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COVID-19 PANDEMIC: A REVIEW OF RENAL MANIFESTATION AND COMPLICATION

Dr. Roy Arslan Ahmed[1], Dr. Ambar Shamrez[3], Dr. Aurang Zaib Bhutta[3]

[1]Nishter Medical University Multan, [2]Services Institute of Medical Sciences, Lahore, [3]Islamic International Medical College, Riphah International University, Islamabad.

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Abstract:

The pandemic of the coronavirus disease-19 (COVID-19), the causative agent of the severe acute respiratory syndrome-2 (SARS-CoV-2), has become a public health emergency of the international concern. The majority of the population is exhibiting signs and symptoms similar to the flu and common cold. Despite that, alveolar damage result in progressive lung failure has also been reported. Although COVID-19 has been reported principally to affect the respiratory system, other system's involvement has also been reported. Renal involvement has also been underlined in the literature. Renal involvement in SARS-COV-2 usually corresponds to three situations: (a) renal manifestation of acute kidney injury, (b) renal manifestation of chronic kidney injury, (c) renal manifestation in patients with a kidney transplant, and the patients on dialysis. The actual disease pathogenesis is unknown; however, direct viral injury, inflammation due to markers, activation of the complement system, and coagulation cascades might be the cause. Patients with renal involvement usually experience a wide range of signs and symptoms such as oliguria, anuria, proteinuria, hematuria, lower abdominal and back pain, or elevation in creatinine and blood urea nitrogen. The pandemic of SARS-CoV-2 has become a unique challenge for the nephrologist. Various renal manifestations have been observed and reported in many cases, and even renal features may precede the classical respiratory signs and symptoms. In this review, we have summarized the information from published literature including, case reports and open-source data sets, to describe the spectrum of renal manifestation and complication observed in COVID-19 cases.

Corresponding author:

Dr. Roy Arslan Ahmed,

Nishter Medical University Multan.

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INTRODUCTION:

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-COV-2), the causative agent of COVID-19 (coronavirus disease-2019), first originated from Wuhan, China, during late December of 2019. It started as an outbreak which led to an epidemic with 44,672 confirmed cases in China by February 14, 2020, with a 2.3% reported mortality rate, which was comparatively lower than the previously known epidemics caused by human coronaviruses (Severe Acute Respiratory Syndrome Coronavirus [SARS-CoV] and the Middle East Respiratory Syndrome Coronavirus [MERS-CoV]) in 2003 and 2012 respectively [1, 2]. It rapidly spread outside of China to the rest of the world, mainly through human-to-human transmission by respiratory droplets or possibly through the fecal-oral route. Consequently, the World Health Organization (WHO) declared COVID-19 as a global pandemic on March 11, 2020 [3]. Currently, with the disease spread to 216 countries, there are over 27,769,074 reported confirmed cases worldwide, including over 19,851,252 deaths as of September 09, 2020 [4]. The infection presents most commonly as fever, dry cough, shortness of breath, sore throat, and diarrhea with severe respiratory involvement in patients with advanced age (over age 80). The overall case fatality rate in this age group is about 14.3% [5]. The severity of this disease is characterized by severe pneumonia, respiratory failure requiring mechanical support, sepsis, myocardial injury, multi-organ failure, and mortality increases in patients having underlying comorbidities such as cardiovascular disease, diabetes, chronic kidney disease, and chronic respiratory disease [6, 7]. However, some patients may have very mild symptoms or act as asymptomatic carriers suggesting that the actual number of cases may be much higher than reported [2]. Given the rapid spread of the virus, researchers across multiple nations have dedicated themselves to understand the virus, disease pathophysiology better, and develop effective drugs and preventive vaccines.

Even though, Although COVID-19 has been reported principally to affect the respiratory system, renal involvement has also been underlined in the published literature, raising the concerns about renal invasion by COVID-19. The amount of literature on renal involvement by COVID-19 is small. However, we believe that a structured summary of existing data at this point would be requisite for nephrologists. Being well informed about the renal clinical presentations would not only provide support to them have a high index of clinical doubts but also take obligatory precautions. In this paper, we have summarized the information from published literature, including case reports and open-source data sets, to describe the spectrum of renal manifestations and complications observed in COVID-19 cases.

