

[CASE REPORT]

Recent Inferior Myocardial Infarction Complicated with a Right Ventricular Thrombus Detected by Three Cardiac Imaging Modalities

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Abstract:

We report the case of a 71-year-old woman diagnosed with recent inferior myocardial infarction complicated with right ventricular infarction and a right ventricular thrombus. Three-dimensional transthoracic echocardiography, contrast-enhanced computed tomography, and cardiac magnetic resonance imaging clearly detected a thrombus. We consider cases with a recent right ventricular infarction to require assessment for thrombus formations in the right ventricle. Fortunately, vigorous anticoagulation therapy resolved the thrombi in both the right ventricle and right coronary artery.

Key words: inferior myocardial infarction, right ventricular infarction, right ventricular thrombus, three-dimensional transthoracic echocardiography

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Introduction

Right ventricle (RV) thrombus formation is uncommon in cases of RV infarction, and the diagnosis is sometimes challenging (1-3). We report the case of a 71-year-old woman diagnosed with recent inferior myocardial infarction complicated with RV infarction and an RV thrombus. Three-dimensional transthoracic echocardiography, contrast-enhanced computed tomography (CT), and cardiac magnetic resonance imaging (MRI) clearly detected a thrombus.

Case Report

A 71-year-old woman had complained of epigastric pain. Her doctor diagnosed her with gastroesophageal reflux disease and prescribed a proton pump inhibitor for one week. During that period, her symptoms worsened, and she had nausea and appetite loss. She was admitted to the hospital, where an electrocardiogram revealed evidence of an inferior myocardial infarction. She was then transferred to our hospi-

tal with a diagnosis of recent inferior myocardial infarction. She had a history of dyslipidemia and denied any smoking history. Intravenous fluid improved her initial blood pressure from 70/55 mmHg to 110/90 mmHg. An electrocardiogram showed inferior myocardial infarction without ST elevation (Fig. 1A). Her lactate dehydrogenase level was 575 U/L, creatine phosphokinase was 257 U/L, troponin-T was 4.5 ng/mL, and B-type natriuretic peptide was 509 pg/mL. Transthoracic echocardiography revealed a severely reduced systolic function of the inferior wall of the left ventricle (LV) and RV, and three-dimensional transthoracic echocardiography clearly detected a thrombus in the RV (Fig. 1B, Supplementary material 1-4). Contrast-enhanced CT also detected the thrombus in the RV (Fig. 1C).

We stabilized her condition with intravenous fluid, antiplatelet and anticoagulation therapy (intravenous unfractionated heparin, then warfarin), an angiotensin-converting enzyme inhibitor, and spironolactone for one week. We then performed coronary angiography, which revealed thrombotic occlusion in the proximal segment of the right coronary artery (RCA) and a distal segment supplied by collateral ves-

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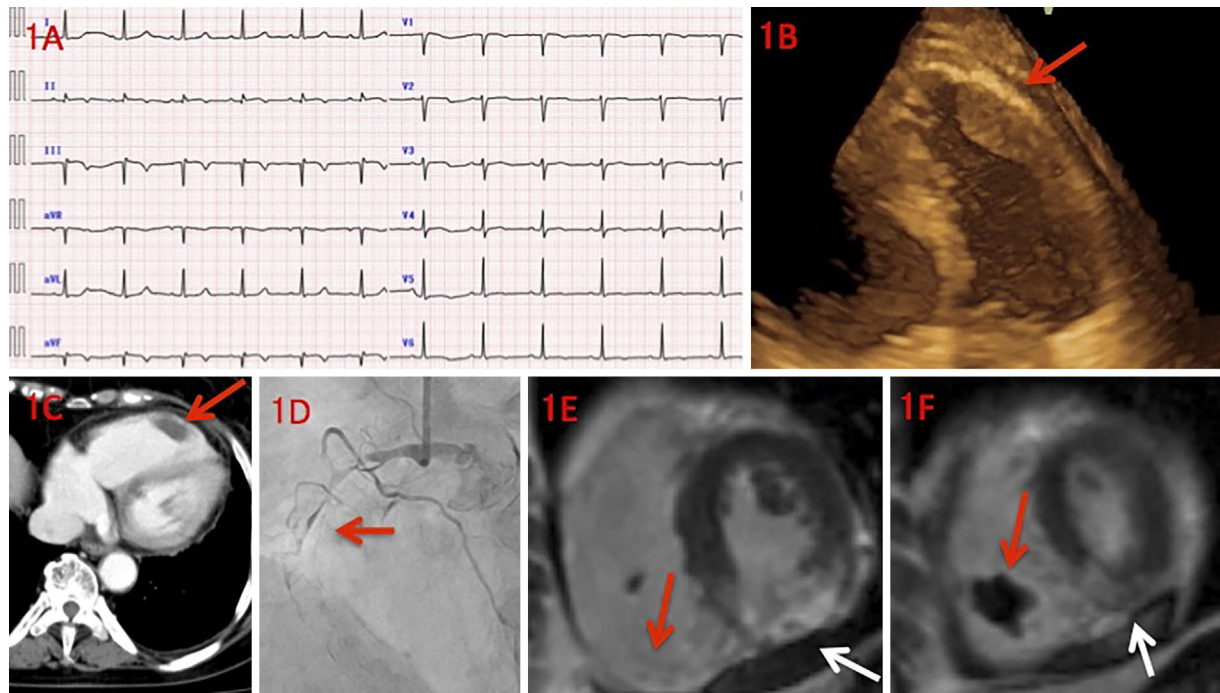


