

A Case Report of Intramyocardial Dissecting Hematoma Similar to Left Ventricular Mural Thrombus

Ruirui Yue*, Yudong Peng, Wei Xu#

Department of Ultrasound, The First Affiliated Hospital of Yangtze University, Jingzhou, China
Email: 859398074@qq.com, #Jzyycsxw@163.com

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Abstract

Background: Intramyocardial Dissecting Hematoma (IDH) is one of the serious and rare complications of acute myocardial infarction (AMI). It is a manifestation of subacute cardiac rupture and has a high mortality rate. With the development of imaging technology, especially echocardiography, this complication has been gradually recognized. **Case Presentation:** The patient had intermittent chest and back pain without obvious inducement and did not seek medical treatment in time. One month later, the patient came to the hospital for treatment due to the aggravation of the condition. Transthoracic echocardiography (TTE) in the other hospital showed segmental wall motion abnormality and hypoechoic mass in the left ventricular apex, which was considered thrombosis. In our hospital, the diagnosis by Transthoracic echocardiography combined with left ventricular opacification (LVO) was: segmental wall motion abnormality, left ventricular apex hypoecho mass, intramyocardial dissecting with hematoma formation were considered. Later, the diagnosis of Intramyocardial dissecting with hematoma formation was confirmed by cardiac magnetic resonance (CMR) examination in a superior hospital. **Conclusion:** In this case report, by analyzing the ultrasound imaging manifestations of left ventricular intramyocardial dissecting hematoma after myocardial infarction and its differential diagnosis with left ventricular mural thrombosis, we deepened the understanding of this rare complication and provided a reliable basis for clinical treatment decisions.

Keywords

Myocardial Infarction, Intramyocardial Dissecting Hematoma, Echocardiography, Left Ventricular Opacification

*First author.

#Corresponding author.

1. Introduction

Intramycardial dissecting hematoma is an extremely rare complication after myocardial infarction and represents a form of subacute cardiac rupture [1]. The condition is extremely perilous. The vast majority occur in the acute phase of myocardial infarction, while a small number occur months, years, or even decades after myocardial infarction. For patients with unstable hemodynamics, it may rapidly evolve into complete rupture, requiring immediate emergency cardiac surgery. However, for patients with stable hemodynamics, the new cavity may retract and close, allowing for conservative treatment. Transthoracic echocardiography can determine the location of the dissection, the size of the tear, and the trend of continued expansion. Combined with contrast-enhanced echocardiography, it can more clearly show the endocardium boundary of the left ventricle, the continuity and integrity of the endocardium and epicardial myocardium, and further confirm the diagnosis [2]. CT has high spatial resolution but limited contrast resolution, and it cannot distinguish between mural thrombi and intramycardial dissecting hematomas. Cardiac magnetic resonance is the gold standard for the diagnosis of intramural hematoma and has high tissue resolution, but it takes a long time, costs a lot, and is often not the first choice because of the severe condition of the patient. In this case, our main aim is to highlight the ultrasonographic findings of intramycardial dissection hematoma after myocardial infarction and its differential diagnosis with left ventricular mural thrombus. When transthoracic echocardiography is difficult to distinguish, the combination of left ventricular contrast echocardiography can help us to make the correct diagnosis.

