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CASE REPORT

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Post myocardial infarction left ventricular intramyocardial dissecting hematoma penetrated right ventricular outflow tract: a rare complication report

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Abstract

Background Intramyocardial dissecting hematoma (IDH) is a rare mechanical complication following myocardial infarction (MI), and only a few isolated cases have been reported to date. IDH presents with diverse clinical manifestations, often resulting in missed or misdiagnosed cases due to limited physician understanding. The diagnosis and treatment of IDH is a major challenge.

Case presentations We report a case of acute extensive anterior MI in a 73-year-old woman, who underwent percutaneous coronary intervention (PCI); the left ventricular intramyocardial dissecting hematoma (LVIDH) penetrated the right ventricular outflow tract (RVOT), resulting in thrombus formation and subsequent RVOT obstruction. Clinically insignificant IDH was detected by transthoracic echocardiography (TTE) at 3 days, 43 days, and 75 days post-PCI, with characteristic changes in the left ventricular wall ultrasound images. This unusual case highlights the important role of continuous transthoracic echocardiography in identifying this rare complication of LVIDH. After a detailed discussion with the patient, the choice between conservative or surgical management of IDH depends on factors such as the size of the hematomae, left ventricular systolic function, and the patient's clinical and haemodynamic status. In this particular case, conservative management was chosen by the patient who declined surgery but unfortunately succumbed to cardiogenic shock.

Conclusions This case describes a rare complication of acute myocardial infarction (AMI) and also focuses on the utility of TTE in the diagnosis of this rare complication. Whether LVIDH is treated conservatively or surgically requires careful evaluation to achieve the best prognosis for the patient.

Keywords Left ventricular intramycardial dissecting hematoma, Post myocardial infarction complication, Transthoracic echocardiography, Mechanical complication

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Introduction

Intramyocardial dissecting hematoma (IDH) is a type of incomplete cardiac rupture, whose clinical symptoms can range from asymptomatic to cardiac death, most commonly caused by myocardial infarction (MI), percutaneous coronary intervention (PCI), coronary artery bypass grafting(CABG), cardiac surgery or spontaneously [1]. It is a rare mechanical complication with a high case mortality rate. Diagnosis and treatment of IDH are challenging [2]. Our case describes a rare complication of acute myocardial infarction (AMI) and found that continuous transthoracic echocardiography (TTE) was effective in identifying this rare complication.

Case presentation

A 73-year-old female patient with a prior history of type 2 diabetes mellitus and hyperlipidemia, was admitted for intermittent chest tightness and pain of one week's duration and worsening over the past three days. Laboratory tests revealed elevated levels of myocardial enzymes: alpha-hydroxybutyrate dehydrogenase (a-HBDH) at 592 IU/L (reference range: 95–250), lactate dehydrogenase (LDH) at 718 U/L (reference range: 120–250), creatine kinase (CK) at 278 U/L (reference range: 40–200), CK-MB at 30 U/L (reference range:0–25), troponin I (cTni) at 6.30 ng/ml (reference range: < 0.1) and myoglobin (Myo) at <30.0 ng/ml(reference range: < 70). The emergency 18-lead emergency electrocardiogram (ECG) showed abnormal QRS complexes in the extensive anterior wall leads (V1-V6, I, aVL) with ST-segment elevation of 0.05 to 0.10 mV and inverted T waves. In addition, there were abnormal QRS complexes in the inferior wall leads (II, III, avF) (Fig. 1a). 6 days after admission, coronary angiography showed significant stenosis of approximately 80–90% in the proximal and proximal-mid-segment of the opening of the anterior descending artery along with its diagonal branch orifice(Fig. 2a and b). Two stents, EXCRO 3.0×36 mm and LEPU 3.5×21 mm, were implanted after balloon pre-dilatation of the stenotic lesions in the proximal and proximal-mid-segment of the opening of the anterior descending branch via the right radial artery pathway, and high-pressure balloon dilatation was performed with no residual stenosis, and TIMI 3 flow (Fig. 2c and d). Dual antiplatelet therapy (DAPT) consisting of aspirin plus clopidogrel was administered postoperatively. Two days after PCI, TTE showed the hypokinesis of the anterior septum, anterior wall, and lateral wall were thickened with spot-like and homogeneous echo. The anterior septal thickness was approximately 16 mm (Fig. 3a and d). An apical aneurysm measuring approximately 20×20 mm was also observed along with minimal pericardial effusion. The left ventricular end-diastolic dimension (LVDD) measured 42 mm and the left ventricular ejection fraction (LVEF) was calculated to be 61%. The patient was discharged after stabilisation.

