International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2020): 7.803

Poor Prognosis of Myocardial Injury in COVID-19 Patients

Igboanugo Somadila Annastatia

Affiliation: Rostov State Medical University
annastatia4good[at]yahoo.com

Abstract: ***The aim of this study is to evaluate the risk of mortality among COVID-19 patients with myocardial injury. A total of 5559 COVID-19 patients were studied from three different case studies found on original research works using PubMed, Medscape and Google Scholar as sources for the materials. Patients who were older (≥60) and had comorbidities were more prone to developing myocardial injury. From the three different case studies, it was confirmed that COVID-19 patients with myocardial injury had high risk of mortality. Most deaths occurred within 6 months from the day of admission into the hospital.***

Keywords: Myocardial injury, mortality, COVID-19

1. Introduction

Severe acute respiratory syndrome - coronavirus-2 (SARS-CoV-2) is the causative agent of coronavirus disease. The virus was declared a pandemic on the 11th of March 2020 by World Health Organization (WHO). [1]

Although SARS-CoV-2 affects mainly the respiratory systems, research has proven that this virus affects other systems of the body as well. The virus has the ability to exacerbate an existing cardiac disease or it can bring about the existence of a cardiac disease.The SARS-CoV-2 has a spike S protein which it uses to bind to its receptor Angiotensin Converting Enzyme 2 (ACE2). As SARS-Cov-2 is binding to ACE2, it uses transmembrane protease serine 2 (TMMPSS2) as its host to prime the spike S protein and thus making it easier for cell entry. After entering the cell, this virus begins to cause damages. [2], [3], [4], [5]

In normal physiology, ACE2 is the enzyme that converts angiotensin II to angiotensin I. Angiotensin II is responsible for vasoconstriction and increase in blood pressure, while angiotensin I is responsible for vasodilation and a counter regulator of renin-angiotensin system (RAS).But as a result of the attack of SARS-CoV-2, ACE2 is shed into circulation, therefore reducing its function. Angiotensin II on the other hand increases its function and thus enhancing inflammation and vasoconstriction.Patients with underlying heart conditions tend to suffer more because of the increased function of the angiotensin II and reduced function of angiotensinI.It is important to note that ACE2 and TMMPSS2 co-express not only in the heart but also in other tissues like the lung, gut smooth muscles, immune cells, neurons, liver and kidney. This can be used to explain the laboratory findings or symptoms of corona virus disease. These symptoms/laboratory findings include fever, fatigue, myalgia, lymphopenia, dyspnea, dry cough, respiratory failure, nausea, diarrhea, myocarditis, heart failure, arrythmias, vasculitis, thrombosis, microangiopathy, anosmia, hypogeusia, encephalopathy, seizures, myopathy, abnormal liver function, renal dysfunction. [2]

Increase in troponin above the 99th percentile explains the existence of myocardial injury in a patient. Recently a large number of researches have shown the prevalence of myocardial injury within patients with COVID-19, showing their worst results and poorer short-term survival. Subsequent studies have suggested that myocardial injury is common in critically ill patients of COVID-19 through various mechanisms mostly due to direct damage of cardiomyocytes and systemic inflammation.[6] The motive of this study is to evaluate the mortality rate of COVID-19 patients with myocardial injury.

2. Method

I searched PubMed, Google Scholar and Medscape for case studies of COVID-19 patients with myocardial injury that were published between March 11, 2020 to November 2, 2021. I focused on the types of myocardial injury, the cardiac biomarkers, risk factors for this illness, structural abnormalities and the proneness to mortality.

