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A Case of Unrecognized Myocardial Infarction With Left Ventricular Thrombus in a Young Patient

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Left ventricular thrombus（LVT）is a possible complication, especially in patients who experience acute myocardial infarction（MI）, even after revascularization. We encountered a case of unrecognized MI an LVT in a 38-year-old man, which was discovered incidentally on electrocardiogram. The patient experienced no symptoms, such as chest pain or dyspnea, and had no relevant medical history or regular medication use. However, he did have a history of smoking（15 cigarettes daily）, and his father had MI. The patient was obese and his low-density lipoprotein cholesterol levels were high（170 mg/dL）. In addition to pharmacotherapy for secondary prevention of MI, anticoagulation therapy with warfarin was started, and percutaneous coronary intervention of the left anterior descending artery was successful. Management of cardiovascular risk is important even at a young age.

Key words: myocardial infarction, risk factors, anticoagulation therapy, young adults

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INTRODUCTION

Left ventricular thrombus（LVT）is a possible complication, especially in patients who experience acute myocardial infarction（MI）, even after revascularization［1］. Before thrombolytic therapy and reperfusion as primary interventions, the incidence of LVT associated with MI was estimated to be 20%-40%. Although the rate is currently significantly lower, at least 5% of patients develop LVT after acute MI［2, 3］. In addition, the prevalence of unrecognized MI can be as high as 19%-44%, with a prognosis very similar to or worse than that of recognized MI. Although most observational investigations are expected to detect unrecognized MI on electrocardiogram（ECG）, the problem is that abnormal Q waves are not always present［4］. When LV wall motion decreases, not only after MI, but also as a result of dilated cardiomyopathy or similar conditions, blood flow disorders and congestion occur in the region, thus increasing the possibility of thrombus formation［5］. Anticoagulation with warfarin reduces the risk of both LVT and subsequent thromboembolism but increases the risk of bleeding［1］. We report a case of unrecognized MI with LVT in a young patient.

CASE REPORT

An ECG abnormality was observed in a 38-year-old man during a medical examination. The patient experienced no symptoms, such as chest pain or dyspnea, and had no relevant medical history or regular medication use. However, he did have a history of opportunistic alcohol consumption and smoking（15 cigarettes daily）, and his father had MI in his 50s. The patient was obese, with a height of

