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CASE REPORT

WILEY

Management of a patient presenting with anterior STEMI with concomitant COVID-19 infection early in the course of the U.S. pandemic

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Abstract

The coronavirus disease-2019 (COVID-19) is a viral illness with heterogenous clinical manifestations, ranging from mild symptoms to severe acute respiratory distress syndrome and shock caused by the severe acute respiratory syndrome coronavirus-2. The global healthcare community is rapidly learning more about the effects of COVID-19 on the cardiovascular system, as well as the strategies for management of infected patients with cardiovascular disease. There is minimal literature available surrounding the relationship between COVID-19 infection and acute coronary syndrome. We describe the case of a woman who presented with an acute anterior ST-elevation myocardial infarction managed by primary percutaneous coronary intervention, who subsequently developed severe COVID-19 infection and ultimately succumbed to multisystem organ failure.

KEYWORDS

cytokine Storm, myocardial infarction, revascularization, SARS-CoV-2

1 | INTRODUCTION

A 79-year-old woman with a history of dyslipidemia and diabetes mellitus developed substernal chest pressure associated with nausea and lightheadedness while carrying groceries up her stairs on the afternoon of March 10, 2020. She had been experiencing low-grade fevers (100.0°F) the day prior to presentation with no other symptoms. Her chest pressure persisted through the day and she attributed it to indigestion. After going to bed and waking up the following morning continued chest discomfort, she presented to a nearby non-percutaneous coronary intervention (non-PCI) capable critical access hospital and a 12-lead ECG was performed revealing Q-waves and ST elevations in the precordial leads consistent with a late presenting anterior ST-elevation myocardial infarction (STEMI) (Figure 1). She was noted to have a fever (temperature 100.8 F) upon presentation. The differential diagnosis included STEMI from acute plaque rupture, as well as other STEMI mimickers including viral perimyocarditis, coronary embolism, and stress cardiomyopathy.

She was treated with aspirin, clopidogrel, and heparin and then transferred to our PCI capable facility for urgent coronary angiography. The projected door to device time was 150 min. Fibrinolytic therapy was deferred based upon the delay in her presentation, advanced age and minimal expected benefit as shown in Figure 2.

The patient resided in the state of Vermont. As of the date of presentation, there was only one known case of coronavirus disease-2019 (COVID-19) infection in the state (located 50 miles from the patient's residence) so no additional precautions were taken during transport or upon arrival to the cardiac catheterization laboratory.

2 | CASE SERIES

On arrival in the catheterization laboratory, she was hemodynamically stable, oxygenating well on room air and had no chest pain. Coronary angiography performed via the right radial approach revealed a culprit 100% obstructive lesion in the mid left anterior descending artery

