Journal of Research in Medical and Dental Science
2022, Volume 10, Issue 8, Page No: 170-175

Available Online at: www.jrmds.in
eISSN No. 2347-2367: pISSN No. 2347-2545

Covid 19 in Patients with Renal Diseases

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ABSTRACT

Whole world has been frightened because of the rapid rise in the number of cases and deaths due to novel corona virus (officially referred to as COVID-19) which was found in late 2019 in Wuhan city, China. Till date, increased spread of novel coronavirus Disease (COVID-19) is even now at an uncontrollable rate and the amount of confirmed patients and mortality rate is still increasing at an exponential rate. Till 1st October 2020, from 216 different nations, regions, and territories, the World Health Organization (WHO) received reports of 33,842,282 confirmed cases and 1,010,635 confirmed deaths. This virus causes a variety of symptoms which can range from mild illness to moderate illness. Difficulty in breathing and death occur in severe cases. The common earliest manifestations include fever, cough, fatigue, myalgia, diarrhoea and headaches. The disease is mainly transmitted through respiratory droplets from a person who is infected, via coughing, sneezing or exhaling. Adding together expertizes across various domains to a variety of new and effective solutions are the need of hour. Very little is known about how corona virus affects the kidneys. There's no such proof that shows if people with renal diseases are more susceptible than general public. Studies have been made over complications of coronavirus disease in patients with renal diseases and how to tackle them. This review article summarizes on the analysis of present situation with subject to coronavirus pandemic, its sequel on people with renal disorders and the seriousness of the disease. Majorly it gives an outline in management of this critical condition.

Key words: Covid-19, Kidneys, Renal diseases

HOW TO CITE THIS ARTICLE: Ajinkya Kalbande, Pramita Muntode, Covid 19 in Patients with Renal Diseases, J Res Med Dent Sci, 2022, 10 (8): 170-175.

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Received: 30-July-2022, Manuscript No. JRMDS-22-70034;

Editor assigned: 01-August-2022, **PreQC No.** JRMDS-22-70034(PQ);

Reviewed: 16-August-2022, QC No. JRMDS-22-70034(Q);

Revised: 19-August-2022, Manuscript No. JRMDS-22-70034(R);

Published: 26-August-2022

INTRODUCTION

No one can deny the fact that the world has changed dramatically in just a matter of few months due to spread of Novel Corona Virus, COVID-19. This COVID - 19 is a member of SARS family Cov-2 Virus and was identified in Wuhan, China, in December 2019 and has rapidly spread across the world. India being the second most populous country after China, the presence of this virus in a patient was first detected on 30 January 2020 in Kerala. Being an agronomical (agricultural-based economy) country, the majority of population stays in rural areas and access to health care facilities is difficult. To restrict the transmission of the virus in India, a series of lockdown were instituted and as on 8 July 2020 there are 7, 46,506 cases and India is ranked 3rd in the world as far as the number of cases are concerned . As the number suggests a diffuse spread of this disease in India, Maharashtra and Tamil Nadu are amongst the worst affected states with both of them nearly contributing around 50 % of total cases. Although substantial amount of data is available on Corona virus pandemic but still some details are yet to be known about it like, its outcomes and susceptibility on people with renal disorders [1].

MATERIALS AND METHODS

A thorough search was made on online research portal PUBMED using the terminology COVID-19, SARS CoV-2, renal disorders, kidney up to 4th June 2021. Full articles were accessed and analysed.

DISCUSSION

SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) was first discovered in December 2019 and is the cause of the present worldwide pandemic and coronavirus disease 2019 (COVID-19). The respiratory signs of coronavirus disease are the most visible, but acute renal injury (AKI) is now recognized as a common complication of the condition, and it is commonly visible

