Review Article

RFP Journal of Biochemistry and Biophysics
Volume 8 Number 1, January - June 2023
DOI: http://dx.doi.org/10.21088/rfpjbb.2582-3558.8123.2

Corona Survivors and Risk of Cardiovascular Disease

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How to cite this article:
Neha Suthar, Sachin C. Narwadiya/Corona Survivors and Risk of Cardiovascular Disease/RFP Jour. of Bio. and Biophy. 2023;8(1):17–21.

Abstract

After surviving from corona extra care of health is requisite in the society. The news of deaths during exercise in Gym were popular as many celebrities and famous personalities lost their lives. Many of them were corona survivors and post corona excessive exercises lead them to death. The present review study is analyzing the facts lying behind the cause of cardiovascular diseases CVDs. The CVDs increasing death rates in India at fast pace.

Keywords:COVID 19; Cardiovascular Disease; Heart Attack; Post COVID-19, Cholesterol.

INTRODUCTION

In 2020, corona virus disease 2019 (COVID-19) was the third leading cause of death with an estimated 345,323 deaths in the US.[1] COVID-19 has attracted the cardiology community perhaps more than any other communicable disease has connections with cardiovascular disease (CVD).[2,3,4]

Early in the pandemic, patients with cardiovascular comorbidities were shown to be most vulnerable to severe infection.[5] The specificity of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) for the angiotensin converting enzyme-2 (ACE-2) protein raised further concerns. of cardiovascular damage and raised concerns about concomitant use of medications including angiotensin converting enzyme inhibitors and angiotensin receptor blockers.[6,7]

In the fight against the new disease, the cardiology community has deployed its most advanced technology, including cardiac magnetic resonance (CMR) imaging, which has characterized the acute and chronic consequences of SARS-CoV-2 infection.[8,9]

Cardiac troponin is a highly specific test for myocardial damage that can be measured by conventional or highly sensitive tests. Notably, elevated troponin (defined as above the 99th percentile of the upper reference limit) does not necessarily correspond to Myocardial infarction MI. According to the 4th universal definition, criteria for MI require a troponin rise/fall with at least one value above the 99th percentile along with

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Received: 07.08.2023

Accepted: 03.09.2023

other symptoms or signs of ischemia.[10]

Type 1 MI occurs because of acute plaque rupture/erosion, which has also been observed in other viral infections, while type 2 MI is caused by "demand is chemia" in the context of oxygen demand/delivery mismatch arising from stressors such as hypoxia, hypoperfusion and tachycardia that can occur in COVID-19 as well as other critical illnesses. Both types of MI have been reported in COVID-19.[2,4,11]

Echocardiography has furthered our understanding of myocardial injury in COVID-19, detailing specific functional patterns of injury.[9] Szekely *et al*. found that the most common echocardiographic abnormality in a series of 100 hospitalized COVID-19 patients was right ventricular (RV) dysfunction. In approximately 40%, RV deterioration is more associated with clinical decompensation.[14] Right ventricular dysfunction was also the most common abnormality observed in an international multicenter cohort of more than 300 hospitalized COVID-19 patients, approximately 26%. Therefore, measurement of troponin in hospitalized patients with covid-19 is integrated into routine clinical practice and management algorithms. For hospitals, this helps predict progress and identify patients who may need more intensive resources, especially during times of shortage.[16] Several societal guidelines, such as the World Health Organization and China's COVID-19 clinical guidelines, recommend troponin measurement for all hospitalized patients. The American College of Cardiology (ACC) and others recommend testing when clinically indicated.[17] The association between increased troponin and mortality raises the debate as to whether myocardial injury is a mediator or a marker of adverse outcomes. Mattkos *et al.* found that in a comparison of covid-19 and non-covid-19 ARDS, increased troponin was associated with mortality and morbidity after controlling for age, sex, and most importantly, multiple organ system dysfunction.[18]

A multicenter international retrospective study of echocardiographic findings in more than 300 hospitalized patients with COVID-19 found that only patients with elevated troponins and echocardiographic abnormalities, not only those with elevated troponins, had a significantly increased risk of mortality. They had a hospital in a rice field.[19]

Exercise in the presence of active myocarditis can increase inflammation and create a pro arrhythmogenic environment. In addition, exercise hearts have abnormalities in size, function, and response to exercise that can make them difficult to distinguish from in flamed or damaged hearts. Strenuous exercise may transiently increase troponin and cause imaging findings suggestive of cardiac fatigue and myocardial inflammation.[21]

The question of when competitive athletes can return to play (RTP) after COVID-19 has become an urgent and important issue for the cardiology field. The urgency stems from the fact that sports organizations, from professional to recreational, were the first to return in full force during the pandemic. This mass rush to return began with little information about how to safely return home after infection. The importance of myocarditis was clear, as it is a potential consequence of COVID-19 and a leading cause of death among young athletes.

