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

Cardiac injury after COVID-19: Primary cardiac and primary non-cardiac etiology makes a difference

With the coronavirus disease 2019 (COVID-19) pandemic entering now the third year, the multiple and devasting ways of the etiologic virus, severe acute respiratory syndrome-coronavirus 2 (SARS-CoV-2), involving the heart and cardiovascular system become more and more clearer [1].

The tremendous effort in research in combination with the numerous clinical observations sheds light on this complex and multifactorial interdependence. Therefore, today we can assume that cardiac injury associated with COVID-19 can result via direct or indirect mechanisms. Furthermore, we can distinguish between acute and post-recovery involvement of the cardiovascular system by the virus [2]. Another important facet of the pandemic is the associated collateral damage in the management of many other diseases. This includes all forms of heart diseases, not only the often issued delayed treatment of myocardial infarctions, has to be considered [3–5].

The direct mechanism involves Corona-virus infiltration into myocardial tissue, causing cardiomyocyte death and further fuel the inflammation process. Indirect mechanisms include stress on cardiac myocytes due to respiratory failure, hypoxemia and cardiac involvement secondary to severe systemic hyperinflammation [6].

In 2003, ACE2 was identified as the receptor for cell entering of SARS-CoV. SARS-CoV-2 also binds to ACE2 [1]. So, with the beginning of the COVID-19 pandemic the question arose, if there is some kind of interplay and further, whether or not the cardioprotective therapy with ACE/ARB in patients with heart failure should be discontinued. The latest, quite well conceived study by Bauer et al., showed no statistical significant interaction [7].

In addition, potential long-term cardiac sequelae of COVID19-associated myocardial injury has been suggested [8]. Evidence of some kind of myocardial abnormalities were noted in a remarkably wide range, from 9% to 78%, of patients who recovered from acute COVID-19 [1]. Although more recent analyses suggest a lower incidence of long-term cardiac involvement.

Considering all the above-mentioned ways of cardiovascular involvement in the context of COVID-19 infections, it is of interest to know what the clinical implications are. The study by Khaloo et al. published in this issue of the *International Journal of Cardiology* provides further insights in this context. [9]. This well conducted multicenter retrospective cohort study included 2450 patients hospitalized for confirmed COVID-19 infection within a single hospital network between March and June 2020. The authors used a logistic regression analysis to identify predictors of mortality. Wide variability in some previous studies that reported an impact of myocardial injury on mortality of patients with COVID-19 has been attributed to the small sample size and the differences in the definition of cardiovascular events. Due to the high sensitivity, with the accompanied, sometimes "false positive results", troponin measurements alone do not allow to distinguish between a primary cardiac event leading to a biomarker elevation or a secondary cardiac event, precipitated by non-cardiac illness such as pulmonary disease, renal failure or sepsis [10].

In the current study, the authors showed that 57% of the study population had elevated high sensitive troponin (hs-TnT) levels. The comprehensive work-up and discrimination of the etiology of the troponin origin includes review of the primary data to confirm the ICD-10-CM code, furthermore putative changes in the 12-lead ECG and in the echocardiogram (e.g. wall motion abnormalities) pathologies. This analysis showed, that 653 (47%) of the 1401 patients with elevated hs-TnT levels in COVID-19 patients had a primary cardiac etiology (e.g. myocardial infarction), whereas 748 patients (53%) had a primary non-cardiac etiology such as renal failure ($n = 304$) or critical illness ($n = 286$). Remarkably, the mortality rate was significantly higher (OR 4.6, 95% CI: 2.7–7.6; $P < 0.001$) in patients with a primary cardiac etiology.

Taken together, this well performed analysis by Khaloo et al. stresses the extraordinary importance of cardiovascular care in COVID-19 patients and underlines that knowing the cause of the troponin etiology, helps not only to choose the right treatment but furthermore allows to predict more precisely the outcome of the given patient. Therefore, this paper has some potential implications on our daily cardiovascular-treatment of COVID-19 patients.

Declaration of Competing Interest

None.

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