Received: 28 January 2022 | Revised: 16 March 2022 | Accepted: 22 March 2022

DOI: 10.1111/anec.12987

CASE REPORT

WILEY

Acute proximal left anterior descending coronary artery occlusion presenting with Normal ECG: A case report

Yuecheng Hu MD[1] | Dongxia Jin MD[1] | Chunwei Liu MD[1] | Rui Zhang MD[1] | Shiying Zhou MD[2] | Le Wang MD[1] | Hongliang Cong MD, PhD[1]

[1]Department of Cardiology, Tianjin Chest Hospital, Tianjin University, Chest Clinical Medical College of Tianjin Medical University, Tianjin, China

[2]Department of Cardiology, People's Hospital of Hotan District in Xinjiang, Xinjiang, China

Correspondence
Yuecheng Hu and Hongliang Cong, Department of Cardiology, Tianjin Chest Hospital, Tianjin University, Chest Clinical Medical College of Tianjin Medical University, No. 261, Taierzhuang South Road, Jinnan District, Tianjin, 300222, China.
Email: hwhitecrane@163.com (Y.H.); hongliangcong@126.com (H.C.)

Funding information
This study was supported by a grant from Science and Technology Funding of Tianjin Chest Hospital (NO.2018XKZ07) and a grant from Tianjin Municipal Health Bureau(CN)(NO.MS20016)

Abstract

The middle-aged male was diagnosed with "acute anterior wall myocardial infarction" based on clinical symptoms, laboratory examination, and coronary angiography (CAG), but his ECG showed no significant change in QRS wave or ST-T within 6 h of admission. Thus, a perfect explanation with the existing theory is difficult, and only the case is presented here.

KEYWORDS
basic, clinical, ventricular tachycardia/fibrillation, non-invasive techniques - T-wave alternans

1 | CASE REPORT

A 43-year-old man presented to our emergency department complaining of persistent chest pain for 4 h after dinner, accompanied by profuse sweating. The electrocardiogram (ECG) immediately revealed sinus tachycardia with no obvious abnormality of the ST-T segment in each lead (Figure 1a). After a while, the patient suddenly lost consciousness because of ventricular fibrillation, and he regained consciousness after extra-cardiac compression and electrical cardioversion. The ECG was rechecked and found to be normal with no significant changes (Figure 1b). The patient continued to complain of excruciating chest pain. Meanwhile, the laboratory tests revealed: Myo 124 ng/ml (0–107 ng/ml); cTnI 0.05 ng/mL (0–0.05 ng/ml); D-Dimer 0.4 ng/ml (<0.5 ng/ml). An urgent contrast-enhanced CT of the chest ruled out the diagnoses of "acute aortic dissection" and "acute pulmonary embolism." The third ECG revealed sinus tachycardia, 0.1 mV ST-segment elevation in leads II, III, and aVF compared to the first and second ECGs, without mirror changes in other leads (Figure 1c). The patient was highly suspected of having "acute inferior myocardial infarction" and was prepared for emergency PCI after receiving loading doses of aspirin and ticagrelor. The fourth ECG showed no obvious abnormality within 2 hours after admission before the operation (Figure 1d). Emergency transthoracic echocardiography showed normal sizes of cardiac chambers decreased anterior wall motion of the left ventricle, with LVEF of 55%. After adequate heparin anticoagulation, the emergency CAG showed total

