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COVID-19 INCIDENCE AND MORTALITY IN HEMODIALYSIS PATIENTS IN IBN SINA HEMODIALYSIS CENTER IN DIYALA PROVINCE

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COVID-19 INCIDENCE AND MORTALITY IN HEMODIALYSIS PATIENTS IN IBN SINA HEMODIALYSIS CENTER IN DIYALA PROVINCE

By Rokan Jumaah Mhmood AL-GBURI

April 2022

We certify that we have read this thesis and that in our opinion it is fully adequate, in scope and in quality, as a thesis for the degree of Master of Science

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ABSTRACT

COVID-19 INCIDENCE AND MORTALITY IN HEMODIALYSIS PATIENTS IN IBN SINA HEMODIALYSIS CENTER IN DIYALA PROVINCE

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Master of Science in Chemistry

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Main goal of this study is to estimate the incidence and prevalence of COVID 19 infection among hemodialysis patients, as well as their associated clinical, radiological, and laboratory risk factors. In this study, individuals with confirmed covid 19 infection ranged in age from (21-75years). The study's findings were inconclusive when compared to the patient age group (p-value > 0.05). Average and standard deviation of age into. Two main groups were hemodialysis with COVID and hemodialysis in control (42.75 ± 17.46, 43.467 ± 15.45) respectively, and a p-value 0.70, regarding hypertension and DM, the result showed a significant effect of hypertension for hemodialysis, especially with COVID infection p-value (0.02), but slightly change with DM p-value (0.031). the mortality of hemodialysis with covid 19 showed 22% from 80 patients but only2% from hemodialysis without covid 19 in the same periode of the study.result of this study illustrates the average and stander deviation of D-Dimer. Two main groups were hemodialysis with COVID and hemodialysis in control (2093.83 ± 1030, 912.26 ± 255) respectively, with and p-value of 0.001. The hematological parameter has a decrease in WBC but slightly increasing Hb and a decrease in lymphocytopenia.

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Keywords: Covid-19, Chronic kidney disease, Hemodialyses, Kidney function test, Kidney diseases, Interleukin-6, D - dimer

ÖZET

DİYALA İLİ İBN SINA HEMODİALİZ MERKEZİNDEKİ HEMODİALİZ HASTALARINDA KOVİD-19 İNSİDANS VE MORTALİTE

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Bu çalışmanın amacı, hemodiyaliz hastaları arasında korona enfeksiyonlarının insidansı ve prevalansının yanı sıra bunlarla ilişkili klinik, radyolojik ve laboratuvar risk faktörleridir. Bu araştırmada, doğrulanmış covid 19 enfeksiyonu olan bireylerin yaşları (21-75) arasında değişmektedir. Çalışmanın bulguları, hasta yaş grubuyla karşılaştırıldığında yetersizdi (p-değeri > 0,05). Yaşın ortalama ve standart sapması. İki ana grup, COVID'li hemodiyaliz ve kontrolde (42,75 ± 17, 46, 43.467 ± 15,45) ve p değeri 0,70 olan hemodiyaliz idi. Hipertansiyon ve DM olarak bir sonuç, özellikle COVID enfeksiyonu p değeri (0,02) ile hemodiyaliz için hipertansiyonun önemli bir etkisini göstermektedir, ancak DM p-değeri (0,031) ile biraz değişir. Covid 19 ile hemodiyalizin mortalitesi 80 hastada %22, Covid 19 olmayan hemodiyalizden ise %2 gösterdi. Bu çalışmanın sonucu, D-Dimer'in ortalama ve standart sapmasını göstermektedir. İki ana grup, COVID'li hemodiyaliz ve kontrolde (2093,83 ± 1030, 912,26 ± 255) ve p değeri 0,001 olan hemodiyaliz idi. Tüm grupların serumlarında üre ve CRE ölçüldü. Hematolojik parametre, WBC ve Hb'de önemli bir artış ve lenfositopenide bir azalma gösterdi.

2022, 47 sayfa

Anahtar Kelimeler: Covid-19, Kronik böbrek hastalığı, Hemodiyaliz, Böbrek fonksiyon testi, Böbrek hastalıkları, İnterlökin-6, D - dimer

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Rokan Jumaah Mhmood AL-GBURI
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CONTENTS

LIST OF SYMBOLS

%	Percent
±	Plus minus
℃	Degrees Celcium
µg	Migrogram
µL	Microliter
g	Gram
h	Hour
kg	Kilogram
mg	Miligram
mL	Mililitter

LIST OF ABBREVIATIONS

A	Absorbance
ADMA	Asymmetric dimethylarginine
ALS	Amyotrophic lateral sclerosis
ANOVA	Analysis of variance
BMI	Body mass index
CBC	Complete blood counts
CDC	Centers for disease control
EIZA	Enzyme ımmunoassay
ERK	Extracellular signal-regulated kinase
HIF-1	Hypoxia factor-1
NOS	Nitric oxide species
OD	Optical density
TAMs	Tumor-associated macrophages
WBCAs	White blood cells

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1. INTRODUCTION

According to the Centers for Disease Control and Prevention (CDC), the SARS-coronavirus 2 outbreak that began in Wuhan, China in December 2019 and has since spread throughout the rest of the country has resulted in the deaths of more than 630,000 people (CDC). As a direct result of the outbreak, the SARS-CoV-2 virus has been passed on to close to 15 million individuals. The majority of patients had pre-existing conditions, with chronic renal failure accounting for more than 20 percent of all deaths (CKD). Given that respiratory failure and significant alveolar damage are both common symptoms of infection with SARS-CoV-2, the mortality rate associated with this virus is relatively high. According to a number of studies, patients who have had dialysis or a kidney transplant have a greater risk of contracting COVID-19 and a poorer prognosis for the disease. This information is based on research that will be published in 2020 by Kataria et al. (Naicker *et al*. 2020).

Because computed tomography scans of the lungs showed low densities, inflammation and edema were considered to be the cause of the patient's symptoms (Gagliardi *et al.* 2020). According to data that was only recently made public, 26.7 percent of COVID-19 patients were hospitalized with hematuria, while 44 percent of patients had proteinuria and hematuria at the time of their admission. Patients presented with an increased blood creatinine level 15.5percent of the time, and an elevated serum urea nitrogen level 14.1% of the time (Naicker *et al.* 2020). Those who are on dialysis have an increased risk of having many comorbid diseases, are often of advanced age, and have different degrees of immunosuppression, all of which make them more vulnerable to SARS-CoV-2 infection and make disease progression more challenging (Taji *et al.* 2021).

It is believed that only 0.2 percent of the overall population is affected by COVID-19; however, the incidence rate for dialysis patients is 3.3 percent across the country. When a patient received hemodialysis in a facility, rather than at home, their risk was nearly twice as high as when they received the treatment at home (Hsu and Weiner 2020). There is a possibility that the immune response of the body to the virus was what caused

the symptoms of SARS-CoV-2. Because of the COVID-19 infection, the immune systems of some people may become overly activated, which can result in a condition known as a cytokine storm. In this particular scenario, receiving cytokine injections can lead to severe inflammation as well as the death of kidney tissue. It is possible that the SARS-CoV-2 virus will cause the formation of microclots in the blood vessels of the kidneys, which will make it difficult for the kidneys to perform their typical functions. (Askari *et al.* 2021).

It is possible that COV-2 associated with SARS plays a role in the progression of chronic kidney disease in the same way that acute renal damage does. Throughout the course of the disease, damage to the patient's tubules or glomeruli has been observed in a sizeable portion of the patient population. Proteinuria and hematuria are both potential side effects of the cellular damage to the tubuloglomerular system that can be brought on by a virus. This damage can be both chronic and irreversible. It is recommended that the renal functions of COVID-19 patients be thoroughly examined, and any drugs that can preserve kidney function should be implemented as quickly as is practically possible. Also, it is important to note that this recommendation does not apply to patients who have been diagnosed with COVID-18. (Cravedi *et al.* 2020).

1.1 Objectives of Study

The objective is to record the incidence of corona infections among hemodialysis patients, and their risk factors according to clinical status, radiological and laboratory conditions related to mortality rate.

1.2 Specific

1- Determine the land and levels of D-Dimer, IL-6 in patients at the dissemination phase of corona infection.
2- Find a correlation of D-Dimer, and IL-6 with some demographaic factor like obesity and ageing also with some clinical pathological cases with hemodiylasis.

2. LITERATURE REVIEW

2.1 Overview of COVID-19

Pneumonia incidences in Wuhan, rose sharply in December 2019 compared to the same month last year. SARS-CoV-2, a new coronavirus strain identified by the WHO as the source of the sickness, was already part of the virus cluster that includes SARS and MERS (Sun *et al*. 2020).

All continents except Antarctica have been hit by the COVID-19 (new coronavirus sickness of 2019). Pandemic produced by the covid-19 virus, COVID-19 has had a disastrous nrgative reflect on the world's population, as evidenced by the available data. Coronavirus has been responsible for almost 15 million fatalities in 212 nations and territories as of 23.07.2020, but only 5.4 percent of those deaths were fatal North and South America and Europe have been particularly hard hit by climate change. If you're referring to the United States (Gagliardi *et al*. 2020).

Approxamitaly, about 4,103,674 cases and nearly 146,000 deaths in United States. a large number of people throughout Europe Around 200,000 people in Russia, Spain, the United Kingdom, and Italy have died as a result of infections since the outbreak was announced (Bartsch *et al*. 2020).

In the entire continent of North America, new cases reported in China have steadily decreased over this time period, according to government data. As a result, it's a good idea to do so. Patients who have been infected with the virus have received the aid of more than ten million Europeans (Bartsch *et al*. 2020).

EU and UK research funding totals €20 million, with an additional £20 million coming from the UK. COVID-19 needs to be vaccinated against. US healthcare is expected to be incredibly expensive, as predicted. The cost of treating a single patient afflicted with the virus is expected to exceed $3 million. During the pandemic, nearly $654.0 billion

was spent. Symptoms of influenza with moderate toxicity are present. COVID-19 has been found in 20% of the cases, despite the fact that it has been found in 80% of the instances. Respiratory failure can lead to lung diseases such as asthma or pneumonia. Strokes and pulmonary embolisms can be caused by thrombophilia (PE) (Rapezzi *et al*. 2020).

Acute coronary syndrome and myocardial infarction Despite the fact that most of the qualities associated with a Acute respiratory failure and significant alveolar damage are among the side effects of COVID-19. In addition, it's possible that you're suffering from kidney illness. Acute kidney damage (AKI) is a common consequence of HIV-infected cases . SARS-CoV-2. One-fifth of those who died had chronic renal disease, which was also detected. (CKD) Since COVID-19 may play a role in the kidney, further investigation is warranted (Boraschi 2020).

2.2 Epidemiology

As of July 23, 2020, COVID-19 has been found in more than 15 million people around the world. Because the virus has spread so much, so many people now have it. COVID-19 is more likely to spread than MERS and SARS because it can be spread through direct contact, feces, or droplets. People who don't have any symptoms or are still in the "incubation period" could also spread the virus, which would explain why the illness is so contagious (Qi *et al*. 2020).

COVID-19 infections can take up to 14 days to show symptoms, and most of them show up four to five days after exposure. In some cases, though, the incubation period has been said to be as long as 27 days. In a later study it was found that the incubation period could last from 0 to 24 days, with 3 days as the middle point (Chakraborty *et al*. 2020).

Because the symptoms of a COVID-19 infection can manifest themselves in different people at different times, it is impossible to predict how long an infected person can

continue to spread the disease to others. There is evidence to suggest that the SARS-CoV-2 virus can be transmitted either before or after the onset of symptoms associated with the disease. [Citation needed] Patient advocacy organizations in China, Italy, and the United States have compiled data suggesting that the virus can affect people of all ages, including children, and that men are more likely to pass away as a result of the illness than women are. Obesity, chronic lung disease (CLD), and kidney cancer (CKD) were common co-morbidities among COVID-19 patients treated at Tongji Hospital (BMI 40), which was the primary source of infection for this strain of virus. Chronic lung disease (CLD) was the most common (Richardson et al. 2020).

On the other side of the world, a group of 5700 hospitalized Americans in the United States provided data that was comparable (Richardson et al. 2020). In spite of the fact that recent findings suggest that children are just as likely as adults to become infected with SARS-CoV-2, there have only been a few fatalities and a small number of admissions to intensive care units. In this study, 336 additional children were examined from 11 different case series. Multiple studies have demonstrated that children have stronger innate immune responses and are less likely to have other health issues at the same time, both of which contribute to the fact that children's infections are less severe. (arterial hypertension, cardiovascular disorders). Acute kidney damage, also known as AKI, is a clinical illness that has been linked to a number of undesirable outcomes, including death, in patients who have COVID-19 (Brienza et al. 2020).

There were approximately the same number of cases of AKI that there were of SARS and COVID-19. During the production of COVID-19, it is possible that the kidney function of both people who do not have renal disease and those who do have renal disease could significantly decrease (Brienza et al. 2020).

