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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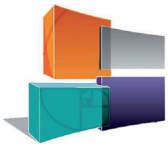

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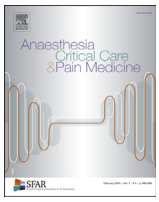

Letter to the Editor

Left ventricular dysfunction in COVID-19: A diagnostic issue

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1. Introduction

The coronavirus disease 2019 (COVID-19) is mainly a respiratory disease potentially leading to acute respiratory distress syndrome (ARDS). Besides, major cardiac complications are being reported [1]. Here is provided the ongoing knowledge available in the literature to manage patients with left ventricular manifestations of COVID-19.

2. Physiopathology

Respiratory droplets are thought to be the main transmission route of SARS-CoV-2. The spike protein surface of the virus uses its receptor-binding domain to mediate the interaction between the angiotensin converting enzyme 2 (ACE2), following activation of the spike protein by transmembrane protease serine 2 found in the lower respiratory human tract (type II alveolar cells), and enables host cell entry [2]. Myocardial localisation of ACE2 has been described, suggesting a possible direct infection of the heart. A cytokine storm could be involved in patients with severe forms of COVID-19, implying a direct myocardial toxicity and/or myocardial ischemia due to myocardial oxygen imbalance [1]. Further, some drugs used to treat COVID-19 are well known to provide cardiac toxicity (Fig. 1).

3. Myocarditis and pericarditis

Direct virus and cytokine-mediated toxicities can explain the occurrence of cardiac myocarditis and/or pericarditis. Severe cases requiring veno-arterial extracorporeal life support have been described. The direct identification of the virus in myocardial biopsies has sometimes been carried out, even if a blood contamination remains possible [3]. The pretty similar presentation of type-2 myocardial infarction and the incompleteness of some reports on the ongoing literature make it difficult to differentiate the prevalence of both presentations.

4. Myocardial ischemia

There are two types of myocardial injury: the first one is due to the obstruction of a coronary plaque (type-1 myocardial infarction) and the second one is related to a mismatch between oxygen needs/supply leading to myocardial ischemia (type-2 myocardial infarction). Such an imbalance can result from non-obstructive coronary disease that reduces the oxygen transport, but also from hypoxemia secondary to ARDS and systemic inflammatory syndrome. In a recent report including 18 patients with typical ST-segment elevation, 8 patients only had an obstructive coronary disease and 5 received percutaneous coronary revascularisation [4]. Among those patients, chest pain and cardiovascular risk factors were more frequent, and D-Dimers and troponin levels were higher. Because of the high hypercoagulability, micro thrombi and/or emboli could also promote myocardial injury. A troponin elevation is a factor associated with poor prognosis and increased death rate in COVID-19. In a recent series of 188 patients (overall mortality rate 23%), patients without cardiovascular disease and troponin elevation experienced a 7.6% mortality rate while patients with cardiovascular disease and without troponin elevation had a 13.3% mortality rate. Finally, patient without cardiovascular disease but with troponin elevation had a 37% mortality rate, and patient with both cardiovascular disease and troponin elevations reached a 69% mortality rate. It has to be compared with high prevalence of type-2 infarction in ICU and the strong correlation between type-2 infarction and outcomes previously reported in ICU patients [5]. A clinical approach assessing serum troponin level, ST-segment modifications and new echographic wall motion abnormalities should be considered to avoid delay in the diagnostic of myocardial infarction.

5. Arrhythmias

Ventricular tachycardia and/or fibrillation occurred in 5.9% of hospitalised COVID-19 patients, and in 17.3% patients with troponin elevation [1]. Again, the main reason seems to be myocardial ischemia secondary to oxygen imbalance. The prevalence of atrial fibrillation is not yet described in COVID-19 but we may infer that it should be high since hypoxemia and old age are factors also associated with both COVID-19 and atrial fibrillation. The abundant communication regarding potential benefits of hydroxychloroquine ± azithromycin in the treatment of COVID-19 has led to a large prescription of both drugs. QT interval prolongation is frequent in ICU patients and known to increase mortality. Thus, it should be properly monitored, and in case of prolongation, those drugs should be stopped. Meanwhile, their use should be evaluated according to the individual benefit/risk.

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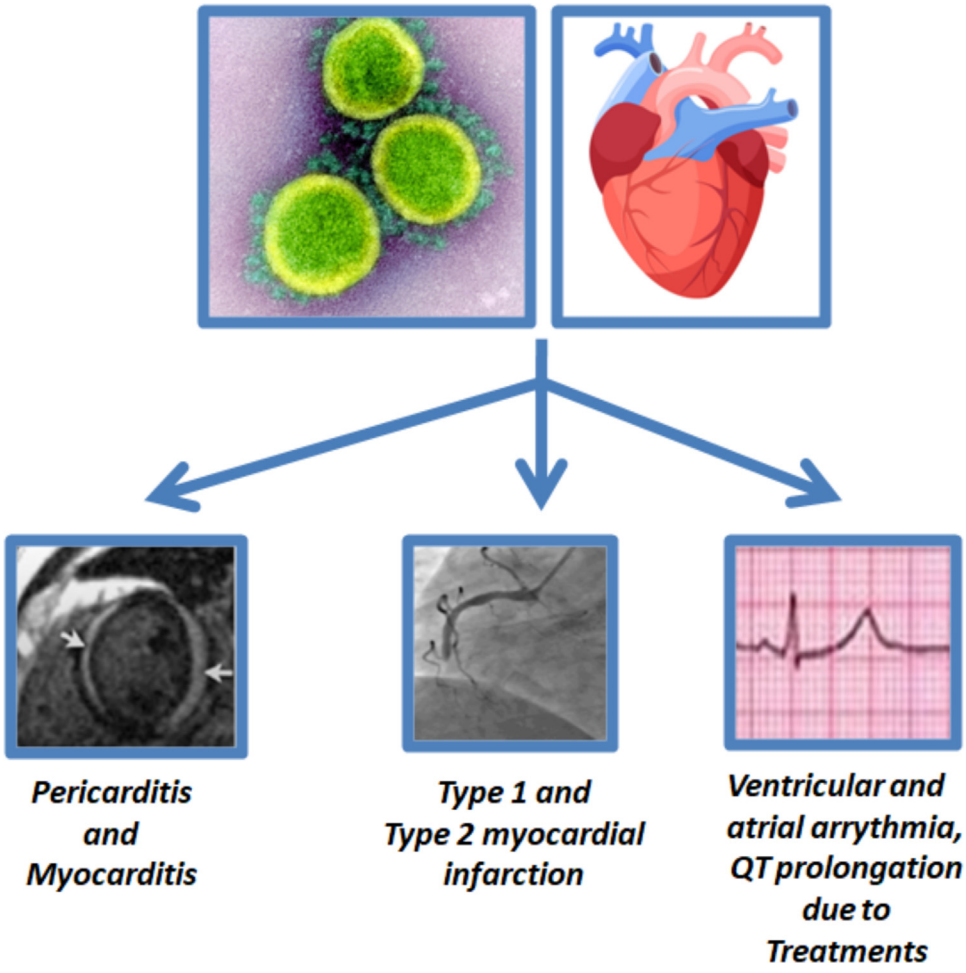

Fig. 1. Mechanism involved in left ventricular dysfunction in COVID-19.

6. Conclusion

Left ventricular dysfunction is frequent in COVID-19 patients and increases mortality. Cardiac assessment including troponin dosage, EKG monitoring and echocardiography is warranted to avoid the delay of diagnostic and to adapt the therapeutic approach adequately.

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Disclosure of interest

The authors declare that they have no competing interest.

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