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Case Report

Simultaneous left and right ventricular apical thrombi after occlusion of the wrapped left anterior descending artery

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ABSTRACT

Left ventricular (LV) thrombus formation is a well-known complication of acute myocardial infarction (AMI) and is reported to occur in 5–8% of patients who have anterior or apical myocardial infarction. However, right ventricular (RV) thrombus has not previously been reported after AMI. We describe a 54-year-old woman who presented with an ST-elevation myocardial infarction due to occlusion of the distal left anterior descending artery, which wrapped around the apex and led to simultaneous LV and RV apical thrombi.

<**Learning objective:** This case emphasizes the following: (1) in patients who have a long left anterior descending artery (LAD) that wraps around the apex, even distal LAD occlusion can cause a large infarct area including the apex, inferior wall, and right ventricular apex, as well as serious complications such as cardiac arrest and left ventricular and right ventricular (RV) thrombus. (2) Cardiac magnetic resonance imaging is useful for detecting apical thrombus especially in the RV.>

Introduction

Since the left anterior descending artery (LAD) supplies blood to a large portion of the myocardium, occlusion of its proximal portion is usually associated with adverse outcomes and complications [1,2]. However, occlusion of the distal LAD can also lead to serious complications if the LAD is long enough to curve around the apex [3]. One such complication is left ventricular (LV) thrombus formation that often accompanies acute myocardial infarction (AMI) [4], but right ventricular (RV) thrombus has not previously been reported. Here, we present a case of simultaneous LV and RV apical thrombi confirmed by cardiac magnetic resonance imaging (MRI) after acute occlusion of the distal LAD.

Case report

A 54-year-old woman was brought to the emergency room with severe ongoing chest pain that had developed 30 min previously. Although she had a history of hypertension, she had not been taking any medication. Soon after admission, she suddenly had a seizure and collapsed and ventricular fibrillation was documented on electrocardiography (ECG) monitoring. Direct current defibrillation was performed twice and initial 12-lead ECG after resuscitation showed ST-segment elevation in leads V3 to 5, II, III, and aVF (Fig. 1A), suggesting both anterior and inferior wall myocardial infarction. After tracheal intubation due to her altered mentality and severe pulmonary edema, emergent coronary angiography (CAG) was performed, revealing total occlusion of the distal LAD (Fig. 1B). There were no luminal stenoses in the left circumflex or right coronary arteries, and the right coronary artery was small with a diminutive posterior descending artery and posterolateral branch (Fig. 1C). The lesion was crossed with Runthrough guidewire (Terumo, Tokyo, Japan) and predilated with a 2.0 × 15-mm Nimbus Pico (ClearStream, Tempe, AZ, USA) balloon (Fig. 1D). After that, we successfully implanted a drug eluting stent (2.5 × 18 mm, Xience Alpine, Abbott, Abbott Park, IL, USA) in the distal LAD, and Thrombosis In Myocardial Infarction (TIMI) flow grade 3 was achieved. Final angiography showed that the distal

