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CASE REPORT

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Interventricular septal dissection secondary to acute inferior myocardial infarction: case series and literature review

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Abstract

Background Interventricular septal dissection is a critical disease characterized by the separation of the intraventricular septum into two layers, forming an intermediate layer with a cystic cavity that communicates with the root of the aorta or ventricle. It has low morbidity and high mortality rates.

Case presentation Case 1: A 58-year-old male with a history of hypertension and smoking presented to a local hospital due to chest tightness and pain for 4 days. Coronary angiography revealed diffuse lesions from the proximal to the middle segment of the left circumflex branch, with 80% stenosis at its most severe point, and complete occlusion of the proximal segment of the right coronary artery. A stent was implanted in the middle of the right coronary artery. Three months later, the patient was misdiagnosed with an aneurysm of the membranous ventricular septum with defect via echocardiography at the local hospital. After the implantation of a stent in the left circumflex branch, the patient came to our hospital for further diagnosis and treatment. The first ultrasound of our hospital misdiagnosed it as ventricular septal rupture, and a senior ultrasound doctor diagnosed the patient with interventricular septal dissection secondary to myocardial infarction. The patient underwent follow-up echocardiography every 1–2 months for 6 months. The patient remains asymptomatic with stable hemodynamics. The original treatment regimen and follow-up continues. Case 2: A 70-year-old male was admitted to a local hospital due to repeated chest distress for more than 20 years that worsened over several hours. Coronary angiography revealed complete occlusion of the right coronary artery. Cardiogenic shock occurred after percutaneous coronary intervention. The initial several echocardiography of the local hospital and our hospital misdiagnosed it as interventricular septal rupture secondary to myocardial infarction. The later echocardiography diagnosed it as interventricular septal dissection with rupture secondary to myocardial infarction. The patient underwent interventricular septal repair and mitral valvuloplasty after 25 days of medical treatment and died of multiple organ failure on the fourth day after the operation.

Conclusions These two cases illustrate a complication of acute myocardial infarction and highlight the importance of echocardiography in its diagnosis. By exploring the etiology, pathogenesis, and key diagnostic points of IVSD, this study aims to provide valuable insights for clinical practice.

Keywords Interventricular septal dissection, Interventricular septal rupture, Myocardial infarction, Echocardiography

Introduction

Interventricular septal dissection (IVSD) is a critical disease characterized by the separation of the intraventricular septum into two layers, forming an intermediate layer with a cystic cavity that communicates with the root of

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the aorta or ventricle. IVSD is caused by congenital or acquired factors, such as aortic sinus aneurysm [1, 2], infective endocarditis [3], coronary artery fistula [4, 5], chest trauma, cardiac surgery, endomyocardial biopsy, and myocardial infarction (MI) [6, 7], and has low morbidity and high mortality rates [7]. To date, few studies on IVSD after MI have been conducted, and any large case series or systematic reviews on IVSD after myocardial infarction has not been published. In this study, the diagnosis and treatment of two patients with acute inferior MI caused by complete occlusion of the right coronary artery (RCA) after stent implantation was retrospectively analyzed, and the pathogenesis, epidemiology, clinical manifestations, diagnosis, treatment and prognosis of the IVSD secondary to MI was reviewed. The aim was to explore the etiology and pathogenesis and key diagnostic points with respect to the disease and to provide valuable clinical practice experience.

Case presentation

Case 1

A 58-year-old male patient was admitted to a local hospital due to a 4-day chest tightness and pain, which progressively worsening over 11 h. The patient had a history of hypertension which was not previously treated, and had a history of smoking. His body temperature was 38.2 °C, his blood pressure was 116/73 mmHg, and his heart rate was 72 beats per minute. His cardiac troponin I (cTnI) level was 52.506 ng/ml, and his B-type natriuretic peptide (BNP) level was 876 pg/ml. An electrocardiogram (ECG) (Fig. 1) revealed ST-segment elevation in leads II, III, and aVF. Transthoracic echocardiography (TTE) revealed hypokinesis of the inferior wall of the left ventricle. Emergency coronary angiography (CAG) revealed 70% stenosis in the middle segment of the left anterior descending artery (LAD), diffuse lesions with 80% stenosis at the narrowest segment of the proximal to middle branches of the left circumflex branch (LCX), and complete occlusion in the proximal segment of the RCA. Percutaneous coronary intervention (PCI) was performed, and a stent (3.0*29 mm) was implanted in the middle segment of the RCA. Postoperative CAG confirmed that the RCA was patent with thrombolysis in myocardial infarction (TIMI) grade 3 blood flow. After surgery, the patient's temperature was 36.3 °C, his blood pressure was 101/73 mmHg, and his heart rate was 76 beats per minute. A review of a Holter ECG revealed a sinus rhythm, premature ventricular and atrial contractions, an intermittent first-degree atrioventricular block, and a second-degree type I atrioventricular block. The patient was treated with aspirin and clopidogrel for dual antiplatelet therapy, statins for lipid-lowering therapy, and pantoprazole for gastroprotection. Six months after surgery, the patient's cTnI level decreased to 0.056 ng/ml, and his BNP level decreased to 432 pg/ml.