According to the findings of various studies, AKI can manifest itself anywhere from seven to fifteen days after a patient's first visit. Following the initial doctor's appointment, anywhere from 7 to 15 days later, up to 23 percent of patients report having this issue. Contrary to what was previously believed, having a chronic illness did not significantly increase the risk of death in patients who had AKI. On the other

hand, individuals who were diagnosed with acute kidney injury (AKI), which is medical shorthand for acute renal damage, had a mortality risk that was 5.3 times higher. A recent analysis of 26 different studies came to the conclusion that there is a 13-fold increase in the risk of death due to acute kidney injury (AKI). There is a correlation between having chronic kidney disease (CKD) when admitted to the hospital and having acute kidney injury (AKI) while the COVID-19 infection is active, which has been linked to an increased risk of dying (Zhang et al. 2020).

COVID-19 has also been shown to be harmful to those who suffer from kidney disease (RRT). In their investigation, researchers from the Italian Society of Nephrology (SIN) found SARS-CoV-2 in 2.8 percent of patients undergoing hemodialysis but none of the 18 patients undergoing peritoneal dialysis (0.65 percent). Dialysis was performed on patients who were undergoing RRT at the patients' own facilities. The majority of patients were sent to hospitals that provided sub- or critical-care services; however, only 4.7% required intensive care. Even though the risk of death was higher for men (31.1 percent) than it was for women (4 percent) and for people who had cardiovascular disease, the amount of time spent on dialysis did not have a correlation with the risk of death, as shown by data from dialysis units in the Piedmont and the Aosta Valley in Italy, two regions in Italy's Northern West. On the other hand, despite the fact that men had a higher mortality rate, this was still the case (29.9 percent vs. 10.7 percent, respectively) (Zhang *et al*. 2020).

2.3 Etiology of Crona Virus

Coronviruses, which have a positive charge, are members of the coronaviridae family of viruses that only contain a single strand of RNA. There were six coronaviruses that might potentially cause sickness in humans in 2019, and there were four bacteria that are often seen in the human body. The first two strains of zoonotic coronavirus, which may be transmitted from animals to people, were discovered in the years 2003 and 2012 respectively. increasing in the number of people who have died from lung illnesses such as SARS in 2003 and MERS in 2014. According to the findings of recent study, coronaviruses such as SARS and MERS may be able to spread to humans via the use of

intermediate hosts such as the civet and camel, respectively. According to the findings of recent study, coronaviruses that may be discovered in bats may infect human cells (Perico *et al.* 2020).

The Huanan Seafood Wholesale Market was fingered as the source of a new respiratory ailment that rapidly spread throughout China. The sickness was originally identified in Wuhan, which is located in the province of Hubei. In January of 2020, a strain of the coronavirus that had not been seen before was located, and this discovery uncovered the root cause of the ailment. There is just one cluster that contains all three strains of the SARS-CoV and MERS-CoV coronaviruses. SARS-CoV-2 is the name given to the third strain of the coronavirus that has been shown to be associated with sickness. SARS-CoV-2 was revealed to be composed of two coronaviruses that had been identified in the past; one came from bats, while the other came from an unidentified source (Sun *et al.* 2020).

In spite of the fact that just a portion of the pangolin coronavirus genome was sequenced, it was discovered that SARS-CoV-2 and the pangolin coronavirus are two separate viruses. Despite this, the genome of the bat coronavirus is 88 percent similar to that of the pangolin coronavirus, while the genome of the pangolin coronavirus is 99 percent identical to that of the bat coronavirus. Since it was discovered that the pangolin served as a connection between bats and people, it cannot be said that it was directly responsible for the SARS-CoV-2 epidemic (Luan *et al*. 2020).

2.4 Pathophysiology of Corona Virus

By looking at SARS-genome CoV-2 along with SARS-CoV and MERS-CoV, scientists were able to learn more about how viruses cause disease. There are 79 coronaviruses that are similar to SARS and 50 that are similar to MERS (Li *et al.* 2020).

In order to get into cells, both SARS-CoV and SARS-CoV-2 need ACE-2. ACE-2 can stop many different things from happening, including those that stop blood vessels from

growing and those that stop cells from forming scar tissue. Both RNA sequencing on a single cell and histochemical tagging show that ACE-2 is present in a large number of cells. The amount of ACE-2 in many cell types was low, which made people wonder if it was needed for SARS and CoV-2 to interact or for the virus to get inside cells (Zou *et al.* 2020). There are many different kinds of receptors that ssRNA viruses can attach to (Qi *et al.* 2020) looked into how ACE-2 and ssRNA viral receptors and membrane proteins work together in thirteen different cell types from different organs. Pearson did some research and found that ACE-2 is linked to 94 different genes. The proteins ANPEP, ENPEP, and DPP4 are all linked to ACE-2 (DPP4). Coronaviruses have been found to target ANPEP and DPP4, but there is no link between ENPEP and virus infection. Type II integral membrane endopeptidase from the M1 endopeptidase family that contains zinc and is an endopeptidase. This protein controls the blood pressure and makes blood vessels in the kidney cortex and terminal ileum. Based on what the research team found, more research needs to be done to show that ENPEP is a coronavirus receptor (Qi *et al.* 2020).

For antiviral purposes, it may be possible to lower the concentrations of ACE-2 in the membranes of the cells. Blocking ACE-2 in humans leads to the development of clinical symptoms as well as disruptions in cell-to-cell communication. A recent study suggests that SARS-CoV-2 may be able to prevent the spread of COVID-19 by reducing ACE-2. [Citation needed] [Citation needed] Those who have COVID-19 and take an ARB may have a lower risk of becoming infected with SARS-CoV-2. The production of ACE-2 by cells can be stimulated by ARBs. If certain cell proteases do not become more active during the first stage of contact between SARS-CoV-2 and ACE-2, the virus may become stalled on the cell membrane and the infection may be stopped. This is something that can only be accomplished with the help of SARS-CoV-2-ACE-2. ADAM17 has the potential to dislodge ACE-2 from the membrane, thereby enabling it to dissolve in solution. The production of SARS-CoV-2 decoy receptors is possible by elevating the total amount of expressed and soluble ACE-2. As a consequence of this, viruses are unable to enter cells (Zou et al. 2020).

Other coronaviruses, including SARS-CoV-2 and ACE 2, also have a spike glycoprotein in common with each other. The combination of the S1 component and the S2 component of the coronavirus spike protein results in the formation of one transmembrane segment as well as two internal segments. S1 is in possession of both a CTD as well as an NTD at the present time (CTD1, CTD2 and CTD3). SARS-CoV RBDs combine with the viral protein to form a complex through their interaction (CTD1). According to testing using atomic-resolution, SARS-CoV-1 and SARS-CoV-2 share 73.7 and 89.2 percent of their respective sequences, respectively. As a result of these novel characteristics, the SARS-CoV-2 RBD virus is more likely to quickly spread throughout the world. According to the findings of the study, the SARS-RBD CoV-2 virus possesses one change that enables it to more effectively adhere to ACE-2 (He et al. 2020). It is simpler to maintain the virus' solubility in order to keep SARS-RBD CoV-2 stable at high temperatures. The SARS-RBD CoV-2 virus has developed a new binding site, and this one is more flexible than the previous one. RBD-ACE-2 indicates that SARS-2 is more sensitive to variations in temperature than SARS-1 (He et al. 2020).

The fusion of the virus membrane with the cell membrane occurs when the RBD of SARS-CoV and ACE-2 work together. Cathepsins are a type of enzyme that can be found in endosomes. They are responsible for facilitating the entry of viruses into cells. In order for viruses to enter the body through a route other than the endosomal pathway, proteases are required to remove S1 and S2 from the S protein. This allows viruses to enter the body. It was discovered by Hoffmann et al. in 2020 that TMPRSS2 is essential for the priming of SARS-CoV-2 proteins.

Both ACE-2 and TMPRSS2 are required for the CoV2 virus in order for it to infect host cells and spread throughout the body. After the transcription machinery within the cell has been activated, the SARS-CoV-2 virus is able to make use of it. When infected cells are exposed to SARS-CoV-2, the production of cytokines occurs; these cytokines have an effect on the immune system. To begin, macrophages and the cells that produce ACE-2 had some sort of interaction with one another. Then there were the functions that macrophages were not yet known to be responsible for performing. Providing further

support for the hypothesis that macrophages act as a form of virus surveillance. According to the findings of a recent study, the SARS-CoV-2 virus did not interact with mitochondrial proteins that are known to be inhibited. Viral cells are unable to perish as a result of this factor. (Hoffmann *et al.* 2020).

2.5 Kidney Damage by Covid -19 infection

In addition to the kidneys and the liver, the ACE-2 enzyme can also be found in the esophagus, the stomach, the ileum, and the colon. These organs are at risk of infection from SARS-CoV-2 due to the symptoms of COVID-19, which include acute kidney injury (AKI), heart failure, and digestive problems. COVID-19 can cause a variety of symptoms (Huang et al. 2020, Huang et al. 2020).ACE-2 is also found in the stomach, ileum, colon, and esophagus, in addition to the kidneys and liver. SARS-CoV-2 could infect these organs because of the symptoms of COVID-19, which include AKI, heart failure, and digestive problems. Symptoms of COVID-19 include (Huang et al. 2020, Huang et al. 2020).

It is possible that SARS-CoV-2 could become ensnared in organs, which would compromise its ability to spread throughout the body. It has been suggested that the SARS-CoV-2 virus can infect kidneys that express ACE-2 (Zou et al. 2020, Zou et al. 2020). None of the cells in the immune system, intercalated duct cells or collecting duct cells were found to express ACE-2 in any of the organs studied (8%). (8 percent).

According to ACE-2 receptor and TMPRSS genes, additional TMPRSS genes (TMPRSS 4, 5, or 9) may promote SARS-CoV-2 to target kidney epithelial cells (Batlle et al. 2020, Batlle et al. 2020). In both Podocyte and PRTC populations, ACE-2 and TMPRSS expression were found to be associated. SARS-CoV-2 nucleocapsid protein was found in urinary tubular structures. According to these studies, cytotoxic renal cells may be the cause of viral AKI. Infecting podocytes and gaining access to tubular fluid may allow the virus to attach to ACE-2 in the proximal tubules. Patients with COVID-19 may experience proteinuria and hematuria as a result of podocyte death. Not clear

whether the SARS-CoV-2 virus directly or inadvertently damages the kidneys (Fabrizi et al. 2020, Fabrizi et al. 2020).

The researchers (Diao et al. 2020, Diao et al. 2020) believe that SARS-CoV-2 is to blame for the kidney damage associated with COVID-19 (TECs). Tube cells from these patients contained viral antigens, CD68+ macrophages in the tubulo-interstitial region, and C5b-9 deposits on the apical brush border of tubular epithelial cells (TECs). Tubulointerstitial macrophages produce proinflammatory cytokines and injury-induced complement-mediated pathways contribute to tubular damage. Fibrosis, cell death, and microvascular damage are all possible side effects of overly aggressive host defenses. Overreacting is a common symptom of an overactive immune system.

Renal damage can occur in those who have SARS or one of its associated illnesses, such as CRS. SARS-CoV and SARS-CoV-2 immunological profiles and functions in patients with COVID-19 were examined using PBMCs. The rise in neutrophils and proinflammatory cytokines in SARS-CoV-2 lymphopenic patients may be explained by a decrease in the absolute T cell count. The concentrations of IFN, IL-2, IL-6, IL-10, and IL-10 increased sharply in the study. Viruses reduce the number of T cells in the body, which leads to an increase in inflammation. IL-2, IL-4, IL-10, TNF, and IFN- levels rise as the number of T cells decreases as a result of pathogen infiltration (Liu et al. 2020, Liu et al. 2020).

Patients with COVID-19 have higher levels of IL-6 and IFN- than healthy individuals. CD4+, CD8+, and NK cells have all decreased in the body. ' The antiviral cytokine interferon (IFN) (IFN). Transitioning from the innate to the adaptive immune system is facilitated by the activation of antiviral and antiproliferative genes by IFN-receptor binding and IFN-IL-6/sIL-6R signaling. IL-6/IFN Patients with SARS and COVID-19 who are infected with SARS-CoV-2 are at risk for developing AKI as a result of a cytokine storm (Liu et al. 2020, Liu et al. 2020).

Organ failure may be exacerbated by inflammation in the circulatory system, which raises extravascular pressure and restricts tissue perfusion. Occasionally, inflammation

may aid in tissue and organ recovery. Acute inflammation can cause damage to organs and tissues. SARS-CoV-2-induced organ damage and cell death result in a decrease in kidney density in COVID-19 patients (Cheng et al. 2020, Cheng et al. 2020).

A ventilator is needed to treat cytokine storms, which cause lung infections. Hypotension caused by septic shock usually necessitates the use of inotropic medications. Inotropics can cause hypotension and vasoconstriction in patients with COVID-19 or ARDS who have insufficient glomerular filtrate (Cheng et al. 2020, Cheng et al. 2020).

