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CASE REPORT

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COVID-19 complicated by acute myocardial infarction with extensive thrombus burden and cardiogenic shock

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Abstract

A patient with coronavirus disease 19 (COVID-19) developed acute myocardial infarction (AMI) complicated by extensive coronary thrombosis and cardiogenic shock. She underwent percutaneous coronary intervention and placement of a mechanical circulatory support device but subsequently died from shock. This report illustrates the challenges in managing patients with COVID-19, AMI, and cardiogenic shock.

KEYWORDS

cardiogenic shock, COVID-19, myocardial infarction

1 | INTRODUCTION

Coronavirus disease 19 (COVID-19) is associated with serious cardiovascular complications, including acute myocardial infarction (AMI). We present the case of a patient with COVID-19 who developed ST-segment elevation myocardial infarction (STEMI) and cardiogenic shock. Emergent percutaneous coronary intervention (PCI) was complicated by extensive thrombus burden with distal embolization and recurrent stent thrombosis within the newly deployed stent, managed with the administration of intracoronary thrombolytics and eptifibatide. To our knowledge, this is the first report that describes the use of intracoronary thrombolytics in a patient with COVID-19 and recurrent intraprocedural thrombotic complications.

2 | CASE

A 40-year-old woman with a history of diabetes mellitus, hypertension, hyperlipidemia, and paranoid schizophrenia presented to the emergency department with retrosternal chest pain for 1 day and cough and shortness of breath for 1 week. She reported exposure to confirmed COVID-19-positive residents in her group home. On admission, she was afebrile (37.5°C), normotensive (124/78 mmHg), but tachycardic (112 beats per minute), and hypoxic (oxygen saturation 84% on room air and 95% on 8 L of oxygen via nasal canula). Electrocardiogram (ECG) showed sinus tachycardia and left ventricular (LV) hypertrophy (Figure 1a). Chest X-ray showed bilateral opacities consistent with pneumonia (Figure 2). Laboratory data are presented in Table 1. The patient was admitted for treatment of COVID-19 pneumonia and to rule out acute coronary syndrome, treated with an aspirin 325 mg and clopidogrel 600 mg oral load. On the morning of hospital day 2, she reported worsening chest pain. ECG showed sinus tachycardia, new right bundle branch block, and ST-segment elevation in the anterior, lateral, and inferior leads (Figure 1b).

The patient received maintenance dual antiplatelet therapy and 4,000 U of intravenous (IV) unfractionated heparin and was transferred to our facility for emergent cardiac catheterization. Upon arrival, heart rate was 113 beats per minute, blood pressure 165/112 mmHg, and oxygen saturation was 93% on 6 L via nasal canula. Transradial coronary angiography demonstrated a thrombotic occlusion at the ostium of the left anterior descending (LAD) artery (Figure 3) and no significant disease in the other vessels in a large right-dominant system.

IV bivalirudin was administered and a 2.0-mm compliant balloon was used to establish flow. Angiogram showed extensive thrombus along the entire length of the LAD and large diagonal arteries