at the time of admission to the hospital. Although early reports from China suggested relatively low rates of kidney involvement [2-5], subsequent reports from the United States and Europe show much higher rates of AKI, particularly in the intensive care setting, with up to 45 percent of ICU patients requiring kidney replacement therapy (KRT) [6-8]. Hospitalized individuals with COVID-19-associated AKI (COVID-19 AKI) have a greater mortality rate than those who do not have renal disease [9]. The death rate for patients admitted to the ICU with COVID-19 AKI needing KRT is very high, as it is in other cases of AKI requiring ICU admission in the setting of multi-organ failure. Anecdotal accounts of a lack of renal recovery in those who survive when compared to other types of AKI are particularly concerning [10]. Long-term patient outcomes, on the other hand, are still unknown because of longer hospital stays and a paucity of recorded follow-up. Due to disparities in the underlying comorbidities of the populations studied, as well as significant variances in the practice and techniques of AKI diagnosis and reporting, determining the exact epidemiology of COVID-19 AKI is difficult. Patients with COVID-19 have an increased risk of AKI when they are older, have a history of hypertension, or have diabetes mellitus. Chronic kidney disease (CKD) is a well-known risk factor for AKI in hospitalized patients, and in 3,099 critically sick patients with COVID-19, it was found to be the most important risk factor for AKI needing KRT. Indeed, multiple epidemiological studies have shown that CKD is a significant and independent risk factor for poorer COVID-19 results.

COVID 19 and kidney

Coronavirus disease is basically considered respiratory disorder, but the renal organs could also be 1 of the targets of COVID 19 virus infection. This is because the virus enters the cells via the angiotensin-converting enzyme 2 receptor, which is present in kidneys in a large number [11].

Patho-physiology

To understand the pathophysiology behind the coronavirus mechanism, the genome sequence of the similar 3 variants: SARS-coronavirus, SARS-coronavirus 2 and MERS-coronavirus were compared. Coronavirus-2 has a sequence that is 79 percent identical to coronavirus and 50 percent identical to MERS- coronavirus [11]. After analyzing some specific proteins like the coronavirus primary proteinase (3CLpro), papain-like protease (PLpro), and RNA-dependent RNA polymerase (RdRp), it was shown that coronavirus and coronavirus-2 have a 96 percent sequence identity [12]. Thus, a relation between the pathophysiological mechanism of coronavirus 1 and coronavirus 2 has been hypothesized [13].

Coronavirus-2, like coronavirus, requires angiotensin converting enzyme 2 (ACE-2) for penetrating the targeted cells, according to several investigations. ACE-2 is a carboxypeptidase that splits angiotensin I enzyme into angiotensin 1–9 and angiotensin II into angiotensin 1–7, preventing vasoconstriction, proliferation, as well as fibrosing effect of Ang II produced by ACE [14]. Single-celled RNA sequencing research revealed vast spread of ACE-2 in several organs, which was further verified by histochemical staining [15]. However, because some cell types had less amount of expression of ACE 2 , it was assumed that coronavirus-2 cellular contact and internalization were not solely dependent on angiotensin converting enzyme 2, but on various auxiliary cell membrane receptors and proteins. Also, ssRNA viruses are known to have a large number of receptors. The appearance of angiotensin converting enzyme 2 on one hunder and nineteen cell types from thirteen human organs, as well as the expression properties of single stranded RNA human viral receptors and membrane proteins, was studied. 94 genes were discovered to be substantially linked with ACE-2 in a correlation study of gene expression origin [15].

The peptidases alanylaminopeptidase (ANPEP), glutamyl aminopeptidase (ENPEP), and dipeptidyl peptidase 4 (DPP 4) had strongest connection with angiotensin converting enzyme 2. While both peptidases alanylaminopeptidase and dipeptidyl peptidase 4 have been identified as targeted receptor for different COVID-19 viruses (human COVID 229E, swine epidemic diarrhoea virus, canine COVID virus, feline COVID, and MERS-CoV for dipeptidyl peptidase 4), link between glutamyl aminopeptidase and viral disease is unknown. Glutamyl aminopeptidase is a type II integral membrane zinc-containing endopeptidase identified in mammals that belongs to the edopeptidases family M1. It's predominantly found in the terminal section of the ileum and the cortex of the kidney, and it's involved in the functional mechanism of the Renin-Angiotensin System (RAAS), blood pressure regulations, and blood vessel development. While some findings show that ENPEP is a COVID 19 receptor, more research is required for validating this. Decreased angiotensin converting enzyme 2 expression on the surface of the cell, on the other hand, could be regarded as a viral defensive mechanism. Downregulation of ACE-2 has previously been linked to quicker human coronavirus cell-to-cell transmission and increased severity of clinical presentation. A certain study suggested that down regulation of ACE 2 can be a phenomenon caused by coronavirus 2 to achieve quicker diffusion within the cells, even for coronavirus. The overexpression of ACE-2 could be defensive when suffering from SARS-CoV-2 infection, according to studies conducted to investigate the efficacy of angiotensin receptor blocker (ARB) medicines in people suffering coronavirus 2 infection. ARBs, significantly boost ACE-2 cellular expression. However, because the coronavirus-2-ACE-2 interactivity is only the 1st course of action in a group of incidents, ACE-2 over-expression is not accompanied with a rise in specific cell protease required for viral internalization and activating, SARS-CoV-2 will be sequestered on the membrane of the cell , which will limit the infection. Also, the membrane-bound ACE-2 can be acted on by the metalloproteinase ADAM17, resulting in soluble version

