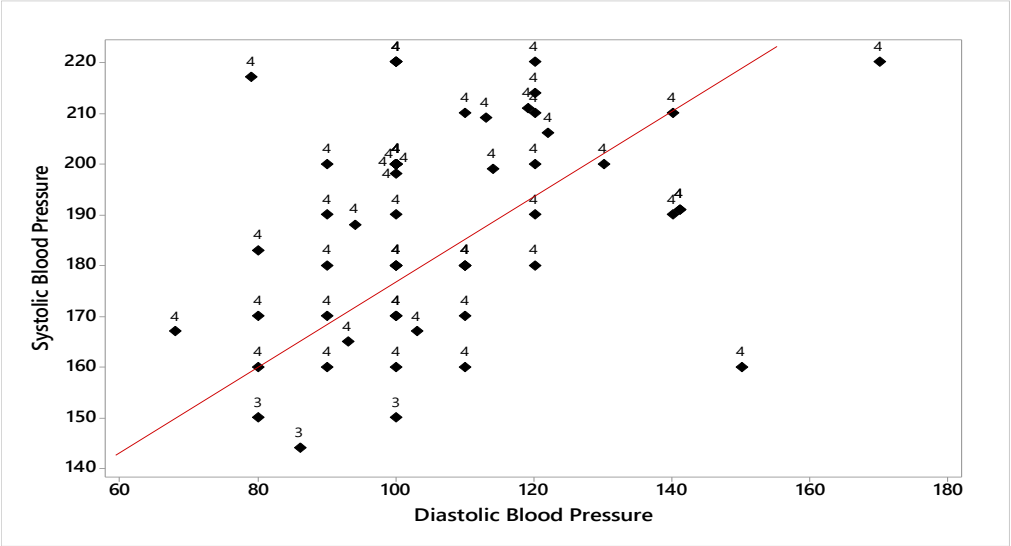
Degree of Hypertension	Number	%
Stage I Hypertension	3	5.2
Stage II Hypertension	55	94.8
Total	58	100

Based on table 9, the results obtained are the highest frequency in stage II hypertension with a total of 55 patients (94.8%) and the lowest frequency in stage I hypertension with a total of 3 patients (5.2%).

Table 10: Distribution of Frequency Based on History of Hypertension

History of Hypertension	Number	%
No History of Hypertension	14	24.1
Have a History of Hypertension	44	75.9
Total	58	100

Based on table 10, the results obtained were patients with the highest frequency in patients with a previous history of hypertension, namely 44 patients (75.9%), and the lowest frequency in patients without a previous history of hypertension, namely 14 patients (24.1%).



* Scatter Plot Linearity Test; linear

Figure 1: Correlation of Systolic and Diastolic Blood Pressure

Based on Figure 1, the results obtained from the scatter plot linearity test between systolic and diastolic blood pressure are linearly correlated, where the higher the systolic blood pressure, the higher the diastolic blood pressure.

Table 11: Frequency Distribution of Hypertensive Patients with CKD

Category	Number	%
No CKD	46	79.3
Accompanied by CKD	12	20.7
Total	58	100

Based on table 11, the results obtained are the frequency of hypertensive patients accompanied by chronic kidney disease, as many as 12 patients (20.7%), and hypertensive patients without chronic kidney disease, as many as 46 patients (79.3%).

Table 12: Correlation of Hypertension and Chronic Kidney Disease

Hypertension	Chronic Kidney Disease				OR	P-Value*
	Not Accompanied by CKD		Accompanied by CKD			
	N	%	N	%		
Stage I Hypertension	3	6.5	0	0.0	1.279 (1.112-1.471)	0.364
Stage II Hypertension	43	93.5	12	100		
Total	46	100	12	100		

* Chi-square test, p > 0.05

Based on table 12, the results obtained are three patients with stage I hypertension without chronic kidney disease (6.5%), stage I hypertension patients with chronic kidney disease, and no stage II hypertension without chronic kidney disease, as many as 43 patients (93.5%), and stage II hypertension patients accompanied by chronic kidney disease as many as 12 patients (100%). The chi-square test was carried out to see the correlation between hypertension and chronic kidney disease. The results obtained were p-value > 0.05, so based on the results of statistical correlation analysis, it was concluded that hypertension had no significant impact on chronic kidney disease.

