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# Acute Myopericarditis Mimicking ST-Elevation Myocardial Infarction in a Young Adult Male: A Case Report

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## Abstract

Myopericarditis can present with clinical features similar to ST-elevation myocardial infarction (STEMI), including chest pain, electrocardiographic (ECG) ST elevations, and elevated cardiac biomarkers. This overlap often leads to the activation of acute coronary syndrome protocols and sometimes the administration of thrombolytic therapy. We report a case of a 24-year-old male initially diagnosed and treated as STEMI based on ECG and enzymatic findings. However, he underwent percutaneous coronary intervention (PCI), which revealed normal coronary arteries, and subsequent cardiac MRI ruled out infarction, confirming the diagnosis of acute myopericarditis. This case highlights the importance of keeping a broad differential diagnosis in young adults presenting with STEMI-like features and reinforces the role of advanced imaging in avoiding unnecessary interventions.

## Keywords

Myopericarditis, STEMI Mimic, Cardiac MRI, Thrombolysis, ACS, ECG, Chest Pain, Troponin, ST-Segment

## 1. Introduction

Acute typical chest pain associated with ST-segment elevation and elevated cardiac biomarkers typically warrants activation of a STEMI pathway. However, non-ischemic myocardial injury, such as myopericarditis, can present with similar features and lead to misdiagnosis. Differentiating between these conditions is crucial, particularly in young patients without traditional cardiovascular risk factors [1]. Studies have shown that up to 26% of patients referred for coronary angiography with suspected STEMI are found to have normal coronary arteries [2]. In one case series, several of these patients were later diagnosed with myopericarditis follow-

ing advanced imaging and clinical correlation [3]. Myopericarditis is often triggered by viral infections, particularly in young males, and can lead to myocardial inflammation with associated chest pain and electrocardiographic changes [4]. The inflammatory process may also involve the pericardium, resulting in overlapping symptoms that resemble acute coronary syndromes and complicate the initial diagnosis [3]. Despite its presence among non-ischemic STEMI mimics, myopericarditis is frequently underrecognized in emergency settings due to nonspecific ECG features and a lack of definitive early markers [5]. Therefore, awareness of its presentation and pathophysiologic basis is critical to avoid unnecessary reperfusion therapy.

## 2. Case Presentation

A 24-year-old previously healthy male presented to the emergency department (ED) in a non-PCI facility with a 2-hour history of intermittent central chest and epigastric pain unrelated to exertion, position, or food intake, and described it as heaviness. Pain at rest and exertion; he denied any exacerbation or relieving factors. He denied radiation, sweating, or dyspnea. This was his first episode of such pain. He reported a sore throat and cough approximately three weeks prior. He had smoked one pack/day for 5 years.

