Cardiovascular outcomes for people hospitalised with COVID-19 in Australia, and the effect of vaccination: an observational cohort study

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The known: Information regarding the frequency of cardiovascular events in people hospitalised with COVID-19, and the impact of vaccination, is limited.

The new: Troponin levels were elevated in 37% of patients with COVID-19 assessed during admission to one of 21 Australian hospitals, but cardiovascular outcomes were infrequent and not influenced by vaccination. Those who had received one or more COVID-19 vaccine doses were less likely to die in hospital or to be intubated.

The implications: There risk of cardiovascular events for people hospitalised with COVID-19 is small but clinically significant. Our findings highlight the value of COVID-19 vaccination for reducing in-hospital mortality.

Abstract

Objectives: To assess the frequency of clinical cardiovascular outcomes for people hospitalised with coronavirus disease 2019 (COVID-19), and the impact of vaccination.

Study design: Observational cohort study.

Setting, participants: All index admissions of adults with laboratory-confirmed COVID-19 to 21 hospitals participating in the Australian Cardiovascular COVID-19 Registry (AUS-COVID), 4 September 2020 – 11 July 2022.

Main outcome measures: Frequency of elevated troponin levels, new arrhythmia, new or deteriorating heart failure or cardiomyopathy, new pericarditis or myocarditis, new permanent pacemaker or implantable cardioverter–defibrillator, and pulmonary embolism. Secondary outcomes: impact of COVID-19 vaccination on likelihood of in-hospital death, intubation, troponin elevation, and clinical cardiovascular events.

Results: The mean age of the 1714 people admitted to hospital with COVID-19 was 60.1 years (standard deviation, 20.6 years); 926 were men (54.0%), 181 patients died during their index admissions (10.6%), 299 required intensive care (17.4%). Thirty-eight patients (2.6%) developed new atrial fibrillation or flutter, 27 (2.6%) had pulmonary embolisms, new heart failure or cardiomyopathy was identified in 13 (0.9%), and pre-existing cardiomyopathy or heart failure was exacerbated in 21 of 110 patients (19%). Troponin was elevated in 369 of the 986 patients for whom it was assessed (37.4%); in-hospital mortality was higher for people with elevated troponin levels (86, 23% *v* 23, 3.7%; $P < 0.001$). The COVID-19 vaccination status of 580 patients was known (no doses, 232; at least one dose, 348). The likelihood of in-hospital death (adjusted odds ratio [aOR], 0.38; 95% confidence interval [CI], 0.18–0.79) and intubation (aOR, 0.30; 95% CI, 0.15–0.61) were lower for people who had received at least one vaccine dose, but not the likelihood of troponin elevation (aOR, 1.44; 95% CI, 0.80–2.58) or clinical cardiovascular events (aOR, 1.56; 95% CI, 0.59–4.16).

Conclusions: Although troponin levels were elevated in a considerable proportion of people hospitalised with COVID-19, clinical cardiovascular events were infrequent, and their likelihood was not influenced by vaccination. COVID-19 vaccination, however, was associated with reduced likelihood of in-hospital death and intubation.

Trial registration: Australian and New Zealand Clinical Trials Registry, ACTRN12620000486921 (prospective).

Coronavirus disease 2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), rapidly spread from China from late 2019, and the World Health Organization declared a pandemic in March 2020.[1] The predominant manifestations of COVID-19 are respiratory, but it can also can affect the heart and vascular tissues by binding ACE2 (angiotensin-converting enzyme 2), the main cell entry point bound by the SARS-CoV-2 spike protein.[2] Cardiovascular implications of COVID-19 include myocarditis, cardiomyopathy, myocardial injury, and arrhythmias.[2]

COVID-19 vaccines dramatically altered the course of the pandemic and prevented more than 14 million deaths within twelve months of their introduction.[3] The impact of vaccination on the numbers of COVID-19-related hospitalisations and deaths has been described, but their impact on cardiovascular outcomes has not been systematically assessed.[4,5]

Australia provides a unique opportunity for the unbiased study of the cardiovascular effects of COVID-19 because of its geographic isolation, social distancing laws that restricted the number of SARS-CoV-2 infections, and a relatively unchallenged health care system.[6] The aim of our multicentre observational study was a comprehensive analysis of cardiovascular outcomes for people hospitalised with COVID-19 and of the impact of vaccination on these outcomes.