Three months later, the patient was readmitted for LCX stenosis resolution. Laboratory tests revealed a cTnI level of 0.007 ng/ml and a BNP level of 389 pg/ml. ECG revealed no significant abnormalities. TTE revealed a false lumen protruding into the right ventricle around the membrane of the ventricular septum, a 6 mm defect measuring on the left ventricular side and

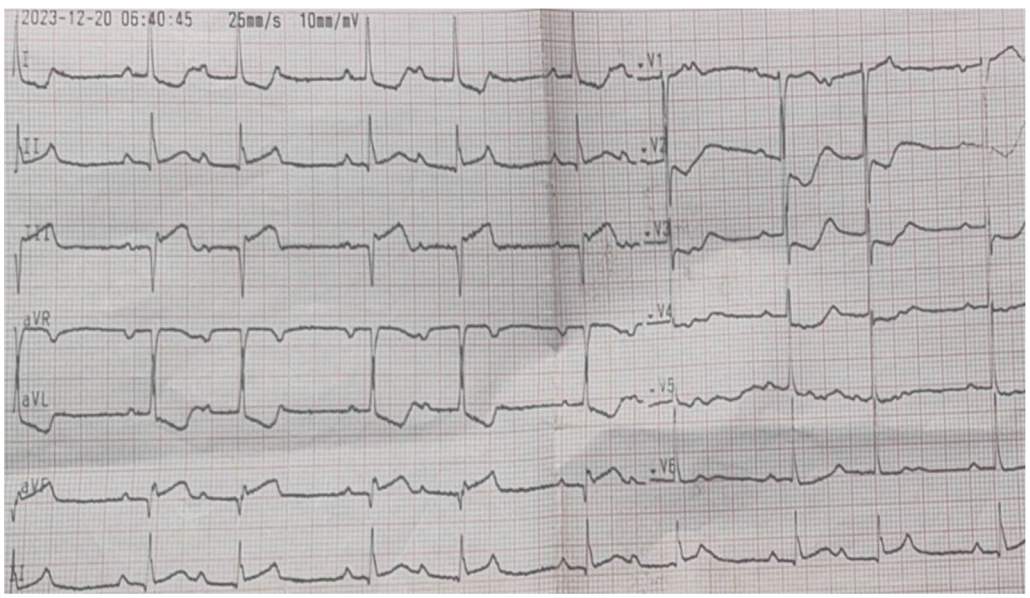

Fig. 1 First ECG performed upon admission to the local hospital showing ST elevation in leads II, III, and aVF

transseptal flow shunting between the false lumen and the left ventricle. The patient was initially misdiagnosed with an aneurysm of the membranous ventricular septum with a defect. CAG was performed again and indicated that there was no more stenosis in the middle part of the RCA where the stent was implanted and that there was 60–70% stenosis in the distal segment, which was consistent with the previous CAG results. A stent (2.75*30 mm) was implanted in the LCX. On the day following the procedure, the patient reported no significant discomfort, and his vital signs were stable. He was advised to undergo follow-up TTE and further treatment at a higher-level hospital.

Four months after experiencing acute myocardial infarction (AMI), the patient came to our hospital for further treatment. The first TTE examination conducted at our hospital revealed a 6-mm rupture in the interventricular septum, with bidirectional shunting through the rupture and weakened motion of the inferior wall of the left ventricle, indicating that interventricular septum rupture (IVSR) had occurred post-AMI. Owing to the patient's history of AMI 3 months prior and stable hemodynamics, the diagnosis was questioned by clinician. The next day, TTE examination performed by a senior sonographer at our hospital revealed a 5.0×4.0 cm septal false lumen at the basal part of the septum communicating with the left ventricular cavity through a 6-mm defect on the left ventricular side, and that the right ventricular septal endocardium is intact by two dimension(2D) imaging(Fig. 2a). Blood flow between the false lumen and the left ventricle was examined by color Doppler flow imaging (CDFI) (Fig. 2b and Video 1), and the shunting from the left ventricle to false lumen during systole and the false lumen to the left ventricle during diastole was examined via pulse Doppler (PW) (Fig. 2c).The movement of the inferior myocardial wall was decreased and left ventricular ejection fraction (LVEF) was 55%. IVSD occurred post -AMI was considered. Following multidisciplinary treatment (MDT), owing to the anatomical complexity and high surgical risk of repairing the interventricular septum, and the stable hemodynamics and small size of the rupture of the patient, the original medical treatment regimen continued without surgical intervention. The patient has returned to our hospital for follow-up for about 6 months, including TTE, cTnI and BNP every 1–2 months. The results revealed no significant abnormalities. Currently, the patient has no symptoms and stable hemodynamics, and the original treatment regimen continues.

