

Atypical ST-Segment-Elevation Myocardial Infarction Presentation in Patients With COVID-19 at a High-Volume Center in New York City

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Atypical presentations of ST-segment-elevation myocardial infarction (STEMI) have been reported in patients who have COVID-19. We have seen this occurrence in our center in Bronx, New York, where multitudes of patients sought treatment for the coronavirus. We studied the prevalence of atypical STEMI findings among patients with COVID-19 who presented during the first 2 months of the pandemic.

Consistent with previous reports, 4 of our 10 patients with COVID-19 and STEMI had no identifiable culprit coronary lesion; rather, they often had diffuse ST-segment elevations on surface electrocardiograms along with higher levels of D-dimer and inflammatory markers. In contrast, 32 of 33 patients without COVID-19 (97%) had a culprit lesion. The patients with COVID-19 and a culprit lesion more often needed thrombectomy catheterization and administration of glycoprotein IIb/IIIa inhibitors.

Our study confirms that patients with COVID-19 often have atypical STEMI presentations, including the frequent absence of a culprit coronary lesion. Our findings can help clinicians prepare for these atypical clinical presentations. (Tex Heart Inst J 2021;48(5):e207446)

Citation:

Alvarez Villela MA, Alkhalil A, Weinreich MA, Koslowsky J, Aoi S, Latib MA. Atypical ST-segment-elevation myocardial infarction presentation in patients with COVID-19 at a high-volume center in New York City. *Tex Heart Inst J* 2021; 48(5):e207446. doi: 10.14503/THIJ-20-7446

Key words:

Cardiovascular diseases/physiopathology; coronary angiography; coronavirus infections/diagnosis; COVID-19; retrospective studies; ST elevation myocardial infarction/complications/epidemiology; time-to-treatment

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Montefiore Health System (Bronx, New York) was at the center of the SARS-CoV-2 coronavirus pandemic: more than 5,000 patients who had COVID-19 were discharged from our hospital as of early May 2020. Throughout this period, we continued to provide primary percutaneous coronary intervention (PCI) for patients who presented with ST-segment-elevation myocardial infarction (STEMI). Delays before first medical contact and a high prevalence of nonobstructive coronary artery disease accompanying STEMI have been described during the current pandemic.^{1,2} We noted and decided to study atypical presentations of STEMI in patients with COVID-19.

Patients and Methods

We retrospectively collected data on all patients who presented with STEMI diagnosed on electrocardiograms (ECG) and who underwent coronary angiography at our center from 11 March 2020 (the date of the first reported case of COVID-19 in our system) through 9 May 2020. Each patient's COVID-19 diagnosis was determined from the results of a real-time polymerase chain reaction (PCR) assay for SARS-CoV-2 from a nasopharyngeal sample tested by our hospital laboratory. During the initial phase of the pandemic, before PCR assays were widely available in our hospital system, 14 patients who presented with STEMI completed a screening questionnaire and were considered to be negative for COVID-19. Telehealth visits were used to confirm the absence of subsequent symptoms in 11 of these patients (79%) after a median duration of 18 days (interquartile range [IQR], 12–25 d). We divided patients diagnosed with both COVID-19 and STEMI into subgroups of those who had or did not have a culprit coronary lesion.

TABLE I. Characteristics of 43 Patients Diagnosed With STEMI During the COVID-19 Pandemic

