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The Evolving Impact of Myocardial Injury in Patients With COVID-19 Amid the Omicron Wave of the Pandemic

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COVID-19 with myocardial injury, defined as troponin elevation, is associated with worse outcomes. The temporal changes in outcomes during various phases of the pandemic remain unclear. We evaluated outcomes during the Omicron phase compared with previous phases of the pandemic. We analyzed patients who were COVID-19-positive with evidence of myocardial injury who presented to the MedStar Health system (11 hospitals in Washington, District of Columbia, and Maryland) during phase 1 of the pandemic (March to June 2020), phase 2 (October 2020 to January 2021), and phase 3 (Omicron; December 2021 to March 2022), comparing their characteristics and outcomes. The primary end point was in-hospital mortality. The cohort included 2,079 patients admitted who were COVID-19 positive and for whom troponin was elevated (phase 1: n = 150, phase 2: n = 854, phase 3: n = 1,075). Baseline characteristics were similar overall. Inflammatory markers were significantly elevated in phase 1 compared with phases 2 and 3. The use of remdesivir and dexamethasone was highest in phase 2. In phase 3, 52.6% of patients were fully vaccinated. In-hospital mortality, though high, was lower in phase 3 than in phases 1 and 2 (59.3% vs 28.1% vs 23.3%; p <0.001). Patients who were vaccinated showed more favorable in-hospital outcomes than did those who were unvaccinated (18.3% vs 24.2%, p = 0.042). In conclusion, patients with COVID-19 with elevated troponin during phase 3 tended to have improved outcomes when compared with patients in earlier waves of the pandemic. This improvement could be attributed to the implementation of the COVID-19 vaccines, advances in COVID-19 treatment options, provider experience, and less virulent variants. **(Am J Cardiol 2023;190:54−60)**

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Patients infected with SARS-CoV-2, resulting in COVID-19, can develop myocardial injury,[1,2] with an overall high prevalence[3] and poor outcomes.[4−6] Furthermore, patients with known cardiovascular disease are at an increased risk of developing the more severe form of COVID-19.[7] Management of patients with ST-segment elevation myocardial infarction (STEMI) amid COVID-19 was controversial at first[8,9]; however, it was soon realized that some of these patients have no culprit vessel on angiography.[10] Given these concerning findings, guidelines reinforced early invasive angiography as the standard of care for STEMI and non−ST-elevation myocardial infarction (NSTEMI) in patients with high-risk features.[11,12] As data accumulated early in the pandemic, hospitals quickly adapted and evolved to better treat patients with COVID-19, including patients presenting with elevated troponin. Several waves of infections caused by SARS-CoV-2 have emerged since December 2019, the most notable of which are the Delta and Omicron waves.[13] The Omicron (B.1.1.529) variant was found to have an increased number of spike protein mutations and increased risk for transmission owing to increased immune evasion, despite the COVID-19 vaccines.[14] However, the effect on clinical outcomes of the Omicron variant as compared with previous variants is less clear. In this analysis, we seek to evaluate the in-hospital outcomes of cardiac involvement for patients with COVID-19 who presented with troponin elevation within our healthcare system during the Omicron wave and ways these outcomes compared with previous waves. Furthermore, we evaluate the impact of therapeutic options, along with vaccination status, on outcomes.

Methods

We analyzed patients who were COVID-19-positive with concomitant troponin elevation who presented to the MedStar Health system (11 hospitals in Washington, District of Columbia, and Maryland) during the pandemic era. Phase 3 (Omicron) of the pandemic was identified as December 1, 2021, to March 31, 2022, whereas phase 1 was defined as March 1 to June 30, 2020, and phase 2 as October 1, 2020,