2. Case Presentation

A 38-year-old male patient presented with intermittent chest and back pain for 1 month without obvious cause, accompanied by throat discomfort, which could be relieved by rest. He had shortness of breath for the past 2 days and woke up at night. Then he came to our hospital for further treatment. Physical examination showed that the temperature was 36.5°C, the heart rate was 85 bpm, the respiration was 20 bpm, and the blood pressure was 150/118mmHg. Laboratory tests showed CKMB 4.51 ng/ml, Myo 320 ng/ml, Tni 0.23 ng/ml, NTProBNP 9205 ng/ml, and D-dimer 2.73 mg/l. Electrocardiogram showed sinus heart rate, acute extensive anterior myocardial infarction, abnormal Q wave in the inferior lead, right axis deviation, and $P_{tfv1} \leq -0.04$ mms (indicating increased right atrial load). Transthoracic echocardiography in the other hospital showed left ventricular segmental wall motion abnormality (suspected ventricular septum, left ventricular anterior wall, and lateral wall myocardial infarction), abnormal echo in the left ventricular cavity (suspected thrombosis), and ejection fraction 39%. The admission diagnosis was Killip grade II myocardial infarction, possible left ventricular mural thrombosis, very high risk of grade 3 hypertension, heart failure, and coronary heart disease. Transthoracic echocardiography in our hospital showed that: 1) Segmental wall motion abnormalities: reduced left ventricular

anterior wall (basal, middle, apical), anterior septum (basal, middle), apical segment of septum and apical cap, estimated left ventricular ejection fraction 40%; 2) There was a hypoechoic attachment in the apex of the left ventricle, with a size of 57 * 20 mm, a few anechoic areas within it (**Figure 1 & Figure 2**), low amplitude swing at the edge, and no obvious blood flow signal inside (**Figure 3**). Ultrasound indication: Segmental wall motion abnormality, left ventricular apical hypoechoic mass adherent thrombus? Intramyocardial dissecting with thrombosis in the dissection? To confirm the diagnosis, the patient underwent a left ventricular opacification examination. Left ventricular opacification showed: 1) segmental wall motion abnormality: decreased perfusion of the left ventricular anterior wall (middle segment, apical segment), the middle segment of the anterior septum, an apical segment of the septum and apical cap; 2) There was a hypoechoic mass attached to the apex of the left ventricle, with a size of 56 * 20 mm, and a few anechoic areas could be seen within it. There was no contrast microbubble in it, and a slightly strong membrane-like echo was seen separating the hypoechoic mass from the cardiac cavity (**Figure 4**). Left ventricular opacification suggested that segmental ventricular wall motion was abnormal and the hypoechoic mass in the left ventricular apex was considered to be intramyocardial dissecting with hematoma formation. After that, the patient was transferred to the superior hospital for further treatment. The conclusion of echocardiography and cardiac magnetic resonance examination in the superior hospital was the same as that in our hospital: segmental ventricular wall motion abnormality and hypoechoic mass in the left ventricular apex were considered as intramyocardial dissecting with hematoma formation. Subsequently, this patient received surgical treatment.

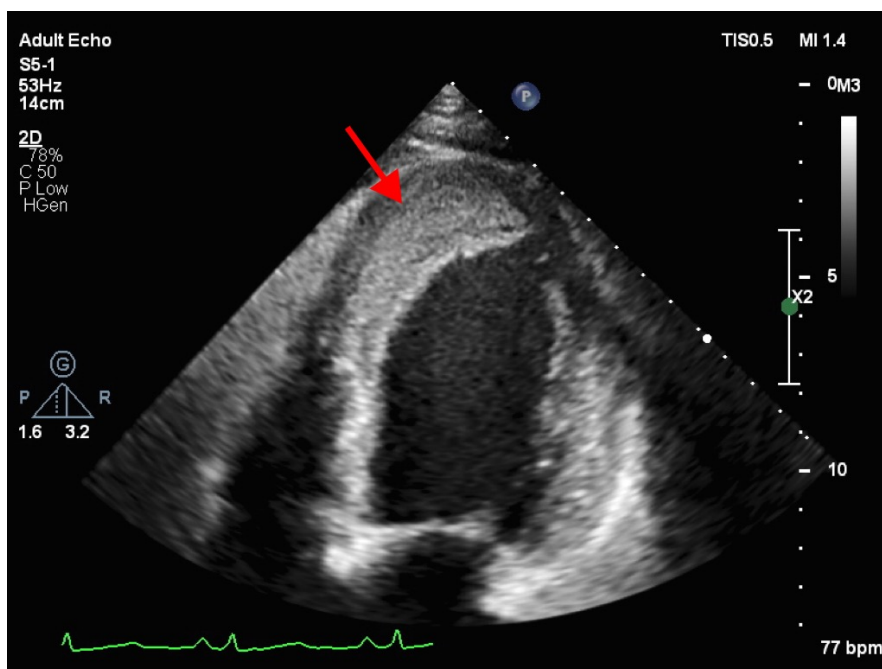


Figure 1. Left ventricular apical intramyocardial dissecting hematoma (apical four-chamber view).

