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SARS-CoV-2, Treatment Options and Relationship with Kidney Disease

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The current global pandemic caused by the SARS-CoV-2 coronavirus with figures established by the Center for Systems Sciences and Engineering of the Johns Hopkins Hospital [1] in the United States until May 7, 2020, of 3,845,718 and 269,567 associated deaths that it emerged in the city of Wuhan in Hubei province [2].

The way this coronavirus binds is through the angiotensin-converting enzyme 2 (ACE2) receptor, a functional receptor for the entry of SARS-CoV-2 through its S protein; These receptors are in the lung, heart, kidney, and intestine, with a strong association with cardiovascular diseases [3]. The replication of the coronavirus already inside the cell is observed in figure 1; The mechanisms of action of multiple drugs today in therapeutic tests act at various levels to control replication.

Transmission is from person to person with direct contact, through droplets spread by coughs or sneezes. The clinical behavior after an average of 5 days of incubation will vary with age and immune system mainly, with a shorter period in those over 70 years of age with symptoms of cough, fever, and headache, with dyspnea as symptom of severity; There are some other symptoms such as arthralgia, myalgia, odynophagia, rhinorrhea, conjunctivitis, and diarrhea. The main pathogenesis is lung affection with severe pneumonia with a production of cytokines and chemocytes that lead to multi-organ failure. The laboratory findings observed are leukopenia, elevation of c-reactive protein, erythrocyte sedimentation rate, D-dimer and the cytokines involved are interleukin (IL) 1-β, IL2, IL6, IL7, IL8, IL9, IL10, and tumor necrosis factor alpha (TNF-α) [5]. Figure 2 exemplifies the clinical phases, associated with the signs and suggested management [6].

The treatments with the greatest scientific evidence without their recommendation being conclusive are the following [7]:

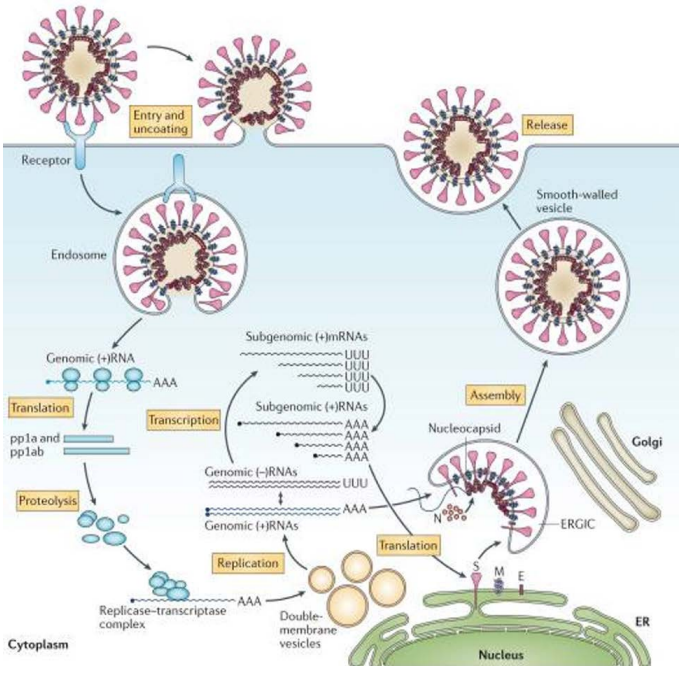

Figure 1: Coronavirus replication. After entry of the virus into the host cell, this viral RNA is not covered within the cytoplasm and the fragments are translated to produce pp1a and pp1ab (translation) and subdivided into 16 non-structural proteins that become the replicase-transcriptase complex (proteolysis). A 3rd and 4th phenomenon (replication and transcription) allows the production of complete copies, which are used as templates for full-length RNA genomes. Subsets of sub-genomic RNAs (discontinuous transcription) are those that encode all structural proteins. All the structured proteins formed are assembled to form the nucleocapsid and the viral envelope in the intermediate compartment between the endoplasmic reticulum and the Golgi apparatus to continue the last step of the release of the nascent virion from the infected cell. Adapted from De Wit et al. [4].

