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THE RELATIONSHIP OF ANGIOTENSIN ENZYME AND OTHER PARAMETERS WITH COVID 19 AND ITS EFFECT ON KIDNEY DISEASE IN IRAQI SOCIETY

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By Yaqoob Khalid Kareem AL-LAMI

January 2023

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ABSTRACT

THE RELATIONSHIP OF ANGIOTENSIN ENZYME AND OTHER PARAMETERS WITH COVID 19 AND ITS EFFECT ON KIDNEY DISEASE IN IRAQI SOCIETY

Yaqoob Khalid Kareem AL-LAMI

Master of Science in Chemistry

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The aim of this study is to evaluate the importance of ACE, other parameters of Covid 19 and its effect on kidney disease. A total of 105 people were enrolled in this study. 55 patients with COVID-19, 50 normal controls. It stimulates ACE2, which plays important role in the body because it plays a role in maintaining a physiological state. And the pathophysiological balance of the body, and therefore the change in its levels is an important indicator in the diagnosis and follow-up of Covid-19. Age had a clear importance, as the study proved that the older the age, the higher the infection rate, which means that the virus affects the elderly more than the young. Urea and serum creatinine were not significantly affected in those infected with the Covid-19 virus, and this explains the absence of a relationship between renal failure disease and the virus that causes the disease. There was no strong indication of the clinical significance of lipids in COVID-19 patients. D-Dimer had a high statistical significance, as he explains these changes in his results as a result of tissue changes that occur in the lungs, and thus the percentage of oxygen decreases, and as a result, blood clots increase in Covid-19 patients. The results of IL-6 indicate that the levels of IL-6 changed significantly due to the importance of the immune system in this disease.

2023, 51 pages

Keywords: Angiotensin Enzyme, Blood urea, Creatinine, Covid-19, Kidney disease.

ÖZET

IRAK TOPLUMUNDA ANJİYOTENSİN ENZİMİ VE DİĞER PARAMETRELERİN COVİD 19 ILE ILİŞKİSİ VE BÖBREK HASTALIĞINA ETKİSİ

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Bu çalışmanın amacı, ACE'nin önemini, Covid 19'un diğer parametrelerini ve böbrek hastalığına etkisini değerlendirmektir. Bu çalışmaya toplam 105 kişi alındı. 55 COVID-19 hastası, 50 normal kontrol. Fizyolojik bir durumun korunmasında rol oynadığı için vücutta önemli rol oynayan ACE2'yi uyarır. Ve vücudun patofizyolojik dengesi ve dolayısıyla seviyelerindeki değişim Covid-19 tanı ve takibinde önemli bir göstergedir. Çalışma, yaş arttıkça enfeksiyon oranının da arttığını, yani virüsün yaşlıları gençlerden daha fazla etkilediğini kanıtladığından, yaşın açık bir önemi vardı. Covid-19 virüsü ile enfekte olanlarda üre ve serum kreatinin önemli ölçüde etkilenmedi ve bu da böbrek yetmezliği hastalığı ile hastalığa neden olan virüs arasında bir ilişki olmamasını açıklıyor. COVID-19 hastalarında lipidlerin klinik önemine dair güçlü bir gösterge yoktu. D-Dimer, akciğerlerde meydana gelen doku değişiklikleri sonucu sonuçlarındaki bu değişiklikleri ve dolayısıyla oksijen yüzdesinin azaldığını ve buna bağlı olarak Covid-19 hastalarında kan pıhtılarının arttığını açıkladığı için istatistiksel olarak yüksek bir öneme sahipti. IL-6'nın sonuçları, bu hastalıkta bağışıklık sisteminin önemi nedeniyle IL-6 seviyelerinin önemli ölçüde değiştiğini göstermektedir.

2023, 51 sayfa

Anahtar Kelimeler: Anjiyotensin enzim, Kan üre, Kreatinin, Covid-19, Böbrek hastalığı.

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Yaqoob Khalid Kareem AL-LAMI

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CONTENTS

LIST OF SYMBOLS

-	Minus
%	Percent
**	Significant
/	Divide
+	Plus
<	Greater than
=	Equal
>	Less than
±	Plus minus
≤	Greater or equal to
≥	Less or equal to
Ca	Calsium
Cu	Copper
dL	Deciliter
g	Gram
kg	Kilogram
L	Liter
m^2	Square meter
µg	Microgram
Mg	Magnesium
mg	Microgram
mIU	Milli-international units
min	Minute
mL	Milliliter
mmol	Millimole
mol	Mole
ng	Nanogram
nm	Nanometer
NS	Non-significant
rpm	Revolutions per minute
Se	Selenium
Zn	Zinc
µL	Microliter

LIST OF ABBREVIATIONS

angII	Angiotensin II
AT1	AngII type 1
CKD	Chronic kidney disease
PAI	Plasminogen activator inhibitor
PKD	Polycystic kidney disease
RAAS	Renin-angiotensin-aldosterone system

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1. INTRODUCTION

Angiotensin II, also known as angII, is the most important biologically active product, and its production requires a pathway that involves a large number of precursor (Basic) peptides and enzymes, some of which are controlled by different sets of genes. In addition, angII is produced as a direct consequence of this action. It is known that angII may increase fluid and salt retention as well as having the capacity to constrict vascular smooth muscle cells. This effect is achieved by directly acting on tubular cells and by boosting the synthesis of aldosterone. In addition to this, it is well known that angII has the potential to narrow blood arteries (Liu *et al.* 2022).

Over the course of several decades, a human kidney's ability to function normally may slowly but steadily deteriorate, even if just slightly. This physiological phenomenon is made clearly clear by the fact that, as one grows older, their glomerular filtration rate steadily drops. In other words, as one gets older, their kidneys become less efficient at filtering blood. We may count ourselves fortunate because the patient's clinical state is often unaffected by the slight decline in renal function that has occurred (Legouis *et al.* 2022).

On the other side, those who have a renal disease may see a significant quickening in the pace at which they are losing their renal function. Even if the original insult or the symptoms of the underlying sickness have passed or the disease's activity has decreased, this might still be the case. As a direct result of this, a variety of metabolic problems will develop, and in the end, a significant proportion of patients will have renal failure that has reached its terminal stage. . This line will depict the progression of the decline in renal function (Netere and Sendekieb 2022).

On the other hand, more recent study has given light on other probable behaviors that angII may engage in. In vitro studies have shown that angiotensin II (angII) has the capacity to increase the proliferation of vascular smooth muscle cells, glomerular mesangial cells, and renal tubular cells. These results were obtained from a variety of different studies. Additionally, it would seem that AngII has a function in the formation

of collagen by accelerating the process of collagen creation while concurrently blocking its breakdown. This would suggest that AngII is involved in the formation of collagen. This is the case because genetic polymorphisms have been shown to be associated with a higher risk of developing chronic renal failure. This is due to the fact that genetic polymorphisms have been shown to have a role in the progression of hypertension, which may lead to kidney failure. Also, Angiotensin Enzym and is involved in many organs of the body such as the kidneys, heart, lungs, and other organs; however, we will investigate its association with Kidney illness and Covid-19 patient here. An investigation will be conducted to see whether or not there is a connection between Angiotensin and Covid 19 and the other indicators seen in renal disease (Park *et al.* 2022).

1.1 Aim of Study

The purpose of this study is to evaluate the importance of angiotensin enzyme, along with other parameters, in relation to its validity in early detection and severity of disease.

2 LITERATURE REVIEW

2.1 Angiotensin Enzyme

The renin-angiotensin-aldosterone system, sometimes known as the RAAS, plays an important part in the pathophysiology of both the cardiovascular system and the kidneys. Angiotensin II or (angII) is the most important biomolecular activation product produced by numerous peptides and precursor enzymes some of which are controlled by different genes. This mechanism also leads to the generation of angII. By acting directly on renal tubular cells and stimulating angII aldosterone production it increases fluid and salt retention and potentially constricts vascular smooth muscle cells. In addition, angII is known to have the ability to constrict blood vessels (Augustine *et al.* 2022).

On the other hand, newer research has shed light on additional possible behaviors that angII may do. The results of several different studies show that angII stimulates the proliferation of glomerular interstitial and renal tubular smooth muscle cells in vitro. Additionally, it would seem that AngII has a role in the buildup of collagen by stimulating the process of collagen production while simultaneously preventing its breakdown. Another observation that highlights the significance of angII in cardiovascular and renal pathology is that it interferes with the fibrinolytic process. This is done by increasing plasminogen activator inhibitor (PAI-1), which is a protein. This is because genetic polymorphisms of the RAAS have been shown to play a role in the development of hypertension, which can lead to kidney failure (Balakumar *et al.* 2022).

Angiotensinogen may be transformed into angiotensin I when the renin enzyme is activated. Afterward, angiotensin I is changed into angiotensin II, which is the primary biologically active product of the RAAS. There are 26 exons included inside the human ACE gene, which may be found on chromosome 17q23. The coding sequence specifies a protein with 1304 amino acids and a signal peptide, both of which are included. The ACE gene product is made up of two identical domains, each of which has two active sites. Rigat *et al.* Presented an important paper in 1990 that served as a catalyst for subsequent

research on the RAAS gene polymorphisms. This publication provides motivation for reported in those with the II genotype (Sharma *et al.* 2022).

Subjects with the ID genotype showed some degree of correlation because the effects of both alleles could be seen in heterozygotes. High ACE activity is hypothesized to lead to AngII conversion. The Role of Angiotensin II in the Pathophysiology of Cardiovascular and Renal Disease Several studies have established an association between the prevalence and incidence of cardiovascular and renal disease and the ACE I/D polymorphism which is not surprising. Effort (Goel *et al.* 2022).

2.1.1 Role of angiotensin enzyme

ACE makes an important contribution to the RAAS cascade by converting AngI to AngII. This is due to plasma ACE and ACE binding mainly to the endothelial surface. AngII is a potent vasoconstrictor acting on the systemic and renal vasculature. The AngII type 1 (AT1) receptor is responsible for mediating this effect. AngII can also activate tissue growth and renal and vascular repair processes by influencing AT1 and AT2 receptors. A progressive decline in renal function is thought to be due to the hemodynamic effects (such as increased systemic and glomerular blood pressure) and tissue growth effects (such as promoting hepatomegaly and interstitial fibrosis) of AngII. Several studies have investigated whether elevated ACE levels in DD heterozygotes have a functional impact on the RAAS phenotype. Plasma levels of RAAS components including AngII renin and aldosterone were found to be similar across genotypes (Hasan *et al.* 2021).

The homozygous status of the D-D gene made this discovery possible. A study using a human hand model provides evidence for this finding. On the other hand genotypes did not differ in response to exogenous Angi when renin was inhibited in other words the effect of background RAAS activity was limited. The association of ACE I/D gene polymorphisms remains unclear despite extensive work in various published linkage studies. This is mainly because association studies show only statistical associations between polymorphisms and increased risk of disease or increased organ damage (Xiao *et al.* 2021).

It is clearly shown by research into the relationship between a polymorphic section of DNA (referred to as the genotype) and a gene allele that is responsible for illness (phenotype). However, there are instances in which the spacing between these DNA pieces is not very close. As a result, it is abundantly evident that association studies are not going to provide solutions to all of our problems. Various research have also looked at populations of people of various racial and ethnic backgrounds (Saleh *et al.* 2022).

This makes it much more difficult to compare the results of different association studies. In conclusion, it is abundantly obvious that further fundamental physiological research that study the effects of (ACE I-D) polymorphism on renal pathophysiology are desperately needed. We won't be able to grasp the influence of the ACE I/D polymorphism on the development and progression of renal illness until we reach that point. Eventually, we will be able to devise therapeutic regimens that are precisely suited to various genetic risk profiles (Wang *et al.* 2022).