During the operation, angiography was performed separately at the left and right coronary ostia. It was revealed that no obvious stenosis was observed in the left main coronary artery (LM). At the proximal segment of the left anterior descending artery (LAD), a 95% stenosis was present at the branching point of D1, and D1 was thick. Atherosclerosis was noted in the proximal segment of the left circumflex artery (LCX), and the right coronary artery (RCA) also had atherosclerosis. Later, with the consent of the patient's family, percutaneous coronary intervention (PCI) was performed on the LAD of this patient. Repeated angiography demonstrated no obvious residual stenosis.

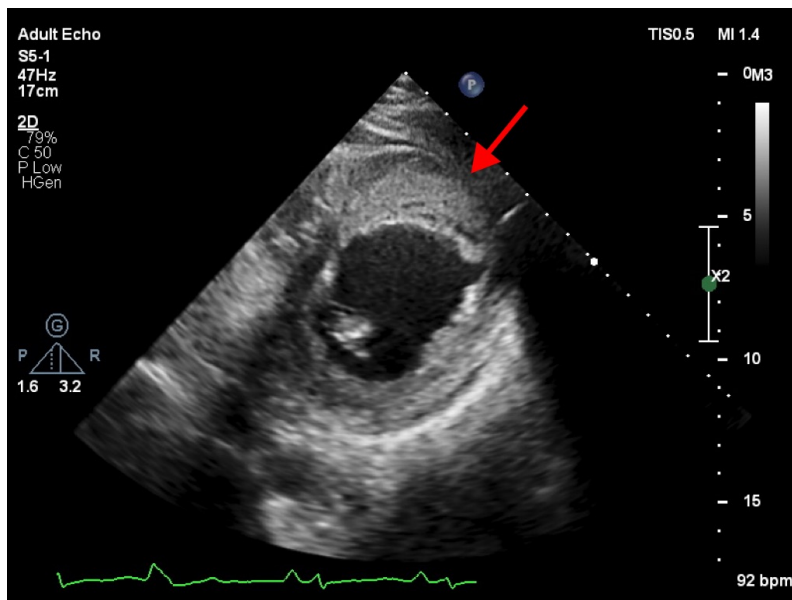


Figure 2. Left ventricular apical intramyocardial dissecting hematoma (left ventricular short axis section).

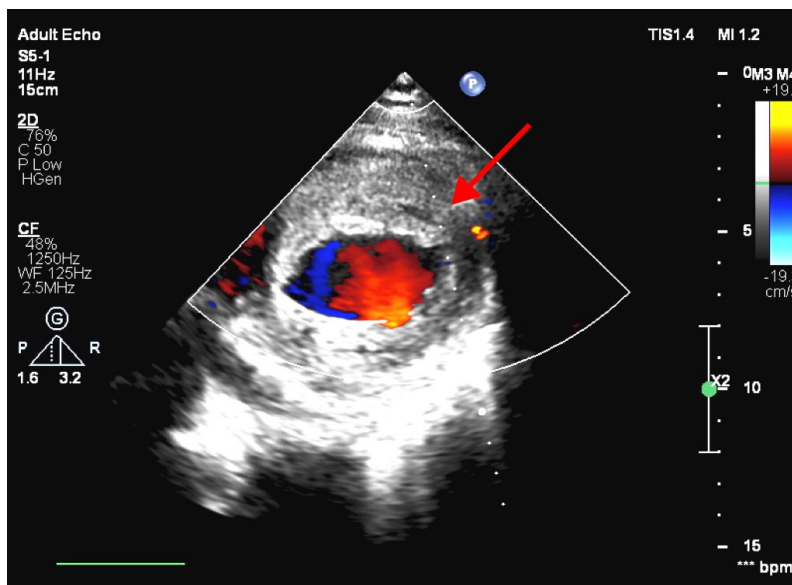


Figure 3. CDFI showed no obvious blood flow signal in the dissection.

