# Persistent electrocardiographic ST segment elevation from previous myocarditis

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#### **ABSTRACT**

Various conditions may present with an electrocardiographic pattern of ST segment elevation simulating myocardial infarction. We present an asymptomatic 16-year-old male patient, whose ECG showed persistent anterolateral ST segment elevation and magnetic resonance imaging showed wall motion abnormalities from previous myocarditis but no evidence of inflammation.

Keywords: Electrocardiogram, myocarditis, ST elevation

### **INTRODUCTION**

Various conditions, apart from the coronary artery disease, may present with an electrocardiographic pattern of ST segment elevation myocardial infarction (STEMI). These include early repolarization variant, pericarditis, myocarditis, Brugada syndrome, aortic stenosis, pulmonary embolism, aortic dissection, and other noncardiac conditions such as cholecystitis, pneumonia, or pancreatitis, which may also mimic these electrocardiographic findings.<sup>[1-3]</sup>

#### **CASE REPORT**

An asymptomatic 16-year-old male patient was referred for evaluation because of his past history. He had undergone aortic coarctation surgery by the Waldhausen technique when he was 15 days old.

At the age of 11, the patient went to the pediatric emergency department for chest pain associated with dyspnea. At admission then, the patient was afebrile, hemodynamically stable, and denied any previous history of flu-like episodes, fever, or diarrhea in the previous months. However, the electrocardiogram (ECG)

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showed a ST elevation in the anterior leads; this is the reason why he was admitted for further evaluation. In the next 24 hours, the chest pain spontaneously and progressively disappeared and two repeated creatine phosphokinase (CPK) assessments were within normal limits. Angiography showed normal coronary arteries, a preserved left ventricular function, absence of the left subclavian artery, and no aortic recoarctation.

Now 5 years later after his hospital admission, the patient is asymptomatic and has had no recurrence of chest pain or dyspnea. There is no history of diabetes, hypertension, dyslipidemia, or smoking. Physical examination is unremarkable with no murmurs or pericardial friction rubs. Analytical data shows normal creatinine, troponin, NT-pro brain natriuretic peptide, and cholesterol concentrations. Antinuclear antibodies are negative. Viral serological tests is positive for Epstein-Barr virus IgG, Coxsackievirus A9 and B (1-6) IgG, Echovirus IgG, Parvovirus B19 IgG, Human Herpes virus 6 (HHV-6) IgG and Rubeola IgG and negative for Epstein-Barr virus IgM, Coxsackievirus A9 and B (1-6) IgM, Echovirus IgM, Parvovirus B19 IgM, Human HHV-6 IgM, Hepatitis A IgG/M, Hepatitis B, Human Immunodeficiency virus (HIV) and Human Cytomegalovirus IgG/M. Meanwhile, bacterial and parasite serological tests are positive for Mycosplasma pneumoniae IgG, Chlamydia pneumoniae IgG, and Rickettsia conorii IgG and negative for Chlamydia pneumonia IgM, Chlamydia trachomatis IgG/M, Chlamydia psittaci IgG/M, Coxiella burnetii IgG/M, Rickettsia typhi IgG/M, Borrelia burdorferi IgG/M, Rickettsia conorii IgM, Leptospira IgM, and toxoplasma IgG/M.

The ECG shows upward concave ST segment elevation

Address for correspondence: Dr. Efrén Martínez Quintana, Avda. Marítima del Sur s/n 35016 Las Palmas de Gran Canaria, Spain. E-mail: efrencardio@gmail.com in the anterolateral leads with T wave inversion and an incomplete right bundle branch block [Figure 1] similar to the ECGs recorded during his hospital admission at the age of eleven previously. The ECG at the time of coarctation surgery was reported as showing right bundle branch block and right atrial enlargement only.

Cardiac magnetic resonance (MR) imaging shows no renarrowing at the aorta, a normal right ventricle, thinning and hypokinesis of the anterior and anteroseptal middle segments of the left ventricular wall and a preserved left ventricular function (70%). Moreover, there was no evidence of myocardial edema, global relative enhancement, or late Gadolinium enhancement. T2-weighted triple-inversion-recovery imaging was used to calculate the myocardial edema ratio (ER), a normal T1-weighted imaging before and after contrast agent administration was used to determine the myocardial global relative enhancement (gRE), and a normal inversion-recovery gradient-echo imaging was used to evaluate areas of late enhancement (LE)<sup>[4,5]</sup> [Figure 2].

