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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Resuscitation

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Letter to the Editor

Myocardial injury in severe COVID-19: Identification and management

Abstract

The World Health Organization declared the 2019 coronavirus (COVID-19) a global pandemic on March 12, 2020. However, inadequate attention seems to have been paid to the heart when managing COVID-19 in terms of detection, monitoring and treatment.

We are of the opinion that these severe patients may have had myocardial injury or acute myocarditis. Signs that supports this opinion is the extremely high myocardial injury markers in severe patients, cardiac arrhythmia and suffer progressive heart failure or unexpected cardiac arrest in recent studies. Some suggestions involved of treatment need to be made. The use of an intra-aortic balloon pump (IABP) plus extracorporeal membrane oxygenation (ECMO) should be placed earlier if the pneumonia progresses rapidly, the ejection fraction decreases or there is heart failure.

Besides, blood purification treatment including continuous kidney substitution treatment (CRRT) is recommended to clear inflammatory factors and block cytokine storm. In addition, the early usage of glucocorticoid and human immunoglobulin has been found to be preferable when acute myocarditis is accompanied by unstable hemodynamics.

Keywords: Myocardial injury, COVID-19, Cytokine storm

On March 12, 2020, the World Health Organization declared the 2019 coronavirus (COVID-19) a global pandemic. We have observed severe COVID-19 patients frequently developing myocardial injury and myocarditis, running into the underlying cardiovascular epidemic.[1] The most common of cardiac injury is elevated cardiac troponin levels at admission, which was reported in many studies.[2,3] Besides, cardiac arrhythmias are also frequently observed in COVID-19 patients. Furthermore, patients with severe COVID-19 have often been found to suffer progressive heart failure or cardiac arrest.

There are three predominant mechanisms or phases of myocardial injury induced by COVID-19 (Fig. 1). Firstly, this may be virally mediated with direct invasion into the myocardial cell via the angiotensin converting enzyme 2 receptor which is mainly expressed in the lungs and heart. Second, the oxygen supply demand imbalance might cause type-2 myocardial infarction, and the observation of hyaline thrombus in small blood vessels of multi-organs indicated that the patient had diffuse intravascular coagulation. The third mechanism is a hyperinflammation response, leading to a cytokine storm. Although autopsy study revealed that degeneration and necrosis could be seen in a small number of myocardial cells,[4] the systemic inflammation response appeared disproportionate to the degree of myocardial injury in patients with multi-organ failure.

Protocols for early management of cardiac injury in patients with severe COVID-19 should be instigated as early as possible. Firstly, in the current treatment of severe patients, the rates of invasive mechanical ventilation and extracorporeal membrane oxygenation (ECMO) have been low, ranging from 2% to 5%, and the outcome has been poor.[2] Indeed, most of these patients had preexisting heart failure. Left ventricular assist device (LVAD) plus ECMO could be placed early if the pneumonia progresses rapidly and is associated with reduced ejection fraction and signs of heart failure.

Acute lung injury is the leading cause of death by other coronavirus, while multiple organ failure caused by a hyper-inflammation response appears to be the predominant cause of death in COVID-19. Selective cytokine blockade, such as IL-6 blockade, has been a potential treatment option. Moreover, continuous renal replacement therapy (CRRT) not only protects the kidneys, but also regulates the volume, corrects the fluid overload and helps to maintain hemodynamic stability in treating critical cases of COVID-19. However, considering the current low usage rate (1.5%−9%)[2] of CRRT, serum cytokine may continue to attack multi-organs.

Hu et al.[5] report a case of fulminant myocarditis. The use of methylprednisolone to suppress the inflammation and intravenous immunoglobulin to regulate the immune status proved to be effective. We do not recommend large doses of glucocorticoid due to its adverse side-effect and poor prognosis in non-severe patients. However, a low dose of dexamethasone and immunoglobulin is preferable when acute myocarditis is accompanied by unstable hemodynamics or shock.

Current management protocols need to incorporate detection, monitoring and treatment of the cardiovascular effects in severe COVID-19. Insight may be provided into the treatment of COVID-19 based on the life-saving role of LVAD plus ECMO, blood purification, cytokine blockade, glucocorticoid and intravenous immunoglobulin.

