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Title: Patterns of heart Injury in COVID – 19 and relation to outcome.

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Article:

We read with much interest the article "Analysis of heart injury laboratory parameters in 273 COVID-19 patients in one hospital in Wuhan, China" by Han et al as published in March 2020. In this retrospective, single center study authors have discussed the role of

acute cardiovascular injury marker including CK-MB, Myoglobulin, cardiac troponin I (cTnI) and NT- proBNP on the outcome of 273 patients with COVID-19 disease. Authors have suggested that elevated concentrations of these enzymes in venous blood were related to disease severity and poor outcome[1]. We believe that this topic is relevant and evolving. We have the following additional comments for further understanding of the disease and its various pathophysiological bases in cardiac involvement.

Non ischemic and ischemic myocardial injury has been attributed to be the two principle pathophysiological basis of acute cardiac injury in COVID-19 patients[2]. Of the two, nonischemic myocardial injury is predominant and is secondary to multiple secondary mechanisms. The various distinct mechanisms for non-ischemic myocardial injuries that have been published in the literature are – [i] cytokine storm as documented by significantly raised inflammatory markers [ii] secondary to hemophagocytic lymphohistiocytosis, [iii] viral myocarditis with reports of progression to fulminant myocarditis, [iv] stress cardiomyopathy, and [v] hypoxia induced cardiac myocyte apoptosis[3-5].

Markers of cardiac injury including CK-MB, MYO, LDH, ultra-TnI, NT-proBNP, D-dimer are elevated in any of the above patterns of cardiac injury. However, in most studies of COVID-19 patients showing increased mortality, an elevated troponin I level has been uniformly associated with uncontrolled inflammation secondary to cytokine storm. This phenomenon of infection related increased cytokine release is identified by an elevated plasma level of several inflammatory markers. Various inflammatory markers that are shown to be elevated in patients with COVID-19 related cardiac injury are C-reactive protein (CRP), procalcitonin, ferritin, D- dimer, Interleukin - 2 (IL-2),

Interleukin – 7 (IL-7), granulocyte – colony stimulating factor, IgG- induced protein 10, chemokine ligand 3 and tumor necrosis alpha[2,3,6]. Did the authors have the details of various inflammatory markers among their patients?

Not only that cytokine storm related cardiac injury has been consistently reported in most studies, but it has also been identified to be predictors of complications like acute respiratory distress, acute kidney injury, the severity of illness, requirement of intensive care unit admission and higher mortality[2,7,8].

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