According to recent studies, COVID-19 renal injury is complex. The enzyme creatine phosphokinase has been linked to rhabdomyolysis (Su et al. 2020, Su et al. 2020). Rhabdomyolysis is caused by myoglobin secreted by an injured muscle. Myoglobin-induced renal tubular necrosis in hypoperfusion is caused by factors such as reduced nitric oxide, renal vasoconstriction, cast formation, and direct cell damage. In other words, these are the people who make ATN tick. The rhabdomyolysis that occurs in COVID-19 patients is particularly difficult to understand (Su et al. 2020, Su et al. 2020).

Increased hyperventilation may exacerbate the hypoxic effects of SARS-direct CoV-2. Patients with COVID-19-induced rhabdomyolysis may experience acute kidney injury (AKI) (AKI) (AKI). In COVID-19 patients who lack platelets or fibrinoid fragments, erythrocyte aggregates obstruct peritubular and glomerular capillary lumens (Su et al. 2020, Su et al. 2020).

Microvascular damage can be exacerbated by oxidative stress, inflammation, and activation of the complement system. Erythrocyte obstruction is almost always the result of endothelial dysfunction. Angiotensin-converting enzyme (ACE) is unique to renal endothelial cells. The SARS-CoV-2 virus does not infect cells in the respiratory endothelium. A disease or medication can alter the expression of ACE-2. SARS-CoV-2 was found to have a direct and indirect effect on the endothelium. The microcirculation is disrupted. Renal endothelial lesions require further investigation. SARS-CoV-2 can

infect endothelial cells by binding to CD147 (basigin) (Su et al. 2020). A study published in 2020 by Su et al.

An increased risk of thromboembolic events in COVID-19 patients may be due to SARS-CoV-2 coagulopathy. Patients with COVID-19 have extremely clotting blood. High TAP-C, fibrinogen, and d-dimer levels were found in blood tests. There have been cases of microangiopathy and disseminated intravascular coagulation in China and the United States. Tissue and coagulation factors may be activated by macrophages, cytokines, and proteins that are damaged. Clots are more likely to form. It is possible that renal and cortical necrosis was caused by hypercoagulability. COVID-19-positive individuals with back pain and microhematuria indicate renal infarction (Su et al. 2020, Su et al. 2020).

SARS-CoV-2 infection causes both short-term and long-term kidney damage. Tubules or glomeruli have been damaged in rare cases. Tubulo-glomerular cell death results in renal tubule scarring as a result of infection with a virus. There is a link between renal failure and the SARS-CoV-2 and COVID-19 viruses.

It is possible for the SARS-CoV-2 virus to become trapped in organs, which would inhibit its ability to disseminate to other parts of the body. It has been hypothesized that the SARS-CoV-2 virus is capable of infecting kidneys that express the ACE-2 enzyme (Zou et al. 2020, Zou et al. 2020). It was discovered that none of the immune system cells, intercalated duct cells, or collecting duct cells expressed ACE-2 in any of the organs that were under investigation (8 percent). (8 percent).

Additional TMPRSS genes (TMPRSS 4, 5, or 9) may encourage SARS-CoV-2 to target kidney epithelial cells, as indicated by the ACE-2 receptor and TMPRSS genes (Batlle et al. 2020, Batlle et al. 2020). ACE-2 and TMPRSS expression were found to be associated in both the Podocyte and PRTC populations. Urinary tubular structures were found to contain the nucleocapsid protein associated with SARS-CoV-2. These studies suggest that cytotoxic renal cells could be the root cause of viral acute kidney injury (AKI). It is possible for the virus to attach itself to ACE-2 in the proximal tubules if it

first infects podocytes and then uses this access to tubular fluid. As a consequence of podocyte death, patients diagnosed with COVID-19 may exhibit symptoms including proteinuria and hematuria. It is unknown whether the SARS-CoV-2 virus causes damage to the kidneys either directly or indirectly (Fabrizi et al. 2020, Fabrizi et al. 2020).

According to the findings of Diao et al. 2020 and Diao et al. 2020, the researchers believe that SARS-CoV-2 is to blame for the kidney damage that is associated with COVID-19 (TECs). These patients' tube cells had viral antigens, CD68+ macrophages in the tubulo-interstitial region, and C5b-9 deposits on the apical brush border of tubular epithelial cells. In addition, these patients' tube cells had C5b-9 deposits on the apical brush border (TECs). Both injury-induced complement-mediated pathways and tubulointerstitial macrophages contribute to tubular damage. Tubulointerstitial macrophages produce proinflammatory cytokines. There is a possibility that overly aggressive host defenses will cause side effects such as fibrosis, cell death, and damage to the microvascular system. An overactive immune system frequently manifests itself in the form of overreaction.

Those who have SARS or one of the illnesses associated with it, such as CRS, have an increased risk of developing renal damage. PBMCs were used to investigate the immunological profiles and functions of SARS-CoV and SARS-CoV-2 in patients diagnosed with COVID-19. A lower absolute T cell count may be the cause of an increase in neutrophils as well as cytokines that promote inflammation in SARS-CoV-2 lymphopenic patients. Throughout the course of the research, there was a discernible rise in the levels of IFN, IL-2, IL-6, IL-10, and IL-10. Viruses cause a decrease in the number of T cells that are found in the body, which ultimately results in an increase in inflammation. As a consequence of pathogen infiltration, the number of T cells will gradually diminish, which will lead to an increase in the levels of IL-2, IL-4, IL-10, TNF, and IFN- (Liu et al. 2020, Liu et al. 2020).

Patients diagnosed with COVID-19 have been shown to have elevated levels of both IL-6 and IFN- compared to healthy controls. There has been a general decline in the

number of CD4+, CD8+, and NK cells throughout the body. ' Interferon, also known as IFN, is an antiviral cytokine (IFN). The activation of antiviral and antiproliferative genes by IFN-receptor binding and IFN-IL-6/sIL-6R signaling makes it easier for the body to make the transition from its innate immune system to its adaptive immune system. IL-6/IFN Infected patients with SARS and COVID-19 who also have SARS-CoV-2 infection have an increased risk of developing acute kidney injury (AKI) due to a cytokine storm (Liu et al. 2020, Liu et al. 2020).

Inflammation in the circulatory system, which raises extravascular pressure and restricts tissue perfusion, may make organ failure worse. This is because inflammation increases extravascular pressure. In some cases, inflammation can actually speed up the healing process of damaged tissues and organs. An acute inflammatory response has the potential to damage internal organs and tissues. Patients with COVID-19 experience a decrease in kidney density as a direct result of the organ damage and cell death caused by SARS-CoV-2 (Cheng et al. 2020, Cheng et al. 2020).

In order to treat cytokine storms, which are the root cause of lung infections, a ventilator is required. In most cases, the administration of inotropic drugs is required in order to treat hypotension brought on by septic shock. Patients with COVID-19 or ARDS who do not have adequate glomerular filtrate may experience hypotension and vasoconstriction as a side effect of inotropic treatment (Cheng et al. 2020, Cheng et al. 2020).

Recent research indicates that the COVID-19 virus causes a complex renal injury. Rhabdomyolysis has been connected to the enzyme creatine phosphokinase in some studies (Su et al. 2020, Su et al. 2020). Rhabdomyolysis is brought on by myoglobin, which is released into the bloodstream by injured muscles. Necrosis of the renal tubules brought on by myoglobin in the presence of hypoperfusion is brought on by a number of factors, including a decrease in nitric oxide, renal vasoconstriction, cast formation, and direct cell damage. In other words, these are the individuals who are responsible for ATN's smooth operation. Rhabdomyolysis is notoriously difficult to comprehend, and it

is especially challenging when it occurs in COVID-19 patients (Su et al. 2020, Su et al. 2020).

It is possible that increased hyperventilation could make the hypoxic effects of SARS-direct CoV-2 worse. Acute kidney injury, also known as AKI, is a potential complication for patients whose rhabdomyolysis was caused by COVID-19 (AKI). erythrocyte aggregates are responsible for the obstruction of peritubular and glomerular capillary lumens in COVID-19 patients who are deficient in platelets or fibrinoid fragments (Su et al. 2020, Su et al. 2020).

Oxidative stress, inflammation, and the activation of the complement system can all contribute to the worsening of microvascular damage. Endothelial dysfunction is almost always the cause of erythrocyte obstruction, which can also be written as erythrocyte obstruction. Renal endothelial cells are the only cells in the body that produce angiotensin-converting enzyme (ACE). There has been no evidence that the SARS-CoV-2 virus infects cells in the respiratory endothelium. The expression of ACE-2 can be changed by a variety of conditions and medications. Researchers discovered that the SARS-CoV-2 virus had both a direct and an indirect effect on the endothelium. There is a disruption in the microcirculation. Lesions found in the renal endothelium require further investigation. Endothelial cells can be infected by SARS-CoV-2 when the virus binds to CD147 (basigin) (Su et al. 2020). A research paper that was published in the year 2020 by Su et al.

Patients diagnosed with COVID-19 may be more susceptible to developing thromboembolic events as a result of the coagulopathy caused by SARS-CoV-2. Patients diagnosed with COVID-19 have blood that clots very easily. Tests on the patient's blood revealed elevated levels of TAP-C, fibrinogen, and d-dimer. Microangiopathy and disseminated intravascular coagulation have both been documented as having occurred in patients in China and the United States. Macrophages, cytokines, and proteins that have been damaged can all be responsible for activating coagulation and tissue factors. It is more likely that clots will form. It is possible that hypercoagulability was the cause of the necrosis that was seen in the renal

and cortical tissue. People who have a positive COVID-19 test and also have back pain and microhematuria likely have a renal infarction (Su et al. 2020, Su et al. 2020).

An infection with SARS-CoV-2 can cause kidney damage in both the short term and the long term. In some unusual instances, either the tubules or the glomeruli were damaged. An infection with a virus leads to the death of tubulo-glomerular cells, which in turn leads to scarring of the renal tubules. There is a connection between renal failure and the SARS-CoV-2 virus as well as the COVID-19 virus. (Figure 2.1) (Naicker *et al.* 2020).

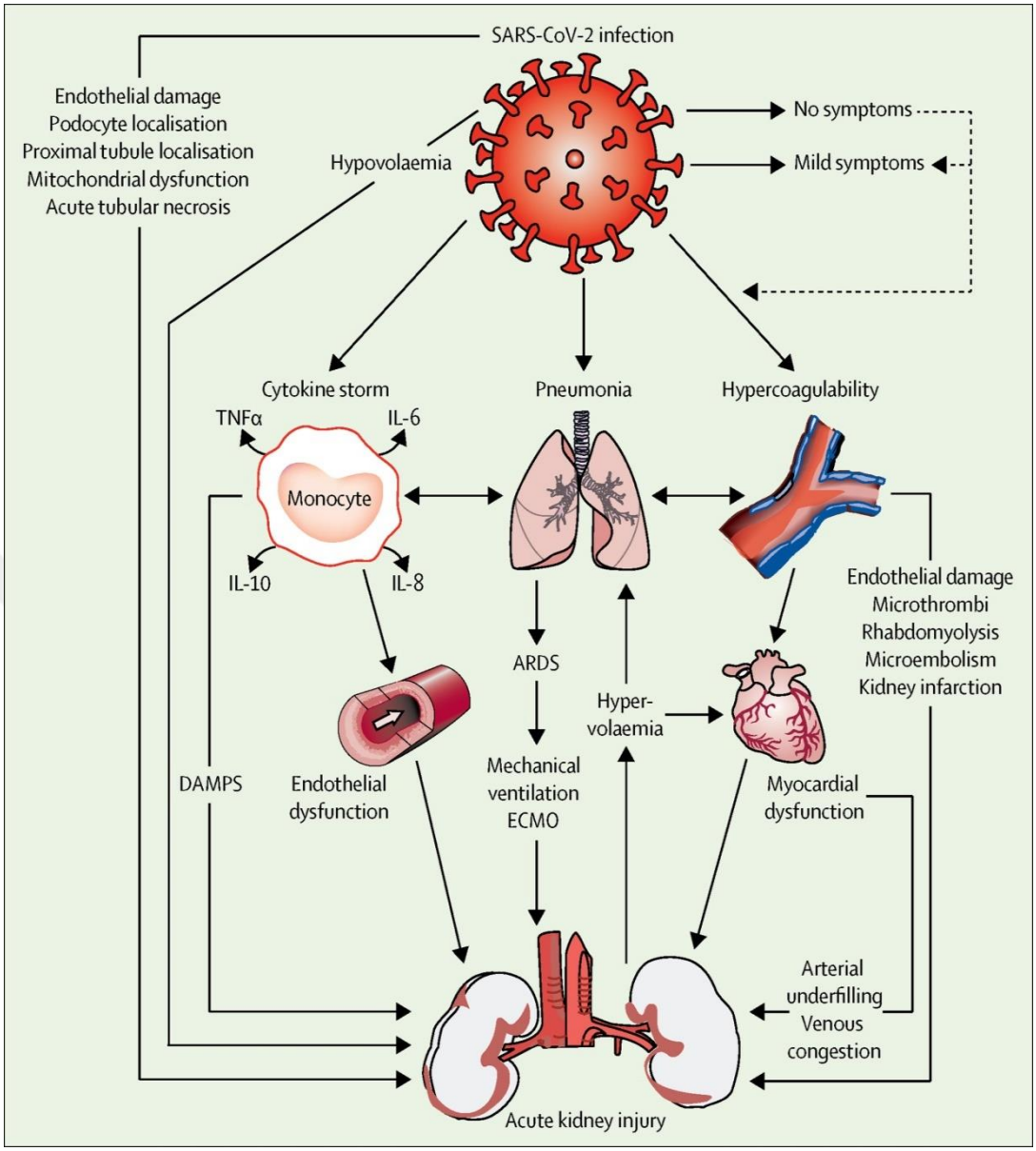

Figure 2.1 Pathophysiology of kidney damage associated with COVID-19 (Naicker *et al.* 2020)

2.6 COVID-19's AKI Treatment Protoco

In the intensive care unit for COVID-19 patients, the primary focus of care is on providing comfort care and emotional support for the patients. Because COVID-19 has a high incidence of renal involvement, it is absolutely necessary to test any and all of the currently available therapies for maintaining kidney function. (Ronco *et al.* 2020).