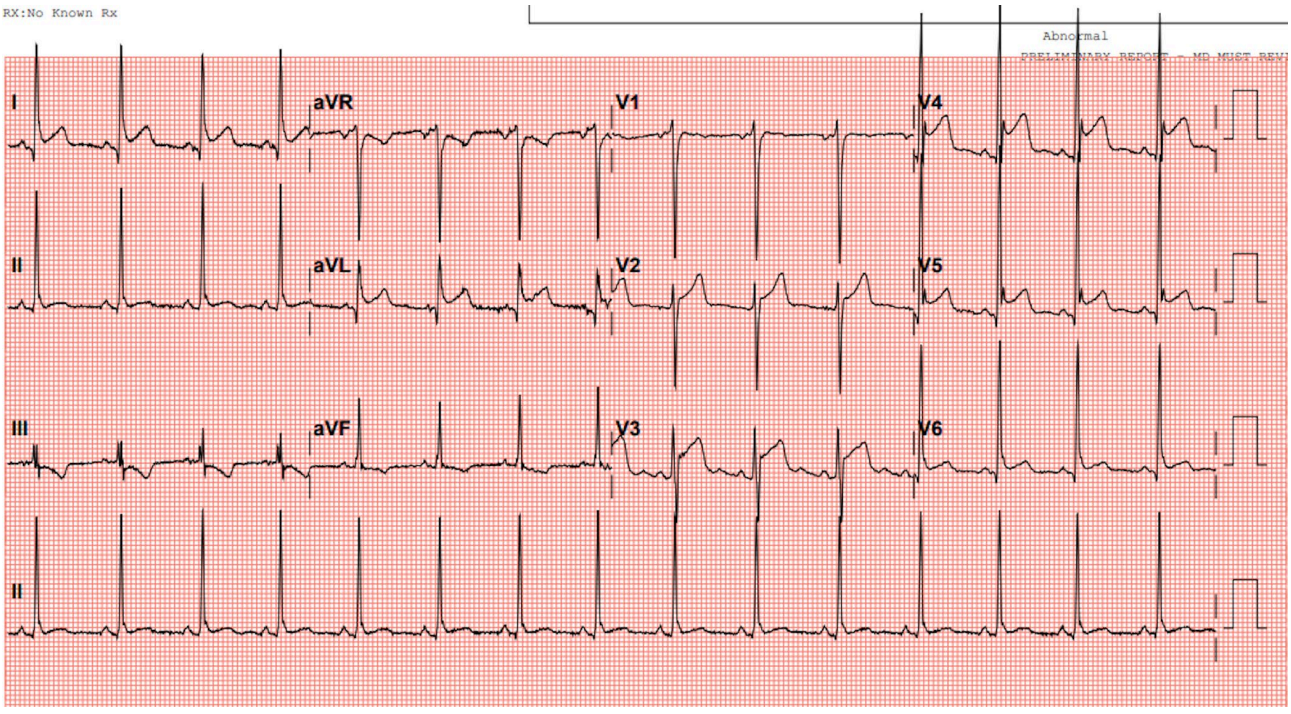


**Figure 1.** ST elevation in anteroseptal and lateral leads (V2-V6, Lead 1, and aVL) with reciprocal ST depression in the inferior leads (lead 3 and aVF).

On arrival, he had sinus tachycardia at 105 beats per minute (bpm), oxygen saturation of 99% on room air, blood pressure of 146/101, and tympanic temperature of 37˚C. On chest auscultation, there was no murmur, added sounds, or

pericardial rub; the rest of the physical examination was unremarkable. ECG (Figure 1) showed sinus rhythm with anteroseptal and lateral ST-segment elevations and reciprocal ST depression with T-wave inversion in leads III and aVF. He was loaded with aspirin 300 mg and given analgesia for pain, which subsequently improved. Due to his ECG changes with typical chest pain, he was transferred to a tertiary hospital with PCI capability. His second ECG (Figure 2) showed dynamic changes as anteroseptal and lateral STEMI. The patient was diagnosed with STEMI, cardiology services were consulted urgently, and received IV Tenecteplase thrombolysis 35 mg. Cardiac biomarkers came to be elevated (Table 1), and other investigations, including D-Dimer, liver function tests, renal function tests, lipid profile, full blood count, and chest x-ray, were normal. A follow-up ECG (Figure 3) 90 minutes post-thrombolysis showed persistent and worsened ST elevations, despite the patient being asymptomatic.