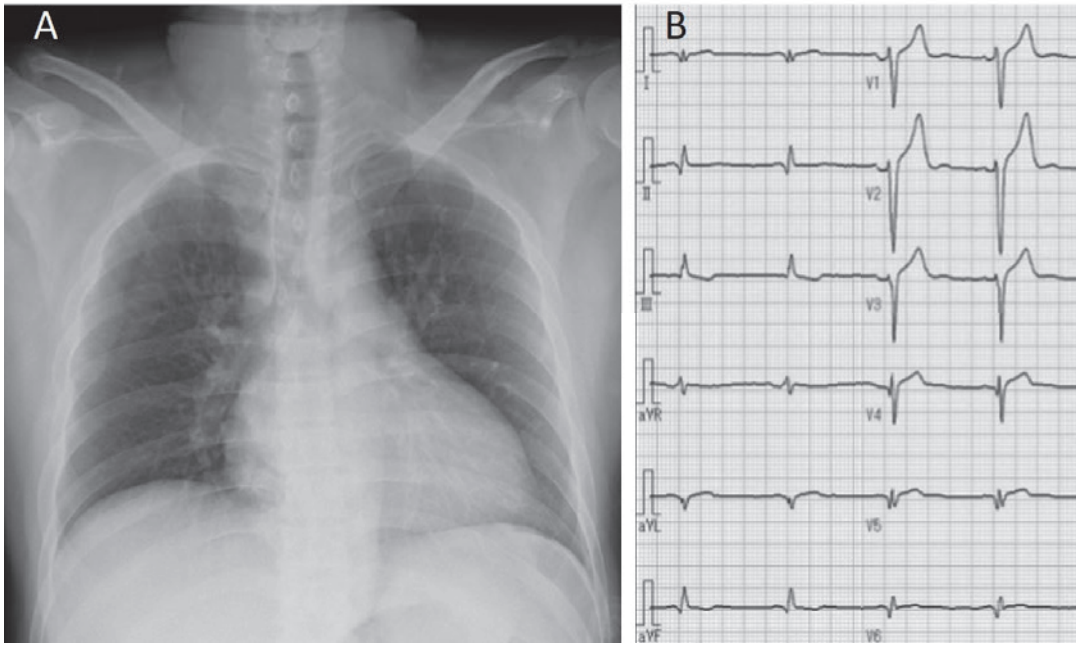

Fig. 1. Chest radiography shows a cardiothoracic ratio of 58%, indicating cardiomegaly, but no congestion or pleural effusion（A）. A 12-lead ECG reveals abnormal Q waves in leads I, II, aVF, and V4 -V6 and a poor R wave in V3（B）.

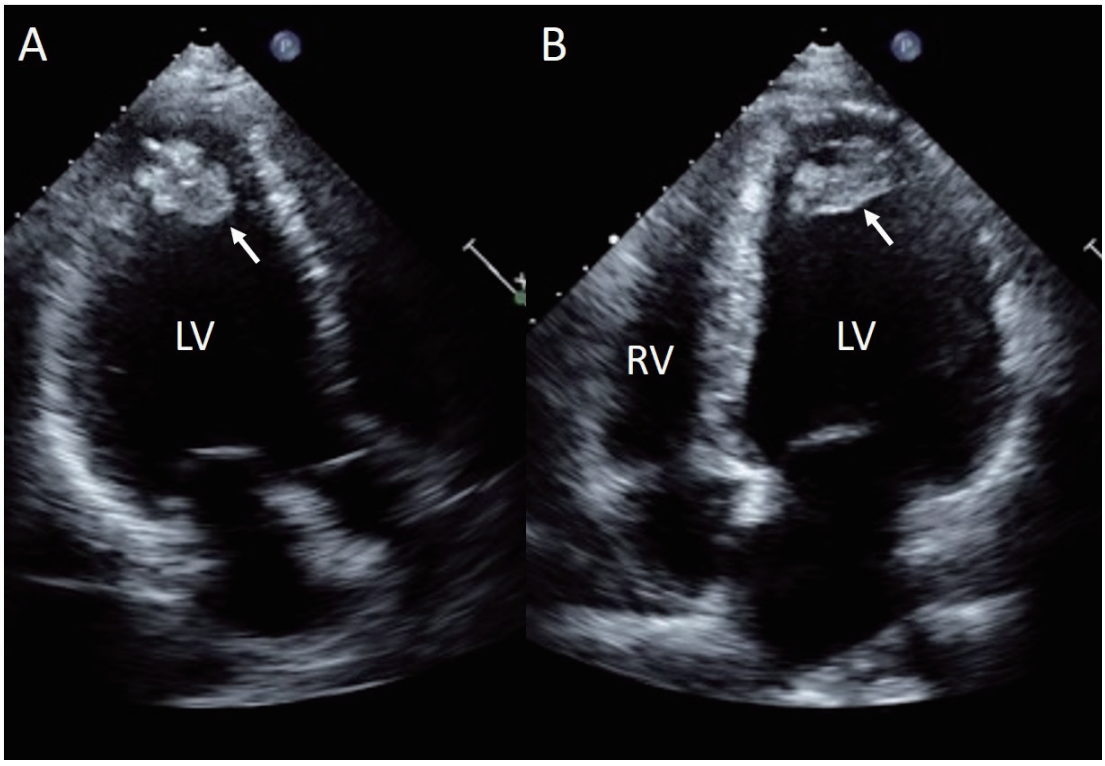

Fig. 2. Apical long axis（A）and 4-chamber（B）views by transthoracic echocardiography show a left ventricular thrombus at the LV apex（arrow）. LV, left ventricle; RV, right ventricle.

Fig. 3. Myocardial perfusion imaging with technetium-99m tetrofosmin at rest and under adenosine stress demonstrate the myocardial ischemia in the left anterior descending artery and diagonal artery regions (arrow heads).