One month after PCI, the patient experienced recurrent chest pain and tightness, accompanied by dizziness and left-sided tinnitus. 36 days post PCI, she suddenly fainted with loss of consciousness and fecal incontinence while using the toilet. After cardiopulmonary resuscitation, she was urgently admitted to our hospital for further treatment. Laboratory tests revealed elevated levels of D-dimer (1.08 ug/ml; reference range: 0-0.5), a-HBDH (317 IU/L), LDH (430 U/L), cTni (2.25 ng/ml) and Myo (85.6 ng/ml). However, CK and CK-MB were within normal limits (CK: 124 U/L; CK-MB: 15 U/L). The ECG showed sinus tachycardia, with the anterior wall ST segments largely back to baseline and the T waves showing a shallow inversion. The high lateral wall ST segments had a concave upward elevation of 0.05 mV, and the T waves were inverted. Correspondingly, there was a reciprocal depression of the inferior wall ST segments (Fig. 1b). At 43 days post PCI, TTE showed increased thickness of hypokinetic and echo-decrease segments including the anterior septum (23 mm), posterior septum(22 mm), anterior wall(22 mm), and lateral wall(19 mm) (Fig. 3e). The left ventricular (LV) long-axis view showed an extremely low echo area space measuring 36×25 mm sandwiched between the myocardium without evident color flow signal on color Doppler imaging(Fig. 3b). The right ventricular outflow tract (RVOT) mass measuring approximately 24×21 mm was attached to the mid-segment of the anterior septal surface with minimal color flow signal at their junction observed on color Doppler imaging(Figs. 3e and 4a and b, Video 1). This mass caused an increase in forward velocity across the pulmonary valve resulting in a flow rate of 2.5 m/s and a pressure difference of 25 mmHg (Fig. 4c and d, Video 2). There was no significant increase in pericardial effusion, but mild tricuspid regurgitation was noted along with mild pulmonary hypertension (RVSP: 43mmHg) (Fig. 3). LVDD was measured to be 35 mm and the LVEF was calculated to be 65%. These findings, combined with the initial TTE image, suggested a left ventricular intramyocardial dissecting hematoma (LVIDH) penetrating the RVOT and causing RVOT obstruction due to thrombosis. Despite the cardiothoracic surgeon's recommendation for surgical intervention, the patient's family refused. Ultimately, the patient was discharged again after symptom relief.

However, half a month later, she presented with chest tightness and pain, shortness of breath, fatigue, and orthopnea. 75 days after PCI, she sought emergency treatment at our hospital with a blood pressure of 60/39 mmHg and a heart rate of 140 bpm, and was admitted to the CCU with a history of cardiogenic shock. Approximately four hours after admission, she suddenly developed dyspnoea with generalized sweating and cold,