3. Results

3.1 First Study

Myocardial injury can either be acute of chronic. An acute myocardial injury is diagnosed when the cardiac troponin level isnormal on the first test and subsequently increases with >50% variations, or when cardiac troponin is raised on both first and subsequent assessments with >20% variation. Chronic myocardial injury is diagnosed when the cardiac troponin level is elevated on both first and subsequent tests with ≤20% variation. Patients were considered to be myocardial injury free if the troponin level were normal on the first assessment and subsequently increased with ≤50% [7]. Annapoorna Kini et al studied 4695 patients from different hospitals in New York metropolitan area in United State. 319 of these patients had a chronic myocardial injury, 1168 of them had acute myocardial injury while the rest (3208) of these patients were myocardial injury free. Patients with chronic myocardial injury showed higher rates of chronic kidney disease and heart failure, whereas patients with acute myocardial injury were prone to hypertension, tachypnoea, lower peripheral oxygen saturation, and increased levels of inflammatory biomarkers like C – reactive protein, ferritin, interleukin, tumor necrosis factor-α

and D-dimer and also lymphopenia [7], [8]. Research confirmed that patients with either of the myocardial injury were old (60 and above), and so we can say that the more advanced you are in age, the higher the risk of developing myocardial injury when infected with SARS-CoV-2.

According to the report made by Annapoorna Kini et al, within 6 months (day of diagnosis to date of death or recovery), a cumulative of 1106 deaths occurred in the overall study cohort. 13% of patients free of myocardial injury died while 43% of patients with chronic myocardial injury died. Also 47.3% of patients with acute myocardial injury died, **see figure 1 below**. It was not stated why the patients who were myocardial injury free died but you can say that the treatments on them were ineffective as a result of other underlying diseases or low immune system.Patients who were <65 years old with acute myocardial injury were associated with worse prognosis when compared to patients who were ≥65 years old.

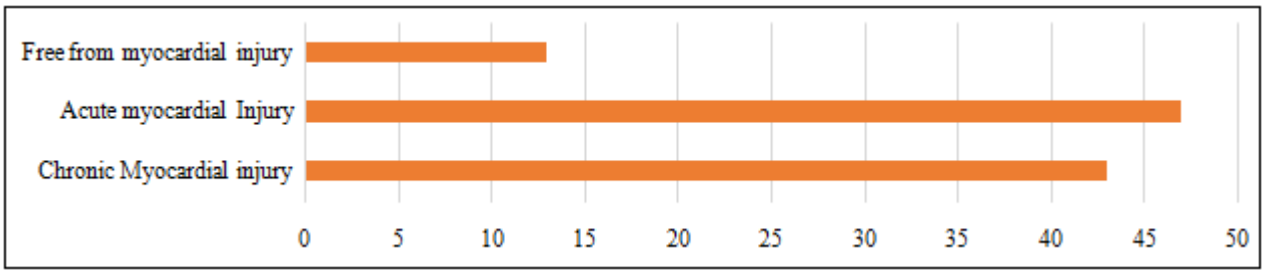

Figure 1: Mortality rate of 1106 patients from different hospitals in New York USA, Mortality rate %

3.2 Second Study

Is it possible to develop myocardial injury during hospitalization? [9], [10] As we read further, we will see the answer to the above question. Gennaro Giustino et al did not only study myocardial injury in COVID-19 patients using cardiac troponin level elevation, they included the use of transthoracic echocardiographic (TTE) evaluations to their studies [6]. Gennaro Giustino et al studied a total of 305 patients in New York, United States and Milan, Italy. Average age of the patients is 63 years of which 67.2% of them were males. A cumulation of 190 patients were confirmed to have biomarkers evidence of myocardial injury, 118 patients out of the 190 had myocardial injury at the time of hospital administration and 72 of them developed myocardial injury during the hospitalization[9]. From the electrocardiographic, echocardiographic and angiographic findings, patients with myocardial injury were susceptible to ST-segment elevation or depression. Patients with myocardial injury had global left ventricle dysfunction (including lower ejection fraction), regional left ventricle wall motion abnormalities, grade II or III diastolic dysfunction, right ventricle dysfunction, and pericardial effusion. They also had greater left ventricle volumes, wall thickness, and left atrial volumes. Furthermore, myocardial injury COVID-19 patients with ST-elevation changes, often had chest pain and had higher degree of troponin elevations.
From the outcome of this study, from 307 number of patients, 18.7%mortality rate occurred, **see figure 2.** [9]Patients with myocardial injury without TTE abnormalities were 17.5% of in-hospital death, patients with myocardial injury with TTE abnormalities were 34.8% of in-hospital death, while patients without myocardial injury were 3.0% of in-hospital death.

