

## A 40-YEAR-OLD WOMAN WITH CHEST PAIN, ST ELEVATION, ELEVATED TROPONIN AND NORMAL CORONARY ARTERIES: A CASE REPORT

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Electrocardiographic changes resembling myocardial ischemia or infarction can be caused by a variety of causes other than ischemia. One of them is acute myocarditis which further confounds clinical judgment by causing elevation in troponins as well. We report a case of myocarditis which underscores the importance of identifying the clinical presentation of acute myocarditis and the electrocardiographic changes that can be associated with it.

### Case Report

A 40-year-old mother of two children with no significant past medical history presented to an outside hospital (OSH) complaining of intermittent chest pain for a week. She described it as “pressure-like” pain in the retrosternal and epigastric regions, with no radiation and reported it had been particularly worsening in the past two days. She notes that her youngest child aged three was sick with an upper respiratory infection in the past week. On arrival at the OSH, patient’s electrocardiogram (EKG) showed significant ST elevations in V1 and V2, ST depression and T wave inversion in the inferior and other precordial leads. (Figure 1) Her initial troponin was 28.6 ng/ml (normal range –

less than 0.5 ng/ml). Anticoagulation was initiated with heparin and an emergent cardiac catheterization was performed at the OSH, which revealed normal coronary arteries.

During the procedure, she developed hemodynamic instability, requiring an infusion of norepinephrine and phenylephrine and a placement of an intra-aortic balloon pump (IABP). Patient was then transferred to our institution for further management. Subsequently, she started experiencing repeated episodes of sustained ventricular tachycardia, requiring multiple boluses and continuous infusions of amiodarone. She also developed an irregular tachyarrhythmia with right bundle branch block (Figure 2 and 3). Laboratory work and other cardiovascular studies from OSH were reviewed. Echocardiogram revealed an ejection fraction of 15-20% with severe global LV dysfunction. A presumptive diagnosis of acute myocarditis was made. On the second day of admission, extra-corporeal membrane oxygenation (ECMO) was initiated due to further hemodynamic deterioration.

Viral titers for adenovirus, cocksackie, Cytomegalovirus (CMV), herpes, echovirus, Epstein-Barr virus, hepatitis, parainfluenza and varicella were sent. Antibody titers for Lyme, mycoplasma and toxoplasma were also sent. The titers were positive only

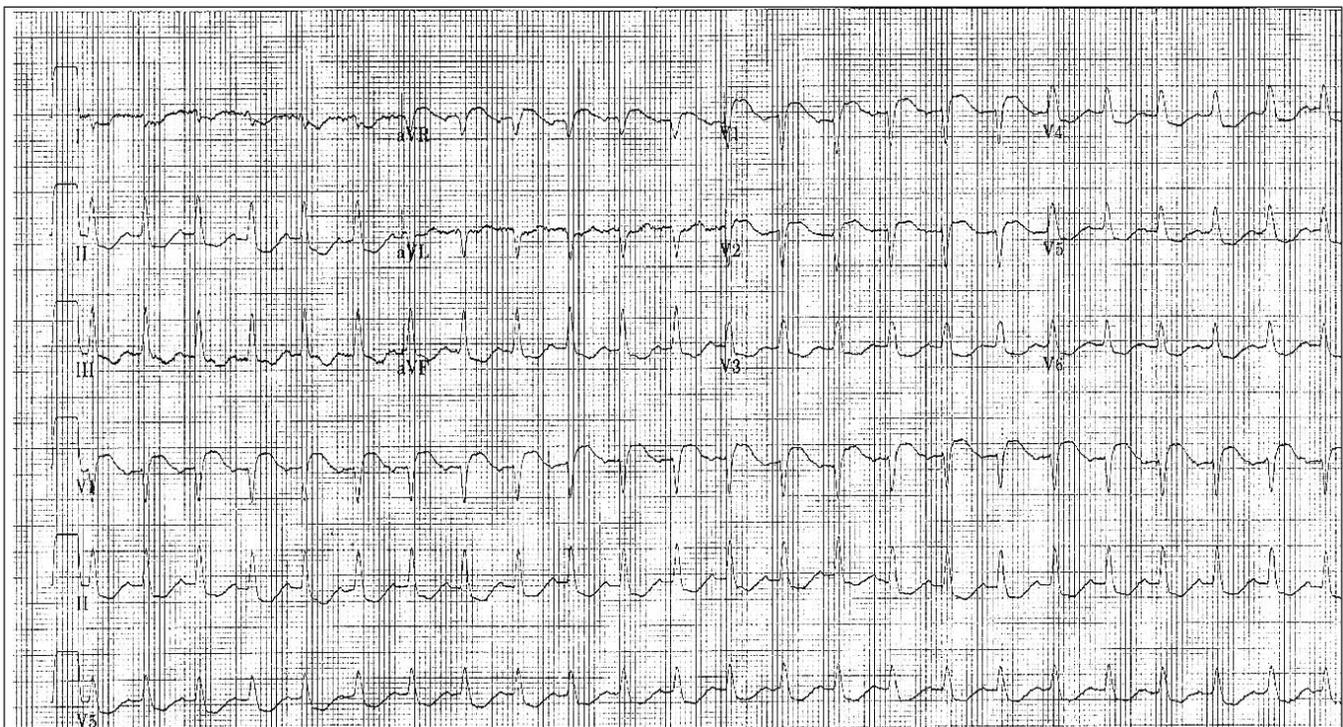


Figure 1. EKG 1 at the time of initial presentation to the OSH. EKG shows sinus tachycardia with ST elevation in V1 and V2, ST depression in V3 to V6, I, II, III, aVF.

