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CASE REPORT

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SARS-COV-2 infection presenting as ST-elevation myocardial infarction

Francesco Castagna MD | Roberto Cerrud-Rodriguez MD | Miguel Alvarez Villela MD | Anna E. Bortnick MD, PhD, MS

Department of Medicine, Division of Cardiology, Montefiore Health System, Albert Einstein College of Medicine, New York, New York

Correspondence
Anna E. Bortnick MD, PhD, MS, Jack D. Weiler Hospital, 1825 Eastchester Road, Suite 2S-46, Bronx, New York, NY 10461.
Email: abortnic@montefiore.org

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Abstract

We describe a patient presenting with chest discomfort, anterolateral ST elevation, and developing acute cardiogenic shock secondary to SARS-COV-2infection—patient zero presenting to our institution's cardiac catheterization laboratory. The emergent presentation with limited clinical information led to exposure of personnel. The diagnosis was complicated by two negative tests for SARS-COV-2, and high-clinical suspicion from the patient's occupational history led to additional testing in order to confirm the diagnosis.

KEYWORDS

acute myocardial infarction/STEMI, angiography, coronary, cardiomyopathy

1 | INTRODUCTION

Cardiac manifestations of SARS-COV-2 are poorly understood, but early data suggest that individuals infected with the novel pathogen responsible for the COVID-19 pandemic are susceptible to myocarditis.[1] Cardiac catheterization laboratories are on the front lines for patients presenting with emergent cardiac syndromes and must be alert to extrapulmonary presentations of SARS-COV-2.

2 | CASE DESCRIPTION

A 51-year-old man, a customer service representative at an international airport, presented to an outside hospital Emergency Department (ED) with 4 days of malaise, progressing to left-sided, non-radiating chest pain, diaphoresis and syncope with fall. His presenting vital signs were: 98°F, 181/100 mmHg, 100 beats per minute, respiratory rate 20 breaths/min, oxygen saturation 95% on room air and he had a normal physical examination. The electrocardiogram (EKG) was concerning for 3.5 mm ST elevation in I and avL, 5 mm isolated ST elevation in lead V2, with deep reciprocal depressions in III, avF and avR (Figure 1a). Computed tomography (CT) of the head was negative for intracranial hemorrhage. He was transferred to our hospital for emergent cardiac catheterization on suspicion of ST-elevation myocardial infarction (STEMI) which revealed widely patent coronary arteries (Figure 1b,c), a preserved left ventricular ejection fraction (LVEF) of 55% and anteroapical hypokinesis on ventriculography. Cardiac catheterization laboratory personnel were wearing usual personal protective equipment, but not N95 masks or protective eyewear. Several hours later, febrile to 103°F and rigoring, he became hypotensive (65/50 mmHg) and tachycardic (110 beats per minute), with mild cough. Bilateral interstitial prominence, consistent with possible pneumonia, was seen on chest X ray.

The past medical history was significant for hypertension and hypercholesterolemia. Six weeks prior to the current presentation, he had visited another ED. He reports being told that he had a "heart attack" and that he was treated with unspecified medication. He ran out of prescriptions for 2 weeks prior to the presentation, could not report which medications he was prescribed, and was only taking aspirin and ibuprofen. He had traveled to three different boroughs of New York City during that time. The differential diagnosis included sepsis, myocarditis, and due to occupational history, SARS-COV-2.

Initial laboratory testing was remarkable for hyponatremia (129 mEq/L), normal white blood cell count (9.6 k/μl) with elevated neutrophils (8.3 f/L) and decreased lymphocytes (0.6 k/μl), microcytic anemia (hemoglobin 11.5 mg/dl, mean corpuscular volume 79.3 fl), mildly elevated liver function tests with aspartate aminotransferase (AST) 82 U/L and alanine aminotransferase (ALT) 128 U/L, elevated