Case 2

A 70-year-old male patient was admitted to a local hospital due to recurrent chest tightness for more than 20 years and worsening over several hours. Emergency CAG indicated 50% stenosis in the mid-segment of the LAD, multiple 20–30% stenosis in the LCX, 40% stenosis in the proximal RCA, and complete occlusion in the mid-segment with visible large thrombus shadows. PCI was performed, and two stents were implanted in the middle segment of the RCA. Postoperatively, the patient was treated with aspirin and clopidogrel for dual antiplatelet therapy, statins for lipid-lowering therapy, and pantoprazole for gastroprotection. Upon returning to the ward, the patient experienced recurrent chest tightness and cold sweats, suggesting cardiogenic shock. Treatments, including antishock measures, vasopressors, and continuous renal replacement therapy (CRRT), were provided to reduce cardiac preloading. However, hemodynamic instability persisted, and intra-aortic balloon counterpulsation (IABP), endotracheal intubation, invasive mechanical ventilation, and low-dose vasoactive drugs were used to maintain systemic circulation. TTE indicated IVSR

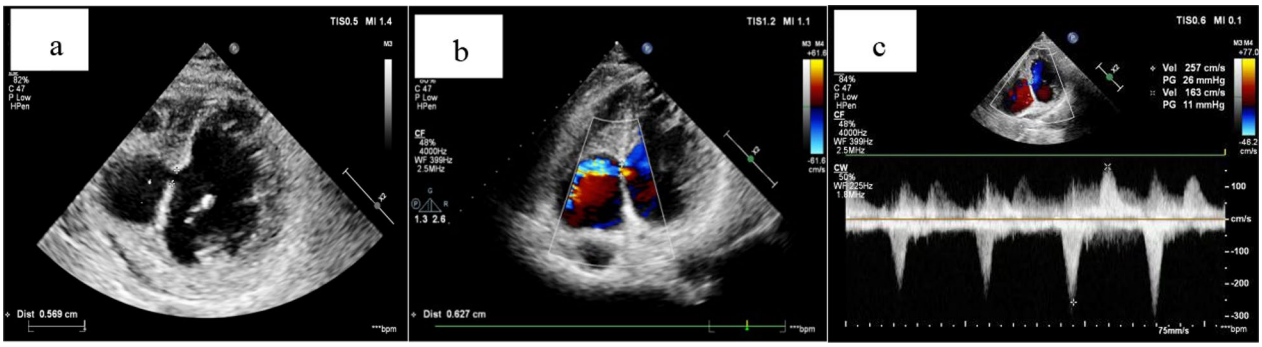

Fig. 2 TTE of the parasternal short-axis view for a patient in our hospital. **a** 2D revealed an 6 mm-laceration in the endocardium on the left ventricular side of the interventricular septum. **b** CDFI revealed bidirectional shunting between the left ventricle and the false lumen through the laceration. **c** PW revealed that the peak systolic flow velocity at the endocardial defect of the basal segment of the posterior septum was 257 cm/s from the left ventricle to the false lumen during systole, and 163 cm/s from the false lumen to the left ventricle during diastole

