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Research Article **Open Access Journal**

Literature Review on Acute Kidney Injury as a Predictor of Discharge in Patients with Covid-19.

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Abstract:
Coronaviruses (CoV) are single-stranded positive-sense RNA viruses, capable of rapid mutation and recombination, this is an extensive group of viruses that has been known for a long time, however, in recent years the appearance of a new member , known as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which causes coronavirus disease 2019 (COVID-19) has quickly become a pandemic that, to date, has affected millions of people in everyone. In addition, authors report that a large proportion of affected patients have underlying comorbidities that can cause greater severity in the manifestations of this pathology and can trigger complications such as acute kidney injury, which is one of the most important complications of COVID-19 to the point of being able to predispose the medical discharge of the patient as well as its mortality.

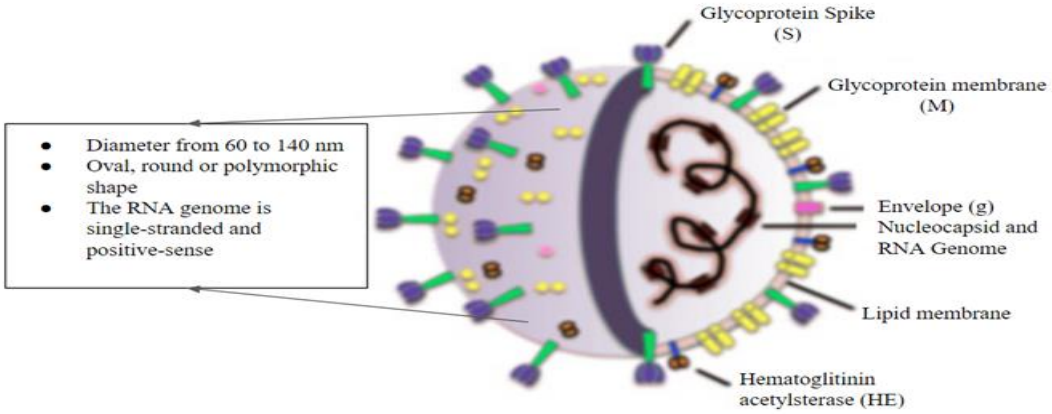
Key Words: SARS-CoV-2, COVID-19, syndrome; Viral pneumonia; complications; Kidney injury, mortality, medical discharge.

Introduction

Coronaviruses are a large family of viruses that have the ability to cause diseases in humans, especially focused on the respiratory system, such as respiratory infections that can range from the common cold to more serious diseases such as Middle East respiratory syndrome or known by its acronym in English as MERS and severe acute respiratory syndrome known by its acronym in English as SARS (1). However, in 2019, severe acute respiratory syndrome coronavirus 2 (SARSCoV-2), the seventh human coronavirus, was discovered in Wuhan, Hubei province, China, during the recent pneumonia epidemic that began in January 2020 (2, 3). Since then, the virus has spread throughout the world and, as of May 20, 2020, has infected more than 4,806,299 people and caused more than 318,599 deaths (4). Among the characteristics of this virus observed in Figure 1, it has been described that morphologically the particle is round or oval and often polymorphic, has a diameter of 60 to 140 nm, has a spike protein found on the surface of the virus and

forms a bar-shaped structure, which is important to recognize because it is the main structure used for typing, it also has the nucleocapsid protein that encapsulates the genome, which is single-stranded RNA of positive polarity and can be used as an antigen diagnosis (5,6).

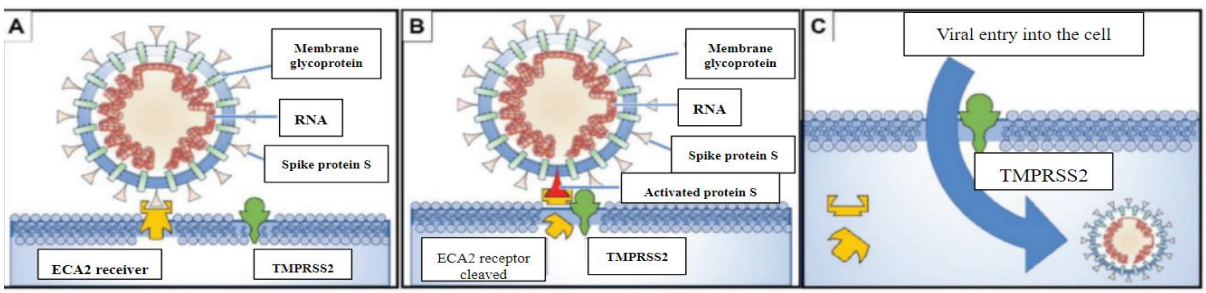
Figure 1. Structural Characteristics of CoVid-19



Source: Santos-Sánchez, Norma Francenia, and Raúl Salas-Coronado. "Origin, structural characteristics, prevention measures, diagnosis and potential drugs to prevent and control COVID-19." Medwave 20.8 (2020).