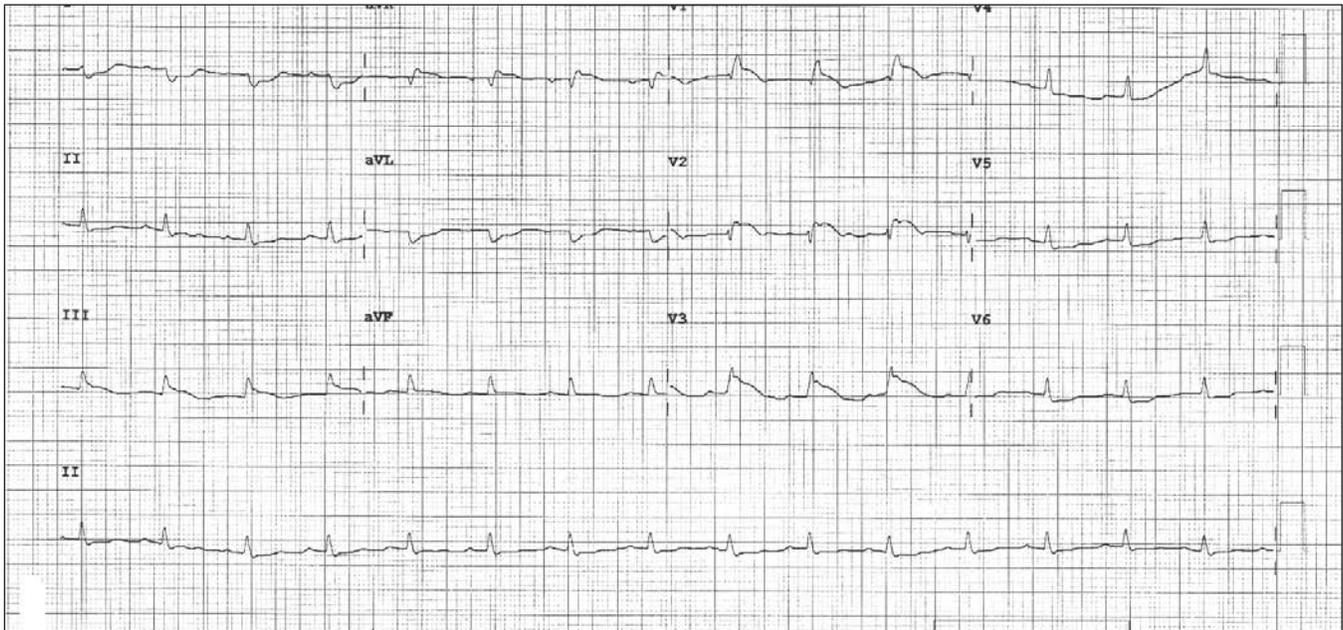


Figure 2. EKG 2 was obtained at our institution 6 hours after the initial EKG at the OSH. It shows ST elevation from V1 to V3 and ST depression in I, aVL, V4 to V6. Also present are minimal ST depressions in II.

for CMV. Methylprednisolone was administered intravenously at a dose of 1 gram daily for three consecutive days. Patient subsequently received a left ventricular assist device (LVAD). An endomyocardial biopsy revealed lymphocytic myocarditis. The Heart Failure service initiated a work-up for cardiac transplantation. Despite full mechanical circulatory support and multiple intravenous inotropic agents, the patient succumbed to her illness, a week after the transfer to our institution.

## Discussion

Myocarditis is defined as myocardial inflammation in the absence of ischemia or infarction. It can present with minimal symptoms with slowly progressive cardiomyopathy or acute cardiogenic shock. Myocarditis can be caused by infectious and non-infectious causes. Viruses are the most common infectious cause, but bacterial and protozoal infections are also responsible. Non-infectious causes include rheumatological conditions and drug induced reactions.<sup>2</sup> Depending on the acuity of clinical presentation, EKG changes and lab tests can provide clues to suspect myocarditis. Cardiac MRI is increasingly being used in suspected myocarditis cases but endomyocardial biopsy remains the gold standard for diagnosis.<sup>3</sup> Viral titers may help in identifying a presumptive source but will usually not alter treatment. The mainstay of therapy is supportive care and a heart failure regimen should be initiated at the earliest time possible. If medical therapy fails, mechanical circulatory support can be considered. Some patients may eventually require a cardiac transplant.<sup>2</sup>