In May 2020, the ACC Division of Sports and Exercise Cardiology published the first set of RTP recommendations. Athletes who experience symptomatic infections should under go a 2 week rest period after resolution of symptoms, cardiac evaluation (ECG, echocardiogram, or high sensitivity troponin) and, if abnormal, additional cardiac imaging. I recommend an examination. If myocarditis is diagnosed, physicians are now referred to the existing American Heart Association (AHA)/ACC myocarditis guidelines, which recommend avoiding exercise for 3 to 6 months. Six months later, the department updated and expanded the guidelines to include specific age based recommendations. An expert consensus statement was subsequently issued, both recommending the use of CMR based screening for all athletes with a history of COVID-19.[26]

Cardiopulmonary exercise tests The CPET was performed on a treadmill with continuous measurements of minute ventilation (V'E), V'O2, carbon dioxide production ($V'CO_2$), heart rate, ECG and oxygen saturation measured by pulse oximetry (SpO_2).[27,28]

The COVID-19 pandemic has caused global health, social, and economic system challenges. To try and reduce transmission rates, most countries have varying levels of societal lockdowns and social restrictions in place.[27]

It creates a unique challenge for the promotion of physical activity and exercise, which we know has profound physical and mental health benefits. There was initial promise of increased population interest in physical activity and exercise at the beginning of the COVID-19 pandemic, recent large scale data from over 455 000 people has demonstrated a 27%

decrease in average daily steps within 30 days of the pandemic declaration.[30,31]

It may therefore be more important now than ever to facilitate physical activity and exercise promotion during and post COVID-19. Despite recent collaborative efforts developing post COVID-19 guidelines for athletes returning to exercise, limited evidence is available for the impact of exercise and cardiac rehabilitation (CR) on clinical outcomes following COVID-19.

Secondary prevention through comprehensive CR has been recognized as the most cost-effective intervention to ensure favorable outcomes across a wide spectrum of cardiovascular diseases.[32,33]

Several limitations are of note. Firstly, the characterization of COVID-19, health conditions, and CR and exercise programs were based on ICD codes from EMRs, and reporting of conditions with ICD codes may vary by patient characteristics and healthcare organizations.[34] Indeed, we do not know the severity of individual COVID-19 cases, which may have affected the results. However, before propensity score matching, there was no difference in relative mortality between the cohorts.[35]

During the emergency linked to the spread of Sars-Cov-2, physiotherapy intervention requires remodulation to guarantee the patient recovers their health, and at the same time, to protect the physiotherapist against the risk of contagion. In addition, it is necessary to consider the restrictions imposed by the authorities to prevent the spread of the infection, which cause increasing difficulties in providing rehabilitation assistance in out patient and home settings, and it is also necessary to lighten the burden of acute care by transferring post-COVID patients to rehabilitative structures.[36]

When the rehabilitation intervention cannot be carried out in direct contact with the patient, telerehabilitation may be helpful as an alternative strategy; this involves the use of video calls or adequately structured platforms. In cardiac rehabilitation, there are already promising experiences described in the literature that provide for the use of tele-rehabilitation for a higher number of patients and for a favorable cost/effectiveness ratio.[37,38]

Remote cardio-rehabilitation is safe and effective even for patients with cardiovascular disease or post cardiac surgery.[39] However, monitoring systems that provide for oximetry, blood pressure control is required as well as electrocardiography, especially in the management of complex patients.[40,41]

Cardiac telerehabilitation is mainly based on exercise training in interval or endurance mode, with calisthenics exercises or with the use of a cycle ergometer or treadmill. The intensity of the exercise is established for each patient based on the initial assessment, the hemodynamic parameters assessed remotely with devices such as the oximeter and telemetry, and the symptoms investigated with the administration of scales, such as the Borg scale for dyspnea and RPE. Exercise training should also include counseling strategies, patient education, psychological support, and nutritional interventions.[42,43]

A form of hybrid treatment may be appropriate for this type of patient, limiting in presence physiotherapy to a minimum, preferring the remote modality and scheduling periodic evaluations and treatments in presence.[44]

While questions remain and will continue to emerge regarding COVID-19 and CVD, the pandemic has proven that the scientific community is exceptionally committed and capable of providing these critical answers.

CONCLUSION

The review study is focused on the cardiovascular prevalence among the corona survivors. The study has outcome that Cardiac troponin is a highly specific test for myocardial damage that can be measured by conventional or highly sensitive tests. The elevated troponin can be a criteria for Myocardial Infarction which require a troponin rise/fall oher symptoms or signs of ischemia.[10]

Type 1 MI occurs because of acute plaque rupture/erosion, which has also been observed in other viral infections, while type 2 MI is caused by “demand ischemia” in the context of oxygen demand/delivery mismatch arising from stressors such as hypoxia, hypoperfusion and tachycardia that can occur in COVID-19 as well as other critical illnesses. Both types of MI have been reported in COVID-19.

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