The recognition of the structure of this particle is relevant to understand its mechanism of action, since, once the virus enters the organism, the spike glycoprotein (S) as seen in Figure 2, uses its two functional subunits to enter with great ease: the S1 subunit and the S2 subunit, where the first allows union with the host cell receptor and the second mediates the incorporation of the virus into the cell membranes (7). In greater depth, it is relevant to know that the spike glycoprotein (S) of the microorganism in question binds to the angiotensin-converting enzyme II (ACE2) receptor, thus forming a complex that, in a certain way, is proteolytically processed by the transmembrane protease type 2 or known as TMPRSS2, for its acronym in English, which leads to the division of ACE2 and the functioning of the spike glycoprotein (S), thus initiating the process of union and fusion of the virus with the cell membrane, ending with the entry of the virus into the host cell (8). However, after the entry of the particle into the cell, the replication process begins as shown in Figure 3, where in the first instance, the particle fuses with the cytoplasmic vacuole, then the viral RNA is directly translated into two polypeptides that are divided to form an RNA polymerase and the latter is responsible for the transcription of a complete positive chain (genome) of the virion and numerous NS and proteases are also synthesized (9). It is important to highlight that 2/3 of the genetic material translates into 16 non-structural proteins and 1/3 corresponds to 4 structural proteins, among these is the S protein, which has two important parts, the first is an S1 subunit, capable of to participate in such a way that the affinity of the virus intervenes for the angiotensin converting enzyme 2 or (ACE2); and the S2 subunit important for the process of cell membrane fusion to be easier; In addition, there is an M protein, which mediates the release of RNA to the cell of the infected individual; and finally, the N and E proteins, which are proteins that are part of the structure and may be responsible for interacting with the innate immunity of the host (10).

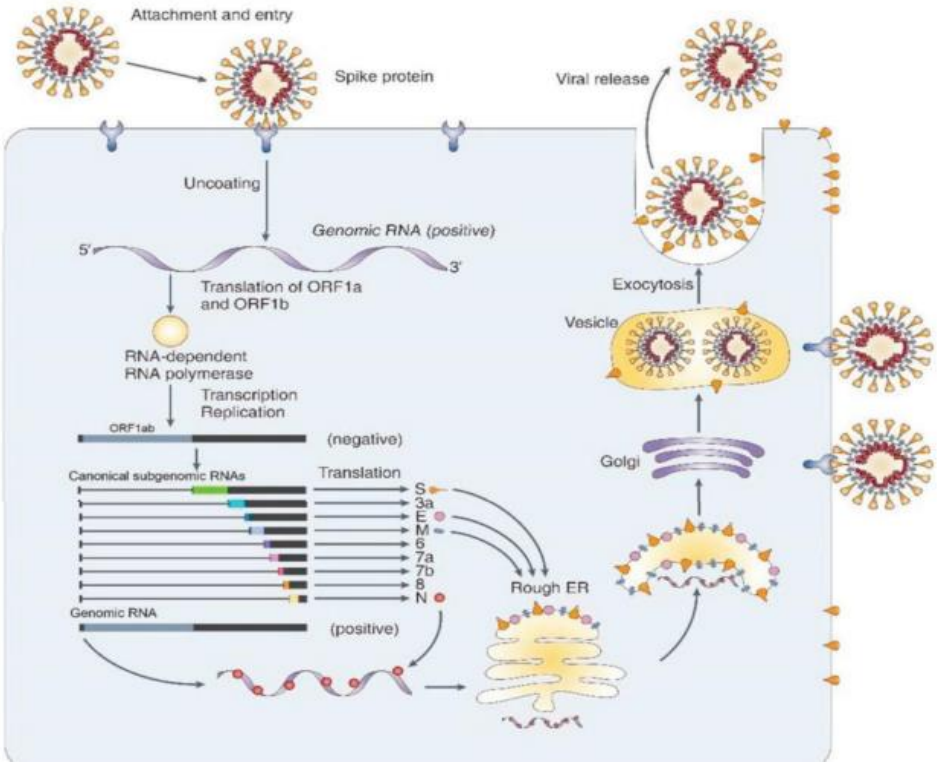
Figure 2. Entry of SARS-CoV-2 into the cell



Modified from: Rabi A. SARS-CoV-2 and Coronavirus Disease 2019: What We Know So Far. Pathogens. 2020; 9(3): 231.