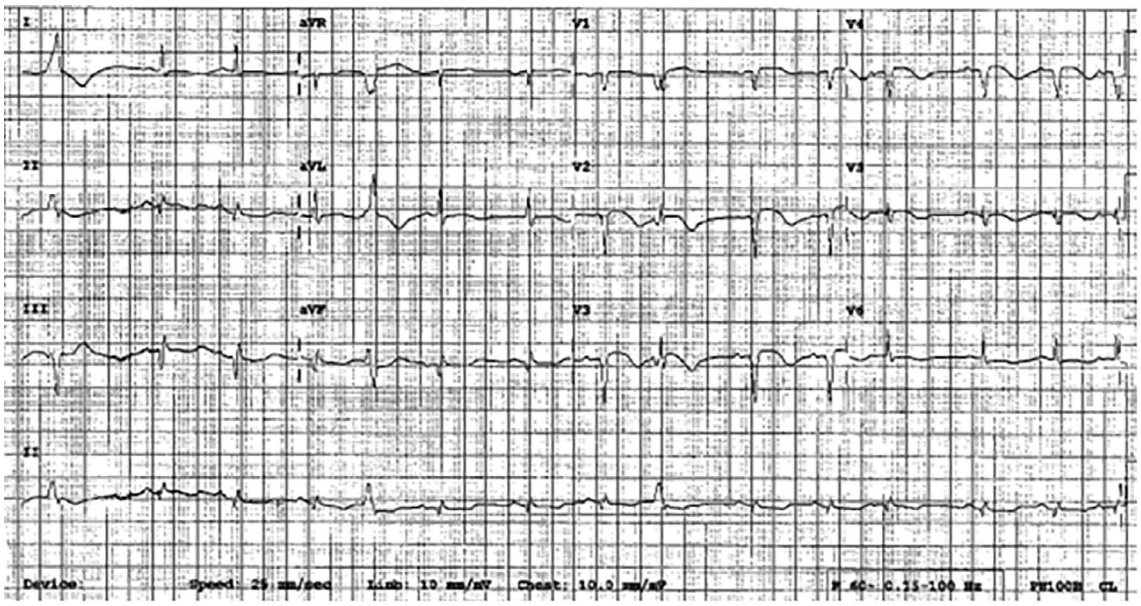

FIGURE 1 Presenting ECG

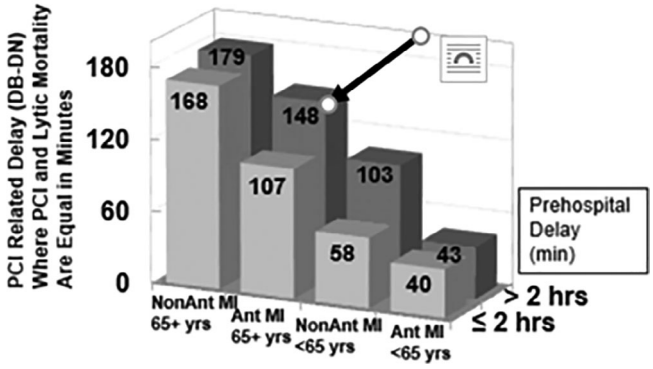

FIGURE 2 Door to needle versus door to balloon equipoise times at Dartmouth-Hitchcock Medical Center stratified by time to presentation, age and MI location[1]

(LAD) with apparent thrombus and a nonculprit 80% obstructive lesion in the left circumflex artery (LCX) as shown in Figure 3. The left ventricular end diastolic pressure was 24 mmHg.

Following discussion with the attending inpatient cardiologist, the decision was made to defer revascularization of the completely occluded LAD based on an estimated ischemic time exceeding 24 hr combined with the absence of ongoing chest discomfort, and instead the nonculprit LCX was intervened upon in the hopes of addressing postinfarction angina and potentially improving mortality.[2,3] The LCX lesion was wired, and a drug-eluting stent was placed and postdilated to optimize expansion as shown in Figure 4.

She remained hemodynamically stable and chest pain free throughout the procedure and was transferred to the cardiac critical care unit for further management. Physical exam at this time revealed no clinical signs of heart failure. Post-PCI transthoracic echocardiography images revealed moderately reduced global left ventricular systolic function with an ejection fraction of 38% and wall motion abnormalities predominantly in the territory supplied by the LAD. The estimated pulmonary artery systolic pressure was 40 mmHg (Figure 5). Labs obtained on admission are displayed in Table 1.

The patient remained stable initially following intervention. She had marked improvement in her postinfarction angina when ambulating. She was initially diuresed and started on appropriate evidence-based postinfarction medication, including an ACE inhibitor (lisinopril), beta blocker (metoprolol), and statin (atorvastatin). Dual antiplatelet therapy was continued.

Over the first 3 days following admission (hospital Days 1–3), she continued to develop intermittent fevers (highest temperature 101.7 F), which were attributed to an inflammatory postmyocardial infarction syndrome.[4] She continued to subjectively improve and maintained normal oxygen saturations on room air through hospital Day 3, at which point she developed a cough. Blood cultures showed no preliminary growth; however, a chest X-ray revealed new patchy airspace opacities in the left lung suggestive of a multifocal pneumonia (Figure 6).