Methods

The Australian Cardiovascular COVID-19 Registry (AUS-COVID; https://www.aus-covid.com) is an observational cohort study in 21 hospitals in Queensland, New South Wales, Victoria, and Western Australia, established in April 2020 to evaluate the cardiovascular effects of COVID-19 in people hospitalised with the disease in Australia. Comprehensive information regarding medical history, medications, serologic, echocardiographic, and imaging findings, and cardiovascular outcomes for each patient are collected in a standardised manner from electronic medical records, de-identified, and entered into a Research Electronic Data Capture (REDCap) database. Our reporting of the study adheres to the Strengthening and Reporting of Observational studies in Epidemiology (STROBE) statement.[7] The study was prospectively registered with the Australian and New Zealand Clinical Trials Registry (ACTRN12620000486921; 17 April 2020).

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Study population

Our analysis includes all index hospitalisations of adults (18 years or older) with laboratory-confirmed COVID-19 recorded in the AUS-COVID registry (admission dates: 4 September 2020 – 11 July 2022); patients transferred from participating to other hospitals were not included because their final outcomes were unknown.

Outcomes

The primary outcomes were the frequency of cardiovascular events, including troponin elevation beyond the upper limit of normal according to the site-specific assay, documented new arrhythmia (atrial fibrillation or flutter, high grade atrioventricular [AV] block, torsades de pointes), new or deteriorating heart failure or cardiomyopathy, new pericarditis or myocarditis, new permanent pacemaker or implantable cardioverter–defibrillator, and pulmonary embolism. Secondary outcomes were the impact of COVID-19 vaccination on in-hospital mortality, intubation, troponin elevation, and clinical cardiovascular events (composite outcome: new onset atrial fibrillation or flutter, high grade AV block, new cardiomyopathy or heart failure, pericarditis, myocarditis or myopericarditis, pulmonary embolism).

Statistical analysis

Continuous variables are summarised as means with standard deviations (SDs), categorical variables as proportions (missing data omitted from denominators). The statistical significance of differences in proportions was assessed in Pearson χ^2 (cell sizes of five or more) or Fisher exact tests (cell sizes lower than five); the significance inter-group differences in means was assessed in independent samples *t* tests for normally distributed continuous variables. Relationships between COVID-19 vaccination status and secondary outcomes were assessed in multivariable binary logistic regression analyses. Vaccination was included as the covariate of interest, and other covariates were included if $P < 0.1$ in univariable analyses; covariates for which the cell size was zero were excluded from the multivariate analysis. $P < 0.05$ was deemed statistically significant; to mitigate the impact of multiple testing with respect to the four secondary outcomes, we employed Bonferroni correction (ie, $P < 0.0125$). We estimated that a sample size of 865 patients for primary outcome assessment would be required for detecting a two percentage point difference in a parameter with a population proportion of 10% ($\alpha = 0.05$). Statistical analyses were undertaken in SPSS Statistics 29.0.0.0 (241) (IBM).

Ethics approval

The study was approved by the Northern Sydney Local Health District Human Research Ethics Committee (HREC 2020/ETH00732), which granted a waiver of the requirement for individual patient consent.

Results

Index admissions for 1714 patients to 21 Australian hospitals with laboratory-confirmed COVID-19 were identified in the AUS-COVID registry. Their mean age was 60.1 years (SD, 20.6 years); 926 were men (54.0%), 68 were health care workers (4.0%), and 152 were nursing home residents (8.9%) (Box 1). A total of 181 patients died during their index admissions (10.6%), 299 required intensive care (17.4%), 148 required intubation (8.6%), and 62 were transferred to non-participating hospitals (outcomes unknown) (3.6%).

Arrhythmias

During hospitalisation, 38 people (2.6%) developed new atrial fibrillation or flutter, three of whom underwent successful direct current cardioversion. High grade AV block was detected in five people (0.3%), all of whom survived to discharge; three people had Mobitz II AV block, one had third degree AV block, and one had both Mobitz II and third degree AV block. Dual chamber permanent pacemakers were implanted into three patients (0.2%); no patients received implantable cardioverter–defibrillators. Temporary pacing wire was inserted into one person with bradycardia–tachycardia syndrome and non-ST segment elevation myocardial infarction; they subsequently died. No patients had torsades de pointes.