When the organism replicates and spreads throughout the host, flu-like symptoms appear, including manifestations such as fever, cough, dyspnea, myalgia, and fatigue. In addition, sudden loss of smell, odynophagia, nasal congestion, mild headache, and loss of taste (without mucus being the cause) have been observed. In more severe cases, it is characterized by producing pneumonia, acute respiratory distress syndrome, sepsis, and septic shock that leads to death in around 3% of those infected, in addition, the mortality rate is 4.48% and continues to rise. (eleven). Generally, in individuals where more severe cases occur, it is related to other previously present diseases that in a certain way complicate the course of the pathology, for that reason, patients with previously associated diseases, elderly individuals, immunocompromised or immunosuppressed, have higher probability of death, therefore, at present, diseases such as those shown in Table 1 are considered risk factors (12,13,14). The main risk factor for mortality is generally age over 80 years. In a specific population studied by McGoogan JM, the percentage of deaths reported in patients older than 80 years was 14.8%; Other risk groups with high mortality are included in patients with previous cardiovascular disease, diabetes mellitus and dyslipidemias that are part of the metabolic syndrome, chronic lung disease, arterial hypertension and even a history of cancer (15,16,17).

Figure 3. Replication process of the SARS-CoV-2 particle



Source: Solis, Jaime Galo Benites. Structure and composition of SARS-COV-2.

Now, when an individual suffers from covid-19, sequelae usually appear that can affect any device, especially at the cardiovascular level, where it causes arrhythmias, can predispose to heart attacks and even triggers hypertensive periods, at the level of the upper airways it leaves a residual degree as a sequel of anosmia and ageusia in addition to cough, in the lower airways it leaves dyspnea on exertion, cough, mucous secretion, difficulty for deep inspiration and chest pain, at the muscular level it promotes generalized weakness and erratic pain, in the neurocognitive part patients usually present lack of attention, memory loss, and insomnia, in the psychological part depression and anxiety appear, at the gastrointestinal level choking is manifested, change in the rhythm of bowel movements, a feeling of fullness that leads to weight loss and at the renal level authors describe renal lesions caused by this microorganism that even

predispose to discharge of the patient (18,20). In fact, the report of AKI in patients with COVID-19 has varied over time, having a variation in prevalence from 0.5% to 28% and increasing(17).

Table 1. Some risk factors associated with morbidity in patients with covid-19.

Neurological	Cerebrovascular disease, dementia, Alzheimer's
Cardiovascular	Hypertension, Acute myocardial infarction, Myocarditis, Arrhythmias
endocrinological	Obesity, Diabetes mellitus, dyslipidemia, metabolic syndrome
Pulmonary	Respiratory failure, Asthma, COPD
Renal	Chronic renal insufficiency
musculoskeletal	Osteoporosis

Acute kidney injury (AKI) is defined as the sudden decrease in glomerular filtration that causes the accumulation of metabolic waste products, more specifically urea and creatinine, with the potential risk of hydroelectrolytic disorders and acid-base disorders (19). This condition has taken on great relevance during the SARS-CoV-2 pandemic, in fact, it has been described as a frequent complication in patients with COVID-19, in addition its appearance is a potential indicator of multiorgan dysfunction and severity of the disease that can Compromise the life of the patient, so it minimizes the patient's departure from the hospital entity and, on the contrary, increases their days of stay, for this reason, it is considered a predictor of discharge in the patient with covid-19. In some studies, imaging and autopsy results reflect a variety of mechanisms involved in kidney injury in patients with sars-cov-2, including cytokine injury, type 1 cardiorenal syndrome, alveolar damage causing hypoxia renal failure, rhabdomyolysis that causes renal toxicity, endothelial damage, fluid loss in the third space, and hypotension that give rise to renal hypoperfusion, among others (20,22). However, these causes of AKI associated with COVID-19 are still under study; however, the authors have already given their explanations and have considered some applications in this regard, which are presented in Table 2 . The first pathway is associated with cytokine storm damage or cytokine release syndrome. It is mainly mediated by proinflammatory interleukin-6 (IL-6) and interleukin-8 (IL-8), although the participation of the vascular endothelial growth factor, monocyte chemoattractant protein-1, and decreased expression of E-cadherin in endothelial cells, which allows for increased vascular permeability that promotes hypotension and pulmonary dysfunction in respiratory distress syndrome acute respiratory. The second pathway is mediated by alveolar damage, and its relationship with myocardial and tubular tissue, since myocarditis due to cytokine release syndrome and acute viral myocarditis contribute significantly to renal hypoperfusion, renal vein congestion and hypotension, causing a marked decrease in the glomerular filtration rate. Similarly, hyperinflation and overproduction of cytokines is involved in bidirectional lung-kidney damage and the injured renal tubular epithelium causes an increase in the concentration of Interleukin-6, which is a cytokine that generates greater alveolar-capillary permeability and thereby promoting the possibility of pulmonary hemorrhage. Similarly, another proposed pathway suggests that the appearance of AKI in patients with positive PCR for covid-19 is mediated by the systemic effect of several mechanisms that include endotoxins, positive fluid balance, hypotension, endothelial cell injury, among others. In addition to these, there are other pathways mentioned in the literature (23,24).

