Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Editorial

Myocardial injury in COVID-19: When observations become reality

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Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) emerged in Wuhan, China, in late 2019 and is still causing a global pandemic [1], with second waves now afflicting several countries. Although COVID-19 presents primarily as a respiratory condition, evidence clearly shows it is a systemic disease [2]. Cardiac injury has been described since the first reports, primarily detected through the elevation of biomarkers of myocardial necrosis [3–7]. Several pathophysiological mechanisms have been hypothesized varying from a direct viral damage to the explanation of myocardial injury as a result of an intense cytokine response causing inflammatory injury [5]. Regardless of the etiology, cardiac injury has been associated with adverse outcomes and mortality in COVID-19 [8].

In this issue, Yang et al. [9] provide an interesting report on the prevalence of myocardial injury, defined by the detection of a rise in cardiac troponin I (cTnI) above the 99th-percentile upper reference limit, in a retrospectively collected cohort of 203 laboratory-confirmed COVID-19 patients (median age 62 years, male prevalence 56%). Besides confirming that the phenomenon is not rare (37%) in the overall considered population, the Authors proved that myocardial injury was more common among those who were critically (47%) vs. moderately ill (12%) upon admission and in non-survivors (50%) vs. survivors (6%), remarking the association of cardiac injury with dismal outcomes. To better appreciate pathophysiological changes of cardiovascular system during the clinical course of COVID-19, the Authors recorded serial biomarker measurements of each patient for temporal trend analysis, demonstrating that cTnI levels as well as other markers of cardiac injury (myoglobin, NT-proBNP, lactate dehydrogenase) and inflammation (C-reactive protein [CRP], d-dimer) were higher at any time point in non-survivors compared to survivors, in line with what reported in other studies [8]. Finally, to facilitate clinical interpretation, they constructed a simple chart with the expected probability of mortality determined by different combinations of variables including d-dimer, CRP and cTnI. The subgroup of patients with baseline d-dimer <1 μg/ml, cTnI <26 pg/ml and CRP <10 mg/l had the lowest mortality (3%). As the three biomarkers all raised above the upper limit of reference range, mortality could approximate 90%.

Yang et al. added another piece of evidence on what has now to be considered a proven reality: the relevance of myocardial injury in COVID-19 and its value as marker of poor prognosis since the initial stages of infection. Unfortunately in this report, no comparison on rates of cardiac events between those experiencing vs. those who did not experience myocardial injury was provided. However, recent data showed that serious cardiac events were more common in critically ill patients with cardiac injury compared to those without cardiac injury, suggesting that cTn elevation does have cardiovascular disease manifestations and consequences [8]. Even though speculations on the possible existence of a direct virus-mediated myocardial injury have been proposed, most of the evidence, including the one here provided by Yang et al. [9], suggests that the most plausible mechanism might be secondary to the systemic inflammation caused by SARS-CoV-2. As the COVID-19 pandemic continues to place healthcare systems under immense stress, the need to identify patients who are at greater risk since the early stage of hospitalization is of paramount importance for front line physicians. Biomarkers of myocardial injury should both be included in the laboratory triage and in the follow-up panels of COVID-19 patients, together with markers of inflammation and coagulation, as it may help identify those characterized by higher risk of severe disease and mortality.

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Declaration of Competing Interest

None.

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