2.6.1 Clinical management

Patients who adhered to the treatment recommendations developed by the Kidney Disease: Improving Global Outcomes initiative had a lower incidence of acute kidney injury (AKI) and a milder manifestation of AKI than patients who did not follow these recommendations (e.g., avoidance of nephrotoxins, regular serum creatinine and urine output monitoring, consideration of haemodynamic monitoring). Nevertheless, additional research is necessary to support these claims. (Ronco *et al.* 2020).

Using lung-protective ventilation, which may lessen the effects of ventilator-induced hemodynamic changes and cytokines on kidneys, may make it feasible to lower the risk of developing a new case of AKI or of an existing case becoming worse (Ronco *et al.* 2020).

It is imperative that medical research on recently found biomarkers of tubular damage get underway as soon as possible to assess their diagnostic and therapeutic relevance (Ronco *et al.* 2020).

The tests for volume responsiveness and tolerance are very important instruments for regulating fluid balance. This strategy is implemented with the goals of normalizing the patient's blood pressure, lowering the risk of acute kidney injury (AKI), and avoiding other adverse effects of volume overload as much as possible. Because COVID-19 patients almost never get pre-hospital fluid resuscitation, volume depletion is likely to be present when these patients show themselves at emergency departments. In order to stop the progression of AKI in these individuals, the hypovolemia has to be treated. High positive end-expiratory pressure and recruitment maneuvers are two potential treatment modalities for COVID-19-associated respiratory distress syndrome (ARDS) (Palevsky 2021).

2.6.2 RRT and extracorporeal support

In patients who have volume overload and refractory hypoxemia, RRT should be undertaken in the event that normal therapy is unsuccessful. Patients diagnosed with COVID-19 and AKI may avert further disease development with early renal replacement therapy (RRT) and subsequent endoscopic kidney surgery (ECOS) (Figure 2.2). It is necessary to conduct clinical studies on actual people using this methodology. It is recommended to provide continuous RRT to individuals who have hemodynamic instability (Palevsky 2021).

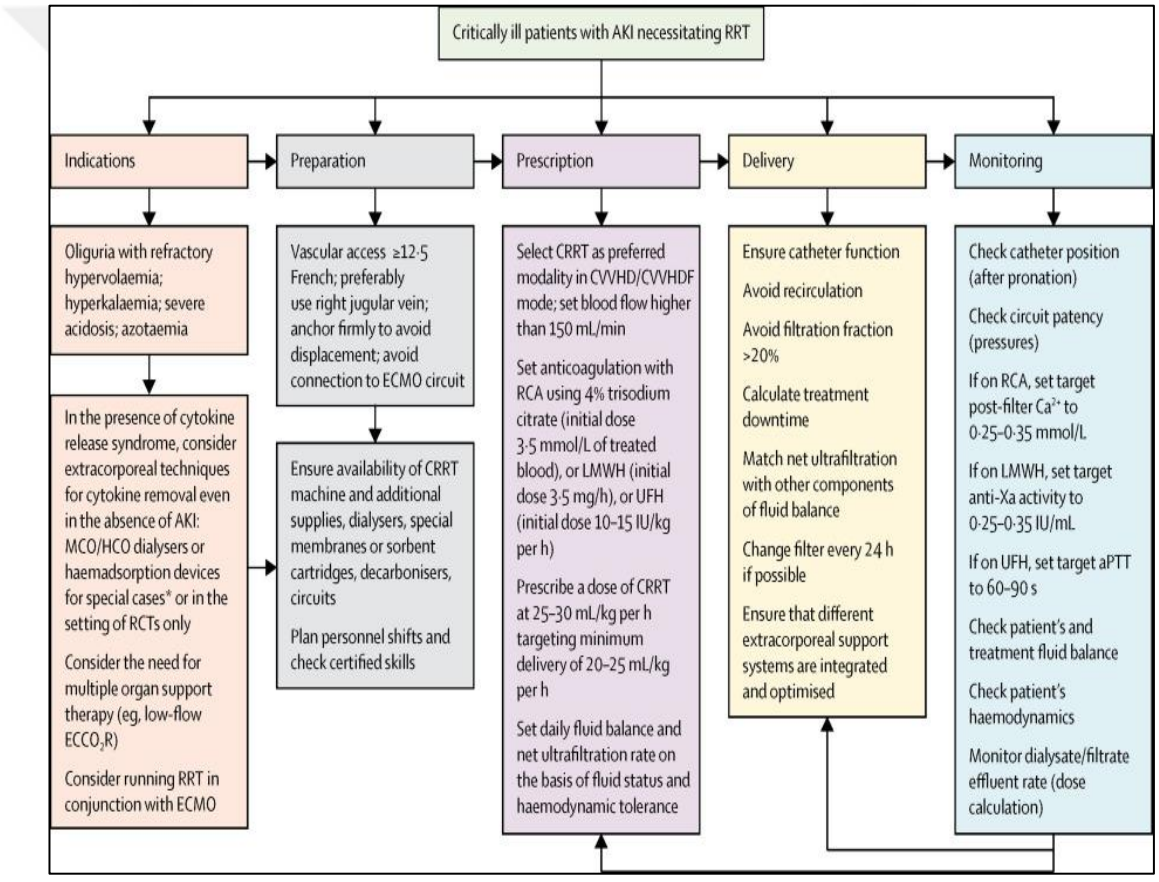

Figure 2.2 Treatment of patients with COVID-19 who have a sudden kidney injury that requires dialysis. (Palevsky 2021)

3. MATERIAL AND METHOD

3.1 Materials

3.1.1 Instruments

Some of important interuments and kits which used in vitro during the present study are sumerized in Table 3.1.

Table 3.1 List of instrument and kits

No	Instrument	Country	Company
1	Biopette Variable.Volume 2-20, 20-100, 100-1000 ulv	China	
2	UV transmission	Germany	Vilberlourmat
3	Incubation	China	Jrad
4	Spectrophotometer	Jaban	apple
5	Centrifuge	Japan	H-19F Kokusan
6	Water bath	Germany	Arneold
7	Autoclave	Germany	Human
8	Tips (blue, yellow)	Maliza	-
9	Lab. Water bath	China	mindery
10	Complete blood picture(CBC)	china	Genex
12	EDTA Tubes	China	-
Kits			
1	IL-6	ITALY	Roche -coubus
2	D-dimer	China	Human ELIZA
3	CBC	China	genex
4	Creatnine	Spain	Linear
5	Urea	Spain	Linear

3.1.2 Subjects

In this study, 450 patients with chronic renal failure stage 5 on regular hemodialysis in Ibn Sina hemodialysis Center in Diyala Governorate will be monitored. D-Dimer, interleukin-6, urea creatinine, CBC, in patients with COVID-19 and renal failure. And studying the impact of Covid 19 on patients with kidney failure from the period 1st December 2021 to 1st march,2022 the study groups were classified into two groups.

Group 1. Patients with kidney failure with COVID 19 (80).

Group 2. Control groub, all the staff of ibn Sina hemodialysis center including doctors. paramedics around (50) persons how will be monitored for infection with COVID 19.

3.1.3 Diagram of project

The groups of the current study are illustrated in Figure 3.1

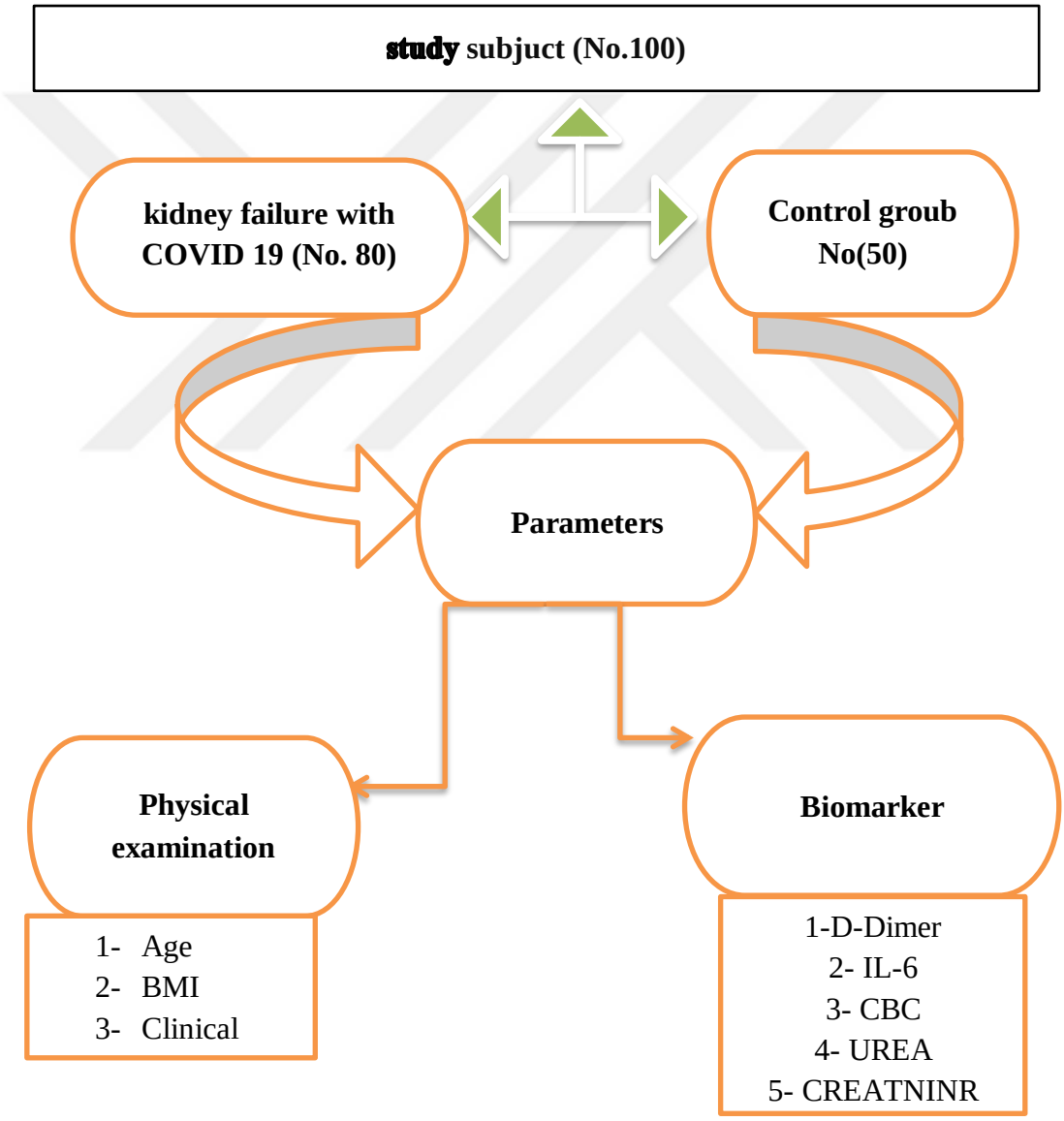

Figure 3.1 Groups of current study

3.2 Methods

3.2.1 Blood sampling

Two divisions of 3 and 2mL of patients and healthy controls were obtained via venipuncture, and specimen were stored at four ℃ until further analysis. We coagulated the first sample using a gel tube and 15 min at room temperature. Serum was obtained by centrifugation for 10 min at 3000 rpm and stored in the freezer (-20 ℃) until needed. The second aliquot is dissolved in EDTA and kept at -20 ℃ until blood tests are required to avoid coagulation.

3.2.2 White blood cells count

A multi-parameter cell counter can be used to measure, derive, and/or calculate WBC types (Hematology Analyzer, Frankford, Germany, 2013). System status monitoring, barcode recognition, and computer-enhanced data processing are all features of the automated sampling system at Medical City Oncology Hospital.

3.2.3 Determination of IL-6 by roche-coubs

Elecsys IL-6 immunoassay measures IL-6 (interleukin-6) in serum and plasma. Patients with COVID-19 who undertake this assay and lab testing can determine their likelihood of mechanical ventilation. Elecsys IL-6 immunoassay is cobas-e compliant (ECLIA). Elecsys IL-6 immunoassay requires FDA Emergency Use Authorization. Appropriate in healthcare.