of angiotensin converting enzyme 2 being released. If a rise in soluble ACE-2 is linked to a rise in ACE-2 expression, which could operate like false receptor for coronavirus 2, inhibiting virus from entering in the cells being targeted. The relationship between coronavirus-2 and ACE was confirmed through research on the spike glycoprotein produced on the envelope of the virus, which is similar for all COVID viruses. A certain intracellular component, trans- membrane segment, a large ecto- domain produced by subunit S1 and will interact with the targeted receptor and another subunit S2 and further fuse to form coronavirus spike protein. S1 is made up of 4 components: one N terminal domain (NTD) and three C-terminal domains (CTD1, CTD2, and CTD3). Receptor of the cell and protein of the virus bind via the receptor-binding domain (RBD), which in this case of SARS-CoV is found in the CTD1 domain. CoV and CoV-2 have an increased sequence similarity (89.2%) and sequence identity (73.7%), according to experiments conducted to examine virus-receptor interaction with atomic resolution. Further detailed examination of CoV-2 receptor binding domain, on the other hand, found distinctive traits that are likely to result in the higher dispersion compared to CoV. A single change in the coronavirus-2 binding domain receptor increases its angiotensin converting enzyme 2 binding affinity [16]. According to Heet al., the properties of CoV-2 receptor binding domain would make the virus more dissolvable, allowing it to more easily attach ACE-2, and also giving it higher stability, and will allow it to survive at higher temperatures. The CoV-2 receptor binding domain, on other hand, is has higher flexibility than the CoV receptor binding domain, particularly at the site for binding. As a result, CoV-2 is substantially has increased sensitivity to temperature than coronavirus in relation to the receptor binding domain-angiotensin converting enzyme-2 link, resulting in a decrease in infectivity as temperature rises. The CoV receptor binding domain-ACE-2 interaction causes certain specific alterations in the subunit S2, causing the viral membrane to fuse with the cell. The virus can enter endosomal cells more easily with a decreased pH. Furthermore, host transmembrane cell proteases split S protein into S1 and S2 subunits, which are required for virus entry via th non-endosomal route present on surface of cell.

As a result, the linked expression between ACE-2 and Trans-membrane Serine Protease 2 is a deciding element in CoV-2 entrance in the host cells. CoV-2 uses cell's native transcription process to reproduce and spread after entering and activating it. Cell having infection of coronavirus 2 can acquire and regulate immunogenic cells by producing chemokine and various different cytokine [16]. The function of macrophages is still unknown. However, certain study conducted recently found that the proteins produced by mitochondria which interact with CoV-2 are down regulated. The technique can be construed in a way for this virus to prevent mitochondria-instigated apoptosis [17].

COVID-19 and acute kidney injury

Over a quarter of patients hospitalized with coronavirus disease 2019 (COVID-19) have been reported to develop acute kidney injury (AKI) [18]. Many coronavirus patients in hospitals, according to current data, have renal impairment and may present with features of blood in urine, proteins loss in urine and injury to kidney. Tubular damage is indicated by low molecular weight proteinuria, Fanconi syndrome, and histological abnormalities. Acute kidney injury is more likely in coronavirus infected cases who are seriously ill or in the end stage, and it is associated with a higher risk of death. Acute tubular damage is the most prevalent pathology found in both live kidney biopsies and autopsy series. Other diseases seen in both live and autopsy tissues include collapsing glomerulopathy and thrombotic microangiopathy [19].