2.1.2 Effect on renal disease

Under these circumstances, it is reasonable to anticipate an increased frequency of a particular genotype if the ACE polymorphism has an influence on the likelihood of developing certain kidney diseases. On the other hand, the start of many renal disorders may be gradual and symptomless, and the illnesses may never be detected at all unless the progression of the disease is observed. Association studies in patients with existing renal illness may not just represent vulnerability but also a progressive course due to the selection bias that results from the investigations. Most of the data collected to date has been on diabetic nephropathy. Therefore meta-analysis can be used to assess the role played by ACE genotype (Monticone *et al.* 2020).

This suggests a possible mechanism for this relationship. European and Japanese studies have shown similar results showing that allele frequencies and genotype distributions are normal in IgA nephropathy. Furthermore, the IgA nephropathy relapse does not rely on the recipient's genotype in cases of renal transplantation. Patients diagnosed with membranous glomerulopathy have been shown to have a normal genotype distribution,

according to reports. According to the findings of a research conducted in Korea, the distribution of genotypes was normal in cases with minimal change nephropathy; (Verhoeven *et al.* 2022).

On the other hand, focal segmental glomerulosclerosis is regarded to be, in the majority of instances, not a primary renal ailment but rather the culmination of a process in which a variety of renal conditions have progressed over time (Van Sandwijk *et al.* 2019). Therefore, it is possible that the latter connection reflects a progressive course rather than a propensity to developing renal illness. Last but not least, a number of studies lend credence to the hypothesis that the D allele is linked to hypertensive renal vascular damage. This hypothesis is supported by the presence of hypertensive nephrosclerosis and biopsy-proven hypertension associated with albuminuria and overt reno-vascular-disease. Taken together ACE genotype does not appear to have a significant effect on susceptibility to developing primary glomerular disease or tubulointerstitial disease(Gauckler *et al.* 2020).

On the other hand situations in which damage to the microvascular or renal system plays an important role in the pathophysiology seem to be associated with the D allele (Podestà and Ponticelli 2020). This observation is consistent with the results of a large meta-analysis conducted by Lorbach and colleagues in hypertension cardiovascular disease and renal disease (2020) (Lorbach *et al.* 2020).

2.1.3 Putative mechanisms

In order to develop effective treatments for renoprotection, it is essential to have a better understanding of the processes that underlie the renal risk that is linked with the D allele. It is important to highlight, howeverIt is currently unknown whether D is actually a risk factor for kidney disease or just a risk marker. Responsibility for increased risk of kidney. Because of this, impacts on frequent mediators of renal function decline are suggested (Porrett *et al.* 2022).

It is believed that a similar route plays a substantial role in the gradual loss of renal function that happens in a large number of people with renal disease, despite the fact that there is no overt activity of the original condition. This common downward spiral, which ultimately results in focal segmental sclerosis and is thought to be the final common pathway of chronic renal disease, has been linked to systemic and glomerular hypertension as well as glomerular protein leakage. Both of these conditions have been linked to the disease. The function of AngII in this common pathway is supported experimentally and by the reprophylactic effects of ACE inhibitors in human kidney disease. The theory that the D allele is responsible for stimulating this common route of kidney injury by increasing the production of AngII is appealing for a number of different reasons (Sun *et al.* 2022).

In accordance with this, it has been shown that the DD genotype makes, it is possible that endothelial dysfunction, which has been linked to having the D allele, is implicated in the increased susceptibility of the cardiorenal macro- and micro-vascular bed. There is a possibility that AngII is involved in the renal and systemic effects that were just mentioned, but there is also a possibility that AngII is engaged in the effects that it has on other routes. AngII, for example, causes a rise in endothelial production of plasminogen activator inhibitor-1 (PAI-1), which has the potential to change the balance of fibrinolytic activity. It has been hypothesized that high PAI-1 levels may be associated with an increased risk of myocardial infarction and that PAI-1 levels may be linked by A.C.E genotype. It has not yet been determined whether or if this has any bearing on renal disease; nonetheless, in light of the hypothesis that intraglomerular thrombosis plays a role in the gradual decline of renal function, this potential warrants more investigation (Khamissi *et al.* 2022).

As can be seen from the above summary, the investigation into the function that normal genetic variability plays in diseases that are caused by a complex interaction of several factors does not have any straightforward solutions. The methodological shortcomings of the research that are now available make it difficult to understand the impact that an individual's ACE genotype plays in renal illness (Pastor-Soler *et al.* 2022).

Thus (ACE) genotype is the primary GRF (genetic risk factor) for progressive loss of renal function although it has moderate predictive power and is not always significant. The genetic basis of this renal effect can be revealed by conducting more in-depth genetic studies in genetically similar groups. Although large epidemiological studies have helped to determine the overall effect on the kidneys it may be more important to determine the mechanism and patient effect of ACE genotype (I/D) on the kidney. This can be done by determining which genotype the patient has. Clinical and pathophysiological research especially in genetically homogeneous populations is well suited to translate the information obtained by new genomic approaches into therapeutic benefits (Herrlich 2022).

2.2 Kidney Disease

2.2.1 Background

It can be said, in general, that the human kidneys, or if both kidneys are correct, are responsible and have a function for a variety of different activities within the human body, among these functions is the filtering of minerals, as well as the function of ensuring fluid balance, and for the safety of the body it works On the disposal of waste, and one of the important roles of the kidneys is to control the volume of blood, and there are other functions among others. The location of the kidneys is in the retroperitoneal region, on both sides of the vertebral column of the human body, on both sides of the spine. The kidneys get adequate amounts from a steady supply of blood. There is a function of the renal artery is to transport contaminated blood or that contains the wastes of metabolic processes to the kidney, and thus the kidneys filter and dispose of it. As a result, from the foregoing, we conclude that the primary function of the kidneys is to eliminate or rid the body of the toxic substance known as urea (which we measure in order to know the activity of the kidneys) from the bloodstream, in addition to other substances such as other waste salts and excess water, where they transform the composition The chemical mixture of a group of these substances into a yellowish liquid known as urine (Maio *et al.* 2022).

The encapsulation process takes place in each kidney with a capsule or called the renal capsule, and it is thick and made of strong fibrous tissue. This layer of fibrous tissue consists of two fats that act as a cushion between the other layers (Gonçalves *et al.* 2022, Gandhi *et al.* 2022).There is another area called the body area that is the site of the adrenal glands located in the kidneys and is the upper back. Every human has two kidneys, the outer layer is known as the renal cortex, It is a layer made up of the inner renal medulla and the outside layer. The human kidney's two sections are both passed through by the nephron. After that, special passageways known as renal veins allow the blood to leave the body (Gonçalves *et al.* 2022, Gandhi *et al.* 2022).

Even when individuals have the same underlying disease, there is a substantial interindividual heterogeneity in the pace at which they lose renal function. This is a finding that scientists find very interesting. This shows that the course of RFL (Renal Function Loss) seems to be mainly independent of the kind of underlying renal illness; however, this does not rule out the involvement of other variables. It should come as no surprise that preventing progressive renal failure in a patient is of the utmost significance, not only for the patient's sake but also to bring down the very high expenses associated with treating patients in this condition. Regrettably, we still do not have the ability to stop the progression of many distinct forms of renal disorders. However, a vast number of studies have shown beyond a reasonable doubt that the course of RFL (enal functFion L) oss is mostly determined by a variety of secondary variables. It is generally accepted that the most prevalent mediators in the last common route that leads to progressive renal impairment are systemic and glomerular hypertension, proteinuria, and hyperlipidemia. It is very uncommon for more than one of these common risk factors to be present at the same time, and the mutual interaction of these risk factors tends to speed up the progression of kidney injury (Dong *et al.* 2022).

2.2.2 Disease types

Different kidney illnesses can affect different parts of the body. For instance, the underlying cause of acute kidney injury is frequently the buildup of circulating mineral waste. Maintaining the proper fluid balance becomes more challenging as a direct result.

Other organs that may suffer damage from the illness include the heart, lungs, brain, and others. Numerous problems, such as limited blood flow, direct kidney damage, and blockage, might result in these symptoms. The disease known as kidney failure, on the other hand, occurs when the kidneys are unable to carry out their typical duty of filtering waste materials from the blood. Poor blood supply to the kidneys, urinary issues, infections, hemolytic uremic syndrome, drugs and alcohol, certain medications, and chemotherapy are the most prevalent and common causes of kidney failure. Numerous other factors can also contribute to kidney failure. Diabetes, excessive cholesterol, kidney infections, glomerulonephritis, and numerous other illnesses are the result. Since chronic kidney disease (CKD) is the primary subject of this investigation, more information regarding CKD is covered in the sections below (Zeng *et al.* 2020).

2.2.3 Chronic kidney disease

It has the potential to overtake tobacco usage as the world's top cause of mortality and has been established as a significant contributor to early cardiovascular disease. Additionally, the twelfth most common cause of mortality in the US is chronic kidney disease. The sixteenth most common cause of impairment worldwide is disability. Recent studies have shown a strong association between chronic kidney disease and higher rates of hospitalization, morbidity and mortality. Moreover, it is a serious disease, especially when kidney function gradually deteriorates over several years. Obesity, high blood pressure, cardiovascular disease and diabetes are all symptoms of being present that need to be addressed (Cheung *et al.* 2021).

Viewed from the other side, it can be recognized by a decreased glomerular filtration rate or by signs of kidney damage or impairment. Chronic kidney disease is the end result of many different factors resulting from many different disease pathways that affect kidney function. Eggers made the discovery in 2011 that approximately two million people all over the world require renal replacement in order to maintain a reasonable quality of life. This is the case despite the fact that the typical cost of renal replacement is approximately ten percent less for those who require it. Eggers further suggested that the number would continue to climb because of the rising prevalence of diabetes among the general

population. CKD is the main cause of mortality in those under the age of 60 and is responsible for a significant portion of the economic loss that occurs in both the public and private sectors (Kalantar-Zadeh *et al.* 2021). It is established that the disease has entered the breeding season with a rate of 5.7% in the first stage. 5.4% is the amount for stages 2 and 3. (Figure 2.1). Stages 4 and 5 together account for 0.4% of the total.

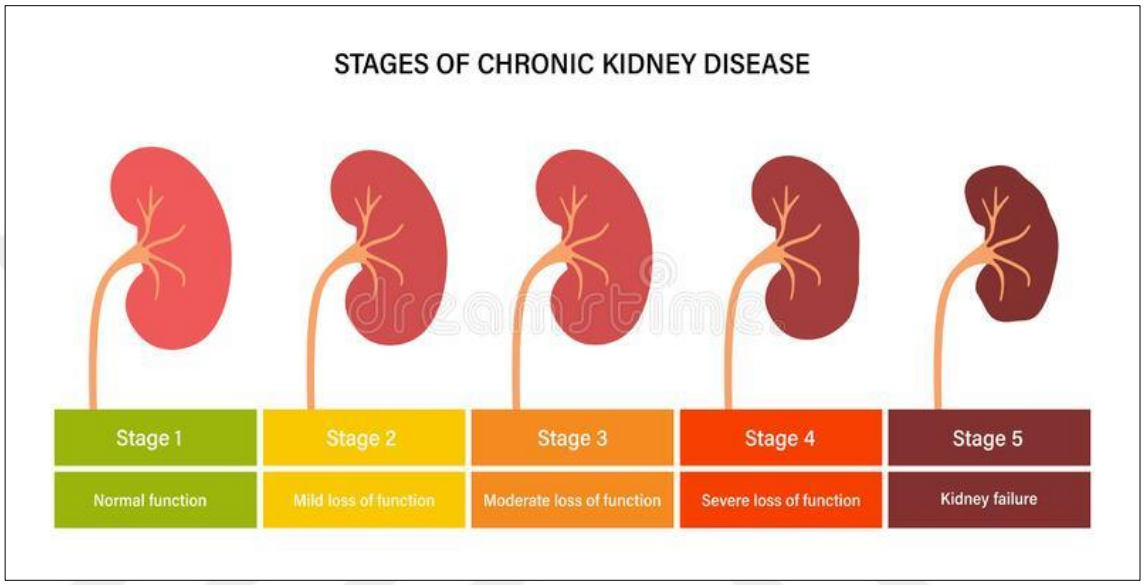

Figure 2.1 Stage of chronic kidney disease

In the United States, 19.2 million people are thought to have chronic renal disease. The adult population in the United States has a crude prevalence rate of 15.1% of the total population. In the same way, it ranks among the major causes of death in the UK. Over 5% of British people have chronic renal disease (Bhatt *et al.* 2021).