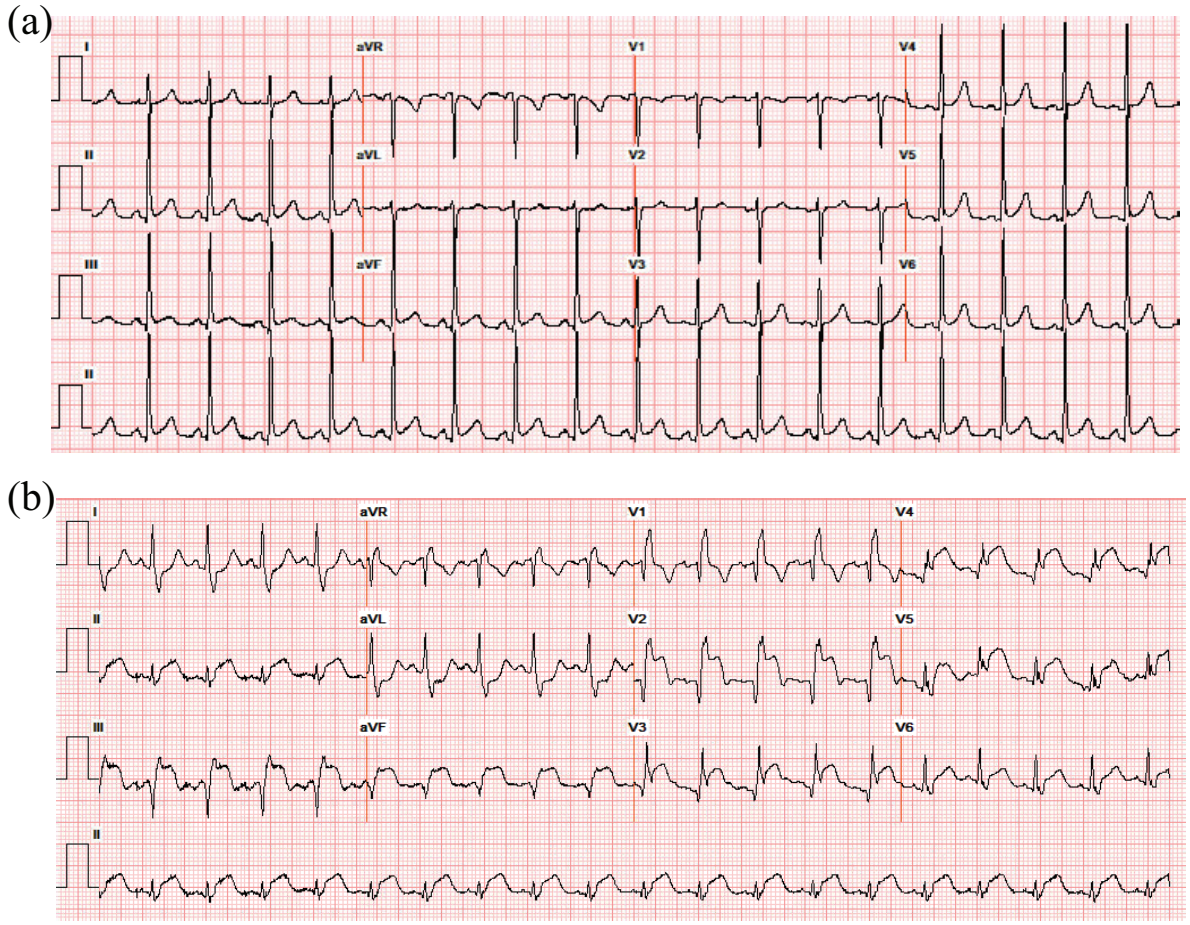

FIGURE 1 Electrocardiogram (a) on admission showing sinus tachycardia and left ventricular hypertrophy and (b) during chest pain, showing new right bundle branch block and diffuse ST-segment elevations

