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An Overview of Kidney Failure in Covid-19 Patients

Anwar Abd-Al-Hameed Kamal[*1], Shrooq Majeed Mohameed Saeed [*2]

[1,2]Department of Physiology, College of Medicine, University of Babylon, Iraq.

Corresponding Author: Anwar Abd-Al-Hameed Kamal

Abstract:
A coronavirus (COVID-19) illness epidemic began in Wuhan, in December and quickly disseminated to other locations across the world. Although severe respiratory failure and diffuse alveolar injury were the major symptoms, organ involvement has to be investigated. Since there is little information on renal illness in people with COVID-19, we looked at how frequently these people have acute kidney damage (AKI). Additionally, we looked at the correlation between COVID-19 patients' mortality rates and signs of impaired renal function. The coronavirus disease outbreak from 2019 (COVID-19) at the start of 2020 caused a pandemic and widespread concern. Since then, the fatality rate has risen in tandem with the exponential rise in the number of infected persons. The mortality risk of COVID-19 in individuals with renal illness has not yet been compiled in research on Kidney disease (KD). As a result, the current study's goal was to provide an overview of kidney failure in patients. Patients with KD with COVID-19 infection had a death rate that was considerably greater than that of KD patients without COVID-19 infection; these patients have a high mortality risk and need a thorough multidisciplinary care plan.

Key Words: kidney disease (KD). CD68, GFR, End-Stage Renal Disease (ESRD).

Introduction

The first severe acute respiratory syndrome new beta-coronavirus (SARS-CoV-2)- related coronavirus disease epidemic (COVID-19) was reported in Wuhan, China, in December 2019 (Al-Hindy et al 2023, Al Sa'ady et al 2022, Karim et al 2023). Kidney failure has an unacceptably high mortality rate, which is defined as a glomerular filtration rate that is consistently less than 15 mL/min per 173 m2. Mortality in poor nations is mostly caused by a lack of access to renal replacement treatment. In addition, individuals on dialysis or following a kidney transplant had much greater rates of cardiovascular and non-cardiovascular mortality than the general population. A variety of socioeconomic variables impact the mortality of patients receiving renal replacement treatment. Creatinine and other waste items are removed from the blood by healthy

kidneys (Rasooly et al.,2015). Your body excretes filtered waste through your urine (Negm et al.,2022). A higher level of creatinine may build up in the blood if the kidneys are not working correctly. The serum creatinine test measures the amount of creatinine in the blood, provides a glomerular filtration rate estimation of how well the kidneys are filtering and can measure urine creatinine. Similarly, creatine is a substance that may be produced either directly from creatine or indirectly by non-enzymatic stimulation. It is typically generated by the body at a very steady pace (depending on muscle mass chemically). A cyclic derivative of creatine is creatinine. Although there is a tiny quantity that the kidneys actively promote in the urine, it is mostly taken from the blood through the kidneys (Kielar et al.,2020). If renal clearance is compromised and blood creatinine levels increase, there is little or no tubular absorption of creatinine. As a result, the purity of creatinine (CrCl), which represents the GFR and the glomerular filtration rate, may be determined using the amounts of creatinine in the blood and urine (Wołyniec et al.,2019). Given that it measures renal function, it is significant from a clinical standpoint. Cimetidine and trimethoprim decrease tubular creatinine secretion, which improves the accuracy of estimations of glomerular filtration rate, especially in the context of acute renal failure. Total creatinine cleaned of DNA or ketosis (in the absence of creatinine secretion it may behave like insulin) When evaluating the blood (plasma) content of creatinine from urea, a more comprehensive estimation of renal function may be demonstrated. In addition to kidney-related issues, the ratio of urea to creatinine may also signal the existence of pre-kidney issues including volume depletion. As an example, the level of urea elevated from the ratio of creatinine may suggest this (Ellis,2011). The body categorizes creatine molecules as a biological waste product produced by the muscles. Creatinine is excreted by the body through the kidneys, where it is filtered and expelled in the urine after traveling through the circulation to the kidneys. In actuality, because creatinine is not an organic acid, it serves no important functions in the body. However, creatinine aids in measuring the effectiveness of renal function in the blood. Since the kidneys are largely in charge of removing creatinine from the body, any problem with them has an impact on the level of creatinine in the blood, whether it is low or high.Chemically, The body converts 2% of creatine each day into creatinine, making creatine a cyclic derivative of creatine. In other words, creatinine is an amino acid that is made by the liver, kidneys, and pancreas naturally in the human body. It may also be consumed through animal products. The kidneys remove creatinine from the blood. Although just a small quantity of creatinine is actively eliminated by the kidneys into the urine, creatinine levels rise in the blood when the kidneys are not filtering the blood as thoroughly. Through a procedure called a creatinine clearance analysis, which represents the glomerular filtration rate, the concentrations of creatinine in the blood and urine may be utilized to determine the purity of creatinine (Narasimhan et al.,2001). As a measure of kidney function, the glomerular filtration rate is crucial for clinical purposes since any impairment of kidney function, regardless of the source, raises the blood's amount of creatinine. A molecule called urea is present in the blood and lymph of humans. It is first excreted through the urine when amino acids and ammonia molecules are removed by the liver. Waste that exits the body, whether it comes from the inside or the outside, is known as urea. Either amino acids are removed from amino acids that are already present in the body or proteins are broken down when biological tissues are being rebuilt. One of the most significant waste products in the human body is urea, which is formed during the breakdown of proteins (Mayne et al.,2021). Since it's crucial to monitor the quantity of urea in the blood for many health issues, having a high level of urea in the blood is a sign that something is wrong with the body. The liver analyzes the protein in meals to make BUN, which is then released into the circulation together with urea. This results in the accumulation of this material in the kidneys, which then try to eliminate it, leaving it behind. Urea is a natural waste product produced by the human body as well. Low concentrations or levels in the blood (Bahrebar,2021). It is important to remember that the kidneys eliminate urea through urine, but if their function is compromised or they are unable to carry out their duties as needed, they will have trouble eliminating the urea already in the blood, which will boost its levels in the body. One of the common procedures used to assess the kidneys' functionality and general health is the examination