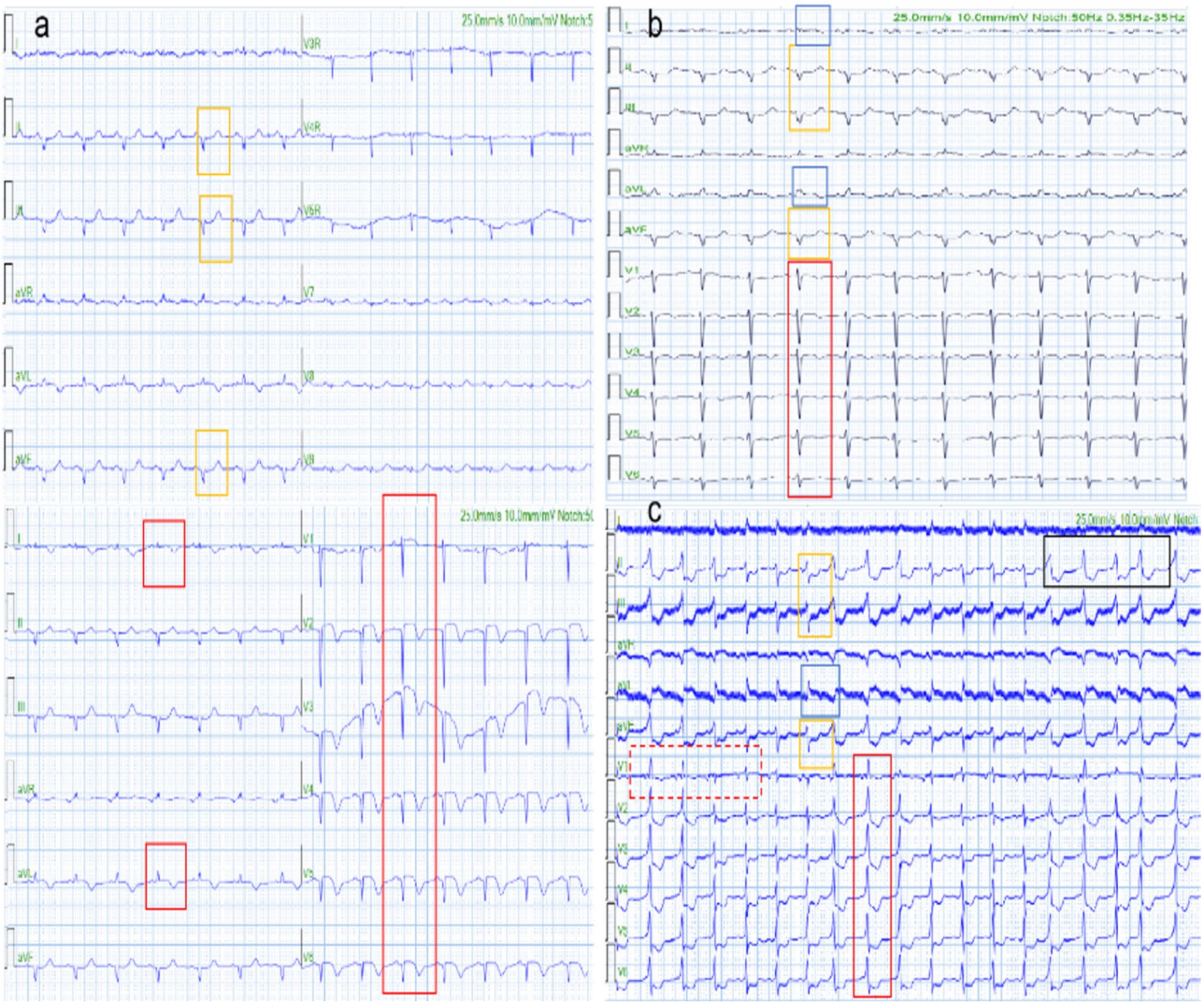

Fig. 1 **a**, The 18-lead emergency ECG at the first visit, showing abnormal QRS complexes in the extensive anterior wall leads (V1-V6, I, aVL), with ST-segment concave upward elevation ranging from 0.05 to 0.10 mV and inverted T waves (red frame), as well as abnormal QRS complexes in the inferior wall leads (II, III, avF) (yellow frame). **b**, ECG at 36 days post-PCI showed sinus tachycardia, with the anterior wall ST segments largely returning to baseline and the T waves showing a shallow inversion (red frame). The high lateral wall ST segments exhibit a concave upward elevation of 0.05 mV, and the T waves are inverted (blue frame). Correspondingly, there is a reciprocal depression of the inferior wall ST segments (yellow frame). **c**, Emergency ECG at 75 days post-PCI showed high lateral ST-segment elevation of 0.05 mv, T wave inversion((blue frame), corresponding reciprocal ST segment depression of inferior and anterior wall((yellow frame and red frame), episodes of brief ventricular tachycardia (black frame) and sinus tachycardia(red dotted frame)

clammy skin, her respiratory rate increased to 42 bpm while fingertip oxygen saturation remained at 96%. Heart rate reached 141 bpm, while blood pressure was stabilized at 105/60 mmHg with the support of norepinephrine. The bedside chest X-ray (Fig. 5) showed a significant left pleural effusion. Despite the drainage of approximately 600 ml of pale yellow fluid via a left thoracic puncture, the patient's dyspnoea was not immediately relieved. As a result, the patient was immediately placed on mechanical ventilation. Laboratory tests revealed elevated levels of D-dimer (1.90 ug/ml, reference range 0-0.5), a-HBDH (1046 IU/L), LDH (1143 U/L), cTni (2.85 ng/ml) and Myo (87.3 ng/ml). However, CK and CK-MB were within normal limits (CK: 12 U/L, CK-MB: 21 U/L). The emergency ECG showed a high lateral ST-segment elevation of 0.05 mv, along with T-wave inversion. Corresponding reciprocal ST-segment depression is seen in the inferior and anterior leads. There are also episodes of brief ventricular tachycardia and sinus tachycardia (Fig. 1c). Emergency bedside TTE performed at 75 days after PCI showed heterogeneous echo patterns in the LV wall with extensive thickening involving the interventricular septum, anterior wall, lateral wall, posterior wall, and inferior wall (Fig. 3f). In particular, the anterior septum showed thickening of up to 25 mm. An extension of the low echo area was previously observed as an extremely