Table 13: Levels of Sodium, Potassium, Chloride, Urea, and Creatinine

Category	Sodium		Potassium		Chloride	
	Number	%	Number	%	Number	%
Decrease	10	20	16	32	4	8
Normal	40	80	34	68	43	86
Increase	0	0	0	0	3	6
Total	50	100	50	100	50	100

Table 14: Level of Urea and Creatinine

Category	Urea		Creatinine	
	Number	%	Number	%
Decrease	0	0	0	0
Normal	38	76	25	50
Increase	12	24	25	50
Total	50	100	50	100

Based on tables 13 and 14, the results obtained are the frequency of patients with decreased sodium levels in 10 patients (20%), potassium levels decreased in 16 patients (32%), and chloride levels decreased in 4 patients (8%). The frequency of patients with chloride levels increased in 3 patients (6%), urea levels increased in 12 patients (24%), and creatinine levels increased in 25 patients (50%).

This study was conducted to look at the impact of hypertension on patients with chronic kidney disease at UKI General Hospital for the period 2019 – 2020. The results obtained were hypertensive male patients, as many as 23 patients (39.7%), and female as many as 35 patients (60.3%). There are more women with hypertension than men. The risk of hypertension in women began to increase during the premenopause period. It is because the hormone estrogen level begins to decrease, where estrogen functions to increase High-Density Lipoprotein (HDL), which protects blood vessels from damage.[36]

The age groups in this study were 46-55 years and 56-65 years, with a total of 15 patients (25.9%). Hypertension sufferers at the age of 45 years because hypertension is a degenerative disease, where with increasing age, the risk of hypertension is higher due to an increase in peripheral resistance and sympathetic activity, arterial walls thicken because collagen accumulates in the muscle layer, so that blood vessels become narrow and stiff. In the elderly, the sensitivity of blood pressure regulators, namely the baroreceptor reflex, is reduced, and renal blood flow and glomerular filtration rate decrease. It can trigger hypertension. The frequency distribution based on BMI was highest in obesity I, with a total of 24 patients (41.4%). Subjects with obese BMI had a 4.02 times greater risk of developing hypertension than subjects with normal BMI. In hypertensive patients with obesity, cardiac output and blood volume will increase; sodium reabsorption in the kidneys will also increase and cause hypertension.[37,38]

The results of the frequency distribution based on clinical manifestations were nausea and vomiting in 67 patients (29%), headaches in 53 patients (23%), weakness in 25 patients (11%), shortness of breath in 15 patients (7%), digestive disorders in 3 patients (1%), blurred vision in 2 patients (1%), and other clinical manifestations in 64 patients (28%) which included chest pain, cough, fever, and cold sweats. The distribution results based on the stage of hypertension obtained among patients with stage I hypertension, as many as three patients (5.2%), and patients with stage II hypertension, as many as 55 patients (94.8%). There were 44 patients with hypertension (75.9%) and hypertensive patients with no previous history of hypertension (24.1%). The linearity scatters plot test results of the patient's systolic blood pressure are linearly correlated with the patient's diastolic blood pressure, where the higher the systolic blood pressure, the higher the diastolic blood pressure.

In this study, there were 58 patients diagnosed with hypertension, 12 (20.7%) of whom had chronic kidney disease, and 46 (79.3%) did not have chronic kidney disease. Specifically, for hypertensive patients with chronic kidney disease, 42% were diagnosed with stage 5 CKD, 17% CKD stages 3a, 3b, and 4, and 8% CKD stage 2. Chronic kidney disease in hypertensive patients can be caused by uncontrolled high blood pressure. Weakens and narrows the blood vessels that supply the kidneys, thereby preventing the kidneys from functioning normally. In addition, high blood pressure levels and hypertension duration can affect chronic kidney disease incidence. A chi-square test was performed to see the correlation between hypertension and chronic kidney disease. The results obtained were p-value > 0.05, so based on the statistical analysis, it was concluded that hypertension had no significant impact on chronic kidney disease.

