

## Serial Electrocardiographic Changes in a Young Female with Acute Myocarditis

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### Authors' contributions

This work was carried out in collaboration among all authors. Author GS designed the manuscript, provided the clinical finding, the laboratory findings and images for the study and wrote the first draft of the study. Authors MB and AJ managed the literature searches. Author NOB revised the manuscript. All authors read and approved the final manuscript.

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Case Report

### ABSTRACT

Electrocardiographic changes in myocarditis mimic a wide range of ECG diagnoses ranging from ST-elevation myocardial infarction to complete heart block. We report a case of acute myocarditis in a young female with a wide range of ECG changes that mimic ST-elevation myocardial infarction and atrioventricular block.

Keywords: Myocarditis; electrocardiographic changes.

### ABBREVIATIONS

ECG : Electrocardiogram;  
AV : Atrioventricular;  
AMI : Acute Myocardial Infarction;  
ACS : Acute coronary Syndrome

### 1. INTRODUCTION

Myocarditis has a wide range of potential clinical presentations, which leads to difficulties in diagnosis and classification. The clinical picture may be one of asymptomatic

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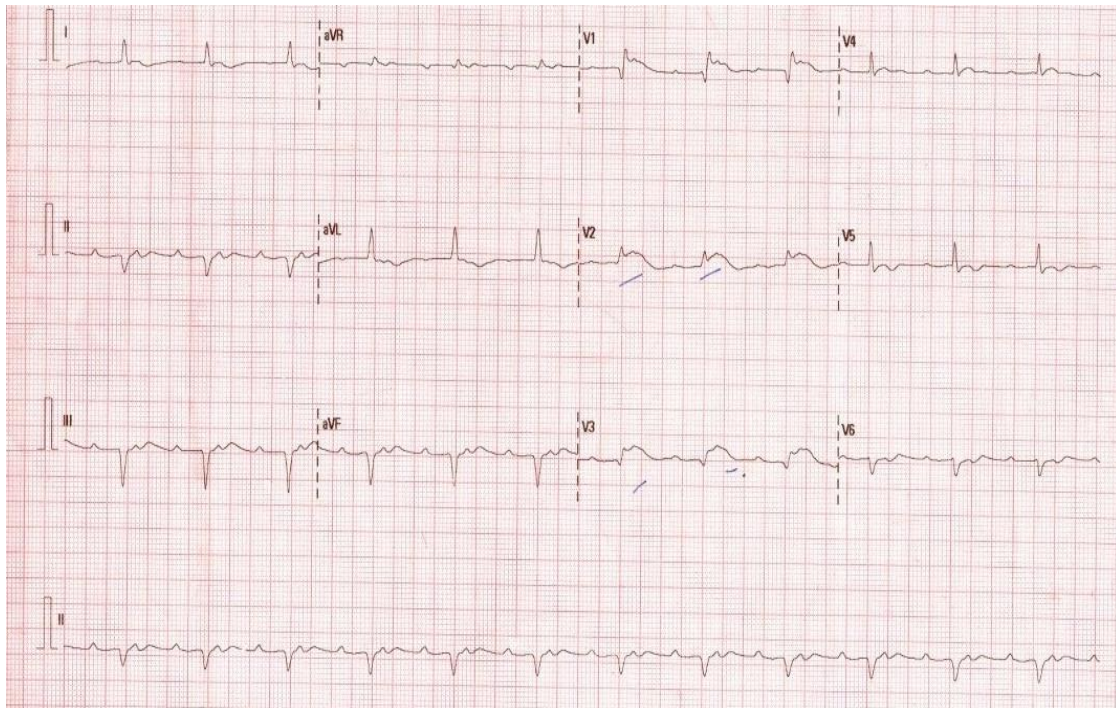
electrocardiographic or echocardiographic abnormalities or may include signs and symptoms of heart failure, chest pain, arrhythmias and/or hemodynamic collapse. Myocarditis typically has a bimodal age of presentation. Acute or fulminant presentation is more commonly seen in young children and teenagers. The presenting symptoms are more subtle and insidious, often with dilated cardiomyopathy and heart failure, in the older adult population. In this article, we present a case of acute myocarditis in a young female with a wide range of ECG changes that mimic both ST-elevation myocardial infarction and atrioventricular block.

