HEMORRHAGIC CARDIAC TAMPONADE IN A PATIENT WITH COVID-19 AND END STAGE RENAL DISEASE

Mojtaba Mirzaei, MD, MPH1, Aneil Bhalla MD1
1. Department of Internal Medicine, Yale University School of Medicine

Category: Case Vignettes

Introduction: The COVID-19 pandemic has caused a significant amount of morbidity and mortality in the United States. It has been suggested that after recovery from COVID-19, patients remain at high risk not only for lung disease, but cardiovascular disease. More data is needed regarding the cardiovascular complications of COVID-19.

Case description: A 47-year-old Hispanic male with a past medical history of coronary artery disease status post stents to the LAD and RCA, uncontrolled hypertension, end stage renal disease on peritoneal dialysis, and COVID-19 infection one month prior presented with two days of progressively worsening dyspnea, nausea, and continuous chest pressure. He denied any fevers, chest pain, or medication nonadherence. On admission, he was found to have a blood pressure of 109/54 mmHg while saturating well on room air. Physical exam was significant for clear lungs on auscultation and distant heart sounds. His initial liver function tests were within normal limits but shortly after admission, his AST and ALT increased to 3878 and 2036, respectively. He initially had a mild lactic acidosis of 2.7 which increased to 6.1 during the same time period. A CXR demonstrated cardiomegaly and patchy ground-glass infiltrates. Subsequent imaging with a CT scan of the chest showed a large pericardial effusion.

Given the CT scan findings, relative hypotension, and profound transaminitis indicative of hypoperfusion to the liver, cardiac tamponade was suspected. He underwent emergent echocardiography and pericardiocentesis which yielded 900 cc of sanguineous fluid and resulted in complete symptom relief. Of note, the patient’s systolic blood pressure on arrival to the operating room was 66 mmHg which improved to 110 mmHg after pericardiocentesis.

Discussion: Cases of cardiac tamponade requiring immediate intervention as a complication of COVID-19, although limited, have been documented. Reported cases have been found to occur often in Hispanic or African American middle-aged men, an observation consistent with our patient’s background.

Cardiac tamponade can be caused by the accumulation of either transudative or exudative fluid in the pericardium resulting in decreased venous return, ventricular filling, and cardiac output. Our patient developed a large and rapidly progressive pericardial effusion after infection with COVID-19, suggestive of a possible underlying pericardial inflammatory process. Further studies are warranted to examine the association between COVID-19 infection and the development of hemodynamically significant pericardial effusions and hemorrhagic tamponade.