Variable	Without COVID-19* (n=33)	With COVID-19 (n=10)	
		Culprit Lesion (n=6)	No Culprit Lesion (n=4)
Age (yr)	63 (55–71)	59 (55–72)	54 (46–62)
Male	20 (60)	6 (100)	3 (75)
Race/ethnicity			
Hispanic	9 (28)	5 (83)	0
Black	15 (45)	1 (17)	3 (75)
White	6 (18)	0	0
Asian	2 (6)	0	1 (25)
Unknown	1	0	0
Comorbidities			
Diabetes	10 (30)	6 (100)	2 (50)
Hypertension	21 (64)	5 (83)	2 (50)
Hyperlipidemia	25 (76)	4 (67)	3 (75)
Active smoker	10 (30)	0	0
Obesity	13 (39)	3 (50)	3 (75)
Coronary artery disease	6 (18)	2 (33)	1 (25)
Chronic kidney disease	3 (9)	0	0
Presenting symptoms			
Chest pain	32 (97)	5 (83)	0
Fever	2 (6)	1 (17)	2 (50)
Cough	3 (9)	2 (33)	2 (50)
Dyspnea	8 (24)	3 (50)	3 (75)
Intubation needed	1 (3)	1 (17)	2 (50)
Cardiogenic shock	2 (6)	2 (33)	1 (25)
Cardiac arrest	1 (3)	0	1 (25)
ST-segment elevation			
Focal	30 (91)	5 (83)	1 (25)
Diffuse	3 (9)	1 (17)	3 (75)
LVEF (%)	45 (35–55)	42 (33–54)	35 (30–66)
Regional WMA	26 (79)	6 (100)	2 (50)
Laboratory values			
Peak troponin T (ng/mL)	5.4 (1.8–9.8)	2.5 (1–3.6)	6.8 (3–13)
C-reactive protein (mg/dL)	4.2 (2.5–13.8)	6 (0.9–14)	13 (10–18.6)
Ferritin (ng/mL)	386 (104–715)	634 (400–1,629)	6,652 (659–12,704)
D-dimer (µg/mL)	0.8 (0.5–4.4)	2.2 (0.9–4.8)	13 (2.3–20)
WBC (× 10 ³ /µL)	10 (8.5–14)	9 (5.6–13)	13 (10–28)
Neutrophils (%)	79 (68–85)	77 (58–89)	78 (69–84)
Lymphocytes (%)	14 (7–23)	8 (4.5–29)	7.5 (7–10)
Procedural details			
PCI performed	32 (97)	6 (100)	0
LAD diagonal branch	18 (55)	4 (67)	0
LCx	1 (3)	0	0
RCA	13 (39)	2 (33)	0
None	1 (3)	0	4 (100)
Thrombectomy catheter use	3 (9)	3 (50)	0
IABP placement	9 (27)	1 (17)	1 (25)
In-hospital death	1 (3)	2 (33)	2 (50)

IABP = intra-aortic balloon pump; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; LVEF = left ventricular ejection fraction; PCI = percutaneous coronary intervention; RCA = right coronary artery; STEMI = ST-segment-elevation myocardial infarction; WBC = white blood cell count; WMA = wall-motion abnormality

*Only 1 patient had no culprit lesion.

Data are presented as median and interquartile range or as number and percentage.

Statistical Analysis

We classified ST-segment elevation (STE) as focal when it was present in 2 contiguous leads, and as diffuse when in noncontiguous leads. We collected peak values for cardiac troponin T, and we diagnosed cardiogenic shock (CS) in accordance with pulmonary artery catheterization findings (a cardiac index <2 L/min/m² and a pulmonary capillary wedge pressure >15 mmHg). In our descriptive statistics, qualitative data are presented as number and percentage and quantitative data as median and IQR.

Results

Of 43 patients who underwent coronary angiography when STEMI was suspected, 33 had negative results for COVID-19 and 10 had positive results (Table I).

Patients Without COVID-19

Of the 33 patients without COVID-19 (median age, 63 yr; IQR, 55–71 yr), 32 (97%) had chest pain (the most prevalent symptom), 2 (6%) had fever, and 1 (3%) was intubated because of respiratory distress. Focal STE was present in 30 patients (91%), and 26 patients (79%) had regional wall-motion abnormalities (RWMA) on transthoracic echocardiograms (TTE). A culprit lesion was identified in 32 patients, all of whom underwent PCI. Mechanical thrombectomy (with use of a Medtronic Export AP aspiration catheter, when performed) and intravenous glycoprotein (GP) IIb/IIIa inhibitor infusion were rarely necessary in this group. Two patients (6%) had CS on presentation, and 9 (27%) underwent intra-aortic balloon pump (IABP) placement. One patient, a 68-year-old woman who had a delayed presentation complicated by ventricular septal and free-wall rupture, died in the hospital. The other 32 patients survived their hospital stays.