There are many different things that may cause chronic kidney disease, and the symptoms can vary from mild to severe. Some of the factors that contribute to it are more prevalent than others. For instance, glomerulonephritis is the underlying illness in 16 percent of patients in the United States who have urinary tract infection, and it is the underlying condition in 18 percent of cases in adults under the age of 65 in the United Kingdom. In addition to immunological illnesses like systemic lupus erythematosus and diabetes mellitus, other prominent causes of kidney failure include infectious diseases like

hepatitis B, medicines and poisons like nonsteroidal anti-inflammatory medications and penicillamine, tumors, and renal transplantation (Navaneethan *et al.* 2021).

Another condition that contributes to the development of chronic kidney disease in humans is diabetes, which may lead to diabetic nephropathy. Patients are considered to have diabetic nephropathy if they have proteinuria that is more than 0.5 grams per day. These patients' risk of developing cardiovascular disease also increases as their renal function deteriorates, which is a further concerning factor. Secondary hypertension is a kind of high blood pressure that occurs in people who already have another medical problem. Less than ten percent of all instances of hypertension may be attributed to secondary hypertension, which can be brought on by conditions such as chronic renal disease, endocrine dysfunction, cardiovascular illness, pregnancy, and drug use (Wheeler *et al.* 2021).

A number of variables contribute to the spread of chronic illnesses like CKD. A deteriorating economic position, unhealthy behaviors, and a lack of access to health care services and facilities are some of the factors contributing to this. There was a connection between the progression of kidney disease and factors such as age, the use of nephrotoxic medicines, and diabetes. In addition, infectious diseases were found and classified according to a risk factor, which included insufficient sanitation, a lack of a clean or safe water supply, and high concentrations of the infectious agent. It was shown that males under the age of 60 who were exposed to lesser amounts of heavy metals had a greater prevalence of predominance when pesticide exposure, dehydration, and alcohol usage were all present (Mafra *et al.* 2021).

On the other hand, risk factors are often researched in individuals who do not display any symptoms of the illness. It is strongly suggested that people who are at a higher risk of developing CKD get themselves evaluated. A condition that is connected with kidney disease or its treatment is referred to as a "complication." Some examples of complications include high blood pressure, anemia, malnutrition, bone disease (osteoporosis), peripheral neuropathy (neuropathy), and a loss in quality of life. The progression of renal disease is associated with an increase in the number of abnormalities

in the blood, which suggests an increase in the chance of adverse effects (Silva *et al.* 2022).

2.2.4 Acute kidney disease

It is a disease that manifests itself due to the sudden deterioration in kidney function. There are several different types of this disease. Acute renal impairment is usually detected early in the patient's disease progression. The gallon is a unit of measure for the amount of waste, electrolytes, and fluids collected in this way. Additionally, it could have less severe consequences, like impairment to the immune system or organs other than the kidneys. The location as well as the consequences have an impact on the size of the effect. It frequently has both immediate and long-term deleterious effects, including increased mortality and morbidity. Acute kidney injury, or AKI, is a public health issue in today's society. As a result, testing and detection are necessary to establish a person's susceptibility (Sabatino *et al.* 2021).

The diagnosis is determined based on the amount of serum keratinize that is present as well as the amount of urine that is present. The diagnosis is made if the serum keratinize level is higher than 0.3 mg/dl, rises by more than 48 hours, or increases by more than 1.5-fold in seven days. The diagnosis and classification of acute renal damage are broken down into many categories. AKI phases, on the other hand, can be identified by changes in the keratinization of the serum or the amount of urine that is produced. These changes are described as an increase in serum keratinization to less than 0.3 mg/dL in 48 hours, a 1.5-fold increase in the keratinization of the serum, and an increase in the amount of urine produced (Cai *et al.* 2021).

2.2.5 Polycystic kidney disease

This condition is characterized by the development of numerous cysts in the kidneys, which eventually lead to kidney failure. Due to the structural changes caused by PKD cysts, the kidneys may increase in size, which can lead to decreased kidney function and,

in severe cases, kidney failure. PKD occurs at irregular intervals. There is a need to test the patient's ability to undergo the transplant. Almost half of the population is affected by PKD, which puts them at increased risk for failure. PKD can lead to liver complications. However, in the early stages of kidney life, the number of cysts is inseparable and they should all be considered harmless cysts. This distinction is only possible at later stages of kidney development (Dond *et al.* 2021).

The most common symptoms reported were headache, back and side discomfort, and back and side pain. The pain may only last for a short time or it may last for a long time, and its intensity may vary from mild to severe. Imaging techniques are used in the diagnosis. CT is now the most common type of diagnostic imaging and has replaced MRI as the most common form of diagnostic imaging. Depending on the circumstances, damage and evolution can take an infinite number of different forms and configurations. Results may vary depending on the age of the patient at the time of surgery. To make a definitive diagnosis, genetic tests that check samples for mutations are finally done. This is usually how you do it when dealing with chromosomal genes (Torres and Harris 2019).

2.2.6 Testing techniques

A diverse array of symptoms is indicative of disease. A discussion of the clinical manifestations of CKD will follow in the next paragraphs of this section. It is occasionally important to postpone treatment planning in order to prepare for dialysis, which has been more prevalent in recent years. However, the kidney function test that is most frequently utilized is the plasma creatinine test. The test is conducted as an inpatient procedure in a hospital as part of a battery of exams that come after a string of operations or outpatient trips to a clinic. On the other hand, walking or coughing can be used to identify COPD. It is unclear that in this scenario, without doing blood or urine tests, the degree of chronic renal disease can be determined (Cornec-Le Gall *et al.* 2019).

A test of the urine may establish whether or not CKD is present as well as its degree of severity. During their monthly cycle and for the two to three days immediately after it, it is advised that women take a test using fresh, "mid-stream" pee. Because of this, the

likelihood of accidently contaminating the urine with cocaine when the temperature is between 2 and 8 degrees Celsius is significantly reduced. A change in urine color can be seen to indicate the severity of chronic kidney disease. Dipsticks can be used to assess the pH, hemoglobin, glucose, protein, leukocyte esterase, nitrites, and ketones among other chemical components of urine. These chemical properties can be used to help diagnose diseases (Douguet *et al.* 2019).

On the other hand, it is hypothetically plausible to imagine or suggest the use of microscopic hematuria as a diagnostic tool for human chronic renal disease. For illustration and explanation, more than 95% of individuals who are in perfect health do not exhibit any symptoms or indications of the presence of red blood cells during the process of urination. When there are elevated levels of a blood component (red blood cells) in the urine, it may be pink or even scarlet in human urine., are all potential methods for determining whether or not a patient has chronic kidney disease (Menezes and Germino 2019).

2.3 Corona Virus

2.3.1 Backgroun

The disease or viral infection caused by the Coronavirus 2019 (COVID-19), which is the virus that invaded the world in 2019, is an infectious disease caused by the Coronavirus-2. It is called Coronavirus Disease 2019 (COVID-19) (Figure 2.2). (Diao *et al.* 2021).

There is, however, a growing body of research revealing that it has strong correlations with certain clinical problems such as type diney diseases. One such ailment is type diney disease. Despite the fact that it is anticipated that people with COVID-19 disease will experience less than a 6 percent overall mortality rate, people with diabetes have a higher risk of dying. People who have diabetes have a greater risk of dying from other causes (Bormashenko *et al.* 2021).

Nevertheless, in order to corroborate these results, further research is required. Despite this, there is evidence that diabetes puts patients at a greater risk of developing issues related to COVID-19 (Rabb 2020).

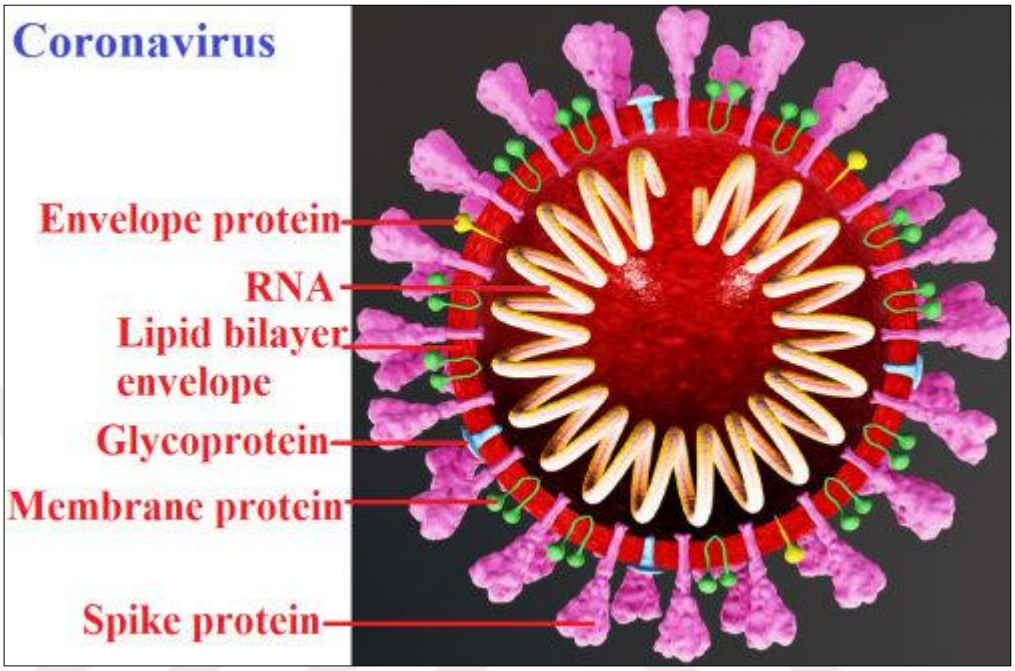

Figure 2.2 Schematic of corona virus (Bormashenko *et al.* 2021)

2.3.2 Mechanisim

There are several different ways that viruses may spread from one host to another. Insects that consume seedlings can spread disease-causing organisms and viruses, such as aphids in plants, from one plant to another. This is one of the ways in which the sickness may be transmitted. Another approach is by the use of a carrier. Sneezing and coughing are two of the most common ways that influenza viruses are passed from person to person. The fecal-oral channel, hand-to-mouth contact, feces, water, and hand-to-mouth contact are the primary modes of transmission for noroviruses. The fecal-oral route is another method by which the rotavirus, which is one of the most common viral causes of gastroenteritis, may spread. Less than 100 particles of norovirus per milliliter of body fluid are required to cause infection in humans. This is the infection threshold for humans (Zhang and Liang 2020).

When animals are exposed to viruses, their bodies mount an immunological response that, in the vast majority of instances, clears the illness completely. Vaccines that have been genetically modified to give protection against a specific viral illness may be able to stimulate the body's immune response to fight off that virus. There are certain viruses, such as those that are responsible for causing acquired immunological disorders and illnesses caused by human papillomavirus, that have the potential to remain in the body and cause infections, such as viral hepatitis. There has been progress made toward the creation of antiviral medicines (Strich *et al.* 2022).