Her white blood cell count remained within normal limits at 9.0 (x 10^3/mcl); the lymphocyte count remained low at 0.5 (x 10^3/mcl). Legionella urinary antigen was negative and respiratory viral PCR panel showed no evidence of infection with any common pathogens (including mycoplasma). At this time, it was reported that another person in her small town had been admitted to the nearby VA Medical Center with a diagnosis of severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection.

Over the following 2 days (hospital Days 4 and 5), she developed myalgias and hypoxemia (O_2 saturation of 75% requiring 2 L of O_2 delivered via nasal cannula). She was further diuresed, but developed worsening hypotension on hospital Day 5, prompting administration of IV fluids and cessation of her ACE inhibitor and beta blocker. Over

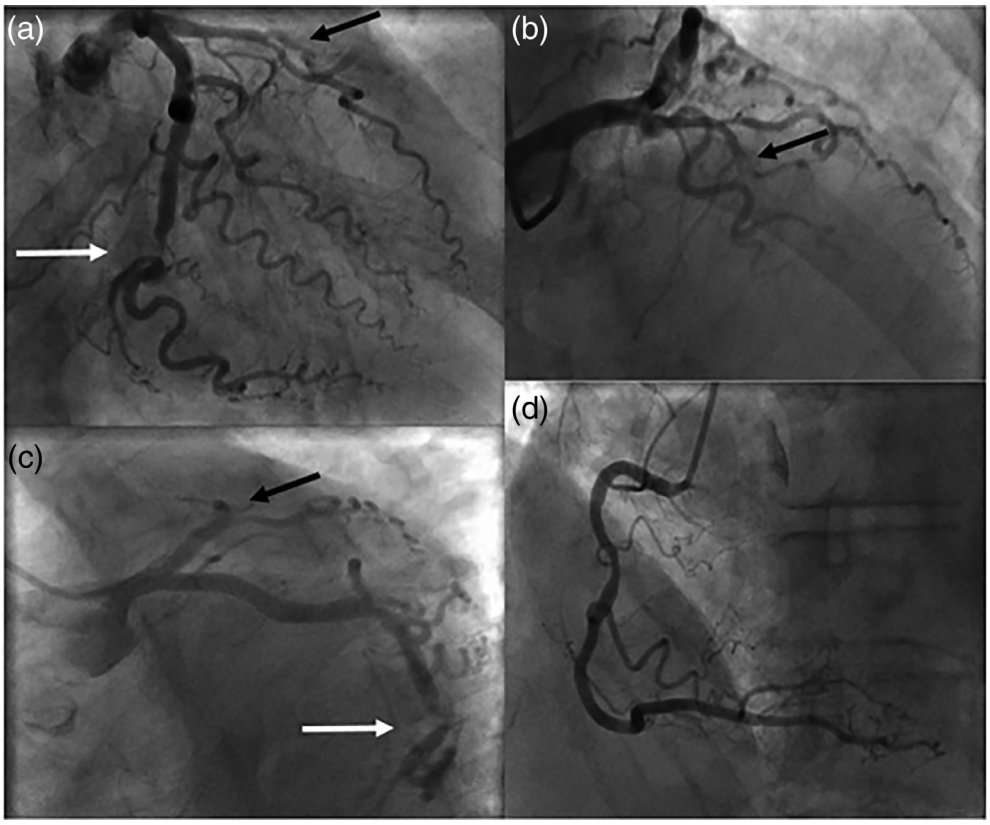

FIGURE 3 Initial angiography from multiple projections of the left coronary system (a–c) revealing the culprit 100% occlusion of the mid left anterior descending artery (black arrows), severe obstructive atherosclerotic disease of the mid left circumflex artery (white arrows), and mild diffuse atherosclerotic disease of the entire right coronary artery (d)