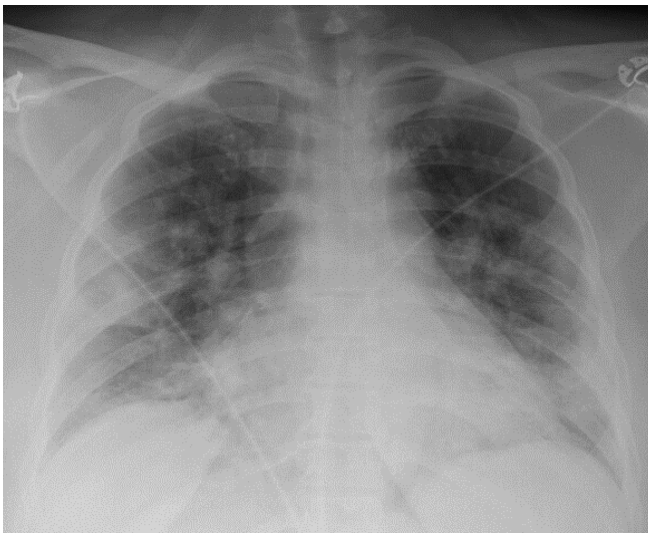

FIGURE 2 Chest X-ray on admission showing bilateral airspace opacities

(Figure 4). Subsequent interventions were performed with intracoronary nitroprusside pretreatment. Mechanical powered aspiration thrombectomy was performed with no improvement in thrombus burden. Alteplase 5 mg was administered into the LAD via a guide extension catheter with an occlusive balloon inflated distally. After 5 min, repeat angiogram demonstrated persistent thrombus. Repeat mechanical powered aspiration thrombectomy was performed with resolution of thrombus in the proximal and mid LAD but there remained persistent thrombus in the distal segment of the LAD. After intubation due to refractory hypoxia, a second wire was placed in the left circumflex (LCx) artery and 3.5 mm drug-eluting stent was deployed in the proximal LAD and postdilated with a 4.0-mm non-compliant balloon. Thrombus embolization was noted, thereafter, in the distal LCx artery (Figure 5a). A catheter was brought proximal to the thrombus to deliver 5 mg of alteplase in the LCx. Due to persistent hypotension, an intra-aortic balloon pump (IABP) was placed. Subsequent reimaging noted new thrombus formation in the recently implanted LAD stent despite adequate activated clotting time throughout the procedure (Figure 5b). Additional intracoronary alteplase 5 mg was administered with subsequent resolution of the recurrent proximal thrombus (Figure 5c). Intracoronary eptifibatide, nitroprusside, and epinephrine were administered in the distal coronary bed of the LAD and LCx. Despite all of these efforts, there remained no reflow in the distal coronary bed of the LAD and LCx at the conclusion of the procedure.

TABLE 1 Laboratory values on admission and within 24 hr of STEMI

	Results on admission prior to STEMI	Results within 24 hr of STEMI	Reference range
Creatinine (mg/dl)	0.73	3.4	0.5–1.2
ALT (U/L)	38	735	0–33
AST (U/L)	90	2,554	5–32
WBC ($\times 10^3$/mcl)	7.8	13.3	4.8–10.8
Hemoglobin (g/dl)	12.7	10.1	12–16
Hematocrit (%)	41.3	32.3	37–47
Platelets ($\times 10^3$/mcl)	377	342	150–450
Lactate, arterial (mmol/L)	1.6	4.9	1.0–1.9
Ferritin (ng/ml)	186	2,694	15–150
hs-CRP (mg/L)	35	194	≤5
ESR (mm/hr)	57	—	0–20
D-dimer (ng/ml)	788	7,658	0–243
LDH (U/L)	610	>4,200	135–214
IL-6 (pg/ml)	347	—	0–15.5
Procalcitonin (ng/ml)	0.67	—	0.002–0.20
Troponin T (ng/ml)	<0.010	—	≤0.010
Troponin I (ng/ml)	—	416[a]	0–0.060
COVID PCR	Positive	—	Not detected

Abbreviations: ALT, alanine transaminase; AST, aspartate aminotransferase; COVID, coronavirus disease; ESR, erythrocyte sedimentation rate; hs-CRP, high-sensitivity C-reactive protein; IL, interleukin; LDH, lactate dehydrogenase; PCR, polymerase chain reaction; STEMI, ST-segment elevation myocardial infarction; WBC, white blood cell.

[a]Peak troponin I was 837 ng/ml.

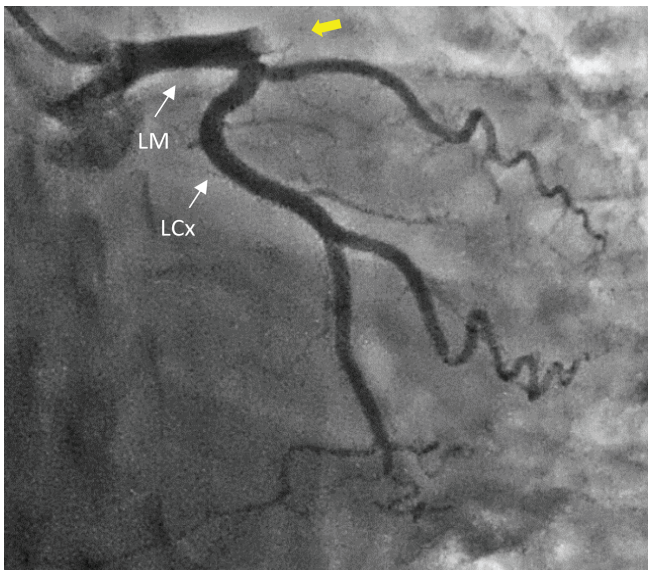

FIGURE 3 Initial coronary angiogram showing thrombotic occlusion of the left anterior descending artery (arrow). LCx, left circumflex; LM, left main