175 cm and weight of 86.4 kg, corresponding to a body mass index of 28.2 kg/m^2. On examination, the patient's body temperature was 36.6°C, with a heart rate of 84 beats/min, a blood pressure of 140/90 mmHg, a respiratory rate of 16 breaths/min, and oxygen saturation of 95% on ambient air. The palpebral conjunctiva was not pale, and the jugular vein was not enlarged. No murmur was heard, and respiratory sounds were clear. Peripheral coldness in the limbs and lower leg edema were not observed. Chest radiography revealed a cardiothoracic ratio of 58%, indicating cardiomegaly, but no congestion or pleural effusion (Fig. 1A). A 12-lead ECG revealed abnormal Q waves in leads I, II, aVF, and V4 -V6 and a poor R wave in V3 (Fig. 1B). Laboratory investigations revealed a mildly elevated creatine kinase level (329 U/L), with no elevation of troponin (0.02 ng/mL), and a slight elevation of brain natriuretic peptide (75 pg/mL). Low-density lipoprotein (LDL) cholesterol levels were high (170 mg/dL) and a hemoglobin A1c level was 6.0%. Renal function was normal with a creatinine level of 0.93 mg/dL, and estimated glomerular filtration rate of 73.9 mL/min/BSA. Transthoracic echocardiography (TTE) revealed an LV ejection fraction of 26%, and wall motion of the LV apex was akinetic, with no valvular abnormalities. A mass, 40 × 20 mm in size was found at the LV apex (Fig. 2), and a high-intensity area was found inside the mass. Although the mass was not mobile, there was a slightly mobile area around it.

The patient was admitted and anticoagulation therapy with intravenous heparin was initiated. Contrast-enhanced computed tomography (CT) revealed a mass with calcification in the apex of the heart, similar to TTE, and there were heterogeneous atrophy and poorly stained areas in both kidneys were present, suggesting renal infarction. Myocardial perfusion imaging with technetium-99m tetrofosmin at rest and under adenosine stress demonstrated reversal of the myocardial ischemia in the left anterior descending (LAD) artery and diagonal artery regions (Fig. 3). Coronary angiography (CAG) revealed complete occlusion of LAD segment 7 and collateral flow from the right coronary artery to the LAD segment via the epicardial and septal branches (Fig. 4A, B). Magnetic resonance imaging (MRI) of the head revealed a very small spot of the new cerebral infarction position of high signal on the fluid-attenuated inversion recovery sequence. Although the patient was young and asymptomatic, he was at a risk of cardiovascular disease, given his history of smoking, findings of hypertension, and dyslipidemia, and family history. Based on CAG findings, it was

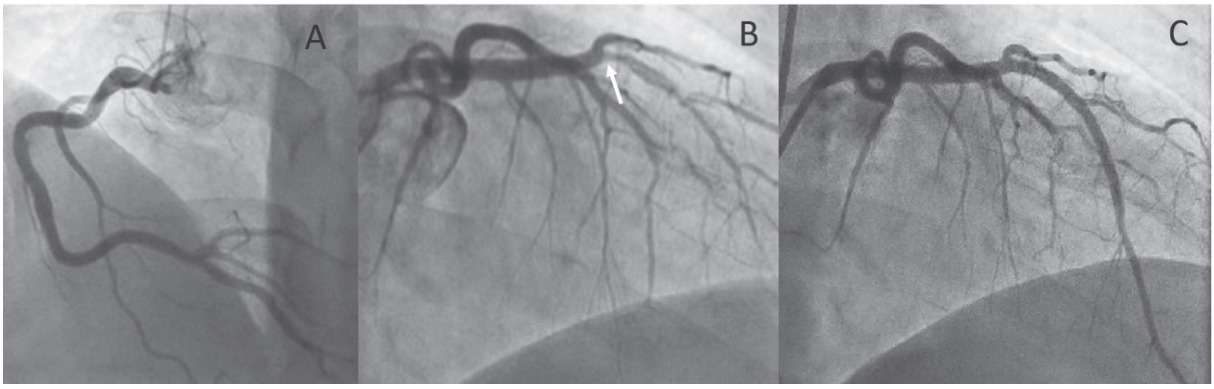

Fig. 4. Coronary angiography reveals no significant stenosis of the right coronary artery (A) and complete occlusion of the left anterior descending artery (LAD) (B, arrow). Percutaneous coronary intervention of the LAD is successful (C).