MATERIAL AND METHODS:

We systematically reviewed the literature on COVID-19 and its relevance to our nephrology practice. A comprehensive literature search was performed using a combination of keywords (MeSH terms and free text words) including 'COVID-19'/'SARS-CoV-2', and "nephrology/renal". Pubmed, EMBASE, and Cochrane Library were searched up to 8th July 2020. A limit of after 2019 was imposed since COVID-19 was first reported in late 2019. Additional articles were sought from the reference lists of the included studies. All articles identified from the literature search were screened by two independent reviewers. We included studies on COVID-19 in adult patients. Case reports, case series, observational studies, non-randomized studies, and randomized trials that were published in English were included in this review. Conferences abstracts, letters to editors, commentaries, and editorials were also included. Studies related to obstetrics and gynecology were excluded.

For eligible studies, study information including first authors, site of study, inclusion and exclusion criteria, sample size, age, and sex were recorded. A standardized form for data entry has been devised to focus on the following areas: (1) renal manifestations of COVID-19; (2) special considerations in renal conditions. Relevant data were analyzed and summarized.

Review:

Mechanism of renal invasion:

The genome of the SARS-CoV-2 consists of single-stranded positive-sense RNA encapsulated within a membrane envelope, which has glycoprotein spikes giving coronaviruses their crown-like appearance [7]. Of the four classes of coronaviruses (alpha, beta, gamma, and delta), SARS-CoV, MERS-CoV, and COVID-19 causative SARS-CoV-2, are included in the class beta. While SARS-CoV, MERS-CoV, and SARS-CoV-2, all attack the lower respiratory tract, SARS-CoV-2 additionally also affects the heart, gastrointestinal system, liver, kidney, and the central nervous system eventually leading to multi-organ failure [8, 9]. Glycosylated spike (S) protein, which is one of the structural proteins encoded by the coronavirus genome, is a major inducer of host immune response. This protein binds to angiotensin-converting enzyme 2 (ACE2) receptor protein located on the host cell surface membrane and mediates the

host cell invasion [9, 10]. ACE2 (entry receptor for SARS-CoV) was particularly confirmed in COVID-19 infection regardless of mutations at key receptor-binding domains. Inhuman transmission and pathogenesis of COVID-19 are based on the interactions, involving virus binding, receptor recognition, cleavage of protease, and membrane fusion.

The exact mechanism of renal involvement is unclear and is most likely due to multifactorial. Renal disease may be caused by COVID-19 disease binding to the ACE2 receptor on kidney cells that allows the virus entry into the cells [11]. Moreover, the normal kidney and gastrointestinal tract have higher ACE2 expression than pulmonary tissue [12]. Detection of coronavirus in the kidneys and urine of COVID-19 patients supports the idea that the virus can directly damage the kidney tissues. Preliminary evidence in postmortem examinations of renal tissue from six patients, it was found that severe acute tubular necrosis and lymphocyte infiltration were present. Additionally, SARS-CoV-2 nucleocapsid protein (NP) has been noticed through immunohistochemistry in renal tubules [13]. Furthermore, in one autopsy of a kidney transplant patient who died of COVID-19, viral inclusion structures were detected in the endothelial cells of the kidney nephron. Viral infection could persuade tubular damage through the confession of the MAC complex (the final step of the complement cascade) on tubules and penetration of CD68+ macrophages in the tubule-interstitial cells [13]. Longwinded damage in proximal tubules with the loss of brush border, vacuolar degeneration, and even necrosis was observed in a study of twenty-six autopsies of patients with COVID-19. In the peritubular and glomerular capillary of these autopsies, diffuse erythrocyte aggregation with endothelial damage, and obstruction without fibrin thrombi or distinct fragmentation of erythrocytes or platelets were observed [14]. Bunches of SARS-CoV-2 was detected with electron microscopy in the tubular epithelium and podocytes. However, this finding could be non-specific as the presence of viral proteins may not represent direct damage mediated by the virus and instead indicate clathrin-coated vesicles. Puelles et al. showed the presence of SARS-CoV-2 RNA and proteins in all kidney areas, especially in glomerular cells in autopsies of three of six COVID-19 patients [15].