secondary to MI, diminished motion of the left ventricular inferior wall, and enlargement of the right ventricle and left atrium. On the 15th day after AMI, the patient was referred to our hospital for further treatment. Since the onset of illness, the patient had been bedridden, conscious but uncooperative, and anuric. The patient's temperature was 37.7 °C, his heart rate was 129 beats per minute, his respiration rate was 11 breaths per minute, his blood pressure was 133/85 mmHg, and his pain score was 0. His white blood cell count and cTnI, creatinine, alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels were 15.70×10^9/L, 1.243 ng/L, 154 μmol/L, 65 U/L, and 48 U/L, respectively. ECG indicated acute inferior wall MI. The initial several diagnosis of the bedside TTE at our hospital was IVSD On the 22nd day after AMI, TTE revealed a 5.2×4.2 cm septal aneurysm, at the basal part of the septum that communicated with the left ventricular cavity by multiple lacerations (entrance) at the left ventricular surface (Fig. 3a), the widest part being approximately 1.5–2.0 cm in size. The false lumen communicated with the right ventricular cavity via multiple lacerations (exit) at the right ventricular surface (Fig. 3a and Video 2), the widest part was approximately 5–6 mm; and the peak flow velocity was approximately 2.6 m/s during systole (Fig. 3b). The patient was considered to have IVSD with rupture. Mechanical ventilation, anticoagulation with heparin sodium, anti-infection treatment with ceftazidime-avibactam, gastric protection, phlegm resolution, and fluid supplementation were provided as symptomatic supportive treatments. On the 23rd day after AMI, computed tomography angiography (CTA) of lower limb arterial revealed possible occlusion and thrombosis. Owing to the patient's critical condition, MDT was provided at our hospital. Considering that the patient had been diagnosed with IVSD with rupture, cardiogenic shock, blood-borne infection, abnormal liver function, and lower limb thrombus, surgery was needed as soon as possible. Owing to hemodynamic instability and progressive worsening, surgery was performed on the 25th day after AMI. Intraoperative transesophageal echocardiography (TEE) revealed mitral valve prolapse with moderate regurgitation caused by minor chordae tendineae rupture, and the other results were consistent with the results of bedside TTE performed on the

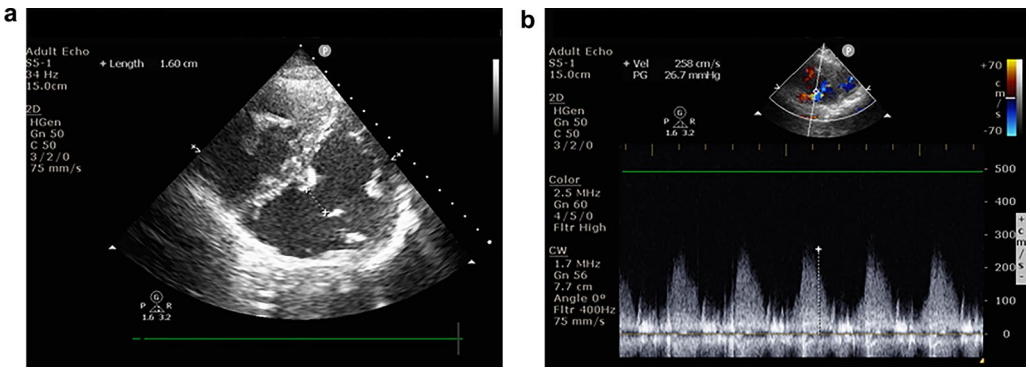

Fig. 3 TTE of the parasternal short-axis view on the 22nd day after AMI. **a** 2D image revealed IVSD with multiple lacerations on both the left and right ventricular sides. **b** PW revealed the peak systolic flow velocity at one of the lacerations of the right ventricular side of the IVSD was 258 cm/s

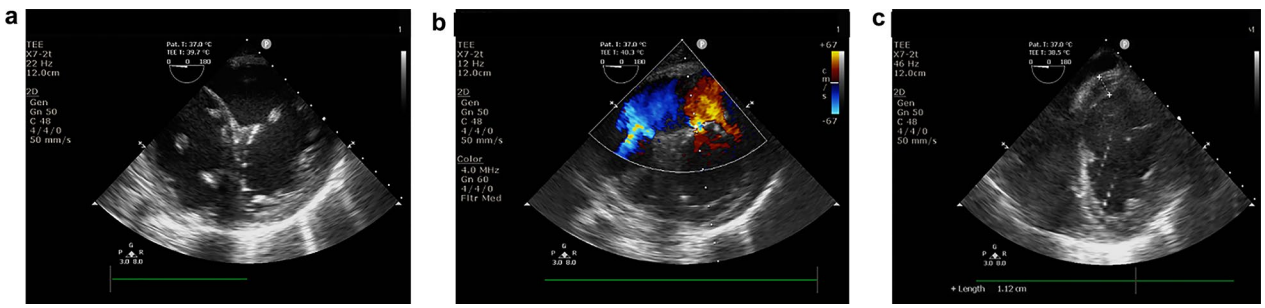

Fig. 4 TEE of the parasternal short-axis view on the 25th day after AMI. **a** 2D image revealed a false lumen with multiple lacerations on both the left and right ventricular sides. **b** CDFI revealed the left-to-right shunting through multiple lacerations on both the left and right ventricular side of the false lumen. **c** 2D image revealed hyperechogenicity in the interventricular septum