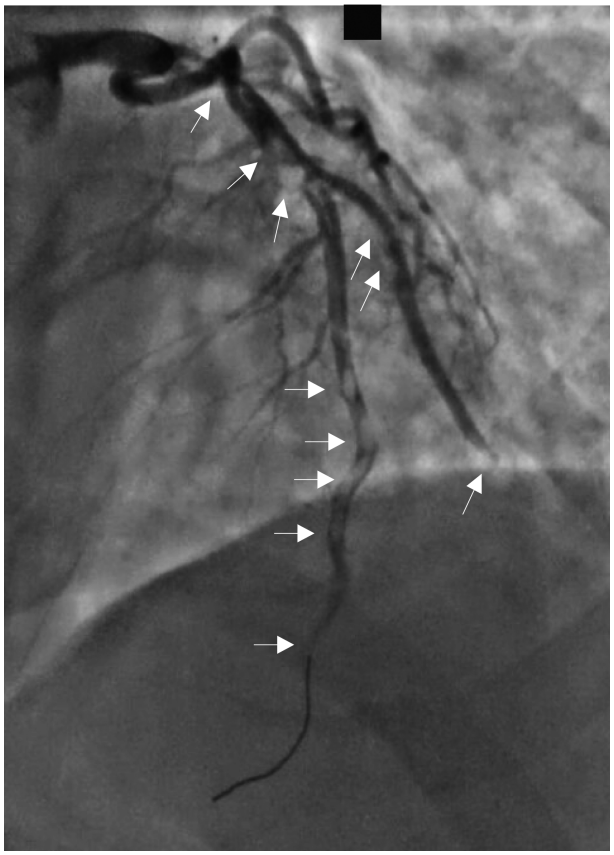

FIGURE 4 Coronary angiogram after 2.0 mm balloon inflation, showing extensive thrombus in the left anterior descending artery and diagonal branch (arrows)

Invasive hemodynamics measurement revealed a cardiac index of 1.7 L^{-1} min^{-1} m^{-2} despite the use of IABP and norepinephrine. Bedside echocardiogram showed akinesis of the LV apex, anteroseptum, and anterior wall, and diffuse hypokinesis of the other segments with an estimated ejection fraction of 20% and no LV thrombus. The IABP was upgraded to an Impella device (Abiomed, Danvers, Massachusetts). The patient was transferred to the intensive care unit for management of cardiogenic shock and COVID-19 pneumonia. Inflammatory markers rose precipitously (Table 1) and the patient died 48 hr after PCI due to mixed (predominately cardiogenic vs. septic) shock.

3 | DISCUSSION

The severe acute respiratory syndrome coronavirus 2, which causes the highly contagious COVID-19, emerged in Wuhan, China, and spread internationally at an alarmingly rapid rate. The World Health Organization declared COVID-19 a pandemic on March 11, 2020 as healthcare systems across the globe became overwhelmed by the disease. Fever and cough are the most common symptoms, present in greater than 80 and 60% of patients, respectively.[1] Less common symptoms include headache, myalgias, fatigue, diarrhea, vomiting, and