considered that LVT occurred in association with decreased LV contractility and old MI. In addition to pharmacotherapy for secondary prevention of MI, anticoagulation therapy with warfarin was continued, and percutaneous coronary intervention (PCI) of the LAD using the drug-eluting stent was successful (Fig. 4C). Medication included enalapril, carvedilol, atorvastatin, ezetimibe, aspirin, clopidogrel and warfarin. LDL cholesterol levels improved from 170 to 66 mg/dL. Five months after PCI, the patient's LV ejection fraction was increased, from 26% to 53%, and the LVT decreased in size from 40 × 20 mm to 33 × 15 mm.

DISCUSSION

As with all thrombus formation, Virchow triad (hypercoagulability, endothelial injury, and blood stasis) plays an important role in LVT formation after MI. The stimulating event and myocardial substrate may modulate risk of LVT formation beyond LV dysfunction alone. Subendothelial tissue injury causes inflammation and collagen exposure, serving as a nidus for platelet aggregation and activation of the clotting cascade. Indeed, persistent hypercoagulable states are observed for up to 6 months after MI [6]. Of 1,035,888 patients hospitalized in the United States with ST-elevation MI (STEMI) between 2003 and 2013, 0.2% developed acute in-hospital LVT [7]. Factors associated with LVT included anterior/anterolateral STEMI, acute or chronic heart failure with reduced ejection fraction, atrial fibrillation, LV aneurysm, left heart valvular disease, acute or chronic deep venous thrombosis/pulmonary embolism and alcohol abuse. Patients with LVT were less likely to be woman [7]. LVT carries a risk of systemic embolism, not only in the acute phase, but also in the chronic phase, and significantly increases patient social burden and deterioration of prognosis [8]. The size of the thrombus is usually not a problem; however, mobile thrombus, the presence of adjacent hyperkinesia, and thrombus protrusion are believed to be significant contributors to progression to systemic embolism [9]. For LVT, once diagnosed, oral anticoagulant therapy should be considered for up to 6 months, guided by repeat echocardiography and consideration of the bleeding risk and the need for concomitant antiplatelet therapy [10]. However, surgical thrombectomy should be considered in patients with hemodynamic compromise due to thrombus, recurrent systemic embolism despite anticoagulant therapy, or a mobile thrombus, history of embolism, or potential for improvement in cardiac function. It is well-documented that ventriculotomy is usually associated with an increased incidence of cardiac arrhythmias and decreased ejection fraction [11]. Despite the presence of cerebral infarction and renal infarction in our patient, he was asymptomatic, and mobility of the thrombus was minimal. The risk of symptomatic cerebral infarction with surgical intervention was greater; therefore, we decided to continue oral anticoagulant therapy. With a lack of clear randomized clinical trials data and great variability in the presentation and associated

complications of LV thrombus, individualized approaches will continue to be necessary.

Risk factors for cardiovascular disease, such as hypertension, dyslipidemia, diabetes and smoking, have also been identified in younger patients. Studies focusing on younger patients have examined the role of additional risk factors such as illicit drug use and coagulation factor dysfunction; however, few studies have addressed MI in patients ≤ 40 years of age. Despite significant progress in primary prevention, the rate of MI has not decreased in young adults [12-14]. Yang et al. compared patients with acute MI aged < 40 years and those aged 41-50 years, and reported similar 1-year and long-term outcomes despite a lower prevalence of hypertension and an average age of 10 years younger [15]. In this patient, secondary prevention of MI is critically important. Key lifestyle interventions include cessation of smoking, optimal blood pressure control, diet advice and weight control, and encouraging physical activity.

CONCLUSION

We encountered an individual of unrecognized MI with LVT at a young age, which was discovered incidentally on ECG, and treated with medication and percutaneous coronary intervention. Management of cardiovascular risk is important even at a young age.

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Research involving human participants and/or animals: In this article, studies of human and animal participants are not included.

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