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Letter to the Editor

Cardiac Troponin-I may be a predictor of complications and mortality in COVID-19 patients



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To the Editor,

Coronavirus disease 2019, commonly referred to as COVID – 19, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has resulted in one of the worst pandemics the world has ever witnessed. From the first case of pneumonia caused by an unknown agent in Wuhan, China, It has now spread worldwide, to more than 215 countries, infecting over 37 lakh people and claiming more than 2.5lakh lives as on 8th May 2020.¹ The world health organisation has described the pandemic as a public health emergency of international concern.

It is reported that up to 15% of COVID – 19 infected cases is complicated by severe pneumonia, which may further progress to complications like acute respiratory distress syndrome (ARDS), Multi organ failure and death.² It is already known that cardiac complications are common in a considerable amount of patients suffering from pneumonia.³ Viral infections are one of the leading causes of infective myocarditis and is commonly seen in influenza and parvovirus B19 infections. Though the SARS beta coronavirus was found to be associated with signs of heart failure, the cardiovascular involvement of the novel SARS – Cov 2 virus is yet to be described completely. It is noted that among COVID 19 infected patients, those with coexisting cardiovascular diseases are the ones with the highest risk of progression to severe disease and death.⁴ As the virus is new, there exists a big knowledge gap about the characteristics of the virus, the natural history of the disease, and its management.

Myocarditis caused by viruses may be focal or global, and might result in inflammation and necrosis of the myocardial wall, eventually resulting in ventricular dysfunction. Chest pain in a patient with influenza like symptoms, with clinical evidence of acute coronary syndrome on ECG or lab values, and wall motion abnormalities, without any evidence of obstruction in coronary angiography should arouse the suspicion of a focal viral myocarditis⁵

Laboratory data from China has pointed out that among patients diagnosed with COVID – 19, the values of cardiac troponin I (cTnI), were significantly high among patients with severe disease than in the ones with milder forms of the disease.⁵ Though recent literature shows that marginal elevation of cardiac Troponin I (cTnI) is

common in most patients infected with COVID - 19, only about 8–12% of cases show values above the 99th percentile of the upper limit. Also significant elevation in the values of cTnI was noted in patients with severe form of the disease who progressed to major complications like multi organ dysfunction and death.⁷

Cardiac involvement in these cases maybe due to direct dissemination of the virus into the blood stream from the lungs, or due to an exaggerated immune response to the virus, resulting in myocardial injury. The second theory may justify the use of corticosteroids to attenuate the inflammatory response, as was seen in some cases of COVID – 19 with myocarditis who improved with the use of steroids.⁸

Hence it may be reasonable at this point to consider that significant elevation in the values of cardiac Troponin I (cTnI) may be linked to severe disease and life threatening complications in patients infected with COVID – 19. We hypothesize that measurement of cTnI values at the time of hospital admission and following up the values longitudinally may help us identify the subset of patients with COVID 19, who may progress towards severe complications and death. This might help in focussing more on this subset of patients which in turn might help in lowering the morbidity and mortality associated with the current pandemic worldwide. Further studies on this topic is urgently required which will help in further investigations into this topic and help in controlling the current outbreak and deaths associated with it worldwide.

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Declaration of Competing Interest

The authors declare no conflict of interest.

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