Twenty-four hour ambulatory monitoring of arterial pressure shows a daytime and a nighttime normal blood pressure. Meanwhile, treadmill test shows a normal

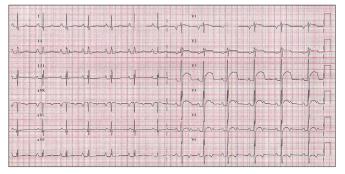


Figure 1: Electrocardiogram showing sinus rhythm, an incomplete right bundle branch block and an ST-segment elevation in leads V1 through V5 with biphasic T waves in V4-V6. No reciprocal ST segment depression or Q waves abnormalities were seen

hypertensive response and no arrhythmias during exercise.

## **DISCUSSION**

The most frequent ST abnormalities found in patients with myopericarditis, in order from most to least frequent, are the T wave inversion, the ST segment elevation and the ST segment depression in the anterolateral and the inferolateral leads.<sup>[6]</sup> However, the initial elevation of the ST segment in the acute phase of myocarditis generally returns to normal within several days to a few weeks, and flat, biphasic or negative T waves occasionally remain as the ST segment elevation subsides. However, fibrous lesions due to myocarditis, located predominantly in the subepicardial and middle layers, may led to persistent ST elevation during hospitalization<sup>[7]</sup> or to very rare chronic persistent upward elevation of the ST segment, as seen in our patient.<sup>[8]</sup>

Cardiovascular MR imaging has emerged as a new noninvasive modality for the diagnosis of myocarditis. However, the area of myocarditis diminishes in size as it is replaced by scar, potentially explaining the observation that contrast enhancement typically decreases significantly over time.<sup>[9]</sup> In this context, Gutberlet et al,<sup>[4]</sup> reported 83 patients with suspected chronic myocarditis and compared cardiac MR imaging with having or not having histologic data of intramyocardial inflammation and viral persistence, determined by polymerase chain reaction (PCR) assay, in the endomyocardial biopsy. Nearly 42% of the 83 patients had an elevated gRE, 52% demonstrated an elevated ER and 24% presented a LE of which 86%, 74%, and 65% of the cases had a positive viral PCR result, respectively. However, there was no significant association between PCR proof of viral infection and gRE, ER, or LE cardiac MR results: The authors concluded that cardiac MR imaging might be helpful in detecting

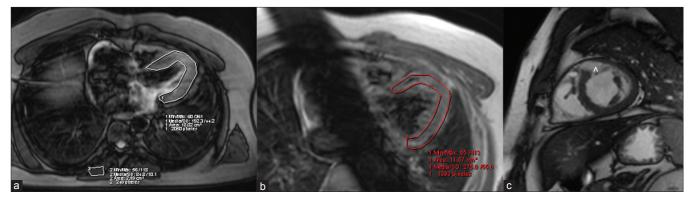


Figure 2: Magnetic resonance imaging examinations to assess myocardial inflammation. (a) Transverse T2-weighted triple-inversion-recovery magnetic resonance image showing a normal edema ratio. (b) Postcontrast transverse T1-weighted fast spin-echo magnetic resonance image showing a normal global relative enhancement. (c) Mid-ventricular short-axis with late gadolinium enhancement showing thinning of the anteroseptal segment of the left ventricle (arrowhead) and normal delayed enhancement in the left ventricular walls. In B an additional saturation section is positioned across the atria to reduce signal from slow-flowing blood. ER and gRE are calculated according to the method of Friedrich *et al.*<sup>[5]</sup>

intramyocardial inflammation noninvasively, but failed to depict viral persistence.

The diagnosis of myocarditis can be challenging, and more so for chronic persistent myocarditis. Myopericarditis remain one of the frequent cause of ST segment elevation in the young. Our patient shows that previous myocarditis may be responsible for persistent ST segment abnormalities, accompanied by the wall motion abnormalities in the cardiac MR.<sup>[10]</sup> The prognostic implication of such findings on ECG or MR imaging remain unknown.

In conclusion, persistent ST segment elevation may be the result of previous myocarditis. In some patients, this may be accompanied by wall motion abnormality in cardiac MR imaging.

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