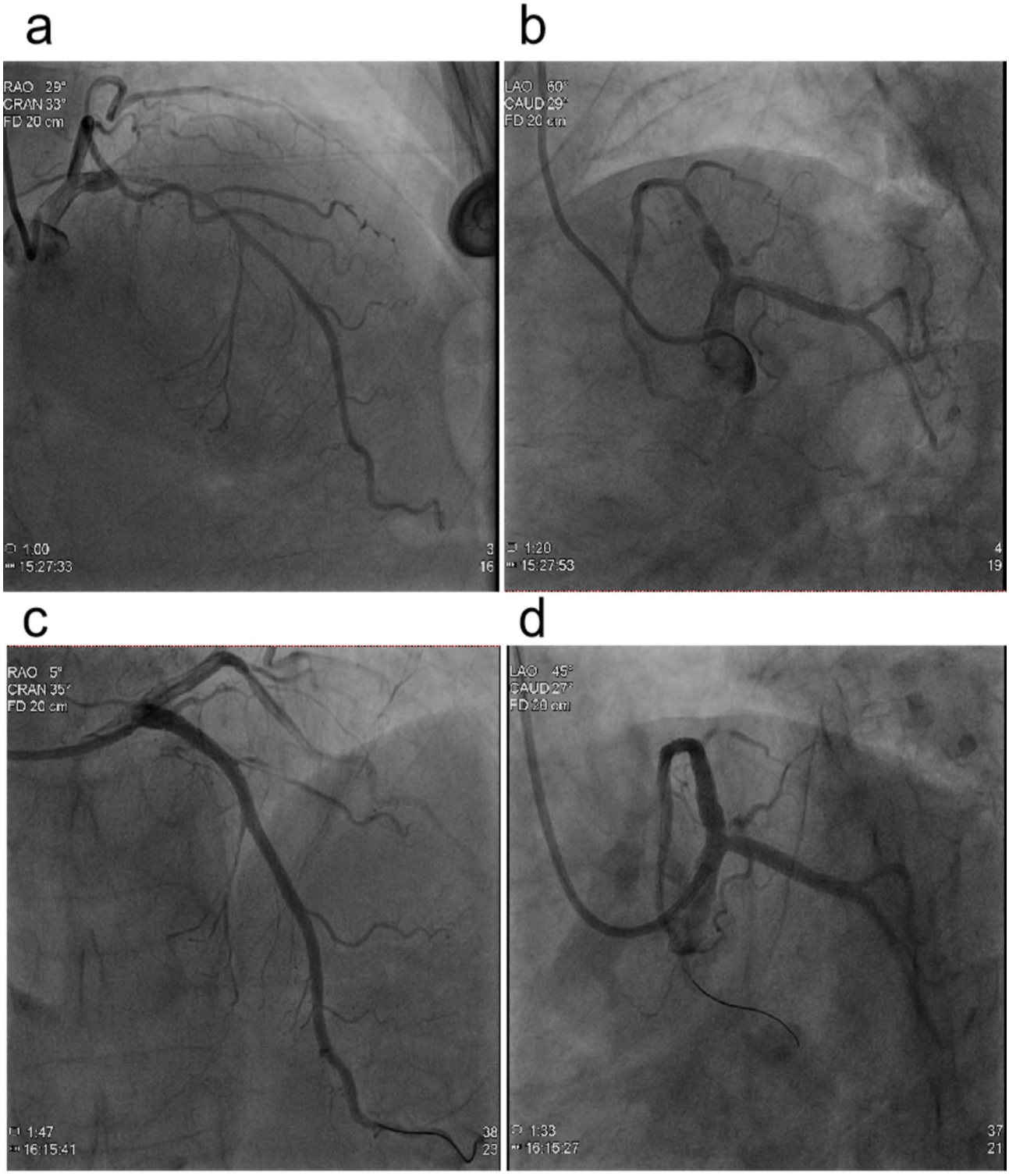

Fig. 2 **a**, **b**, Pre-intervention image: coronary angiogram showed significant stenosis of approximately 80-90% in the proximal and proximal-mid-segment of the opening of the anterior descending artery along with its diagonal branch orifice. **c**, **d**, Post-intervention image: Two stents were successfully deployed to the proximal and proximal-mid-segment of the stenotic left anterior descending artery (LAD) ostium. The final angiography demonstrated optimal results with TIMI 3 flow