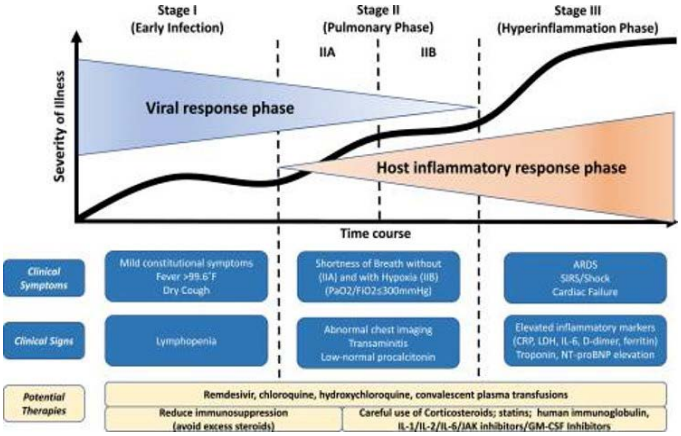

Figure 2: Progressive phases of SARS-CoV-2 disease. The signs associated with each one is exemplified in 3 phases, in addition to the symptoms referred to; In the last boxes, possible treatment alternatives are presented. ARDS: acute respiratory distress syndrome; SIRS: systemic inflammatory response syndrome; LDH: lactic dehydrogenase; CRP: c-reactive protein; IL: interleukin; NT-proBNP: N-terminal pro-brain natriuretic peptide. Adapted from Hasan et al. [6].

(a) Antiviral therapy:

- Remdesivir: Inhibitor of RNA-dependent RNA polymerase replication.
- Lopinavir/Ritonavir: Coronavirus protease inhibitor.
- Interferon beta: Synergy with Lopinavir/Ritonavir, used in severe pneumonia.

(b) Antimalarials: They manage to suppress the production and release of TNF and IL6. The potential risk of cardiac arrhythmias must be taken into account.

- Chloroquine: Inhibits pH-dependent (alkalinizing) steps in replication.
- Hydroxychloroquine: Antimalarial agent with greater potency than chloroquine.

(c) Steroids: The use of steroids despite multiple studies is not recommended due to the inhibition of viral clearance and the lengthening of the duration of viremia.

(d) Convalescent plasma or immunoglobulins: Consists of taking the plasma of recovered patients to treat people who are seriously ill with the same condition.

(e) IL6 inhibitor: In this case Tocilizumab is suggested for patients with severe pneumonia, reducing oxygen consumption.

Kidney disease in the context of SARS-CoV-2 infection

Renal involvement in the patient with COVID-19 infection is approximately between 5 to 15% with the development of acute kidney injury. The mechanism of kidney damage is sepsis, cytokine storm or direct cellular damage by the virus [7]. Patients with chronic kidney disease are immunocompromised patients. Acute kidney injury in patients infected with COVID-19 is induced by sepsis, so the need to maximize the effect of renal replacement therapy in the face of the exaggerated inflammatory response, high-volume hemofiltration therapies, together with membranes, have been proposed. coupled plasma filtration absorption; However, even with little evidence to support it [8].

Uremic patients on dialysis combine intrinsic fragility and a burden of comorbidities. Chronic kidney disease constitutes a relevant comorbidity and dialysis centers represent a risk as a potential vector in the spread of this pandemic. Hemodialysis patients have greater exposure than the general population because their treatment generally requires three sessions per week, making it difficult to maintain social distancing measures [9].

Kidney transplant recipients maintain a state of permanent immunosuppression and need to periodically go to hospitals for check-ups. There is not enough information to establish at the moment the prognostic factors and the course that COVID-19 infection will take in transplant recipients. Minimizing immunosuppression seems a reasonable measure in recipients diagnosed with COVID-19, in particular, cases complicated with pneumonia. The administration of protease inhibitors requires the temporary interruption of calcineurin and mTOR inhibitors due to the risk of significant interactions. In very severe cases of COVID-19, complete withdrawal of immunosuppression should be considered after evaluating the risk of rejection and graft loss [10].

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