



Case report

Acute myocarditis mimicking myocardial infarction can misdirect the diagnostic approach☆

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ABSTRACT

Acute myocarditis is a well-recognized but rare manifestation of mostly viral infections. It can present with various clinical manifestations and may mimic myocardial infarction (MI) since patients usually present with chest pain, and the electrocardiographic changes similar to those observed in acute ST-elevation MI. We, herein, present such an extreme case of acute myocarditis characterized by dynamic ST segment elevation with reciprocal changes in the electrocardiogram.

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Introduction

Acute myocarditis is a well-recognized but rare manifestation of mostly viral infections with a broad spectrum of symptoms and clinical features.¹ The diagnosis of acute myocarditis is one of the most challenging issues in cardiology due to the nonspecific pattern of clinical presentation. It can present with various clinical manifestations and may mimic myocardial infarction (MI), since patients usually present with chest pain, and the electrocardiographic changes similar to those observed in acute ST-elevation MI.^{2–4} In this report, we present such an extreme case of acute myocarditis characterized by dynamic ST segment elevation with reciprocal changes in the electrocardiogram (ECG).

Case report

A 25-year-old man with no previous cardiac history presented to the emergency department with intermittent retrosternal crushing chest pain radiating to back. On admission, he was pain-free and had a two-

day history of symptoms of acute upper respiratory infection including fever, cough, sore throat and nausea. There was no history of smoking, diabetes, hypertension, dyslipidemia or family history of coronary artery disease. On examination, initial heart rate was 84 bpm, temperature 37.4 °C, and arterial blood pressure 115/75 mm Hg. Cardiac examination revealed normal findings, with no additional sounds, murmurs or pericardial rub. ECG on admission revealed sinus rhythm, normal QRS axis and minimal ST-segment elevation in leads II, aVF, I, aVL, V5, and V6 with reciprocal ST segment depression at lead V1 (Fig. 1). An initial echocardiogram showed mild hypokinesia in the apical region of the left ventricle with an ejection fraction of 55%. Cardiac biomarkers revealed serum Troponin I of 37.7 ng/mL (normal < 0.04 ng/mL), creatine kinase (CK) of 2250 U/L (normal 20–171 U/L), and CK-MB of 181 IU/L (normal < 0–24 IU/L). White blood cell count was 13,100/mL, and erythrocyte sedimentation rate was 28 mm/h. Other routine biochemical blood tests were within normal limits. With all these findings, our initial diagnosis was acute myocarditis, and we decided to start angiotensin-converting-enzyme inhibitor and beta-blocker therapy because of the mild ventricular dysfunction. After 30–35 min of admission, the patient's chest pain increased progressively. The ECG taken at that time showed 4–5 mm ST-segment elevation in inferior and lateral leads with ST segment depression in right precordial leads (Fig. 2A). Posterior ECG showed that the ST-segment depression in right precordial leads was due to the reflection of ST elevation of the posterior wall (Fig. 2B). Concurrent echocardiography showed an apparent regional hypokinesia in the middle and basal segments of the inferior and posterior walls with an ejection fraction of 45%, whereas

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Fig. 1. ECG on admission revealed sinus rhythm and minimal ST-segment elevation in leads II, aVF, I, aVL, V5, and V6 with reciprocal ST segment depression at lead V1. ECG, electrocardiogram.

the ST-segment elevation in especially leads I, aVL was concave seen in pericarditis. Therefore, we performed coronary angiography due to the acute changes. Coronary angiography showed normal epicardial coronary arteries (Fig. 3A) and we observed mild hypokinesia in the apical region of the left ventricle. Over the following days, ST-segment elevations decreased gradually, and ECG revealed biphasic T waves in the leads I, II, aVL, V5 and V6 seen in the subacute phase of myocarditis. The patient discharged on perindopril 5 mg q.d. and metoprolol succinate 50 mg q.d. (Fig. 3B). Follow-up echocardiography at two weeks revealed normal findings without wall motion abnormality.

Discussion

The symptoms and clinical features of myocarditis range from an asymptomatic state to fulminant cardiogenic shock and sudden death. The diagnosis of acute myocarditis, especially when it mimics MI, is challenging due to wide variation in the clinical presentation such as our patient. Although endomyocardial biopsy (EMB) is the gold standard for diagnosis, current guidelines recommend the EMB only in a limited number of clinical scenarios such as life-threatening clinical presentations of myocarditis.⁵ In addition, selective coronary angiography is recommended in patients with suspected myocarditis presented with an acute coronary syndrome-like findings. CT coronary angiography may also be an alternative to conventional angiography.⁶ Non-invasive imaging techniques such as cardiac magnetic resonance

(CMR) imaging can be useful for diagnosis and delineating the extent of the disease.^{5,6} However, its usage may be limited regarding availability in acute cases. In the present case, history and clinical presentation (young age, low coronary risk profile, concomitant flu-like symptoms in the few days before admission) were consistent with myocarditis. But the localized ST-segment elevation along with the “reciprocal-like” changes in addition to segmental left ventricular dysfunction and worsening chest pain raised the possibility of an acute MI. Then, the patient underwent coronary angiography for eliminating an acute coronary syndrome. As is seen in our case, confirming the diagnosis with coronary angiography in patients with typical symptoms of acute MI is of great importance especially in young cases with myocarditis or other non-coronary reasons (such as Takotsubo syndrome).⁷

Conclusion

The diagnosis of acute myocarditis is one of the most challenging issues in cardiology. A detailed history, examination and ECG interpretation are usually adequate for diagnosis. However, there may be an extreme similarity regarding ECG findings in cases mimicking acute MI. Therefore, the clinicians should keep in mind that coronary angiography is necessary before taking an action with treatment modalities such as thrombolysis due to serious side effects.