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to January 31, 2021. These dates were chosen because they captured the 3 waves of the COVID-19 pandemic in the United States to date. Phase 1 occurred before public rollout of the COVID-19 vaccines, whereas phase 2 represents the very early stages of the vaccine rollout. A positive test for the infection was based on polymerase chain reaction testing and the patient having respiratory symptoms and/or chest x-ray or computed tomography findings. Drawing of troponins was not standardized and was at the discretion of the provider. Previous analysis at our institution indicated that the rate of troponin elevation in our patient population with COVID-19 was ∼9%.[15] The troponin values recorded were the peak values during the hospitalization of the patients who were COVID-19-positive. In our analysis, we included cardiac troponin I (cTnI; upper limit of normal, 0.03 ng/mL) or high-sensitivity cardiac troponin (hs-cTn; upper limit of normal, 30 ng/L), which are common troponin markers collected in our healthcare system. We identified significant presence of cTnI as an elevation >1 ng/mL or hs-cTn >30 ng/mL. Furthermore, we rescaled the hs-cTn from ng/L to ng/mL to simplify reporting.

Baseline patient characteristics were collected for each cohort. In this analysis, the co-morbidities were identified using International Classification of Diseases, Tenth Revision codes. Laboratory data, including inflammatory markers, were compared among the 3 groups. The primary end point was in-hospital mortality. The secondary end points included intensive care unit (ICU) admissions and use of mechanical ventilation. Hospital admission, ICU admission, and use of ventilation were not protocolized and were all under the discretion of the providing team at the respective hospital. Finally, vaccination (Pfizer, Moderna, or J&J/Janssen) status was recorded at time of admission to the hospital. Partial vaccination status refers to receiving only 1 of the 2 doses of the Pfizer or Moderna vaccine. In our secondary analysis, we compared outcomes based on patients within the Omicron phase who were vaccinated with outcomes of those who were not. The study was conducted in accordance with the Declaration of Helsinki and was approved by our institutional review board.

Descriptive statistics such as percentages, median and quartiles, or mean and SD were used to describe the study population. Analysis of variance was used to compare means of continuous variables. In the presence of variables that violate the normality assumption, the nonparametric Kruskal-Wallis test was performed. Chi-square test was used to compare categorical variables. Statistical significance was considered to be a $p < 0.05$. All analyses were done in R (v. 4.1.3). One author (BCC) has full access to all the data in the study and takes full responsibility for their integrity and the data analysis.

Results

The cohort included 2,079 patients admitted who were COVID-19-positive and for whom cTnI or hs-cTn was elevated (phase 1: n = 150, phase 2: n = 854, phase 3 [Omicron]: n = 1,075). Baseline characteristics are displayed in Table 1. Most patients were men with a mean age of 70.14 (60.48 to 79.87) years. The proportion of Caucasian patients was higher in the Omicron phase than in phase 1 and phase 2, whereas the proportion of Hispanic patients was the lowest in the Omicron phase. Rates of co-morbidities, such as hypertension, hyperlipidemia, chronic kidney disease, asthma, chronic obstructive pulmonary disease, coronary artery disease, congestive heart failure, previous pulmonary embolism, and atrial fibrillation, were similar among the groups. The baseline incidence of diabetes was significantly less in the patients in the Omicron phase than in the other 2 cohorts.

In addition, Table 1 depicts the proportion of patients who underwent coronary angiography and those who subsequently received revascularization with either percutaneous coronary intervention or coronary artery bypass grafting. Overall, the use of an invasive strategy was low and did not differ among the 3 phases. Lastly, Table 1 outlines the overall vaccination status and medical therapy given to patients with COVID-19 for their infection. The use of remdesivir and dexamethasone was lowest early in phase 1, peaked during phase 2, and was lower during the Omicron phase. As expected, patients were not fully vaccinated during phase 1, and a very small proportion were partially vaccinated in phase 2. In our patient population, 52.6% were fully vaccinated and 5.5% were partially vaccinated in the Omicron phase.

During hospital admission, white blood cell count, C-reactive protein, lactate dehydrogenase, and ferritin were all significantly higher in phase 1 than in phase 2 or the Omicron phase. A similar pattern was seen for maximum measured creatinine. Troponin values did not differ significantly among the 3 groups. Laboratory data are displayed in Table 2.

In terms of our primary end point, in-hospital mortality was significantly lower (23.3%) in the Omicron phase than in phase 1 (59.3%) and phase 2 (28.1%; $p < 0.001$). Regarding our secondary end points, 64.0% of patients in phase 1 received mechanical ventilation, compared with 32.1% in phase 2, and the rate was even lower, at 27.3%, in the Omicron phase ($p < 0.001$) (Figure 1). Lastly, the number of ICU admissions per patient was significantly lower in the Omicron phase than in the other phases (Table 3).