Principle

After 18 minutes, the first stage of the incubation process was finished. Incubation of a sample in a volume of 30 liters with a monoclonal antibody specific to IgG, or sample volume of 18 liters (cobas e 801 analyzer). In the second stage of the incubation

process, streptavidin-coated microparticles are added to a monoclonal IL-6-specific antibody. This step is performed in order to produce an antibody-antigen combination. The microparticles in the reaction mixture are drawn into the measuring cell by suction, which results in magnetic confinement on the electrode surface. [Cause and effect] The magnetic confinement of the microparticles on the electrode surface. After that, the unattached compounds are eliminated using the ProCell/ProCell M/ProCell II M system. In order to detect the chemiluminescent emission, a voltage is first applied to the electrode, and then a photomultiplier is used. A two-point calibration and a master curve, which are both supplied by the reagent barcode or e-barcode, are used to derive the results.

3.2.4 Determination of D-dimer

The Human ELISA for D-Dimer is used to determine the concentration of (D-Dimer) in human serum as well as plasma and medium used in cell culture. The diagnostic test will be able to identify recombinant as well as naturally occurring Hu D-Dimer. The overarching conception upon which the process is founded; the quantity of target that is bound between two antibodies may be measured using an enzyme-linked immunosorbent test (ELISA) that uses solid-phase sandwich technology. In the wells of the microplate that was delivered to you, it has already been pre-coated with a target-specific antibody. After inserting samples, standards, or controls into these wells, the immobilized (capture) antibody is incubated with the antibody for a period of time. Antibodies are placed in a sandwich with a substrate solution, which binds to the enzyme-antibody-target combination and produces a signal that can be measured. The strength of this signal is directly related to the amount of target that was present in the sample that was being analyzed.

3.2.5 Assay of renal function tests (principle of the test for determination of urea concentration)

The urea concentration in serum and urine was estimated by using the diagnostic kit supplied by Cam Tech Medical Company and by spectrophotometric method, as urea

binds with water to turn into ammonia and carbon dioxide in the presence of the enzyme urease, and thus the ammonia produced is estimated through interaction with (salicylate. And Hypochlorite to be 2, 2-Dicarboxy Indophenol) that produced green in color, through which it is sensitive to urea concentration. The colored complex is read at a wavelength of 580 nm (263), the calculation urea are shown in Equation (3.1)

Urea (mg/ dL) = (Abs. sample/ Abs.standard) x standard conc (mg/dL) (3.1)

Principle of the test determination of creatinine (CRE) concentration

The creatinine concentration is estimated according to the reaction of (Jaffe) in the blood serum and urine by using the diagnostic kit supplied by Biolabo-France Company, where CRE reacts with alkaline picrate to give a yellow complex. The absorbance of the colored complex is read at a wavelength of 490-510 nm. The calculation of creatinine are shown in Equation (3.2).

Creatinine conc. = (A1-A2) sample / (A1-A2) standard) x standard conc. (3.2)

3.2.6 Complete blood count test (cbc)

1. WBC or leukocyte count.
2. Including. Neutrophils, lymphocyte.
3- Hemoglobin (HGB).
4- Platelet count.

Test procedure

1. Drawing a few milliliters of blood from a vein.

2. In order to get a blood sample, first the plunger of the syringe is slowly and carefully retracted, or the needle is affixed to a specialized vacuum container that collects the blood, and then the vein is punctured.
3. Whole blood is placed in EDTA tube and then transfer to CBC instrument.

4. The results of a full blood count may be available within a few hours following the collection of the blood samples.

3.3 Statistical Analysis

As a result, it was concluded that the data had been properly transformed and standardized. IBM SPSS version 25.0 and a student independent T-test with a median standard deviation were also used to estimate the probability, we regarded it significant when the likelihood was less than 0.5%.

4. RESULTS AND DISCUSSION

4.1 Characteristics of the Study Population

Patients who had COVID-19 confirmed at Ibn Sina Teaching Hospital in dialya, Iraq, All of the patients who had COVID-19 were positive for the virus that was confirmed by PCR test and number of the cases that followed are 450 patients. IgM levels were then used to group subjects into subgroups based on how high their COVID-19-IgM values to study sample as following:

- Study sample of hemodialysis with COVID19 (IgM >4.0), N= 80.
- Study sample of hemodialysis without COVID infection (N= 370).
- Healthy subject as control group (IgM <1.0) N= 50.

Discussion of present study was in two parts

1. The first part was the related risk factors that participate in COVID-19.
2. The second part is study of the biochemical marker in the blood of these groups.
3. The third part study of the hematological marker in the blood of these groups.

4.1.1 Age and BMI distribution in covid 19 and control groups

In this study, the age range for patients with confirmed infection for covid 19 range from (21-75)years. It is important to note that this is just a control group. The results showed non-significant difference when we compared for the age group of patients (p-value > 0.05). Table 4.1 and Figure 4.1 illustrate the average and standard diviation of age into two main group hemodialysis with COVID and hemodialysis in control (42.75

± 17.46, 43.467 ± 15.45) respectively, and a p-value 0.70, also nonsignificant were appeared in comparison between both patients groups and healthy control (39.26 ± 9.91) p-value (0.61 ±0.59) respectively.

In present study when we talking about BMI theres no significant shown between hemodialysis with COVID-19 and hemodialysis without COVID19 groups (20.46 ± 3.2, 19.6 ± 1.56) respictively and p-value (0.88), but a slightly significance when we compared between both patients groups with healthy control (23.76 ± 2.5) p-value (0.03, 0.033) respictively.

For another clinical pathological studied such as hypertension and DM the result showed a significant affect of hyper tension for hemodialysis espically with COVID infection p-value (0.02), but slightly change with DM p-value (0.031), wherase the number of mortality was a 22% of 80 sample for hemodiylasis with covid 19 and 2% from hemodyalisis without covid infection in the time period of the study, p value <0.05.

Table 4.1 Clinical parameters for patients with COVID 19 and control groups

Clinical charachtristic	**Hemodialysis with COVID 19**	**Hemodialysis without infection Conrol**	**Healthy Control**	**p-value**		
				1x2	**1x3**	**2x3**
Age	42.75 ± 17.46	43.467 ± 15.45	39.26 ± 9.91	0.70	0.61	0.59
BMI	20.46 ± 3.2	19.6 ±1.56	23.76 ± 2.5	0.88	0.03	0.033
Hypertension	13 (26%)	7 (14.66%)	-	0.02	-	-
DM	18 (22%)	12 (24.5%)	-	0.031	-	-
Mortality	18(22%)	1 (2%)	-	0.001		

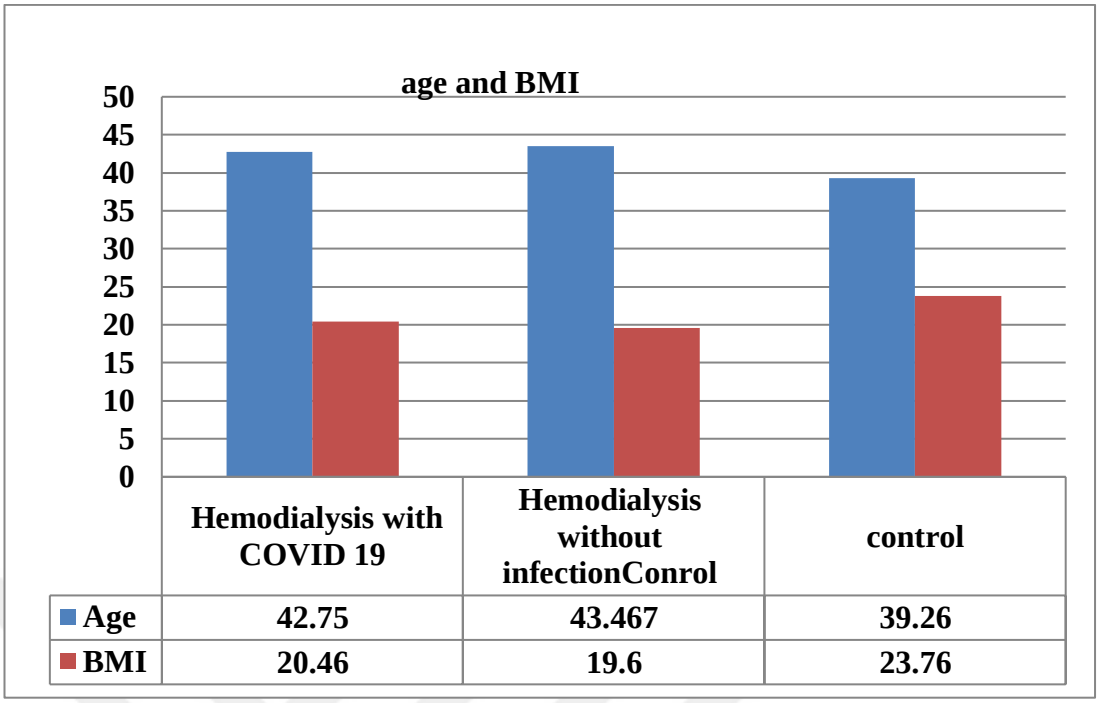

Figure 4.1 Mean and SD of age and BMI for hemodialysis patients and control groups

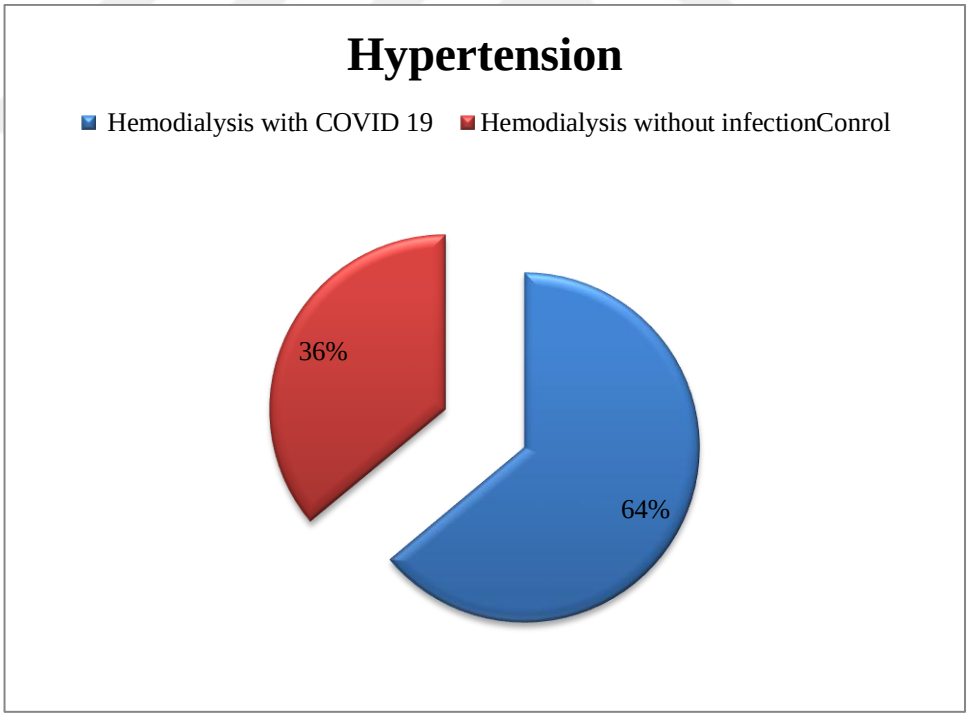

Figure 4.2 distribution of hypertension frequency for hemodialysis patients and control groups

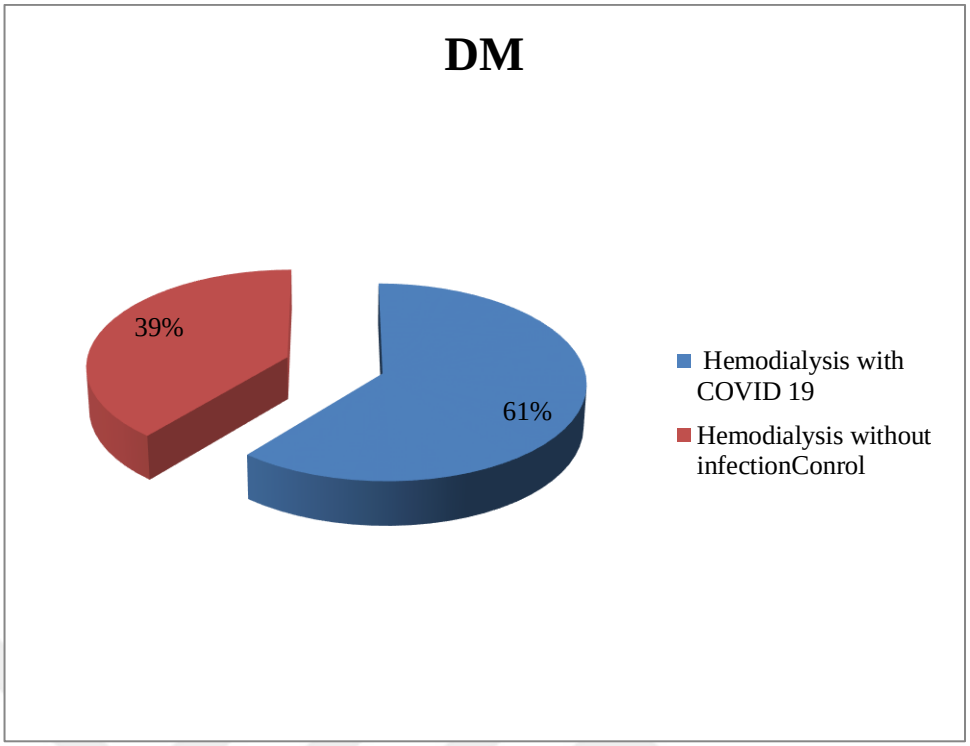

Figure 4.3 distribution of DM frequency for hemodialysis patients and control groups

Anyone of any age may get COVID-19, an infectious viral disease. According to our findings, those over the age of 30 are more prone to get ill. As a person grows older, their illness worsens. The findings indicate that there is some preference favoring males. On admission, patients with severe illness had fewer lymphocytes and eosinophils than healthy patients (P = 0.001 and 0.000, respectively). Eosinopenia was seen in 52.8 percent of individuals with moderate, severe, or critical illness. According to one research, persons with severe or critical illnesses had less CD4+, CD8+, and B cells. There were no statistically significant differences between the four groups, however the number of natural killer (NK) cells decreased as the illness progressed (Hillion *et al.* 2020).