Table 2. Proposed pathways by which covid-19 can cause acute kidney injury.

First way	**Second way**	**Third way**

Cytokine storm damage or cytokine release syndrome mediated mainly by proinflammatory Interleukin-6 (IL-6) and others, which can be released after covid-19 infection.	Mediated by the close relationship between alveolar, myocardial and tubular damage due to the release of cytokines causing cardiorenal syndrome type 1	Systemic effect of various factors such as positive fluid balance, endothelial injury, hypotension and third space fluid loss, rhabdomyolysis and endotoxins
Fourth way	**Fifth way**	**Sixth way**
Endothelial injury due to deposition of viral particles in the lung and kidney tissue, which may indicate that viremia is a possible cause of renal endothelial damage and a probable determining factor of AKI	SARS-CoV-2 can directly infect the proximal renal tubular epithelium and podocytes through its dependence on angiotensin-converting enzyme 2 (ACE-2), taking into account that its expression is more common in renal tissue.	COVID-19-associated macrophage activation, increased ferritin levels, cytokine storm, and release of pathogen-associated molecular patterns and damage-related molecular proteins can release tissue factor and activate coagulation factors that create a predisposition to hypercoagulability

Source modified from: Ronco C, Reis T. Kidney involvement in COVID-19 and rationale for extracorporeal therapies. Nat Rev Nephrol. 2020;16(6):308-10-http://dx.doi.org/10.1038/s1581-020-028-7

Materials and Methods

A detailed bibliographic search of the most relevant published information is carried out in the databases pubmed, scielo, elsevier, medline, national and international libraries specialized in the topics covered in this review article. The following descriptors were used: SARS-CoV-2, COVID-19, syndrome; Viral pneumonia; complications ; Kidney injury, mortality, medical discharge. The data obtained range between 5 and 40 records after the use of the different keywords. The search for articles was conducted in Spanish and English, limited by year of publication, and studies published from 2018 to the present were used.

Results

According to the results of the articles found, which meet the inclusion criteria in this study, COVID-19 manifests itself mainly as acute respiratory disease with lower respiratory compromise, but it can affect different organs without limitation, such as the kidney, which leads to worse prognosis. The prevalence of extrapulmonary manifestations has been estimated at 92% of patients, where 58% have renal involvement (25). Chu et al. found in their study that 6.7% of patients (36) developed acute kidney injury out of 536 patients who were included, which had a mean duration of 20 days (range 5-48 days) after the onset of kidney disease. covid 19, despite a normal serum creatinine level (SCr) at the beginning of the clinical picture. In addition, they concluded that patients with viral infection and AKI had a higher mortality rate than those without renal complications, according to the results of 33 patients (91.7% vs. 8.8%) ($p < 0.0001$) (26). The causes of acute kidney injury associated with COVID-19 are under study; however, and despite the recent appearance of the disease, there are already some postulations about it. Within the machinery used by the virus to cause kidney damage, the following aspects have been described: damage by cytokines, reciprocal interaction with other organs, systemic effects related to mechanical ventilation, and acute respiratory distress syndrome (ARDS) (25). In a study of 22 cases, it is evident once again how covid can lead to the development of kidney injury, whose patients were positive for the SARS-CoV-2 test, of them 12 (55%) acute kidney injury, within this complicated group four (18%) AKI I, five (23%) AKI II and three (14%) AKI III.