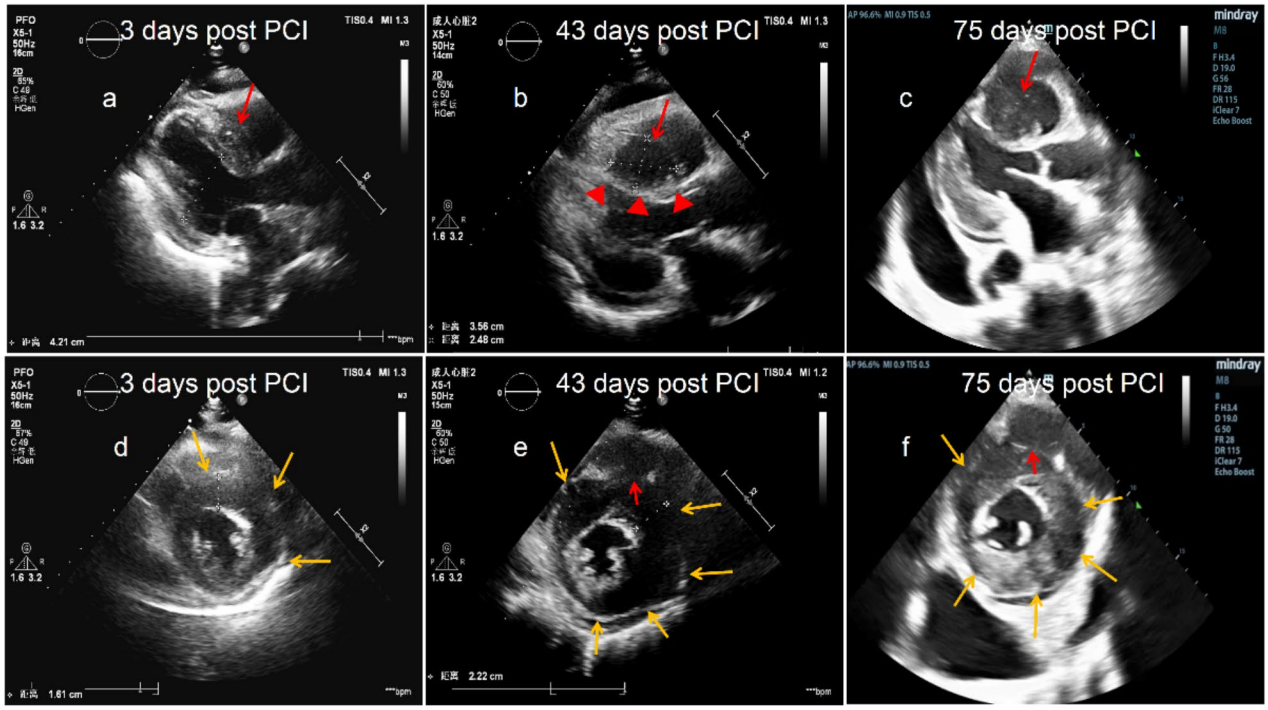

Fig. 3 **a**, **b**, **c**, TTE(LV long-axis view)at 3 days, 43 days, and 75 days post PCI showing the anterior septum thickness change, range, and echo change of the extremely low echo area in the anterior septum (red arrow). b, The extremely low echo area measuring 36×25 mm sandwiched between the myocardium (red triangle arrow). **d**, **e**, **f**, TTE (LV short-axis view) at 3 days, 43 days, and 75 days post PCI showing the echo and thickness changes of LV wall, and the thickening range of the LV wall was enlarged, and the inferior wall was gradually involved (yellow arrow). At 43 days and 75 days post PCI, the mass (red short arrow) of RVOT was connected to the middle segment of the anterior septum surface. TTE image showing no increase in pericardial effusion

low echo area in the LV long axis, but with unclear borders towards the muscular layer (Fig. 3c). Compensatory motion was enhanced in the interventricular septum, while reduced motion amplitude was observed in the inferior wall, and no motion was observed in other segments of the LV wall. Additionally, there was an increase in mass within the RVOT (approximately measuring 34×23 mm) (Video 3), moderate mitral regurgitation and mild aortic regurgitation together with a small amount of pericardial effusion(Fig. 4e and f). The LVDD was reduced to 35 mm, and the cardiac function was significantly reduced (LVEF: 20%). Based on three consecutive TTE image changes and clinical data analysis, we diagnosed this case as LVIDH rupture into the ROVT with thrombosis leading to cardiogenic shock. Considering the significant reduction in cardiac function and multiple organ damage as high risk factors, the cardiologists together with the cardiothoracic surgeons decided on a conservative management approach. Unfortunately, her family chosed to discharge her without further treatment, and subsequent telephone follow-up revealed that she had died.