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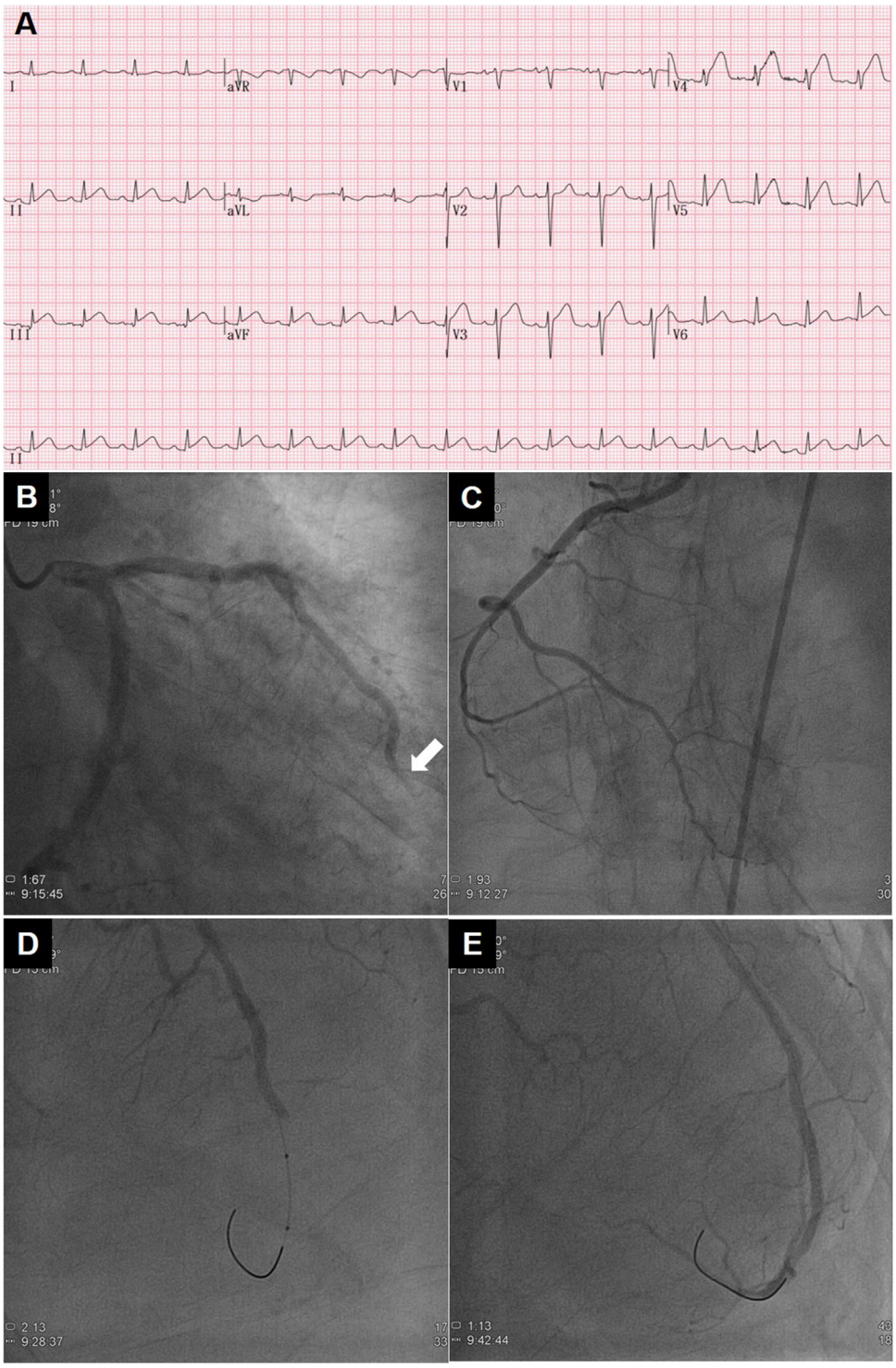

Fig. 1. 12-lead electrocardiography (ECG), initial coronary angiography (CAG), and primary percutaneous coronary intervention. (A) ECG showed ST-segment elevation of leads V3 to 5, II, III, and aVF. (B) Left CAG showed total occlusion of the distal left anterior descending artery (white arrow). (C) The right coronary artery was short, non-dominant, and tapers by supplying the right ventricular branches. (D) Predilatation using a 2.0 × 15-mm balloon was performed at the distal left anterior descending artery (LAD). (E) Coronary angiography after successful stent implantation revealed that the distal LAD was wrapped around the apex and long enough to supply the inferior wall of the apex.

LAD was wrapping around the apex and was long enough to supply the inferior wall of the apex and inferior septum (Fig. 1E). The echocardiography showed mild to moderate LV systolic dysfunction (ejection fraction: 40%) with akinesia of the apex. To determine myocardial viability of the infarct related area, cardiac MRI was performed on day 15 and subendocardial delayed enhancement of the LV apex, inferior septum, inferior wall, and RV apex was observed. Interestingly, the apical thrombi were simultaneously detected in the LV and RV apex (Fig. 2A–C). Follow-up echocardiography was performed on the same day, but we could only observe a 1.7 × 0.23-cm sized thrombus at the LV apex but not at the RV apex (Data Supplement Movie 1). At first, the