## 2. CASE REPORT

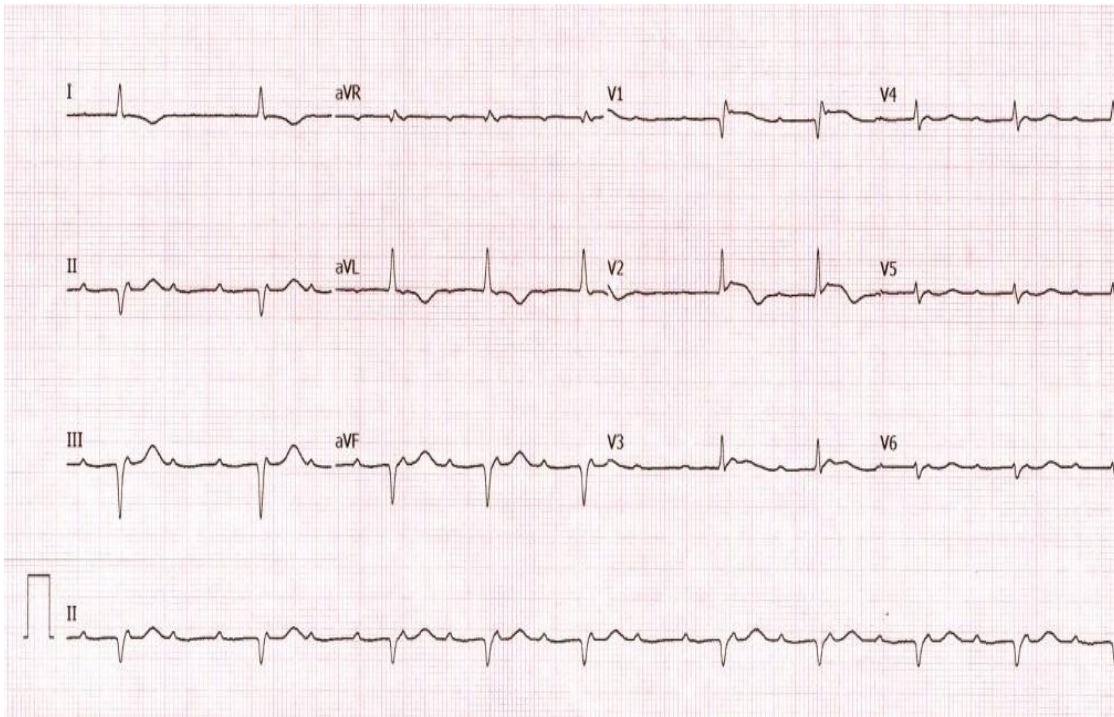
A sixteen-year-old female was admitted with 5 days of fever. On clinical examination, pulse was 72/min (low in proportion with the body temperature), blood pressure – 96/60 mmHg. Cardiovascular and respiratory system examination was normal. Blood investigations showed hemoglobin 10.6 g/dl (Normal- 13-16 g/dl), Total white blood count was 14,300 per cubic millimeter, (Normal – 4000-11000 per cubic

millimeter) with neutrophil predominance. Serum creatinine and electrolytes were normal. Electrocardiogram was done as the pulse rate of the patient was low with respect to the body temperature. The first ECG on admission (Fig. 1) showed a P rate of 150/min 2:1 2<sup>nd</sup> degree AV block and ST elevation in V1-3.

