MYOPERICARDITIS AS A RARE PRESENTATION OF COVID INFECTION Joseph Bahgat, MD¹, Andrey Vavrenyuk, MD¹, Zachary Estep, MD¹, Maryana German, APRN², Asha Shah, MD³, Arzhang Fallahi MD², Anna Koulova MD²

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Case Vignettes

Background

Myopericarditis is characterized by inflammation of the myocardial layer as well as concurrent involvement of the pericardium. Myocardial injury in COVID-19 infection is common in approximately 20-30% of hospitalized patients. However, COVID-19 induced myocarditis is an uncommon phenomenon with studies showing only about 5% occurrence in highly selected cases undergoing endomyocardial biopsy. Furthermore, only about 10% of COVID-19 associated myocarditis cases have pericardial involvement with pericardial effusion. The effusions that occur in COVID-19 are typically sterile, suggesting a secondary inflammatory process, rather than pericardial infection. We present a patient with myopericarditis as presenting sign of COVID infection.

Case Presentation

45-year-old female with a five day history of COVID infection presented to the emergency department with progressive chest pain radiating to her left shoulder, worsening dyspnea on exertion, generalized fatigue, myalgias, and low grade fevers. Initial vital signs were afebrile, BP 107/93, HR 110, saturating 99% on room air. Physical examination findings revealed JVD and bibasilar crackles on auscultation. Labs were remarkable for a troponin of 0.69 and BNAT 175. D-dimer was negative. PCR for SARS-CoV-2 was positive. EKG demonstrated sinus rhythm, low voltage, and slight elevation in lead I and aVL. Chest x-ray showed bilateral opacities. Chest CTA was negative for aortic dissection, but revealed a medium sized pericardial effusion as well as bilateral multifocal infiltrates consistent with COVID-19 infection. Transthoracic echocardiogram (TTE) revealed a moderate sized pleural effusion.

Decision Making

In the context of her clinical findings, the patient underwent pericardiocentesis with removal of 300 cc of straw-colored, sterile fluid and was monitored in the ICU. Repeat TTE post-procedure showed complete resolution of the effusion. During admission, she received convalescent plasma, but did not receive steroids or remdesivir given lack of hypoxia and respiratory symptoms. She was discharged on colchicine and NSAIDs. Cardiac MRI is planned to evaluate for residual myocardial inflammation or fibrosis.

Conclusion

Our patient manifested with a predominantly cardiac involvement of COVID-19 infection in the form of myopericarditis which is uncommon. COVID-19 prominently affects the cardiopulmonary system; however, the degree of cardiac involvement is not necessarily related to the severity of respiratory involvement. Much remains unknown about the nature of myocardial injury in patients with COVID-19.