22nd day after AMI (Fig. 4a, b, and Video 3). Interventricular septum repair and mitral valvuloplasty were performed via cardiopulmonary bypass under general anesthesia. Postoperative TEE revealed a high echo in the posterior interventricular septum measuring 11 mm in thickness (as shown in Fig. 4c) without any abnormal shunting signal. A septal aneurysm, measuring approximately 4.6 × 3.0 cm in size, at the posterior interventricular septum, which was smaller than that before surgery, and decreased regurgitation of the mitral and tricuspid valves were noted. Postoperative physical examination revealed that the patient's temperature was 35.8 °C, his heart rate was 76 beats per minute, his blood pressure was 127/57 mmHg, and his blood oxygen saturation was 99%. Four days after surgery, the patient died of multiple organ failure.

Discussion and conclusions

IVSD is a subacute mechanical complication of AMI with low morbidity rate [8], and its precise morbidity rate remains unknown. IVSD occurs within 1 to 17 days (average of 7.5 days) after AMI, with peak incidences on the first day and the 7th day [8]. Current research on post-MI IVSD is limited to case reports. There are only several systematic review on myocardial dissection post-MI. Leitman et al. reported that only 42 cases of intramyocardial dissection of any etiology had been diagnosed and published in English literature to date in 2018 [9]. Hajsadeghi et al. identified that only 37cases of patients with intramyocardial dissection post -MI in English literature published from January 1990 to February 2019 [10]. We searched the Chinese and English literature published from January 1995 to October 2024, a total of 35 cases of patients with IVSD post-MI, including the two patients mentioned in this article are identified. The eligibility criteria were as the following:

1. Article type of case reports, case series, prospective or retrospective cohorts, and letters
2. Cases of IVSD in the context of AMI
3. Articles published in English or Chinese with available full text

The morbidity rate of IVSD with rupture after MI is rarer. Risk factors include advanced age, female sex, previous stroke, ST elevation, single-vessel coronary disease, high Killip grade, and complete occlusion of coronary artery without collateral circulation [8]. IVSD is almost always associated with major coronary artery lesions at more proximal portions. Zhong et al. reported a rare case of IVSD with perforation at the mid-septum due to occlusion of the first septal branch of the coronary artery [11]. Anterior MI due to occlusion of the LAD can lead to apical dissection, whereas the occlusion of RCA leading to inferior or lateral wall infarction may result in dissection at the basal segment of the septum and posterior wall junction. Reports suggest that myocardial dissection following an inferior wall AMI is more common than that following other ventricular wall AMIs [12]. This is because during inferior wall infarction, the septal branches of the RCA supply the basal segment of the septum, whereas the LAD supplies the middle and apical segments. The shearing force at the junction of the moving septum (LAD-supplied) and nonmoving septum (RCA-supplied) contributes to the formation of IVSD at the basal segment of the septum [13, 14]. Intramyocardial dissection typically originates at the site of MI, where blood enters the spiral myocardial interface because transmural blood flow impacts the ischemic myocardium. If it progresses along a curve, dissection is likely; if straight, rupture is more probable. If both are present, there is a tendency to form a dissection with rupture.

The clinical manifestations of IVSD are varied, ranging from asymptomatic to chest pain, shock and sudden cardiac arrest. The patient of Case 1 is asymptomatic, result in the neglect of IVSD. The patient in Case 2 had a sudden onset of chest pain and cardiac shock after stent implantation, and the lesion of the ventricular septum was detected by echocardiography immediately.

The diagnosis and the differential diagnosis of IVSD secondary to MI are challenging. It needs to be differentiated from the dissecting hematoma, IVSR and ventricular septal defect. Echocardiography allows for real-time dynamic monitoring and bedside examination through multiple sections, which can make a definitive diagnosis of IVSD. The diagnostic criteria of echocardiography for IVSD are as follows. 2D ultrasound revealed an interruption in the endocardium on the left ventricular side of the interventricular septum and a false lumen protruding toward the right ventricle. CDFI revealed shunting between the left ventricle and the false lumen through the detect. PW revealed bidirectional shunting through the detect if there is no detect in the right ventricular side of the interventricular septum, and left-to-right shunting if there is a detect in the right ventricular side of the interventricular septum. Cardiac magnetic resonance can be used as an adjunct in the diagnosis of IVSD, which has advantage in the detection of hematoma, reperfusion, and microvascular obstruction. IVSD is easy to misdiagnose and miss, and the main reasons are as follows. Firstly, during ultrasound diagnosis, a lack of comprehensive understanding of the concept of intramyocardial dissection and its complications can lead to the neglect of detailed observations of endocardial continuity. Secondly, attention is often focused on ventricular wall motion, the infarct site, and whether ventricular aneurysm, thrombus