Kidney biopsies in two Afro-American patients with high-risk APOL1 genotype and SARS-COV-2 infection showed a collapsing focal segmental glomerulosclerosis. However, COVID-19 RNA was not detected in the kidney tissue raining the intriguing hypothesis that cytokine storm increased APOL1 expression resulting in podocyte injury [16, 17]. Other indirect mechanisms that potentially lead to tubular injury are sepsis, cytokine storm syndrome, shock/hemodynamic instability, rhabdomyolysis, and hypoxia of kidney tissue.

Kidney biopsies would be supportive to better understand the histologic pattern of injury (tubular, glomerular, and vascular) and the pathogenesis that could lead to AKI and CKD. Unfortunately, this is very difficult to obtain given the respiratory and hemodynamic instability of AKI patients and the use of anticoagulation which increases the risk of bleeding. In addition, non-essential procedures in infected patients are not being done in most hospitals given the significant risk of exposure to personnel.

Acute kidney injury in COVID-19 patients:

AKI is not uncommon among patients with more severe COVID-19 disease, particularly in those recovering in the intensive care unit, and is being considered as a negative prognostic factor for survival. In a study of 333 patients with COVID-19 disease, those who presented with renal dysfunction and abnormality had higher mortality rates than patients without renal involvement (11.2 and 1.2%, respectively) [18]. Similarly, in an observational study of 287 patients, 55 patients presented with AKI. These patients were significantly older, more likely male, and with other comorbidities, including chronic renal insufficiency, hypertension, and cerebrovascular disease, and tended to have more severe pneumonia. Out of 287 patients, 14.3% presented with AKI at stage-one, while 4.9% of patients presented with stage-2 or 3. Higher mortality rates, especially with severe AKI were observed (mortality: 3.0, 7.3, and 64.3% for non-AKI, AKI stage 1, and AKI stages 2/3, respectively). It was observed that most patients recovered from AKI stage 1. However, patients who progressed to AKI stage 2 or 3 had a very high mortality rate [19]. Furthermore, in another study of 701 patients, 11.9% of those with elevated baseline creatinine developed AKI compared to 4.0% in patients with normal baseline creatinine. In-hospital mortality was significantly higher in patients with proteinuria, hematuria, elevated baseline creatinine and urea, and AKI stage 2–3.

The current treatment and management of COVID-19 associated AKI include supportive management, avoiding the use of nephrotoxic drugs, and early start, when possible, of renal replacement therapy. SARS, MERS, and sepsis have been successfully treated in

the past with continuous renal replacement therapy (CRRT). In these cases, CRRT by hemofiltration and hemodiafiltration can contribute to the improvement of multi-organ failure. Therefore, CRRT may be beneficial in patients with COVID-19 and sepsis syndrome, but careful considerations are required [20]. Filters with membranes made of acrylonitrile and sodium methyl sulfonate plus polyethyleneimine or polymethylmethacrylate could adsorb cytokines, but they should be replaced every 24 hours. Prone positioning should be applied early to reduce mortality in other causes of severe ARDS. Prone positioning could potentially improve the response to positive oxygenation. However, it further complicates the placement of a central venous catheter and could hinder the ability to do CRRT for increased hemodynamic instability. High dose diuretics and K-binding resins have been used to delay the need for dialysis. Careful fluid management to reduce the risk of pulmonary edema in patients with severe ARDS from COVID-19 is the first goal, so in the absence of hypotension and shock, a negative fluid balance of 0.5–1.0 L per day is recommended [21].

