


CASE REPORT

Electrocardiogram evolution of acute anterior ST-segment elevation myocardial infarction following pericarditis

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Abstract

Electrocardiogram is a powerful tool for differentiating acute ST-segment elevation myocardial infarction (STEMI) and pericarditis. However, an unusual ECG presentation of the simultaneous occurrence of the two conditions has not been reported previously. In this article, we report a case of ECG evolution of acute anterior STEMI following pericarditis with pericardial effusion (PE) and find that QRS complex widening in ECG lead with maximal ST-segment elevation is also applicable for identifying STEMI even in patients with prior pericarditis. Undoubtedly, our case can help prevent emergency physicians from making incorrect diagnoses and administering inappropriate treatments.

KEYWORDS

ECG evolution, pericarditis, ST-segment elevation myocardial infarction

1 | INTRODUCTION

Acute ST-segment elevation myocardial infarction (STEMI) and pericarditis are two distinct disorders, but they result in similar chest pain and manifest as ST-segment elevation on ECG. Over the past decades, many ECG features have been found to discriminate STEMI and pericarditis, including the morphology of ST-segment elevation (Spodick, 1974), PR-segment depression (Spodick, 1974), Spodick's sign (Witting et al., 2020), the orientation of ST-segment vector (Bischof et al., 2016), and QRS complex duration (Rossello et al., 2014). However, when two conditions occur simultaneously, what will happen to the ECG signals? This article reports a case of ECG evolution of acute anterior STEMI following pericarditis with pericardial effusion (PE) for the first time to provide a reference for emergency physicians to use to help them make correct clinical decisions.

2 | CASE REPORT

A 68-year-old woman with a medical history of hypertension, exertional angina, and ongoing tobacco use was admitted to the primary emergency department (ED) with severe chest pain accompanied by nausea and sweating for 2 h. Her blood pressure was 140/90 mmHg, and her heart rate was 74 bpm. Neither cardiac murmurs nor a friction rub was auscultated, and lung auscultation revealed no rales. The first electrocardiogram (ECG) showed a sinus rhythm, ST-segment elevation, and PR-segment depression in the anterior (V2–6) and inferior (II, III, and aVF) leads with concomitant PR-segment elevation in lead aVR. Moreover, low voltages (<5 mV) in six limb leads were observed (Figure 1a). Cardiac biomarkers showed an initial cardiac troponin level of 0.39 ng/ml (normal level <0.034 ng/ml), and the results of other laboratory investigations, including a complete blood count,

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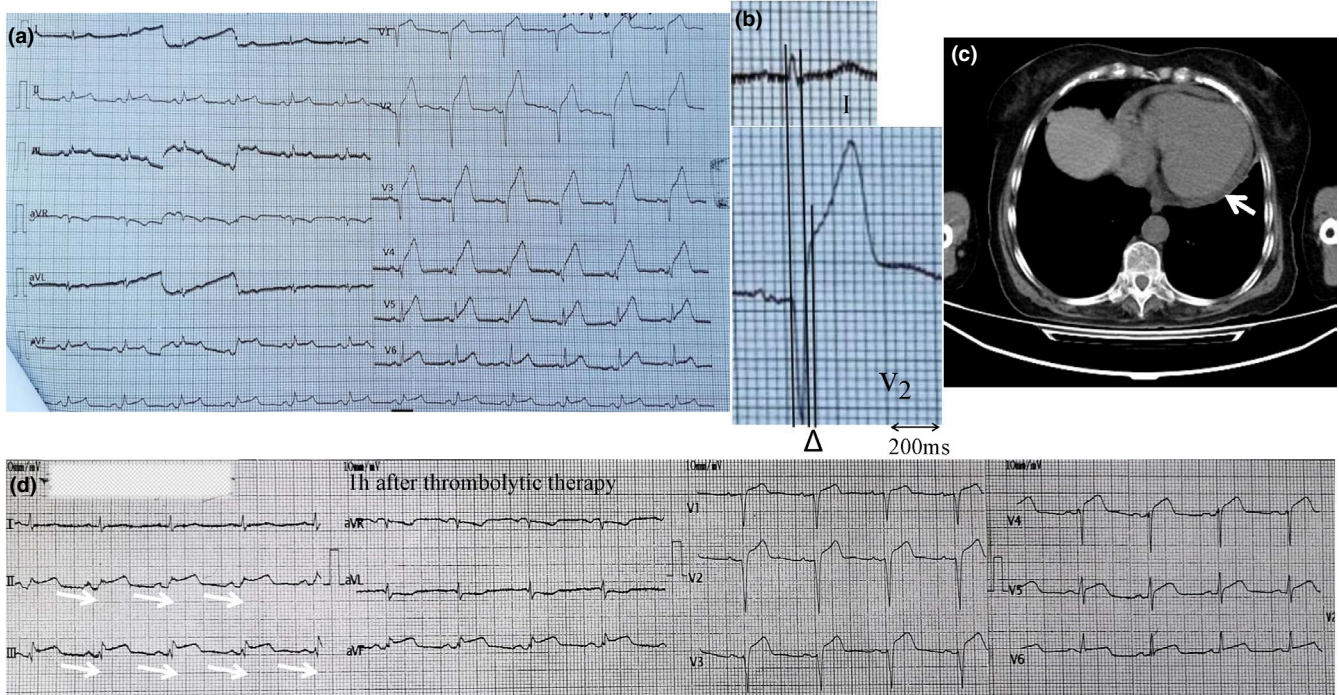


