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.

rates of rehospitalisation and all-cause mortality compared to patients with ACEi/ARB at discharge only (0.6% vs 0.7%, p=0.003; 0.9% vs 3.8%, p<0.0001, respectively). Patients continuing ACEi/ARB therapy to 12 months had fewer deaths than those who had discontinued therapy at 6 months (0.3% vs 0.6%, p=0.012).

Conclusion: Treatment with ACEi/ARB therapy on discharge and their persistence beyond 6 months beyond may reduce the rate of rehospitalisations and death in patients after myocardial infarction. Mortality benefits appear magnified when persistence extends to 12 months.

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Induced Pluripotent Stem Cell-Derived Models of Spontaneous Coronary Artery Dissection

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Spontaneous coronary artery dissection (SCAD) is the cause of up to 1/3 of myocardial infarcts in women under 50 years old, with patients often presenting with no traditional cardiovascular risk factors. The dissection event is characterised by detection of an intramural haematoma due to an intima tear or rupture of the vasa vasorum. SCAD is poorly understood, due largely to the limited studies examining genetic risk variants, and the lack of disease models, and the inability to obtain coronary artery biopsies from survivors. Our team has have developed the first biobank of SCAD induced pluripotent stem cells (iPSCs), including cell lines generated from SCAD survivors, and age- and sex-matched controls. To study the cellular mechanisms underlying the pathophysiology of SCAD, we have used this cohort. We have generated iPSC-derived vascular smooth muscle cells (VSMC; TAGLN+, αSMA+, CNN1+, SM-MHC+) and endothelial cells (CD31+, VWF+, VE Cadherin+), to investigate disease phenotypes. We have identified differences in expression of VSMC cytoskeletal proteins in VSMCs, as well as significantly increased rates of cell proliferation (mean SCAD doubling times compared to controls, calculated from Incucyte live cell tracking). Together these findings suggest perturbations in cellular activation states may predispose patients to SCAD. These iPSCs represent an important tool for understanding SCAD pathophysiology and will be essential in future drug screening.

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Is Myocarditis a Disease of the COVID-19 Pandemic? A Comparison of COVID-19 vs COVID-19 Vaccine-Associated Myocarditis

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Background: Myocarditis is of growing concern since the COVID-19 pandemic, particularly with the earlier COVID-19 variants (e.g. delta) and the concern with mRNA COVID-19 vaccine use.

Aim: To compare the incidence of myocarditis among hospitalised populations, before and during the COVID-19 pandemic, in comparison to patients with confirmed COVID-19 and those who received mRNA COVID-19 vaccine.

Methods: Retrospective analysis of medical records from 2018 to 2022 February of hospitalised patients with an ICD-10 code of myocarditis from a busy tertiary hospital in Australia.

Results: Annualised incidence (per 100,000 hospital admissions) of myocarditis was calculated: 3.06 (2018), 4.96 (2019), 8.72 (2020), 33.03 (2021, estimate), 0 (until February 2022). Annualised incidence of myocarditis in COVID-19 patients peaked in 2021 at 184.84 (2021). 2,353 patients with COVID-19 were included. 48 patients with myocarditis were included (age 41.8±20.5, 54% male). 3 patients had COVID-19 associated myocarditis; all of these patients did not receive COVID-19 vaccine. 9 patients (7 had Pfizer, 2 had Moderna, age 24.3±6.3, 78% male) had COVID-19 vaccine associated myocarditis. 36 patients had myocarditis not related to COVID-19 or COVID-19 vaccine.

Conclusion: Based on our single-centre data, the incidence myocarditis has increased since 2018, especially from 2020 to 2021 during the COVID-19 pandemic. At its peak in 2021, the majority of the myocarditis in the hospitalised population was neither related to active COVID-19 infection nor the mRNA vaccine.

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