Figure 1. A: Electrocardiogram shows an abnormal Q wave and inverted T wave in leads III and aVF. B: Three-dimensional transthoracic echocardiography shows a thrombus (16×34 mm; red arrow) in the RV on admission. C: Contrast-enhanced computed tomography shows a thrombus (red arrow) in the RV. D: Coronary angiography shows occlusion in the proximal segment (red arrow) of the right coronary artery (left anterior oblique view). E: Cardiac magnetic resonance imaging (late gadolinium enhancement) shows delayed enhancement of the inferior wall of the LV (white arrow) and almost all parts of the RV (red arrow). RV end-diastolic volume, 121 mL; RV end-systolic volume, 93 mL; RV ejection fraction, 23%. F: Cardiac magnetic resonance imaging (late gadolinium enhancement) shows delayed enhancement of the inferior wall of the LV (white arrow) and a thrombus in the RV (red arrow). RV: right ventricle, LV: left ventricle

sels from left coronary arteries (Fig. 1D, Supplementary material 5). Subsequent cardiac MRI showed the thrombus in the RV and late gadolinium enhancement in the inferior LV and RV (Fig. 1E and F). Stress myocardial perfusion ²⁰¹thallium scintigraphy showed no redistribution at 4 hours but partial redistribution at 24 hours in the inferior wall of the LV (Fig. 2A). Since myocardial perfusion scintigraphy demonstrated partial viability of the inferior wall of the LV, we performed coronary CT angiography one week after coronary angiography for revascularization and observed recanalization of the RCA (Fig. 2B). We then performed a second coronary angiogram, which also revealed recanalization of the RCA, with tiny thrombi (Fig. 2C, Supplementary material 6). At a two-month follow-up, the patient remained free from heart failure and angina symptoms. Follow-up echocardiography showed the disappearance of the thrombus in the RV and a slight improvement in the RV systolic function (Fig. 2D, Supplementary material 7-9).

Discussion

This was a case of inferior myocardial infarction complicated by a thrombus in the RV. The delayed presentation of RV myocardial infarction affects thrombus formation (2, 3) because primary percutaneous coronary intervention can im-

prove the RV wall motion (4), whereas delayed reperfusion can lead to thrombus formation (5). Cases with a recent RV infarction require assessment for thrombus formations in the RV. Furthermore, because anticoagulation prevents thrombus formation, the administration of medical therapy without anticoagulation promotes thrombus formation (1). Although a few cases of RV infarction with thrombus formation have been reported (1-3), all thrombi were located in the apex of the RV, as was observed in our case. Therefore, we believe that RV thrombus formation occurs due to RV apex infarction, especially in the subacute phase or in cases without the administration of anticoagulation therapy. Because the RCA or left anterior descending artery supply the RV apex in most cases, these two arteries are likely to be responsible for RV thrombus formation (3, 6).