formation, or myocardial perforation are present, resulting in the neglect of careful observation of endocardial continuity. Thirdly, the false lumen of the IVSD is located in the right ventricle, and sometimes, the size and shape of the IVSD resemble those of the right ventricle, leading to the inability to differentiate in certain views. In the parasternal short-axis and apical four-chamber views, the position of the right ventricular free wall are not be carefully observed, and the outer edge of the false lumen was mistaken for the free wall of the right ventricle, resulting in an incorrect diagnosis of IVSR. This diagnosis of IVSR (bidirectional shunt at the ventricular level) was also incorrect. If IVSR occurs after AMI, the shunting direction is from left to right because the pressure in the left ventricle is greater than that in the right ventricle. Bidirectional shunting is only observed in patients with ventricular septal defect when pulmonary hypertension occurs. In Case 1, in the first TEE the patient was misdiagnosed with an aneurysm of the membranous septum, which is a congenital anomaly where the membranous septum protrudes into the right ventricle. It is crucial to differentiate congenital diseases from secondary diseases. In both cases, some of the early TTE misdiagnosed it as a simple IVSR. As a sonographer, the following should be noted. First, the patient's medical history should be assessed thoroughly to confirm the presence and specific scope of MI. Secondly, multi-section scanning should then be performed to assess the integrity of endocardial continuity in the MI area because the interruption of endocardial continuity is the basis of dissection.

For IVSD secondary to MI, different treatment strategies are employed including conservative medical treatment, traditional surgical septal repair, and novel transcatheter septal closure. All these management strategies are on the basis of the patient's clinical presentation, the size of dissection and the rupture, hemodynamic stability and whether there is a hematoma or an obstruction of the right ventricular outflow tract or the left ventricular outflow tract [15, 16]. Yu et al. reported a rare case about left ventricular intramyocardial dissecting hematoma penetrated right ventricular outflow tract post-MI [17]. The choice of surgery timing is very difficult for patients with IVSD combined with rupture. The high risk of the severe difficulties in suturing due to myocardial edema in the early stage and progressively aggravated hemodynamic instability resulting in multiple organ failure should be weighed. Vega et al. suggested that if the surgery could be delayed for more than 3 days after the occurrence of VSR, a 50% reduction in mortality could be expected [18]. The surgery involves resecting the aneurysmal tissue and repairing the septum through incisions in the ascending aorta and right ventricular outflow tract. Recently, transcatheter septal closure was introduced as a new type of surgery, and its survival rate is comparable to that of traditional surgery. When hemodynamic stability is maintained and the rupture is small, close monitoring without immediate surgery may be considered [18]. If the rupture does not significantly increase during follow-up, continued observation is warranted. The size of the myocardial dissection may gradually decrease over time, potentially resolving spontaneously or leaving only a small mass similar to an old thrombus [19]. Myocardial dissection may also progressively extend to adjacent areas [20]. If complicated by rupture of the papillary muscle, chordae tendineae or interventricular septum, the prognosis of myocardial dissection is extremely poor. Vargas-Barron et al. [15] followed 15 patients who underwent left ventricular myocardial dissection after MI for more than 12 months and reported an overall mortality rate of 47%. Thirteen of these patients had IVSD, with a mortality rate of 78%. The cause of death was progressive aggravated ventricular enlargement and heart failure. In Case 1, IVSD was discovered during a follow-up TTE 3 months after the patient's underwent stent implantation. Dynamic TTE revealed that the patient had stable hemodynamics, with a 6-mm rupture and no significant change in the size of the aneurysmal false lumen, so surgery was not performed at that time. In Case 2, in view of the patient's progressively worsening heart failure and unstable hemodynamics, conventional surgery was performed on the 25th day after AMI. When perforation occurs, the condition is critical, and the mortality rate is 90%.