2.3.3 Symptoms

Symptoms of COVID-19 often show between 4 and 7 days after the virus has been infected. In this particular research, 14 days was shown to be the median amount of time that passed before the symptoms of COVID-19 led to fatalities. The length of the treatment depends on a number of factors, including the patient's age and immune system condition. Patients who were older than 70 years old had a lower life expectancy than those who were younger than 70 years old. An infection with COVID-19 may cause a variety of symptoms, including but not limited to the following: fever, cough, drowsiness, sputum production, nauseousness, vomiting, headache, hemoptysis, diarrhea, dyspnea, and lymphopenia are some of the symptoms. Despite the fact that the patient's chest CT scan revealed they had pneumonia, it also showed that the patient had grand-glass opacities, RNA anemia, acute respiratory distress syndrome, sudden cardiac death, and other conditions that all contributed to the patient's passing (Kalbande and Muntode 2022).

In spite of the fact that the trial participants exhibited upper respiratory symptoms such as rhinorrhea, sneezing, and sore throat, COVID-19 did not have any effect on the lower airway. It is essential to keep in mind that individuals diagnosed with COVID-19 had gastrointestinal symptoms including diarrhea, but patients diagnosed with MERS-CoV or SARS-CoV did not have these symptoms. As a result of this, samples of feces and urine need to be analyzed in order to rule out any other probable transmission routes. These pathways might include healthcare staff, patients, and other persons. It is thus essential to

create technology that can detect the multiple routes of transmission, such as samples of feces and urine, in order to develop preventative and/or reduction tactics, as well as medications to treat and manage the illness (Hosseini *et al.* 2022).

2.3.4 Transmission

At a seafood market in Wuhan, China, direct contact with sick animals is thought to have been the cause of the first known cases of the illness (animal-to-human transmission). On the other hand, clinical examples that have been recorded have had a broad variety of different exposure histories. This lends greater support to the idea that the virus could spread through the air from one person to another. Human-to-human transmission is presently the mode of transmission that is currently the most prevalent in history as a direct result of this. Asymptomatic persons pose a possible threat to public health because they might transfer the disease to others (Kumar *et al.* 2022).

According to the findings of the study, the transmission of illness between people may occur when they are in close, physical contact with one another. This also boosts the chance of transmission in such situations because of the high aerosol concentrations that are present in limited places. This indicates that a patient has the capability of transmitting the virus to two other people in addition to themselves. According to the most recent scientific research, the incubation period for the virus might last anywhere from three to seven days. The preceding instances served as the foundation for these discoveries, which are based on those examples. In addition, more research is necessary to get a deeper comprehension of the dynamics of transmission and the length of the incubation period.

3 MATERIALS AND METHODS

In order to understand and study the variables that can occur to patients with Covid 19, we have studied a set of variables on a number of patients and compared them with normal people without the virus. To the results, conclusion and recommendations.

3.1 The Instruments

The instruments listed below devices that can be used in the preparation of the search: -

A- polymerase chain reaction (PCR)

B- Elisa

C-Spectrophotometer cobas c111 D-Centrifuge

D- and other instruments

3.2 Blood sample and Chemicals Used

About Ten milliliters of venous blood will be drawn and put in an anti-coagulated tubes containing sodium EDTA and the second plain tube for serum will be taken from all participants and sera will be separated by centrifugation at 1500xg for 10 min at $4c^0$, and divided into small aliquotes, and stored at -20c until its use for the following analyses

3.3 Biochemistry Study

Elisa kits will be used for the determination of Angiotensin II and biochemical kit to estimation Blood Urea, Creatinine, proteinuria, and other parameter. Molecular study of covid-19: COVID-19 will extraction DNA according to manufacturer instructions (Positive smear results were obtained from samples of people infected with the Corna virus after conducting a molecular diagnosis by the central laboratory).

3.4 Subjects and Methods

A total 100 subjects will be enrolled in this study.

Group A: includes 55 covid -19 Kidney disease patients an age range of 15 to 70 year. Group B: include 50 normal control.

3.5 The Tests

The tests listed below will be carried out in the research work according to the mode of operation specified by the manufacturer:-

A-Dtection of covid -19

B- Dtection of Angiotensin Enzyme

C-Blood Urea

D-Serum creatinine

E- proteinuria.

3.6 The Angiotensin II Enzyme (Product Number (Sigma): RAB0010)

The array angiotensin II immunoassay (EIA) is a quantitative in vitro assay based on the principles of a competitive enzyme immunoassay for angiotensin II peptides. EIA-ANGII-1 can theoretically detect all active angiotensinogens including ANGI ANGII ANGIII and ANGIV. However no inactive angiotensinogen was detected.

3.6.1 Biochem/physiol actions

Examination of in vitro models showed that mechanical stretch-induced Ang II release plays an important role in regulating the stretch-induced hypertrophic response. Ang II contributes significantly to the pathogenesis of hypertension and cardiovascular disease and is engaged in blood pressure regulation. Additionally, vascular smooth muscle cell

migration, development, and mitosis are all significantly influenced by Ang II. It causes the thickening and fibrosis of blood vessel walls and heart muscle by increasing the production of type I and III collagen in fibroblasts. Variations in the Ang II gene increase the risk of hypertension myocardial infarction and left ventricular hypertrophy. Ang II expression is associated with proliferative and inflammatory responses to diabetic heart failure cardiac remodeling and arterial injury. Drugs such as angiotensin II-converting enzyme inhibitors or angiotensin II receptor antagonists can be used to treat Ang II-induced hypertension and heart failure.

3.6.2 Kit components

20X Wash Buffer (Item B) Sigma RABWASH3, EIA Angiotensin II Peptide standard (Item C) Sigma RAB0010C, EIA Assay Diluent A (Item D) Sigma RABDIL9 EIA 5X Assay Diluent B (Item E) Sigma RABDIL10, Anti-Angiotensin II Detection Antibody (Item N) Sigma RAB0010F, Biotinylated Angiotensin II Peptide (Item F) Sigma RAB0010G, HRP-STREPTAVIDIN Sigma RABHRP3, TMB Substrate solution (Item H) Sigma RABTMB2, Stop Solution (Item I) Sigma RABSTOP2, Met. Corr. 1; H290 Angiotensin II Positive Control Sample, Lyophilized (Item M) Sigma RAB0010K, EIA Angiostensin II 1x Assay Diluent E.

3.6.3 Sandwich method assay and procedure it

1. Place all reagents and the rest of the supplies and samples at room temperature (approximately 20-25 degrees Celsius) before starting use. It is recommended that all standards for testing (Fig. 3.1) and samples be done in at least twice in order to increase accuracy.
2. Add 100 µl of each test standard and study sample into the appropriate wells. All wells should be covered and incubated for about 2.5 hours at 25°C or overnight at 4°C and should be with gentle shaking.
3. Wash 4 times with 1X kit washing solution. Wash by filling each well with Wash Buffer (300 µL) using an automated kit or a multichannel pipette. The washing

process is the complete removal of the liquid and the rest of the plankton at each step necessary for performance and good results or good results. After washing and applying the last wash, any residual detergent or washing-up liquid should be removed by blotting, vacuuming, or pouring, using blotting paper. The plate should be turned over and wiped appropriately with clean paper towels (blotting paper).

4. After the washing and drying process is completed, we proceed to the next step. 100 µL of 1x congated prepared for each well is added in order to complete the reaction. After this step, we cover the wells and incubate them for an hour (60 minutes) at a temperature of 25 degrees, while shaking them gently in order to ensure homogeneity.
5. After this step, and in order to ensure good results, we repeat the washing procedure, and the washing process must be as we mentioned in the third step.
6. Then we add 100 microliters of streptavidin solution prepared in the correct way to all samples. Then we cover the wells in all wells of the study and incubate them for forty-five minutes at a temperature of 25 (room) with gently shaking them in order to ensure homogeneity.
7. After this step, and in order to ensure good results, we repeat the washing procedure, and the washing process must be as we mentioned in the third step.
8. We add 100 µl of TMB (staining) one-step substrate reagent (item H) to each well of the study wells. We cover the wells of the standards and samples and incubate them for thirty minutes at room temperature (25°C) in the dark, with gently shaking them in order to ensure homogeneity.
9. We repeat the addition, but here we add 50 µL of the reaction stop solution (item 1) to each well of the study wells. The absorbance reading at 450 nm is done immediately after this step in order to ensure accuracy.

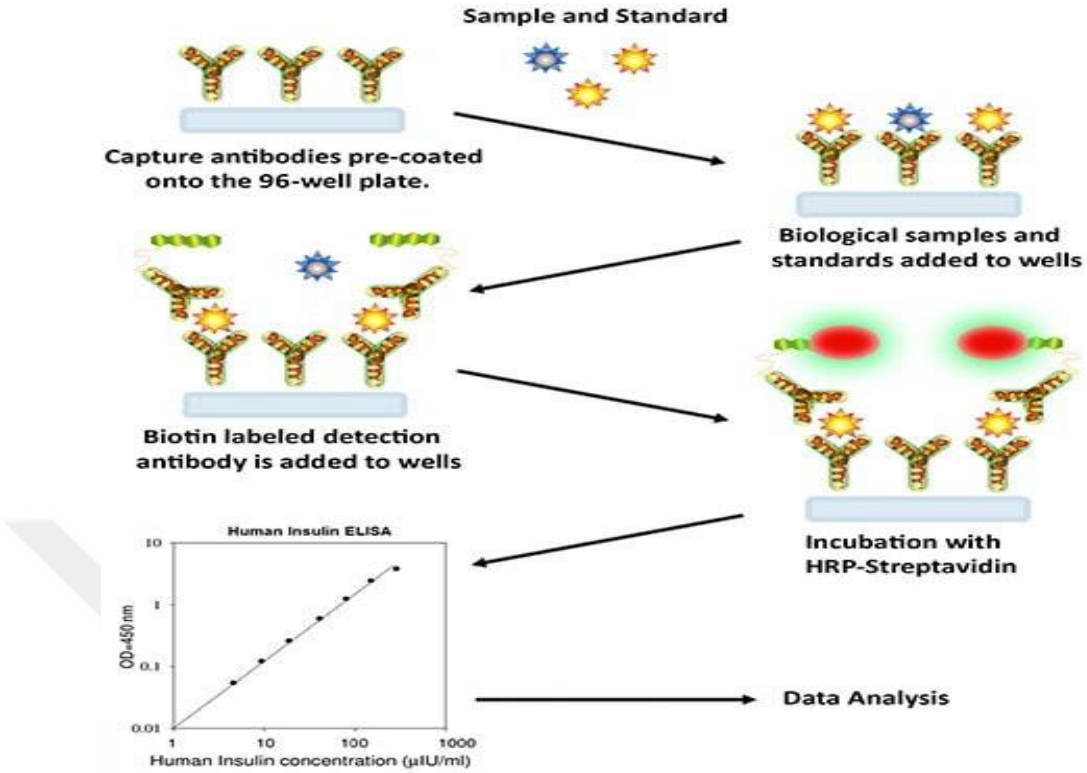

Figure 3.1 The sample and standard curve for AGEII

3.7 Urea Assay Kit

to quickly, accurately, and sensitively quantify the urea concentration of different samples. This item should only be used for study and not for medical diagnosis. Stability and storag: We store the kits upon arrival in the dark at 4°C. The shelf life of the set is 1 year from the date of receipt unless the parts are replaced. Some work classes before teaching at the university. The obtained material is stable for two month.

