

CASE REPORT**Troponin-Positive Chest Pain with Unobstructed Coronary Arteries**Mitchell Padkins¹, Meltiady Issa²¹Mayo Clinic School of Graduate Medical Education. Department of Medicine. Rochester, Minnesota²Mayo Clinic. Department of Medicine Division of Hospital Medicine. Rochester, MinnesotaCorresponding author: Mitchell Padkins, M.D. Mayo Clinic. 200 First Street SW. Rochester, Minnesota 55905
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We present a 29-year-old man who presented with crushing substernal chest pain with radiation to his jaw and associated diaphoresis. He was hemodynamically stable and electrocardiogram was unremarkable. Laboratory studies revealed initial high-sensitivity troponin of 1950 ng/L (reference: <15 ng/L), 2-hour troponin 2165 ng/L, and 6-hour troponin 2413 ng/L. NT-pro-BNP was elevated at 1692 pg/mL (reference: <51 pg/mL), and C-reactive protein was 50.9 mg/L (reference: <8 mg/L). Transthoracic echocardiogram revealed an ejection fraction (EF) of 30% with severe global left ventricular hypokinesia. Left heart catheterization revealed normal coronary arteries. Cardiac magnetic resonance imaging showed acute myocarditis. With aggressive diuresis and anti-inflammatory therapy for concomitant pericarditis, his symptoms improved. After one month of beta-blocker and angiotensin converting enzyme inhibitor therapy, his EF improved to 63% with normal left ventricular function.

Keywords: Myocarditis, Cardiac Magnetic Resonance Imaging, Lake Louise Criteria**CASE PRESENTATION**

A 29-year-old man presented to the Emergency Department with crushing substernal chest pain with radiation to his jaw and associated diaphoresis, dyspnea, nausea, and vomiting. He had a history of hypertension, hypertriglyceridemia, and tobacco use. The patient was hemodynamically stable and EKG was unremarkable. Laboratory studies revealed initial high-sensitivity troponin of 1950 ng/L (reference: <15 ng/L), 2-hour troponin 2165 ng/L, and 6-hour troponin 2413 ng/L. NT-pro-BNP was elevated at 1692 pg/mL

(reference: <51 pg/mL), and C-reactive protein was 50.9 mg/L (reference: <8 mg/L). Given his presentation, acute coronary syndrome (ACS) was high on the differential and he was started on aspirin, heparin, and clopidogrel. Transthoracic echocardiogram revealed an ejection fraction (EF) of 30% with severe generalized left ventricular hypokinesia. Cardiac catheterization revealed normal coronary arteries without obstructive lesions. Given these findings, cardiac magnetic resonance (CMR) imaging was performed and findings were consistent with acute myocarditis (Figure 1). With

aggressive diuresis and preload reduction, the patient's chest pain improved and he was started on anti-inflammatory therapy for concomitant pericarditis. After one month of beta-blocker and angiotensin converting enzyme inhibitor therapy, his EF improved to 63% with normal left ventricular function.

CASE DISCUSSION

ACS patients with unobstructed coronary arteries represent a clinical dilemma that carries high mortality (1). The most common etiology in these patients is myocarditis with a range of prevalence from 15-75% (2). This myocardial inflammatory process has a variable presentation that can range from mild symptoms to severe heart failure (1). The etiology is usually viral in origin; however, bacterial pathogens have been identified along with a host of medications such as antipsychotics (1, 2). Coronary angiography is commonly performed to exclude obstructive coronary

vessel lesions if there is a high suspicion for ACS. The diagnostic gold standard is an endomyocardial biopsy, but the advent of CMR has helped clarify this diagnosis without pursuing invasive measures. Recent publications have outlined the recommendations for using CMR to diagnose myocarditis and developed the Lake Louise Criteria for diagnosis (3). CMR has been validated against endomyocardial biopsy and, if biopsy is performed, CMR can localize myocardial inflammation for increased biopsy yield (4, 5). CMR can be used to detect various characteristics of myocarditis, including inflammatory hyperemia, edema, and myocyte necrosis, which makes it useful to support the diagnosis and monitor disease progression (3).

In summary, CMR should complement the evaluation of patients with ACS and unobstructed coronary arteries if available. A correct diagnosis is important for prognosis and treatment.

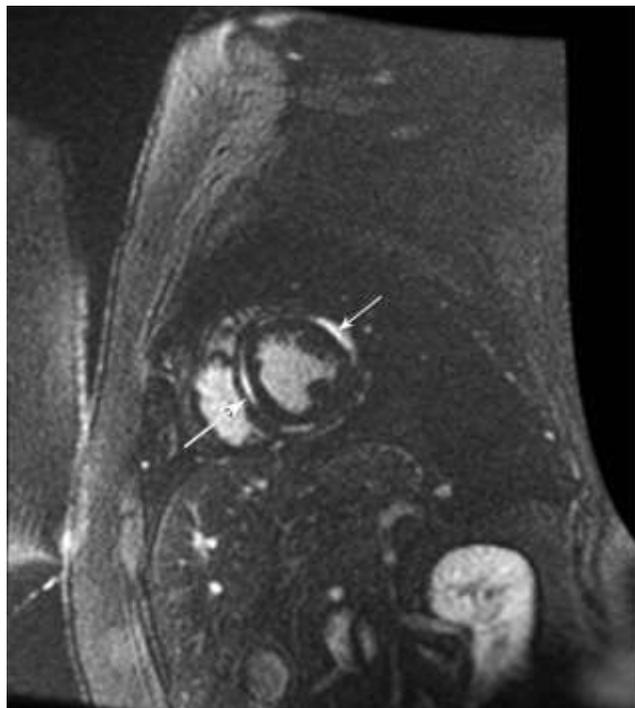


Figure 1. Short-axis CMR demonstrating gadolinium-delayed enhancement. Abnormally increased signal is present in the epicardium of the anterolateral wall (short arrow) and mid-ventricular septum (long arrow) corresponding to areas of increased space and myocardial damage. These findings do not correspond to a vascular territory and is consistent with myocarditis.

Notes

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