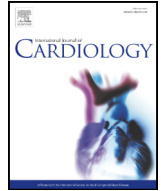
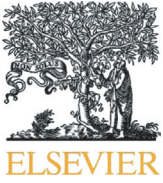




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Letter to the editor

## COVID-19 myocardial injury: We have much more to discover

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We read with great interest the work by Deng Q. et al. who provide valuable, front-line data regarding myocardial injury in Coronavirus Disease – 19 (COVID-19) hospitalized patients [1]. Myocardial involvement has been recognized as a component of COVID-19 in a noteworthy proportion of patients (ranging up to 40% of the cases) and has been related to mortality. Indeed, cardio-protection in COVID-19 - regarding both myocardial necrosis and inflammation - is a target of ongoing research [2]. Do myocardial necrosis' biomarkers act as an unpleasant bystander who provides clinicians an early sign of adverse outcomes or are they indicative of direct myocardial involvement? Deng. et al. [1] suggest, that myocardium is mainly hit in a secondary fashion (i.e. oxygen demand/supply in myocardial cell level). Of course, lack of biopsy and/or MRI data in their cohort is noted as limitation for this argument; however, authors supported this viewpoint on already published autopsy reports [1]. Still, our knowledge on the field changes rapidly. To date, a series of cases proving diverse mechanisms of direct myocardial injury has been published: histological proved virus-positive SARS-CoV-2 myocarditis [3], virus-negative lymphocytic myocarditis associated with SARS-CoV-2 respiratory infection (clinically presented as reverse-takotsubo) [4] and true takotsubo syndrome [5]. These reports do not

contradict (but, indeed, are in line) with the findings of Deng et al. [1], but COVID-19 being a new entity for clinicians and researchers, they do remind us of the Socratic paradox: "The only thing I know is that I know nothing".

### Declaration of competing interest

The authors report no relationships that could be construed as a conflict of interest.

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