Discussion and conclusions

Intramyocardial dissecting hematoma (IDH), also known as intramural hematoma, myocardial dissecting hematoma, or myocardial hematoma, is a rare and life-threatening mechanical complication of MI with high mortality. It can also occur following chest trauma, PCI, CABG, cardiac surgery, or even spontaneously. IDH can occur in the LV free wall, right ventricular (RV) wall, interventricular septum, or left atrial wall. The diagnosis of IDH after MI is challenging and must be considered in conjunction with clinical symptoms and signs [3]. IDH are localised in the early stages and may subsequently expand and rupture into surrounding tissues, and become self-absorbing or transform into thrombi. Vargas-Baron et al. proposed that IDH could be diagnosed as long as the following three or more echocardiographic features were satisfied: (1) One or more new cavities with cavity echo were formed in the tissue; (2) Thinning and active endocardial border around the cavitary defect; (3) ventricular myocardium is found in areas outside the cystic region; (4) Echo changes of new cavities suggestive of blood; (5) Partial or complete absorption of the cystic structures; (6) Continuity between the dissecting hematoma and one of the chambers; (7) Communication between the two ventricles through the myocardial

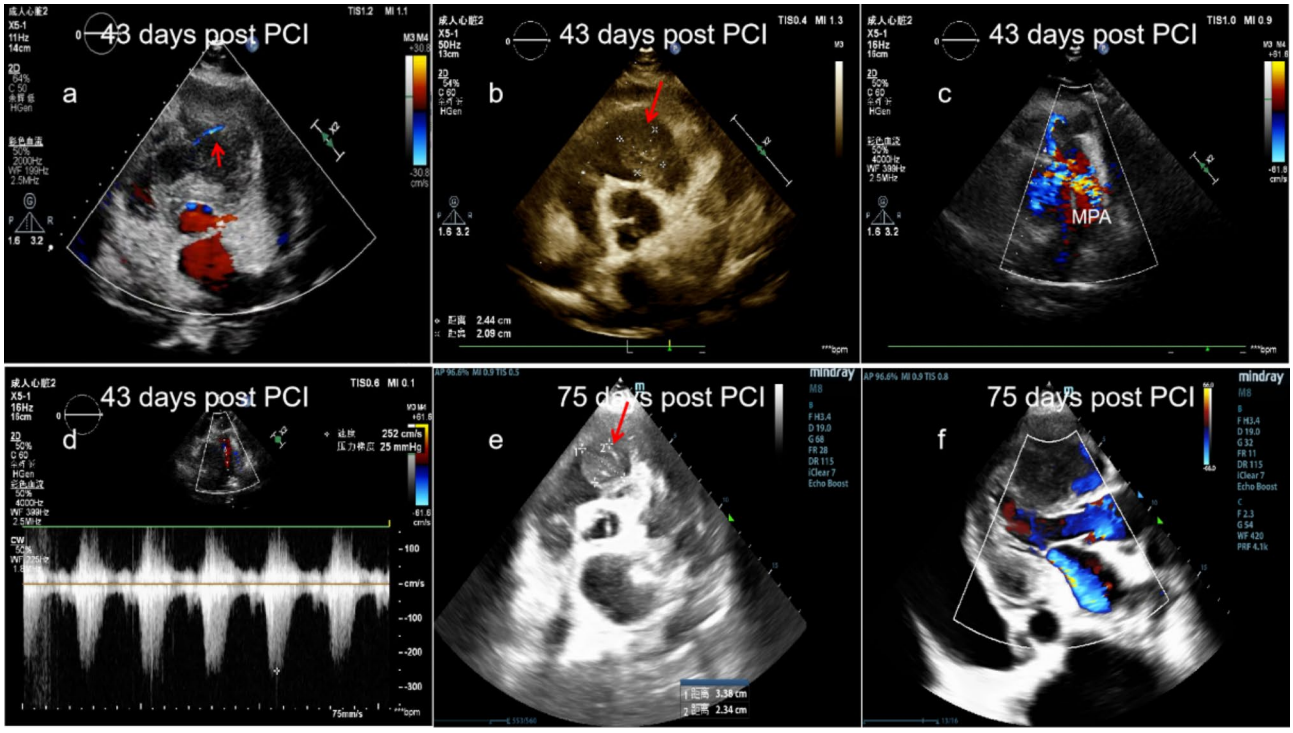

Fig. 4 **a**, **b**, At 43 days post PCI, the RVOT mass (red arrow) measuring approximately 24×21 mm was connected to the middle segment of the anterior septum surface with minimal color flow signal at their junction observed on color Doppler imaging (red short arrow). **c**, **d**, The mass caused an increase in forward velocity across the pulmonary valve resulting in a flow rate of 2.5 m/s and pressure differential of 25 mmHg. e, f, At 75 days post PCI, TTE image showing an increase in mass (red arrow) within the RVOT (approximately measuring 34×23 mm), moderate mitral regurgitation, and minimal pericardial effusion. MPA, main pulmonary artery