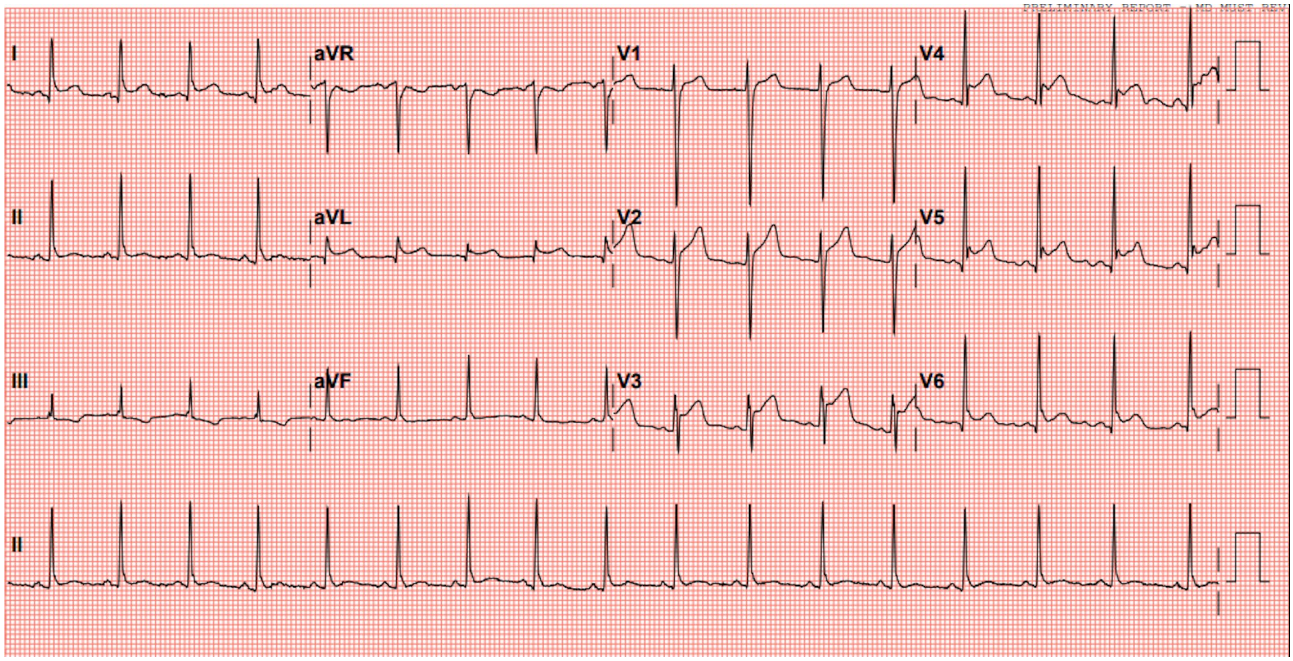


Figure 2. ECG on presentation to the tertiary hospital, showing dynamic changes in the anteroseptal and lateral leads.

He was started on nitroglycerin infusion and underwent rescue PCI, which revealed normal coronary arteries. Given the clinical stability, young age, and recent viral illness, myopericarditis was considered. The patient was treated with colchicine, a beta-blocker (bisoprolol), and an angiotensin-converting enzyme inhibitor (lisinopril).

Echocardiography revealed a preserved ejection fraction (55% - 60%) with trace mitral and tricuspid regurgitation. Cardiac MRI showed no evidence of myocardial infarction, edema, or fibrosis, confirming the diagnosis of myopericarditis. His symptoms resolved after 2 days of admission, and his ECG changes improved (Figure 4). He was discharged with bisoprolol and colchicine for 3 months with outpatient cardiology follow-up.