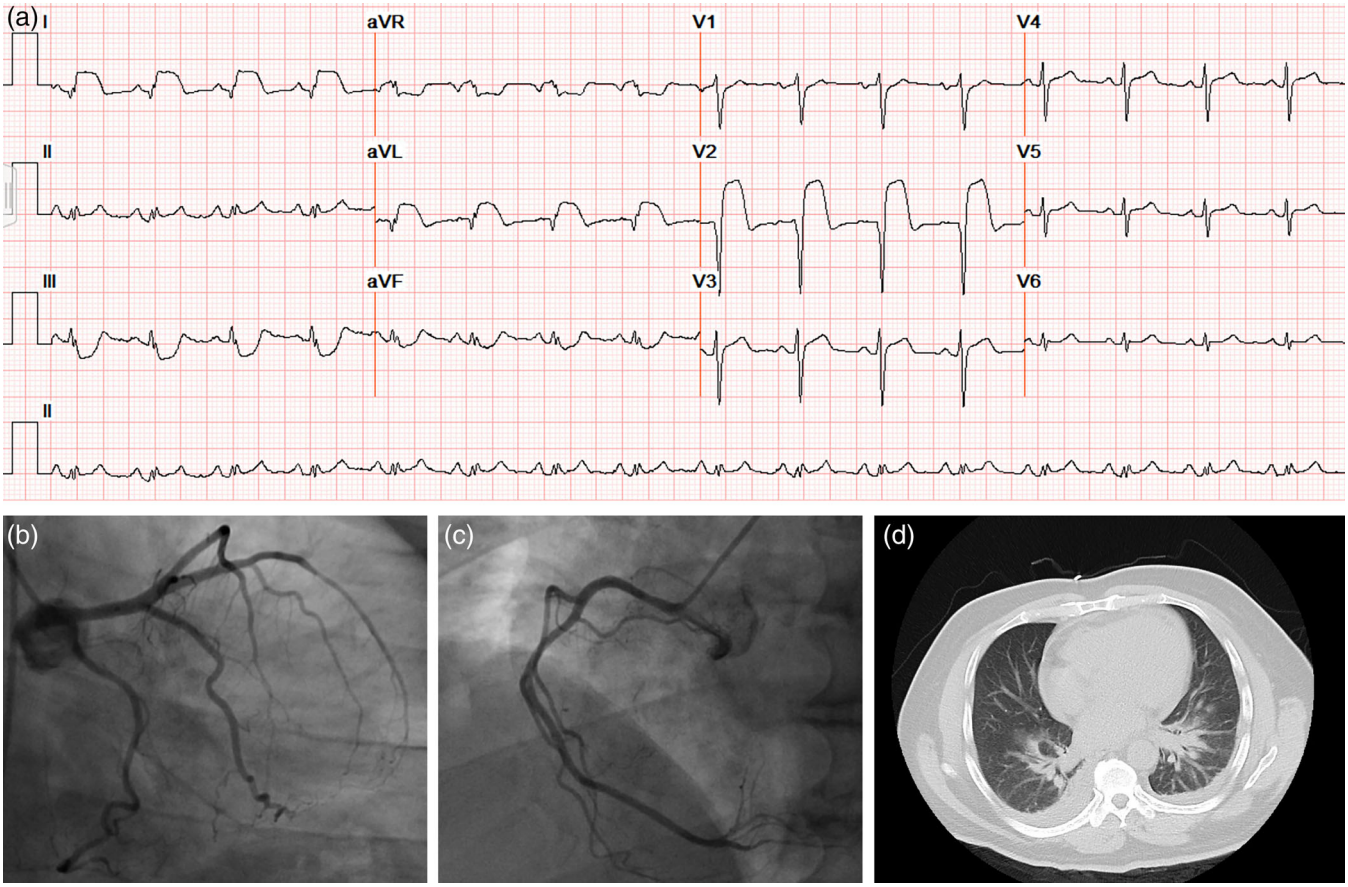

FIGURE 1 (a) Presenting 12-lead EKG with anterolateral ST elevation and reciprocal inferior depression. (b, c) Coronary angiogram showing widely patent coronary arteries. (d) Computed tomography of the chest demonstrating perihilar groundglass opacities, thickening of interlobular septa, and minimal bilateral pleural effusions

N-terminalpro-B natriuretic peptide 3,307 (pg/ml), as well as elevated cardiac biomarkers with creatine kinase 647 U/L and troponin T 1.65 ng/ml (ref <0.1 ng/ml).

Routine blood and urine cultures, rapid tests for Legionella, Streptococcus pneumoniae, Mycoplasma pneumoniae, Chlamydiae pneumonia, and an extensive viral panel, inclusive of human rhinovirus, influenza A, B, A H1, A H3, and H1N1, parainfluenzae virus 1–4, respiratory syncytial virus A and B, human metapneumovirus, adenovirus C, and coronavirus (non-SARS-COV-2), were negative.

CT chest was remarkable for perihilar groundglass opacities, thickening of interlobular septa, and minimal bilateral pleural effusions, interpreted as consistent with congestive heart failure (Figure 1d). LVEF declined to 40% on subsequent echocardiography with the finding of apical akinesis (Video S1), and he developed a small pericardial effusion.

Broad spectrum antibiotics were administered empirically and the patient was admitted to our Cardiac Intensive Care Unit (CICU). His course was complicated by high fever (103°F), worsened pulmonary congestion, progressing to hypoxia, requiring 2 L of oxygen by nasal cannula, elevated lactate (2.6 mmol/L), with a Fick cardiac output (CO) of 3.1 L/min and cardiac index (CI) of 1.8 L min^{-1} m^{-2}, consistent with cardiogenic shock. He had marginal improvement on intravenous dobutamine 2.5 mcg kg^{-1} min^{-1} and nitroglycerin 10 mcg/ml with a Fick CO 3.5 L/min and CI 2.0 L min^{-1} m^{-2}.

SARS-COV-2 infection was suspected based on occupational history and worsening clinical course. Multiple nasopharyngeal samples were obtained for SARS-COV-2 testing. The first two run at independent laboratories (Lab A and Lab B) were negative and the third, positive (Lab C). A fourth confirmatory sample, sent to Lab A, was also positive. The patient was initially treated with lopinavir/ritonavir 400 mg/100 mg tablet by mouth every 12 hr for 4 days and hydroxychloroquine 500 mg by mouth every 12 hr, then hydroxychloroquine alone 400 mg by mouth daily. Lopinavir/ritonavir was discontinued as it may have limited efficacy in treatment of SARS-COV-2 infection and adverse events.[2] The patient recovered and was discharged home on day 26 on aspirin, statin and metoprolol.