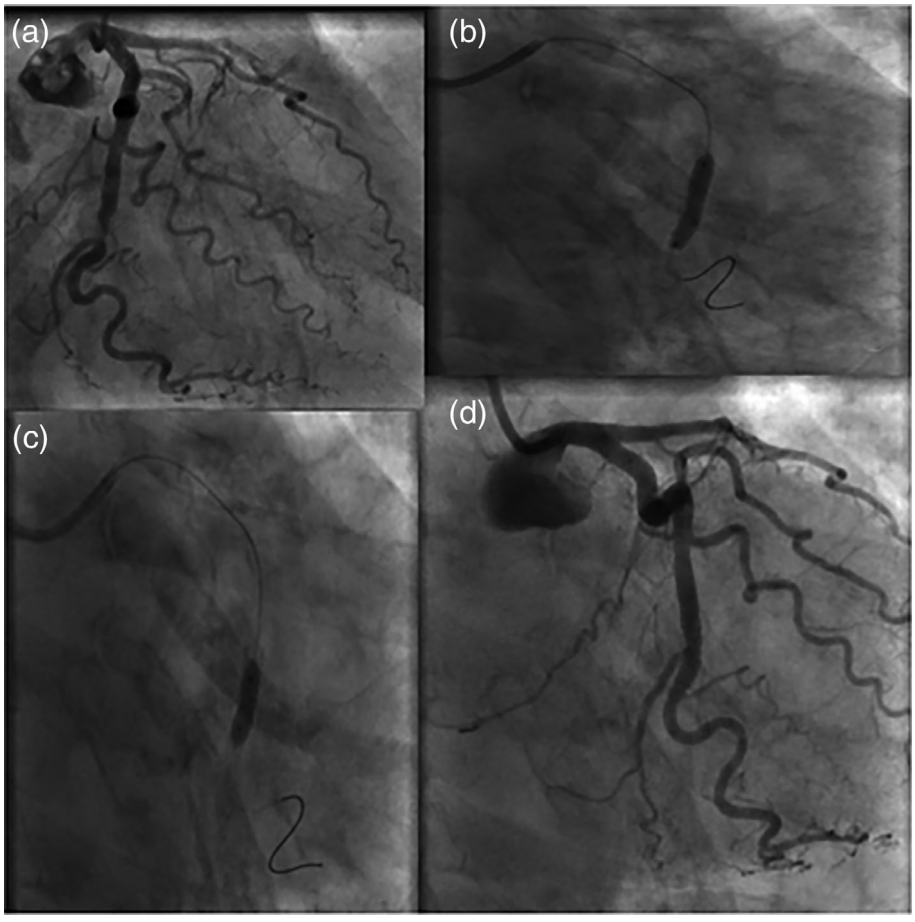

FIGURE 4 Representative images taken from the right anterior obliqueand AP caudal projections demonstrating percutaneous coronary intervention of the mid left circumflex artery lesion, With preintervention angiograms (a), Drug eluting stent deployment (b), Stent post deployment dilation (c), and Final angiogram (d)