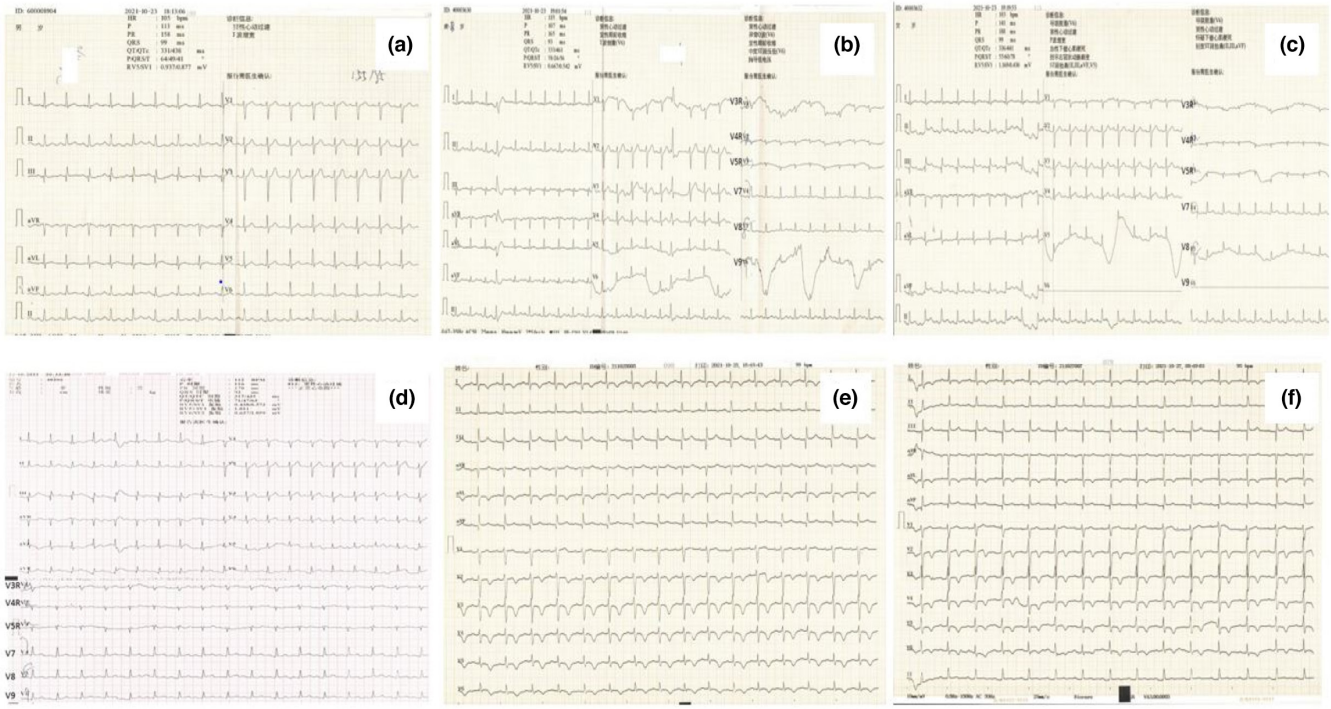

FIGURE 1 ECGs dynamic changes of the patient. Note: (a) The first ECG of the patient after admission. It showed sinus tachycardia without obvious abnormality. (b) The second ECG of the patient after admission, about 50 min away after the first ECG. It showed sinus tachycardia with an occasional ventricular premature beat. Compared with the previous figure, R and ST-T of each lead have no obvious abnormal changes. (c) The third ECG of the patient after admission, about 70 min after the first ECG. It showed sinus tachycardia. Compared with the first and second images, ST segments of the leads of II, III, and aVF were raised by 0.1 mV, while R and ST-T of the anterior wall leads showed no significant abnormal changes. (d) The fourth ECG of the patient after admission, about 140 min from the first ECG. It showed sinus tachycardia. Compared with the first and second ECGs, the R and ST-T of each lead showed no significant abnormal changes. Compared with the third ECG, ST segments of leads II, III and aVF returned to baseline. (e) The patient's ECG on the second day after emergency PCI. Compared with the preoperative ECGs, the T wave showed symmetry inversion in the lead of V2-6, I, and aVL, without R wave attenuation. (f) The ECG of the patient on the 4th day after emergency PCI. Compared with the ECG on the second day after the operation, the T-wave inversion in lead V2-6 was shallower gradually, and the R wave was not attenuated

occlusion near the opening of the proximal left anterior descending coronary artery (LAD), with heavy thrombus load (Figure 2a–c). A large number of thrombus and 1.0 cm strips of gelatinous tissue, composed of a large number of fibrin, red blood cells, and inflammatory cells, were aspirated (Figure 3a,b). A stent was successfully implanted at the culprit lesion (90% residual stenosis) of LAD, with the final flow of TIMI III. And the final angiogram depicted the anatomy of LAD wrapping around the apex of the left ventricle (Figure 3c,d). After the operation, the patient was monitored continuously for one week. On the second and fourth days following emergency PCI, the ECGs revealed a classic dynamic ischemic change of T-wave inversion in precordial leads (V2 to V6) and lateral extremity leads (I and aVL), but no R wave attenuation was observed (Figure 1e,f). The peaks of myocardial enzymes were as follows: cTnT 12.1 ng/mL (<0.1 ng/ml), CK 3124 U/L (25–190 U/L), and CK-MB 284 U/L (0–25 U/L), respectively. Transthoracic echocardiography performed before discharge revealed normal atrial and ventricular sizes as well as decreased left ventricular anterior wall motion with LVEF of 40%. The patient was diagnosed with "acute anterior wall myocardial infarction" and was discharged with dual antiplatelets, statin, beta-blocker, and sacubitril/valsartan, as per the guidelines.