4.1.2 Relation D-Dimer and IL-6 parameter with COVID-19

In this study, the biochemical range for patients with confirmed infection for covid 19. The results showed highly significant study when we compared for the D-Dimer group of patients (p-value <0.001). Table 4.2 and Figure 4.4 illustrate the the average and

stander diveation of D-Dimer into. Two main group hemodialysis with COVID and hemodyalsis in control (2093.83 ± 1030, 912.26 ± 255) respictively, and p-value 0.001. also a significant were appered in comparsion between both patients groups and healthy control (367.8 ± 163) p- value (<0.001, 0.01) respictively.

Our result shown theres a significant in IL-6 shown between hemodialysis with COVID-19 and hemodialysis in control groups (261.89 ± 220.8, 44.105 ± 33.87) respictively and p-value (<0.001), also significant when we compared between both patients groups with control (23.84 ± 10.6) p-value (<0.001, 0.037) respictively.

Table 4.2 The Mean and SD of D-Dimer and IL-6 parameter with COVID-19

Clinical charachtristic	Hemodialysis with COVID 19	Hemodialysis without infectionConrol	Control	p-value		
				1x2	1x3	2x3
D-DIMER	2093.83 ± 1030	912.26 ± 255	367.8 ± 163	0.001	<0.001	0.01
IL-6	261.89 ± 220.8	44.105 ± 33.87	23.84 ± 10.6	<0.001	<0.001	0.037

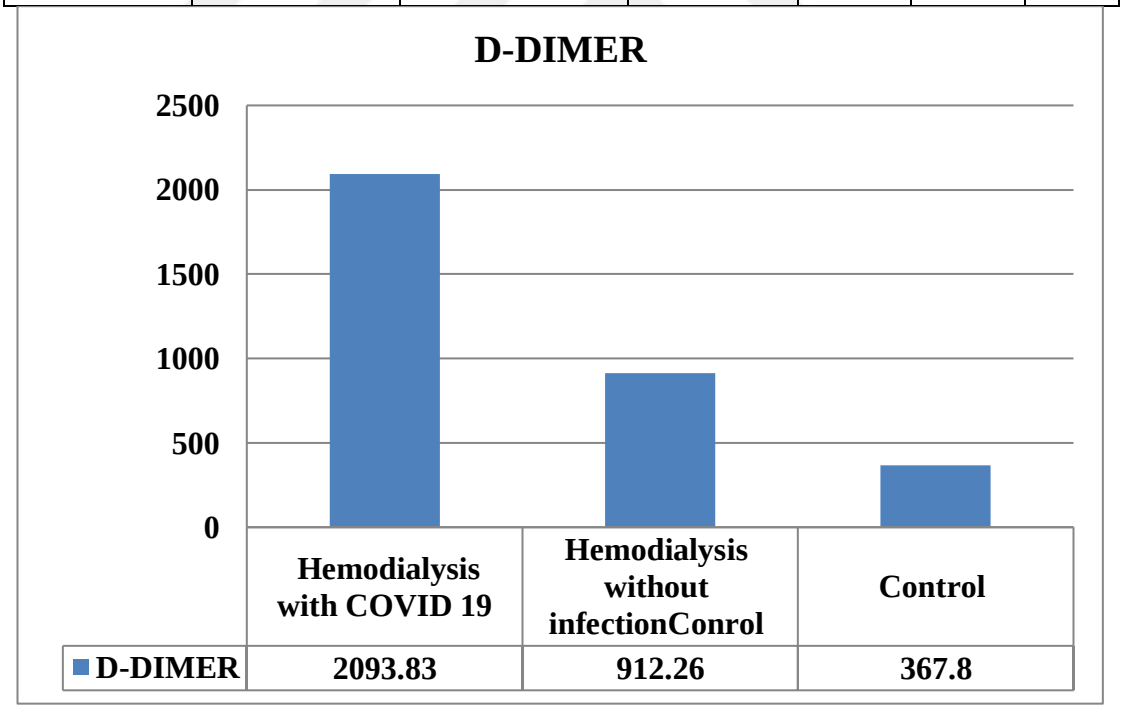

Figure 4.4 Mean and SD of D-Dimer for hemodialysis patients with covid and control groups

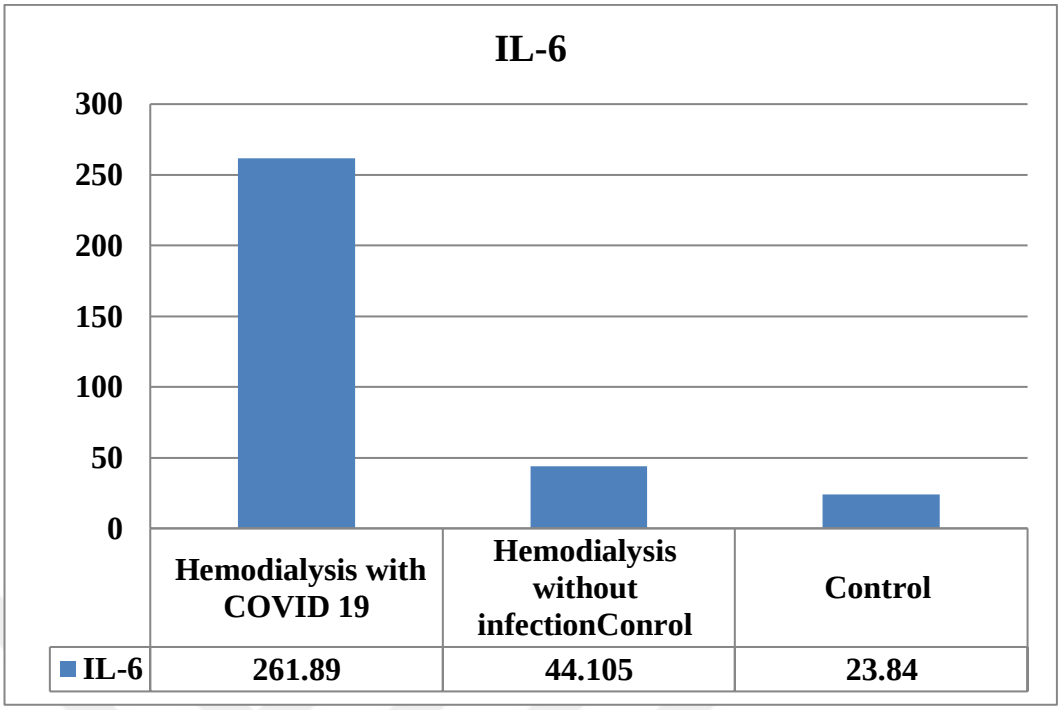

Figure 4.5 Mean and SD of IL-6 for hemodialysis patients with COVID and control groups

The D-dimer test can only be used to accurately predict mortality and the severity of Covid-19 illness in patients who are hospitalized; this test has no bearing on any other clinical outcomes whatsoever. Researchers set out with the intention of developing an indicator for patients with the Covid-19 genotype that would help predict mortality, acute kidney injury (AKI), and the requirement for hemodialysis or intubation (D-dimer). Men have lower levels of serum albumin, lymphocytes, and platelets than women do, but they have higher levels of white blood cells (WBC), aspartate aminotransferase (AST), liver dihydrogen phosphate (LDH), and creatinine kinase (CK). Males also have higher levels of the enzymes aspartate aminotransferase (AST), and liver dihydrogen phosphate (LDH). Patients who presented with mild to moderate COVID-19 illness had clinical symptoms and laboratory results that were nearly identical to those of patients who presented with severe to critical COVID-19 disease at the time of admission. As a consequence of this, it can be challenging to differentiate between patients who have conditions ranging from mild to moderate and those who have conditions ranging from severe to critical solely based on the outcomes of routine laboratory tests carried out upon admission (Herlambang et al. 2021).

Il-6 levels rise more quickly than those of other markers, which can serve as an early indicator of inflammation. When IL-6 is present in the body, which it must be in order for the virus to replicate, the infection caused by CoVid-19 can spread more quickly. Patients undergoing chronic hemodialysis (HD) have an increased likelihood of experiencing inflammation due to the fact that the uremic milieu produces pro-inflammatory mediators, which then require more time to be eliminated. Each and every HD session carries with it the potential for inflammation of the body's tissues. If something like this occurs during treatment, it could result in undesirable adverse effects. Previous studies have established a correlation between the levels of IL-6 found in HD patients and the length of their lifespans. Patients undergoing dialysis who have abnormal levels of IL-6 are at an increased risk of developing acute inflammatory disorders such as COVID-19. According to research carried out by Jofré et al. in 2006, individuals who have HD and who experience complications during their dialysis treatment are at an increased risk of experiencing inflammation.

Patients who were diagnosed with COVID-19 in the past have benefited from the utilization of MCO membranes. This is something that could happen given how cytokines are removed from the body by contemporary dialysis equipment. By utilizing MCO membranes for a period of four weeks prior to the age of COVID-19, a reduction in the total amount of TNF-alpha and IL-6 mRNA was achieved. According to the findings that we obtained from the research that we conducted, we discovered that patients who were able to remove IL-6 using MCO membranes survived COVID-19. Some of the patients, but not all of them, exhibited the same symptoms. A recent study found that patients undergoing dialysis who had high IL-6 levels had a lower chance of recovering from their condition. [Citation needed] Blood samples taken from patients who passed away prior to starting dialysis treatment revealed higher levels of the cytokine IL-6. Some individuals with high blood pressure were more concerned about their overall health than the majority of those who also suffered from the same condition. (Jofré *et al*. 2006).

4.1.3 Relation of renal function parameters with COVID-19

Urea and CRE were measured in serum of all groups. The mean ± SD of urea and CRE for these groups were shown in Table 4.3 and Figure 4.6, cases of COVID-19 had a significant difference (P< 0.05) increased in urea concentration and CRE levels when compared to the control group, according to the statistical analysis of the results.

As previously mentioned, COVID-19 can cause respiratory problems, but it can also have an impact on other systems like the kidneys; heart; intestines; blood; and nervous system.