Due to the hypercoagulability state associated with COVID-19, systemic anticoagulation with unfractionated heparin or regional citrate anticoagulation is advised [22]. Since these patients experience higher filter clotting, it might be useful to choose pre-dilution replacement fluid administration for hemofiltration, and consider using a heparin bolus together with pre-filter heparin at a higher rate than usual -monitoring PTT before and during hemodialysis to avoid bleeding- in order to maintain higher blood flows.

Asymptomatic renal involvement:

The current studies report that respiratory symptoms of COVID-19 represent the most common manifestation, which is highly suggestive of the droplet and contact transmission. However, the incidence of renal involvement and manifestations differs significantly among different populations. In some patients, only lab abnormalities have been observed in the absence of any renal symptom. Elevated blood urea nitrogen, increased serum creatinine, hematuria, and proteinuria, the increased osmolality of urine, elevated D-dimer, or urine dipstick abnormalities can be manifested in ant patient with COVID-19 without any symptomology.

Li et al. analyzed kidney function in 193 COVID-19 patients and found that 31% of patients had an elevated level of blood urea nitrogen (BUN) and 22% had increased serum creatinine. These authors also found also that in 147 patients, 60% exhibited proteinuria and 48% exhibited hematuria. D-dimer was elevated in 70% of 182 patients and high levels of D-dimer were common in severe and deceased cases. In another retrospective study of 333 patients, about 75% experienced urine dipstick abnormalities [18].

Chronic kidney injury in COVID-19 patients:

The patients with kidney disease who appear most at risk for COVID-19 are those with a kidney transplant, due to immunosuppression, and those who undergo in-center hemodialysis treatments thrice weekly, due to inability to self-isolate. Patients with kidney disease also have other comorbidities, including hypertension, diabetes mellitus, and cardiovascular disease, which are risk factors for poor outcomes in COVID-19. Based on a contrite meta-analysis of early and preliminarily available data by Henry et al., CKD seems to be associated with an enhanced risk of severe COVID-19 infection. Patients with CKD should hence be advised to take extra precautions to minimize the risk exposure to the virus. Physicians should also be engaged in close monitoring of CKD patients with suspected COVID-19, for timely detecting signs of disease progression. Finally, the presence of CKD shall be regarded as an important factor in future risk stratification models for COVID-19. Patients with chronic kidney disease (CKD) are known to have a higher risk of upper respiratory tract infection and pneumonia due to their persistent pro-inflammatory state with functional defects in innate and adaptive immunity. No further study so far has found that chronic kidney disease is statistically correlated with severe COVID-19. However, a significant association of CKD with severe COVID-19 was observed when data of different studies were combined.

COVID-19 in kidney transplant recipients:

There is limited data on kidney transplant recipients in COVID-19 patients. The transplant community is mystified in trying to understand the best therapeutic approach, in the absence of any strong clinical data. The initial presentation in transplant recipients differs from other hosts and many patients did not report contact with infected individuals. The usual symptoms at disease onset have been fever, cough, asthenia, myalgia, and diarrhea. In a study of 36 transplant recipients, however, the temperature was less common than in general COVID-19 patients [23]. In multiple series, transplant patients show numerous radio-opacity and patchy shadows on chest radiographs often at presentation [24]. However, in one case series of fifteen kidney transplant patients, 33% had no acute radiographic findings [25]. Laboratory exams often showed lymphopenia with lower CD3, CD4, and CD8

T cells especially in those patients who had received anti-thymocyte globulin in the weeks before the infection.

Until more data is available, the rules to prevent viral infection in the general population apply to transplant patients (hand hygiene, sanitization, social distancing, and avoiding areas where infected patients could be present). Transplant patients with potential COVID-19 infection should not access the transplant center due to the risk of viral spread. In countries with periodic COVID-19 infection, deceased donor transplants should last. However, donors at risk of infection should not be accepted since viremia was reported in at least 15% in one case series, and, transmission from the donor is possible. Suspension of all transplants that require T or B cell depletion (i.e., hyper-immune patients with the greater title of donor-specific HLA antibodies) should be considered even in countries where the incidence of COVID-19 positive individuals is low.

COVID-19 infection in dialysis patients.

