CHEST PAIN AS THE PRESENTING SYMPTOM OF BIOPSY-PROVEN LYMPHOCYTIC MYOCARDITIS

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Case Vignettes

BACKGROUND: Enteroviruses, adenoviruses, and parvovirus B19 have been long associated common causes of lymphocytic myocarditis, and in presentations mimicking acute myocardial infarction – have been implicated in 71% of patients with normal coronary anatomy. Here we present a patient with biopsy-proven lymphocytic myocarditis.

CASE PRESENTATION: 28-year-old female with no significant medical history presented with progressive chest discomfort with radiation to the left shoulder and neck. She had no prodromal symptoms or evidence of infection prior to chest pian. COVID testing was negative, and she received her COVID vaccine 4 months prior. Troponin was 4 ng/dL. She was admitted for non-ST-elevation myocardial infarction. Cardiac catheterization (LHC) showed normal coronary arteries, and transthoracic echocardiogram (TTE) revealed normal left ventricular ejection fraction and she was diagnosed with MINOCA. She was treated with dual-antiplatelet therapy, metoprolol, and lisinopril and discharged home with plans for outpatient cardiac MRI to evaluate for myocarditis. Four days later, she developed constant, severe chest pain at rest and returned for evaluation. ECG showed sinus tachycardia with new nonspecific ST-segment changes, while telemetry noted frequent PVCs and episodes of ventricular bigeminy. Beta blockade was intensified and repeat LHC ruled out coronary dissection. Repeat TTE identified mid-inferior hypokinesis. Given high suspicion for myocarditis she underwent cardiac MR which showed preserved biventricular size and function, and increased T1, T2 signals in multiple territories in a non-coronary distribution representing a large area of edema and inflammation consistent with myocarditis and normal pericardium. Inflammatory markers were unremarkable and autoimmune and endocrine testing was within normal limits. Her chest pain recurred with rising troponin and declining ejection fraction on serial ECHO as well as increasing frequency of non-sustained ventricular tachycardia. She was emergently transferred for endomyocardial biopsy.

DECISION MAKING: Troponin peaked at 41 ng/dL. CRP was normal at 2.5, ESR was normal at 19, and brain natriuretic peptide was normal at 29. TSH was within normal limits. HIV testing was negative, and lupus was ruled out with negative ANA and anti-dsDNA. IVIG was considered, but ultimately not given due to unclear evidence of benefit. With declining ejection fraction and increasing ventricular arrhythmias, she had strong indications for endomyocardial biopsy to rule out giant cell myocarditis. Pathology confirmed lymphocytic myocarditis, with resultant heart failure with reduced ejection fraction. Amyloid and iron staining were negative. She was treated with neurohormonal blockade for reduced ejection fraction with beta blocker, angiotensin-receptor antagonism, and spironolactone, as well as colchicine for pericarditis with chest pain resolution and stabilized ejection fraction.

CONCLUSION: In patients with rapidly declining cardiovascular function in the setting of myocarditis of unknown cause, giant cell myocarditis must be considered and ruled out via endomyocardial biopsy as these patients are high risk for requiring transplant and immunosuppression. This patient's myocarditis was determined to be viral in the setting of positive parvovirus B19 IgG and equivocal IgM and supportive care was able to achieve a good outcome.