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

Impact of Myocardial Injury in Hospitalized Patients With COVID-19 in 2 Peak Time Periods

SARS-CoV-2, the etiologic agent in COVID-19, has infected over 185 million people worldwide (1). Prior to widespread immunization, the virus caused significant mortality, despite several pharmacological and nonpharmacological treatment strategies that were developed.

Myocardial injury is prevalent in patients hospitalized with COVID-19 and has been linked to increased in-hospital mortality, although these data have mostly been gathered early in the pandemic (2,3). The aim of this study was to investigate myocardial injury in hospitalized patients with COVID-19 across 2 pre-vaccination peak time periods.

Patients with laboratory-confirmed SARS-CoV-2 infection who were at least 18 years of age and had a troponin measurement within 24 hours of admission were collected from The Mount Sinai Health System in New York, New York, and examined across 2 time periods: March 2020 to June 2020 (Period 1 [P1]) and October 2020 to December 2020 (Period 2 [P2]). Approval was obtained from the Mount Sinai Institutional Review Board. Rank-sum tests were used for continuous variables and chi-square tests were used for categorical variables. Survival event rates were estimated as time from admission to death or discharge. Cox proportional hazard models were used to evaluate the association between elevated troponin (>0.03) at the time of admission and 30-day mortality. Separate multivariable models were used to evaluate the association between peak troponin elevation during the hospitalization and in-hospital mortality (including beyond 30 days). Models were adjusted by age, race, sex, body mass index, smoking status, and history of coronary artery disease, congestive heart failure, diabetes, hypertension (HTN), chronic kidney disease (CKD), and atrial fibrillation.

A total of 7,238 patients were included in the 2 peak time periods (P1, n = 5,698; P2, n = 1,540). Patients from P1 had higher prevalence of HTN (35% vs 31%; $P < 0.001$), diabetes (24% vs 20%; $P < 0.003$), and CKD (12% vs 9.4%; $P < 0.002$), whereas P2 had more White patients (48% vs 29%; $P < 0.001$). Rates of in-hospital mortality and intubation were higher in P1 than P2 (23% vs 13%; $P < 0.001$; and 12% vs 7.6%; $P < 0.001$, respectively); however, there was no difference in rates of ICU admission (18% vs 16%; $P = 0.14$) or hospital length of stay (P1: 7.0 days [IQR: 3.0-13.0 days] vs P2: 6.0 days [IQR: 3.0-13.0 days]; $P = 0.20$). Regarding disease severity, admission Sequential Organ Failure Assessment scores were greater in P1 than P2 (1.99 vs 1.51; $P < 0.001$). Median peak troponin elevation during the hospitalization were higher in P1 when compared with P2 (0.030 [IQR: 0.000-0.146] vs 0.020 [IQR: 0.000-0.066]).

In multivariable-adjusted models, positive troponin on admission was associated with increased risk of 30-day mortality in both P1 (adjusted HR: 1.53; 95% CI: 1.19-1.96; $P < 0.0008$) and P2 (adjusted HR: 1.80; 95% CI: 1.03-3.24; $P < 0.0390$). When modeled as a continuous variable, peak troponin elevations (per 1-ng/mL increase) were associated with increased hazard of in-hospital mortality in P1 (HR: 1.008; 95% CI: 1.006-1.011) and with greater magnitude in P2 (HR: 1.042; 95% CI: 1.022-1.061).

This is the first study to assess the relationship between mortality and myocardial injury across 2 peak time periods of COVID-19. Although mortality was significantly lower in P2, myocardial injury as evidenced by troponin elevation was associated with increased 30-day mortality across both time periods. Interestingly, although peak troponin elevations during the hospitalization were associated with increased mortality in both time periods, such risk appeared to be greater in P2 compared with P1.

The decreased mortality seen in the pandemic has been reported previously and is possibly related to earlier infection detection, increased testing, novel treatments, viral variants, increased preparedness, and/or lower patient volumes (4). Even with this decreased mortality, peak troponin was a more robust predictor of mortality in P2 than in P1, whereas P2 had lower troponin elevation when compared with P1. Possibly, cardiac injury of any degree plays an important role in mortality even in less severe “waves” of this illness. Although earlier detection and novel

treatments in P2 may explain the lower overall mortality, patients who failed to respond to therapy may have had higher troponin caused by severe disease.

Among hospitalized patients with COVID-19 treated in a large health care system, troponin elevation both at the time of hospital admission and during the hospitalization remain independently associated with increased risk of mortality, irrespective of the time period and overall mortality rate.

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