# Letters

## COMMENT & RESPONSE

**In Reply** Myocardial injury as indicated by elevated serum troponin levels has been detected in many patients with coronavirus disease 2019 (COVID-19), with significant differences having been noted between patients who died and survived.[1] Our recent study suggested that patients with underlying cardiovascular disease (CVD) were more prone to experience myocardial injury and faced a higher risk of death during the course of COVID-19 compared with patients without underlying CVD.[1] For patients with underlying CVD, such as hypertension, coronary artery disease, and cardiomyopathy, viral infection can further damage myocardial cells possibly through several mechanisms, including direct damage by virus, systemic inflammatory response, destabilized coronary plaque, and aggravated hypoxia.

The exact pathophysiological mechanism underlying myocardial injury caused by COVID-19 is not fully understood; important causes may be via direct damage of myocardial cells by the virus and by the systemic inflammation. In our recent study,[1] plasma troponin T levels exhibited a significant positive linear correlation with plasma high-sensitivity C-reactive protein levels, indicating that myocardial injury may be closely associated with inflammatory pathogenesis during the progress of disease. Laboratory parameters in our study showed that the level of lymphocytes was reduced in most patients with COVID-19. This suggests that viral infection may mainly act on lymphocytes, especially T lymphocytes, similar to the situation of severe acute respiratory syndrome coronavirus (SARS-CoV), notably because SARS-CoV-2 and SARS-CoV are highly homologous in genome. Recent data showed that 52% of patients infected with COVID-19 had elevated interleukin 6 levels and 86% had elevated C-reactive protein levels.[2] This indicates a significant inflammatory state in patients with COVID-19. Huang et al[3] highlighted that in patients with COVID-19, the imbalance of Th1 and Th2 responses resulted in a cytokine storm, which may contribute to myocardial injury.

Accordingly, as for the issue in question raised by Giannopoulos et al whether we could use treatments specifically targeting the COVID-19–associated inflammation storm to improve outcomes, as known to all, no approved preventive vaccines or specific therapies are available for COVID-19 at present. However, the exaggerated inflammatory cell infiltration and cytokine release after infection may cause reduction in coronary blood flow, decreases in oxygen supply, destabilization of coronary plaque, and microthrombogenesis. Hence, we speculated that patients with COVID-19 might benefit from treatments specifically targeting the COVID-19–associated inflammation storm. Indeed, in addition to antiviral medications, numerous immune-modulating medications to regulate inflammatory response are currently being investigated in patients with COVID-19. In clinical practice, glucocorticoid is generally used to inhibit severe inflammation in high-risk patients. Besides, chloroquine, which has been used as an antimalarial agent, blocks virus infection by increasing the endosomal pH required for virus/cell fusion and has been demonstrated in vitro to have inhibitory activity in SARS-CoV-2.[4] Yet, for patients with COVID-19 experiencing an inflammation storm, more evidence is needed to verify the effectiveness of glucocorticoid and immunosuppressive therapy. For us, it may be reasonable to triage patients with COVID-19 according to the presence of underlying CVD and evidence of myocardial injury for prioritized treatment and particularly for treatments specifically targeting on inflammation.

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