Hunting the Hidden: Surgical Treatment of Chronic Silent Thrombus in the Left Ventricle. A Case Report.

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Case report

Keywords: Left ventricular thrombus, Systemic embolism, Myocardial bridging, Surgical treatment, Case report

Posted Date: November 8th, 2021

DOI: https://doi.org/10.21203/rs.3.rs-1009598/v1

Abstract

Background: Silent left ventricular thrombus is dangerous. The current standard anticoagulation therapy is not effective, and the outcomes are frustrated.

Case presentation: A 33-year-old man with silent left ventricular thrombus, which was detected incidentally by transthoracic echocardiography. After admission, anticoagulation with low-molecular-weight heparin therapy was carried out. Unfortunately, acute left temporal embolism emerged 5 days later, then the patient was transferred to the neurology department for further treatment. One month later, the patient received coronary artery bypass grafting (CABG), ventricular aneurysm resection and left ventricular thrombectomy and was discharged uneventfully after surgery.

Conclusions: For the patients with giant or hypermobile left ventricular thrombus or recurrent systemic emboli, surgical treatment should be a priority.

Background

Left ventricular thrombus (LVT) is uncommonly seen in unselected patients by echocardiogram, it is, however, more commonly found in patients with heart failure and acute myocardial infarction[1]. Silent LVT detected by transthoracic echocardiography (TTE) is rare and dangerous in those with normal cardiac function without any history of cardiac diseases. The current standard treatment strategy for LVT is anticoagulation therapy, including vitamin K antagonist (VKA), direct oral anticoagulants, low molecular heparin, and intravenous unfractionated heparin[2, 3]. Nevertheless, surgical intervention should be considered if systemic embolism emerged[2].

Case Presentation

A 33-year-old man was referred to the cardiac surgery department due to left ventricular thrombus, which was detected incidentally by TTE during routine preoperative examination for urinary calculi. In the outpatient department, the patient's blood pressure, heart rate, and percutaneous oxygen saturation were all within normal range, without any signs of acute or chronic cardiac failure. This patient, however, had a more than 10-year history of drinking and smoking.

Electrocardiogram (ECG) demonstrated sinus rhythm with abnormal Q-wave in the inferior wall (Figure 1). TTE showed apical space-occupying lesions (2*1.5cm) and mild tricuspid regurgitation. Ejection fraction (EF) was 63%. Coronary angiography (CAG) revealed 30% stenosis in the middle of left anterior descending branch, 30% stenosis in the distal left circumflex branch, and myocardial bridging (MB) in the middle of right coronary artery (RCA) with 70% systolic stenosis (Figure 2). Tc-99m myocardial perfusion scintigraphy (resting and activity state) demonstrated that severe myocardial hypoperfusion and hypokinesis in apex of left ventricular (LV), abnormal perfusion cardiac muscle accounting for 19% of total left ventricular myocardium, and only 4% of them were hibernating. Cardiac magnetic resonance

imaging (MRI) with delayed enhancement confirmed the left ventricular aneurysm formation after myocardial infarction with apical thrombus (Figure 3).

During Hospitalization, this patient presented with stroke symptoms during anticoagulation with low-molecular-weight heparin therapy. MRI revealed acute left temporal embolism. Then, he was transferred to the neurology department for further treatment. One month later, he accepted Coronary Artery Bypass Grafting (CABG), ventricular aneurysm resection and left ventricular thrombectomy under cardiopulmonary bypass. Seven days later, the patient was discharged uneventfully with warfarin and aspirin therapy for 1 year.

Discussion And Conclusions

LVT is most commonly found in myocardial infraction, low ejection fraction, LV aneurysms, and ventricular wall akinesis or dyskinesis[4, 5]. The thrombus formation refers to Virchow's triad: blood stasis, endothelial injury, and hypercoagulability[6]. McCarthy et al. reported that in unselected patients, the incidence of LVT detected by TTE is 0.1%. Most LV thrombi are formed within 2 weeks. However, some occur even more later, especially in the patients with LV systolic dysfunction[6]. Cullen JG et al. reported that systemic embolism was found in patients even with calcified thrombi[7].

Post-myocardial infraction has been demonstrated as the most common risk factor for the development of LVT in the previous studies, and heart failure, alcohol abuse, and tobacco use has also been documented[1, 8]. In this case, the patient has a history of drinking and smoking for over 10 years. Approximately two years ago, the patient was completely drunk and transferred to the emergency room for rescuing care. CAG showed myocardial bridge in middle of RCA with 70% systolic stenosis, which was in concordance with the ECG examination. MRI and myocardial perfusion scintigraphy demonstrated left ventricular aneurysm with apical thrombus. Montone RA et al. reported that coronary spasm with MB is the independent risk factor of myocardial infarction and non-obstructive coronary arteries[9]. In this case, we speculated that MB in RCA, alcohol and tobacco abuse contributed to myocardial infraction. Essential thrombocytopenia (ET) is another important incentive that we should take into account, which have been demonstrated to be responsible for the onset of acute myocardial infarction[10, 11]. In this case, the patient's complete blood cell count showed platelet count was $435x10^9$/L. Further test was carried out, gene mutation of JAK2 V617F, JAK2 exon 12, MPL, CALR was not detected in this patient, which have ruled out the possibility of ET.