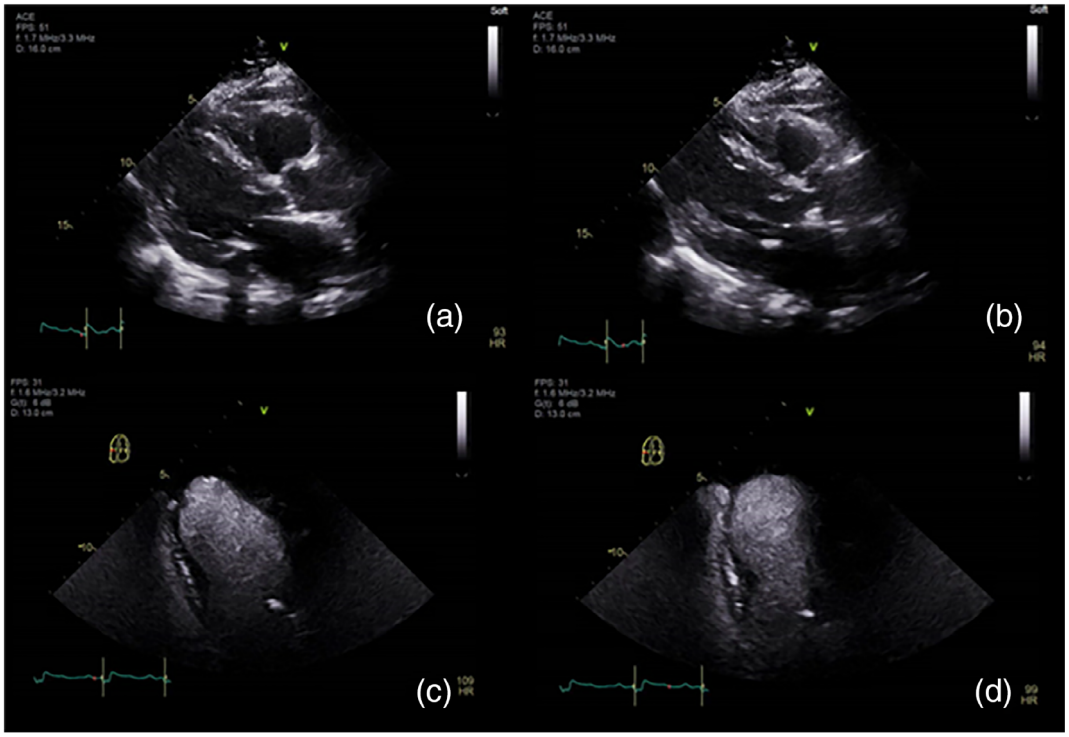

FIGURE 5 Representative parasternal long axis (a and b) and contrast enhanced apical four chamber (c and d) transthoracic echocardiography images taken at end diastole (a and c) and end systole (b and d) depicting wall motion abnormalities in the apex

TABLE 1 Admission laboratory values

Admission laboratory values		
	Value	**Normal ranges**
White blood cells	9.2 ($\times 10^3$/mcl)	
Hemoglobin	12.9 (gm/dl)	
Platelets	213 ($\times 10^3$/mcl)	
Lymphocytes	**0.7 ($\times 10^3$/mcl)**	0.9–3.2 ($\times 10^3$/mcl)
Sodium	134 (mmol/L)	135–145 (mmol/L)
Potassium	4.3 (mmol/L)	
Creatinine	0.76 (mg/dl)	
Troponin-T	**3.6 (ng/ml)**	< 0.01 (ng/ml)
CK	**2,540 (unit/L)**	0–160 (unit/L)
AST	**290 (unit/L)**	0–30 (unit/L)
ALT	**67 (unit/L)**	0–30 (unit/L)

Note: Abnormal values are in bold.

the course of the day, she became increasingly hypotensive, requiring vasopressor support (low dose phenylephrine). She became increasingly hypoxic requiring increasing supplemental oxygen and subsequent intubation and mechanical ventilation using an acute respiratory distress syndrome (ARDS) protocol (volume control using tidal volumes of 6 ml/kg of ideal body weight, PEEP of 12 cm H_2O and FIO2 of 40%). Her pulmonary compliance was reassuring, with an inspiratory plateau pressure of 21 cm H_2O and driving pressure of 9 cm H_2O. On the evening of the fifth hospital day, her test for SARS-CoV-2 infection returned positive. She was noted to have a profound inflammatory response as demonstrated by a CRP of 193 mg/L (normal < 5 mg/L) and was started on hydroxychloroquine in an effort to modulate her immune response. Steroids, IL-6 antagonists (tocilizumab) and antiviral agents (remdesivir) were deferred.

Unfortunately over the following day (hospital Day 6), she developed worsening hemodynamic instability with distributive shock requiring escalation of the vasopressor regimen (high dose norepinephrine and vasopressin) as well as worsening oliguric renal failure. Empiric antibiotics were started to treat a possible secondary bacterial pneumonia. Further definition of her hemodynamics with a PA catheter was considered, but risks to the patient and hospital personnel were felt to outweigh potential benefits. Central venous saturations remained in the range of 70–75% (without inotropic or mechanical circulatory support), suggesting that peripheral vasodilation rather than impaired cardiac output was the cause of her hypotension. Her renal function continued to deteriorate over the course of her hospitalization and after multiple conversations with her family regarding her prognosis, her goals of care were shifted to comfort measures only and she died.