dissection; (8) Blood flow in the myocardial dissection was recorded by Doppler [4]. The TTE images of our case were consistent with the above-mentioned 1st, 3rd, 4th, 5th, 6th and 7th. In addition, this case is the first report of RVOT obstruction caused by the spontaneous rupture of the LVIDH to the RVOT thrombus. In this case, we found that TTE was more reliable in diagnosing IDH in the LV wall. The main advantage of TTE is that it can be scanned continuously and multi-sectionally, clearly distinguishing IDH, myocardial tissue, and hematoma rupture [5]. In this case, our sonographic diagnosis was based on dilated LV wall thickness, newly formed very hypoechoic areas and echogenic changes in the interventricular septum surrounding myocardial tissue of the ventricular septum and rupture of the RV septal surface to form thrombi. TTE image changes in this patient 3 days, 43 days, and 75 days after PCI in this patient were considered to be RVOT obstruction due to spontaneous rupture of the IDH in the LV wall with thrombus formation in the RVOT.

The pathophysiological mechanisms leading to the development of IDH in patients with AMI are complex. Initially, the inflammatory response following AMI may exacerbate myocardial tissue damageand facilitate the formation of IDH [6]. Subsequently, myocardial cell necrosis and apoptosis are closely linked to the generation of IDH [7]. In addition, post-AMI changes in cardiac structure and function, such as increased ventricular wall stress and ventricular remodelling, may affect the integrity of the myocardial microvasculature, further promoting hematoma progression [8]. Moreover, the performance of PCI after a myocardial infarction may result in further damage to the coronary microvasculature, causing blood to leak from the injured vessels and form a hematoma [9]. The likelihood of myocardial hemorrhage is higher in patients who receive reperfusion therapy at a later stage [10]. Finally, the use of antiplatelet and anticoagulant therapies after PCI may increase the risk of bleeding, especially if the microvasculature is compromised, potentially leading to the formation of IDH [11]. In our patient, PCI was performed six days after admission, and the occurrence of postoperative LVIDH may be due to the combined effects of the above mechanisms, affecting the recovery of cardiac function and the patient's prognosis.The true cause of the patient's LVIDH could not be confirmed as there was no autopsy report.

IDH is a serious mechanical complication of MI. The best management of IDH depends on a variety of factors. Patients with small lesions located at the apex, who are hemodynamically stable, without risk predictors such