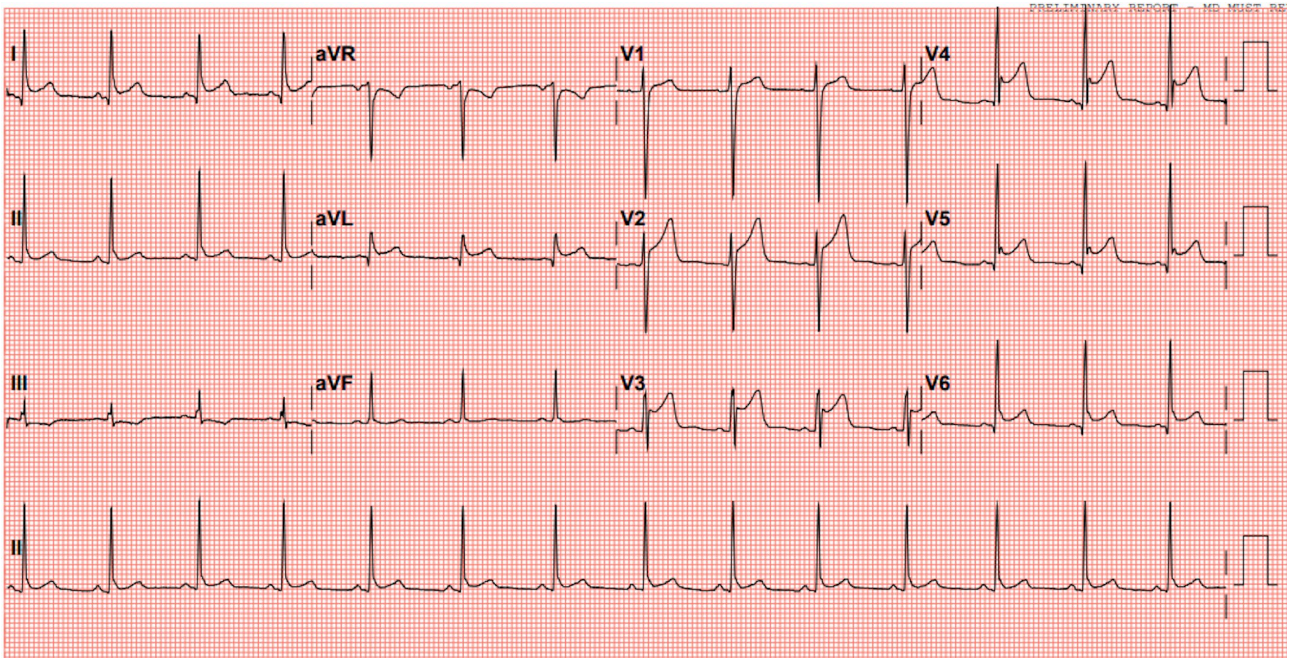


Figure 3. ECG 90 minutes post thrombolysis, showed persistent and worsening ST elevation in the anteroseptal and lateral leads.

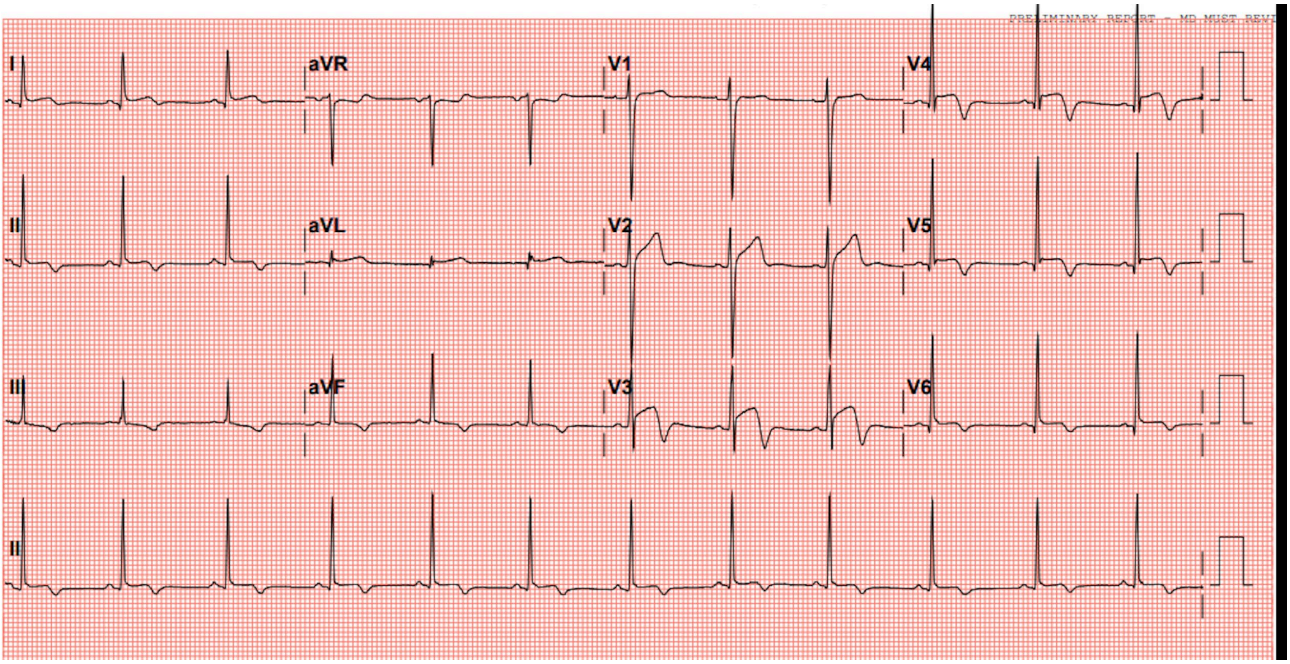


Figure 4. The ECG at discharge shows resolution of acute ST and extensive T wave changes.

Table 1. Patient laboratory results.

| Test | Result | Reference range |
|---|---|---|
| Troponin T | 318 (at 0 hour) → 356 (at 3 hours) → 347 ng/L (at 6 hours) | <14 ng/L |
| CK-MB (Mass) | 39.3 ng/mL | <6.23 ng/mL |

**Continued**

| | | |
|---|---|---|
| NT-proBNP | 62.02 pg/mL | <125 pg/mL |
| C-Reactive protein | 22.2 mg/L | <5.0 mg/L |
| Procalcitonin | 0.08 ng/mL | <0.05 ng/mL |

## 3. Discussion

STEMI mimics present a significant diagnostic challenge, particularly in young adults with minimal cardiovascular risk factors. Conditions such as myopericarditis, pericarditis, esophageal spasm, gastroesophageal reflux, and even acute pancreatitis can present with chest pain, ST-segment elevation, and elevated cardiac biomarkers, making it difficult to differentiate them from true ischemic events [2].