The number of samples used in the frequency distribution of sodium, potassium, chloride, urea, and creatinine levels was 50, which was due to the incomplete attachment of laboratory results in 8 other patients in the medical records. The analysis of urea and creatinine levels showed that 12 patients (24%) had increased urea levels, and 25 patients (50%) had increased creatinine levels. The results explain that although the statistical analysis of hypertension has no significant impact on chronic kidney disease, based on the mechanism, Biological studies have reported an increase in urea and creatinine levels, resulting in a decrease in kidney function. The excretion of urea into the urine is hampered by impaired renal excretion, so the level of urea in the blood increases. Serum creatinine levels are influenced by kidney function; when kidney dysfunction occurs, the creatinine filtration ability will decrease so that serum creatinine levels will increase.

CONCLUSION

Based on research conducted at the UKI General Hospital for 2019 - 2020, a total of 58 patients with hypertension were found, 12 (20.7%) of whom were accompanied by chronic kidney disease. The statistical analysis of the correlation between hypertension and chronic kidney disease is p > 0.05, so it can be concluded that hypertension has no significant impact on chronic kidney disease at UKI General Hospital for the period 2019 – 2020. Lifestyle changes can provide beneficial effects in hypertensive patients, and recommended that readers maintain a healthy diet. Balanced nutrition so that blood pressure can be controlled and does not impact decreasing kidney function.

Declaration of Interest

The authors report no conflicts of interest.

REFERENCES

[1] MCH, Overview of the Use of Books, and Knowledge of Pregnant Women About. "The SDGs. World Heal Organ. 2016; 1,121. 3. Ministry of Health of the Republic of Indonesia. Infodatin Center for Data and Information of the Ministry of Health of the Republic of Indonesia. Center of Data and Information of the Ministry of Health of the Republic of Indonesia: Causes of Maternal Death. 2014. p. 1-2. 4. Health Office Aceh. Aceh Health Profile 2016. 2017; 1-160."

[2] Adrian, Steven John. "Essential hypertension: the latest diagnosis and management in adults." Mirror of the World of Medicine 2019; 46(3):172-178.

[3] Nuraini B. Risk Factors of Hypertension. Faculty of Medicine, University of Lampung. 2015; 4(5).

[4] Rahardjo, Pudji. "The Link between Hypertension and Kidney Disease." In: Republika newspaper/magazine. University of Indonesia (2007).

[5] Hill, Nathan R., Samuel T. Fatoba, Jason L. Oke, Jennifer A. Hirst, Christopher A. O'Callaghan, Daniel S. Lasserson, and FD Richard Hobbs. "Global prevalence of chronic kidney disease-a systematic review and meta-analysis." PloS one 2016; 11(7):e0158765.

[6] Ministry of Health, R. I. "The Role of Government in the Prevention and Control of Kidney Disorders in Children." Tropical Diseases in Indonesia, November (2018): 1-18.

[7] Sudikno, Hidayat Syarief, Cesilia Meti Dwiriani, Hadi Riyadi, and Jl Percetakan Negara. "Risk Factors of Central Obesity in Adults Age 25-65 Years in Indonesia (Basic Health Research Data Analysis 2013) (Risk Factors Central Obesity in 25-65 Year-Old Indonesian Adults [Analysis of Data of Basic Health Research 2013])." researcher. Nutrition and Food 2015; 38(2):111-120.

[8] Giles, Thomas D., Barry J. Materson, Jay N. Cohn, and John B. Kostis. "Definition and classification of hypertension: an update." The journal of clinical hypertension 2009; 11(11):611-614. https://doi.org/10.1111/j.1751-7176.2009.00179.x

[9] Ostchega, Yechiam. Blood pressure randomized methodology study comparing automatic oscillometric and mercury sphygmomanometer devices: National Health and Nutrition Examination Survey, 2009-2010. No. 59. US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics, 2012.

