How to cite: Gomes VA. COVID-19 Cardiac Repercussions. Rev Bras Fisiol Exerc 2020;19(2supl):S2-S4. DOI: https://doi.org/10.33233/rbfe.v19i2.4059

RBFEx
ISSN: 2675-1372

Brazilian Journal of Exercise Physiology

Editorial

COVID-19 Cardiac Repercussions

Repercussões Cardíacas da COVID-19

Vinícius Afonso Gomes[1,2,3,4]

1. CISVIVER, Salvador, BA, Brazil.
2. Grupo de Pesquisa Ciências da Saúde em Fisioterapia. Universidade Salvador (UNIFACS), Feira de Santana, BA, Brazil.
3. Centro Universitário UNIRUY WYDEN, Salvador, BA, Brazil.
4. Universidade Federal da Bahia (UFBA), Salvador, BA, Brazil.

Although it is best known for damage to the respiratory system, today we know that the new coronavirus (COVID-19) can also compromise the heart [1]. This fact started to gain strength when a retrospective study pointed out that 33% of deaths in these cases were attributed to cardiorespiratory failure, and 7% to isolated heart failure [2].

A report of a previously healthy woman who developed acute myopericarditis with systolic dysfunction during a COVID-19 condition, drew attention. In this case, it was possible to detect systemic inflammatory responses associated with markers of myocardial injury, such as elevated serum levels of highly sensitive troponin T and creatine kinase-MB. Furthermore, abnormalities in ventricular contraction were found, without any sign of obstruction of the acute coronary flow [3].

Despite the strong evidence, there is still no proof of the presence of the virus within the myocardium, however the occurrence of direct and indirect cardiac lesions attributed to it is plausible. Indirect injuries can be caused by cardiac overload resulting from hypoxemic respiratory failure and systemic inflammation. Whereas direct lesions would be caused by successful tissue infection resulting in the death of cardiomyocytes [4].

Another fact that suggests direct cardiac injury by COVID-19 was the presence of an inflammatory infiltrate of mononucleated cells found in autopsies in cardiac tissue [5]. In 2009, an outbreak caused by a variation of the coronavirus led researchers to investigate the presence of the viral genome in cardiac autopsies. Through the real-time polymerase chain reaction (qPCR), it was possible to find the genome in 35% of patients who died of acute respiratory syndrome [6]. This shows that it is possible to expect similar behavior in cases of COVID-19, as the genomes of both viruses are extremely similar [7].

Previous knowledge states that viral respiratory infections can be the "trigger" for adverse effects of the heart8. In the case of arrhythmias, its manifestation can be observed in several ways, ranging from "simple" isolated premature ventricular contractions, to the successful ventricular fibrillation of asystole9. Regardless of the condition that generates arrhythmias, it is known that episodes of hypoxemia, sympathetic hyperactivity and pro-inflammatory effects, can make them more frequent [8,9].

Correspondence: Vinícius Afonso Gomes. Centro Universitário Uniruy/Wyden - Campus Paralela Av. Luís Viana, 3230 - Paralela, Salvador - BA, 41720-200 - 3° andar NAP. E-mail: vinifisioterapia@yahoo.com.br

If in respiratory infections, healthy hearts can develop arrhythmias, what to expect from those with some affection already installed? The additional inflammation that is generated on the atheromatous plaque, together with the increased demand for oxygen and the reduction of its availability, increase the chances of myocardial infarction [10,11]. The clot formed in cases where the plaque ruptures limits the passage of blood, which causes ischemia and cardiac dysfunction [12].

Still with regard to ischemia, it is necessary to remember that pericytes are contractile, branched cells that have an important role in reducing the permeability of blood vessels [13]. Infectious conditions can promote lesions of these structures, thus causing ruptures of the microcirculation, with subsequent myocardial ischemia [4]. It is true that this process is still speculative, lacking studies that can confirm its hypothesis.

The chances of heart failure decompensation in a COVID-19 infection are high. Its causes are also among those that trigger arrhythmias and infarction, especially myocarditis [8]. The risk of a heart with previous dysfunction deteriorating becomes greater when trying to compensate someone's respiratory system with all the consequences of a systemic inflammation [7]. Despite the relevance of decompensating heart failure, there is another scenario that is even more worrying.

Retrospective cohorts have shown that signs of cardiac injury, such as increased levels of troponin at the onset of the disease, are associated with a worse prognosis [2,14]. In the study by Guo *et al.* [7] the direct relationship between troponin T and the levels of highly sensitive C-reactive protein (CRP) was proven, an important inflammatory marker that reinforces the link between inflammation and myocardial injury. This fact should not go unnoticed, as the risk of death in cases of myocardial injury exceeds that of factors such as age, presence of diabetes mellitus, previous chronic lung and heart disease [7,15].

In short, the heart can be greatly affected in cases of COVID-19, contributing to a significant portion of cases of morbidity and mortality. The worst outcomes seem to be associated with those cases where the infectious process directly affects the heart, increasing the levels of troponin T and CRP. We must emphasize that there are several consequences, including arrhythmias, infarction and heart failure decompensation, triggered mainly by the exacerbated inflammatory response and myocarditis.

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