IVSD is a subacute mechanical complication of AMI. This complication was poorly understood, leading to frequent misdiagnoses and missed diagnoses. TTE can be used to promptly and accurately identify the location of rupture of myocardial dissection, assess its size and shunt volume, and evaluate myocardial motion in real time, allowing for direct diagnosis of IVSD or IVSR. For clinicians, it is crucial to closely monitor patients via echocardiography after PCI to avoid missing severe complications and delaying treatment. Ultrasound is valuable in terms of early diagnosis and provides valuable information for clinical treatment. As a retrospective study with a limited number of cases, our research has inherent constraints. The low morbidity and high mortality of IVSD secondary to MI have restricted the development of dedicated trials. A key limitation is that the diagnosis of IVSD relied solely on TTE or TEE image changes, without follow-up using contrast echocardiography, cardiovascular magnetic resonance imaging, or other multimodal imaging techniques. As this study is a retrospective study, the number of cases is limited. The low morbidity and high mortality of IVSD secondary to MI have limited the possibilities to

develop dedicated trials. The disadvantage of this article is that the diagnosis of IVSD was only considered on the basis of TTE or TEE image changes and the patient was not followed up with contrast echocardiography, cardiovascular magnetic resonance imaging or other multimodal imaging.

Abbreviations

IVSD	Interventricular septal dissection
MI	Myocardial infarction
RCA	Right coronary artery
cTnI	Cardiac troponin I
BNP	B-type natriuretic peptide
ECG	Electrocardiogram
TTE	Transthoracic echocardiography
CAG	Coronary angiography
LAD	Left anterior descending artery
LCX	Left circumflex branch
PCI	Percutaneous coronary intervention
TIMI	Thrombolysis in myocardial infarction
AMI	Acute myocardial infarction
IVSR	Interventricular septal rupture
2D	2-Dimension
CDFI	Color Doppler flow imaging
PW	Pulse Doppler
LVEF	Left ventricular ejection fraction
MDT	Multidisciplinary treatment
CRRT	Continuous renal replacement therapy
PASP	Pulmonary arterial systolic pressure
IABP	Intra-aortic balloon counterpulsation
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
CTA	Computed tomography angiography
TEE	Transesophageal echocardiography

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13019-024-03271-z.

Supplementary Material 1.

Supplementary Material 2.

Supplementary Material 3.

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Author contributions
YM proposed the study and was the guarantor. PC performed the research and drafted the manuscript. PC and YM revised the manuscript. YM and XW provided part of the imaging figures. All the authors contributed to the design and interpretation of the study.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Patient consent statement
The patient in case 1 and the legal client of patient in case 2 authorized the study, both of whom signed the written authorization consent form.

Ethical approval and consent to participate
Patients signed an informed consent process that was reviewed by the Ethics Committee of Zhejiang University, which certified that the study was performed in accordance with the ethical standards of the 1964 Declaration of Helsinki.

Competing interest
No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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References

1. Fennich H, Doghmi N, Rim F, Belhaj S, Cheikhi F, Cherti M. Spontaneous rupture of right aortic sinus of Valsalva leading to massive cystic dissection of interventricular septum and complete heart block. Echocardiography. 2018;35(12):2109–12. https://doi.org/10.1111/echo.14166.
2. Stróżyk A, Kołaczkowska M, Fijałkowska J, Siondalski P, Fijałkowski M. Sinus of Valsalva rupture in a patient with a mechanical aortic prosthesis: aneurysm dissecting into the interventricular septum. Kardiol Pol. 2018;76(12):1742. https://doi.org/10.5603/KP.2018.0235.
3. Li HY, Wang M, Zhang WF, Wu JT, Wang TY, Sheng W, Niu ZZ. Diagnosis, treatment and follow-up analysis of 6 cases of dissecting aneurysm of the interventricular septum. Chin J Cardiovasc. 2021;26(5):463–7. https://doi.org/10.3969/j.issn.1007-5410.2021.05.012.
4. Wu Q, Jin YP, Zhou L, Liu YF, Wu DZ. A dissecting aneurysm of interventricular septum resulting from congenital coronary artery fistula. J Clin Ultrasound. 2019;47(1):55–8. https://doi.org/10.1002/jcu.22639.
5. Huang ZX, Pu ZX, Shentu W, Li J, Shen ZH, Tan YB, Cheng L. A case report of asymptomatic right coronary fistula with giant interventricular aneurysm. Chin J Cardiol. 2019;47(6):496–7. https://doi.org/10.3760/cma.j.issn.0253-3758.2019.06.014.
6. Damluji AA, Van Diepen S, Katz JN, Menon V, Tamis-Holland JE, Bakitas M, Cohen MG, Balsam LB, Chikwe J, Cardiology AHaCOC, Council on Arteriosclerosis T, Biology V, Surgery COC, Anesthesia, Cardiovascular CO, Nursing S. Mechanical complications of acute myocardial infarction: a scientific statement from the American Heart Association. Circulation. 2021;144(2):e16–e35. https://doi.org/10.1161/CIR.0000000000000985.
7. Gong FF, Vaitenas I, Malaisrie SC, Maganti K. Mechanical complications of acute myocardial infarction: a review. JAMA Cardiol. 2021;6(3):341–9. https://doi.org/10.1001/jamacardio.2020.3690.
8. Jones BM, Kapadia SR, Smedira NG, Robich M, Tuzcu EM, Menon V, Krishnaswamy A. Ventricular septal rupture complicating acute myocardial infarction: a contemporary review. Eur Heart J. 2014;35(31):2060–8. https://doi.org/10.1093/eurheartj/ehu248.
9. Leitman M, Tyomkin V, Sternik L, Copel L, Goitein O, Vered Z. Intramyocardial dissecting hematoma: two case reports and a meta-analysis of the literature. Echocardiography. 2018;35(2):260–6. https://doi.org/10.1111/echo.13796.
10. Hajsadeghi S, Amirfarhangi A, Pakbaz M, Pazoki M, Tanha K. Postinfarction intramyocardial dissection, an interesting case report and systematic review. Echocardiography. 2020;37(1):124–31. https://doi.org/10.1111/echo.14565.
11. Roberts WC, Burks KH, Ko JM, Filardo G, Guileyardo JM. Commonalities of cardiac rupture (left ventricular free wall or ventricular septum or papillary muscle) during acute myocardial infarction secondary to atherosclerotic coronary artery disease. Am J Cardiol. 2015;115(1):125–40. https://doi.org/10.1016/j.amjcard.2014.10.004.
12. Psaltis PJ, Dundon BK, Teo KS, Worthley SG. Utility of cardiac magnetic resonance imaging in detection of post-infarction intra-myocardial dissection. Heart Lung Circ. 2008;17(5):415–6. https://doi.org/10.1016/j.hlc.2007.07.010.
13. Kalvin L, Yousefzai R, Khandheria BK, Paterick TE, Ammar KA. Ventricular septal dissection complicating inferior wall myocardial infarction. Case Rep Cardiol. 2017;2017(1):9680891. https://doi.org/10.1155/2017/9680891.