FIGURE 1 (a): An emergency ECG recording shows ST-segment elevation and PR-segment depression in the anterior (V2–6) and inferior (II III aVF) leads with concomitant PR-segment elevation in lead aVR. Also, low voltages (<5 mV) in six limb leads were observed. (b): Magnified recording from lead I with isoelectric ST segment and V2 with maximal ST-segment elevation showing differences in the QRS complex duration (Δ). (c): Pericarditis with pericardial effusion as revealed by computed tomography (arrow). (d): An ECG recording 1 hour after thrombolytic therapy demonstrates that the amplitudes of the ST-segments in V2–V6 have been reduced to half of their original amplitude, though the amplitude of ST-segment elevation in inferior leads increased and PR-segment depression in inferior leads as well as PR-segment elevation in lead aVR did not exhibit significant changes. Moreover, Spodick's sign, a downsloping of the ECG baseline (the TP segment), indicating pericarditis, became obvious (arrow)

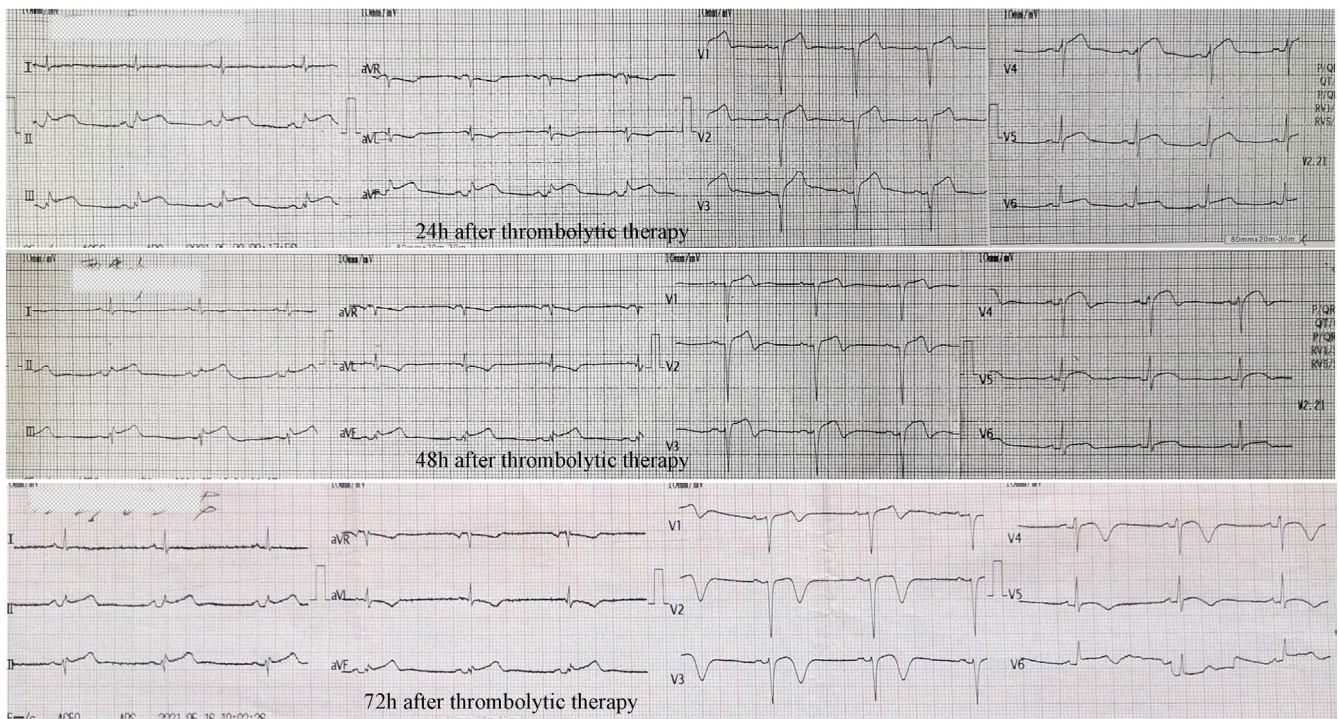


FIGURE 2 Variation in ECG after thrombolytic therapy mainly shows a dynamic evolution of ST-T waves in leads V1–V6 rather than in six limb leads