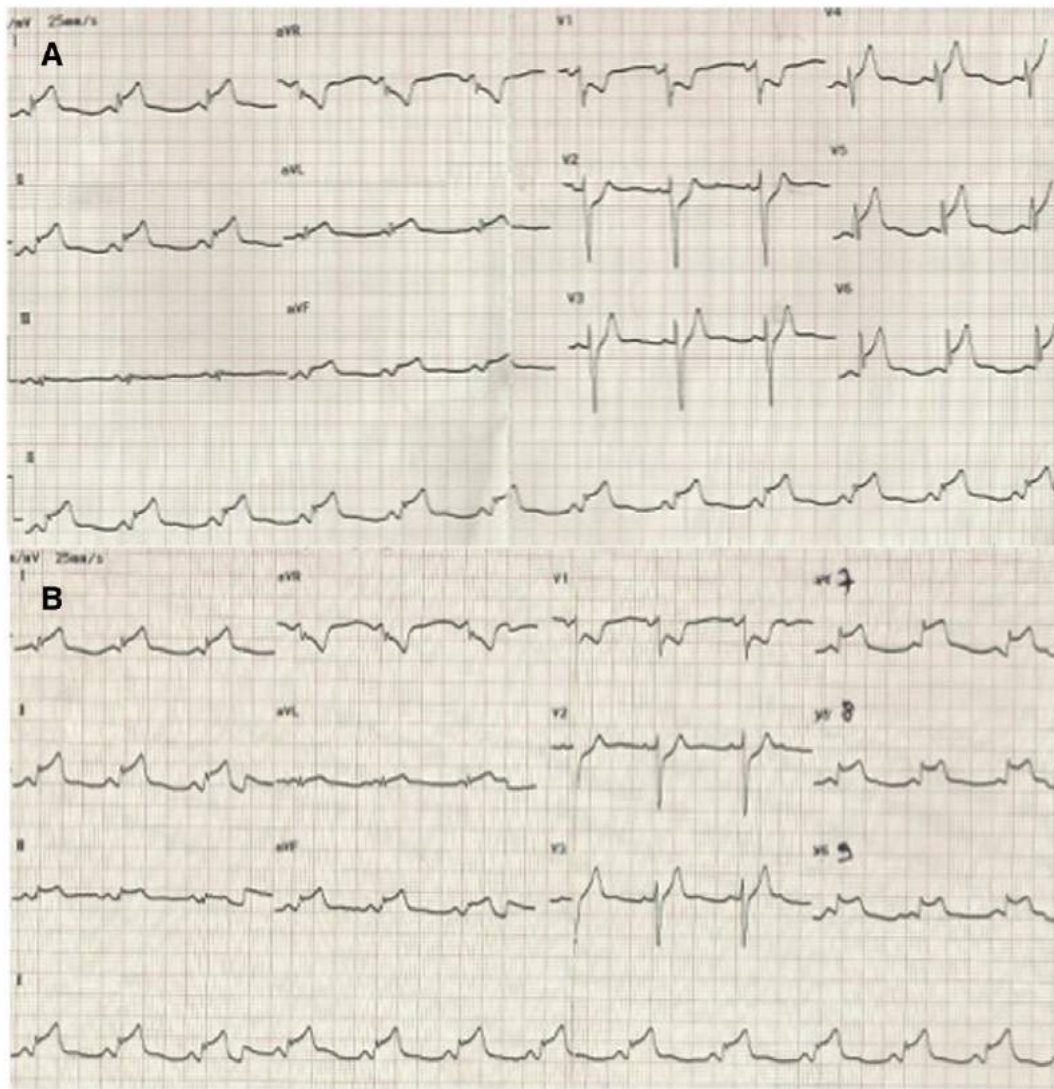


Fig. 2. ECG taken when the patient had chest pain showed 4–5 mm ST-segment elevation in leads I, II, III, aVF, aVL, and V4–V6 with ST segment depression in right precordial leads (A). Posterior ECG showed that the ST-segment depression in right precordial leads was due to the reflection of ST elevation of the posterior wall (B). ECG, electrocardiogram.