Of which 33% of these require hemodialysis and with a mortality of 15.1% according to SAPS-3 (27). Therefore, the tubular injury will be the result of COVID-19 infection at the kidney level, as a result of the fact that human ACE2 is specifically expressed in tubules instead of glomeruli(28). Zhenli et al. They present renal alterations in 59 patients infected by COVID-19, 63% of them presented proteinuria in very early stages. In addition, 19% and 27% of the patients had high values of plasma creatinine and urea nitrogen, whose damage in patients with inflammation and edema 100% (27/27) was reflected in the computed tomography that showed radiographic abnormalities of the kidneys (29). It is important to highlight that AKI is multifactorial in patients with severe symptoms of SARS-CoV-216 infection. In addition, it is important to highlight some of the comorbidities that influence the risk of developing AKI in the infected population, among them are: age, hypertension, diabetes, coronary heart disease, chronic kidney disease, hypovolemia and manifestations of septic shock, and the use of nephrotoxic drugs Similarly, some treatment alternatives such as mechanical ventilation for patients with covid-19 are largely associated with the development of acute kidney injury, which in turn increases the risk of mortality. (30) Li et al. reported that there is an increase in mortality from covid-19 of 5.3 times in those people who develop this injury. Proteinuria and increased serum creatinine levels are sensitive biomarkers among patients with AKI and COVID-19 that have a fatal outcome (31). The study by Echavarría et al. concluded from their observational retrospective cohort study that included 266 patients with COVID-19, that kidney injury influences the prognosis of patients with COVID-19; patients found to be outside the acute kidney injury category with respect to serum creatinine are more likely to be discharged for clinical improvement (32). Tarragon et al. present the clinical evolution of patients hospitalized for COVID-19 with acute renal failure (AKI) who required care by Nephrology, based on clinical and analytical studies they concluded that hypovolemia and dehydration are a frequent cause of AKI in patients with COVID-19. 19, where patients who developed AKI during admission had higher values of C-reactive protein, LDH and D-dimer, more severe lung involvement, and more need for ICU admission (33). Likewise, in a cohort of 333 patients admitted with COVID-19, proteinuria and hematuria were demonstrated in 65.5% and 41.7% of patients, respectively, and were associated with mortality in unit patients. intensive care unit (ICU). Kidney involvement was also evident on CT; mean CT value and CT texture analysis parameters were significantly lower in COVID-19 patients than in healthy controls who did not have kidney disease (34). Even the pediatric population can also be the target of this complication, although with low frequency, so it is suggested to use the definition of AKI as the increase in serum creatinine above 0.3 mg/dl in 48 hours despite adequate resuscitation or more than 50% increase from baseline in the past 7 days or urine volume less than 0.5 mL/kg/hour for 8 hours, as validated by the KDIGO-AKIN criteria for this population. In addition, monitoring of serum creatinine, hematuria and proteinuria (microalbuminuria), electrolytes (sodium, potassium, phosphorus), uric acid, urea and bicarbonate, at least once every 48 hours and ideally every day as a measure of prevention of hypovolemia (35). For this reason, different authors determine that pediatric patients on dialysis should be classified as high risk for contracting the disease, even in hospital, since different mechanisms are combined such as immunocompromised, uremia, secondary malnutrition and immunosuppressive therapy; frequent contact with health workers and other patients in renal units; the presence of relatives and patients during dialysis, and the lack of adherence to prevention measures (36).

Discussion

Covid 19 is a respiratory infectious disease with a variety of complications at the cardiac, hematological, digestive, neurological and renal levels thanks to the distribution of its receptors. Patients with acute renal failure have a worse prognosis profile due to multisystemic damage. A cohort study of 701 patients hospitalized in Wuhan demonstrated that AKI greater than stage 2 was independently associated with death in hospital. The risk of mortality was more than 4 times higher among patients with stage 3 AKI (hazard ratio 9.81, 95% CI: 5.46-17.65) (37). Which is similar to our results and what was reported by Taxbro et al. of a 38-year-old man who was diagnosed with SARS-CoV-2, on day 4 in the ICU developed severe rhabdomyolysis and kidney injury, urine turned tea-colored, myoglobin