erythrocyte sedimentation rate, and C-reactive protein, were all at normal levels. All the evidence mentioned above may suggest acute large anterior wall ST-segment elevation myocardial infarction, but PR-segment deviations and the absence of reciprocal ST-segment depression may confuse emergency physicians because acute pericarditis cannot be ruled out. Bedside echocardiography was performed and revealed left ventricular wall hypokinesia with pericardial effusion (PE), which was also confirmed on a chest CT image (Figure 1c). According to the criteria raised by Xavier Rossello et al. (Rossello et al., 2014) to differentiate acute pericarditis and myocardial infarction by QRS complex duration, the prolongation of the QRS complex duration in ECG lead with maximal ST-segment elevation appeared in this patient, which represented transmural myocardial ischemia (Figure 1b). It was reasonable to speculate that the patient was undergoing STEMI and that pericarditis was present before STEMI. Therefore, thrombolytic therapy with recombinant human TNK tissue-type plasminogen activator was performed immediately because the anticipated door-to-balloon time was more than 120 min. After 1 h, the patient's chest pain was completely relieved. When the ECG signal showed the amplitude of the signal in the ST-segments in V2–V6 decreased to half of the original amplitude, the amplitude of the ST-segment elevation in leads II, III, and aVF increased and PR-segment depression was noted in inferior leads; also, PR-segment elevation in lead aVR did not exhibit significant changes. Moreover, Spodick's sign, a downsloping of the ECG baseline (the T-P segment), became obvious (Figure 1d). In the following week, the ECG mainly demonstrated a dynamic evolution of ST-T waves in leads V1–V6 rather than in leads II, III, and aVF, and PR-segment depression in inferior leads along with PR-segment elevation in lead aVR remained prominent. (Figure 2). Coronary angiography performed 7 days after presentation at a higher-level hospital

showed stenosis of 90% of the proximal-to-mid left anterior descending artery (LAD) (Figure 3a), which was successfully treated with a drug-eluting stent (Figure 3b). Also, coronary angiogram of the left circumflex and right coronary arteries exhibited no significant stenosis. Postoperative ECG showed that ST-segment elevation and PR-segment depression in inferior leads still existed along with PR-segment elevation in lead aVR (Figure 3c). Simultaneously, a mild increase in pericardial effusion was detected by echocardiogram (Figure 3d). The patient was asymptomatic and discharged on the second postoperative day. Two months later, the follow-up echocardiogram showed trivial pericardial effusion.

3 | DISCUSSION

Although the ECG at the onset of chest pain was of low quality, the features of pericarditis were exhibited, including widespread ST-segment elevation and PR-segment depression, especially in inferior leads, along with ST-segment depression in lead aVR. However, the V2 lead with maximal ST segment elevation showed a longer QRS complex than the I lead with isoelectric ST segments, which implied transmural myocardial ischemia (STEMI) (Rossello et al., 2014). Of course, left ventricular wall hypokinesia on echocardiography contributes to the diagnosis of STEMI. Moreover, it is well known that early pericarditis occurs between the 2nd and 4th days after AMI and usually only lasts 1–2 days, and significant pericardial effusion (PE) is not observed (Lichstein, 1983); therefore, we speculate that the PE of the patient was present before STEMI. Another important aspect could not be excluded: probable Wellens' syndrome of the patient due to severe stenosis of the proximal-to-mid left anterior descending artery (LAD) before STEMI may have resulted in pericarditis with PE, which has been reported previously (Yun-Tao