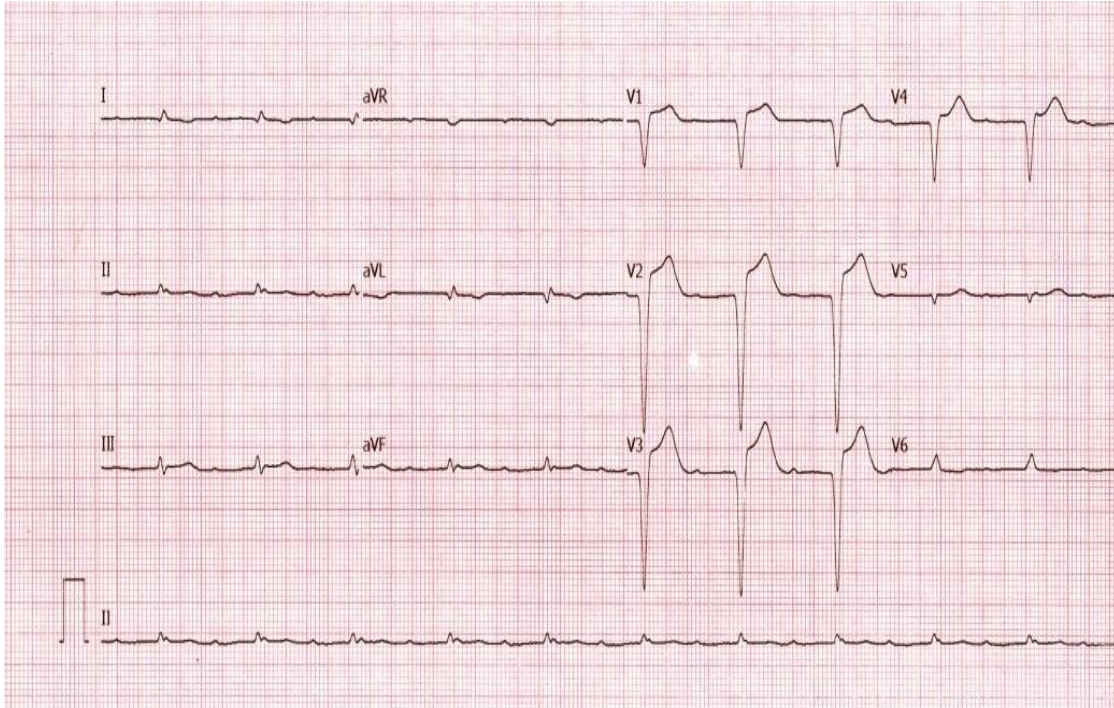
Echocardiography showed mild global hypokinesia with an ejection fraction (EF) of 45%. Quantitative Troponin T was 208 pg/ml (Normal < 14 pg/ml). C- reactive protein was 31 mg/L (Normal < 5 mg/L), Erythrocyte sedimentation rate was 55 mm/hour (Normal 0-20 mm/Hour). Serial ECGs of the patient are shown in Figs. 2-7. Cardiac MRI confirmed the diagnosis of Myocarditis with diffuse STIR (Short T1 inversion recovery) hyperintense signal in the mid and apical regions of the left ventricle (Fig. 8A, 8B). She was monitored in the intensive care unit for any worsening of the clinical condition and ECG changes. Supportive treatment was given to her. She recovered with normal sinus rhythm and with mild decrease in EF. The patient is asymptomatic on subsequent follow-ups, however the ECG changes (shown in Fig. 7) persisted.



