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PREVALENCE OF COVID-19 AND CARDIAC PATIENTS

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INTRODUCTION:

A new strain of *coronaviruses* emerged in Wuhan, China in December 2019, and soon started getting infected many with the unknown pneumonia-like illness (1). It was later named Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), as the signs and symptoms shown by the disease were similar to the 2003 SARS outbreak in Taiwan, China (2). Thereafter, it spread rapidly, and was soon declared as a pandemic (1). Classically, coronavirus disease (COVID-19) presents with pneumonia-like symptoms including fever, cough, pharyngodynia, and fatigue (2); however, in some cases, signs of cardiac damage and myocyte-necrosis have been noticed after COVID-19 (1). Therefore, it is crucial to establish the relationship between the two and increase its awareness amongst the frontline healthcare workers, and in particular, cardiologists.

Recently, an unusual case of COVID-19 was seen in Italy where a previously healthy 53-year-old woman presented with severe fatigue, no dyspnea but complained of only cough and fever from a week before. On physical examination, she was afebrile, hypotensive and had a heart rate (HR) of 100 beats per minute. Her blood tests displayed high levels of myocyte necrosis markers and high sensitivity troponin T level of 0.24 ng/mL (1); a sign of myocardial injury (3). Her electrocardiogram (EKG) showed low voltage in the limb leads, minimal diffuse ST-segment elevation (more prominent in the inferior and lateral leads), and an ST-segment depression with T-wave inversion in leads V1 and aVR, and high levels of myocarditis necrosis markers. Looking at the case, urgent coronary angiography was performed, but it showed no coronary obstruction (1). The patient was admitted as a case of myopericarditis but then decided to be tested for COVID-19 and astonishingly reported positive while she reported negative for any common cardiotropic infectious agents. Similar findings were also noticed in Brooklyn, NY in the United States, where a 64-year-old woman presented with similar signs and symptoms but was diagnosed as a case of COVID-19 on further workup and more reports have been seen around the globe (4).

Much is known about other viruses' effects on the heart but little is documented regarding the growing SARS-CoV-2 (1). One effect of viruses on the heart is myocarditis which is defined as inflammation of the heart tissue with adjacent myocyte necrosis, and infectious agents are known to be the most common cause of it. This has the potential to result in heart failure. More than 20 common viruses, including *coxsackieviruses*, *adenoviruses*, *influenza viruses*, and *human immunodeficiency virus*, have been identified as sources of myocarditis in humans (5). It has also been noticed with another coronavirus strain; Middle Eastern Respiratory Syndrome (MERS) (4). The proposed mechanisms of injury leading to myocarditis include; excessive immune-response, autoimmune-mediated or direct virus-induced cardiomyocyte injury (5). The most likely mechanism for SARS-CoV-2 seems to be the rapid replication and potential dissemination of the virus making its way to the heart and causing local injury (1). Previous severe acute respiratory syndrome (SARS) has also been linked to tachyarrhythmias and heart failure (1).

Patients with cardiac complications associated with COVID-19 might show confusing findings and may present with severe fatigue, hypotension and rapid heart failure. In addition, ST-segment elevation on EKG, increased wall thickness on magnetic resonance imaging (MRI) and echocardiography, hypokinesis, interstitial edema involving both ventricles and severe left ventricular dysfunction might be noticed. Increased Troponin T, mimicking a heart attack, and N-terminal pro-brain natriuretic peptide (NT-proBNP) levels might be observed and should be kept in mind as a possible case of COVID-19 (1). A study including 187 patients correlating the cardiovascular implications on mortality from COVID-19 in Wuhan, showed that 37.50% of the patients with elevated troponin T but without prior history of cardiovascular diseases died after being affected by COVID-19 showing the importance of suspecting COVID-19 in heart patients (3).

Whilst more is researched about the virus's effects on the heart cells, it is crucial for physicians particularly cardiologists to screen all their patients and be vigilant of the likely signs and symptoms a patient may present with. Studies pertaining to COVID-19 and heart should be extensively carried out to help create a better picture. Scientific literature regarding COVID-19 is constantly being reinforced with new findings (6) and cardiologists should be actively updated about it as that can greatly influence their treatment regimens for the disease. Moreover, it is also crucial for workers and physicians in departments of cardiology to carry out specific preventive measures of COVID-19 as respiratory droplets are the main mode of transmission of COVID-19 (6) and they might be unknowingly be infected by the virus and even act as carriers of the disease to most vulnerable; cardiac patients (3).

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