References

- Lieberman EB, Hutchins GM, Herskowitz A, Rose NR, Baughman KL. Clinicopathologic description of myocarditis. *J Am Coll Cardiol* 1991;**18**:1617–1626.
- Moitard N, Mewion N, Bonnefoy E, Abdellaoui M, Revel D, Kirkorian G. Acute myocarditis mimicking lateral myocardial infarction. *Anaesth Intensive Care* 2008;**36**(5):739–742.
- Basic D, Gupta S, Kwong RY. Parvovirus B19-induced myocarditis mimicking acute myocardial infarction. *Circulation* 2010;**121**(7):e40–e42.
- Testani JM, Kolansky DM, Litt H, Gerstenfeld EP. Focal myocarditis mimicking acute ST-elevation myocardial infarction. *Tex Heart Inst J* 2006;**33**(2):256–259.
- Fridrich MG, Strohm O, Schulz-Menger J, Marciniak H, Luft FC, Dietz R. Contrast media enhanced magnetic resonance imaging visualizes myocardial changes in the course of viral myocarditis. *Circulation* 1998;**97**(18):1802–1809.
- Caforio ALP, Pankuweit S, Arbustini S, et al, European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J* 2013;**34**(33):2636–2648.
- Buchholz S, Rudan G. Tako-tsubo syndrome on the rise: a review of the current literature. *Postgrad Med J* 2007;**83**(978):261–264.

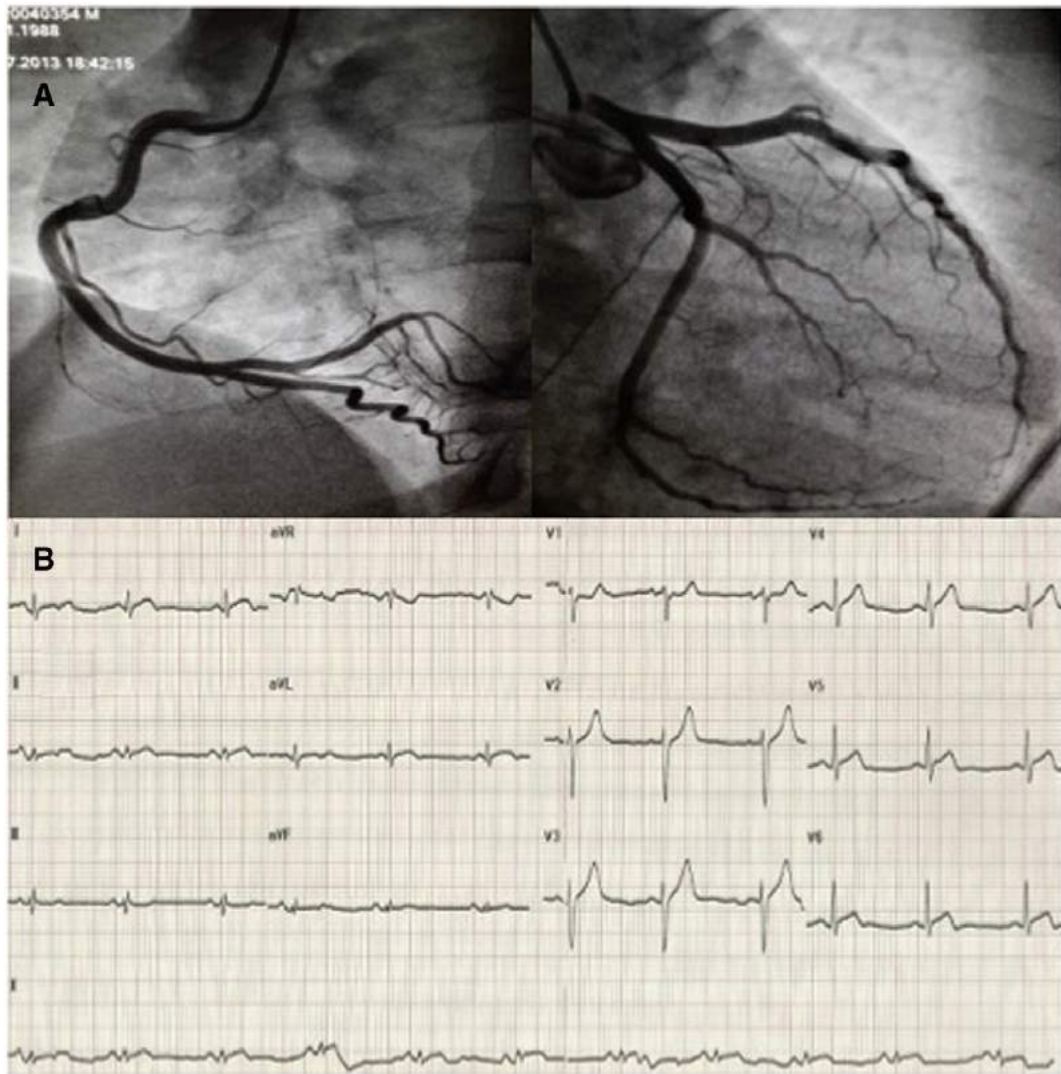


Fig. 3. Coronary angiography showed normal epicardial coronary arteries (A). ECG on discharge revealed biphasic T waves in the leads I, II, aVL, V5 and V6 (B). ECG, electrocardiogram.