3.7.1 Serum, plasma and urine samples

1. Serum and urine samples can be tested directly by adding sample to the wells.

2. Serum and plasma = we recommend to use 0.1 – 1 µL of plasma or serum.

3. Urine = we recommend further dilutions of 1/1000 – 1/50000 µL per well.

3.7.2 Assay procedure and detection

Before use, combine all of the ingredients and the produced reagents at room temperature. It is advised to run all standard controls and samples through two tests. Mix the necessary ingredients to conduct several experiments (sample standards and background controls). To achieve homogeneity, prepare a master mix of the reaction mixture. The calculation listed below is what we advise. Component XL x (number of standard samples 1). 1. Mix each standard with 50 mL of the reaction mixture. Fill the background sample control wells with 50 L of the background reaction mixture. For 60 minutes, mix and incubate at 37°C in a light-protected environment. Utilize a microplate reader to measure the result (OD570 nm). Calculations: Samples that provide a signal that is higher than the ideal standard should be further diluted by reanalyzing the proper buffer and multiplying the concentration by the proper dilution factor.

3.7.3 Sample preparation

It is advisable to dilute the sample several times and make different amounts to ensure the reading is within the standard value range and to use a fresh sample. Complete sample preparation steps before storing samples if analyzes cannot be run simultaneously.

If this is not possible we recommend freezing the cells or tissues in liquid nitrogen after extraction and storing the samples at -80 °C. When ready to test thaw the test piece on ice. This can affect the stability of the sample. The reading may be lower than expected.

3.8 Comparison and Selection of Methods

As for the rest of the all tests, they were conducted according to the instructions of the producing companies.

3.9 Statistical Analysis

In order to understand and interpret the results of our study, statistical analysis was used, SPSS 25. The means and standard deviation of the variables were found by using the t-test, and in order to find the correlation rate, Pearson's test was used. The level of significance in the study was at $P < 0.05$.

4. RESULTS AND DISCUSSION

A total 105 subjects were be enrolled in this study. Patient group: includes 55 covid -19 kidney disease patients an age range of 15 to 70 year. Control group: include 50 normal controls. The aim of this study is to evaluate the importance of Angiotensin Enzyme, And Other Parameters with Covid 19 And İts Effect on Kidney Disease in relation to its validity in early detection and severity of disease. Angiotensin Enzyme and is involved in many organs of the body such as the, kidneys, heart, lungs and other organs, but we will study its relationship with kidney disease and Covid -19 patient The potential relationship between Angiotensin and Covid 19 and other parameters in kidney disease will be investigated.

4.1 Results

4.1.1 Angiotensin II results with age

The results of the relationship of angiotensin enzyme with age with Covid 19 disease and its effect on kidney disease indicate that the virus infects elderly people as in the group of patients (52.8667) when compared with the control group (34.4667) more than young people at P = <0.05, and that there was a direct relationship between the infection rate the disease in the elderly and changes in the level of angiotensin enzyme as in Table 4.1 and Figure 4.1.

Table 4.1 The results of the relationship of angiotensin enzyme with age

Group Statistics						P-value
	Group	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
Age	Control group	50	34.4667	9.62041	2.48398	0.001
	Patient Group	55	52.8667	11.88557	3.06884	0.001
Pearson Correlation		0.397*				0.001

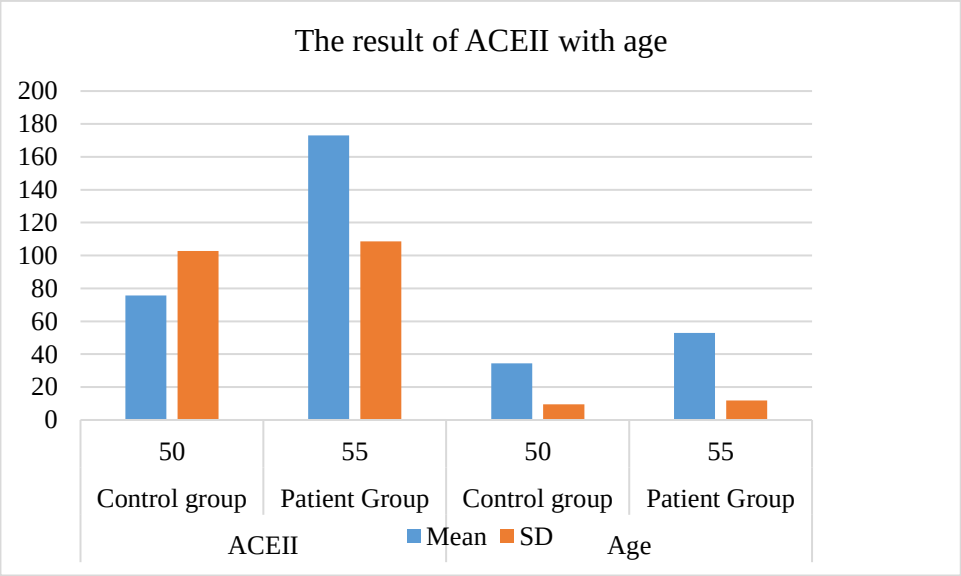

Figure 4.1 The result of ACEII with age

4.1.2 Angiotensin enzyme results with weight

The results of weight results and a relationship with the angiotensin enzyme, which are mentioned in Table 4.2 and Figure 4.2, indicate that weight was not a major factor in the development of kidney failure in people who were infected with Covid 19 virus, as there were very slight differences in the averages of the group of patients (89.2667) when compared In the control group (86.5333) at P = <0.05, and there was no relationship between the average weight and changes in the level of angiotensin enzyme.

Table 4.2 The results of the relationship of angiotensin enzyme with weight

Group Statistics						
	Group	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**P-value**
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
Weight	Control group	50	86.5333	6.62103	1.70954	0.335
	Patient Group	55	89.2667	8.51441	2.19841	0.335
Pearson Correlation		0.564**				0.001

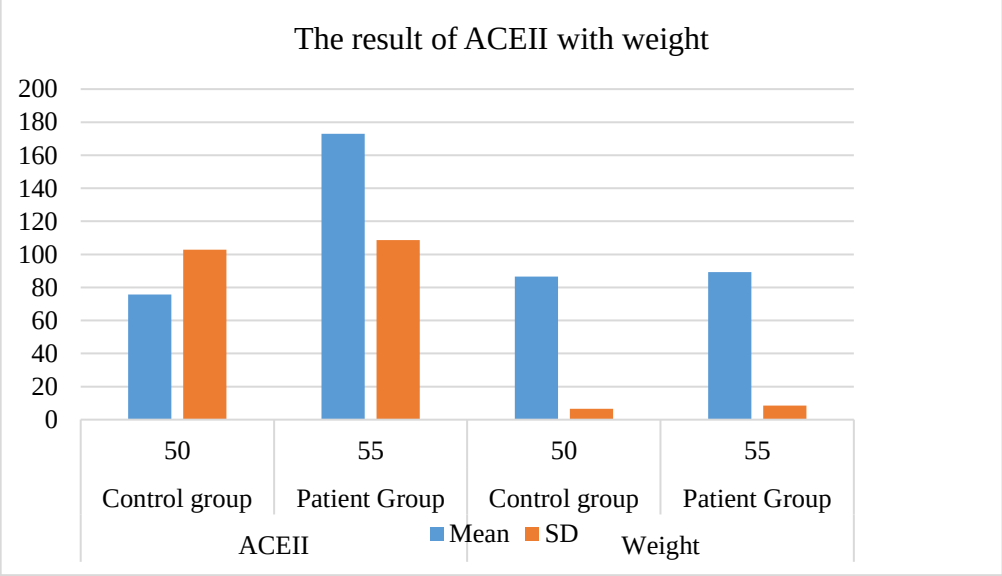

Figure 4.2 The result of ACEII with weight

4.1.3 Angiotensin enzyme results with body mass index

As for the body mass index and the relationship to the angiotensin enzyme, which is mentioned in Table 4.3 and Figure 4.3, indicating that there were slight changes and were not statistically significant when compared to the control group, and it was also not a major factor in the development of kidney failure in people who were infected with the Covid virus. 19, where there were very slight differences in the averages of the group of patients (25.2000) when compared with the control group (24.7575) at P = <0.05, and there was a slight direct relationship between body mass index and changes in the level of angiotensin enzyme.

Table 4.3 Angiotensin enzyme results with body mass index

Group Statistics						
	Group	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**P-value**
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
	Control group	50	24.7575	3.62693	.93647	0.701

BMI	Patient Group	55	25.2000	3.42679	.88479	0.701
Pearson Correlation		0.421*				0.020

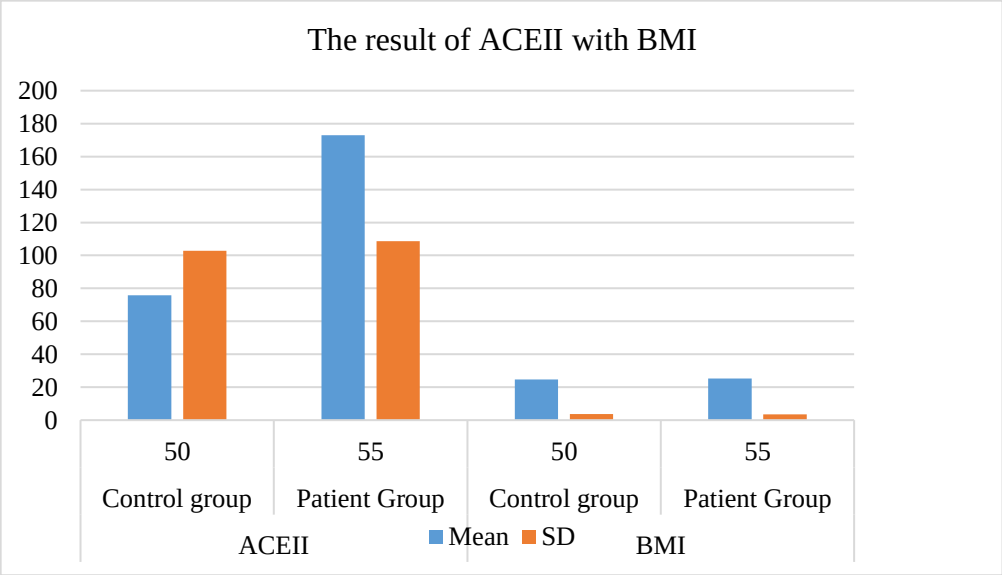

Figure 4.3 The result of ACEII with BMI

4.1.4 Results of angiotensin enzyme with urea

Also, the results of the relationship of the angiotensin enzyme in Table 4.4 and Figure 4.4 with urea with Covid 19 disease and its effect on kidney diseases indicate that urea averages did not change much, which is one of the diagnostic tests for kidney patients and did not have a clear clinical significance. To acute kidney failure, where there were very slight differences within the normal level in the averages of the group of patients (41.0667) when compared with the control group (30.9032) at P = <0.05, and there was a direct relationship between the urea rate and changes in the level of angiotensin enzyme.