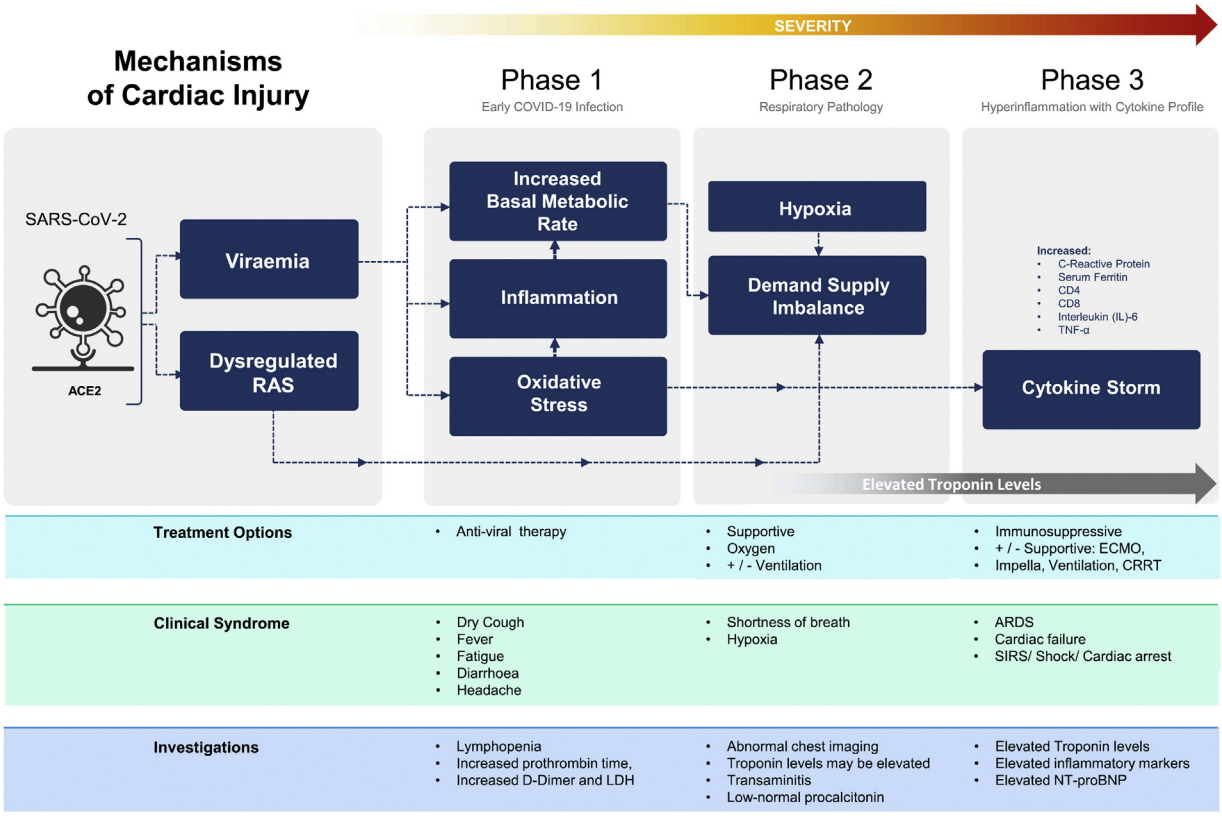

Fig. 1 – The Mechanism, manifestation and treatment underlying myocardial injury in COVID-19. RAS, renin–angiotensin system; TNF-α, tumor necrosis factor-α; LDH, lactic dehydrogenase; ECMO, extracorporeal membrane oxygenation; CRRT, continuous renal replacement therapy; ARDS, acute respiratory distress syndrome; SIRS, systemic inflammatory response syndrome; NT-proBNP, N-terminal pro-brain natriuretic peptide.

Conflict of interest statement

We declare no competing interests.

REFERENCES

1. Yang C, Jin Z. An acute respiratory infection runs into the most common noncommunicable epidemic-covid-19 and cardiovascular diseases. JAMA Cardiol 2020;5:743–4.
2. Wei Z, Geng Y, Huang J, et al. Pathogenesis and management of myocardial injury in coronavirus disease 2019. Eur J Heart Fail 2020;22:1994–2006.
3. Shi S, Qin M, Shen B, et al. Association of cardiac injury with mortality in hospitalized patients with covid-19 in Wuhan, China. JAMA Cardiol 2020;5:802–10.
4. Xu Z, Shi L, Wang Y, et al. Pathological findings of COVID-19 associated with acute respiratory distress syndrome. Lancet Respir Med 2020;8:420–2.
5. Hu H, Ma F, Wei X, Fang Y. Coronavirus fulminant myocarditis saved with glucocorticoid and human immunoglobulin. Eur Heart J 2020 ehaa190.

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