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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Letter to the Editor

Letter to the editor in response to Buja et al. 2020

We read with great interest Buja et al.'s [1] article summarizing the results of 23 autopsies in patients with coronavirus disease 2019 (COVID-19), including 3 previously unreported cases in Houston, Texas. All subjects had comorbidities and evidence of cardiac pathology, most notably cardiomegaly (57%). Histopathological evaluation found that 8/23 (35%) cases had individual cardiomyocyte injury without inflammatory infiltrates, 3/23 (13%) had lymphocytic epicarditis/pericarditis, and 1/23 (4%) had myocarditis.

Discrepancies exist between Buja et al.'s [1] findings and the latest autopsy series in patients with COVID-19. A recent review by Edler et al. [2] reported the results of 80 autopsies carried out in Germany between March 20 and April 18, 2020 on patients infected with SARS-CoV-2. The first 12 cases were histologically evaluated: 10 of these (83%) were known to have pre-existing cardiovascular disease. In comparison to Buja et al.'s [1] findings, there were no cases of lymphocytic epicarditis/pericarditis in Edler et al.'s [2] series. However, one case (8%) of infiltrative myocarditis was reported by Edler et al. [2], in a 71-year-old obese (BMI 36.8 kg/m^2) male with cardiac insufficiency. Unfortunately, the medical and demographic history of the patient with myocarditis in Buja et al.'s [1] study was unknown; however, we appreciate that patients with epicarditis were described in detail (all obese, males with underlying cardiovascular disease). Furthermore, Buja et al. [1] noted that elevated serum troponin is a pathological correlate of virus-related myocarditis. This finding is supported by Lippi et al.'s [3] meta-analysis, which established that cardiac troponin I (cTnI) is significantly elevated in COVID-19 patients with severe disease. Measuring cardiac enzymes, including troponin and B-type natriuretic peptide, may prove useful in screening for cardiac injury and predicting an adverse outcome, thereby guiding management. It is thought that myocardial injury is a complication of COVID-19 due to inflammatory effects (cytokine storm) and whether or not direct intracellular SARS-CoV-2 infection occurs is contentious. Alternatively, cardiac injury may be the result of coronary artery ischemia in patients with pre-existing disease or a consequence of increased coronary artery thrombosis. It would be valuable for all future autopsy series' to detail the cardiovascular history and histopathological profiles of patients with myocarditis, and test patients for the SARS-CoV-2 genome in the myocardium.

Fox et al. [4] carried out autopsies on ten African American patients who tested positive for SARS-CoV-2, with the aim of investigating the pathogenesis of SARS-CoV-2 on different organs. They remarked that the myocardium showed areas of dispersed myocyte necrosis, a finding that was demonstrated by Buja et al. [1]. Fox et al. [4] also observed a rise in cardiac troponin I in 6/10 (60%) cases, supporting Buja et al.'s [1] finding that a rise in troponin levels correlates to cardiomyocyte damage. However, it should be noted that Fox et al. [4] did not find lymphocytic infiltrates or significant evidence of viral myocarditis. While Buja et al. [1] alerted us to the comorbidities that each patient possessed, we were only given further information about the cardiovascular history and function of the second case from Houston, which included their left ventricular ejection fraction and electrocardiogram reports. This makes it difficult to discern whether the cardiac necrosis present was due to COVID-19 or other causes. While it was not possible for Buja et al. [1] to provide information on the cardiovascular function of each patient before they contracted COVID-19, it would be of benefit for future studies to include these data and describe the course of disease in patients with fewer comorbidities.

Schaller et al. [5] conducted autopsies on 10 patients in Germany with proven COVID-19 infection, with an emphasis on discerning the cardiopulmonary effects. These autopsies demonstrated mild lymphocytic myocarditis in 4/10 (40%) cases and signs of epicarditis in 2/10 (20%) cases. While this supports the findings by Buja et al. [1], it is important to note that Schaller et al. [5] could not attribute these myoepicardial changes to myocarditis rather than systemic inflammation. Therefore, looking for markers of systemic inflammation in their cohort may be of benefit.

In conclusion, further research is required to elucidate the cardiac involvement in patients with COVID-19. The establishment of rigorous safety protocols has paved the way for additional studies to be carried out and data from larger sample sizes is necessary to further understanding of the pathogenesis of COVID-19.

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Dilen Parmar*, Tanaya Gandhi
University College London Medical School, 74 Huntley Street, Bloomsbury, London, WC1E 6DE, United Kingdom

Mahmood Ahmad, Ali Kirresh
Royal Free London NHS Foundation Trust, Pond Street, Hampstead, London, NW3 2QG, United Kingdom

George Seligmann
University College London Medical School, 74 Huntley Street, Bloomsbury, London, WC1E 6DE, United Kingdom

*Corresponding author. University College London Medical School, 74 Huntley Street, Bloomsbury, London, WC1E 6DE, United Kingdom
E-mail address: dilen.parmar@nhs.net (D. Parmar)
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