The pathophysiology of myopericarditis involves inflammatory injury to the myocardium, often triggered by viral infections, which results in myocyte necrosis, edema, and leakage of intracellular enzymes such as troponin [4]. The inflammatory process may also lead to localized ST-segment elevations on ECG, mimicking transmural infarction [6]. These ECG changes, mainly when focal, are often indistinguishable from those of STEMI and may be associated with elevated cardiac enzymes, further complicating diagnosis [5]. This underscores the importance of including myopericarditis in the differential diagnosis for STEMI, especially when the clinical picture is atypical [4].

Our case exhibits notable similarities to previously published cases [1] [5] [7]. These reports also describe young male patients presenting with chest pain, ST-segment elevations on ECG, and elevated troponins, ultimately diagnosed with myopericarditis based on imaging or normal angiographic findings. Furthermore, similar to our patient, some of these reported cases involved the administration of fibrinolytic therapy before establishing the correct diagnosis, emphasizing the significant diagnostic overlap and the associated risk of inappropriate treatment. This overlap is also noted in the broader literature, where acute non-rheumatic streptococcal myocarditis and acute perimyocarditis are recognized as mimics of STEMI [5]. While ECG and cardiac biomarker elevation are crucial in the initial assessment of suspected ACS, these findings are not specific and can be present in various cardiac inflammatory conditions, including myopericarditis. The case highlights the potential for over-activation of acute coronary syndrome protocols and the inadvertent administration of thrombolytic therapy in patients with underlying myopericarditis [1] [5].

The differential diagnosis in this case included acute coronary syndrome, pericarditis, esophageal spasm, and gastrointestinal causes such as gastritis or pancreatitis. The resolution of symptoms, young age, history of viral illness, absence of risk factors, and normal coronary angiogram favored a diagnosis of myopericarditis, and laboratory results and chest X-ray excluded other differentials.

This case contributes to the literature by being among the few reports documenting worsening ST elevation after thrombolysis in myopericarditis, a finding

that is more typical of evolving infarction. To reduce misdiagnosis and avoid unnecessary thrombolysis or PCI, emergency physicians and cardiologists should maintain a high index of suspicion for myopericarditis in young patients with atypical symptoms and no risk factors. Incorporating early cardiac MRI, repeated ECGs, and biomarker trending may provide safer and more accurate triage of suspected STEMI cases [8]. Further research into the evolution of ECG patterns in myopericarditis compared to STEMI could aid in earlier differentiation. The development and validation of clinical decision tools that integrate clinical presentation, laboratory findings, ECG features, and early imaging results may help refine the diagnostic approach and reduce the incidence of unnecessary fibrinolysis.

This case report has certain limitations. The absence of endomyocardial biopsy and subsequent lack of tissue confirmation means that the definitive gold standard for myocarditis diagnosis was not pursued [4]. While non-invasive findings supported the diagnosis of myopericarditis, endomyocardial biopsy can provide valuable information regarding the etiology and specific histological characteristics of myocardial inflammation [3]. Furthermore, the lack of a viral serology panel limits our ability to identify a potential infectious trigger for the myopericarditis. Finally, the absence of long-term follow-up data restricts our understanding of potential late complications or the recurrence of cardiac inflammation in this patient.

We suggest further research into ECG evolution patterns in myopericarditis and the development of clinical decision tools incorporating imaging, laboratory, and symptom-based criteria. Such efforts could reduce inappropriate fibrinolysis and optimize patient outcomes.

## 4. Conclusion

Acute myopericarditis should be strongly considered in young patients with atypical chest pain, especially with recent viral illness and absence of cardiovascular risk factors. A multidisciplinary and stepwise diagnostic approach, incorporating advanced imaging techniques such as cardiac MRI, is essential to achieve an accurate diagnosis and optimize patient management, thereby avoiding potentially harmful interventions.

## Conflicts of Interest

The authors declare no conflict of interest.

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