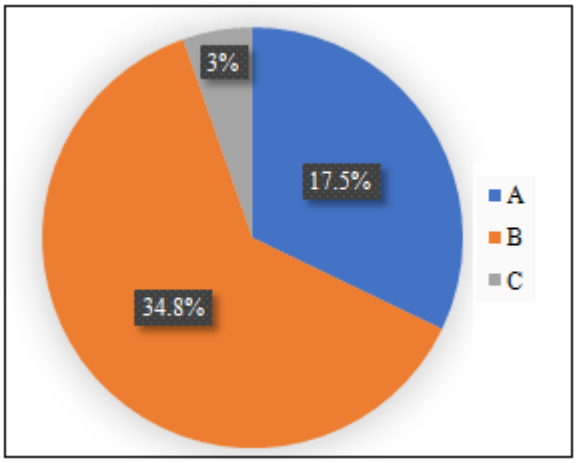

Figure 2: The pie chart shows 18.7% mortality rate in 307 COVID-19 patients
A = myocardial injury COVID-19 patients without transthoracic echo-cardiographic abnormalities
B =. Myocardial injury COVID-19 patients with transthoracic echo-cardiographic abnormalities
C = COVID-19 patients without myocardial injury

3.3 Third Study

Reviewing another study on COVID-19 patients with myocardial injury, OrlyEfros et al [11] studied 559 COVID-19 patients over 18 years old, who were hospitalized at the Sheba Medical Center. 320 patients were tested for troponin levels and 91 of them were found to have an elevated troponin level. Patients with elevated level of troponin were older and had underlying disease like ischemic heart disease, heart failure, diabetes mellitus, chronic kidney disease, cerebrovascular accident and hypertension. These patients were likely to be given beta-blockers and HMG-CoA-reductase inhibitors, they had lower systolic pressure, had higher creatinine level and lower hemoglobin and albumin levels. From this study, there was 30 days mortality among COVID-19 patients with elevated troponin levels.

4. Discussion

In this study, we figured out factors that are associated with increased troponin level in COVID-19 patients, and confirmed that elevated troponin level is associated with a poor prognosis and in other sense, high risk of death among such patients. From the three reviews, COVID-19 patients with myocardial injury have little or no survival. As described in this study, patients with high troponin levels were older and had underlying comorbidities [10], [12], [13], [14], [15], [16],and they were likely to be prescribed with HMG-CoA-reductase inhibitors and beta-blockers.COVID-19 patients with myocardial injuries are more prone to electrocardiographic and echocardiographic abnormalities than COVID-19 patients without myocardial injury. There were various echocardiographic abnormalities which included global left ventricle dysfunction, regional wall motion abnormalities, right ventricle dysfunction, diastolic dysfunction and pericardial effusions. From transthoracic echocardiographic evaluations, COVID-19 patients with myocardial injury had left ventricle, right ventricle and pericardial abnormalities. Transthoracic echocardiographic evaluations in COVID-19 patients with myocardial injury provide a useful prognostic information in the sense that, there was a very significant mortality rate among COVID-19 patients with myocardial injury with TTE abnormalities than COVID-19 patients with myocardial injury without TTE abnormalities. [9]

SARS-CoV-2 virus can navigate its way into the heart through various mechanisms such as a direct viral damage mediated by the ACE2 receptor expressed on cardiomyocytes and an indirect damage secondary to inflammatory response. Patients with heart failure has a higher expression of ACE2 and this may explain their high risk for myocardial injury from the COVID-19 virus. Using polymerase chain reaction, the viral proteome has been detected in the myocardium, but this only indicates the presence of SAR-CoV-2 and not necessarily active infection. [17]

Some COVID-19 patients already developed myocardial injury at the time of admission while some developed it during hospitalization. Both chronic and acute myocardial injury has a poor prognosis. There was high mortality rate in patients with any of these injuries. Generally, acute myocardial injury has been associated with pre-existing diseases like atherosclerosis, while chronic myocardial injury has been associated with pre-existing diseases and cardiovascular risk factors like renal failure and structural heart diseases.