2 | DISCUSSION

The patient was suspected of having an acute myocardial infarction (AMI) in the inferior wall due to occlusion of the right coronary artery (RCA) or the left circumflex coronary artery, based on unrelieved chest pain, enhanced CT findings, reversible ventricular fibrillation, transient ST-segment elevation of leads II, III, and AVF, despite the lack of myocardial enzymes (LCX). The diagnosis of AMI was confirmed by angiograms of emergency CAG and acute occlusion by massive thrombus, but the culprit artery was the LAD, not the LCX or the RCA. Fortunately, the patient was treated and discharged with the diagnosis of "acute extensive anterior wall myocardial infarction" after timely LAD revascularization. But the most perplexing aspect was that the patient's ECGs, particularly the anterior wall leads, showed no significant and dynamic change in either R wave or ST-T.

ST-segment elevation occurs in two or more adjacent leads, typically due to acute complete occlusion of a coronary artery based on the diagnostic criteria for AMI (Jung & Elsässer, 2018). However, the first ECG can only diagnose about half of AMI patients. In the early stages of AMI, the ECG can be normal or close to normal; approximately 40% of confirmed AMI showed continuous dynamic

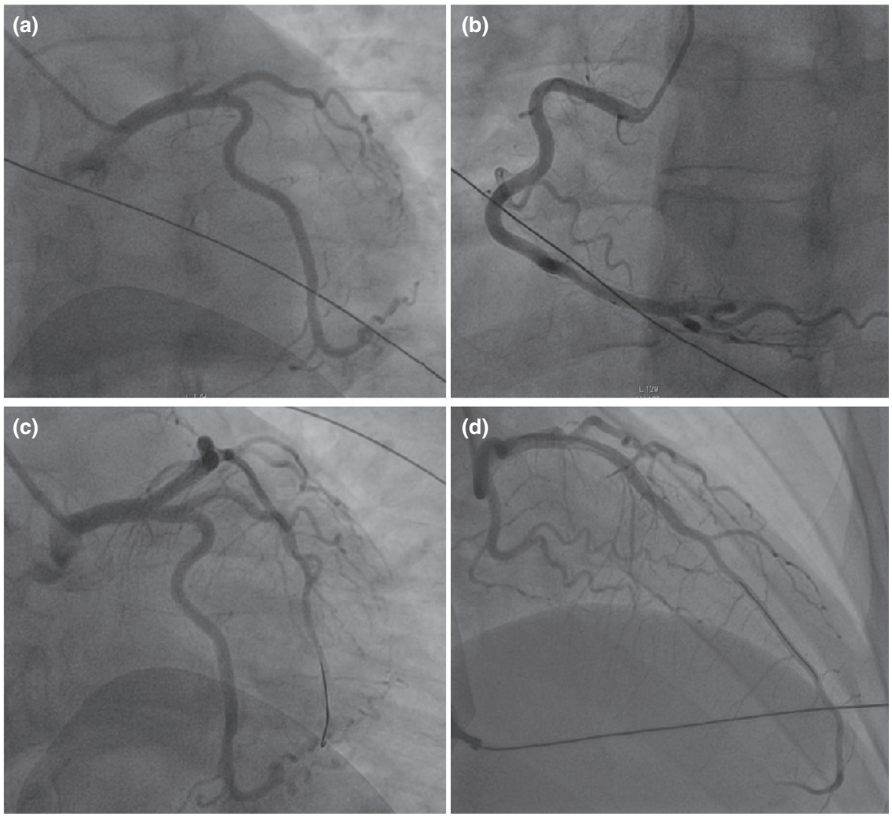

FIGURE 2 Results of coronary angiography, thrombus aspiration, and stent implantation in patients. (a and b) show left coronary angiography, indicating that the proximal segment of LAD was occluded near the opening, without obvious stenosis of LCX and RCA. (c) depicts the restoration of TIMI3 blood flow after stent implantation at the occlusion of LAD. (d) presents angio-figure of LAD after PCI