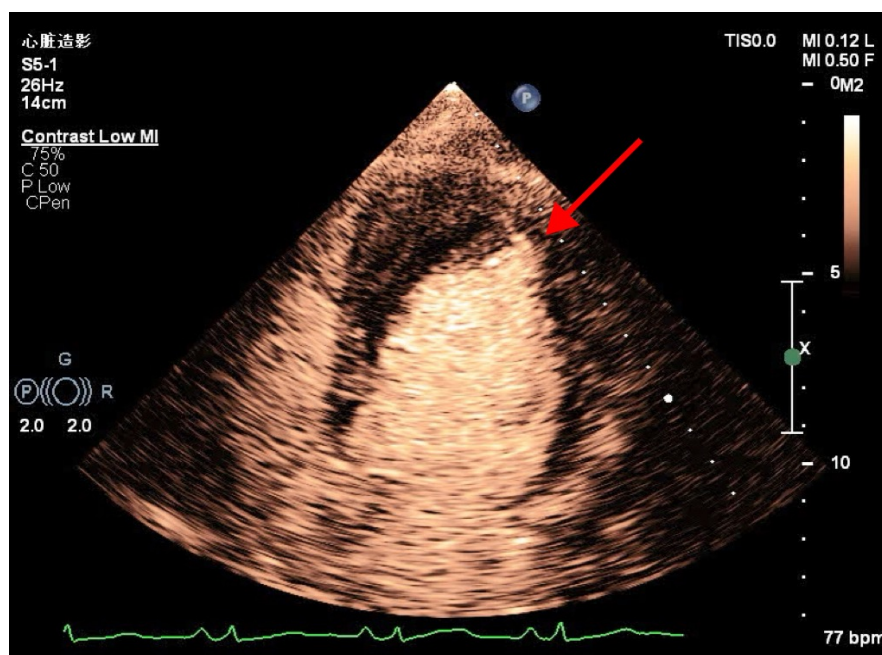


Figure 4. Left ventricular opacification shows intact continuity of the epicardium, and the tunnel into the dissection is visible at the point indicated by the arrow.

3. Discussion

Generally, after myocardial infarction, whole-layer necrosis of the myocardium will occur in the infarction area, the ventricular wall will expand and become thin, and the necrotic myocardium will gradually be replaced by fibrous scar tissue, and the normal contraction ability will be lost. Under the action of cardiac cavity contraction and pressure, the ventricular wall of the infarction area will bulge out, forming a ventricular aneurysm, and in rare cases, non-transmural myocardial infarction, that is, subendocardial myocardial infarction, will occur: The myocardium in the lower endocardial part was necrotic and ruptured, and the high-pressure blood flow in the left ventricle constantly impacted the weak myocardium, resulting in myocardium tear, and blood entered the spiral myocardial interface to form a dissection and hematoma, but the epicardium was still continuous at this time. Intramyocardial dissection hematoma can occur in the left ventricular free wall, right ventricular free wall, interventricular septum, or left atrial wall, and its manifestations are diverse. There are no typical clinical symptoms with characteristic significance. Therefore, intramyocardial dissection hematoma should be alerted to after myocardial infarction [3]. This case is a left ventricular apex intramuscular dissection hematoma. Transthoracic echocardiography can better display the location, extent, and blood flow of the dissection. Since one month had passed since the initial onset of chest pain before admission, transthoracic echocardiography did not observe the expansion and contraction of the left ventricular apical anechoic area with the heart cycle flow. Instead, it showed a hypoechoic mass in the left ventricular apex, which was easily confused with a left ventricular apical mural thrombus, leading to a misdiagnosis. Transthoracic echocardiography

showed that there was a little anechoic area in the hypoechoic mass at the apex of the left ventricle, and a low amplitude swing was seen at the edge, which was suspected to be a tear. After left ventricular opacification, it was found that the epicardium was continuous and intact, and a slightly strong membrane echo was seen on the endocardium to separate the hypoechoic mass from the cardiac cavity. A comprehensive analysis of the two can make the ultrasound diagnosis of intramyocardial dissection hematoma clearer.