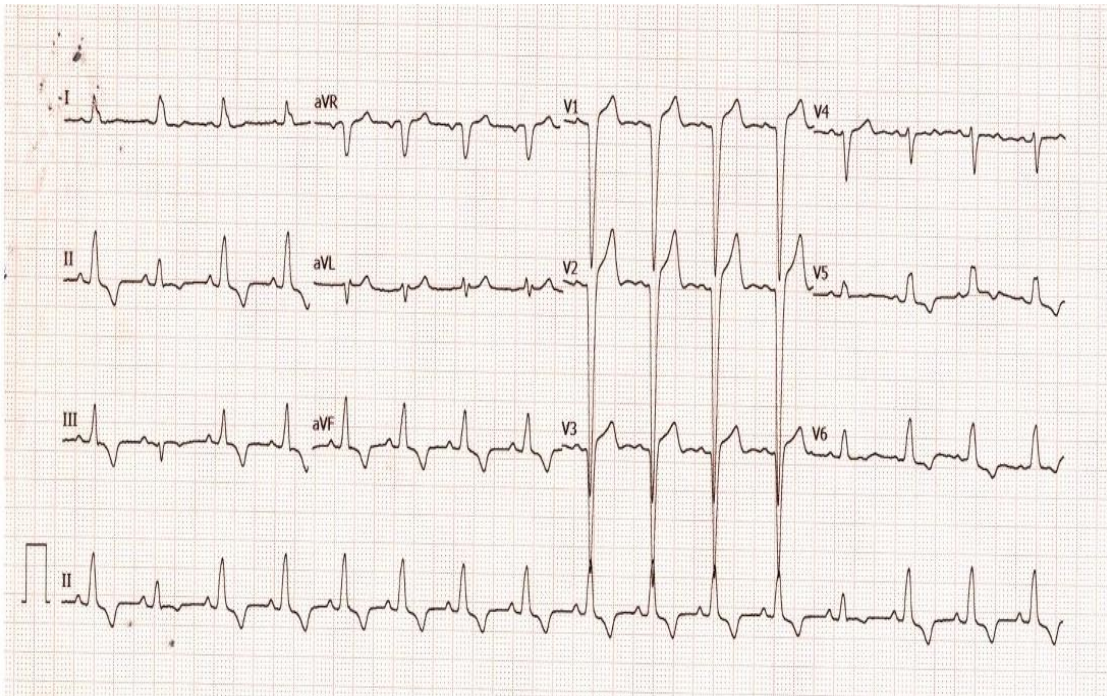
**Fig. 1. P rate of 150/min, 2:1 2<sup>nd</sup> degree AV block, prolonged PR interval, and ST elevation in V1-3 (On admission)**



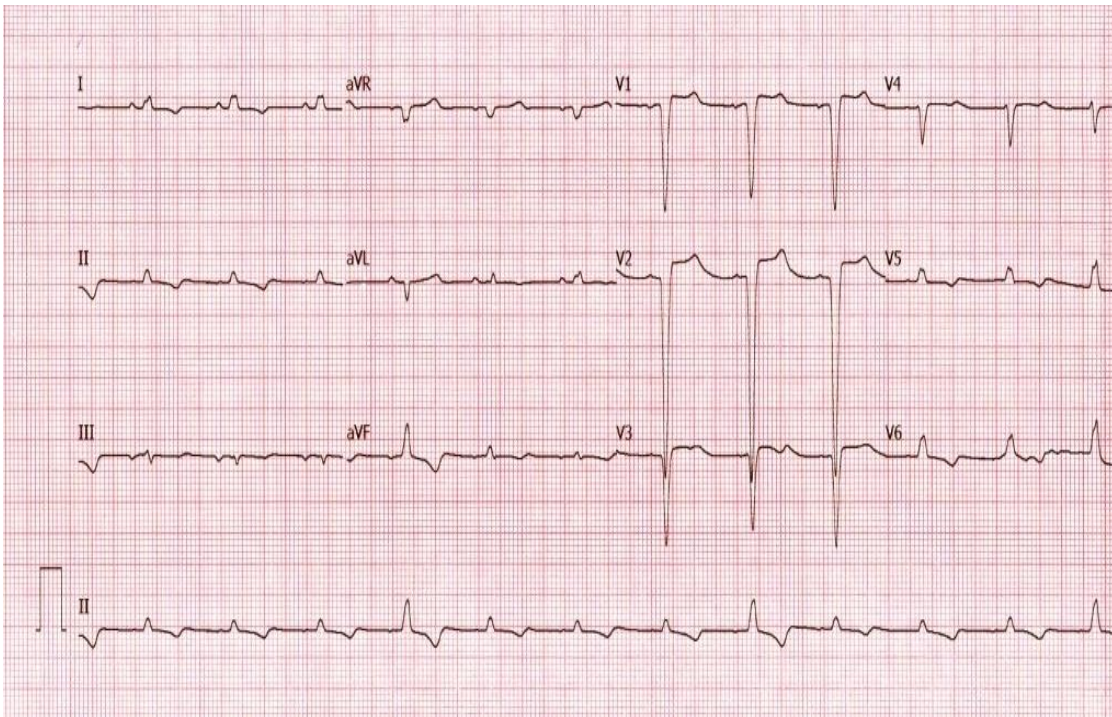
**Fig. 2. P rate of 150 min, 3:1 AV block, Prolonged PR interval, and ST elevation in V1-3 (Day 2)**