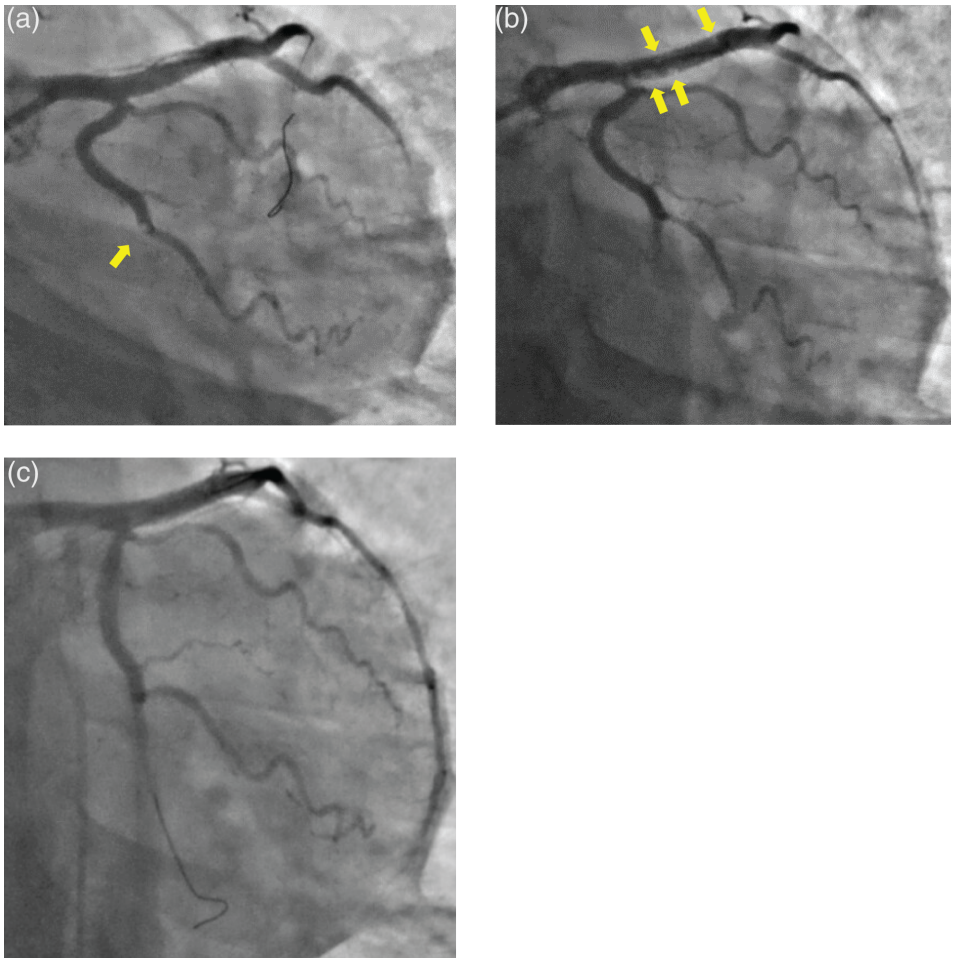

FIGURE 5 Angiogram of (a) the left circumflex artery showing distal occlusion by thrombus embolization (arrow), (b) implanted drug-eluting stent (arrows denote thrombus), and (c) final result

loss of sense of smell and taste. Cardiovascular manifestations are well-recognized complications of COVID-19 and range from minimal cardiac biomarker elevations to acute myocardial infarction (AMI) fulminant myocarditis, and cardiogenic shock.

Establishing a diagnosis of AMI due to coronary thrombosis is particularly challenging in patients with COVID-19, who often manifest electrocardiographic ST-segment changes and regional echocardiographic abnormalities consistent with AMI in the absence of coronary occlusion.[2,3] Other than AMI, two patterns of myocardial injury have emerged. One manifests as a small rise in troponin level (2.5–4.4 pg/ml) in survivors of COVID-19, and a more pronounced rise (290 pg/ml) in troponin level and inflammatory biomarkers in non-survivors.[4] A second pattern is characterized by viral myocarditis or stress cardiomyopathy, manifesting as elevated cardiac biomarkers (troponin and N- terminal pro b-type natriuretic peptide) and LV dysfunction.[4] In our case, the patient presented with diffuse ST-segment elevations and acute thrombosis in one vessel.

The pathogenesis of cardiac injury in patients with COVID-19 is complex and only partially understood. Proposed mechanisms include uncontrolled release of pro-inflammatory cytokines, hypoxemia causing direct damage to cardiac myocytes, and direct endothelial damage by viral cells.[5,6] Acute infections are known triggers of AMI by a variety of mechanisms, including coronary vasoconstrictor, increased platelet activity, endothelial dysfunction, and generalized inflammation leading to a prothrombotic state.[7] Several derangements of hemostatic parameters and venous and arterial thrombotic complications are associated with COVID-19.[8] Elevated D-dimer, prolonged coagulation parameters, and low fibrinogen levels consistent with disseminated intravascular thrombosis have been observed in COVID-19 and are associated with poor prognosis.[9] Finally, cardiac injury in patients hospitalized with COVID-19 is associated with acute respiratory distress syndrome, acute kidney injury, electrolyte disturbances, and greater risk of inhospital mortality.[10]

The management of STEMI in patients with COVID-19 remains controversial, with some regions advocating for initial treatment with thrombolytic therapy in patients without contraindications.[11] However, the United States Society statements advocate against routine IV thrombolytic therapy due to associated bleeding risks, particularly in the potential setting of myocarditis, unless timely PCI is not feasible.[12] Given the very high burden of thrombus in a portion of COVID-19 STEMIs, intracoronary thrombolytic therapy, though, may play a pivotal role. Subsequent to the case illustrated in this report, we successfully treated another patient with COVID-19 and inferior STEMI with high thrombus burden and cardiogenic shock. In this case, unfractionated heparin and IV antiplatelet agents were administered and a 2.0-mm compliant balloon was used to establish flow but extensive thrombus was noted. We subsequently used intracoronary