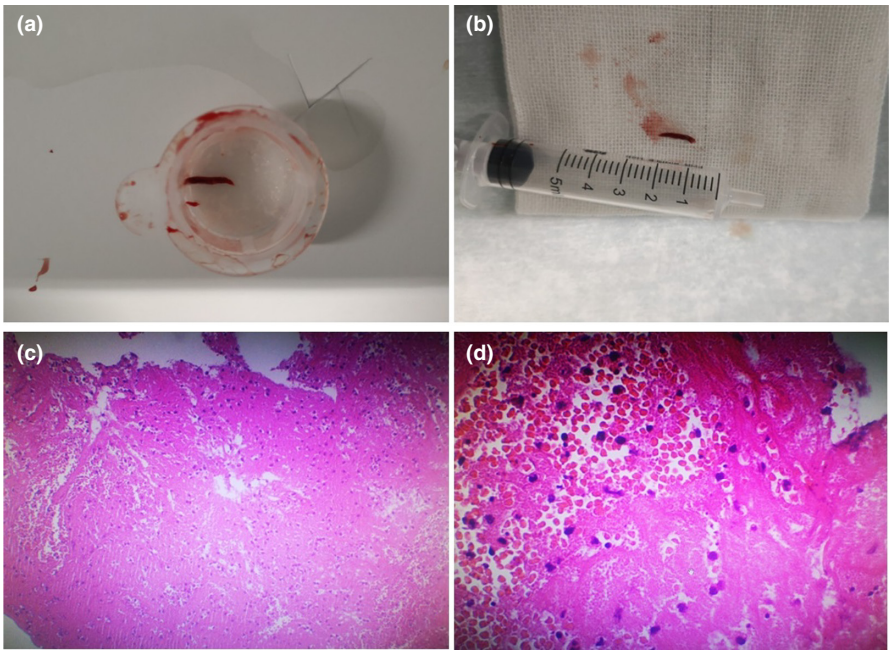

FIGURE 3 Extractive tissue of patient's LAD and HE staining pathological section of the tissue. (a and b) show large pieces of tissue extracted from LAD, with about 1.0 cm length jelly-like. (c and d) show the pathological sections of the tissue stained by HE. A large number of mixed thrombus composed of fibrin, red blood cells, and inflammatory cells are shown in (c) (10×10 magnification). A large number of red blood cells and inflammatory cells are shown in (d) (10×40 magnification)

evolution without any ST-segment elevation. This dynamic evolution may be reflected in changes to the T wave, ST segment, or QRS complex; however, it is extremely rare for patients with acute occlusion near the opening of the proximal LAD to have no dynamic change.

The mechanisms of patients diagnosed with ST-segment elevation AMI(STEMI) without typical ECG changes are discussed below. (1) Serious stenosis of multiple coronary arteries, or myocardial infarction in multiple parts, and the changes of vector offset each other, resulting in the illusion of "normal." (2) ECG failed to capture abnormal changes caused by transient coronary occlusion due to spontaneous recanalization. (3) The blood supply range of LCX is primarily the lateral and posterior wall of the left ventricular in patients with non-dominant LCX occlusion, particularly in patients with RCA dominant type. As a result of the complete occlusion of the LCX, the ECG is insensitive to reflecting the ischemic area. Furthermore, in some distal or small occlusions, a complete occlusion results in only a small ischemia vector that does not meet the elevation-amplitude criteria. (4) The culprit vessel is usually severely stenotic, but other vessels provide adequate

collateral circulation(Freund et al., 2020). When the culprit vessel is suddenly blocked, its collateral circulation may be able to supply blood as quickly as before. (5) As described by Gorgels AP, lack of ST-segment changes may also be due to such a critical amount of transmurality of ischemia that neither subendocardial related ST depression occurs, nor ST elevation due to transmural ischemia(Gorgels, 2013). (6) The ST elevation area necessitates minimal amplitudes in specific leads, which increases the possibility that smaller injury vectors may be missed as non-ST elevation MI(Man et al., 2014). (7) The coronary is not occluded completely before primary PCI, and after a period, the coronary is completely occluded, as detected by CAG. Thus, ST elevation of ECG is missed, and reperfusion may attenuate ST-segment elevation, causing T-wave inversion. (8) For adults, the high-frequency response of an ECG machine is 100 Hz; however, on rare occasions, frequency components above 100 Hz caused by myocardial ischemia are filtered, and myocardial ischemia may be missed.

