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The clinical spectrum of myocardial injury associated with COVID-19 infection

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ABSTRACT

Although respiratory symptoms are the dominant features of COVID-19 infection, myocardial injury has been described in these patients. Reported cardiac manifestations of COVID-19 infection include myocarditis, arrhythmia and acute coronary syndrome including ST elevation myocardial infarction (STEMI). STEMI is a medical emergency and timely intervention is of utmost importance to prevent mortality and long-term morbidities. In this report, we present a wide spectrum of clinical presentations, management, and outcomes for five patients with COVID-19 infection and ST elevation on ECG.

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COVID-19; myocardial injury; myocarditis; thrombosis; ST elevation

Systemic inflammation, cytokine storm and a hypercoagulable state have been proposed as possible explanations for coronary atherosclerotic plaque rupture and subsequent ST elevation myocardial infarction (STEMI) in patients with COVID-19 infection [1]. Though there have been a few recently published reports [2,3], there remain gaps in knowledge regarding the natural history of STEMI in this setting. We retrospectively identified five patients with COVID-19 infection and ST elevation (STE) who underwent emergent left heart catheterization (LHC) in our health system between 20 February 2020 and 31 May 2020. This project was undertaken as a Quality Improvement Initiative and as such does not constitute human subjects research.

Mean age was 56.8 years and all patients were male. Race distribution included two Hispanics, one African American, one Asian and one White. Baseline characteristics, imaging and clinical outcomes are shown in Table 1. Coronary angiography demonstrated culprit thrombotic lesions in three patients, two of whom underwent percutaneous coronary intervention (PCI). Acute stent thrombosis was detected in one patient requiring further intervention. Patient #1 presented with cardiac arrest, 4 weeks after COVID-19 diagnosis as an outpatient. Post-resuscitative coronary angiography demonstrated distal left main artery disease. Given his prolonged arrest, coronary artery bypass graft surgery was deferred until after completion of therapeutic hypothermia in order to assess neurologic function. An intra-aortic balloon pump (IABP) was implanted for coronary perfusion and hemodynamic support. After rewarming, he developed ventricular fibrillation (VF) requiring defibrillation. Mechanical support was escalated to Impella-CP given refractory shock. His hospital course was complicated by mixed shock with concomitant sepsis syndrome, with two subsequent episodes of sustained ventricular tachycardia. He did not demonstrate neurologic recovery and developed multi-organ failure. Family opted for comfort care measures.

In this cohort, two patients did not have chest pain or STE on initial presentation. One of these patients (#2) presented with altered mental status and within 2 hours developed chest pain with ECG showing inferior STE. He underwent emergent PCI of the right coronary artery. Patient #3 presented with cough and fever for 3 days and was intubated for persistent hypoxia despite non-invasive ventilation. He developed cardiac arrest shortly after a repeat ECG showed inferolateral STE. He was successfully defibrillated for VF. Emergent left and right heart catheterization showed non-obstructive CAD, severe global hypokinesis, right atrial pressure of 19 mmHg, pulmonary capillary wedge pressure of 30 mmHg and cardiac index of 1.9 L/min/m^2. Inotropic support was initiated, and he was transferred to our hospital for further management of myocarditis and acute hypoxic respiratory failure. He underwent veno-venous extracorporeal membrane oxygenation (VV ECMO) placement due to persistent hypoxia and hypercarbia despite maximal ventilator support. His VV ECMO was decannulated 2 weeks later. Serial echocardiograms showed significant improvement in his cardiac function. He remains hospitalized at this time in improved condition. Lastly, patient #4 was a 32-year-old male who presented with chest pain and anterolateral STE in ECG. Angiogram showed normal coronaries with evidence of apical ballooning consistent with stress-induced cardiomyopathy.

In this small group of patients, we found a wide spectrum of clinical presentations, management, and outcomes for patients with COVID-19 infection and STE

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Table 1. Baseline characteristic, imaging, and clinical outcome of patients.

Patient	Chief complaint	comorbidities	STE on initial presentation	ECG changes	Culprit lesion	PCI	LVEF	WMA	MCS	Outcome
1	Cardiac arrest	None	Yes	aVR and V1 STE	Left main artery	No	25–30%	Global hypokinesis	IABP→Impella	Death
2	Altered mental status	Former smoker	No	Inferior STE	Right coronary artery	Yes	44%	Regional	No	Death
3	Cough/fever	DM, HTN, Hypothyroidism, non-smoker	No	Inferolateral STE	None	No	30–35%	Global hypokinesis	VV-ECMO	Still hospitalized
4	Chest pain	HTN, current smoker	Yes	Anterolateral STE	None	No	40–45%	Apical ballooning	No	Discharged
5	Chest pain	CAD, atrial fibrillation, former smoker	Yes	Anterolateral STE	Left anterior descending artery	Yes	30%	Regional	IABP	Discharged

STE:ST elevation, PCI: percutaneous coronary intervention; LVEF: left ventricular ejection fraction; WMA: wall motion abnormalities; MCS: mechanical circulatory support; IABP: intra-aortic balloon pump; DM; diabetes; HTN: hypertension; CAD: coronary artery disease; VV-ECMO: veno-venous extracorporeal membrane oxygenation.

on ECG. There were three patients with acute thrombotic coronary lesions, one patient with initial PCI complicated by stent thrombosis, one patient with myocarditis complicated by cardiogenic shock and one patient with stress-induced cardiomyopathy. Stress-induced cardiomyopathy [4] and higher incidence of stent thrombosis [5] have also been previously reported in COVID-19 patients and may be explained by the associated cytokine storm and hypercoagulable state. Systemic inflammation and hypercoagulopathy also increase the risk of acute coronary syndrome and microvascular disease [1]. Heightened clinical vigilance is recommended for the risk of acute coronary thrombosis in the management of patients with COVID-19 infection given reports of increased inflammation, platelet activation and endothelial dysfunction in this critically ill patient cohort.

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