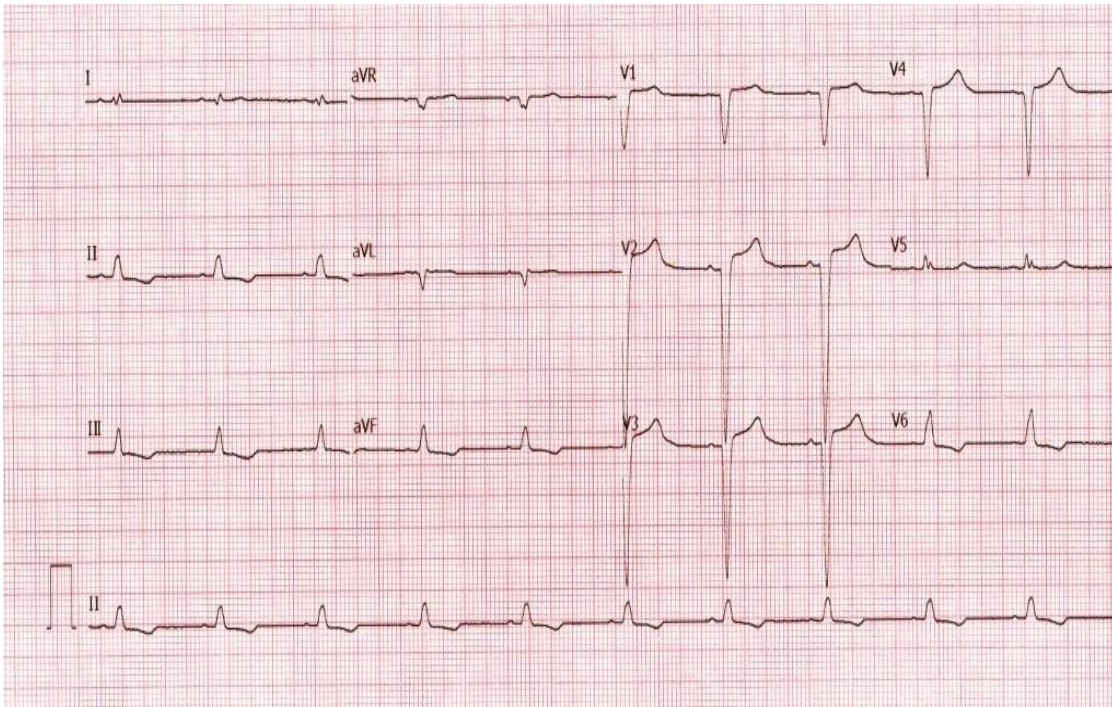
**Fig. 3. P rate 150/min, 2:1 AV block with prolonged PR, ST elevation and loss of R in V1-V4 (Day 3)**