Table 4.4 Results of angiotensin enzyme with urea

Group Statistics						
	Group	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**P-value**
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
Urea	Control group	50	30.9032	11.20902	2.89416	0.037
	Patient Group	55	41.0667	5.90964	1.52586	0.037

Pearson Correlation	0.424*	0.020

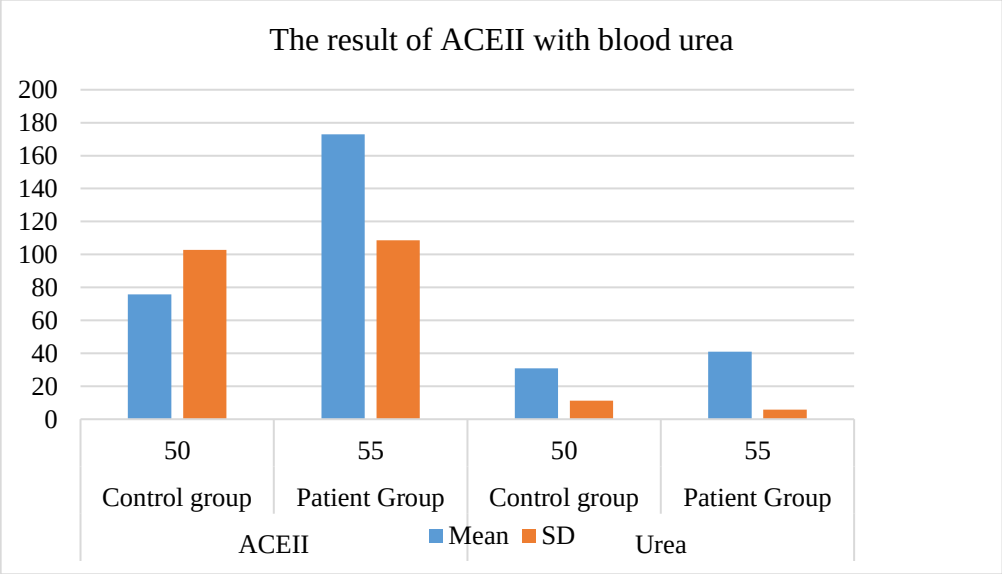

Figure 4.4 The result of ACEII with urea

4.1.5 Results of angiotensin-enzyme with creatinine

As for serum creatinine and angiotensin-enzyme relationship, which is mentioned in Table 4.5 and Figure 4.5, which is the second important test in diagnosing kidney failure, its results indicated that there were no significant statistically significant differences with Covid 19 disease, as there were very slight differences within the normal level. In the averages of the patient's group (0.8558) when compared with the control group (0.8558) at b = 45, and there was an inverse relationship between the creatinine level and changes in the level of angiotensin enzyme.

Table 4.5 Results of angiotensin-enzyme with creatinine

Group Statistics						
	Group	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**P-value**
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
Creatinine	Control group	50	0.9131	.36042	.09306	0.062
	Patient Group	55	0.8558	.29458	.07606	0.062
Pearson Correlation			0.248			0.186

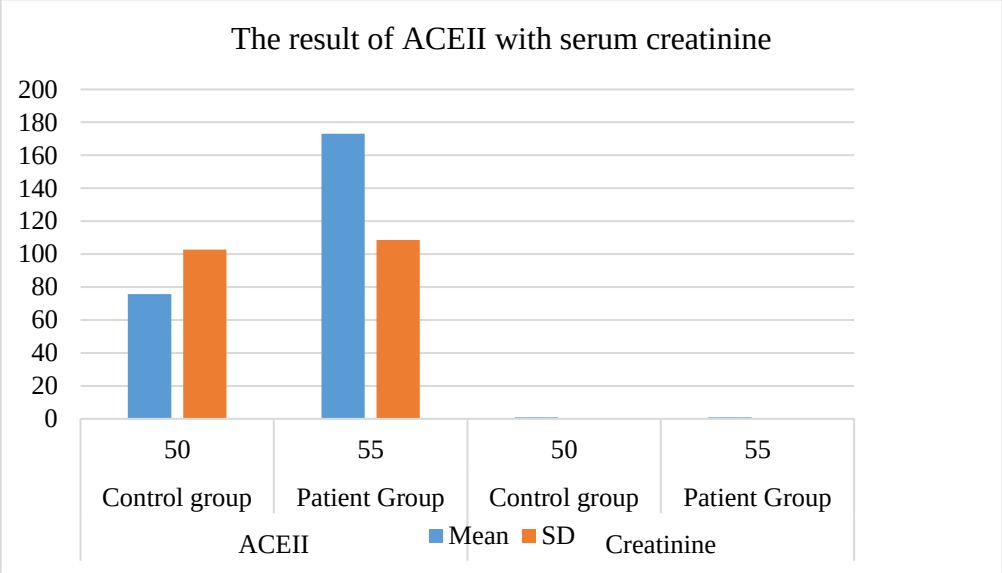

Figure 4.5 The result of ACEII with creatinine

4.1.6 Results of angiotensin enzyme with total cholesterol

The results of our study also indicated that the averages of total cholesterol and the relationship with the angiotensin enzyme mentioned in Table 4.6 and Figure 4.6, indicating that there were changes of statistical significance when compared to the control group, and it is possible that the increase in cholesterol metabolism is a factor in the development of kidney failure. In people who were infected with Covid 19 virus, where there were slight differences in the averages of the patients group (208.0667) when compared with the control group (187.2000) at P = <0.05, and there was a direct relationship between the total cholesterol rate and the changes in the angiotensin enzyme level.

Table 4.6 Results of angiotensin enzyme with total cholesterol

Group Statistics						
	Group	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**P-value**
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
Tc	Control group	50	187.2000	25.25923	6.52190	0.048
	Patient Group	55	208.0667	61.81247	15.95991	0.048
Pearson Correlation		-0.130				0.492

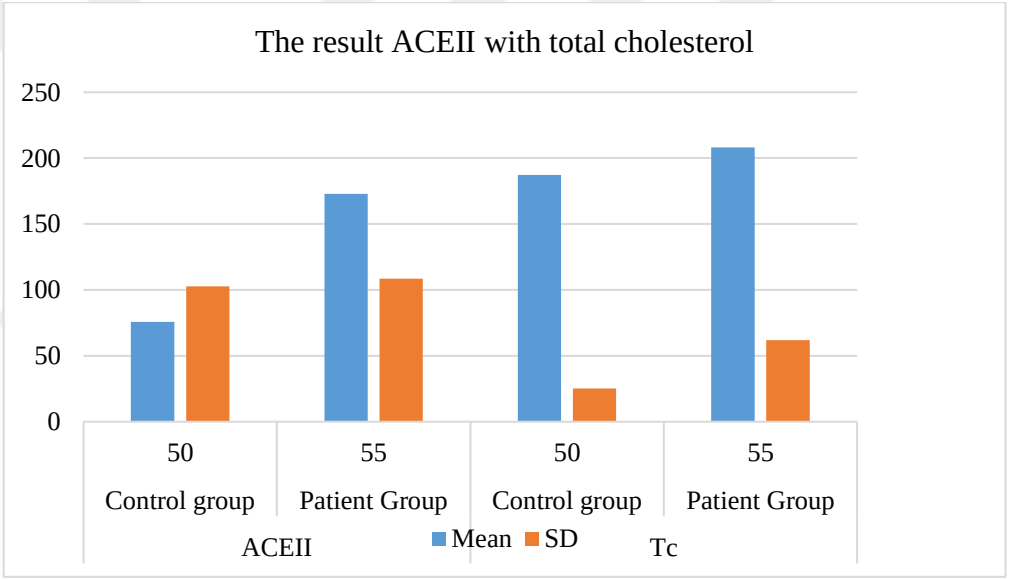

Figure 4.6 The result of ACEII with cholesterol

4.1.7 Results of angiotensin enzyme with triglycerides

Also, the results of the relationship of the angiotensin enzyme with triglycerides in the disease of Covid 19 and its effect on kidney diseases indicated that the averages of triglycerides did not change and did not have a clear clinical significance, as there was a clear equality in the averages of the group of patients (108.4667) when compared with the control group (105.8667) at P = <0.05, and there was no clear relationship between

the rate of triglycerides and changes in the level of angiotensin enzyme as in Table 4.7 and Figure 4.7.

Table 4.7 Results of angiotensin enzyme with triglycerides

Group Statistics						
	Group	N	Mean	Std. Deviation	Std. Error Mean	**P-value**
ACE II	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
Tg	Control group	50	105.8667	14.71086	3.79833	0.951
	Patient Group	55	108.4667	19.02204	4.91147	0.951
Pearson Correlation		0.467**				0.009

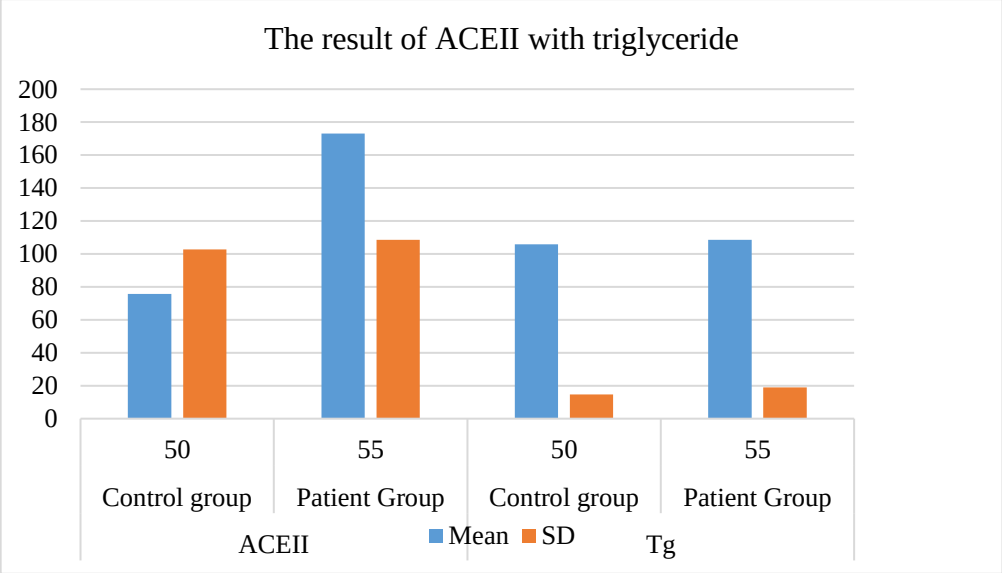

Figure 4.7 The result of ACEII with triglyceride

4.1.8 Angiotensin II results with high-density lipoprotein

The results also indicated in Table 4.8 and Figure 4.8 that the averages of high-density lipoprotein and angiotensin-releasing enzyme were related, indicating that there were no statistically significant changes when compared to the control group, and they had no role in the development of kidney failure in people who were infected with the Covid virus. 19 According to the results of our study, where there were no differences between the averages of the patient group (51.2667) when compared with the control group (48.2655)

at P = <0.05, and there was no relationship between the rate of high-density lipoprotein and changes in the level of angiotensin enzyme.

Table 4.8 Angiotensin II results with high-density lipoprotein

Group Statistics						
	Group	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**P-value**
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
HDL	Control group	50	48.2655	8.42989	2.17659	
	Patient Group	55	51.2667	8.48079	2.18973	
Pearson Correlation					-0.182	0.336

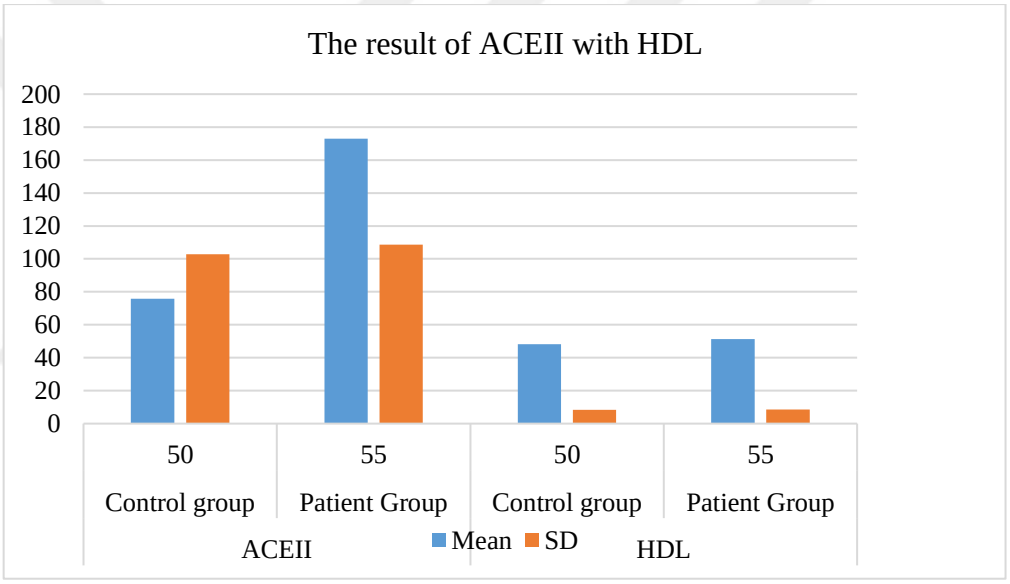

Figure 4.8 The result of ACEII with HDL

4.1.9 Results of angiotensin enzyme with LDL

The results of the relationship of the angiotensin enzyme with LDL in the disease of Covid 19 and its effect on kidney disease indicate that the averages of LDL change very slightly and had no clear clinical significance, as there was a clear convergence in the averages of the group of patients (135.1067) when compared with the control group (108.7611) at b = 45 And that there was a slight direct relationship between LDL rate and changes in the level of angiotensin enzyme as shown in Table 4.9 and Figure 4.9.