levels gradually increased (from normal levels to admission to a peak level of >21,000 µg/L) and renal function deteriorated (lowest recorded relative glomerular filtration rate of 18 mL/min/1.73 m 2) (38). Similarly, other authors confirm that acute kidney injury in Covid-19 is an indicator of adverse prognosis. Its origin (acute kidney injury in Covid-19) is multifactorial, including acute tubular damage and infiltration by lymphocytes and macrophages, having identified the virus within the glomerular endothelium and in tubular cells. Being evident in the echinocytes that are indicative of acute kidney injury (39). As presented in a 40-year-old patient, who underwent tests on admission that revealed acute kidney injury (creatinine 3.1 mg/dl; pH 7.37; bicarbonate 21 mmol/l; sodium 128 mmol/l; potassium 3.84 mmol/l; urine status with 150 mg/dl protein and 250 erythrocytes/µl). D-dimer (4950 µgFEU/l; reference range <500 µgFEU/l) and lactate dehydrogenase (424 U/l) were also elevated. Sonographically, both kidneys appeared enlarged with a strongly echogenic cortex, consistent with acute nephritis, which confirms the severe complications of the infection (40). Likewise, renal histopathology examined in a series of autopsies of 26 patients who died with COVID-19 was shown: all patients had evidence of acute tubular injury (of varying severity); Clusters of erythrocytes and pigmented casts were also present (41). However, NaKR et al. conclude in their study that the incidence of AKI was not high in patients with COVID-19, since their results were: AKI occurred in 3 (4.5%) of the 66 patients, and 1 patient with stage 3 AKI underwent hemodialysis. At follow-up, all 3 patients recovered normal renal function. Compared with patients with mild COVID-19, AKI (n = 3) occurred in patients with severe COVID-19 (42). Furthermore, it is noteworthy in an ambidirectional cohort study of patients with confirmed COVID-19, that 6 months after acute infection, COVID-19 survivors mainly presented fatigue or muscle weakness, sleep difficulties, and anxiety or depression. Patients who were more severely ill during their hospital stay had more severely impaired pulmonary diffusion capabilities and abnormal chest imaging manifestations, and are the primary target population for long-term recovery intervention (43).

Conclusion

Infection by the new coronavirus has become a global human threat, not only because of the severe respiratory disease, but also because of the associated multisystemic complications. The kidney is one of the affected white organs and based on the measurement of serum elements its function can be measured, therefore, acute kidney injury has been evidenced in patients with a positive PCR test for covid-19, generating a clinical picture much more serious and less likely to be discharged from the hospital, than those who have creatinine levels outside the range of injury.

References

1. Carr D. Sharing research data and findings relevant to the novel coronavirus (COVID-19) outbreak [Internet]. London: Wellcome Trust 2020 [citado 4/01/2022]. Disponible en: https://wellcome.ac.uk/press-release/sharingresearch-data-and-findings-relevant-novelcoronavirus-covid-19-outbreak
2. Zhou P, Yang XL, Wang XG, et al. A pneumonia outbreak associated with a new coronavirus of probable bat origin. Nature. 2020;579(7798):270–273.
3. Wu F, Zhao S, Yu B, et al. A new coronavirus associated with human respiratory disease in China. Nature. 2020; 579(7798):265–269.
4. World Health Organization Coronavirus Disease 2019 (COVID-19) Situation Report-97. Available from: https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200426-sitrep-97-covid-19.pdf
5. Santos-Sánchez, Norma Francenia, and Raúl Salas-Coronado. Origen, características estructurales, medidas de prevención, diagnóstico y fármacos potenciales para prevenir y controlar COVID-19. *Medwave* 20.8 (2020).
6. Gutierrez Choque, Bismar Jorge, and Carla Jimena Aruquipa Quispe. Covid-19: aspectos virológicos y patogénesis. *Revista Científica Ciencia Médica* 23.1, 2020: 77-86.
7. Wan Y, Shang J, Graham R, Baric RS, Li F. Receptor Recognition by the Novel Coronavirus from Wuhan: an Analysis Based on Decade-Long Structural Studies of SARS Coronavirus. J Virol. 2020; 94(7): 1-9.
8. Rabi A. SARS-CoV-2 and Coronavirus Disease 2019: What We Know So Far. Pathogens. 2020; 9(3): 231
9. Iwata-Yoshikawa N, Okamura T, Shimizu Y, Hasegawa H, Takeda M, Nagata N. TMPRSS2

