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.

Relevance of myocardial injury biomarkers to the prognosis of COVID-19 patients. Response

Relevancia de marcadores de daño miocárdico en la evolución de los pacientes con COVID-19. Respuesta

To the Editor,

We read with interest the letter by Alcaide et al., which comments on our article about biomarkers of myocardial injury in the prognosis of COVID-19.[1] We would like to thank the authors for their remarks.

We agree on the importance of early identification of patients at higher risk, and this was one of the reasons for our study. Myocardial injury in COVID-19 patients is likely related to inflammation and oxidative stress, as well as to an imbalance between oxygen supply and demand in the myocardium.[2] In patients with serious forms of COVID-19, elevation of the N-terminal fraction of the pro-B type natriuretic peptide (NT-proBNP) has also been reported in the literature, although to a lesser extent. Our study was among the first to observe this, and later studies describe similar results.[3] This elevation may be caused by a combination of hemodynamic deterioration, stress on the myocardial wall, inflammation, and myocardial ischemia. However, we found no published studies showing a lack of association between higher NT-proBNP levels and worse prognosis of COVID-19.

Our study found that elevated troponin T and NT-proBNP had high negative predictive value for mortality at 30 days (97.88% and 98.14%, respectively) and at 50 days. The negative predictive value was also high for the combined endpoint of mortality and mechanical ventilation, although not quite as high (91.94% and 91.27%, respectively). The mortality + mechanical ventilation group included patients with serious infection, some of whom survived. In this case, the negative predictive value was lower than that of the mortality group. Table 1 of the additional material shows the drop in prevalences of elevated troponin T and NT-proBNP as the seriousness of the infection declined.

We are aware of the limitations of our study and, therefore, mentioned them in our article: *a)* the study was conducted between February and April 2020, and at that time SARS-CoV-2 molecular detection tests were performed in hospitals, leading to a selection bias; in an attempt to mitigate this bias, our study included patients referred for home hospitalization; *b)* although this was a single-center study, subsequent studies with a larger sample size and in other geographic areas report similar results,[3] and *c)* the pharmacological treatments were listed in table 2 of the article,[1] and a higher prevalence was observed for treatments with antibiotics, systemic corticoids, and low-molecular-weight heparins in patients with elevated troponin T and NT-proBNP. However, these patients had a worse short-term prognosis.

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A. Calvo-Fernández contributed to the article text. J. Marrugat and B. Vaquerizo contributed with article review and correction.

CONFLICTS OF INTEREST

None.

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