3 | DISCUSSION

Cardiac injury attributed to infection with SARS-CoV-2 has been reported in early cohort studies from China and carries a markedly

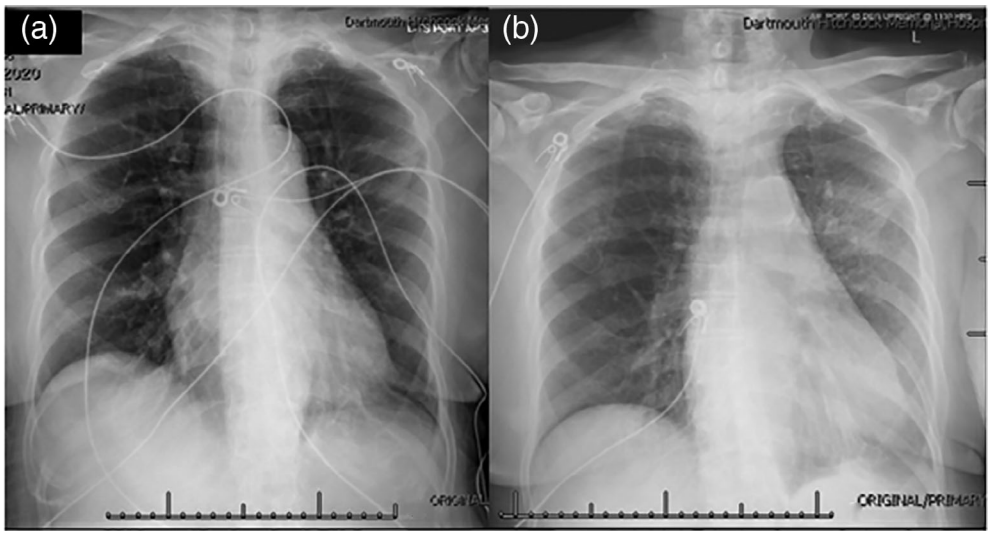

FIGURE 6 Admission CXR (a) showing no acute cardiopulmonary process compared to CXR from hospital Day 3 (b) showing multiple left sided patchy airspace opacities in an atypical distribution

elevated risk of mortality compared to those patients without cardiac injury (51.2 vs. 4.5%, $p < .001$).[5] There are multiple proposed mechanisms regarding cardiac involvement in the setting of COVID-19 infection, with the two most commonly cited being:

1 Primary cardiomyocyte involvement mediated by spike protein binding to ACE2 (highly expressed in the heart) leading to myocarditis.
2 Secondary cardiac injury and stress cardiomyopathy due to a combination of ARDS, systemic inflammatory response syndrome and shock.

To our knowledge, there are no published cases of infection with the SARS-CoV-2 virus occurring simultaneously with an acute coronary syndrome (ACS). While there have been published cases of patients with ST elevations on ECG and no obstructive disease on coronary angiography,[6] there are currently only anecdotes shared on social media pointing to plaque-rupture-mediated MI in the setting of COVID-19 infection.[7] It is well understood that immune dysregulation and the enhanced expression of pro-inflammatory cytokines are directly related to illness severity and clinical outcomes in influenza infection,[8] and now COVID-19 infection.[9] Moreover, the concept that inflammation is a primary driver of atherosclerosis and atherogenesis is supported by several large randomized clinical trials including JUPITER[10] and CANTOS.[11] Additionally, it has been shown that an increased inflammatory burden is a significant risk factor for development of MI in patients with autoimmune diseases such as rheumatoid and psoriatic arthritis, and that reduction in inflammation is associated with a reduction in major adverse cardiac events.[12-14] This case raises the question as to whether the inflammation and cytokine storm that accompany COVID-19 infection leads to enhanced coronary artery plaque susceptibility to rupture in a high flow setting. In addition, increased thrombogenicity driven by inflammation may present another mechanism for acute coronary syndrome due to SARS-CoV-2.