[10] Beaney, Thomas, Aletta E. Schutte, George S. Stergiou, Claudio Borghi, Dylan Burger, Fadi Charchar, Suzie Cro et al. "May Measurement Month 2019: the global blood pressure screening campaign of the International Society of Hypertension." Hypertension 2020; 76(2):333-341. https://doi.org/10.1161/HYPERTENSIONAHA.120.14874

[11] Saeed, Khwaja Mir Islam. "Prevalence and Associated Factors of High Blood Pressure in Urban Setting of Kandahar City in

Afghanistan in 2015." Jundishapur Journal of Health Sciences (JJHS) 2017; 9(3).

[12] Turana, Yuda, Jeslyn Tengkawan, Yook Chin Chia, Satoshi Hoshide, Jinho Shin, Chen-Huan Chen, Peera Buranakitjaroen et al. "Hypertension and dementia: a comprehensive review from the HOPE Asia Network." The Journal of Clinical Hypertension 2019; 21(8):1091-1098. https://doi.org/10.1111/jch.13558

[13] Purba, Elida Normiaty, Heru Santosa, and Fazidah Aguslina Siregar. "The relationship of physical activity and obesity with the incidence of hypertension in adults aged 26-45 years in Medan." Open Access Macedonian Journal of Medical Sciences 2019; 7(20):3464. https://doi.org/10.3889/oamjms.2019.447

[14] Mittal, Bharati V., and Ajay K. Singh. "Hypertension in the developing world: challenges and opportunities." American Journal of Kidney Diseases 2010; 55(3):590-598. https://doi.org/10.1053/j.ajkd.2009.06.044

[15] Wideman, R. F., D. D. Rhoads, G. F. Erf, and N. B. Anthony. "Pulmonary arterial hypertension (ascites syndrome) in broilers: a review." Poultry Science 2013; 92(1):64-83. https://doi.org/10.3382/ps.2012-02745

[16] Deshmukh, Abhishek, Gagan Kumar, Nilay Kumar, Rahul Nanchal, Freij Gobal, Ankit Sakhuja, and Jawahar L. Mehta. "Effect of Joint National Committee VII report on hospitalizations for hypertensive emergencies in the United States." The American journal of cardiology 2011; 108(9):1277-1282. https://doi.org/10.1016/j.amjcard.2011.06.046

[17] Iwakiri, Yasuko, and Roberto J. Groszmann. "Pathophysiology of portal hypertension." The Liver: Biology and Pathobiology (2020): 659-669.

[18] Hall, John E. "Renal dysfunction, rather than nonrenal vascular dysfunction, mediates salt-induced hypertension." Circulation 2016; 133(9):894-906. https://doi.org/10.1161/CIRCULATIONAHA.115.018526

[19] Flemming, Nicole Bernadette, Linda Alba Gallo, and Josephine Maree Forbes. "Mitochondrial dysfunction and signaling in diabetic kidney disease: oxidative stress and beyond." In Seminars in nephrology, vol. 38, no. 2, pp. 101-110. WB Saunders, 2018.

[20] Rajesh, S. "A Study of Thyroid Function Abnormalities in Patients with Chronic Kidney Disease." PhD diss., Coimbatore Medical College, Coimbatore, 2016.

[21] Mirilas, Petros, and John E. Skandalakis. "Surgical anatomy of the retroperitoneal spaces part II: the architecture of the retroperitoneal space." The American surgeon 2010; 76(1):33-42. https://doi.org/10.1177/000313481007600108

[22] Mompeó, Blanca, Eva Maranillo, Arturo García-Touchard, and Jose R. Sanudo. "The morphogenesis of the renal plexus: renal artery and sympathetic fibers." Clinical Anatomy 2019; 32(2):272-276. https://doi.org/10.1002/ca.23297

[23] Borges, S. A., AV Fischer Da Silva, and A. Maiorka. "Acid-base balance in broilers." World's Poultry Science Journal 2007; 63(1):73-81. https://doi.org/10.1017/S0043933907001286

[24] Pottel, Hans, Liesbeth Hoste, and Pierre Delanaye. "Abnormal glomerular filtration rate in children, adolescents and young adults starts below 75 mL/min/1.73 m2." Pediatric Nephrology 2015; 30(5):821-828. https://doi.org/10.1007/s00467-014-3002-5

[25] Chen, Aiqun, Ying Sun, Wenchan Li, Jihong Yang, Zhiming Yao, Lei Shi, and Jing Jia. "Application of GFR estimation equations in elderly patients with measured GFR below 60 mL/min/1.73 m2." Aging clinical and experimental research 2020; 32(3):415-422. https://doi.org/10.1007/s40520-019-01218-2

[26] McLafferty, Ella, Carolyn Johnstone, Charles Hendry, and Alistair Farley. "Fluid and electrolyte balance." Nursing Standard 28, no. 29 (2014).