The impact of COVID-19 in patients with dialysis is poorly understood. However, the effects of COVID-19 in dialysis patients have been observed. Wang et al. studied the outbreak of COVID-19 in the hemodialysis (HD) center of Renmin Hospital, Wuhan University. They recognized COVID-19 in 37 individuals among 230 HD subjects (16.09%) and 4 patients among 33 staff (12.12%). They presented with mild signs and symptoms, and no one required hospitalization. During the surveillance, seven HD patients died −six with COVID-19 and one without- but mortality was not directly related to pneumonia. The causes of death, in fact, were heart failure, renal failure, hyperkalemia, and cerebrovascular disease [26]. In a report of five HD patients, diarrhea was the most common symptom with no fever, cough, and dyspnea, thus making diagnosis harder [27]. In one retrospective multicenter study of 7,154 hemodialyzed patients, about 2% were established having COVID-19 infection and only about 50% of them presented fever while about 20% of patients were asymptomatic. The mortality rate among these patients is greater than the general population with COVID-19 peaking at about 31% [28]. The same mortality rate was underlined in another retrospective study of 59 dialyzed patients.

Circulating levels of CD4 and CD8 T cells, NK cells, and pro-inflammatory cytokines are significantly lower in COVID-19 patients with hemodialysis, compared to non-HD COVID-19 individuals [27]. Consistently, HD patients with COVID-19 disease are more likely to present mild symptoms with a lower risk of developing ARDS compared to COVID-19 patients not on HD. However, the reduced inflammatory response in HD patients suggests that they may be at higher risk of being infected with COVID-19. Therefore, additional prevention measures are essential in managing the epidemic in HD centers.

Future Considerations:

In patients with COVID-19 and without pneumonia, complete withdrawal of immunosuppressant - particularly calcineurin inhibitors (CNI) is discouraged by ERA-EDTA. Reduction of the dosage of CNI, and withdrawal of mycophenolate, azathioprine, or mTOR-inhibitors should be individualized considering the severity, nature, and age of the disease. In severe patients, withdrawal of immunosuppression could be done while converting those patients to hydrocortisone. This approach may advance viral clearance but could lead to immune reconstitution and kidney rejection. It should be considered that reducing immunosuppression may exacerbate inflammation, so this approach should be cautioned in the absence of anti-inflammatory agents. In some patients, tacrolimus reduction may be preferred over complete withdrawal because of direct alleged CNI antiviral properties or CNI anti-inflammatory action.

Interim guidance for outpatient HD facilities has been recently released by the Centers for Disease Control and Prevention (CDC). Early recognition and isolation of individuals with a respiratory infection, isolation of infected patients from other hem-dialyzed patients, and the use of personal protective equipment are high priority. During routine clinical visits, face masks, and eye shields are sufficient, while during high-risk procedures, N95 respirators and other respiratory protection devices are required. Chinese Society of Nephrology and the Taiwan Society of Nephrology have recently published detailed guidelines for managing COVID-19 outbreaks in dialysis units.

CONCLUSION:

The pandemic of COVID-19 presents for a nephrologist some exceptional challenges. We perceive that SARS-CoV-2 may have various renal manifestations, and in many cases, the renal signs and symptoms may precede typical respiratory symptoms. Holistic knowledge of the spectrum of the renal and urological consequences of COVID-19 is crucial to get a hold on the spread of the virus. The most vital clinical information which we meet is that diarrhea and abdominal pain may be a presenting feature of COVID-19. Therefore, a high catalog of suspicion for

such patients will be important to prevent or, at least, minimum exposure to health care providers and other patients. With the gradual settling of the outbreak, it can be predicted that several post-infectious renal complications including AKI, CKD will surface up. The proper caution has to be practiced while treating and managing patients with renal co-morbidities, particularly those patients on dialysis and transplant recipients needing immune-modulator therapy since framed guidelines are lacking at this point. The above review of the renal manifestations of COVID-19 will help the nephrologist have a basic preparation, which is of extreme importance to prevent infections.

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