In our secondary analysis (Table 4), we examined patients with COVID-19 with troponin elevation in the Omicron phase and compared outcomes on the basis of vaccination status. Of note, there was a small component of patients for whom the vaccination status was unknown; thus, the overall cohort for this analysis was n = 794. Patients in the Omicron phase who were fully vaccinated, or even partially vaccinated, showed better in-hospital outcomes, including in-hospital mortality and need for mechanical ventilation.

Discussion

The primary findings of our analysis suggest that in-hospital outcomes (in-hospital mortality, admissions into the ICU, and mechanical ventilation) have declined throughout the course of the pandemic in patients who were COVID-19-positive with concomitant troponin elevation, with the lowest being the Omicron phase. Although patients with preexisting co-morbidities are at increased risk of COVID-19−related adverse outcomes,[16,17] the observations in our analysis are likely because of changes in treatment strategies, with the addition of remdesivir and dexamethasone and the implementation of the COVID-19 vaccines. In

Table 1
Baseline characteristics: baseline characteristics of patients with COVID-19 with troponin elevation in "Phase 1," "Phase 2," "Omicron Phase," and the COVID-19 pandemic overall

	Phase 1 (n = 150)	Phase 2 (n = 854)	Omicron phase (n = 1,075)	Overall (n = 2,079)	p Value*
Demographics					
Age [Q1, Q3] (years)	68.12 [58.16, 75.52]	71.30 [61.03, 80.16]	69.56 [60.31,79.93]	70.14 [60.48, 79.87]	0.093
Male	50.7%	55.6%	55.7%	55.3%	0.493
Height (cm)	167.64 [162.00, 175.00]	170.00 [162.56, 178.00]	170.00 [163.00, 178.00]	170.00 [163.00, 178.00]	0.300
Weight (kg)	84.10 [70.00, 98.80]	87.00 [72.00,99.95]	83.40 [68.55, 97.95]	85.00 [70.00, 99.00]	0.062
Race/ethnicity					
White	20.3%	31.6%	33.1%	31.5%	**0.007**
Black	67.6%	58.7%	62.2%	61.2%	0.072
Asian	0.7%	1.3%	0.7%	0.9%	0.353
Hispanic	12.0%	6.2%	2.3%	4.6%	**<0.001**
Native American	0.0%	0.0%	0.1%	0.0%	1.000
Hawaiian Pacific Islander	0.0%	0.0%	0.2%	0.1%	0.575
Co-morbidities					
Hypertension	48.7%	54.0%	49.1%	51.1%	0.087
Hyperlipidemia	60.0%	60.0%	55.3%	57.6%	0.104
Diabetes mellitus	56.7%	55.4%	47.1%	51.2%	**<0.001**
Chronic kidney disease	43.3%	47.9%	46.4%	46.8%	0.550
Hemodialysis	19.3%	16.4%	14.2%	15.5%	0.172
Chronic obstructive pulmonary disease	13.3%	17.2%	17.2%	16.9%	0.475
Asthma	4.7%	6.0%	6.3%	6.1%	0.720
Coronary artery disease	32.0%	33.5%	31.0%	32.1%	0.502
Stroke	20.7%	13.8%	14.8%	14.8%	0.093
Congestive heart failure	37.3%	38.6%	41.6%	40.1%	0.330
Atrial fibrillation	19.3%	22.8%	20.8%	21.5%	0.451
Prior pulmonary embolism	0.0%	0.1%	0.1%	0.1%	1.000
Medical therapy					
Dexamethasone	10.7%	74.1%	67.6%	66.2%	**<0.001**
Remdesivir	8.0%	34.8%	30.5%	30.6%	**<0.001**
Revascularization strategy					
Coronary angiography	5.3%	2.9%	3.3%	3.3%	0.285
Percutaneous coronary intervention	2.0%	1.5%	1.2%	1.4%	0.555
Coronary artery bypass grafting	0.7%	0.6%	0.2%	0.4%	0.277
Vaccination status					
Fully vaccinated	0.0%	0.0%	52.6%	44.0%	**<0.001**
Partially vaccinated	0.0%	3.4%	5.5%	5.2%	0.657
Not vaccinated	100%	96.6%	41.8%	50.8%	**<0.001**

* Boldface denotes statistical significance.
Values reported as median [interquartile ratio], percentage, or mean±SD.