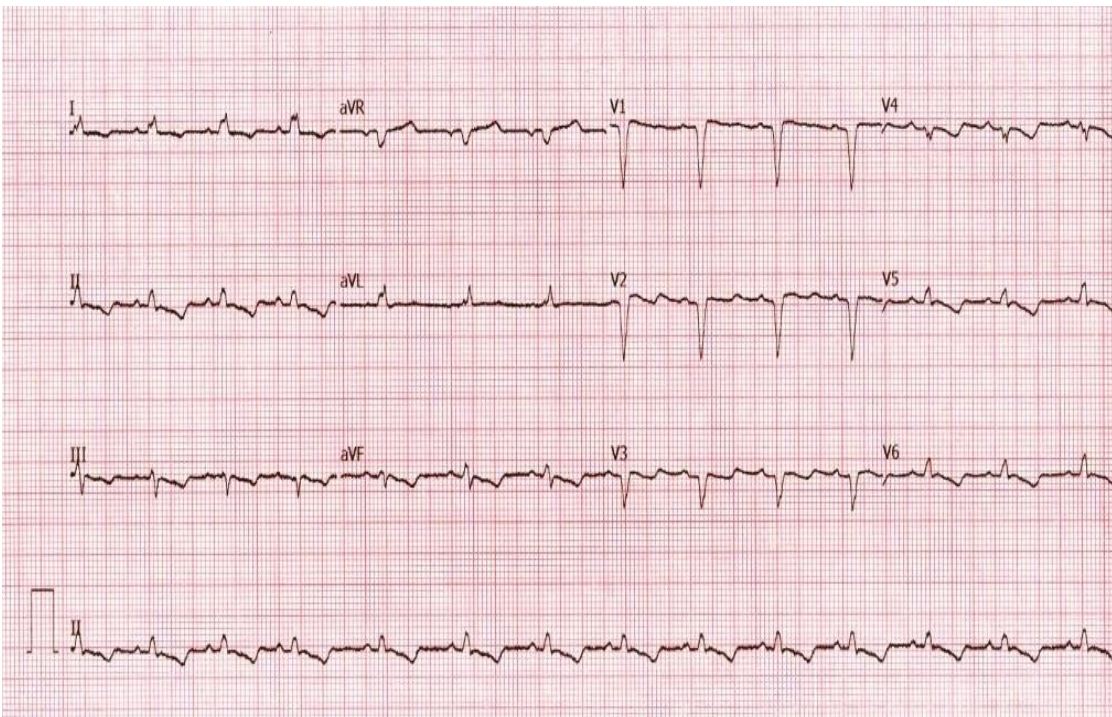
**Fig. 4. Sinus rhythm, P rate 100/min R rate- 100/min, poor R progression in V1-4, incomplete LBBB (Day 5)**



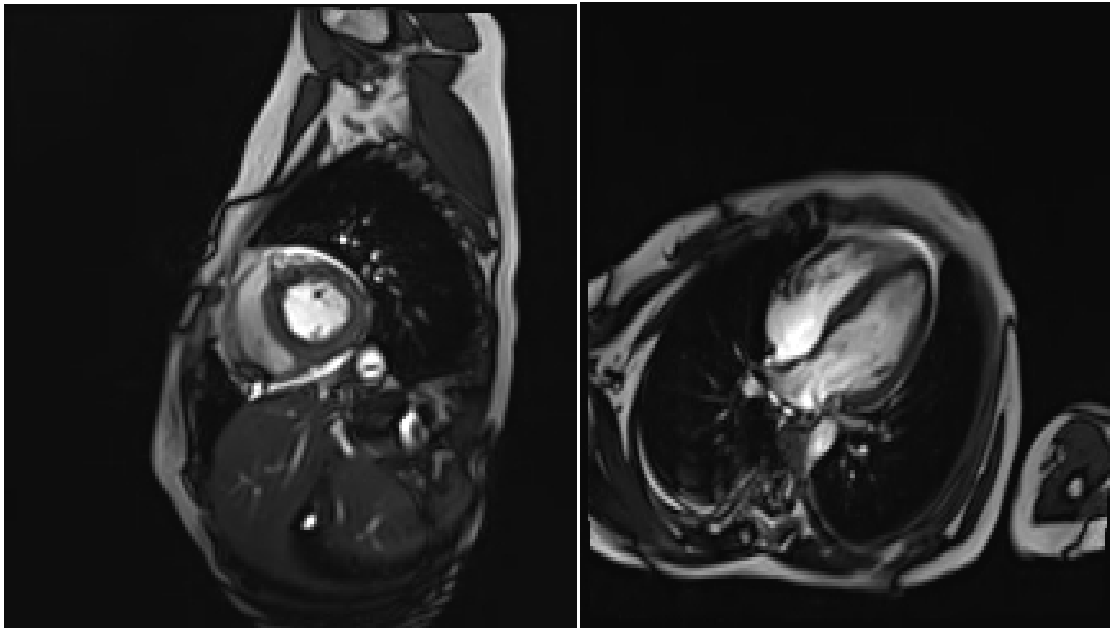
**Fig. 5. Sinus rhythm with rate 75/min, ST elevation and Poor R progression in V1-4 (Day- 7)**



