# A thrombosed left ventricular aneurysm simulating an intra -myocardial dissection hematoma: a diagnostic dilemma

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**Abstract**

A thrombosed LV aneurysm simulating an Intra-myocardial dissection hematoma (IDH) is a rarely reported condition. We describe a rare case of a 52-year-old man suffering from a thrombosed LV aneurysm, who was initially diagnosed as an IDH by conventional echocardiography. Finally, the patient was diagnosed with thrombosed LV aneurysm through multimodal imaging. We discuss the diagnostic process and surgery of the thrombosed LV aneurysm.

## Introduction

LV aneurysms is usually defined as a well-delineated, thin, scarred, or fibrotic wall devoid of muscle or containing necrotic muscle, caused by healed transmural myocardial infarction (MI)[1], thrombus is a serious complication of aneurysm, appropriate antithrombotic therapy can reduced the incidence of thromboembolism and mortality of patients with thromed LV aneurysms[2]. Intra-myocardial dissection hematoma (IDH) is an extremely rare and unusual form of impending heart rupture, with the character of neocavitation in myocardium layer caused by blood infiltrating among the myocardial fibers[3], which may contribute to ventricular rupture. Due to the high mortality of IDH, Surgery is recommoned if needed. Becaused of the serious complications and different threatment for these two diseases, an early differential diagnosis plays a vital role in developing treatment. In theory, differential diagnosis of thrombosed LV aneurysm and IDH is easy by echocardiography. However, if the shape of the intracardiac mural thrombus is similar to IDH, differential diagnosis will become difficult. Thus, we present the importance of multimodal imaging in diagnosing a rare case of a 52-year-old man with thrombosed LV aneurysm who was initially misdiagnosed as IDH.

## Case report

A 52-year-old man was referred to our institute with progressively increasing symptoms of dyspnea for 2 weeks. He revealed a history of anterior MI 2 years ago and received medical treatment then, which resulted in reduced symptoms.

Physical examination indicated blood pressure of 139/96 mmHg, heart rate of 86 beats/min and a respiratory rate of 18/min. An electrocardiogram (ECG) showed sinus tachycardia and ST-segment elevation in leads V1-V5 . Laboratory examinations revealed highly elevated levels of creatine kinase isozyme CK-MB (98 U/L, normal, 0–25 U/L) and troponin I (0.178 ng/mL; normal range, <0.045 ng/mL). Coronary angiography revealed a 90% occlusion of the proximal anterior descending coronary artery (Figure 1A).

The patient prepared to be treated by percutaneous transluminal coronary intervention (PCI). However, transthoracic echocardiography (TTE) revealed a small chamber (39 mm×25 mm in size) attached to the

apical wall with a free-floating isoechoic band (Figure 1B-D, Supplemental Videos 1-3), except for left cardiac enlargement (LAD 43mm, LVEDD 55mm), reduced ejection fraction (LVEF 40%) and abnormal motion of extensive anterior wall. Intro-myocardial dissection hematoma caused by myocardial infarction was on the top of our differential diagnosis. Furthermore, an apical aneurysm with mural thrombus was not excluded.

To identify the diagnosis, a left heart contrast echocardiography and magnetic resonance imaging (MRI) were performed. The left heart contrast echocardiogram showed that there was no entry of contrast agents into the small chamber (Figure 1E, Supplemental video 4). In the meanwhile, CMRI demonstrated a slightly short T1 and long T2 abnormal signal (37 mm×29 mm in size) located in the apex (Figure 1F-G) and first pass perfusion MRI showed that the abnormal signal had no enhancement (Figure 1H).

The preoperative diagnosis was an thrombosed LV aneurysm. The patient underwent apical aneurysm resection and thromboembolectomy under general anaesthesia and cardiopulmonary bypass. The apical thrombus was removed and the apical aneurysm was sutured (Figure 2A-B). A histopathological evaluation of excised tissue with haematoxylin and eosin staining revealed the presence of the thrombus (Figure 2C). The patient recovered well and no complications, for example serious arrhythmia, occurred. At the 3-month follow-up, the patient showed no obvious signs of heart failure. An ultrasonic cardiogram showed that the LV aneurysm had disappeared.

**Discussion**

Conventional treatment of thrombosed LV aneurysm including antithrombotic treatment to reduced the incidence of systemic embolism and mortality[2]. While IDH is a very rare presentation of cardiac rupture, the antithrombotic treatment may cause deadly tamponade. Thus, Early identification and prompt treatment is crucial in management for both diseases.

Conventional echocardiography, which is sensitive and specific in the diagnosis of thrombosed LV aneurysm. In general, the endocardial flap and the communication of intracavitary helps distinguish the IDH from the thrombosed LV aneurysm. However, in this case, the shape and the high-activity of the thrombosed LV aneurysm was similar to the cavity caused by IDH. This condition could caused much difficulty for us, and making a definitive judgment was thereby challenging.

Contrast echocardiogram is better than plain echocardiogram in identifying thrombosed LV aneurysms and increases the sensitivity and specificity for the diagnosis of mural thrombi. CMR is the most sensitive and specific study for identifying and assessing thrombosed LV aneurysm preoperatively. To confirm the diagnosis, the contrast echocardiogram and CMR were performed. What were the findings in our patient? There were no ultrasound contrast agents entering into the small chamber (Figure1E). Similarly, magnetic resonance first pass perfusion imaging revealed an abnormal signal intensity area in the apex, which was not enhanced (Figure1H). Therefore, we preliminarily concluded that the patient suffered from a thrombosed LV aneurysm rather than an IDH.

Similar case like this has not been reported in previous studies. However, the indication for aneurysmectomy is certain because previous studies have showed that aneurysmectomy might improve the outcome[4,5]. Therefore, we proposed a surgery treatment strategy. After sugery, our patient did not experience any new impairment of his functional status, neither with complex arrhythmias nor with thrombotic events.

In conclusion, it is sometimes difficult to distinguish a thrombosed LV aneurysm from IDH by conventional echocardiography if the thrombus shape changes. A definitive diagnosis involves multimodal imaging.

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## Figure legend

*Figure 1 Multimodal imaging*

*A:Left coronary angiography demonstrating a proximal occlusion of the left anterior descending artery (arrow).B:Two-dimensional TTE, apical four-chamber view, showing a small chamber with isoechoic band in the apex (Arrow); C: Doppler echocardiography, apical four-chamber view, revealing small amount of dotted blood flow signal in the small chamber; D:3-D TTE, apical four-chamber view, showing the chamber in the apex (arrow). E: TTE, apical four-chamber view, with ultrasound contrast showing no contrast agends entering the small chamber ( arrow). F-G: CMRI, two-chamber view, showed a slightly short T1 (F) and long T2 (G) abnormal signal in apx; H: Magnetic resonance first pass perfusion imaging revealed no enhancement in the abnormal signal (Arrow).*

*Figure 2 Histological findings.*

*A:The thrombus extraction with resection of the aneurysm(arrow)B:The banded thrombus inside measured approximately 4.1cm×3.6cmC:Hematoxylin and eosin staining revealed thrombus composition consisting of platelets, neutrophils and red blood cells*