severe acute respiratory syndrome coronavirus 2 viral particles were found in renal tubular cells and podocytes, implying direct viral infection, as well as acute tubular necrosis and rhabdomyolysis-associated AKI and glomerulopathies, according to histopathologic study of autopsied kidney tissue [1,19]. The viral infection of kidney cells has been described inconsistently in analyses of kidney biopsy samples from patients with COVID-19 and AKI. Patients with high-risk APOL1 genotypes, particularly those without significant respiratory symptoms, have been found to have collapsing glomerulopathy. Although regional inflammation, endothelial damage, and renal micro thrombi have been documented, their role in the pathophysiology of COVID-related AKI is unknown. Anti-inflammatory drugs (for example, steroids and IL-6 receptor blockers) seem to limit the development of severe AKI in patients with COVID-19 [18].

Angiotensin converting enzyme and SARS COV 2

ACE-2 was identified has functional host receptor for coronavirus 2. This straind of the virus is the one that is causing the present global coronavirus disease outbreak. Angiotensin converting enzyme is widely found in a wide range of cells throughout human body. In human physiology, the breakdown of Ag II, the key participant in the RAAS and the primary substrate of ACE2, is a critical counter-regulatory enzyme to ACE. Alterations in ACE2 expression and COVID-19 severity and development have been associated to age, sex, ethnicity, medication, and several co-morbidities, such as cardiovascular disease and metabolic syndrome. Despite the fact that angiotensin converting enzyme-2 is broadly dispersed in many of human tissues and many determinants of the same are well understood, not all angiotensin converting enzyme-2-expressing organs are involved in coronavirus pathogenesis, indicating that there are other pathways which play a role in functioning of cells and destruction of tissues. Pathologic changes due to coronavirus 2 infection must be found in order to understand coronavirus pathology and physiologyand establish better treatment modalities [20].

Characteristics and outcomes of covid 19 patients with pre-existing CKD history

A research was conducted on 20 patients having Covid 19 pneumonia with a history of CKD, in Tongji hospital, Wuhan between January 20th and March 1st 2020. Clinical baseline data, laboratory findings and chest CT was taken into account. In the review, it was found that SARS-CoV- 2 infected cases with history of chronic kidney disease were older aged people and their most common comorbid condition was hypertension [2]. Pyrexia as well as cough was the commonest symptoms. Laboratory reports showed that low lymphocyte count, raised levels of D-dimer and increased indications for infections like [10] hypersensitive C response protein (hsCRP), interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-α) were routinely seen in the patients. CT scans showed Ground glass opacity (GGO) and consolidations. Few patients had died and some had suffered acute kidney injury during their hospital stay [21].

In a separate study, 4,264 critically ill patients with COVID-19 were admitted to intensive care units (ICUs) (at 68 hospitals across the United States) (143 patients with pre-existing kidney failure receiving maintenance dialysis; 521 patients with pre-existing non-dialysis-dependent CKD; and 3,600 patients without pre-existing CKD).

When compared to other groups, dialysis patients had a shorter duration from symptom onset to ICU admission (median of 4 [IQR, 2-9] days for maintenance dialysis patients; 7 [IQR, 3-10] days for non-dialysis-dependent CKD patients; and 7 [IQR, 4-10] days for patients without pre-existing CKD). Dialysis patients reported changed mental status at a higher rate (25%) than those with non-dialysis-dependent CKD (20%; standardized difference = 0.12) or those without pre-existing CKD (12%; standardised difference = 0.36). Within 28 days of ICU admission, half of dialysis and non-dialysis-dependent CKD patients died, compared to 35% of patients without pre-existing CKD. Compared to patients without pre-existing CKD, dialysis patients had higher risk for 28-day in-hospital death (adjusted HR, 1.41 [95% CI, 1.09-1.81]), while patients with non-dialysis-dependent CKD had an intermediate risk [22].

COVID-19 in hemodialysis patient

Hemodialysis patients more than seventy years of age were found to be at an increased risk of dying from symptomatic COVID-19, while the patients younger than seventy years had a milder form of disease. HD patients over 70 years old had longer dialysis duration, higher CRP, and were more likely to die from COVID-19, whereas those under 70 years old had a milder condition. In majority of cases the common clinical features were diarrhoea (80 percent), pyrexia (60 percent), and weariness (60 percent) [23]. All of the patients had lymphopenia. All of the patients' lungs had ground glass opacity on computed tomography scans. [23] Complications such as acute respiratory distress syndrome, shock, or multiple organ dysfunctions are unlikely to occur. The mortality rate is also relatively low [19,23].

