ESC European Society of Cardiology

European Heart Journal (2020) **41**, 1855
doi:10.1093/eurheartj/ehaa333

Immune-mediated mechanism in coronavirus fulminant myocarditis

Xin Wei, Yuan Fang, and Hongde Hu *

Department of Cardiology, West China Hospital, Sichuan University, 37 Guoxue Alley, Chengdu, 610041, PR China

Online publish-ahead-of-print 22 April 2020

This Commentary refers to: 'Coronavirus fulminant myocarditis saved with glucocorticoid and human immunoglobulin', by H. Hu *et al.*, doi: 10.1093/eurheartj/ehaa190.

We are pleased to share our point of view on the case presented by Hu *et al.*[1] The pathogenesis of viral myocarditis includes: (i) inflammatory damage mediated by overactivation of the autoimmune system; (ii) direct damage by the circulating virus to the heart; and (iii) secondary causes such as fever, hypoxia, etc.[2,3]

Most cases of fulminant myocarditis are sporadic, suggesting that immune over-reaction is the decisive mechanism. Moreover, due to the patient's good response to glucocorticoid and i.v. immunoglobulin therapy, presumably immune-mediated inflammatory injury is still the main cause of the myocarditis. The patient improved rapidly after treatment.

According to previous literature, influenza virus, Middle East respiratory syndrome (MERS), and other viruses have been reported to be able to directly damage the heart, while there is no clear evidence of SARS-CoV-2 causing direct damage to the heart as yet.[4] Endomyocardial biopsy (EMB) was not performed according to the indications of the ACC and ESC.[5] Therefore, there is no evidence to support that the virus can cause direct damage to the myocardium. Damage to the myocardium caused by the circulating virus may be involved in the pathogenesis at the early stage of disease onset.

Fever, hypoxia, coronary artery factors, and other factors can also be excluded in this case.

We deduce that immune-mediated inflammatory injury is the main mechanism of this coronavirus fulminant myocarditis, viral infection is the initiating factor of myocarditis, and other secondary factors are unlikely to cause myocardial damage. Although this case is not a SARS-CoV-2 infection, it has some value for the future study on the mechanisms of myocardial damage in COVID-19 patients.

Funding

National Key R&D Program of China. Award number: 2017YFC1307800.

Conflict of interest: none declared.

References

1. Hu H, Ma F, Wei X, Fang Y. Coronavirus fulminant myocarditis saved with glucocorticoid and human immunoglobulin. *Eur Heart J* 2020;doi: 10.1093/eurheartj/ehaa190.
2. Madjid M, Safavi-Naeini P, Solomon SD, Vardeny O. Potential effects of coronaviruses on the cardiovascular system: a review. *JAMA Cardiol* 2020; doi: 10.1001/jamacardio.2020.1286.
3. Channappanavar R, Perlman S. Pathogenic human coronavirus infections: causes and consequences of cytokine storm and immunopathology. *Semin Immunopathol* 2017;**39**:529–539.
4. Su S, Wong G, Shi W, Liu J, Lai ACK, Zhou J, Liu W, Bi Y, Gao GF. Epidemiology, genetic recombination, and pathogenesis of coronaviruses. *Trends Microbiol* 2016; **24**:490–502.
5. Cooper LT, Baughman KL, Feldman AM, Frustaci A, Jessup M, Kuhl U, Levine GN, Narula J, Starling RC, Towbin J, Virmani R; American Heart Association; American College of Cardiology; European Society of Cardiology. The role of endomyocardial biopsy in the management of cardiovascular disease: a scientific statement from the American Heart Association, the American College of Cardiology, and the European Society of Cardiology. *Circulation* 2007;**116**:2216–2233.

* Corresponding author. Tel: +86 18980601232, Email: huhongde@vip.163.com