of urea or blood urea nitrogen. Since urea is naturally made by the liver after it breaks down the proteins in meals, it is then delivered to the kidneys, where it is subsequently removed by the kidneys by excreting it with the urine (Tynkevich et al .,2014).If there is an issue with the kidneys, it might result in the material staying in the body and a high blood level. The urea analysis aids in the diagnosis of certain medical disorders and assesses the efficacy of the dialysis regimen (Steen,2014).

SARS-CoV-2 with Kidney Disease:

Since it was initially discovered in Wuhan in December 2019, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has swiftly spread around the world. Despite enormous worldwide public health efforts, the pandemic began on March 11, 2020, with the breakout. More than 120 million individuals were impacted by the coronavirus disease 2019 (COVID-19) after a year, and approximately 3 million people died as a result (Lai et al.,2020,). Acute viral pneumonia with respiratory failure, septic shock, and multiple organ dysfunction are all included in the clinical spectrum of SARS-CoV-2 infection, as are asymptomatic, moderately and severely sick respiratory conditions, and respiratory disease without other symptoms. Advanced age, immunosuppressive drugs, and underlying comorbidities such as chronic renal illnesses, malignancies, diabetes, and cardiovascular and chronic lung diseases are all linked to a greater risk of the severe clinical presentation of COVID-19. The increasing loss of renal function in KD leads to altered innate and adaptive immunological responses, including reduced leukocyte phagocytic activity, diminishing dendritic cells that deliver antigens, faster T cell turnover, and increased apoptosis of CD4+ and CD8+ lymphocytes (Charitos et al.,2020). When the immune system is compromised, infections are more likely to occur and progress more severely, which appears to account for a significant portion of mortality, particularly in patients with end-stage renal disease (ESRD). Immune activation is seen in individuals with chronic renal disease in addition to secondary immunodeficiency (Marti et al.,2001). Increased synthesis of pro-inflammatory cytokines causes systemic inflammation, which worsens the patients with corona infection by causing atherosclerotic cardiovascular disease and other diseases (Cozzolino et al.,2018).

Pathogenesis:

Following the coronavirus spike (S) protein binding to ACE2 receptors, the transmembrane serine proteases family (TMPRSS) breaks and activates the S protein, enabling the virus to release incorporation, a peptide that promotes membrane fusion (Pan et al.,2020, Suhad et al 2023).

Mechanism:

Kidney illness brought on by COVID-19 infection may result from a synergistic interaction between the systemic inflammatory response brought on by cytokines and the direct cytotoxic impact of the virus. Patients with severe illnesses, those with ARDS, and those who require ICU hospitalization have more significant KD. (ATN) caused by multiorgan trauma is another potential cause of KF, as well as a potential prerenal etiology caused by volume loss as a result of reduced intake and a high temperature. Additionally, drug toxicity, hemodynamic harm, and contrast exposure may be factors. Similar to other causes of KF, the workup for COVID-19 infection should include KF testing. In their study, the authors examined many causes of KF, including pre-renal azotemia, ischemia, toxicity, or a combination of both (Mohamed et al.,2020).

Direct Viral Damage:

In situ hybridization and immunohistochemical analysis of kidney tissues from SARS autopsy patients revealed the presence of corona (RNA) fragments (Ding et al.,2004). MERS-CoV infections also have been shown to cause kidney epithelial cell damage by apoptosis mediated through receptors (Yeung et al.,2016). These investigations imply that kidney epithelial cells directly experience cytotoxicity as a result of coronavirus damage. Immunohistochemistry revealed the presence of corona (NP) protein in the tubule of the kidney when Diao et al. examined the kidney tissue from six patients who had died via autopsy. This discovery could be related to a potential direct tubular damage caused by the virus. A quantitative real-time polymerase chain reaction was used to examine the patient's blood for the presence of SARS-CoV-2 RNA (qRT-PCR) (Diao et al.,2020).

Immune-Mediated Damage:

The corona infection and likely immune response resulted in higher levels of cytokines. The noticeably elevated cytokine levels are proof that cytokine storms may develop in extreme circumstances. Instead of SARS-CoV-2, healthy tissues are harmed during the cytokine storm by the immune system. (CD68)+ macrophage infiltration of the significant ATN was seen on the autopsies of six kidney tissues. In all six instances, complement 5b-9 deposition was present in the tubules, whereas deposition in the glomeruli and capillaries was infrequent. In kidney tissue, several CD8+ T - T-cells and CD56+ (NKC) cells were seen (Wrapp et al.,2019).

Conclusions:

According to the analysis, COVID-19 infection can affect the kidneys and be linked to a significant death rate. Early recognition of this component by healthcare professionals is crucial, and prompt implementation of proper management is essential. Patients who have undergone a kidney transplant and those with underlying kidney disease are more likely to get COVID-19 infection.

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