Ultrasound imaging findings of intramyocardial dissecting hematoma: 1) The echo of myocardium on the endocardial surface was interrupted, the interrupted endocardium could move slightly, and the myocardial layer was separated; 2) A long strip or irregular shape of the anechoic cavity formed in the middle of the separated myocardial layer. The echo of the new cavity was consistent with that of blood; 3) With time, a hematoma formed in the middle of the separated myocardial layer, and the echo of abnormal solid mass appeared between the myocardium. The intensity of the echo could be higher, equal to or lower than that of the myocardial echo; 4) The red and blue blood flow signals at the myocardial echo discontinuity were observed by colour Doppler; 5) Left ventricular opacification showed interruption of myocardial echo on the endocardial surface, integrity of myocardial continuity on the epicardial surface, and filling defects in the left ventricular cavity, which were the same as those in conventional transthoracic echocardiography [4].

Ultrasound imaging features of mural thrombus: 1) The echo of the irregular mass was attached to the surface of the endocardium, and the endocardium was continuous and intact; 2) The fresh thrombus was hypoechoic and showed high mobility, while the old thrombus was hyperechoic and low mobility due to organization; 3) Color Doppler showed blood flow signal filling defect in the intracardiac mass; 4) Left ventricular opacification could clearly show the endocardial boundary and the echo was continuous [5].

Transthoracic echocardiography combined with left ventricular opacification is simple, feasible, economical, safe, and non-invasive. It can accurately display the location and size of the dissection, and observe hemodynamic changes in real time. Left ventricular opacification utilizes microbubbles to accurately assess myocardial perfusion, determine whether there is communication with the left ventricle, and judge whether the nature is hematoma based on the microbubble filling defect area. If no microbubbles are seen in the pericardial cavity, it can suggest the integrity of the epicardial myocardial continuity. Compared with ordinary echocardiography, this has significant application value in the diagnosis, evaluation, prognosis, and treatment of myocardial infarction [6]. Furthermore, intramyocardial dissecting hematoma requires differentiation from thick intraventricular trabeculae and pseudoaneurysms. Thick intraventricular trabeculae can form a reticular pattern at the apex of the ventricle, and the intact endocardium can be traced. Colour Doppler can detect the blood flow communicating between the ventricular cavity and the trabecular space, with the flow signal typically being relatively dim

and not prone to forming a high-speed blood flow signal. Pseudoaneurysms are formed by the adhesion of the surrounding tissues and the pericardium after a full-thickness rupture of the myocardium. At the neck of the pseudoaneurysm, the continuity of the full-thickness myocardium from the endocardium to the epicardium is disrupted, the diameter at the neck is significantly narrowed, and thrombosis often occurs within the pseudoaneurysm. Colour Doppler can observe the blood flow signal entering the pseudoaneurysm through the narrow neck during systole, and a vortex can form within the pseudoaneurysm.

4. Conclusion

The ultrasonographic findings of IDH in this case were similar to those of left ventricular mural thrombosis, and the two were easily confused, which made diagnosis difficult. Ultrasound has the advantages of being portable, rapid, accurate, and economical, and is currently the simplest method to diagnose IDH. Through the understanding of its echocardiographic characteristics, IDH can be diagnosed and identified, which provides a reliable basis for clinical treatment decisions.

Consent for Publication

Written informed consent was obtained from the patient for publication of this Case Report and any accompanying images.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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