Cardiomyopathy, heart failure

De novo cardiomyopathy or heart failure was identified in 13 patients (0.9%), six of whom died. Five of seven people in whom electrocardiography was undertaken had left ventricular ejection fractions of less than 50%, and the *N*-terminal prohormone of B-type natriuretic peptide (NT-proBNP) level was elevated in one patient for whom electrocardiography was not undertaken; the diagnosis of heart failure in five patients was based on clinical and radiological information alone. Takotsubo cardiomyopathy was diagnosed in one patient (0.1%). Pre-existing cardiomyopathy or heart failure was exacerbated in 21 of 110 patients (19%), of whom 13 died; none had undergone echocardiography, and the NT-proBNP or B-type natriuretic peptide (BNP) level was elevated in four of eight patients in whom it was measured (NT-proBNP > 1800 ng/L, BNP > 500 ng/L).

Pericarditis, myocarditis, myopericarditis

Pericarditis was diagnosed in two patients (0.1%), and myocarditis or myopericarditis in five (0.3%); echocardiography identified moderate pericardial effusion in two of these patients. One patient (0.1%) underwent cardiac magnetic resonance imaging (MRI); none underwent biopsy, pericardial aspiration, or pericardial window. All patients with pericarditis, myocarditis, or myopericarditis survived to discharge.

Pulmonary embolism

During hospitalisation, 27 patients (2.6%) were diagnosed with pulmonary embolisms, five of whom died. Sixteen of 299 patients admitted to intensive care (5.4%) had pulmonary embolisms; one was already receiving a direct oral anticoagulant, none of these patients was already receiving warfarin.

Troponin level elevation

Troponin was assessed at least once in 986 patients (57.5%); the level was elevated in 369 (37.4%): severe elevation (more than five times the upper limit of normal) in 104 patients and mild elevation (one to less than five times the upper limit of normal) in 265 patients. In-hospital mortality was higher for people with elevated troponin levels (86, 23% *v* 23, 3.7%; $P < 0.001$); mortality was higher for people with severely elevated troponin levels than for those with mild elevations (51 of 104, 49% *v* 35 of 265, 13%). Electrocardiography had been undertaken in 53 people with elevated troponin levels (14%); four had regional wall motion abnormalities. Exercise stress test results for one patient with elevated troponin level were negative, as was a myocardial

1 Baseline characteristics of 1714 people admitted to 21 AUS-COVID registry hospitals with laboratory-confirmed COVID-19, 4 September 2020 – 11 July 2022

Characteristic	All patients	Patients with known vaccination status	
		Vaccinated*	Unvaccinated
Number of patients	1714	348	232
Age (years), mean (SD)	60.1 (20.6)	66.5 (18.4)	53.3 (19.2)
Gender (men)	926 (54.0%)	195 (56.0%)	127 (54.7%)
Health care workers	68 (4.0%)	4 (1.2%)	0
Nursing home residents	152 (8.9%)	9 (2.6%)	1 (0.4%)
Other medical conditions			
Hypertension	722 (44.4%)	197 (57.6%)	66 (31.6%)
Coronary artery disease	183 (11.3%)	62 (18.1%)	16 (7.7%)
Heart failure or cardiomyopathy	110 (6.8%)	28 (8.2%)	7 (3.3%)
Atrial fibrillation or flutter	169 (10.4%)	52 (15.2%)	16 (7.7%)
Permanent pacemaker/implantable cardioverter–defibrillator	59 (3.6%)	16 (4.7%)	5 (2.4%)
Severe valvular disease	42 (2.6%)	10 (2.9%)	2 (1.0%)
Stroke or transient ischaemic attack	98 (6.0%)	22 (6.4%)	9 (4.3%)
Hypercholesterolemia	466 (28.7%)	130 (38.0%)	49 (23.4%)
Diabetes mellitus	389 (23.9%)	114 (33.3%)	42 (20.1%)
Peripheral arterial disease	23 (1.4%)	7 (2.0%)	0
Currently or recently smoked (past year)	134 (8.3%)	32 (9.4%)	21 (10.0%)
Chronic obstructive pulmonary disease	124 (7.6%)	33 (9.6%)	10 (4.8%)
Asthma	192 (11.8%)	42 (12.3%)	25 (12.0%)
Chronic kidney disease (eGFR < 60 mL/min/1.73 m^2)	131 (8.1%)	37 (10.8%)	15 (7.2%)
No information available	90	6	23
COVID-19 vaccination status (as documented)			
Unknown	269 (31.7%)	—	—
Single dose	106 (12.5%)	106 (30.5%)	—
Two doses	204 (24.0%)	204 (58.6%)	—
Three or more doses	38 (4.5%)	38 (10.9%)	—
No doses	232 (27.3%)	—	—
No information available	865	—	—
COVID-19 vaccine type (for 348 people who received at least one vaccine dose)			
Unknown	—	232 [48.2%]	—
Vaxzevria (AstraZeneca)	—	162 [33.7%]	—
Comirnaty (Pfizer)	—	95 [19.8%]	—
Spikevax (Moderna)	—	5 [1.0%]	—