4 | CONCLUSION

Cases of ACS/STEMI occurring concurrently with (and possibly precipitated by) COVID-19 infection are being reported and anecdotally carry a grim prognosis. Further research into the underlying pathophysiology of atherosclerotic plaque rupture and thrombogenesis in the setting of immune dysregulation may help guide management strategies and allow us to better understand potential mechanisms of coronary involvement and acute ischemia in COVID-19 disease, which may compound the already appreciated myocardial injury attributed to SARS-CoV-2.

CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

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REFERENCES

1. Rezaee ME, Conley SM, Anderson TA, Brown JR, Yanofsky NN, Niles NW. Primary percutaneous coronary intervention for patients presenting with ST-elevation myocardial infarction: process improvements in rural prehospital care delivered by emergency medical services. Prog Cardiovasc Dis. 2010;53(3):210-218.
2. Mehta SR, Wood DA, Storey RF, et al. Complete revascularization with multivessel PCI for myocardial infarction. N Engl J Med. 2019; 381(15):1411-1421.
3. Menon V, Pearte CA, Buller CE, et al. Lack of benefit from percutaneous intervention of persistently occluded infarct arteries after the acute phase of myocardial infarction is time independent: insights from occluded artery trial. Eur Heart J. 2009;30(2):183-191.
4. Kawashima C, Matsuzawa Y, Akiyama E, et al. Prolonged fever after ST-segment elevation myocardial infarction and long-term cardiac outcomes. J Am Heart Assoc. 2017;6(7):e005463.
5. Shi S, Qin M, Shen B, et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. JAMA Cardiol. 2020. https://doi.org/10.1001/jamacardio.2020.0950.

6. Inciardi RM, Lupi L, Zaccone G, et al. Cardiac involvement in a patient with coronavirus disease 2019 (COVID-19). JAMA Cardiol. 2020. https://doi.org/10.1001/jamacardio.2020.1096.
7. Kirtane AA. Interventionalists, this will happen in your lab! https://twittercom/search?q=%23RadialFirst&src=tyah. Accessed 2020.
8. Wang Z, Zhu L, Nguyen THO, et al. Clonally diverse CD38+HLA-DR+CD8+T cells persist during fatal H7N9 disease. Nat Commun. 2018;9(1):824.
9. Thevarajan I, Nguyen THO, Koutsakos M, et al. Breadth of concomitant immune responses prior to patient recovery: a case report of non-severe COVID-19. Nat Med. 2020;26:453-455.
10. Ridker PM, Danielson E, Fonseca FAH, et al. Rosuvastatin to prevent vascular events in men and women with elevated C-reactive protein. N Engl J Med. 2008;359(21):2195-2207.
11. Ridker PM, Everett BM, Thuren T, et al. Antiinflammatory therapy with canakinumab for atherosclerotic disease. N Engl J Med. 2017;377(12):1119-1131.
12. Yang ZS, Lin NN, Li L, Li Y. The effect of TNF inhibitors on cardiovascular events in psoriasis and psoriatic arthritis: an updated meta-analysis. Clin Rev Allergy Immunol. 2016;51(2):240-247.
13. Roubille C, Richer V, Starnino T, et al. The effects of tumour necrosis factor inhibitors, methotrexate, non-steroidal anti-inflammatory drugs and corticosteroids on cardiovascular events in rheumatoid arthritis, psoriasis and psoriatic arthritis: a systematic review and meta-analysis. Ann Rheum Dis. 2015;74(3):480-489.
14. Lee JL, Sinnathurai P, Buchbinder R, Hill C, Lassere M, March L. Biologics and cardiovascular events in inflammatory arthritis: a prospective national cohort study. Arthritis Res Ther. 2018;20(1):171.

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