According to the above analysis, it is speculated that (1) In this case of acute extensive anterior wall myocardial infarction, ECGs were changed from normal before PCI (Figure 1a–d) to only T-wave inversion, with no apparent R wave attenuation (Figure 1e–f) in the anterior wall leads with D to B time of more than 6 h after revascularization. Angio-figures revealed that, due to fixed stenosis of the LAD, the area near the opening of the proximal LAD was acutely and completely occluded, with no collateral circulation. It is unlikely that the "transition" between ST depression to ST elevation continues for several hours. As a result, we concluded that the patient's basic ECG showed ST-segment depression and T-wave inversion in the anterior wall leads. Unfortunately, we could not get the basic ECG. Because of the persistent chest pain caused by continuous myocardium ischemia, the ECGs (Figure 1a–d) could be classified as "pseudonormalization." T-wave inversion in leads I, aVL, and V2-V6 (Figure 1e,f) may be associated with post-ischemic changes. (2) Based on the wrapping of the apex by the LAD, it was possible to deduce that the ST-segment elevation of leads II, III, and aVF (Figure 1c) was caused by the complete occlusion of the distal of the LAD. Why was it transient? Did the thrombus do dissolve by itself? The answer is that a spasm could cause distal LAD, RCA, or LCX due to increased sympathetic excitability following persistent chest pain. In any case, it was too far-fetched to prove.

In short, this patient has a precise diagnosis of acute anterior wall myocardial infarction based on symptoms, coronary angiography, myocardial enzyme test, and pathology. Nonetheless, no typical ECG changes were observed in the anterior wall leads, with only transient ST-segment elevation in the inferior wall leads. However, a perfect explanation is difficult to make. Else, Aslanger EK (Aslanger et al., 2021) reported whether STEMI is a transitional fossil in MI classification? STEMI criteria were not intended to diagnose acute coronary occlusion (ACO), and MI was diagnosed using CK-MB rather than ACO on angiography, so these criteria could not distinguish ACO from non-ACO. Based on evidence gathered over the last 20 years, there is room for significant improvement in the ECG diagnosis of ACO rather than relying on a single set of STEMI millimeter criteria that are inaccurate when used universally. Above all, distinguishing patients with ACO is critical in emergency cardiology. And this case demonstrated that (1) a basic ECG is required to provide baseline information for diagnosis and treatment; (2) The diagnosis of AMI cannot be ruled out even if the ECG is normal with no dynamic changes and when the typical symptoms persist. Furthermore, clinicians must improve their diagnostic skills and pay close attention to the use of ultrasound, biomarkers, computed tomography, and even angiography if clinical suspicion is high.

ACKNOWLEDGMENTS

The patients were informed for this case reported and provided informed consent.

CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

ETHICS STATEMENT

Ethics approval was not sought as this report contains 1 case report for which patient consent was obtained.

DATA AVAILABILITY STATEMENT

Data openly available in a public repository that issues datasets with DOIs

ORCID

Chunwei Liu https://orcid.org/0000-0003-1885-5114
Hongliang Cong https://orcid.org/0000-0002-9310-556X

REFERENCES

Aslanger, E. K., Meyers, P. H., & Smith, S. W. (2021). STEMI: A transitional fossil in MI classification? *Journal of Electrocardiology*, *65*, 163–169.

Freund A, Stiermaier T, de Waha-Thiele S, Eitel I, Schock S, Lurz P, Thiele H, Desch S. Coronary collaterals in patients with ST-elevation myocardial infarction presenting late after symptom onset. *Clinical Research in Cardiology* 2020;*109*(10):1307–1315.

Gorgels, A. P. (2013). ST-elevation and non-ST-elevation acute coronary syndromes: Should the guidelines be changed? *Journal of Electrocardiology*, *46*(4), 318–323.

Jung, C., & Elsässer, A. (2018). Update ESC-Leitlinie 2017 – Akuter Myokardinfarkt (STEMI) [Update ESC Guideline 2017 - Acute Myocardial Infarction (STEMI)]. *Dtsch Med Wochenschr*, *143*(11), 797–801 German.

Man, S., Rahmattulla, C., Maan, A. C., van der Putten, N. H. J. J., Dijk, W. A., van Zwet, E. W., van der Wall, E. E., Schalij, M. J., Gorgels, A. P., & Swenne, C. A. (2014). Acute coronary syndrome with a totally occluded culprit artery: Relation of the ST injury vector with ST-elevation and non-ST elevation ECGs. *Journal of Electrocardiology*, *47*(2), 183–190.

How to cite this article: Hu, Y., Jin, D., Liu, C., Zhang, R., Zhou, S., Wang, L., & Cong, H. (2022). Acute proximal left anterior descending coronary artery occlusion presenting with Normal ECG: A case report. *Annals of Noninvasive Electrocardiology*, *27*, e12987. https://doi.org/10.1111/anec.12987