Table 2
Laboratory data: laboratory data of COVID-19-positive patients with troponin elevation in "Phase 1," "Phase 2," "Omicron Phase," and the COVID-19 pandemic overall

	Phase 1 (n = 150)	Phase 2 (n = 854)	Omicron phase (n = 1,075)	Overall (n = 2,079)	p Value*
Maximum troponin (ng/mL)	2.38 [1.40, 5.95]	0.13 [0.05, 0.69]	0.12 [0.05, 0.51]	0.14 [0.06, 1.00]	**<0.001**
Time to maximum troponin (hours)	20.74 [6.26, 75.20]	3.57 [0, 26.3]	2.97 [0, 18.23]	4.13 [0, 25.08]	**<0.001**
N-terminal-pro-hormone B-type natriuretic peptide (ng/L)	3,038.00 [597.50, 13,583.00]	3,280.00 [665.25, 21,168.00]	4,853.00 [995.00, 17,062.00]	3,104.00 [604.00, 16,758.00]	0.834
Maximum creatinine (mg/100 mL)	3.14 [1.66, 6.04]	1.90 [1.20, 4.02]	1.92 [1.21, 4.30]	1.95 [1.22, 4.35]	**<0.001**
Maximum white blood cell (K/μL)	9.60 [8.60, 9.90]	9.30 [8.00, 9.80]	9.10 [7.60, 9.80]	9.20 [7.80, 9.80]	**<0.001**
C-reactive protein (mg/100 mL)	98.80 [59.32, 190.00]	75.55 [35.71, 96.50]	58.62 [16.36, 91.23]	70.09 [27.22, 96.92]	**<0.001**
Lactate dehydrogenase (U/L)	608.00 [473.00, 796.50]	434.00 [321.00, 593.00]	412.00 [284.50, 569.00]	438.00 [307.50, 609.00]	**<0.001**
Ferritin (ng/mL)	895.70 [611.28, 3,594.85]	754.30 [343.05, 1,446.13]	581.50 [247.95, 992.20]	706.90 [308.25, 1,300.62]	**<0.001**

* Boldface denotes statistical significance.
Values reported as median [interquartile range].