COVID-19 = coronavirus disease 2019; eGFR = estimated glomerular filtration rate. * At least one COVID-19 vaccine dose. ◆

perfusion (MIBI) scan in another. Computed tomography coronary angiography was not undertaken for any patients with elevated troponin levels. Coronary angiography was undertaken in nine people with elevated troponin levels; one had triple vessel disease and subsequently underwent coronary artery bypass graft surgery, and three had ST-segment elevated myocardial infarctions, of whom one with thrombolysis was managed and survived, and two were deemed unsuitable for coronary angiography or thrombolysis (one died, one survived to discharge).

Cardiovascular outcomes by COVID-19 vaccination status

Of the 580 patients with known vaccination status, 232 had received no COVID-19 vaccine doses (40.0%) and 348 had received at least one dose (60.0%). The mean age of those who had been

2 Outcomes for 580 people with known COVID-19 vaccination status admitted to 21 AUS-COVID registry hospitals with PCR-confirmed COVID-19, 4 September 2020 – 11 July 2022

	Vaccination doses					Odds ratio (95% CI)*	
Vaccination status	Three or more	Two	One	Vaccinated (at least one)	Unvaccinated (none)	Unadjusted	Adjusted
In-hospital mortality	2/38 (5%)	16/204 (8%)	6/106 (6%)	24/348 (7%)	19/232 (8%)	0.83 (0.44–1.55)	0.38 (0.18–0.79)†
Intubation	1/38 (3%)	9/204 (4%)	7/106 (7%)	17/348 (5%)	22/232 (10%)	1.43 (0.80–2.56)	0.30 (0.15–0.61)‡
Troponin elevation§	13/24 (54%)	82/130 (63%)	20/62 (42%)	115/216 (53%)	41/141 (29%)	2.78 (1.77–4.36)	1.44 (0.80–2.58)¶
Clinical cardiovascular events**	2/26 (7%)	8/153 (5%)	5/84 (6%)	15/263 (5%)	6/182 (3%)	1.73 (0.66–4.54)	1.56 (0.59–4.16)††
New onset atrial fibrillation or flutter	1/28 (4%)	5/171 (3%)	2/91 (2%)	8/290 (3%)	3/193 (2%)	—	—
High-grade atrioventricular block	1/38 (3%)	0/204	0/106	0/348	1/232 (< 1%)	—	—
New cardiomyopathy or heart failure	1/34 (3%)	0/182	0/98	1/314 (< 1%)	0/202	—	—
Pericarditis, myocarditis or myopericarditis	0/38	0/204	1/106 (1%)	1/348 (< 1%)	2/232 (1%)	—	—
Pulmonary embolism	0/38	5/204 (2%)	2/106 (2%)	7/348 (2%)	1/232 (< 1%)	—	—

* Vaccinated *v* unvaccinated. † Adjusted for vaccination status, age, coronary artery disease, heart failure or cardiomyopathy, atrial fibrillation or flutter, stroke or transient ischaemic attack, hypertension, hypercholesterolemia, chronic kidney disease. ‡ Adjusted for vaccination status, age, hypertension, hypercholesterolemia, diabetes. § 216 vaccinated patients and 141 unvaccinated patients for whom troponin measurements during hospitalisation were recorded. ¶ Adjusted for vaccination status, age, sex, coronary artery disease, heart failure or cardiomyopathy, atrial fibrillation or flutter, severe valvular disease, stroke or transient ischaemic attack, hypertension, hypercholesterolemia, diabetes mellitus, chronic obstructive pulmonary disease, chronic kidney disease. ** 263 vaccinated patients and 182 unvaccinated patients without pre-existing diagnoses of atrial fibrillation or flutter, heart failure, or cardiomyopathy. †† Adjusted for vaccination status, sex, diabetes mellitus. ◆