Table 4.9 Results of angiotensin enzyme with LDL

Group Statistics						
	Group	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**P-value**
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
LDL	Control group	50	108.7611	18.26739	4.71662	0.058
	Patient Group	55	135.1067	59.84150	15.45101	0.058
Pearson Correlation		-0.049				0.796

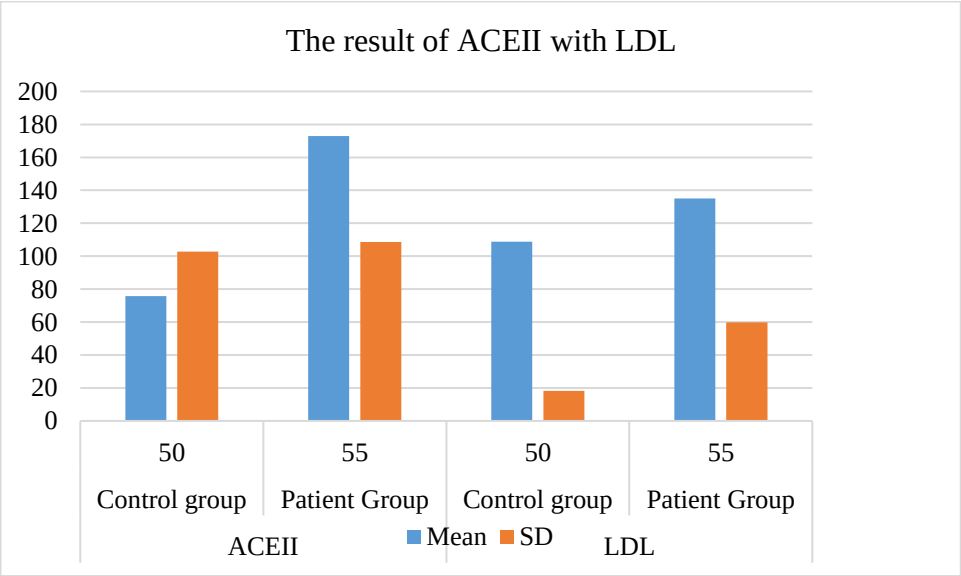

Figure 4.9 The result of ACEII with LDL

4.1.10 Results of the angiotensin enzyme with very low-density lipoprotein

The results in Table 4.10 and Figure 4.10 indicate that there were no statistically significant or significant differences between the averages of very low-density lipoprotein and angiotensin-enzyme relationship, indicating that there were no statistically significant changes between the group of patients (21.6933) when compared to the control group (21.1733), and it has no role in the development of kidney failure in people who were infected with Covid 19 virus, according to the results of our study, at P = <0.05, and there was no relationship between the rate of very low-density lipoprotein and changes in the level of angiotensin enzyme.

Table 4.10 Results of the angiotensin enzyme with very low-density lipoprotein

Group Statistics						
	Group	N	Mean	Std. Deviation	Std. Error Mean	**P-value**
ACEI I	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
VLD L	Control group	50	21.1733	2.94217	.75967	0.679
	Patient Group	55	21.6933	3.80441	.98229	0.679
Pearson Correlation		0.467**				0.009

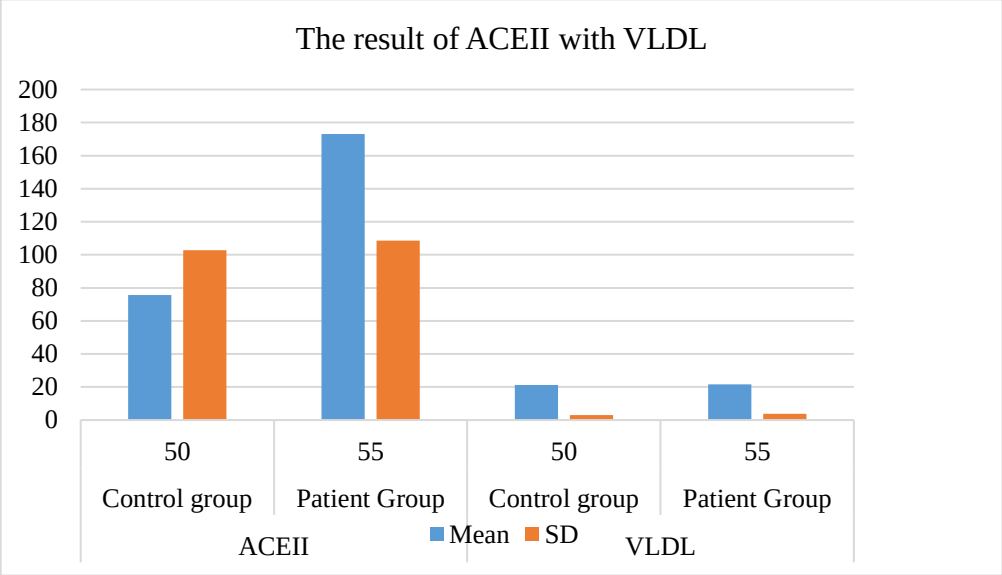

Figure 4.10 The result of ACEII with VLDL

4.1.11 Angiotensin enzyme results with D-Dimer

The results in Table 4.11 and Figure 4.11 indicate that D-Dimer, which is considered one of the most important tests in diagnosing and following up on infection with Covid 19 virus, according to the results of our study and other studies that were mentioned in the section discussing results, indicates that there is a statistical significance and significant differences between the averages D-Dimer is very high and it was associated with a strong relationship with angiotensin enzyme, indicating that there were statistically significant changes between the group of patients (2665.0667) when compared to the control group (853.4000), at P = <0.05, and there was no strong direct relationship between the rate of D- Dimer and changes in the level of angiotensin enzyme.

Table 4.11 Angiotensin enzyme results with D-Dimer

Group Statistics						
	Group	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**P-value**
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
D-Dimer	Control group	50	853.4000	1095.18862	282.77649	0.000
	Patient Group	55	2665.0667	1197.83933	309.28079	0.000
Pearson Correlation		0.617**				0.000

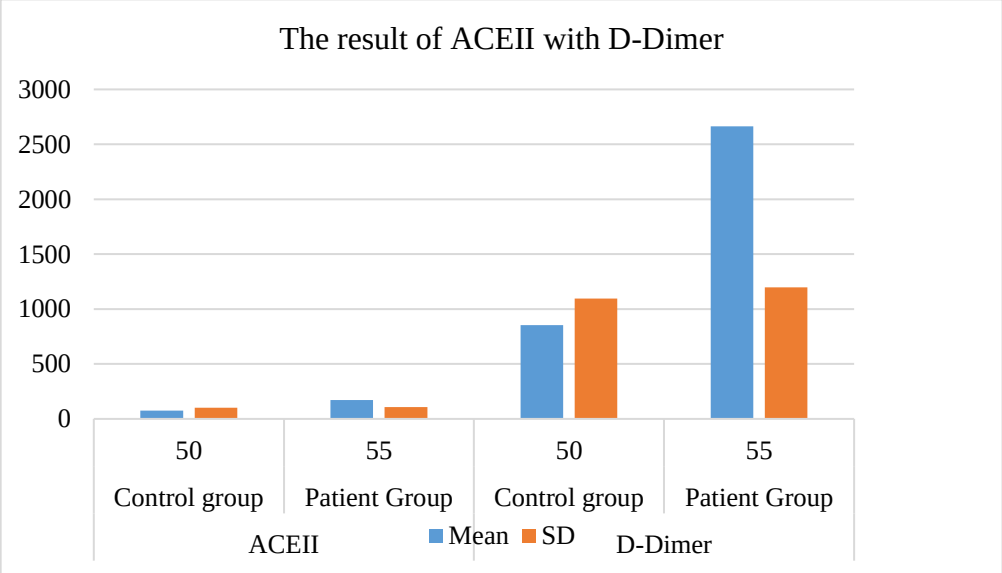

Figure 4.11 The result of ACEII with D-Dimer

4.1.12 Results of angiotensin II with ınterleukin 6

The results of the relationship of the angiotensin enzyme with interleukin-6 with the disease of Covid 19 and its effect on kidney diseases indicate that the averages of interleukin-6 change very significantly, as there was a significant increase in the averages of the patients group (144.4667) when compared to the control group (60.9674) at P = <0.05, and that it was There is a direct relationship between the rate of interleukin-6 and changes in the level of angiotensin enzyme as in Table 4.12 and Figure 4.12.

Table 4.12 Results of angiotensin I with interleukin 6

Group Statistics						
	Group	N	Mean	Std. Deviation	Std. Error Mean	**P-value**
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
IL6	Control group	50	60.9674	107.62107	27.78764	0.020
	Patient Group	55	144.4667	75.04653	19.37693	0.020
Pearson Correlation		0.534**				0.002

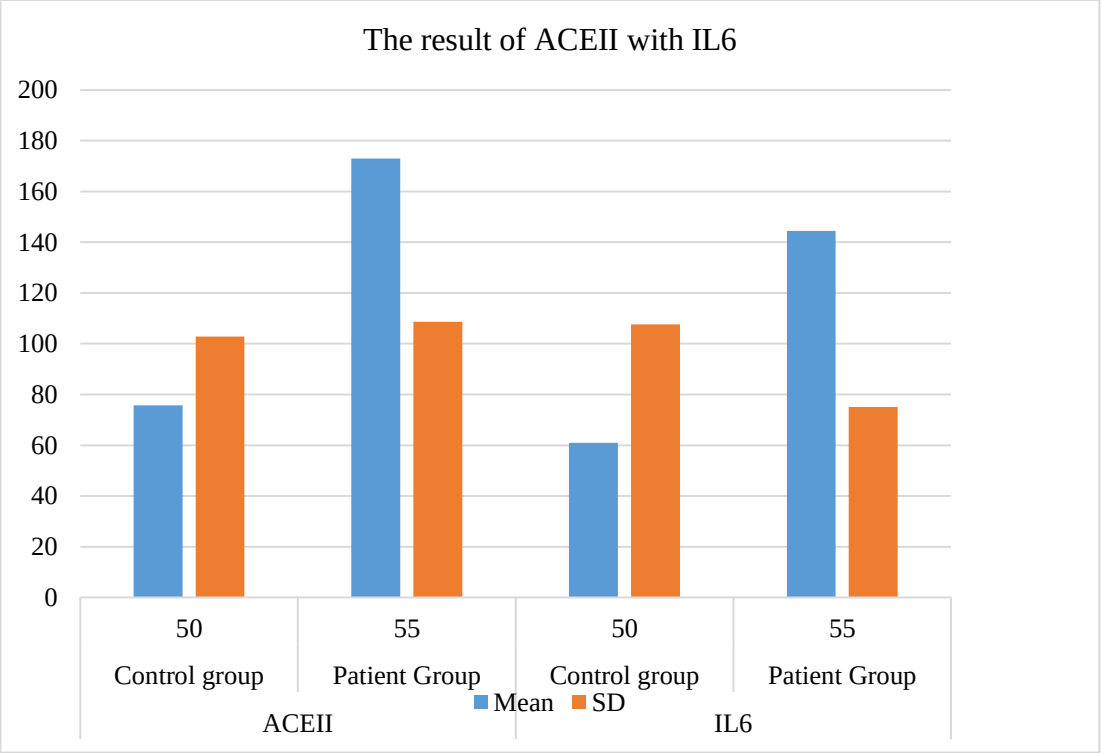

Figure 4.12 The result of ACEII with IL6

4.1.13 Results of the ACEII with white blood cells and hemoglobin

The results of the relationship of the angiotensin enzyme with white blood cells and hemoglobin in Covid 19 disease and its effect on kidney disease indicate that the averages of white blood cells and hemoglobin did not change much, as there were slight changes towards the increase for white blood cells and towards the decrease in respect of hemoglobin As in the group of patients (9.5332 and 12.5341) when compared with the control group (8.1328 and 13.3322) at P = <0.05 and there was a direct relationship between the occurrence of the disease and changes in the level of white blood cells and hemoglobin with angiotensin enzyme as in Table 4.13 and Figure 4.13.