Kidney biopsy findings in patients with COVID-19

The kidney injury caused by Coronavirus Disease 2019 (COVID-19) is hypothesized to be caused by a number of processes. Pathologic analyses have previously been restricted to patient reports and autopsy series [24]. Number of cases with coronavirus disease was found to develop a vast variety of glomerular and tubular disorders. There is no direct evidence that COVID-19-related kidney injury is caused by direct viral infection of the kidneys. Instead, cytokine-mediated effects and heightened adaptive immune responses are implicated [24].

COVID-19 infection in recipients of kidney transplant

Even though covid 19 is a disease of lungs, in major cases, it can cause kidney failure and multi organ failure as well. It is still not know if the people with people with immunocompromised status have an increased chance of acquiring a serious systemic disorder. All the patients of kidney transplant showed the primary respiratory symptoms and fever. Other features that could be commonly seen were, chest crepitation's, hypoxia, lymphopenia and high C reactive protein level. In severe cases high levels of ferritin, troponin and D Dimer levels were found and these have found to be prognostic. Thus, Covid -19 infection in patients with kidney transplant can be dire and the patients need intensive care along with supportive therapy and reduction in immunosuppression [25].

Lupus nephritis and covid-19 infection

For immune-compromised patients with inflammatory autoimmune systemic illnesses, Covid-19 infection is a major threat. Immune dysfunction, immunosuppressive therapy, and excess co-morbidities are most likely to blame. Covid-19 is more common in LN patients who have not received Hydroxycholoroquine medication, which may play a preventive role against the most devastating forms of COVID-19. Men, older patients, and those with comorbidities (lung illnesses, hypertension) and active LN (3 -class IV LN, 2 -class V LN, 1 -class III LN, and 1 -class II LN, according to the 2003 ISN/RPS classification) were more likely to require hospitalization. Male sex, previous lung disease, serum creatinine level, proteinuria, glucocorticoids use >5 mg/day, were significantly associated with hospitalization [26].

Impact of kidney failure on the severity of COVID-19

In terms of the severity of COVID-19 when admitted to the medical facility, it was discovered that pre-existing renal disease was independently related with increased in-hospital mortality, particularly in patients with severe kidney failure, and our findings are consistent with earlier research. Using data from international databases and large multimorbidity studies from various countries, the Global Burden of Disease (GBD) collaboration recently estimated the risk factors for

severe COVID-19 worldwide, concluding that CKD is the condition that carries the highest risk for the disease's severe presentation and COVID-19-related death.

Yang et al. found that in-hospital mortality rates for non-CKD, non-dialysis dependent CKD, and dialysis patients were 9 percent, 50 percent, and 66.7 percent, respectively, in 836 patients. It's worth noting that the proportions of people with a moderate COVID-19 presentation were similar in those without and with non-dialysis dependent CKD (73.7 percent and 75 percent, respectively), but much lower in dialysis patients (40 percent), who were more frequently scored as severe cases on admission, which may have influenced the fatal outcome [25].

CONCLUSION

During this pandemic, it is of utmost importance to be safe, stay indoors and follow healthy sanitary practices. The advancement of our country's healthcare facilities has resulted in greater assistance in the treatment of COVID sufferers. The most commonly affected organs are lungs, manifesting as severe acute respiratory syndrome, but the kidneys are also frequently affected. COVID-19 infection can not only create new kidney damage, but it can also make treatment and care more difficult, as well as raise the mortality risk in patients who already have kidney disease. Kidney tubular damage is the commonest symptom of coronavirus 2 infection in the kidney. Main clinical sign is proteinurea. To reduce mortality rate of the patient, kidney complications should be given increased attention for diagnosing and treating COVID-19. Many interventional procedures are currently being investigated in ongoing clinical trials that include antiviral medicines, biological response modifiers, and renin–angiotensin–aldosterone system inhibitors, among other groups of pharmaceuticals and tactics. COVID-19 will ultimately be combated through prevention, and necessary efforts, such as the development of efficient vaccinations, are being pursued to that end.

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