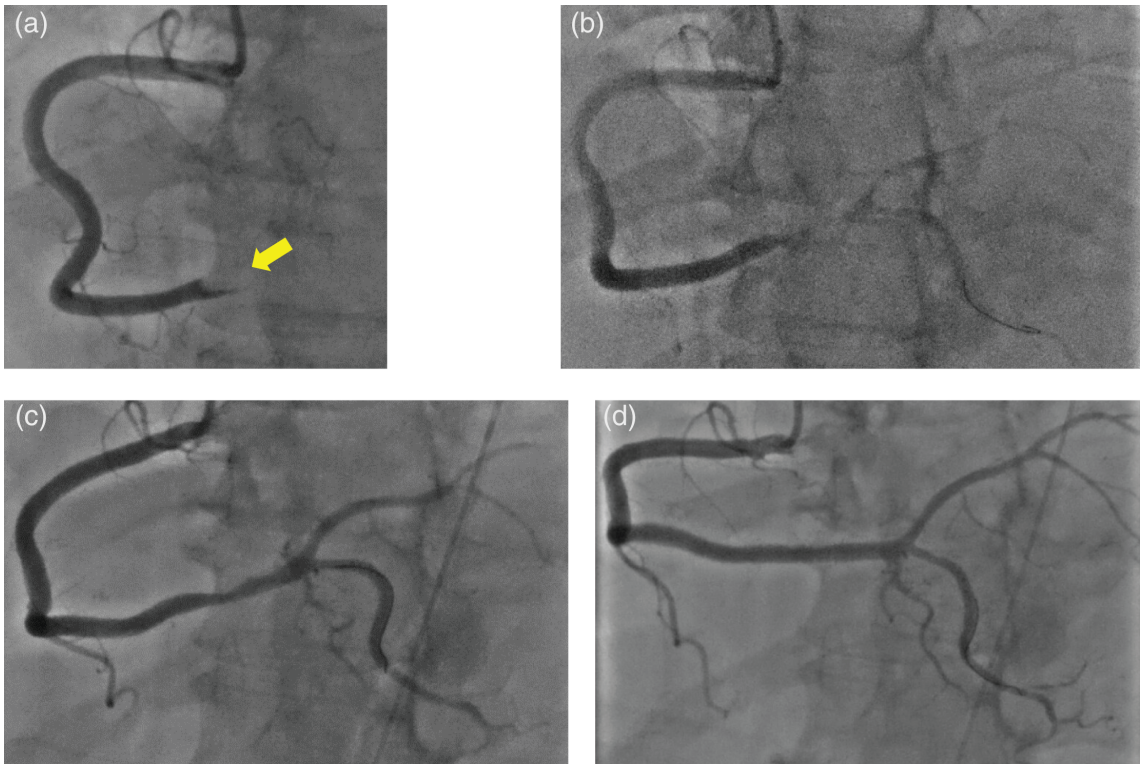

FIGURE 6 Angiogram of the right coronary artery: (a) initial (arrow denotes occlusion), (b) following a 2.0-mm balloon and administration of unfractionated heparin and intravenous antiplatelet agents, (c) following intracoronary tPA and mechanical powered aspiration thrombectomy, and (d) final results. tPA, tissue plasminogen activator

thrombolytic therapy with excellent results (Figure 6). To date, in the absence of clinical studies, no consensus exists on the optimal antiplatelet and antithrombotic regimen in patients with STEMI and COVID-19 who undergo PCI. The case presented in this report illustrates the challenges in treating extensive coronary thrombosis in patients with STEMI and COVID-19.

4 | CONCLUSIONS

Increased thrombotic risk is a well-recognized complication of COVID-19. The management of AMI complicated by extensive thrombus burden in patients with COVID-19 is unclear, and presents a particular challenge with regards to procedural technique and choice of adjunct pharmacotherapy.

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CONFLICT OF INTEREST

R. H., S. B., and E. C. declare no conflict of interest. B. S. reports grants from the VA Office of Research and Development and NIH/NHLBI; is on advisory board for Philips Volcano and Radux Medical; and is a consultant for Terumo Medical.

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