ECRONICON OPEN ACCESS

EC CARDIOLOGY
Short Communication

The Cause and the Effect: Clinicians should be Aware of the Relationship between SARS-CoV-2 and Arrhythmias

Hilaryano Ferreira[1]* and Humberto Morais[2]*

[1]*Department of Cardiology, Hospital Professor Doutor Fernando Fonseca, Lisbon, Portugal*
[2]*Department of Cardiology, Hospital Militar Principal/Instituto Superior, Luanda, Angola*

***Corresponding Author:** Humberto Morais, Department of Cardiology, Hospital Militar Principal/Instituto Superior, Luanda, Angola.

Received: February 20, 2021; **Published:** February 27, 2021

Paracelsus long stated that "all things are poisons, for there is nothing without poisonous qualities. It is only the dose which makes a thing poison".

So, what do you do when you have the lethal combination of COVID19 disease and Long QT (genetic or acquired) syndrome? Should you, or should you not add to this combination azithromycin and hydroxychloroquine? How danger could it be? Are we dying from the disease, or from the cure?

Using its main receptor, the Angiotensin 2 Converting Enzyme (ACE2), SARS-CoV-2 is able to enter and replicate in human host. SARS-CoV-2 causes a multiple proinflammatory chemokines, IL-1B, IL-6, TNF, and IL1RA response, due to the reduction of INF-I and INF-III responses. In some cases, there's a lack of immune response which is insufficient to block virus replication and elimination further exacerbating inflammatory response that may cause cytokine storm, manifested by critical illness with multiple organ dysfunction syndrome [1].

With 30 - 35 percent of the patients presenting concomitant diseases, of which up to 40 percent being cardiovascular disease (with hypertension being the most prevalent) [2], clinicians other than cardiologists must be aware of cardiovascular manifestations caused by this systemic inflammatory response. This is crucial in identifying, triaging and stratifying patients to better manage available resources.

Acute myocardial injury defined by elevation of biomarkers (cardiac Troponin) is a predictor of in-hospital death in about 30 percent cases [3].

When it comes to arrythmias it's a rather complicated matter and one has to ask itself, is it a cause-and-effect relation? Is SARS-CoV-2 a direct cause for arrythmias, or a mere bystander in patients with an underlying substrate, i.e., structural heart disease and concealed genetic phenotypes. Furthermore, the risk of arrythmia may increase in patients with SARS-CoV-2 that are treated with approved drugs such as azithromycin and/or hydroxychloroquine.

Long QT syndrome is a heterogenous group of disorders characterized by QT prolongation and T-wave abnormalities on the electrocardiogram, which is mainly associated with syncope; however malignant arrhythmias like torsade de points leading to sudden cardiac death may occur [4]. Clinicians should be aware of this condition as there known factors that further increase the risk of QT prolongation, such as, advanced age, hypokalaemia, and structural heart disease.

Among patients with COVID-19, about 6 percent of the patients already had a prolonged QTc (>500) at the time of hospital admission. Chorin, Ehud., *et al*. demonstrated in a small cohort of 84 patients being treated with azithromycin and hydroxychloroquine, the baseline QTc interval in all patients was 435 milliseconds pre-treatment [5,6].

In this edition of the journal Sondhi., *et al*. provide an excellent insight into why and how to do QTc surveillance in patients with COVID-19 treated with chloroquine and its analogue hydroxychloroquine, in order to decrease the risk of arrhythmias in patients treated with these drugs [7]. In addition, Ferreira., *et al.* present a systematic review of arrhythmias in Covid. The authors draw attention to the fact that, although arrhythmia-related mortality is relatively uncommon in patients with Covid patients. The pandemic is still ongoing, and more studies need to be carried out in order to acquire more data and establish a direct cause and effect relationship, as well as the long-term effect of the disease on the conduction system [8].

Bibliography

1. García LF. "Immune Response, Inflammation, and the Clinical Spectrum of COVID-19". *Frontiers in Immunology* 11 (2020): 1441.
2. Pericàs JM., *et al*. "COVID-19: from epidemiology to treatment". *European Heart Journal* 41.22 (2020): 2092-2112.
3. Shi S., *et al*. "Characteristics and clinical significance of myocardial injury in patients with severe coronavirus disease 2019". *European Heart Journal* 41.22 (2020): 2070-2079.
4. Shah SR., *et al*. "Long QT Syndrome: A Comprehensive Review of the Literature and Current Evidence". *Current Problems in Cardiology* 44.3 (2019): 92-106.
5. Richardson S., *et al*. "Presenting Characteristics, Comorbidities, and Outcomes Among 5700 Patients Hospitalized With COVID-19 in the New York City Area". *The Journal of the American Medical Association* 323.20 (2020): 2052-2059.
6. Chorin E., *et al*. "The QT interval in patients with COVID-19 treated with hydroxychloroquine and azithromycin". *Nature Medicine* 26.6 (2020): 808-809.
7. Sachin Sondhi., *et al*. "QTc Surveillance in COVID- 19 Era: Why and How?". *EC Cardiology* 8.3 (2021): 22-29.
8. Hilaryano Ferreira., *et al*. "Tachyarrhythmias in COVID19 Patients: A Systematic Review and Meta-Analysis". *EC Cardiology* 8.3 (2021): 14-21.

Volume 8 Issue 3 March 2021