

# Cardiology and respiratory failure in the context of COVID-19: a multidisciplinary perspective

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After reading with great interest the article entitled: "Exploring the impact of the COVID-19 pandemic on provision of cardiology services: a scoping review" redacted by Farah Yasmin *et al.* [1], published by Reviews in Cardiovascular Medicine, I would like to add the following.

With the emergence of the SARS-CoV-2 and the collapse of health systems around the world, it has become clear that COVID-19 goes beyond an exclusively respiratory disease. Some pre-existing comorbidities, especially cardiac ones, cast a shadow on the prognosis of patients if they are not identified in a timely manner.

That is why a meticulous understanding of cardiac hemodynamics and the diagnostic and therapeutic consequences are essential for an adequate triage and management of these patients since, despite the already known pulmonary involvement, multiple direct and indirect inflammatory and mechanical factors increase mortality, especially in populations with risk factors or a significant cardiac history [2].

First, COVID-19 can lead to myocardial injury secondary to the release of pro-inflammatory cytokines such as members of the interleukin and tumor necrosis factor families, which through the activation of inflammatory cascades lead to cardiotoxicity due to oxidative stress or apoptotic pathways. A second theory establishes that within the context of the infection mechanism the virus directly infiltrates the cardiac cells leading to the activation of local triggers that lead to myocardial damage. The third mechanism establishes that one of the main mechanisms for heart failure in the context of COVID-19 may be related to situations of thrombosis. There is the possibility that elevations in some cardiac markers such as troponins and natriuretic peptides in critically unwell individuals infected with COVID-19 are secondary to right heart strain from multiple pulmonary emboli [3, 4].

Direct cardiac injury, increased thrombotic activity, stress cardiomyopathy, and others are some of the most notable cardiac complications that appear in the course of infection. Thus, the integration of clinical observations and findings in auxiliary examinations can promote our knowledge and understanding of the underlying mechanisms identifying and treating cardiovascular complications appropriately [5, 6].

Different publications in the medical literature report a higher mortality and poor prognosis in patients with these cardiovascular associations. That is why a multidisciplinary clinical approach in the cardiovascular and respiratory system as a perfect binomial, an early measurement of markers of heart injury (troponin and natriuretic peptides), electrocardiogram (ECH), echocardiography (ECHO) and its correct interpretation by a team of specialists during the hospitalization process will not only reduce the hospital stay, but will also reduce the probability of death [7, 8].

The effects of SARS-CoV-2 on the heart are diverse. The prognosis worsens when the heart is directly involved. The ECH is the primary preferred tool for assessing cardiac involvement. Findings indicated that ST-elevation, bradycardia, atrial fibrillation, left ventricular ejection fraction, and T-wave inversion could be a possible predictor of worse outcome of COVID-19. Moreover, High levels of brain natriuretic peptides, indicating cardiac stretch, are associated with a higher risk of mortality in patients with ARDS (Acute respiratory distress syndrome) independently of other commonly used prognostic indicators. Further studies are required to determine if a similar relationship exists between cardiac troponins and mortality in ARDS [9–11].

#### Author contributions

CZC wrote and compiled the information in the manuscript, LSMA was in charge of the correction of the language and writing.

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### **Conflict of interest**

The authors declare no conflict of interest.

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