vaccinated was higher (66 [SD, 18.4] *v* 53.3 [19.2] years), and the proportions with cardiovascular risk factors and established cardiovascular disease were larger (Box 1). In multivariable analyses, the likelihood of death (adjusted odds ratio [aOR], 0.38; 95% confidence interval [CI], 0.18–0.79) and intubation (aOR, 0.30; 95% CI, 0.15–0.61) were lower for people who had received at least one dose, but not those of troponin elevation (aOR, 1.44; 95% CI, 0.80–2.58) or clinical cardiovascular events (aOR, 1.56; 95% CI, 0.59–4.16) (Box 2; Supporting Information, tables 1 to 4).

Discussion

We report findings for a large cohort of Australian adults hospitalised with COVID-19 for whom cardiovascular outcomes and vaccination status had been systematically recorded. Our study included patients from periods during which the Alpha (B.1.1.7), Delta (B.1.617.2), and Omicron (B.1.1.529) SARS-CoV-2 variants were predominant in Australia, and from before and after the introduction of COVID-19 vaccines (22 February 2021).[8] In an earlier article, we reported that clinical cardiovascular events were infrequent in a smaller cohort of people hospitalised with COVID-19 during a period in which the Alpha (B.1.1.7) SARS-CoV-2 variant was predominant and prior to the availability of COVID-19 vaccines.[9] Our new study confirmed that the overall incidence of clinical cardiovascular outcomes in people hospitalised with COVID-19 was low. Elevated troponin levels were found in a large proportion of patients, and were associated with greater likelihood of in-hospital death. People who had received at least one COVID-19 vaccine dose were less likely to die in hospital or require intubation, but vaccination status did not influence the likelihood of clinical cardiovascular events or elevated troponin levels.

Arrhythmias

The proportion of patients who developed new atrial fibrillation or flutter (2.6%) was smaller than reported by comparable overseas investigations. In an American study of 27 851 consecutive people without histories of atrial fibrillation hospitalised with COVID-19, 5.4% developed new atrial fibrillation; however, this study also included larger proportions than in our cohort of patients who were admitted to intensive care (with new onset atrial fibrillation: 64.5%; without new onset atrial fibrillation: 27.8%) or required mechanical ventilatory support (with new onset atrial fibrillation: 47.6%; without new onset atrial fibrillation: 15.7%).[10] In the CAPACITY-COVID study, a predominantly European registry study of 3011 people hospitalised with COVID-19, 4.7% developed atrial fibrillation; a larger proportion (27.8%) required intensive care than in our cohort (17.4%).[11]

The pathophysiology of COVID-19-related atrial arrhythmias probably involves a predisposition exacerbated by direct myocardial injury, systemic inflammation, and the adverse cardiovascular effects of targeted COVID-19 treatment. Contributory factors such as hypoxia, hypotension, and elevated intracardiac pressures are more likely in people who require intensive care or mechanical ventilatory support.[10] The higher incidence of atrial fibrillation in other studies might therefore reflect the poorer medical condition of people hospitalised with COVID-19 overseas compared with patients in Australia.

Heart failure or cardiomyopathy

Fewer than 1% of patients (13 people) experienced new heart failure or cardiomyopathy; exacerbation of known heart failure or cardiomyopathy was experienced by 19% of people with such conditions, of whom 62% died (13 people). Other studies

have reported that 1.8%[11] to 5.1%[12] of people hospitalised with COVID-19 experienced de novo heart failure or cardiomyopathy, but the generalisability of these findings is limited because the studies examined small, highly selected patient groups. Most studies, including ours, probably underestimate the rate of heart failure and cardiomyopathy because echocardiography and BNP investigations were not routinely undertaken in people hospitalised with COVID-19, and because these conditions can develop after the patients have been discharged from hospital.

Pericarditis, myocarditis, myopericarditis

The small proportions of patients who developed pericarditis (0.1%) or myocarditis or myopericarditis (0.3%) were similar to those reported by other cohort studies,[11,13] but these findings may underestimate the frequency of these conditions. Only 0.1% of people in our study were assessed with cardiac MRI, despite the number of patients in whom troponin levels were elevated without clear cause.

