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COVID-19 and Cardiological Aspects: Deciphering the Cardiology Riddle

Tania T. Muñoz H

Department of Cardiology, Military Hospital "Dr. Carlos Arvelo" Caracas Venezuela

Correspondence should be addressed to Tania T. Munoz H, electroshock_2010@hotmail.com

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Letter to Editor

SARS-CoV has been analyzed by many experts in various areas of medicine, but all the path physiological mechanisms involved aren´t yet clear because they are multilateral and different from other pathogens that at the time led to pandemics. SARS CoV-2 is internalized in different cell groups after binding to the homologous angiotensin 2-converting enzyme (ACE2), the same that leads the cardiovascular pathway protective catalyzing the reaction: angiotensin 1 and angiotensin 2 in angiotensin 1-9 and angiotensin 1-7, respectively. It participates in a complex and coupled system together with the enzyme ACE producing angiotensin 2, oligopeptide responsible for the deleterious cardiovascular effects such as vasoconstriction, inflammation, proliferation (fibrosis, hypertrophy and myocardial remodeling). The entry of the virus into the cells would produce the reduction of ACE2 by internalizing it, thus neutralizing the protective mechanism that they mediate. Numerous medical publications have demonstrated the increased expression of these receptors in morbid conditions such as systemic arterial hypertension (SAH), diabetes mellitus (DM), heart failure, chronic kidney disease (CKD), senile age, explaining the increased viral load present in this group of patients. The question arises as to whether the opposite case: patients with normal blood pressure levels or below the estimated average range by age, will then have lower receptor expression and lower viral load. Attention is drawn to cases of patients with severe pneumonia hospitalized in an ICU, presenting with significant elevation of the sepsis marker (procalcitonin) and inflammatory reactants of acute phase, cytokine storm, peripheral vascular hypoperfusion, thrombotic immune response, macrophagocytic lymphohistiocytosis without evident shock and characteristic vasoplegia that does not respond to the administration of high doses of vasoactive drugs. These patients were maintained with MPAP slightly above 65 mmHg, SPAP rarely exceeding 100 mmHg and DPAP around 70 mmHg without drugs. Molecular analysis of the expression of these receptors in vitro and human experiments, showed different results regarding their complex regulation of levels of angiotensin 1, angiotensin 2, angiotensin 1-7, angiotensin 1-9 in the presence of inhibitors of ACE and ARA 2. In vitro reported increased expression of ACE2 via messenger RNA and increased bradykinin levels (BK-(1-9)) in the presence of enalapril and increased A1-7 and A 1- 9 with ARA2 drugs. However, in humans these findings were not replicated. A hypothesis postulates that SARS-CoV2 would act as a competitive agonist of the ACE2 receptor promoting its internalization and then the presence of these drugs would instead induce

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intracellular signals for the over-expression of ACE 2, increasing Angiotensin 1-7 and consequently , promoting its protective effect.

Based on these arguments, the following questions arise: Could SARS-CoV2 cause elevation of levels blood pressure by reducing ACE2 levels and elevation of angiotensin 2, thus explaining the absence of shock in some severely compromised patients? In less compromised patients, could the increase in systolic blood pressure that does not respond adequately and promptly to antihypertensive treatment be conditioned by the decrease in ACE2 derived from the massive internalization of the virus? Will adult patients with blood pressure levels below the average range for age have less cellular ACE2 and less viral susceptibility? Would monitoring blood pressure figures in patients and individualizing each case allow us to some extent to detect what stage of the disease it is in and could it have any prognostic value? This riddle must be solved quickly because there is no doubt that this virus likes cardiology.