LVT remains a severe complication associated with a high risk of systemic embolism. According to the latest guidelines[12, 13], several anticoagulation therapies are introduced. The current standard therapy for LVT is chronic warfarin therapy for at least 3months. Recently, direct oral anticoagulants (DOACs) are introduced[3, 14]. The therapeutic dilemmas are: which one is the best? How long the treatment course should take? What's the dose? Several studies have suggested that even following strict anticoagulant treatment, the prognosis of patients is frustrated [15, 16]. Lattuca et al. demonstrated that prolonged

anticoagulation therapy duration could reduce the occurrence of major adverse cardiovascular events, but the bleeding complications raised[15].

Surgical treatment is another considerable option for patients with LVT, especially for those with giant or hypermobile LVT or recurrent systemic emboli developed undergoing anticoagulant therapy[2, 17]. Lee et al. reported that the rate of post-treatment thromboembolism in operative treatment group is less than anticoagulation and antiplatelet group[18].

Although surgical intervention has some intrinsic risks, patients would benefit from it. We highlight that for the patients with giant or hypermobile LVT or recurrent systemic emboli, surgical treatment should be a priority.

Abbreviations

CABG: Coronary artery bypass grafting; LVT: Left ventricular thrombus; TTE: Transthoracic echocardiography; VKA: Vitamin K antagonist; ECG: Electrocardiogram; CAG: Coronary angiography; MB: Myocardial bridging; MRI: Magnetic resonance imaging; ET: Essential thrombocytopenia; LAD: Left anterior descending branch; RCA: Right coronary artery

Declarations

Ethical approval and consent to participate:

The authors declare that this case report is in agreement with the Ethics Committee of Zhejiang University. Written consent have been obtained for this study

Disclosures:

The authors have reported that they had no relationships relevant to the contents of this paper to disclose

Consent for publication:

We announce that all presentations of this case report have consent to publish.

Availability of data and materials:

All data generated or analysed during this study are included in this published article

Competing interests:

The authors declare that they have no competing interests

Funding:

None

Authors' contributions:

Lijun Jiang finished the surgery for the patient. Fengpu He wrote the original manuscript. Yiping Jiao improved the use of English in the manuscript. They all read and approved the final manuscript.

Acknowledgements:

Not applicable

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Figures

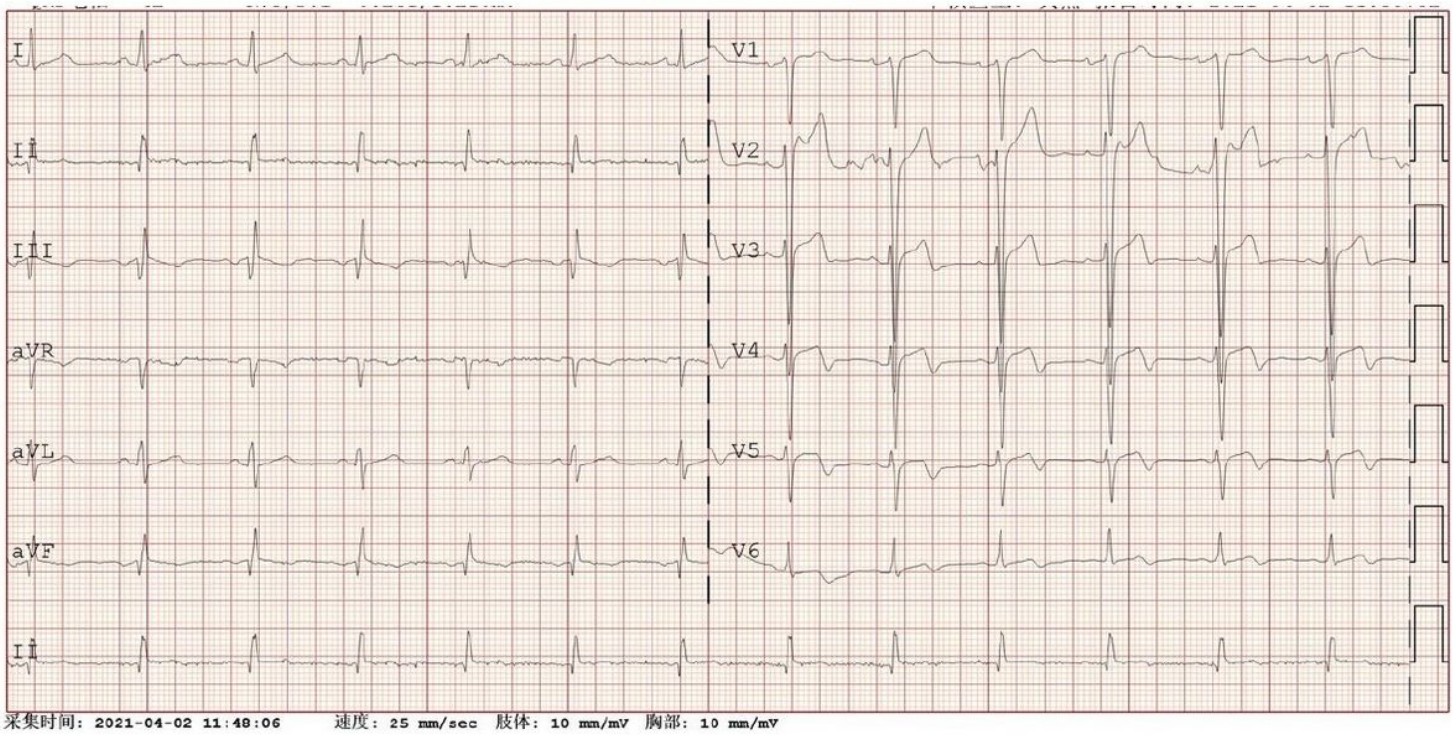

Figure 1

Preoperative electrocardiogram shows sinus rhythm，abnormal Q-wave in inferior wall (II, III, avF).