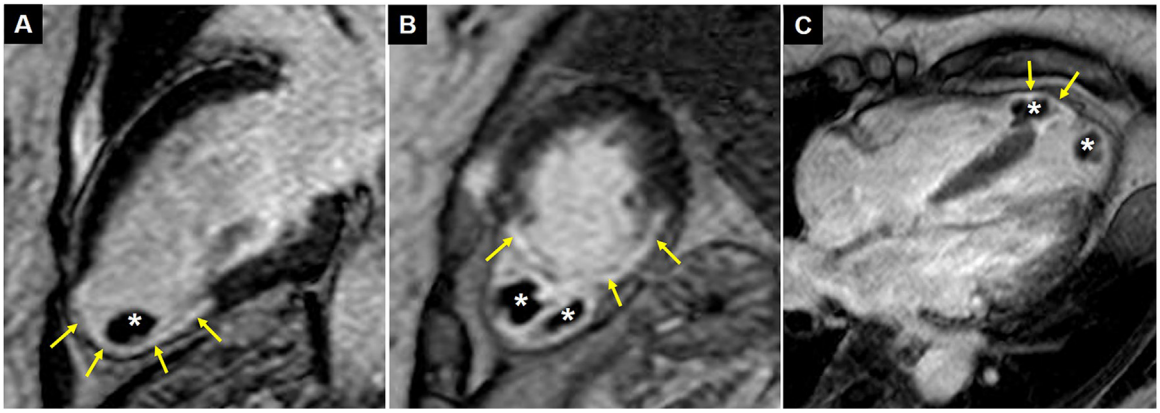

Fig. 2. Cardiac magnetic resonance imaging showing simultaneous left and right ventricular apical thrombi. (A) Delayed hyperenhancement of the apex and inferior wall (yellow arrows) and left ventricular (LV) apical thrombus (asterisk) were observed. (B) Delayed hyperenhancement was seen at the inferior septum and inferior wall of the apex (yellow arrows) and right ventricular (RV) apical thrombi (asterisks) were also detected. (C) A four-chamber view showed a delayed enhancement of the RV apex (yellow arrows) and simultaneous LV and RV apical thrombi (asterisks).