Contributes to Virus Spread and Immunopathology in the Airways of Murine Models after Coronavirus Infection. J Virol. 2019; 93(6).
10. OMS. Noticias ONU. Retos de salud urgentes para la próxima década [Internet]. Ginebra: OMS; 13 enero 2020 [Citado 7/01/2022]. Disponible en: https://news.un.org/es/story/2020/01/1467872
11. Salazar, Martín, et al. COVID-19, hipertensión y enfermedad cardiovascular. *Hipertensión y riesgo vascular*, 2020, vol. 37, no 4, p. 176-180.
12. Tenorio-Mucha, Janeth, and Yamilée Hurtado-Roca. Revisión sobre obesidad como factor de riesgo para mortalidad por COVID-19. *Acta Médica Peruana*, 2020, vol. 37, no 3, p. 324-329.
13. C. Wu, X. Chen, Y. Cai, *et al*. Risk factors associated with acute respiratory distress syndrome and death in patients with coronavirus disease 2019 pneumonia in Wuhan.China. JAMA Intern Med., (2020), pp. 1-10 http://dx.doi.org/10.1001/jamainternmed.2020.099
14. Tort, Arturo Rodríguez, et al. La deficiencia de vitamina D es un factor de riesgo de mortalidad en pacientes con COVID-19. *Revista de Sanidad Militar*, 2020, vol. 74, no 1, p. 106-113.
15. Wu Z, McGoogan JM. Characteristics of and Important Lessons from the Coronavirus Disease 2019 (COVID19) Outbreak in China: Summary of a Report of 72314 Cases from the Chinese Center for Disease Control and Prevention. JAMA - J Am Med Assoc. 2020;2019:25---8, http://dx.doi.org/10.1001 /jama.2020.2648.
16. Guo Y-R, Cao Q-D, Hong Z-S, et al. The origin, transmission and clinical therapies on coronavirus disease 2019 (COVID-19) outbreak - an update on the status. Mil Med Res. 2020;7:1---10, http://dx.doi.org/10.1186/s40779-020-00240-0.
17. Minchola, Kevin, et al. Síndrome metabólico como determinante en la complicación de pacientes COVID-19.*Revista Médica de Trujillo* 16.3 , 2021.
18. molina-molina, María. Secuelas y consecuencias de la COVID-19. *Medicina respiratoria*, 2020, vol. 13, no 2, p. 71-77.
19. Sharfuddin AA, Weisbord SD, Palevsky PM, Molitoris BA. Lesión renal aguda. In: Skorecki K, Chertow GM, Marsden PA, Maarten W, Yu ASL, Wasser WG, editors. Brenner y Rector El riñón. 10 ed. España: Elsevier; 2018. p. 958-1011.
20. Pei G, Zhang Z, Peng J, Liu L, Zhang C, Yu C, et al. Renal Involvement and Early Prognosis in Patients with COVID-19 Pneumonia. J Am Soc Nephrol. 2020;31(6):1157-65. http://dx.doi.org/10.1681/ASN.2020030276.
21. Chen N., Zhou M., Dong X., Qu J., Gong F., Han Y. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. *Lancet.* 2020;395(10223) :507–513.
22. Naicker S, Yang CW, Hwang SJ, Liu BC, Chen JH, Jha V. The Novel Coronavirus 2019 epidemic and kidneys. Kidney Int. 2020;97(5):82-8. https://doi.org/10.1016/j.kint.2020.03.001.
23. Ronco C, Reis T. Kidney involvement in COVID-19 and rationale for extracorporeal therapies. Nat Rev Nephrol. 2020;16(6):308-10-http://dx.doi.org/10.1038/s1581-020-028-7
24. Moore JB, June CH. Cytokine release syndrome in severe COVID-19. Science. 2020;368(690):73-4. http://dx.doi.org/10.1126/science.abb8925.
25. Vargas, Juan Guillermo, et al. "Lesión renal aguda en COVID-19: puesta al día y revisión de la literatura." Acta Colombiana de Cuidado Intensivo (2020).
26. Chu K.H., Tsang W.K., Tang C.S., Lam M.F., Lai F.M., To K.F. Acute renal impairment in coronavirus-associated severe acute respiratory syndrome. Kidney Int. 2005;67: 698–705.
27. Lesión renal aguda en pacientes con COVID-19 en la Unidad de Terapia Intensiva del Hospital Ángeles Pedregal
28. de Francisco ALM, Pérez Canga JL. Nefrología al día. 9 de Junio de 2020. Coronavirus y Riñón. ACTUALIZACIÓN COMPLETA . Disponible en: https://www.nefrologiaaldia.org/305
29. Zhen Li,, Ming Wu,+, Jie Guo et al Caution on Kidney Dysfunctions of 2019-nCoV Patients medRxiv preprint doi: https://doi.org/10.1101/2020.02.08.20021212. [Pubmed]