**Fig. 6. Sinus rhythm with a rate of 75/min persisting ST elevation in V1-4, poor R progression, incomplete LBBB (Day-9)**



**Fig. 7. ECG on discharge - sinus rhythm with P and R rate of 84/min, poor R progression (Day-11)**



**Fig. 8A, 8B. MRI of the patient showing hyperintense signal in the mid and apical region of the left ventricle**

### 3. DISCUSSION

Myocarditis is an inflammatory disease of the myocardium leading to necrosis and degeneration of adjacent myocytes not typical of the ischemic damage associated with coronary artery disease [1]. The diagnosis of myocarditis is often difficult to establish due to the wide range of potential clinical presentations. The direct viral invasion of the myocardium and immune-mediated myocardial injury are the main mechanisms by which viruses might produce myocarditis [2,3]. Clinical findings of myocarditis may mimic acute coronary syndrome with chest pain, ST-changes in ECG, and cardiac enzyme elevation [4-7].

The ECG is usually abnormal in patients with myocarditis, though ECG signs are neither specific nor sensitive. The ST-T changes are due to the severe inflammatory changes of myocardium and/or pericardium leading to necrosis and edema of the myocardium. These changes are not localized as in myocardial infarction. The ST-elevations without reciprocal ST depression is quite distinct from acute myocardial infarction, which can be a diagnostic hallmark in myocarditis. The ST-T segment elevation in myocarditis is typically concave (rather than convex in myocardial ischemia). Thrombolysis in these patients can be fatal.

There are cases in the literature in which coronary angiogram was done in young age patients, suspecting myocardial infarction even in the absence of coronary risk factors. Abnormal Q waves can be present in myocarditis, which is transient unlike in AMI, where Q waves are persistent. Abnormal Q waves have a close relation with left ventricular function and may disappear after the resolution of the inflammatory process [8,9].

As compared to the AMI, conduction abnormalities are more common in myocarditis. Various degrees of conduction abnormalities were present in 43% of patients in one study [10] and 64% in another study [9]. Complete and advanced AV blocks were present in 15% of patients in one series. Pathological mechanisms for AV block, as seen in this case, are most often attributed to the inflammation in the conduction tissue, right and left bundle branches, especially in the terminal portion. Conduction abnormalities in myocarditis do not indicate severe myocardial involvement as seen in AMI, these abnormalities may disappear after the inflammatory process subsides. Some viruses may have selective tropism for the specialized conduction system [11]. Abnormal QRS complexes and left bundle branch block (LBBB) pattern are the only ECG variables which suggest more advanced disease process and are closely related to mortality [9].

Serial ECGs or 24 hours continuous ECG monitoring is very important in patients with suspected myocarditis or in confirmed cases of myocarditis. Our patient had changes in QRS complexes, ST-segment changes, and conduction abnormalities. The importance of recording serial ECGs is shown in our patient, with recovery in various ECG variables. Serial ECGs in patients with myocarditis offer additional and independent clinical information regarding the phase of the disease and prognosis.

#### 4. CONCLUSION

Myocarditis should be suspected in any young patient who presents with ECG changes mimicking ACS or AV blocks. History of fever may not present in all patients. Diagnosis of myocarditis is based on the history, clinical examination, ECG changes, echocardiography, and cardiac enzymes and confirmed on Cardiac MRI. The ECG changes in myocarditis are variable and nonspecific. Endomyocardial biopsy may not be possible in all patients. Treatment in stable patients is supportive.

#### CONSENT

Written informed consent was obtained from the patient for publication of this report and any accompanying images.

#### ETHICAL APPROVAL

As per international standards, ethical approval has been collected and preserved by the authors.

#### COMPETING INTERESTS

Authors have declared that no competing interests exist.

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