Patients With COVID-19

The 10 patients with COVID-19 had more severe lymphopenia and higher C-reactive protein (CRP), ferritin, and D-dimer levels than the COVID-negative patients did. Six (60%) had a culprit coronary lesion and 4 (40%) did not.

Patients With a Culprit Lesion. The median age of the 6 patients with a culprit coronary lesion was 59 years (IQR, 55–72 yr). All had diabetes, and 5 (83%) had hypertension. Five had focal STE, and all 6 had RWMA on TTE. Five presented with chest pain, 2 had CS, 1 had fever, 1 needed intubation, and 1 underwent IABP placement. Thrombectomy was performed in 3 patients, with use of the AngioJet (Boston Scientific Corporation) in 2 and an Export AP aspiration catheter in one, and intravenous GP IIb/IIIa inhibitors were infused. One patient in this subgroup had very late right coronary artery stent thrombosis, profound lym-

phopenia (leukocytes, 3%), a CRP level of 12.5 mg/dL, and a D-dimer level of 10 μ g/mL. He and one other patient died in the hospital (mortality rate, 33%).

Patients Without a Culprit Lesion. The median age of the 4 patients without a culprit lesion was 54 years (IQR, 46–62 yr). Two had fever, and 2 needed intubation because of respiratory distress. Three each had diffuse STE on ECG and a low left ventricular ejection fraction on TTE. None presented with chest pain. One underwent thrombolysis before arrival at our center. One had CS on presentation and underwent IABP placement. Two died in the hospital (mortality rate, 50%). These 4 patients had substantially higher levels of D-dimer and inflammatory markers than our other patients did.

Symptom Onset. The median times from symptom onset to first medical contact were 72 hours (IQR, 30–114 hr) in the patients without a culprit lesion, 6 hours (IQR, 4–42 hr) in those with a lesion, and 8 hours (IQR, 1.5–24 hr) in the patients without COVID-19.

Discussion

Of our patients with COVID-19 and STEMI, 40% had no identifiable culprit lesion. They presented without chest pain, had a higher prevalence of fever, and had notably elevated inflammatory markers and D-dimer levels. The presence of diffuse STE can help clinicians discriminate these STEMI cases from those caused by coronary artery occlusion.^{1,3} Given our limited experience at the time, we did not use these criteria to select patients for coronary angiography.

The underlying mechanism of myocardial injury in patients with COVID-19 and STEMI is undetermined⁴; nevertheless, some points have become clear. First, our center did not initially perform endomyocardial biopsies when patients had no culprit lesion. There is now histologic evidence that patients with COVID-19 have myocardial injury and inflammation.⁵ Second, of all the patients who underwent PCI, a larger number with COVID-19 needed catheter thrombectomy and GP IIb/IIIa inhibitor infusion, suggesting that they had a higher thrombus burden, perhaps related to the prothrombotic state of COVID-19.⁶ Finally, compared with the group of patients without COVID-19, the group with COVID-19 had a higher incidence of CS, and a greater percentage died in the hospital.

Our study confirms that patients with COVID-19 often have atypical presentations of STEMI, including the frequent absence of a culprit coronary lesion. Our findings may help clinicians prepare for these atypical clinical presentations.

Acknowledgments

We thank Drs. Anna E. Bortnick, Juan Terre, Jose M. Wiley, Mark A. Menegus, and our other colleagues at

Montefiore Medical Center for their relentless dedication to patient care during the COVID-19 outbreak in New York City in spring 2020.

Published: 15 December 2021

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