Table 4.3 the Mean and SD of urea and creatinine parameter with COVID-19

Clinical charachtristic	Hemodialysis with COVID 19	Hemodialysis without infection Conrol	Control	p-value		
				1x2	1x3	2x3
Creatnine	4.55± 1.99	5.38±2.01	0.7±0.2	0.471	<0.001	<0.001
Urea	103.91±6.31	91.81±8.11	33.00 ±6.2	0.27	<0.001	>0.001

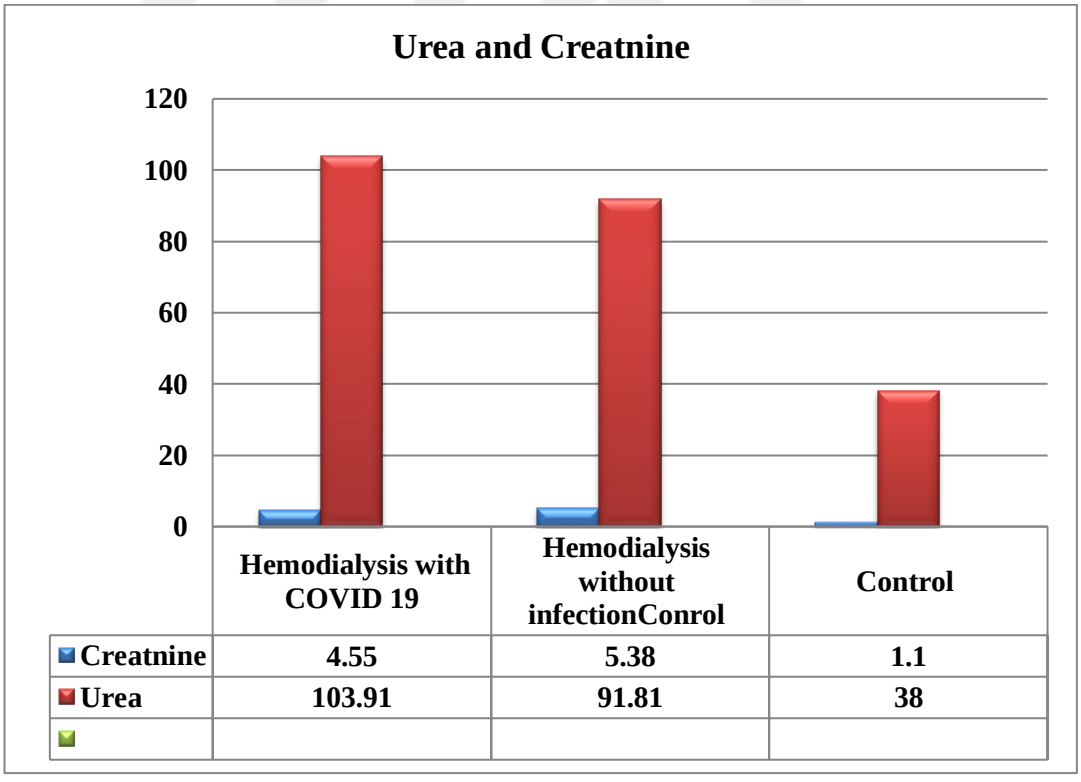

Figure 4.6 Mean and SD of urea and creatinine for hemodialysis patients with COVID and control groups

Since the earliest symptoms of viral pneumonia, which were the most common, researchers have had an interest in the SARS-CoV-2 infection. Because they serve as a reservoir for the virus, the renal tubules have the potential to become a new site of infection for SARS-CoV-2. This potential is due to the tubules' ability to store the virus. These tubules contain a high concentration of the enzyme ACE2. According to the findings of this research, the Middle Eastern Respiratory Syndrome-CoV virus, also known as MERS-CoV, has been associated with infection of renal tubular epithelial cells. Granules have been detected in the urine of SARS-CoV-2 infected patients as well as those who have been in contact with the virus. This virus appears to specifically target the kidneys, as demonstrated by the findings of an autopsy that utilized electron micrographs of kidney tissue. There is a need for additional research because the SARS-CoV-2 virus causes damage to the kidneys (Su et al. 2020).

The effects of COVID-19 on both short-term and long-term clinical outcomes related to acute kidney injury were investigated in this study. Patients who started out the study with normal serum creatinine levels saw a rapid decline in their eGFR and Ccr values. This was one of the findings of the study. According to a recent study, patients diagnosed with COVID-19 had normal levels of BUN and Scr, but abnormal rates of eGFR and Ccr in the range of 67 percent and 41 percent, respectively. After a patient is admitted to the hospital, the percentage of patients who have a decreased eGFR or Ccr rises to 22 or 24 percent, respectively, of the total patient population. An earlier investigation came to the conclusion that the number is significantly lower. The fact that there were more patients present who were representative of the general population might be able to explain this finding. Early renal impairment was frequently observed in COVID-19 patients, even in those patients whose illness was either mild or moderate. Twenty-five percent of patients hospitalized with COVID-19 develop acute renal failure at some point during their illness. The mortality rate associated with this disease has been shown to be quite high (Su et al. 2020).

In previous studies, more emphasis was placed on acute kidney injury as opposed to the early stages of renal function recovery (such as decrease of eGFR or Ccr). Acute kidney injury, also known as AK, is something that can be avoided by recognizing and treating

renal failure as soon as it develops. A high value for the eGFR It was discovered that people infected with the COVID-19 strain had a more dire prognosis. They had normal levels of BUN and Scr, but an unusual pattern of eGFR decline was observed in them. When it comes to determining the patient's prognosis, BUN, Scr, or acute renal injury may not be as reliable of an indicator as eGFR (Su et al. 2020).

Both of these indicators of glomerular and podocyte attack, which are increasingly being recognized as an increased risk factor for renal disease, are important. The risk of developing renal disease is increasing. [This is just one example:] [This is just one example:] The following is an illustration of a scenario in which this might be pertinent: [According to the findings of the research conducted on approximately 710 people who were infected with COVID-19, 44 percent of those individuals had proteinuria and 26.9 percent of those individuals had blood in their urine. In patients suffering from COVID-19, the researchers are keeping their fingers crossed that urinalysis will be able to pick up any early warning signs of renal impairment. Researchers were unable to determine the clinical significance of their findings or the onset of the disease. During the course of our research, we came to the conclusion that hematuria and proteinuria are both symptoms that point to an increased likelihood of developing COVID-19. (Yan *et al.* 2020).

4.1.4 Relation of hematological parameter with COVID-19

Table 4.4 showed highly significant results regarding the relation of COVID-19 infection with white blood cells count and decreases count with the increase in the severity of the COVID-19 case. Also the results showed that there was decrease in white blood cells count COVID-19 patients in comparison with that of control group.

Table 4.4 The Mean ± SD of Hematological parameter in patients groups with COVID-19 and control group

	Control Mean ± SD	**Patients Mean ± SD**	**P-value**
WBC COUNT	6.64 ±1.36 (5.41-8.22)	3.6 ± 0.73 (3.2-4.4)	T=2.09 0.041 NS

Neutrophils (109 /L)	2.69 ± 0.41 (2.28-4.89)	8.85 ± 0.85 (3.45-6.84)	T=8.05 P<0.001
Lymphocytes (109/L)	1.65 ± 0.32 (1.21-2.14)	0.595 ± 0.08 (0.71-1.31)	T=6.91 P<0.001
Hemoglobin (g/L)	13.7 ± 6.29 (12.8-15.07)	13.41 ± 6.23 (12.6-14.9)	T=0.118 P= 0.436
Platelets (109 /L)	228.5 ± 77.206 (187-289)	414 ± 62.95 (169-314)	T=0.565 p=0.931

Studies have shown that our patients have a similar biological profile, which includes the presence of lymphopenia in very ill patients upon admission and an increase in the severity of this illness as they progress through our facility. This is one of the characteristics that defines our patient population.

Even in cases where the total number of white blood cells is normal, a COVID-19 infection can still lead to a reduction in lymphocyte count, which is known as lymphopenia. Mild lymphopenia (less than one million lymphocytes/L) and leukopenia (less than three million lymphocytes/L) were present in patients who were diagnosed with COVID-19. On the other hand, it was discovered that the number of monocytes and neutrophils was within the normal range. (Liu *et al.* 2020).

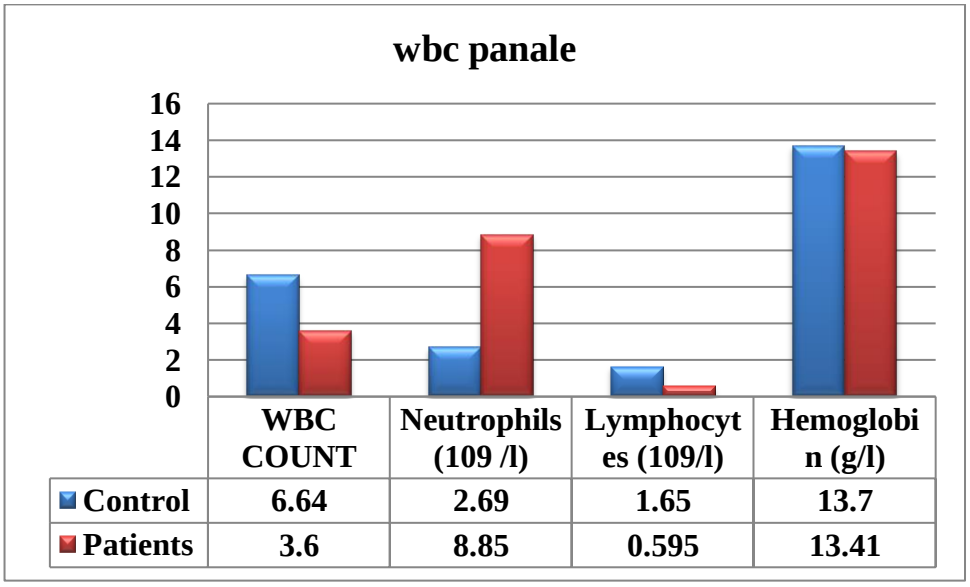

Figure 4.7 The levels of WBC and Hb parameter in patients groups with COVID-19 and control group

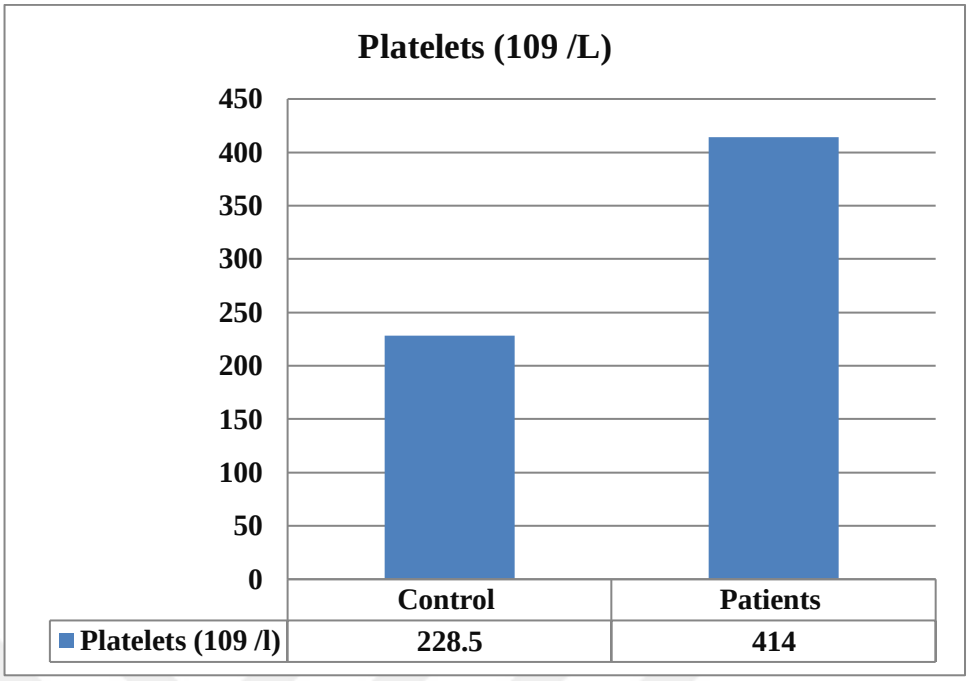

Figure 4.8 The levels of platelet parameter in patients groups with COVID-19 and control group

According to the findings of our investigation, a number of earlier studies, including but not limited to the one we conducted ourselves, have come to the same conclusions (Li et al. 2020). One hundred eighty-seven percent of patients diagnosed with COVID-19 had lymphopenia (less than one million lymphocytes per liter) for the duration of their illness, and sixty-four percent of patients had transitory leukopenia in the first week of their illness.

Within one week of the first reported case of COVID-19 infection in the United States, there was a correlation found between the illness and a slight drop in white cell counts (Lin et al. 2020). According to their initial blood counts, a quarter of the 41 COVID-19 patients who were admitted to Jin Yin-tan Hospital had leukopenia and lymphopenia when they first arrived (accounted for 63 percent). A decrease in the number of lymphocytes can occur in the body as a consequence of an infection caused by a virus. This condition is referred to as lymphocytosis. These three coronaviruses, SARS-CoV, MERS-CoV, and SARS-CoV-2, are all examples of coronaviruses that can trigger the immune system to destroy lymphocytes. In addition, it's possible that the process could be set in motion by combining these two different mechanisms (Lin et al. 2020).

Researchers discovered that people who had COVID-19 had lower peripheral lymphocyte counts; the reason for this lymphocytopenia in COVID-19 patients is not known at this time. According to the limited autopsy and pathological results, the infected patient had necrosis of the spleen and lymph nodes, decreased bone marrow hematopoiesis, and lymphocytic infiltration in the alveolar septum. Additionally, the patient had lymphocytic infiltration in the alveolar septum. These findings were determined after the patient's lungs were examined, and they are as follows: (Yuan et al. 2020). As a consequence of this, the patient's peripheral blood contained a lower number of CD4+ and CD8+ T cells, but the cells that were there were hyperactive. CD4+ T cells had a greater number of cytotoxic particles and a greater number of CCR6+Th17 cells, whereas CD8+ T cells had a greater number of cytotoxic particles and a greater number of CCR6+Th17 cells (Yuan et al. 2020).

An overactive immune response occurred in patients who were infected with COVID-19. This is demonstrated by the increase in pro-inflammatory regulatory T cells and the high level of CD8+T cell cytotoxicity that occurred in these patients. [Citation needed] A few of the participants in this study displayed symptoms of this condition. As a direct consequence of this, patients who were infected with COVID-19 experienced severe immunological impairment (Rouse and Sehrawat 2010).

