

POST-VIRAL CARDIAC TAMPONADE IN A PATIENT WITH COVID-19

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Case Vignette

A 21-year-old female with a history of untreated hypothyroidism, asthma, and obesity presented to the emergency department with complaints of shortness of breath, dry cough, and chest wall tenderness. Her dyspnea had been occurring for one day. She also had a one-week history of watery diarrhea, one episode of non-bloody vomiting, persistent nausea, and subjective fevers. She was admitted for a workup of dyspnea. COVID-19 viral PCR, urine Streptococcus and Legionella antigens were negative. Blood and sputum cultures were negative. The patient was given nebulizer treatments and required 6 L of supplemental oxygen via nasal cannula. On the third day of her hospitalization, she desaturated to the mid-80s and was placed on a Venti-mask with little improvement. On exam, the patient was noted to have jugular venous distension and decreased air entry without pericardial rub. Her EKG showed poor R wave progression and low voltage in the precordial leads. CT angiogram was negative for pulmonary embolism; however, it showed multifocal consolidations and a large posterior pericardial effusion. An echocardiogram revealed a 2.5 to 3 cm effusion, diastolic right ventricular collapse, and 50% respiratory variation across the tricuspid inflow. The patient was diagnosed with cardiac tamponade, and a pericardial window was performed. The pericardial fluid was serosanguinous in appearance. Repeat nasopharyngeal COVID-19 viral PCR was positive. Unfortunately, due to laboratory limitations, viral PCR could not be run on the pericardial fluid.

Only a handful of case reports discuss the development of cardiac tamponade in the setting of SARS-CoV-2 infection. The first documented case report of cardiac tamponade in a COVID-19 patient occurred in Italy in February 2020. In that instance, SARS-CoV-2 was detected in the pericardial fluid by rRT-PCR amplification of SARS-CoV-2 RNA. Aside from this case reported in Italy, few other COVID-19 post-viral cardiac tamponade cases have demonstrated the presence of SARS-CoV-2 RNA in the pericardial fluid. Effusions are typically hemorrhagic, which is rare for a viral pericardial effusion, with the exception of coxsackievirus. The proposed mechanism behind post-COVID19 cardiac tamponade is pericardial inflammation caused by direct cytotoxic effects of the virus. Another potential mechanism is systemic inflammation leading to the accumulation of pericardial fluid. Treatment with colchicine is the cornerstone of management. Non-steroidal anti-inflammatory drug (NSAID) use for COVID-19 associated tamponade is often avoided, although studies have not revealed adverse outcomes with the use of NSAIDs in these patients. Although the primary manifestation of COVID-19 infection is respiratory illness, about 12% of patients experience cardiac complications. It is vital to assess for cardiac injury, as cardiac complications are associated with increased mortality in these patients.

References

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