Pulmonary embolism

A range of complex coagulation abnormalities in some people with COVID-19 portends a hypercoagulable state, and hospitalisation and associated venous stasis increase the risk of venous thromboembolism.[14] Overseas studies have found pulmonary embolisms in 6.2%[15] to 21%[16] of people admitted to intensive care and 2.2% of patients who did not require intensive care.[15] We confirmed that people hospitalised with COVID-19 are at risk of pulmonary embolism, but the relatively small proportion with embolisms (2.6%) again probably reflects the better general condition of Australian patients than those in overseas studies. Moreover, the cited investigations were entirely undertaken before COVID-19 vaccines became available, unlike our study; the impact of vaccination on the risk of pulmonary embolism is therefore unclear.

Troponin elevation

Elevated troponin levels, indicative of myocardial injury, were recorded for 37.4% of patients; this high frequency is consistent with the findings of other studies. In a multicentre study including 6247 people hospitalised with COVID-19 in the United States, troponin levels were elevated in 29% of people within 48 hours of admission.[17] Mechanisms leading to troponin elevation include direct SARS-CoV-2-mediated myocardial injury, oxygen supply–demand mismatch, myocarditis, endothelial dysfunction, and microthrombi formation.[18] Occlusive coronary disease has been reported in people with COVID-19,[19] but it is relatively infrequent; in our study, three patients (0.8%) had ST-segment elevation myocardial infarctions. In-hospital mortality was higher among patients with elevated troponin levels, and was higher with severe than mild troponin level elevation. The prognostic value of myocardial injury (not just type 1 myocardial infarction) and associated factors should be investigated further.

Cardiovascular outcomes by vaccination status

When COVID-19 vaccines were introduced in Australia, health care workers, older adults (including nursing home residents), and adults with certain medical conditions received priority. This may explain the higher mean age, the larger proportion of nursing home residents, and the larger proportions with cardiovascular risk factors and baseline cardiovascular disease in the vaccinated group in our study than among people who had received no vaccine doses. The likelihood of in-hospital death and intubation were nonetheless lower for people who had received at least one dose, supporting the efficacy of vaccination for reducing mortality and disease severity in people with COVID-19.[3,5]

In contrast, vaccination did not influence the likelihood of clinical cardiovascular events or troponin elevation. However, clinical cardiovascular outcomes were relatively infrequent in our study, and some may have been missed because investigations were undertaken only when clinically indicated rather than routinely. The literature on the impact of vaccination on cardiovascular outcomes in people hospitalised with COVID-19 is limited. Despite good vaccination rates in higher income countries, comparing outcomes for vaccinated and unvaccinated people remains relevant given the ongoing need for booster vaccinations to mitigate waning vaccine-elicited and natural immunity, and the possibility that new SARS-CoV-2 variants could emerge.[20,21] Moreover, evidence for longer term effects of COVID-19 on all cardiovascular outcomes is growing, and the impact of vaccination on these late effects requires investigation.[22,23]

Limitations

We included all people admitted with COVID-19 to the participating hospitals, minimising selection bias, and our cohort was the largest for which systematically recorded cardiovascular outcomes and vaccination status have been reported. However, the degree of missing data was a limitation, a consequence of extracting data from electronic medical records and during a pandemic in which normally routine investigations were not always undertaken. Information about vaccination status, ascertained in hospital electronic medical records, was also incomplete. We assumed that all data were missing at random. Further, subclinical complications may not be captured given that investigations were undertaken only when clinically indicated. Similarly, investigations of outcomes such as troponin elevation and myocarditis, including echocardiography and cardiac MRI, were not routinely undertaken because of changes to inpatient clinical practice during the pandemic. Moreover, we report only outcomes until discharge from the index hospitalisation, and outcomes such as cardiomyopathy may not develop until later. This is particularly salient when considering the impact of vaccination on cardiovascular outcomes, as its benefit may be more apparent in the longer term. The overall low frequency of cardiovascular outcomes in our study limited our evaluation of the effect of vaccination on such events. Finally, we could not assess associations between different SARS-CoV-2 variants and cardiovascular outcomes, as the virus was not routinely subtyped.

Conclusion

The overall frequency of clinical cardiovascular outcomes in people hospitalised with COVID-19 was low; however, troponin levels were elevated in a substantial proportion of patients. In-hospital death and intubation were less likely for people who had received at least one COVID-19 vaccine dose, but not clinical cardiovascular outcomes and troponin elevation.

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Supporting Information

Additional Supporting Information is included with the online version of this article.