Table 4.13 Results of the ACEII with white blood cells and hemoglobin

Group Statistics						
	Group	N	Mean	Std. Deviation	Std. Error Mean	**P-value**
ACEII	Control group	50	75.7153	102.79370	26.54122	0.018
	Patient Group	55	173.0000	108.62780	28.04758	0.018
WBC	Control group	50	8.1328	1.95015	.50353	0.109
	Patient Group	55	9.5332	2.61940	.67633	0.109
Pearson Correlation		-0.118				0.534
Hb	Control group	50	13.3322	1.25941	.32518	0.101
	Patient Group	55	12.5341	1.31810	.34033	0.101
Pearson Correlation		-0.193				0.307

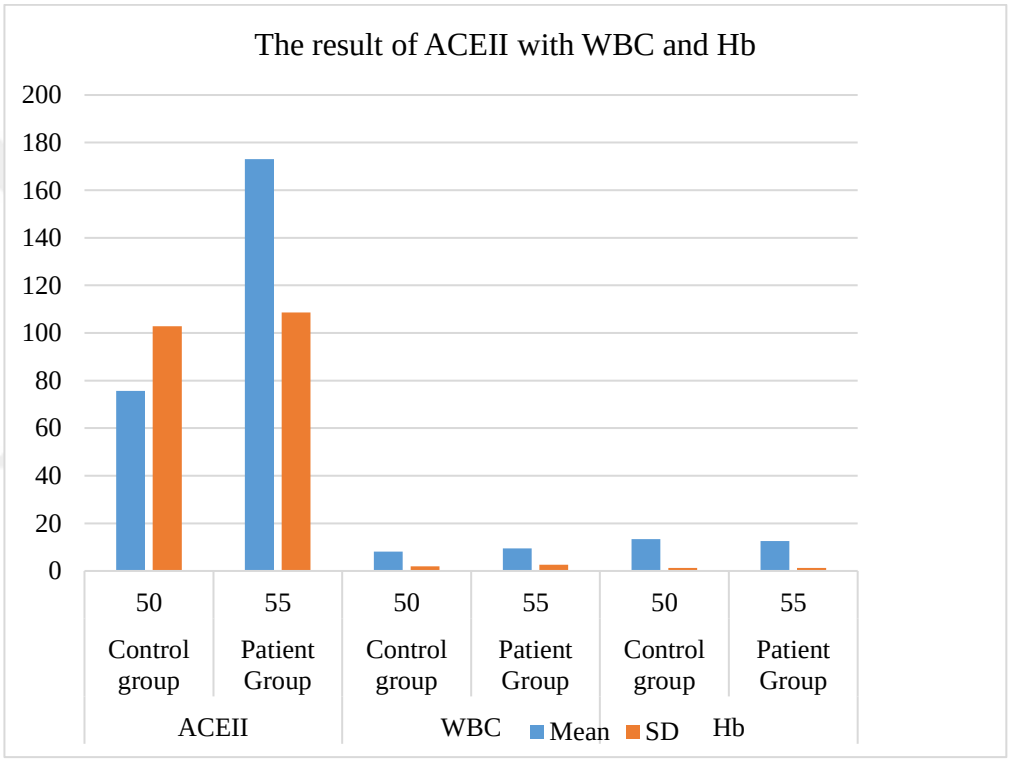

Figure 4.13 The result of ACEII with WBC and Hb

4.2 Discussion

In the year The SARS-CoV-2 pneumonia outbreak that started in Wuhan China in late 2019 has become a global pandemic that has spread to countries around the world. Both SARS-CoV-2 and SARS-CoV enter host cells via the angiotensin-converting enzyme 2 (ACE2) receptor which is expressed in various human tissues. ACE2 converts angiotensin II to angiotensin (1-7) and ACE2 / angiotensin- (1-7) / MAS axis prevents the negative effects of the renin-angiotensin system (RAS) which maintains physiological conditions. Pathophysiological balance of the body. In addition to direct viral inflammation and immune factors associated with the pathogenesis of Covid-19 post-infection reduction of ACE2 levels and imbalance between RAS and ACE2/angiotensin-(1-7)/MAS may contribute. The pathogen of covid-19. The SARS-CoV-2 spike glycoprotein that binds to ACE2 is a target for the development of drug-specific antibodies and vaccines. Free viruses and macrophages can spread to other organs and localize ACE-2-expressing cells to cause multiorgan infections (Ni *et al.* 2020).

Angiotensin-converting enzyme 2 (ACE2) has emerged as a key regulator of the renin-angiotensin system in cardiovascular (CV) disease and plays a central role in infection with coronaviruses and influenza viruses. The present review focuses mainly on the results showing a role for ACE2 in the association of coronaviruses and influenza viruses with CV disease. The risk of infection with coronavirus or influenza virus is thought to be high at least in part because of the high expression of ACE2 in high CV risk populations. The use of coronavirus and influenza virus vaccines in countries at high CV risk may be a potential strategy to prevent CV disease and coronavirus/influenza virus infection (Chen and Hao 2020).

An activated renin-angiotensin system causes a thrombotic state resulting from an imbalance between coagulation and fibrinolysis. Angiotensin II is the central effector molecule of the activated renin-angiotensin system and is hydrolyzed by ACE II to angiotensin (1-7). Novel coronavirus infection (classified as COVID-19) is caused by the novel SARS-CoV-2 coronavirus and is characterized by an exaggerated inflammatory response that can lead to severe manifestations such as acute respiratory distress

syndrome sepsis and death in a percentage of patients. Mostly elderly people with pre-existing co-morbidities. SARS-CoV-2 uses the ACE 2 receptor to enter target cells resulting in activation of the renin-angiotensin system. After ACE II downregulation the vasoconstrictor angiotensin II is overproduced and its anti-angiotensin (1-7) molecules are reduced. Angiotensin II increases thrombin formation and inhibits fibrinolysis. Elevated levels were closely associated with viral load and lung damage in patients with severe COVID-19. Therefore the complex clinical picture of patients with severe complications of Covid-19 results from the different effects of highly expressed angiotensin II on thrombosis and inflammation of vasculopathy. Future treatment options should focus on blocking the anticoagulant and proinflammatory properties of angiotensin II in patients with Covid-19 (Miesbach 2020).

This meta-analysis of more than 500000 COVID-19 patients from different countries highlights an age-specific effect on mortality with relevant thresholds >50 years and especially >60 years. Elderly patients should be prioritized when implementing preventive measures (Bonanad *et al.* 2020).

Many methods have been used to combat the spread of COVID-19 but health systems (particularly intensive care units) are overwhelmed by evidence of high-capacity beds and existing ventilators. Selecting appropriate classification criteria for rare ICUs appears to be very difficult and requires a widely accepted tool for classifying patients in terms of access to rapid life-saving resources. One of these hotly debated parameters is age. In some cases it may be more appropriate to choose treatment for younger patients than for older patients. Demonstrate that age should not be used as the only criterion to withhold/not initiate life-saving interventions even in the context of epidemics and declining medical resources. This approach is based on fundamental ethical principles such as the WMA Code of Ethics and the Hippocratic Oath and should be followed by all physicians treating patients (Hostiuc *et al.* 2021).

We set out to look at independent predictors of COVID-19 disease severity and survival from routine blood measures, particularly the blood urea nitrogen (BUN)/creatinine (Cr) ratio. BUN/Cr ratio (OR = 1.70; 95% confidence interval) and neutrophil to lymphocyte

ratio (NLR) were independent predictors of disease severity in a multivariate model adjusted for potential confounders. BUN/Cr ratio and NLR were independent predictors for survival of COVID-19 disease in the multivariate Cox proportional hazard model. The best levels of the BUN/Cr ratio, with areas under the curve (AUC) of 0.98 and 0.95, respectively, were at 33.5 and 51.7, where there was a higher likelihood of severe illness and fatality. The best NLR thresholds, with AUC values of 0.87 and 0.85, respectively, had a higher likelihood of serious illness and fatality at 3.27 and 5.72. The severity and prognosis of COVID-19 patients are independently predicted by BUN/Cr and NLR. With COVID-19, routine BUN/Cr and NLR screening can assist in identifying cases that are at high risk (Ok *et al.* 2021).

The 2019 coronavirus disease or the current COVID-19 pandemic has infected more than 2 million people and killed more than 100000 at the time of writing this summary. The disease is caused by acute respiratory syndrome coronavirus 2 (SARS-CoV-2). So far many vaccines and treatments are being developed but the disease is only slowing down under extreme social distancing measures that are difficult to maintain. SARS-COV-2 is a virus enveloped by a lipid bilayer. Lipids are fundamental cellular components that perform diverse biological roles from structural building blocks to signaling molecules and energy storage centers. The role of lipids in viral infection involves endocytosis and exocytosis of viral membranes in syncytial virus-replicating host cells. Because lipids play such an important role in the viral life cycle we asked whether drugs that target lipid metabolism such as statins could be used against SARS-CoV-2 and other viruses. In this review we discuss the role of lipid metabolism in viral infection and the potential of targeting lipid metabolism to disrupt the viral life cycle (Abu-Farha *et al.* 2020).

5. CONCLUSIONS

1. It stimulates ACE2, which is an amine that converts angiotensin II into angiotensin-1-7, which in turn counteracts the negative effects of the renin-angiotensin system, which plays important roles in the body as it plays a role in maintaining the physiological state of the body. And the pathophysiological balance of the body, and therefore the change in its levels is an important indicator in the diagnosis and follow-up of the disease (COVID-19).
2. Age had a clear importance, as the study proved that the older the age, the higher the infection rate, meaning that the virus affects the elderly more than the young.
3. Urea and serum creatinine were not significantly affected in people infected with Covid 19 virus, and this explains the absence of a relationship between kidney failure disease and the virus that causes the disease.
4. There was no strong indication of the clinical significance of lipids in COVID-19 patients, but there were slight differences for total cholesterol.
5. Didimer had statistical significance and high statistical significance, as it explains these changes in its results as a result of the tissue changes that occur in the lungs, and thus the percentage of oxygen decreases, and as a result, blood clots increase in patients with Covid 19.
6. The results of interleukin-6 indicate that the levels of interleukin 6 changed significantly to the importance of the immune system in this disease, as it is necessary to give immunostimulants and vitamins to people during the period of infection.
7. The levels of white blood cells and hemoglobin were affected in Covid 19 patients, as white blood cells are the important line of defense in the human body.

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