A 71-year-old well controlled hypertensive female presented on day 4 of acute COVID-19 illness with atypical chest pain and worsening exertional dyspnea. On examination, heart rate was 110 beats/min, blood pressure was 125/82 mmHg and there were decreased heart sounds with visible jugular venous distention. She was not in respiratory distress at rest and was not requiring supplemental oxygen. Chest radiography showed bilateral diffuse opacities (Figure 1). Transthoracic echocardiography (TTE) confirmed a moderate pericardial effusion with right ventricular systolic compression, paradoxical right ventricular septal motion, end-diastolic right atrial collapse, and a plethoric inferior vena cava (IVC) with no respiratory variation. Her ejection fraction (EF) was 55%. Initial workup revealed mildly elevated troponin T levels of 0.14 ng/mL, NT-Pro BNP of 2500 pg/mL. Initial EKG showed a low voltage sinus rhythm without ischemic features (Figure 2). Given the patient’s hemodynamic stability, echocardiographic lack of right ventricular collapse and posterior location of maximal effusion, she was initially managed medically with intent to consider surgical pericardial window if there was no improvement. Intravenous fluids, high dose aspirin, colchicine, and systemic steroids were initiated. However, over the ensuing 12 h the patient developed worsening hypotension with development of a friction rub on examination. Repeat EKG then showed diffuse ST elevation most prominent in the anterior and lateral leads (Figure 3). Serial biomarkers showed a rising troponin T to 1.5 ng/mL with developing lactic acidosis. Point-of-care echocardiography revealed a worsening pericardial effusion, severely impaired right ventricular (RV) function, end diastolic collapse of the right atrium, and a newly reduced EF of 30%–35% with global hypokinesis (Video 1). The interventional cardiology team was called for emergent pericardiocentesis however prior to intervention the patient went into cardiac arrest with pulseless electrical activity. The return of spontaneous circulation was achieved after three cycles of advanced cardiac life support (ACLS) and emergent bedside subxiphoid pericardiocentesis with a total of 200 mL of fluid removed. Repeat bedside echocardiography showed resolution of the effusion and an EF of 5%–10%. Soon afterwards the patient had another cardiac arrest, refractory to optimal ACLS, and expired within 24 hours of presentation.

A 51 year old obese, hypertensive, African American female presented with pleuritic chest pain and worsening dyspnea on exertion. She had been on
day 5 of COVID-19 illness. At presentation, heart rate was 115 beats/min, blood pressure was 95/50 mmHg and cool extremities were noted. EKG was significant for low voltage complexes and diffuse ST elevations (Figure 4). Initial troponin T was 0.93 ng/mL. CT chest revealed scattered, patchy, bilateral ground glass opacities (Figure 5). TTE revealed a moderate effusion with inflammatory exudate, late diastolic collapse of the right atrium, compression of the right ventricle, excessive respiratory variation of the mitral and tricuspid valves, and an EF of 20% (Video 2). High flow intravenous fluids were initiated with stabilization of her blood pressure and multidisciplinary team involving interventional cardiology and cardiothoracic surgery were planning a pericardial window the following day. However, at 12 h of admission to the ICU, the patient developed a ventricular fibrillation cardiac arrest. Optimal ACLS was briefly unsuccessful as despite defibrillation and emergent bedside pericardiocentesis there was subsequent refractory Pulseless electrical activity arrest and the patient expired at 13 h of presentation. COVID-19 most commonly involves the respiratory system, however there is growing evidence that cardiac involvement is very common, especially in hospitalized patients. One such entity that has been described is COVID-19 myocarditis. Myocarditis is defined as inflammation of the heart muscle, and is commonly caused by viral infections. Viral myocarditis has been recognized as a cause of congestive heart failure for many years and a number of different viruses have been implicated as the
cause including adenovirus, enteroviruses (coxsackievirus) and parvovirus.\cite{2} As the COVID-19 pandemic evolves, more and more cases of COVID-19 myocarditis have been reported. While the exact incidence is unknown, some reports estimate that 7% of all COVID-19 deaths may be attributable to myocarditis (these patients were diagnosed clinically and without tissue confirmation).\cite{3} One proposed mechanism of the pathophysiology of COVID-19 myocarditis involves the ACE-2 receptor which can be found on cardiomyocytes among other cells, which SARS-CoV-2 utilizes for cell entry. Intracellular SARS-CoV-2 might impair stress granule formation via its accessory protein. Without the stress granules, the virus replicates and damages the cell. Primed CD8+ T lymphocytes migrate to the cardiomyocytes and cause myocardial inflammation through cell-mediated cytotoxicity. In the cytokine storm syndrome, in which pro-inflammatory cytokines are released into the circulation, T-lymphocyte activation is augmented and releases more cytokines. This results in a positive feedback loop of immune activation and myocardial damage.\cite{3} This is similar to typical viral myocarditis which occurs in phases, with phase 1 involving viral replication, and phase 3 involving the immune system infiltrating the myocytes in order to clear the infection, unfortunately it is this phase that has been implicated in the congestive heart failure that can sometimes follow the acute infection.\cite{2} COVID-19 virus has yet to be obtained via a tissue biopsy in these patients, which may lead one to believe that the inciting inflammatory response is the culprit.

Commonly in myocarditis, the pericardium becomes inflamed resulting concurrent pericarditis which can then lead to an effusion. Cardiac tamponade is potential condition that can occur with sudden and/or excessive accumulation of fluid in the pericardial space. This can restrict the appropriate filling of the cardiac chambers which disrupts normal hemodynamics and decreases cardiac output. Typically, if the fluid accumulates slowly it takes a big effusion to cause cardiac tamponade, however if an effusion accumulates at a rate where the myocardium cannot accommodate the increased pericardial pressure, then tamponade can occur from even very
small effusions. Cardiac tamponade is a clinical diagnosis, classic physical findings in are included in Beck’s triad (hypotension, jugular venous distension, and muffled heart sounds). Pulses paradoaxes may also be present. EKG findings of diffuse ST elevation, PR depressions, and low voltage QRS complexes can also be seen. If tamponade is suspected then a TTE should be performed. Classic echocardiographic findings of tamponade include collapse of the RA and the RV. This happens during their relaxation phase when intra-chamber pressures are lower than pericardial pressures. Atrial collapse is usually observed before ventricular collapse in progressive cardiac tamponade. Additional findings include a plethoric IVC, increased respiratory variation in the mitral and tricuspid valves, and septal bowing. If clinical suspicion is high, then emergent pericardiocentesis or pericardial window should be performed as soon as possible.

In conclusion, COVID-19 has only been reported in a handful of case reports as being a cause of cardiac tamponade. COVID-19 has been reported to trigger an exaggerated systemic inflammatory response in patients and has been implicated in many cases of myocarditis/pericarditis which makes the proposed mechanism of this disease process very plausible. It is vitally important that tamponade remains in your differential in COVID-19 patients as it can cause disastrous hemodynamic collapse very quickly in an already critically ill patient population.

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