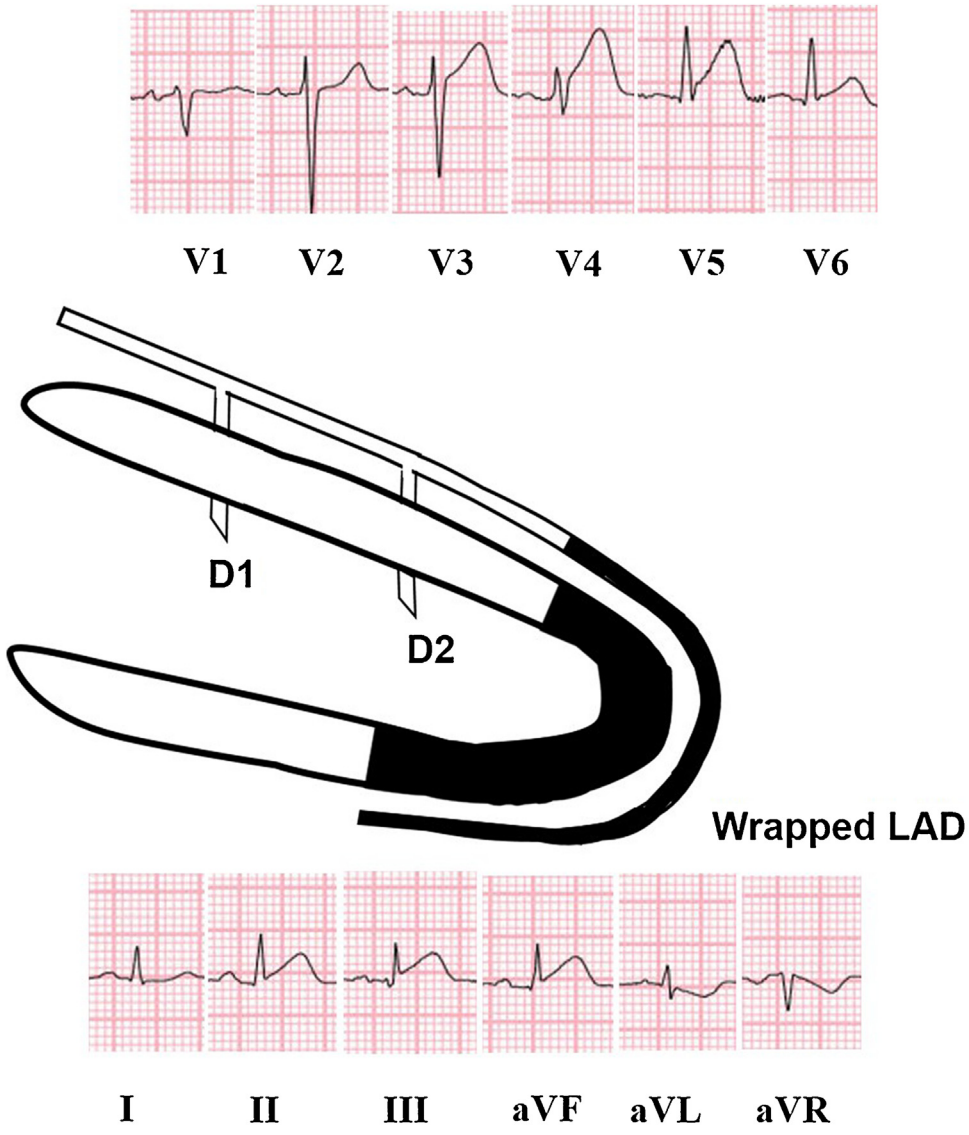

Fig. 3. Schematic demonstration of the infarction area following occlusion of the distal wrapped LAD and electrocardiographic changes in our patient. LAD, left anterior descending artery; D1, the first diagonal branch; D2, the second diagonal branch.

patient was given aspirin and ticagrelor for antiplatelet therapy after primary percutaneous coronary intervention. When we detected LV and RV thrombi on hospital day 15, we added warfarin and changed the dual antiplatelet therapy regimen to aspirin and clopidogrel. With concurrent use of warfarin, aspirin, and clopidogrel, the patient was discharged without any complications.

Discussion

Isolated acute inferior wall myocardial infarction on ECG despite LAD occlusion has rarely been reported [5]. Previous work reported that simultaneous ST-segment elevation in the precordial and inferior leads can occur due to occlusion of a variant LAD that wraps around the cardiac apex to supply both the anterior and inferior wall of the LV [6]. Sasaki et al. reported the ST changes of the inferior leads in patients with LAD infarction according to LAD length and the site of occlusion [7]. They summarized that if the patient has a wrapped LAD and the location of the occlusion is proximal to the first diagonal branch, the ST-segment is elevated in anterior leads and remains isoelectric in the inferior leads. However, if the patient has a wrapped LAD and the location of the occlusion is distal to the first diagonal branch, the ST-segment is elevated in the anterior and inferior leads simultaneously. Our case corresponded with the latter condition and might result in mainly elevated ST-segment of V4 to 5 as well as inferior leads (II, III, and aVF) due to the infarction of the apical aspect and inferior wall (Fig. 3). This was confirmed by cardiac MRI, which showed an infarct area of anterior aspect of the apex, inferior wall, and inferoseptum of the mid LV, and even the RV apex wall. Another interesting finding following cardiac MRI was simultaneous LV and RV apical thrombi. LV thrombus formation is a well-known complication after AMI, most frequently in anterior or apical myocardial infarction, with a reported incidence rate of 5–8% [4]. However, RV thrombus after LAD occlusion has not been reported previously. Although RV thrombi were clearly visualized on the cardiac MRI, they were not observed in the echocardiography performed on the same day due to the limited echo window of the RV apex. Although echocardiography is the most often used tool for assessing LV thrombus, 10–46% of echocardiograms are inconclusive and delayed enhancement cardiac MRI is nowadays considered the gold standard [4]. Detection of an RV thrombus would be more difficult because the RV apex is more often hard to clearly define with echocardiography. The present case emphasizes the essential role of cardiac MRI for assessing RV apical thrombus as well as LV thrombus.

Kobayashi et al. reported that the feature of LAD wrapping around the LV apex is related to a larger infarct size and adverse clinical outcomes in patients with anterior AMI [8,9]. Ilia et al. also reported that longer LAD is a strong predictor of poor prognosis in patients undergoing percutaneous coronary intervention at the proximal LAD due to AMI [3]. Consistent with prior reports, although the culprit lesion was the distal LAD, our patient experienced severe complications such as cardiac arrest and apical thrombi because the distal LAD wrapped around the LV and RV apex and supplied the inferior wall. This resulted in a larger than expected infarct area. To conclude, we believe we are the first to report an RV apical thrombus after AMI and have demonstrated the usefulness of cardiac MRI for detecting RV apical thrombi.

Conflict of interest

The authors declare no conflict of interest.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.jccase.2018.12.015.

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