30. González Camilo, Yama Erika, Yomayusa Nancy, Vargas Juan, Rico Jorge, Ariza Amaury et al . Consenso colombiano de expertos sobre recomendaciones informadas en la evidencia para la prevención, el diagnóstico y el manejo de la lesión renal aguda por SARS-CoV-2/COVID-19. Rev. colom. nefrol. [Internet]. 2020 Dec [cited 2022 Jan 10] ; 7(Suppl 2): 89-117. Available from: http://www.scielo.org.co/scielo.php?script=sci_arttext&pid=S2500-5006 2020000300089&lng=en. Epub May 10, 2021. https://doi.org/10.22265/acnef.7.supl. 2. 73.
31. Li Z, Wu M, Yao J, Guo J, Liao X, Song S, et al. Caution on Kidney Dysfunctions of COVID-19 Patients. medRxiv.2020. https://doi.org/10.1101/2020.02.08.20021212.[Lin ks]
32. Pérez-Echavarría, Ana Irene, et al. "Lesión renal aguda como predictor de alta hospitalaria en pacientes con COVID-19." Medicina Interna de México 37.5 (2021): 721-727.
33. Tarragón, Blanca, et al. "Fracaso renal agudo en pacientes hospitalizados por COVID-19." nefrología 41.1 (2021): 34-40.
34. Ertuğlu LA, Kanbay A, Afşar B, Elsürer Afşar R, Kanbay M. COVID-19 and acute kidney injury. Tuberk Toraks. 2020 Dec;68(4):407-418. English. doi: 10.5578/tt.70010. PMID: 33448738.
35. Baquero Rodriguez, Richard, et al. "Consenso colombiano de expertos sobre recomendaciones informadas en la evidencia para la prevención, diagnóstico y manejo de la infección por SARS-CoV-2/COVID-19 en paciente pediátrico con enfermedad renal crónica o lesión renal aguda asociada a COVID-19." Revista Colombiana de Nefrología 7 (2020): 50-69.
36. Staico MF, Zaffanello M, Di Pietro G, Fanos V, Marcialis MA. The kidney in COVID-19: protagonist or figurant? Panminerva Med [Internet]. 2020. Available from: http://www.ncbi.nlm.nih.gov/pubmed/32432445 [Links]
37. Cheng Y, Luo R, Wang K, Zhang M, Wang Z, Dong L, et al. La enfermedad renal está asociada con la muerte hospitalaria de pacientes con COVID-19. Riñón Int 2020; 97: 829-38
38. Taxbro K, Kahlow H, Wulcan H, Fornarve A. Rhabdomyolysis and acute kidney injury in severe COVID-19 infection. BMJ Case Rep. 2020 Sep 2;13(9):e237616. doi: 10.1136/bcr-2020-237616. PMID: 32878841; PMCID: PMC7470512.
39. Crossette-Thambiah C, Hazarika B, Bain BJ. Covid-19 and acute kidney injury. Am J Hematol. 2021 Jun 1;96(6):747-748. doi: 10.1002/ajh.26089. Epub 2021 Feb 2. PMID: 33421165; PMCID: PMC8013605.
40. Junginger A, Brucke M, Reiser B, Stahn O, Braun G. Hämoptysen und akutes Nierenversagen bei COVID-19 [Hemoptysis and acute kidney injury in COVID-19]. Med Klin Intensivmed Notfmed. 2020 Dec;115(Suppl 3):146-151. German. doi: 10.1007/s00063-020-00762-4. Epub 2020 Dec 14. PMID: 33315119; PMCID: PMC7734111.
41. Alhenc-Gelas F, Drueke TB. Blockade of SARS-CoV-2 infection by recombinant soluble ACE2. Kidney Int. 2020 Jun;97((6)):1091–3
42. Na KR, Kim HR, Ham Y, Choi DE, Lee KW, Moon JY, Kim YS, Cheon S, Sohn KM, Kim J, Kim S, Jeong H, Jeon JW. Acute Kidney Injury and Kidney Damage in COVID-19 Patients. J Korean Med Sci. 2020 Jul 20;35(28):e257. doi: 10.3346/jkms.20 20.35. e257. PMID:32686373;PMCID:PMC7371456.
43. Huang C, Huang L, Wang Y, Li X, Ren L, Gu X, Kang L, Guo L, Liu M, Zhou X, Luo J, Huang Z, Tu S, Zhao Y, Chen L, Xu D, Li Y, Li C, Peng L, Li Y, Xie W, Cui D, Shang L, Fan G, Xu J, Wang G, Wang Y, Zhong J, Wang C, Wang J, Zhang D, Cao B. 6-month consequences of COVID-19 in patients discharged from hospital: a cohort study. Lancet. 2021 Jan 16;397(10270):220-232. doi: 10.1016/S0140-6736(20)32656-8. Epub 2021 Jan 8. PMID: 33428867; PMCID: PMC7833295.

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