Older patients with cardiac and other comorbidities are considered higher risk for morbidity and mortality if infected by SARS-CoV-2 and so they should avoid person to person contact in this COVID-19 era. [18] It is strictly advised that they should adhere to wearing of masks, drinking of warm water, hand sanitizing and other preventive measures for COVID-19. Also, they should seek immediate medical care when there are symptoms of the virus.

5. Conclusion

Myocardial injury in COVID-19 is associated with high level of cardiac troponin, structural abnormalities which is detected by transthoracic echocardiogram, and lastly increased risk of in-hospital mortality rate. The high mortality rate is more prominent in patients with structural abnormalities. Each type of myocardial injury in COVID-19 patients is attributed to a strong predictor of mortality at six months and in some cases 30 days. Acute myocardial injury is more prevalent and so it may be associated with worse survival among younger patients.

References

[1] Abdullah A. Balkhair. COVID-19 Pandemic: A new chapter in the history of infectious diseases. Oman Medical Journal. 2020; 35(2): e123. doi: 10.5001/omj.2020.41

[2] Peter P. Liu, MD; Alice Blet, MD, PhD; David Smyth, PhD; Hongliang LiMD, PhD. The science underlying COVID-19 implications for the cardiovascular system. Circulation. 2020; 142:68-78. https://doi.org/10.1161/CIRCULATIONAHA.120.047549

[3] Anjali Bhatla BA; Michael M. Mayer BS; Srinath Adusumalli MD, MSc; Matthew C. Hyman MD, PhD; Eric Oh MS; Ann Tierney MS; et al. COVID-19 and Cardiac Arrhythmias. Heart Rhythm. 2020; 17(9):1439-1444. https://doi.org/10.1016/j.hrthm.2020.06.016

[4] Gennaro Giustino, MD; Sean P. Pinney, MD; Anuradha Lala, MD; Vivek Y. Reddy, MD; Hillary A. Johnston-Cox, MD, PhD; Jeffrey I. Mechanick, MD; et al. Coronavirus and cardiovascular diseases, myocardial injury, and arrhythmia. Journal of the American College of Cardiology. 2020; 76(17):2011-2023. https://doi.org/10.1016/j.jacc.2020.08.059

[5] Han-Bin Lin, PhD; Peter P. Liu, MD. COVID-19 and the heart. Journal of the American College of Cardiology. 2020; 5(9):884-887. https://doi.org/10.1016/j.jacbts.2020.07.005

[6] Marco Schiavone, Alessio Gasperetti, Massimo Mancone, Aaron V. Kaplan, Cecilia Gobbi, GiosueMascioli, et al. Redefining the prognostic value of high-sensitivity troponin in covid-19 Patients: The importance of concomitant coronary artery disease. Journal of Clinical medicine. 2020; 9(10):3263. **https://doi.org/10.3390/jcm9103263**

[7] AnnapoornaKini, Davide Cao, Matteo Nardin, Samantha Sartori, Zhongjie Zhang, Carlo Andrea Pivato, et al. Types of myocardial injury and mid-term outcomes in patients with COVID-19. European Heart Journal. 2021; 7(5):438-446. https://doi.org/10.1093/ehjqcco/qcab053

[8] Yader Sandoval, MD; James L. Januzzi, JR, MD; Allan S. Jaffe, MD. Cardiac troponin for assessment of myocardial injury in COVID-19. Journal of the American College of Cardiology. 2020; 76:1244-1258. https://doi.org/10.1016/j.jacc.2020.06.068

[9] Gennaro Giustino, MD, Lori B. Croft, MD, Giulio G. Stefanini, MD, PhD, Renato Bragato, MD, Jeffrey J. Silbiger, MD, Marco Vicenzi, MD, et al.