Although transthoracic echocardiography, cardiac MRI, and CT were able to detect an RV thrombus, these modalities have their respective advantages and disadvantages. While transthoracic echocardiography might be able to detect a thrombus in the RV in the emergency room at the time of admission, routine long axis-, short axis-, and four-chamber views might miss a thrombus in the RV, as was observed in our case. Contrast-enhanced CT can clearly detect a thrombus in the RV and also identify pulmonary embolism, if embolized (3), in the emergency room, sometimes

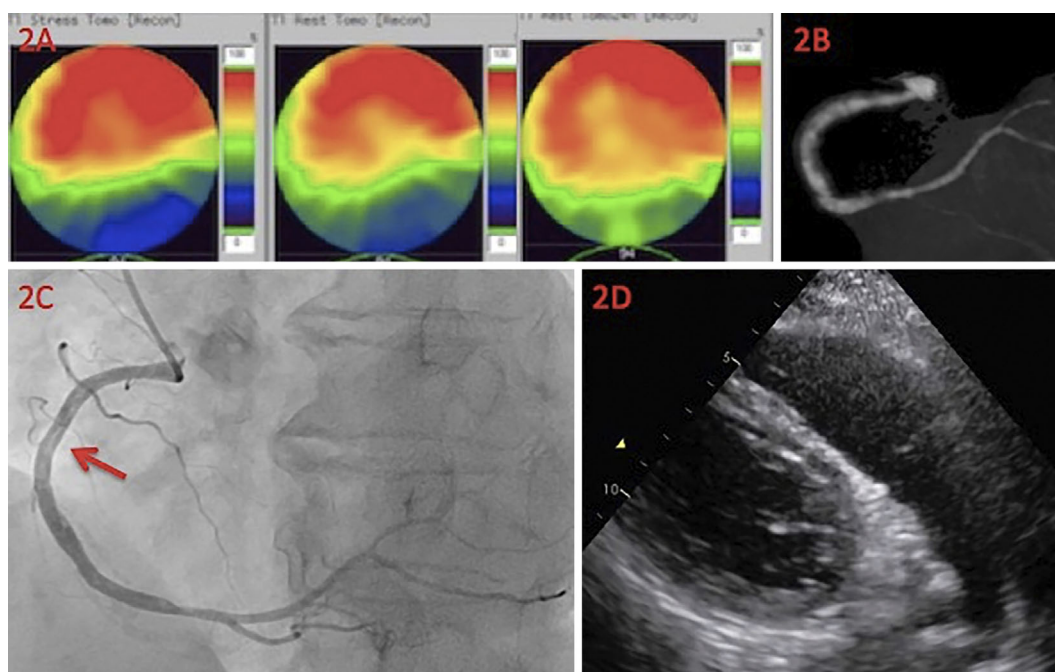


Figure 2. A: Stress myocardial perfusion ^{201}Tl scintigraphy (intravenous dipyridamole); left panel: an image immediately after the stress test shows the absence of any ^{201}Tl uptake in the inferior wall; middle panel: an image 4 h after the stress test shows the absence of redistribution of ^{201}Tl in the inferior wall; right panel: an image 24 h after the stress test shows the partial redistribution of ^{201}Tl in the inferior wall. B: Coronary computed tomography angiography shows recanalization of the right coronary artery. C: Coronary angiography shows recanalization with tiny thrombi (red arrow) in the proximal segment of the right coronary artery (left anterior oblique view). D: Transthoracic echocardiography shows disappearance of the thrombus in the RV at 2-month follow-up. RV: right ventricle, LV: left ventricle

coincidentally, but plain CT might not detect a thrombus. Transthoracic echocardiography therefore needs to be performed prior to CT in order to visualize a thrombus in the RV. Cardiac MRI performed to investigate myocardial infarction scars may incidentally detect a thrombus in the RV, but the procedure cannot be performed in a critical setting, such as in patients with cardiogenic shock.

We encountered a case in which a large RV thrombus was detected using three-dimensional transthoracic echocardiography, cardiac MRI, and contrast-enhanced CT. Fortunately, vigorous anticoagulation therapy resolved the thrombi in both the RV and RCA.

Conclusion

We encountered a case in which a large RV thrombus was detected using three-dimensional transthoracic echocardiography, cardiac MRI, and contrast-enhanced CT.

The authors state that they have no Conflict of Interest (COI).

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