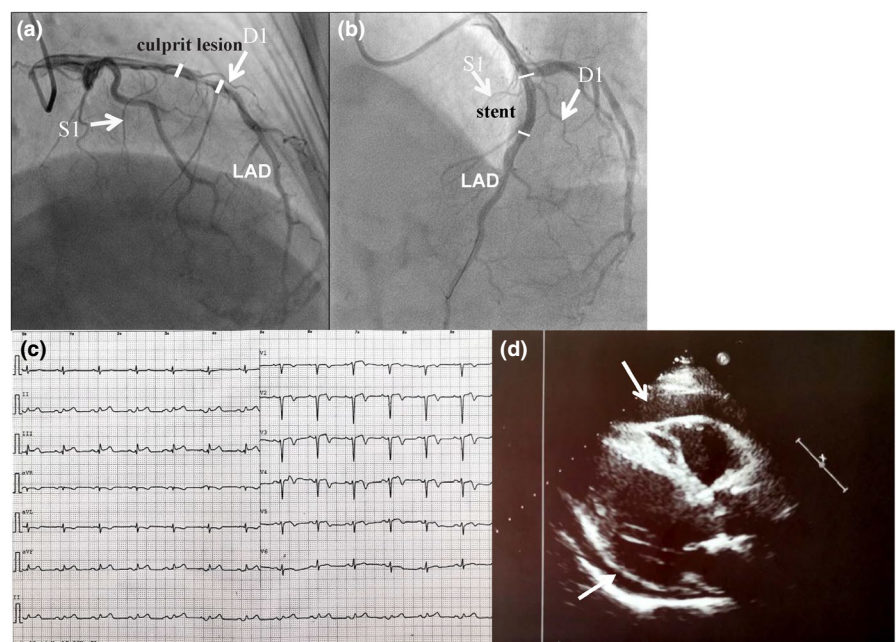


FIGURE 3 (a): Coronary angiogram showed that the culprit lesion was located on the proximal-to-mid left anterior descending artery (LAD) and the stenosis was close to both the first diagonal branch (D1) and the first septal perforator (S1). (b): Coronary angiogram after LAD stenting. (c): An ECG recording after stenting shows ST-segment elevation. Also, PR-segment depression in inferior leads still exists, accompanied by PR-segment elevation in lead aVR. (d): A mild increase in pericardial effusion was revealed by echocardiogram (arrow)

& Chen, 2017). A mild increase in PE 1 week later may be partially related to Dressler syndrome, which is often seen from 3–4 days to 2–6 weeks after AMI (Leib et al., 2021).

In fact, this kind of ECG of concomitant inferior STE in anterior MI can be caused by the presence of a wrap-around LAD anatomy, distal LAD occlusion, or both (Bozbeyoglu et al., 2019), alternatively, by a rare situation of simultaneous thrombosis of the LAD and the RCA (Khareshi et al., 2015). However, it is interesting that the ECG characteristics mentioned above appeared in our patient whose culprit lesion was located at the proximal-to-mid left anterior descending artery (LAD) which is close to both the first diagonal branch (D1) and the first septal perforator (S1). According to previous literature (Engelen et al., 1999; Vasudevan et al., 2004), almost all acute occlusions of this lesion may result in inferior ST-segment depression, which will improve gradually with the restoration of blood flow. In contrast, our case showed that ST-segment elevation in inferior leads tends to become increasingly remarkable after reperfusion. The most likely explanation for this ECG phenomenon is pericarditis with PE. It is well known that pericarditis is a diffuse inflammation of the entire pericardium, and the resulting ST axis should be at approximately 45° toward the apex of the heart (Bischof et al., 2016). Using a hexaxial reference lead system of the 12-lead ECG, the angles between the 45° axis and inferior leads have been shown to range from 15° to 75°, and none of them exceed 90°; thus, pericarditis may lead to ST-segment elevation of inferior leads. In this case, the patient suffered from both acute occlusion of the proximal-to-mid LAD and pericarditis. ST-segment elevation derived from pericarditis may counteract and surpass ST-segment depression related to acute myocardial infarction of the anterior wall in inferior leads; this effect will be amplified following the improvement of inferior ST-segment depression after reperfusion. Therefore, inferior ST-segment elevation is more pronounced after intravenous thrombolytic therapy. To the best of our knowledge, this is the first report of ECG evolution due to acute occlusion of the proximal-to-mid LAD when pericarditis with PE has been noted.

4 | CONCLUSION

Our case findings indicate that the criteria used to discriminate STEMI from pericarditis by the prolongation of the QRS complex in ECG leads with maximal ST-segment elevation are also applicable for identifying STEMI even in patients with prior pericarditis. Additionally, it should be noted that pericarditis may interfere with localizing the occlusion site in the left anterior descending coronary artery by ECG in patients with acute anterior myocardial infarction.

ACKNOWLEDGEMENT

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CONFLICT OF INTEREST

There are no conflicts of interest that should be stated.

ETHICAL APPROVAL

This case report was approved by the Ethical Committee of the Fourth Hospital of Harbin Medical University and followed the ethical Declaration of Helsinki. Patient had the opportunity to read the present case report and had no objections to the case report.

AUTHOR CONTRIBUTIONS

All authors reviewed and approved the manuscript. Xiaopeng Bai: Conceptualization, Writing - original draft. Yufeng Wang and Shuai Shi: Investigation, Methodology, Data collection. Lixiu Sun and Jia Zhao: Investigation, Writing - review & editing. Bingchen Liu: Idea conceptualization, gave suggestions on this manuscript, final approval.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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