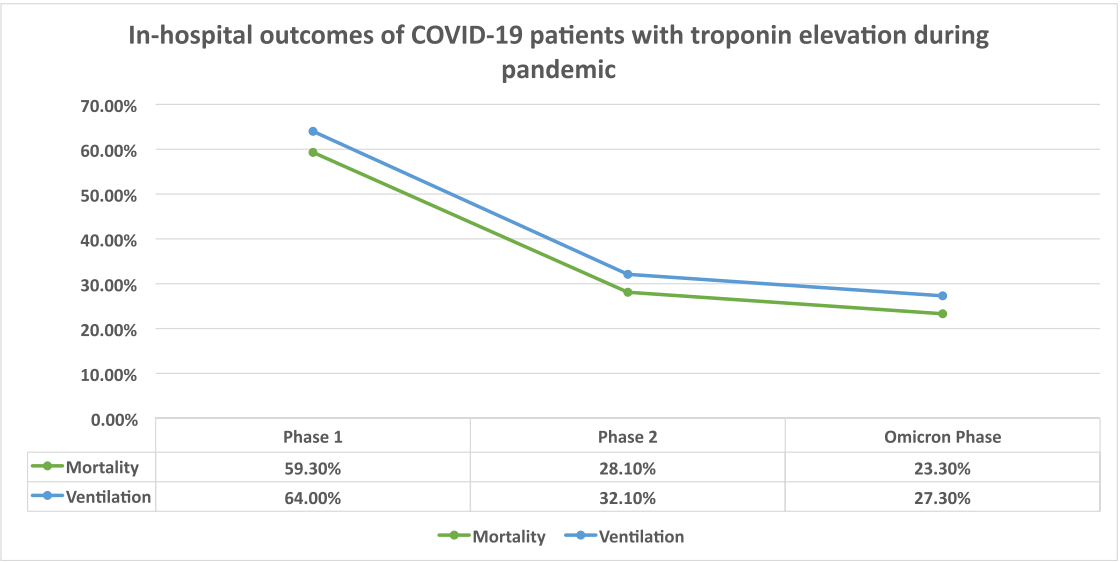

Figure 1. Overall in-hospital outcomes in patients with COVID-19 with troponin elevation during the pandemic—phase 1, phase 2, and Omicron phase.

addition, because it is known that patients infected with SARS-CoV-2 have elevated inflammatory markers[18] and that higher levels of these markers are associated with increasing severity of the illness and worse outcomes,[19] it is not surprising that our analysis revealed that patients in the phase 1 cohort had more significant elevations in all these markers than did the phase 2 and Omicron phase cohorts. This can potentially highlight that the Omicron variant was not as virulent as other strands of SARS-CoV-2. This also highlights the positive impact of the COVID-19 vaccines, which were rolled out before the Omicron phase.

The favorable outcome in the Omicron phase can be attributed to a less aggressive variant, half the population being vaccinated, better therapeutics being available, disease awareness, and early admission of these patients to the hospital for treatment. Early in the pandemic, there were patients with fears of going to the hospital owing to risk of SARS-CoV-2 infection. Patients with symptoms would wait at home longer during the early phase owing to fear of contracting the virus in the emergency department and because of lockdown uncertainty, resulting in a more severe presentation and worse outcomes.[20] In addition, there was a degree of racial and ethnic disparity noted in our analysis, with the proportion of Caucasian patients being higher in the Omicron phase than in phase 1 and phase 2, whereas the proportion of Hispanic patients was the lowest in the Omicron phase. This finding is interesting, and although it cannot be fully explained on the basis of our analysis, any potential racial or ethnic barriers in care need to be identified and addressed while caring for patients with COVID-19 with troponin elevation.

Furthermore, throughout the course of the COVID-19 pandemic, treatment strategies have evolved significantly as guidelines have changed and clinical knowledge has improved. In the early stages of the pandemic, the standard of care was initially supportive, including the use of supplemental oxygen, prone positioning,[21,22] conservative fluid management,[23] prophylactic antibiotics, management of co-morbidities, and avoiding mechanical ventilation whenever possible. More recently, the use of corticosteroids, in particular dexamethasone, is now recommended in patients with COVID-19 who require supplemental oxygen to decrease all-cause mortality.[24] Other treatment strategies include convalescent plasma infusions.[25] Finally, in October 2020, the antiviral medication remdesivir received emergency use authorization from the US Food and Drug Administration because the medication reduced time to recovery in those hospitalized with COVID-19.[26] However, more recent data on remdesivir may not support this finding as strongly.[27] Our analysis revealed positive response to dexamethasone and remdesivir with its increased use during phase 2 and the Omicron phase, with concomitant improvement in inflammatory markers and subsequent improvement in clinical outcomes.

Table 3
Primary and secondary outcomes: in-hospital outcomes and intensive care unit level of care of COVID-19-positive patients with troponin elevation in "Phase 1," "Phase 2," "Omicron Phase," and the COVID-19 pandemic overall

	Phase 1 (n = 150)	Phase 2 (n = 854)	Omicron phase (n = 1,075)	Overall (n = 2,079)	p Value*
Overall in-hospital mortality	59.3%	28.1%	23.3%	27.9%	**<0.001**
Received ventilation	64.0%	32.1%	27.3%	31.9%	**<0.001**
ICU admissions per patient	1.26±1.16	0.72±0.98	0.57±0.89	0.68±0.97	**<0.001**

* Boldface denotes statistical significance.
Values reported as percentage or mean±SD.