3 | DISCUSSION

Although the principal manifestations of SARS-COV-2 infection have been documented as respiratory, myocardial injury was reported in 5 out of the first 41 cases diagnosed in Wuhan, China (Table 1).[1] Other early reports from the region indicate that patients admitted to

TABLE 1 Previous reports of cardiac involvement with SARS-COV-2 infection

Author	Age/ sex	Past history	Symptoms	Cardiac biomarkers	EKG	Coronary imaging	Echocardiogram	Treatment	Outcome
Zeng[1]	63 M	NA	Fever, dyspnea, productive cough	Troponin-I (11.37 g/L) Myoglobin (390.97 Ng/ml) NT-ProBNP (22,600 pg/ml)	Sinus tachycardia, no ST changes	None	LVEF 32%, diffuse myocardial dyskinesia, normal right heart function	Ventilatory support, Lopinavir-ritonavir, interferon α-1b, methylprednisolone, immunoglobulin, Piperacillin-tazobactam, renal replacement therapy, ECMO	LVEF 68%
Hu[2]	37 M	NA	Dyspnea, chest pain, diarrhea, hypotension	Troponin-T (>10,000 ng/L) CKMB (112.9 ng/L) NT pro-BNP (21,025 ng/L)	ST- elevation (III, AVF)	Normal coronary arteries by CTA	LVEF 27% and LV dilation, normal right heart function	Methylprednisolone, immunoglobulin, norepinephrine, diuretic, milrinone, piperacillin-sulbactam	LVEF 66% Cardiac biomarkers improved after 1 week

Abbreviations: CK-MB, Creatine kinase-myocardial b fraction; CTA, computed tomography angiography; ECMO, extracorporeal membrane oxygenation; LVEF, left ventricular ejection fraction; NT pro-BNP, N-terminal pro-B-type natriuretic peptide; NA, not applicable.

intensive care with SARS-COV-2 had higher levels of high sensitivity-cardiac troponin (hs-cTn) and creatine kinase-myocardial band fraction, pointing to a higher severity of disease associated with cardiac injury.[3] Per the National Health Commission of China, 12% of patients who died without a prior cardiac history had elevated hs-cTn and suffered inpatient cardiac arrest.

Several mechanisms have been proposed to explain the etiology of cardiac involvement in SARS-COV-2 infection; all require further investigation. The coronavirus spike protein is known to bind angiotensin converting enzyme 2, suggesting potential for direct entry into myocardial cells, although virus in myocardial cells has not been reported, albeit, in limited studies. Autopsy findings are limited, often incomplete and focused on lung pathology.[1,3,5] In a small case series from the United States, no evidence of myocarditis was noted on autopsy of two individuals.[6] Endomyocardial biopsy may be useful in understanding the histopathology of disease more so than in deciding whether or not to use high dose steroids, which is being liberally adopted as an immune modulatory strategy in COVID-19. Endomyocardial biopsy of a case of fulminant myocarditis revealed viral particles in interstitial cells of the heart, associated with mild inflammation, but particles were not in myocytes or endothelia, per se.[7] Fibrosis and necrosis were not observed. The authors concluded that virus could have directly infected cells or been transported by macrophages originating from lung.

Alternatively, cytokine storm may induce a stress cardiomyopathy which would account for resultant EKG and echocardiographic abnormalities, or severe hypoxia may lead to endomyocardial ischemia. To date, there is no evidence of coronary vasospasm or plaque rupture accounting for cardiac dysfunction in COVID-19, thus the mechanism of SARS-COV-2 associated troponin elevation appears to be demand ischemia or type 2 myocardial infarction, although thrombosis may be increased due to heightened inflammation. Whether there is complete myocardial recovery is unclear and cardiac MRI may be a sensitive modality for follow-up of recovered patients.

Notably, our patient had two negative tests for SARS-COV-2 at two different laboratories. This might indicate that virus was not being highly aerosolized early on in his course or relate to variations in sample collection or processing. The sensitivity and specificity of testing unclear and potentially varies by laboratory, as testing is decentralized and has only recently begun in the US epidemic. In addition, our patient was not a candidate for compassionate use of remdesivir (other trials were not available at the time of his presentation) but there are several randomized controlled trials in progress for remdesivir, convalescent plasma, hydroxychloroquine and anti-IL-6 antibodies.

4 | CONCLUSIONS

Myocardial injury presenting as STEMI and complicated by cardiogenic shock may be a clinical presentation of the novel SARS-COV-2 virus. Cardiac catheterization laboratories and cardiac intensive care units, as front line providers for acute cardiac care, should be alert to SARS-COV-2 associated cardiomyopathy presenting as STEMI and implement protocols for management.[8]

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CONFLICT OF INTEREST

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ORCID

Anna E. Bortnick https://orcid.org/0000-0001-7983-5243

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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