Charaterization of myocardial injury in patients with COVID-19. Journal of American College of Cardiology. 2020; 76:2043-2055. https://doi.org/10.1016/j.jacc.2020.08.069

[10] Shaobo Shi, MD; Mu Qin, MD; Bo Shen, MD; Yuli Cai, MD; Tao Liu, MD; Fan Yang, MD; et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. JAMA Cardiology. 2020; 5(7): 802-810. doi:10.1001/jamacardio.2020.0950

[11] OrlyEfros, Noam Barda, Eshcar Meisel, Avshalom Leibowitz, Alexander Fardman, GaliaRahav et al. Myocardial injury in hospitalized patients with COVID-19 infection – risk factors and outcomes. PLOS ONE. 2021 16(2): e0247800 https://doi.org/10.1371/journal.pone.0247800

[12] Anuradha Lala, Kipp W. Johnson, James L. Januzzi, Adam J. Russak, Ishan Paranjpe, Felix Richter, et al. Prevalence and impact of myocardial injury in patients hospitalized with COVID—19 infection. Journal of the American College of Cardiology. 2020; 76(5): 533-546. https://doi.org/10.1016/j.jacc.2020.06.007

[13] Fei Zhou MD, Ting Yu MD, Ronghui Du MD, Guohui Fan MS, Ying Liu MD, Zhibo Liu MD, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. Lancet. 2020; 8(6): e46-e47. https://doi.org/10.1016/S0140-6736(20)30566-3

[14] Tao Guo, MD;Yongzhen Fan, MD; Ming Chen, MD;Xiaoyan Wu, MD; Lin Zhang, MD; Tao He, MD; et al. Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019 (COVID-19). JAMA Cardiology. 2020; 5(7): 811-818. doi:10.1001/jamacardio.2020.1017

[15] Carlo Mario Lombardi, MD, Valentina Carubelli, MD, PhD, Annamaria Iorio, MD, Riccardo M. Inciardi, MD, Antonio Bellasi, MD, PhD, Claudia Canale, MD, et al. Association of troponin levels with mortality in Italian patients hospitalized with coronavirus disease 2019. JAMA Cardiology. 2020; 5(11): 1274-1280. doi:10.1001/jamacardio.2020.3538

[16] Tushar Kotecha, Daniel S knight, Yousuf Razvi, Kartik Kumar, Kavitha Vimalesvaren, George Thornton, et al. Patterns of myocardial injury in recovered troponin-positive COVID-19 patients assessed by cardiovascular magnetic resonance. European Heart Journal. 2021; 42(19): 1866-1878. https://doi.org/10.1093/eurheartj/ehab075

[17] Victor G. Puelles, MD, PhD; Marc Lütgehetmann, MD;Maja T. Lindenmeyer,PhD;Jan P. Sperhake, MD;Milagros N. Wong, MD;Lena Allweiss, PhD;etal. Multiorgan and renal tropism of SARS-CoV-2. The New England Journal of Medicine. 2020; 383: 590-592. DOI: 10.1056/NEJMc2011400

[18] Michael N. Young, MD; Alexander Iribarne, MD; David Malenka, MD. COVID-19 and cardiovascular health. Journal of the American College of Cardiology. 2021; 77(2): 170-172.https://doi.org/10.1016/j.jacc.2020.11.027

Author Profile

I am **Igboanugo Somadila Annastatia**, a fifth-year medical student at Rostov State Medical University in Russia. I aspire to become a cardiologist and I am a writer. With my writing skills, I hope to make more researches and write more about medicine.