14. Ronco D, Ariza-Solé A, Kowalewski M, Matteucci M, Di Mauro M, López-De-Sá E, Ranucci M, Sionis A, Bonaros N, De Bonis M. The current clinical practice for management of post-infarction ventricular septal rupture: a European survey. Eur Heart J Open. 2023;3(5):oead091. https://doi.org/10.1093/ehjopen/oead091.
15. Vargas-Barrón J, Romero-Cárdenas A, Roldán F-J, Molina-Carrión M, Avila-Casado C, Villavicencio R, Martínez-Sánchez C, Lupi-Herrera E, Zabalgoitia M. Long-term follow-up of intramyocardial dissecting hematomas complicating acute myocardial infarction. J Am Soc Echocardiogr. 2005;18(12):1422. https://doi.org/10.1016/j.echo.2005.03.010.
16. Miura A, Uemura Y, Takemoto K, Uchikawa T, Koyasu M, Ishikawa S, Mitsuda T, Imai R, Iwamiya S, Ozaki Y. Early spontaneous remission of intramyocardial dissecting hematoma. Intern Med. 2017;56(9):1067–70. https://doi.org/10.2169/internalmedicine.56.7967.
17. Yu QQ, Liu R, Bao RH, Cai M, Rao B, Zhou C. Post myocardial infarction left ventricular intramyocardial dissecting hematoma penetrated right ventricular outflow tract: a rare complication report. J Cardiothorac Surg. 2024;19(1):601. https://doi.org/10.1186/s13019-024-03084-0.
18. Vega JDS, Salinas GLA, Florez JMV, Solé AA, De Sá EL, Sanz-Ruiz R, Palacios VB, Roubin SR, Varela SG, Forés JS. Optimal surgical timing after post-infarction ventricular septal rupture. Cardiol J. 2022;29(5):773–81. https://doi.org/10.5603/CJ.a2022.0035.
19. Malhotra A, Patel K, Sharma P, Wadhawa V, Madan T, Khandeparkar J, Shah K, Patel S. Techniques, timing and prognosis of post infarct ventricular septal repair: a re-look at old dogmas. Braz J Cardiovas Surg. 2017;32(3):147–55. https://doi.org/10.21470/1678-9741-2016-0032.
20. Vargas-Barrón J, Roldán F-J, Romero-Cárdenas Á, Molina-Carrión M, Vázquez-Antona CA, Zabalgoitia M, Rios M-a M, Pérez JE. Dissecting intramyocardial hematoma: clinical presentation, pathophysiology, outcomes and delineation by echocardiography. Echocardiography. 2009;26(3):254–61. https://doi.org/10.1111/j.1540-8175.2008.00804.x.

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