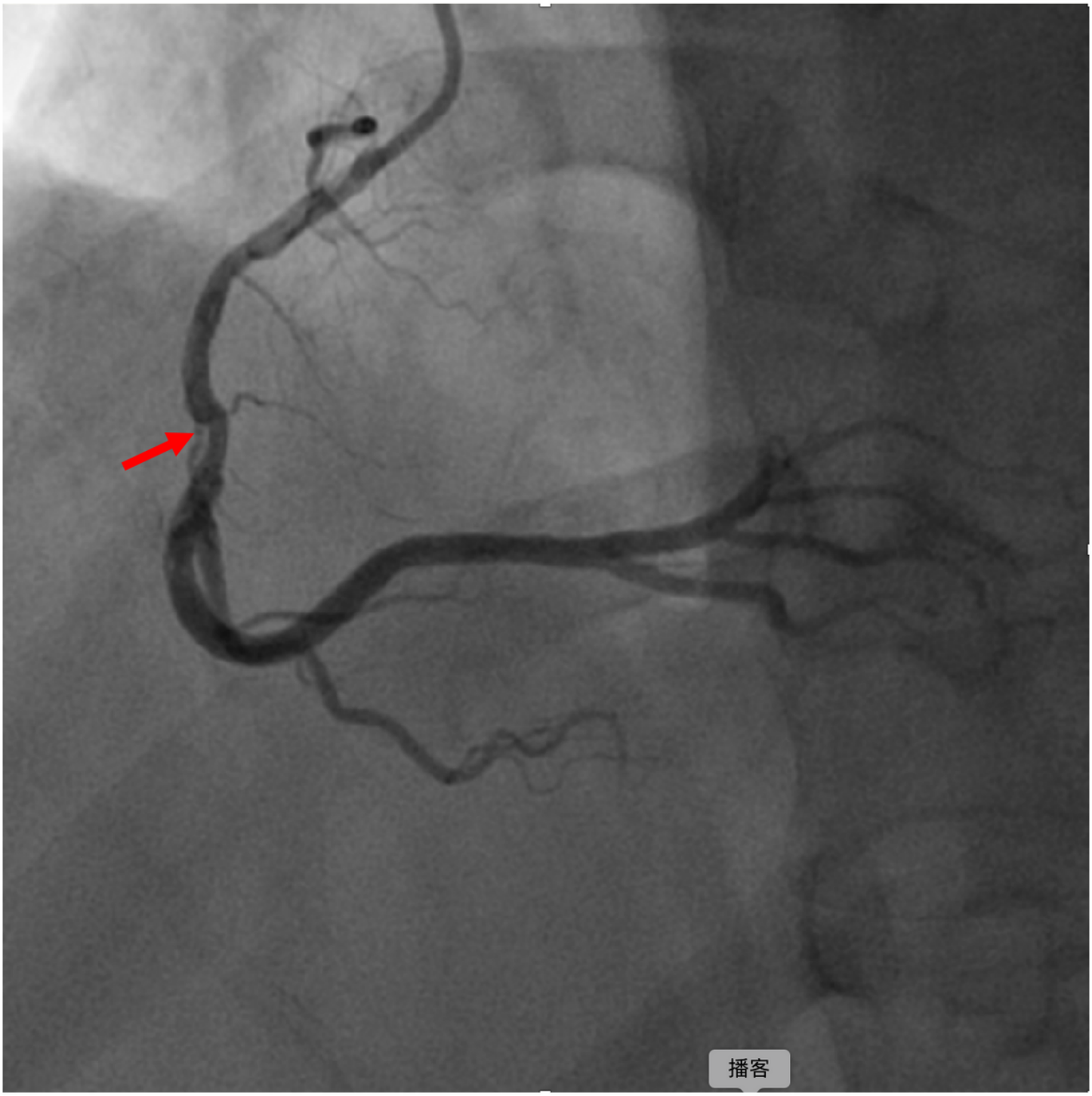

Figure 2

Myocardial bridging (MB) in middle of RCA with 70% Systolic stenosis (arrow).

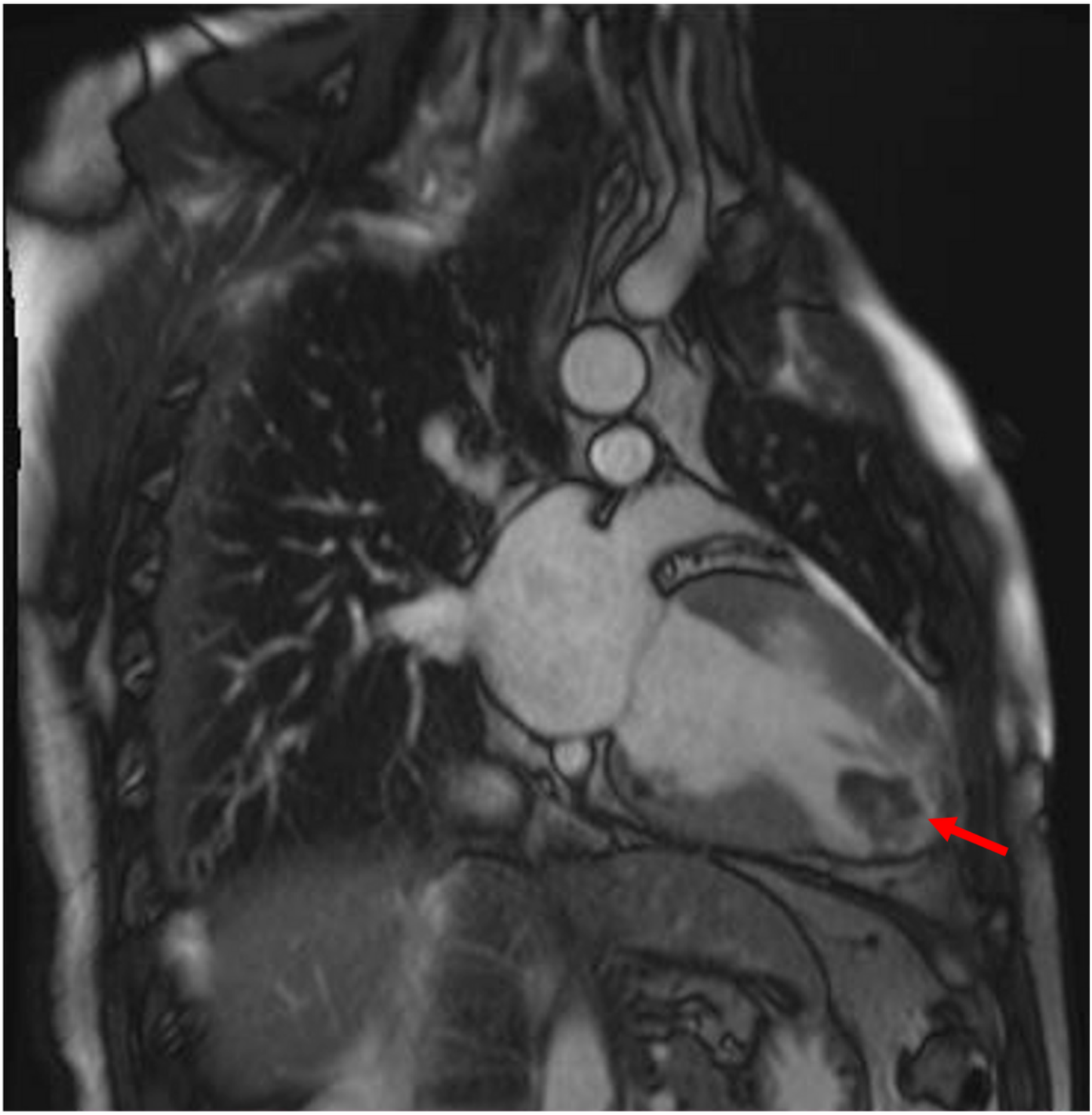

Figure 3

Presence of LV thrombus from magnetic resonance imaging study (arrow).

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