We are conditioned to think that ST segment elevation indicates underlying coronary artery disease. While coronary artery disease resulting in acute coronary syndrome is one of the most important causes of ST segment elevation, it is crucial to remember that it can also be caused by many other conditions viz., left bundle branch block, early repolarization, myocarditis, pericarditis, etc.<sup>4</sup> Myocarditis can cause a spectrum of EKG changes that include ST segment elevation, ST depression, any degree of heart block, T wave inversion, abnormal Q waves, bundle branch blocks, atrial and ventricular arrhythmias.<sup>3,5</sup> Numerous cases of myocarditis mimicking myocardial infarction have been reported.<sup>6-10</sup> However, in myocarditis the ST elevation does not correspond to any coronary artery distribution and ST depressions while present are usually not reciprocal. These can serve as distinguishing features between myocarditis and an infarct.<sup>5</sup> But, unfortunately, EKG changes are non-specific and in cases with focal myocarditis, they can closely resemble an acute myocardial infarction.<sup>7</sup> Consequently, recognizing acute myocarditis remains a serious challenge and inappropriate management of the disease can lead to fatal consequences.<sup>11</sup>

In acute myocarditis, elevation of troponin can also be misleading. Elevated troponins can occur in any condition that causes myocardial damage and does not necessarily imply coronary artery disease.<sup>12</sup> Even though the cause of troponin elevation is significant myocardial injury as in the present case, it is crucial to always assess EKG changes and troponin elevation, with respect to the clinical presentation. This case highlights the importance of understanding the pathological

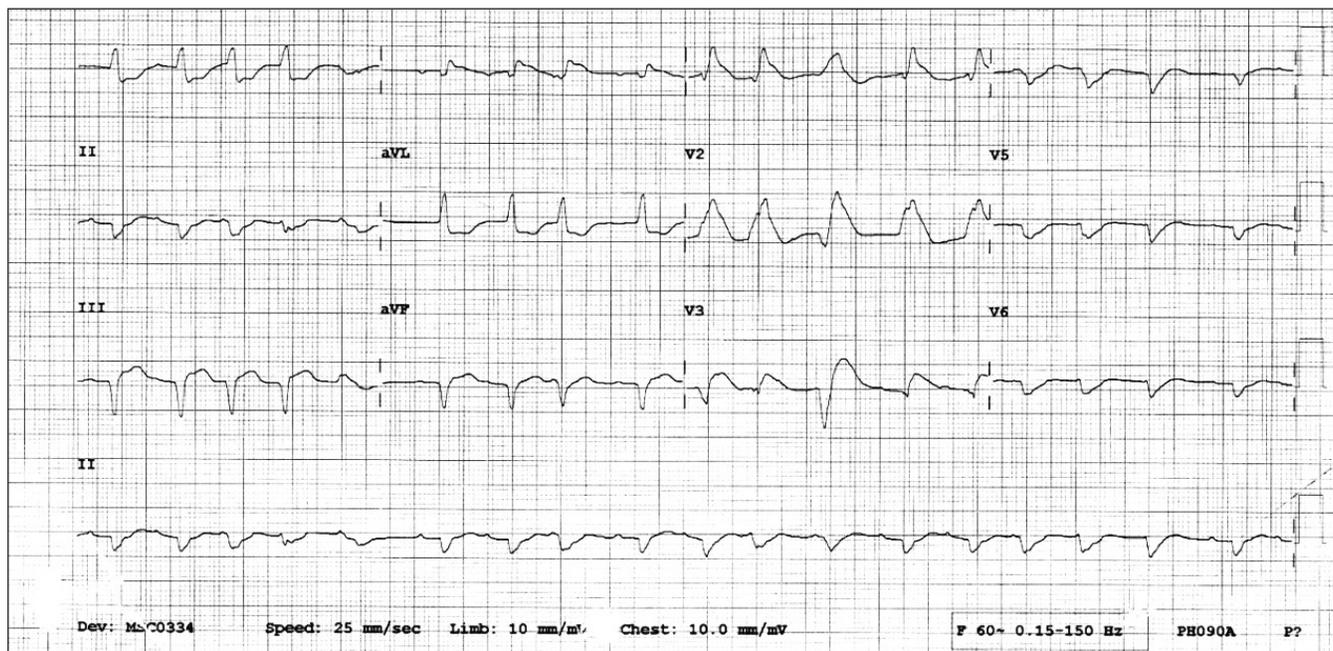


Figure 3. EKG 3 was obtained on hospital day 2. Sinus tachycardia with right bundle branch block and left anterior fascicular block. There are frequent PVCs which preempt AV nodal conduction resulting in shorter PR intervals. Note the QRS duration nearly 260 milliseconds indicating severe myocardial injury in addition to the bundle and fascicular blocks

process of EKG changes and troponin elevation and the possible clinical challenges that arise, when faced with a combination of these abnormalities.

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