Table 4
Baseline characteristics, laboratory data, and outcomes based on vaccination status: baseline characteristics, laboratory data, and outcomes of COVID-19 patients with troponin elevation during the "Omicron Phase" based on vaccination status

	Fully vaccinated (n = 418)	Partially vaccinated (n = 44)	Not vaccinated (n = 332)	p Value*
Demographics				
A[Q1, Q3] **(years)**	70.56 [61.2,79.44]	67.93 [59.71,77.14]	66.86 [56.26,77.3]	**<0.001**
Male	58.4%	45.5%	55.7%	0.239
Height (centimeters)	171 [164,178]	166 [160,175]	170 [164,177]	0.208
Weight (kilograms)	84.6 [68.62,97.15]	72.44 [60.23,88.88]	86 [72.05,99.85]	**0.018**
Race/ethnicity				
White	32.8%	31.8%	43.1%	**0.012**
Black	62.9%	65.9%	53.2%	**0.019**
Asian	0.2%	2.3%	0.9%	0.195
Hispanic	1.5%	0.0%	2.5%	0.397
Native American	0.2%	0.0%	0.0%	0.637
Hawaiian Pacific Islander	0.2%	0.0%	0.0%	0.637
Co-morbidities				
Hypertension	49.5%	54.5%	51.2%	0.773
Hyperlipidemia	61.2%	61.4%	50.9%	**0.015**
Diabetes mellitus	51.2%	56.8%	41.9%	**0.018**
Chronic kidney disease	51.0%	52.3%	38.9%	**0.003**
Hemodialysis	16.5%	25.0%	12.0%	**0.040**
Chronic obstructive pulmonary disease	19.4%	20.5%	16.3%	0.475
Asthma	4.8%	18.2%	5.7%	**0.002**
Coronary artery disease	34.4%	36.4%	28.9%	0.228
Stroke	12.9%	15.9%	12.3%	0.801
Congestive heart failure	47.4%	52.3%	39.2%	**0.043**
Atrial fibrillation	23.7%	25.0%	18.4%	0.181
Prior pulmonary embolism	0.0%	0.0%	0.3%	0.498
Laboratory data				
Maximum troponin (ng/mL)	0.11 [0.06,0.4]	0.18 [0.09,0.44]	0.11 [0.05,0.57]	0.153
Time to maximum troponin (hours)	1.73 [0,16.44]	2.22 [0,15.5]	2.63 [0,18.9]	0.452
Maximum creatinine (mg/100 mL)	1.85 [1.23,4.09]	2.3 [1.26,6.91]	1.71 [1.1,3.77]	**0.048**
Maximum white blood cell (K/μL)	9 [7.3,9.7]	9.3 [7.95,9.8]	9.1 [7.68,9.8]	0.283
C-reactive protein (mg/100 mL)	53.79 [16.04,90.47]	17.23 [9.12,52.05]	60.94 [22.28,92.98]	**0.044**
Lactate dehydrogenase (U/L)	368 [267,521]	387 [312,459]	448 [304.25,628]	**0.003**
Ferritin (ng/mL)	507.8 [230.15,961.68]	539.3 [244.6,879.18]	663.9 [282.5,993.7]	0.194
Medical therapy				
Dexamethasone	62.2%	77.3%	72.9%	**0.003**
Remdesivir	32.8%	27.3%	27.1%	0.223
Revascularization strategy				
Coronary angiography	2.6%	6.8%	3.9%	0.219
Percutaneous coronary intervention	0.7%	2.3%	2.1%	0.171
Coronary artery bypass grafting	0.2%	0.0%	0.3%	1
In-hospital outcomes				
Overall in-hospital mortality	18.3%	11.4%	24.2%	**0.042**
Received ventilation	20.6%	25.0%	29.5%	**0.018**
Intensive care unit admissions per patient	0.51+/−0.87	0.5+/−0.9	0.56+/−0.88	0.686

* Boldface denotes statistical significance.
Values reported as median [interquartile ratio], percentage, or mean±SD.