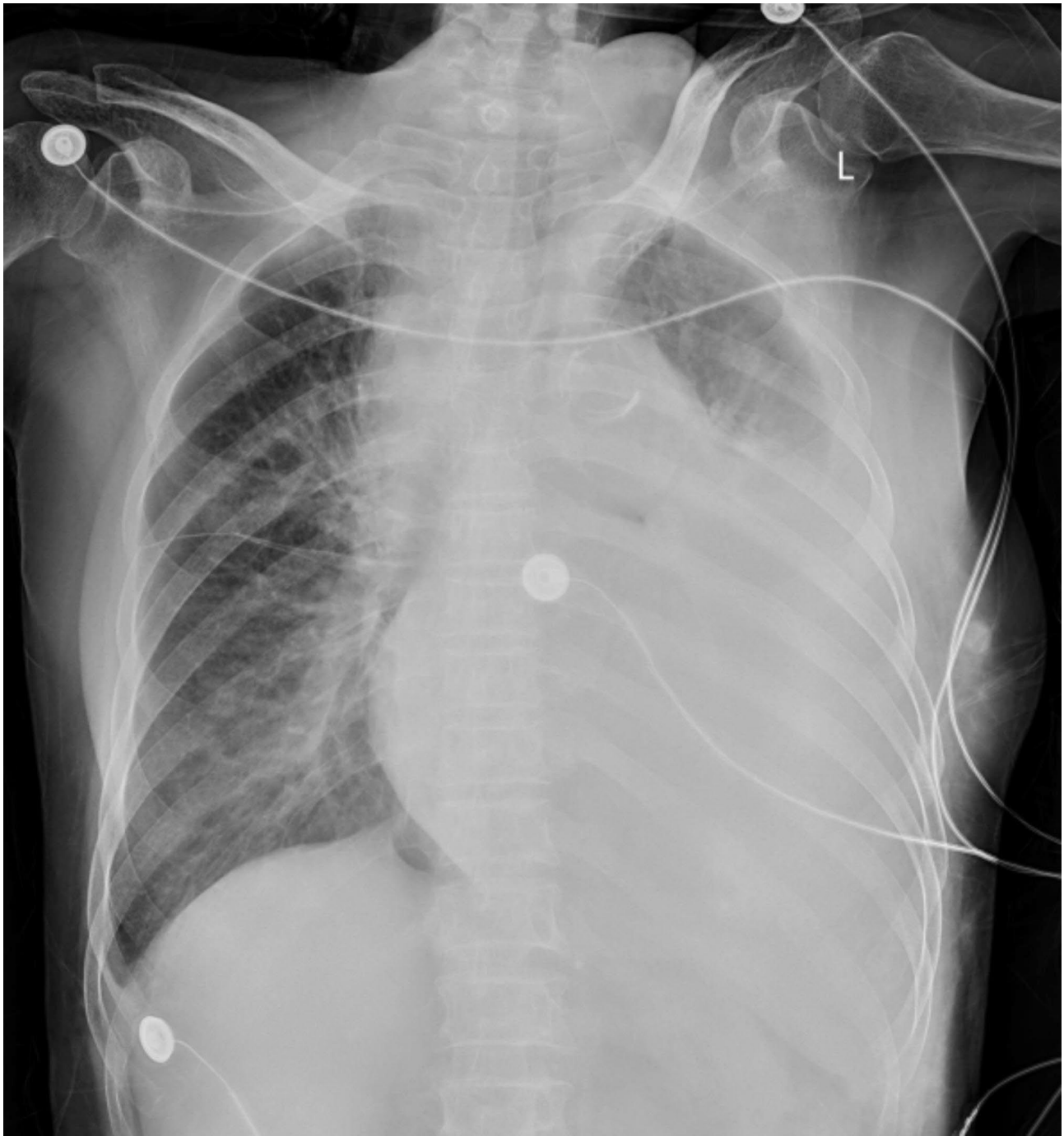

Fig. 5 Bedside chest X-ray showed significant left pleural effusion

as ventricular septal perforation, pericardial effusion, and with patent culprit vessels, are often candidates for conservative management. Conversely, continuous TTE shows an enlarged IDH, and patients with ventricular septal rupture, low ejection fraction, and hemodynamics instability, especially those with anterior MI, should be treated surgically [12]. Our patient was treated conservatively without aggressive surgical treatment and eventually died. IDH is a difficult problem to diagnose and treat. Prompt diagnosis requires vigilance and a high index of suspicion by healthcare professionals, and the decision to treat conservatively or surgically is based on hematoma size, LV systolic function, and the patient's clinical and hemodynamic status after careful discussion with the patient whether to treat conservatively or

surgically. Low EF, age > 60 years, and late diagnosis were all predictors of in-hospital mortality [13].

This case highlights the importance of TTE in the early detection of IDH. When persistent ST-segment elevation and pericardial effusion occur in patients with MI after PCI, the echocardiologist should pay attention to multisectional scanning and carefully observe the wall thickness, echo, endocardial and epicardial continuity, while closely monitoring and following up with the patient. The disadvantage of this case is that IDH was only considered on the basis of three consecutive TTE image changes and the patient was not followed up with cardiovascular magnetic resonance imaging (CMR) and other multimodal imaging.

Abbreviations

IDH	intramyocardial dissecting hematoma
MI	myocardial infarction
PCI	percutaneous coronary intervention
LVIDH	left ventricular intramyocardial dissecting hematoma
RVOT	right ventricular outflow tract
TTE	transthoracic echocardiography
AMI	acute myocardial infarction
CABG	coronary artery bypass grafting
a-HBDH	alpha-hydroxybutyrate dehydrogenase
LDH	lactate dehydrogenase
CK	creatine kinase
ECG	emergency electrocardiogram
DAPT	dual antiplatelet therapy
LVDD	left ventricular end-diastolic dimension
LVEF	left ventricular ejection fraction
LV	left ventricular
RVOT	right ventricular outflow tract
RV	right ventricular

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Author contributions

Qinqin Yu performed data collection, and wrote the manuscript. Chang Zhou, Ronghui Bao and Rong Liu provided guidance for the ultrasonic diagnosis. Mei Cai and Bin Rao provided help in writing the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The ethics committee of Yichang Central People's Hospital approved the study (approval number: 2024-041-01).

Consent for publication

We received consent from the patient.

Competing interests

The authors declare no competing interests.

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