[27] Eckardt, Kai-Uwe, Josef Coresh, Olivier Devuyst, Richard J. Johnson, Anna Köttgen, Andrew S. Levey, and Adeera Levin. "Evolving importance of kidney disease: from subspecialty to global health burden." The Lancet 2013; 382(9887):158-169. https://doi.org/10.1016/S0140-6736(13)60439-0

[28] Habeeba, Shareefa, Rafya Fatima, Noor Us Sabah, Nausheen Sehar, Umama Yezdani, and Mohammad Gayoor Khan. "Prospective Study on Evidence Based Management of chronic Kidney Disease with Comorbidities." Asian Journal of Pharmaceutical Research and Development 2020; 8(2):117-122. https://doi.org/10.22270/ajprd.v8i2.667

[29] Richardson, Caroline R., Jeffrey R. Borgeson, R. Van Harrison, Jennifer A. Wyckoff, Anne S. Yoo, James E. Aikens, Dina H. Griauzde et al. "Management of Type 2 Diabetes Mellitus." (2021).

[30] Awad, Sami M. "Chronic renal failure in Al-Anbar of Iraq." Saudi Journal of Kidney Diseases and Transplantation 2011; 22(6):1280.

[31] Abd el Fattah Abdallah, Mahmoud, Aber Halim Baki, Sally Adel Hakim, and Cherry Reda Kamel. "Epidemiological study of end stage renal disease at Ain Shams University Hospital. A five year retrospective study." Life Science Journal 2014; 11(12).

[32] Neuwirt, Hannes, Paul Perco, Alexander Kainz, Irmgard Mühlberger, Johannes Leierer, Suzie-Jane Braniff, Bernd Mayer, Gert Mayer, and Michael Rudnicki. "A 3-biomarker-panel predicts renal outcome in patients with proteinuric renal diseases." BMC medical genomics 2014; 7(1):1-14. https://doi.org/10.1186/s12920-014-0075-8

[33] Ammirati, Adriano Luiz. "Chronic kidney disease." Revista da Associação Médica Brasileira 66 (2020): s03-s09.

[34] Webster, Angela C., Evi V. Nagler, Rachael L. Morton, and Philip Masson. "Chronic kidney disease." The lancet 2017; 389(10075): 1238-1252. https://doi.org/10.1016/S0140-6736(16)32064-5

[35] Stevens, Paul E., Adeera Levin, and Kidney Disease: Improving Global Outcomes Chronic Kidney Disease Guideline Development Work Group Members*. "Evaluation and management of chronic kidney disease: synopsis of the kidney disease: improving global outcomes 2012 clinical practice guideline." Annals of internal medicine 2013; 158(11):825-830. https://doi.org/10.7326/0003-4819-158-11-201306040-00007

[36] El Khoudary, Samar R., Brooke Aggarwal, Theresa M. Beckie, Howard N. Hodis, Amber E. Johnson, Robert D. Langer, Marian C. Limacher et al. "Menopause transition and cardiovascular disease risk: implications for timing of early prevention: a scientific statement from the American Heart Association." Circulation 2020; 142(25):e506-e532.

[37] Dominguez, Ligia J., Carmen Sayón-Orea, Alfredo Gea, Estefania Toledo, Mario Barbagallo, and Miguel A. Martínez-González. "Increased Adiposity Appraised with CUN-BAE Is Highly Predictive of Incident Hypertension. The SUN Project." Nutrients 2021; 13(10):3309. https://doi.org/10.3390/nu13103309

[38] Soraya, Fira, Elyana Asnar, and Muhammad Aminuddin. "EKG Profile of Hypertensive Patients at Cardiac Polyclinic." Journal of Ners 2016; 11(1):40-44. https://doi.org/10.20473/jn.v11i1.1179