Lymphopenia is a condition that is experienced by people who are infected with COVID19. This condition can be brought on by a variety of different factors. Researchers from China conducted a study in which they compared the hemograms and lymphocyte subpopulations of patients suffering from mild and severe forms of the illness. All patients, regardless of severity, are being considered for participation in this study. Patients who were suffering from severe illnesses had a ratio of neutrophils to lymphocytes that was higher, as did inflammatory indicator levels (CRP, ferritin, interleukin-6, interleukin-8, and interleukin-10). Severe patients had a higher percentage of CD4 lymphopenia, a higher percentage of CD4-nave cells, and a higher percentage of CD4 suppressor T cells in their lymphocyte populations than non-severe patients did. Severe patients also had a higher percentage of CD4 suppressor T cells (Santos et al. 2022).

According to previously conducted research, Covid-19 may have an effect on lymphocytes, particularly T cells, leading to a reduction in the number of CD4 and CD8 cells. Virus particles are distributed throughout the respiratory mucosa by ciliated bronchial epithelial cells that have been infected with angiotensin-converting enzyme-2 receptor. The cytokine storm that is triggered by this causes damage to WBCs as well as other immune cells such as lymphocytes (Santos et al. 2022).

The ability to replace those lymphocytes that have been killed by the virus may be critical to their long-term survival, according to their meta-analysis, which found that COVID-19 can be predicted by a patient's CD4 lymphocyte count. These findings are extremely significant because COVID-19 is an infectious disease that has the potential to be fatal.

5. CONCLUSIONS AND RECOMMENDATIONS

According to the findings of this research, it is possible to draw the following conclusions:

5.1 Conclusions

1. There is a correlation between how terrible COVID-19 is and a person's age, particularly if they are above 55. COVID-19 is more frequent in adults over the age of 65, according to these research.
2. Men are infected about seven times more often than women, who account for around one-third of all cases.
3. Comorbidities revealed a significant difference between hemodialysis instances of diabetes, high blood pressure, patients experienced significant improvements.
4. The study also indicated that there are a significant high levels in urea and creatinine levels especially in the severe cases of COVID-19 patients
5. Present study shown a significant increase in D-Dimer and IL-6 accompiened with hemodialysis with COVID 19 cases.

5.2 Recommendations

1- Estimation of ACE especially in asthma and other lungs condition cases.
2- Estimation of some immunological factor who affected by hematological change such as CD4+, CD8+, especially in severe cases.
3- Utilizing a molecular technique for growth analysis, for example (constant PCR, Microarray) to reach more sensitivity.

RFERENCES

Askari, H., Sanadgol, N., Azarnezhad, A., Tajbakhsh, A., Rafiei, H., Safarpour, A. R. and Omidifar, N. 2021. Kidney diseases and COVID-19 infection: causes and effect, supportive therapeutics and nutritional perspectives. Heliyon, 7(1): e06008.

Bartsch, S. M., Ferguson, M. C., McKinnell, J. A., O'shea, K. J., Wedlock, P. T., Siegmund, S. S. and Lee, B. Y. 2020. The Potential Health Care Costs and Resource Use Associated With COVID-19 In The United States: A simulation estimate of the direct medical costs and health care resource use associated with COVID-19 infections in the United States. Health Affairs, 39(6): 927-935.

Batlle, D., Soler, M. J., Sparks, M. A., Hiremath, S., South, A. M., Welling, P. A. and Swaminathan, S. 2020. Acute kidney injury in COVID-19: emerging evidence of a distinct pathophysiology. Journal of the American Society of Nephrology, 31(7): 1380-1383.

Boraschi, P. 2020. COVID-19 pulmonary involvement: is really an interstitial pneumonia. Academic Radiology, 27(6): 900.

Brienza, N., Puntillo, F., Romagnoli, S. and Tritapepe, L. 2021. Acute kidney injury in coronavirus disease 2019 infected patients: a meta-analytic study. Blood Purification, 50(1): 35-41.

Chakraborty, I. and Maity, P. 2020. COVID-19 outbreak: Migration, effects on society, global environment and prevention. Science of the Total Environment, 728: 138882.

Cheng, Y., Luo, R., Wang, K., Zhang, M., Wang, Z., Dong, L. and Xu, G. 2020. Kidney disease is associated with in-hospital death of patients with COVID-19. Kidney İnternational, 97(5): 829-838.

Cravedi, P., Mothi, S. S., Azzi, Y., Haverly, M., Farouk, S. S., Pérez-Sáez, M. J. and Riella, L. V. 2020. COVID-19 and kidney transplantation: results from the TANGO International Transplant Consortium. American Journal of Transplantation, 20(11): 3140-3148.

Diao, B., Wang, C., Wang, R., Feng, Z., Zhang, J., Yang, H. and Chen, Y. 2021. Human kidney is a target for novel severe acute respiratory syndrome coronavirus 2 infection. Nature Communications, 12(1): 1-9.

El Aidaoui, K., Haoudar, A., Khalis, M., Kantri, A., Ziati, J., El Ghanmi, A. and El Kettani, C. 2020. Predictors of severity in Covid-19 patients in Casablanca, Morocco. Cureus, 12: 9.

Fabrizi, F., Alfieri, C. M., Cerutti, R., Lunghi, G. and Messa, P. 2020. COVID-19 and acute kidney injury: a systematic review and meta-analysis. Pathogens, 9(12): 1052.

Gagliardi, I., Patella, G., Michael, A., Serra, R., Provenzano, M. and Andreucci, M. 2020. COVID-19 and the Kidney: From Epidemiology to Clinical Practice. Journal of Clinical Medicine, 9(8): 2506.

Gagliardi, I., Patella, G., Michael, A., Serra, R., Provenzano, M. and Andreucci, M. 2020. COVID-19 and the kidney: from epidemiology to clinical practice. Journal of Clinical Medicine, 9(8): 2506.

He, J., Tao, H., Yan, Y., Huang, S. Y. and Xiao, Y. 2020. Molecular mechanism of evolution and human infection with SARS-CoV-2. Viruses, 12(4): 428.

Herlambang, H., Saputra, N. E., Supian, A. I. and Rahman, M. A. 2021. The Perception of Jambi Community About th e Impact of Covid-19 and Efforts to Overcome It, 3: 200-205.

Hillion, S., Arleevskaya, M. I., Blanco, P., Bordron, A., Brooks, W. H., Cesbron, J. Y. and Renaudineau, Y. 2020. The innate part of the adaptive immune system. Clinical Reviews in Allergy & İmmunology, 58(2): 151-154.

Hoffmann, M., Kleine-Weber, H., Krüger, N., Müller, M., Drosten, C. and Pöhlmann, S. 2020. The novel coronavirus 2019 (2019-nCoV) uses the SARS-coronavirus receptor ACE2 and the cellular protease TMPRSS2 for entry into target cells. BioRxiv. 10: 1-18

Hsu, C. M. and Weiner, D. E. 2020. COVID-19 in dialysis patients: outlasting and outsmarting a pandemic. Kidney International, 98(6): 1402-1404.

Huang, C., Wang, Y., Li, X., Ren, L., Zhao, J., Hu, Y. and Cao, B. 2020. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. The Lancet, 395(10223): 497-506.

Jofré, R., Rodriguez-Benitez, P., López-Gómez, J. M. and Pérez-Garcia, R. 2006. Inflammatory syndrome in patients on hemodialysis. Journal of the American Society of Nephrology, 17(12): S274-S280.

Kataria, A., Yakubu, I., Winstead, R., Gowda, M. and Gupta, G. 2020. COVID-19 in Kidney Transplantation: Epidemiology, Management Considerations, and the Impact on Kidney Transplant Practice. In Transplantation Direct, 6(8): e582.

Li, K., Fang, Y., Li, W., Pan, C., Qin, P., Zhong, Y. and Li, S. 2020. CT image visual quantitative evaluation and clinical classification of coronavirus disease (COVID-19). European Radiology, 30(8): 4407-4416.

Li, X., Zai, J., Zhao, Q., Nie, Q., Li, Y., Foley, B. T. and Chaillon, A. 2020. Evolutionary history, potential intermediate animal host, and cross-species analyses of SARS-CoV-2. Journal of Medical Virology, 92(6): 602-611.

Lin, L., Lu, L., Cao, W. and Li, T. 2020. Hypothesis for potential pathogenesis of SARS-CoV-2 infection–a review of immune changes in patients with viral pneumonia. Emerging Microbes and İnfections, 9(1): 727-732.

Liu, J., Li, S., Liu, J., Liang, B., Wang, X., Wang, H. and Zheng, X. 2020. Longitudinal characteristics of lymphocyte responses and cytokine profiles in the peripheral blood of SARS-CoV-2 infected patients. EBioMedicine, 55: 102763.

Luan, J., Lu, Y., Gao, S. and Zhang, L. 2020. A potential inhibitory role for integrin in the receptor targeting of SARS-CoV-2. Journal of Infection, 81(2): 318-356.

Naicker, S., Yang, C. W., Hwang, S. J., Liu, B. C., Chen, J. H. and Jha, V. 2020. The Novel Coronavirus 2019 epidemic and kidneys. Kidney International, 97(5): 824-828.

Naicker, S., Yang, C. W., Hwang, S. J., Liu, B. C., Chen, J. H. and Jha, V. 2020. The novel coronavirus 2019 epidemic and kidneys. Kidney International, 97(5): 824-828.

Palevsky, P. M. 2021. COVID-19 and AKI: Where do we stand?. Journal of the American Society of Nephrology, 32(5): 1029-1032.

Perico, L., Benigni, A. and Remuzzi, G. 2020. Should COVID-19 concern nephrologists? Why and to what extent, the emerging impasse of angiotensin blockade. Nephron, 144(5): 213-221.

Qi, F., Qian, S., Zhang, S. and Zhang, Z. 2020. Single-cell RNA sequencing of 13 human tissues identify cell types and receptors of human coronaviruses. Biochemical and Biophysical Research Communications, 526(1): 135-140.

Rapezzi, C. and Ferrari, R. 2020. The Cardiologist at the time of Coronavirus. a Perfect Storm, 41(13): 1320-1322.

Richardson, S., Hirsch, J. S., Narasimhan, M., Crawford, J. M., McGinn, T., Davidson, K. W. and Northwell COVID-19 Research Consortium. 2020. Presenting characteristics, comorbidities, and outcomes among 5700 patients hospitalized with COVID-19 in the New York City area. Jama, 323(20): 2052-2059.

Ronco, C., Reis, T. And Husain-Syed, F. 2020. Management of acute kidney injury in patients with COVID-19. The Lancet Respiratory Medicine, 8(7): 738-742.

Rouse, B. T. and Sehrawat, S. 2010. Immunity and immunopathology to viruses: what decides the outcome. Nature Reviews Immunology, 10(7): 514-526.

Santos, V. B. D., Stein, A. T., Barilli, S. L. S., Garbini, A. F., Almeida, R. C. D., Carazai, D. D. R. and Wittke, E. I. 2022. Adult patients admitted to a tertiary hospital for COVID-19 and risk factors associated with severity: a retrospective cohort study. Revista do Instituto de Medicina Tropical de São Paulo, 64: 1-5.

Su, H., Yang, M., Wan, C., Yi, L. X., Tang, F., Zhu, H. Y. and Zhang, C. 2020. Renal histopathological analysis of 26 postmortem findings of patients with COVID-19 in China. Kidney İnternational, 98(1): 219-227.

Su, H., Yang, M., Wan, C., Yi, L. X., Tang, F., Zhu, H. Y. and Zhang, C. 2020. Renal histopathological analysis of 26 postmortem findins of patients with COVID-19 in China. Kidney İnternational, 98(1): 219-227.

Sun, P., Lu, X., Xu, C., Sun, W. and Pan, B. 2020. Understanding of COVID-19 based on current evidence. Journal of Medical Virology, 92(6): 548-551.

Taji, L., Thomas, D., Oliver, M. J., Ip, J., Tang, Y., Yeung, A., Cooper, R., House, A. A., McFarlane, P. and Blake, P. G. 2021. Covid-19 in patients undergoing long-term dialysis in ontario. CMAJ, 193(8): E278-E284.

Yan, L., Zhang, H. T., Goncalves, J., Xiao, Y., Wang, M., Guo, Y. and Yuan, Y. 2020. An interpretable mortality prediction model for COVID-19 patients. Nature Machine İntelligence, 2(5): 283-288.

Yan, Q., Zuo, P., Cheng, L., Li, Y., Song, K., Chen, Y. and Gao, H. 2021. Acute kidney injury is associated with in-hospital mortality in older patients with COVID-19. The Journals of Gerontology: Series A, 76(3): 456-462.

Zhang, J., Litvinova, M., Wang, W., Wang, Y., Deng, X., Chen, X. and Yu, H. 2020. Evolving epidemiology and transmission dynamics of coronavirus disease 2019 outside Hubei province, China: a descriptive and modelling study. The Lancet Infectious Diseases, 20(7): 793-802.

Zou, X., Chen, K., Zou, J., Han, P., Hao, J. and Han, Z. 2020. Single-cell RNA-seq data analysis on the receptor ACE2 expression reveals the potential risk of different human organs vulnerable to 2019-nCoV infection. Frontiers of Medicine, 14(2): 185-192.

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