Contents lists available at ScienceDirect

American Journal of Emergency Medicine

The authors respond: Cardiac troponin levels in Covid-19 patients

Dear editor,

We want to thank the authors for their interest in our article "Prognostic significance of cardiac troponin level in Covid-19 patients without known cardiovascular risk factors" [1]. We gave our answers based on their comments.

Coronavirus disease 2019 (Covid-19), caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), is mainly characterized by viral respiratory findings [1]. However, shortly after the onset of global pandemic, the current data indicates that the SARS-CoV-2 has an enhanced potential to lead to myocardial injury, especially in patients with a history of cardiovascular disease.

Several mechanisms have been blamed for cardiac injury and cardiac troponin.

elevation in Covid-19 patients. It has been considered that direct viral myocardial.

injury through angiotensin-converting enzyme-2 (ACE-2) receptor signaling pathway may have a key role in triggering the inflammation in the cardiac tissue [2]. Because ACE-2 receptor, which is a main entry point for the SARS-CoV-2 to infect the host cells [3], is highly expressed in the myocardium [4], the SARS-CoV-2 virus can theoretically bind and enter the cardiac myocyte with using ACE-2 receptor, thereby leading to myocardial injury. Moreover, cardiac magnetic resonance imaging findings showed the presence of edema and late gadolinium enhancement in the myocardium in patients with Covid-19 [3,4], which further supports the hypothesis that the SARS-CoV-2 can cause myocardial damage secondary to cytokine storm [5]. Upsurge of interleukin-6 (IL-6) and IL-1 levels as well as imbalance between type 1 and 2 T helper cells can impair cardiac contractility and precipitate cardiac myocyte injury [6,7]. In addition, Covid-19-induced microthrombus formation in the coronary microvasculature may be the other cause of myocardial damage in Covid-19 patients [8]. Therefore, the underlying mechanisms could be either one of the direct myocardial injury, cytokine storm, microthrombus formation or synergistic effects of all them.

In order to illustrate the causes of cardiac troponin elevation in Covid-19 cases, imaging modalities, such as coronary angiography (CAG) and cardiac magnetic resonance image (MRI), should be applied for further evaluation. However, such imaging studies could not be performed in most cases in our study because of highly infectious nature of the virus. Therefore, the main cause of cardiac troponin elevation remains unclear in most patients.

In our study, Covid-19 patients who did not have a typical and/or persistent chest pain and had normal electrocardiography (ECG) findings, and those patients without progressive cardiac troponin elevations

were managed conservatively, including initiation of anti-platelet (either acetylsalicylic acid (ASA) or P_2Y_{12} inhibitor), low-molecular-weight heparin (LMWH), beta-blocker, and statin therapy. Covid-19 patients with dynamic ECG changes and progressive cardiac troponin elevations underwent CAG. Cardiac MRI was performed if CAG yielded a normal or non-significant coronary artery disease.

Declaration of Competing Interest

None to declare.

References

[1] Çınar T, Hayıroğlu Mİ, Çiçek V, et al. Prognostic significance of cardiac troponin level in Covid-19 patients without known cardiovascular risk factors. Am J Emerg Med. 2020. https://doi.org/10.1016/j.ajem.2020.12.033 S0735-6757(20)31152-9.

[2] Zhang H, Penninger JM, Li Y, et al. Angiotensin-converting enzyme 2 (ACE2) as a SARS-CoV-2 receptor: molecular mechanisms and potential therapeutic target. Intensive Care Med. 2020;46(4):586–90.

[3] Wang Q, Zhang Y, Wu L, et al. Structural and functional basis of SARS-CoV-2 entry by using human ACE2. Cell. 2020;181(4):894–904 e9.

[4] Chen L, Li X, Chen M, et al. The ACE2 expression in human heart indicates new potential mechanism of heart injury among patients infected with SARS-CoV-2. Cardiovasc Res. 2020;116(6):1097–100.

[5] Akhmerov A, Marbán E. COVID-19 and the Heart. Circ Res. 2020;126(10):1443–55.

[6] Mehta P, McAuley DF, Brown M, et al. HLH across Speciality collaboration, UK. COVID-19: consider cytokine storm syndromes and immunosuppression. Lancet. 2020;395 (10229):1033–4.

[7] Weiskopf D, Schmitz KS, Raadsen MP, et al. Phenotype and kinetics of SARS-CoV-2-specific T cells in COVID-19 patients with acute respiratory distress syndrome. Sci Immunol. 2020;5(48):eabd2071.

[8] Fernandes Jr CJ, de Assuncao MS. Myocardial dysfunction in sepsis: a large, unsolved puzzle. Crit Care Res Pract. 2012;2012:896430.

Tufan ÇınarAssociate Prof.
^aHealth Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Istanbul, Turkey
*Corresponding author at: Health Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Tibbiye Street, Uskudar, Istanbul, Turkey.
E-mail address: drtufancinar@gmail.com

Mert İlker HayıroğluAssociate Prof.
^aHealth Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Istanbul, Turkey
^bHealth Sciences University, Dr. Siyami Ersek Training and Research Hospital, Department of Cardiology, Istanbul, Turkey

Vedat Çiçek MD
^aHealth Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Istanbul, Turkey

Şahhan Kılıç MD
^aHealth Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Istanbul, Turkey

Süha Asal MD

[a]*Health Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Istanbul, Turkey*

Selami Doğan MD

[a]*Health Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Istanbul, Turkey*

Mehmet Şeker MD

[a]*Health Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Istanbul, Turkey*

Murat Selçuk MD

[a]*Health Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Istanbul, Turkey*

Mehmet UzunProf.

[a]*Health Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Istanbul, Turkey*

Ahmet Lütfullah OrhanProf.

[a]*Health Sciences University, Sultan II, Abdülhamid Han Training and Research Hospital, Department of Cardiology, Istanbul, Turkey*

13 March 2021