Furthermore, particular attention has been directed toward the management of acute coronary syndrome during the COVID-19 pandemic. In patients with STEMI or NSTEMI with high-risk features, in which the etiology of their acute myocardial infarction is suspected to be true plaque rupture and not myocarditis or stress-induced cardiomyopathy in the setting of SARS-CoV-2 infection, our cardiac catheterization laboratory implemented procedures to ensure the safety of medical personnel during primary percutaneous coronary intervention. Per guidelines,[12] our institution trained staff in the catheterization laboratory on proper personal-protective-equipment use, designated 1 laboratory for patients who were COVID-19-positive or were under investigation, and performed extensive cleaning after each procedure. Furthermore, the healthcare system implemented new treatment and risk-stratification algorithms, using noninvasive diagnostic testing such as echocardiogram and cardiac magnetic resonance imaging in patients with low-risk features, ensuring that only patients with high-risk features with COVID-19 and with suspected

plaque rupture were brought to the catheterization laboratory.[28] Noninvasive imaging allowed the diagnosis of disease processes such as stress-induced cardiomyopathy or pericarditis, which are prevalent in patients with COVID-19, and allowed patients to avoid going to the catheterization laboratory. The implementation of noninvasive testing, in part, likely resulted in the low rate of an invasive strategy for the patients in our analysis.

Finally, we would be remiss not to further explain the positive impact that the COVID-19 vaccines have had on outcomes. In our analysis, the COVID-19 vaccines were administered fully before and during the Omicron phase of the pandemic. Therefore, in-hospital outcomes were less severe. In addition, a recent analysis has indicated that side effects from the COVID-19 vaccines are minimal.[29] It is important for providers to continue to emphasize the importance of all patients getting vaccinated against this potentially deadly disease.

There are limitations to our study. First, the analysis is retrospective and relies on the International Classification of Diseases, Tenth Revision codes to identify the patient population. Because inclusion in our analysis depended only on a positive COVID-19 test result and a positive troponin result, it did not distinguish between type I and type II NSTEMI, nor did it analyze whether patients had electrocardiographic changes and/or symptoms consistent with myocardial ischemia. In addition, the drawing of troponin in patients with COVID-19 was not standardized and was at the discretion of the provider. Furthermore, although guidelines did not initially recommend drawing troponin in the absence of chest pain, some sources now recommend that providers obtain troponin in addition to other inflammatory markers. Further analysis of these data would have allowed us to more comprehensively separate patients with obstructive coronary artery disease from those with other etiologies of myocardial injury (e.g., myocarditis or stress-induced cardiomyopathy).[30] The low rate of an invasive approach to managing these patients may suggest a higher rate of other etiologies for myocardial injury; however, it is difficult to truly know in this retrospective analysis. We also did not capture treatment methods for each patient (pharmacology, mechanical support, and so on). As such, although we believe that treatment methods differed among our 3 cohorts on the basis of the date of their hospitalization, we cannot be certain. Finally, our data captured patients in the Mid-Atlantic region of the United States, and our findings may not represent the broader US outcome data.

In conclusion, patients who were COVID-19-positive with elevated troponin during the Omicron phase of the pandemic tended to have improved outcomes, including improved in-hospital mortality, fewer ICU admissions, and decreased use of mechanical ventilation, compared with patients in the earlier phases. Despite a similar baseline incidence of many co-morbidities, those in the Omicron phase had decreased inflammatory markers. This improvement in outcomes likely reflects advances in available COVID-19 treatment options, provider experience with the novel disease, less virulent variants, and the implementation of the COVID-19 vaccines.

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Disclosures

Dr. Rogers reports being a proctor and consultant for Medtronic and Edwards Lifesciences; serving on the Advisory Board of Medtronic; and holding equity interest in Transmural Systems Inc. Dr. Waksman reports serving on the advisory boards of Abbott Vascular, Boston Scientific, Medtronic, Philips IGT, and Pi-Cardia Ltd.; being a consultant for Abbott Vascular, Biotronik, Boston Scientific, Cordis, Medtronic, Philips IGT, Pi-Cardia Ltd., Swiss Interventional Systems/SIS Medical AG, Transmural Systems Inc., and Venus MedTech; receiving institutional grant support from Amgen, Biotronik, Boston Scientific, Chiesi, Medtronic, and Philips IGT; and being an investor in Med Alliance and Transmural Systems Inc. The remaining authors have no conflicts of interest to declare.

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