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Acute Extensive Anterior Myocardial Infarction Complicated by Intramyocardial Dissecting Hematoma of the Interventricular Septum with Perforation: A Case Report

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Case Report

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Abstract

Background

Intramural dissecting hematoma (IDH) is a rare but life-threatening subacute cardiac rupture type that occurs after acute myocardial infarction or during its remodeling process. IDH may resolve spontaneously, or it may extend and destroy adjacent structures, leading to serious mechanical complications. Owing to its low incidence and limited literature, there is currently no standardized diagnostic, therapeutic, or management consensus.

Case presentation:

A 67-year-old male with acute extensive anterior ST-segment elevation myocardial infarction underwent emergency coronary angiography and percutaneous transluminal coronary angioplasty. After the procedure, the patient still had precordial discomfort and was transferred to our hospital. One week later, repeat angiography with intra-aortic balloon pump (IABP) support revealed suspected left anterior descending branch (LAD) dissection, prompting two-stent implantation. The next day, echocardiography showed an apical septal myocardial dissecting hematoma with complex perforation. Surgical ventricular septal repair was planned electively. On day 25 after infarction, cardiogenic shock and multiple organ failure developed; after IABP and biventricular assist device support, acute cerebral infarction occurred, and the family ultimately withdrew treatment.

Conclusion

Ventricular septal IDH with perforation after acute myocardial infarction is a rare and catastrophic mechanical complication. Early recognition, appropriate timing of intervention, and adequate use of mechanical circulatory support are key to improving prognosis.

Background

Intramural dissecting hematoma (IDH) is a rare but life-threatening mechanical complication of acute myocardial infarction, with a low incidence but extremely poor prognosis [1]. IDH often occurs in the free wall of the ventricle or the interventricular septum. Its essence is a dissecting hematoma formed by blood accumulation between the myocardial fiber layers, while the endocardium and epicardium remain intact [2]. As the disease progresses, IDH may extend and destroy surrounding tissues, or it may resolve spontaneously or become thrombosed [3]. Because its effects on cardiac structure and hemodynamics vary, clinical manifestations range from asymptomatic to cardiogenic shock. Currently, there is no standardized management algorithm for post-infarction IDH. The choice between conservative treatment and surgical intervention must consider the IDH anatomy, cardiac function, hemodynamic stability, and the experience of the institution with interventional and surgical approaches. We report a

complex case of ventricular septal IDH with perforation after acute extensive anterior ST-segment elevation myocardial infarction, aiming to share experience for clinical decision-making in such critically ill patients.

Case Presentation

A 67-year-old male patient presented to a local hospital with chest pain for several hours. He had a history of hypertension (untreated) and smoking. Initial laboratory tests showed: Troponin I (TNI) 45.97 ng/ml, N-terminal pro-B-type natriuretic peptide (NT-proBNP) 610 pg/ml. Electrocardiogram (ECG) revealed ST-segment elevation in leads V1-V6. Transthoracic echocardiography (TTE) showed a left ventricular apical aneurysm. Emergency coronary angiography revealed 90% stenosis in the proximal left anterior descending branch (LAD), occlusion in the mid-segment, 90% stenosis in the proximal first diagonal branch, 50% stenosis in the distal right coronary artery, and 20% stenosis in the distal left circumflex artery. Percutaneous transluminal coronary angioplasty was performed on the LAD.

After the procedure, the patient still had precordial discomfort and was transferred to our hospital approximately 23 hours after symptom onset. On admission, physical examination: blood pressure 111/74 mmHg, heart rate 100 bpm, oxygen saturation 100%, and a blowing systolic murmur at the left sternal border. Repeat TTE showed reduced motion in the anterior septum, anterior wall (mid-segments), and apical segments; left ventricular apical aneurysm; EF 40%; moderate tricuspid regurgitation, mild mitral and aortic regurgitation; and left atrial enlargement. Laboratory tests: alanine aminotransferase 55 U/L, aspartate aminotransferase 328 U/L, TNI 228842 ng/l, creatine kinase-myocardial band 212.4 ng/ml, myoglobin 496.1 μg/l, BNP 522.9 pg/ml, D-Dimer 4.19 mg/l. ECG still showed extensive anterior ST-segment elevation myocardial infarction with Q waves, and new left anterior fascicular block and accelerated junctional rhythm (Fig. 1).

During hospitalization, the patient received high-flow oxygen, aspirin plus ticagrelor, atorvastatin plus ezetimibe, diuretics, gastric protection, ventricular remodeling therapy, and rate control. He still had dyspnea but was hemodynamically stable. On day 7 post-infarction, under intra-aortic balloon pump (IABP) support, repeat coronary angiography showed 95% stenosis in the proximal LAD with suspected dissection. Two stents were implanted in the proximal LAD (Fig. 2). The next day, the patient's dyspnea did not worsen, but a harsh 3-4/6 holosystolic murmur was heard at the left sternal border. Repeat TTE revealed several interruptions in the apical septum near the anterior wall; one interruption appeared to communicate with the right ventricle through a channel approximately 0.5 cm wide and 2.1 cm long, with left-to-right shunt, peak velocity 3.8 m/s, and gradient 56 mmHg. This was considered an apical septal intramural dissection with rupture (complex septal rupture) (Fig. 3).

The patient was transferred to the cardiac surgery department for septal repair. Follow-up TTE showed global cardiac enlargement and moderate-to-severe tricuspid regurgitation, with otherwise similar findings. While awaiting surgery, the patient still had dyspnea. On day 25 post-infarction, his condition deteriorated acutely with worsening heart failure, cardiogenic shock, oliguria, and acute respiratory

failure, and he was transferred to the cardiac surgical intensive care unit. The next day, dyspnea did not improve, and acute liver failure developed. Emergency IABP and biventricular assist device (BiVAD) support were instituted. Post-operatively, the patient developed acute cerebral infarction, and the family ultimately withdrew further treatment.

Discussion

Intramural dissecting hematoma is also referred to as myocardial dissection or intramural hematoma. IDH is strictly confined between the spirally arranged myocardial fibers and does not communicate with the ventricular cavity or pericardial space; it may gradually resolve or form a thrombus [4]. When IDH extends further and disrupts adjacent structures, it may present as an open dissection or mural thrombus, ventricular outflow tract obstruction, free wall rupture, ventricular septal perforation, pseudoaneurysm formation, or chordal rupture [5–8]. Therefore, IDH is also considered an early stage of various mechanical complications after myocardial infarction. Its pathogenesis is complex and is related to increased vascular fragility in the infarct zone, decreased myocardial contractility and tension, sustained elevation of intraventricular pressure, microvascular obstruction or spasm, reperfusion injury, and the use of antiplatelet drugs [9,10].

The diagnosis of IDH remains challenging. TTE is the preferred non-invasive diagnostic examination for most IDH patients, especially those who are hemodynamically unstable. Close follow-up with TTE helps to assess the evolution of IDH and guide treatment strategies. Contrast echocardiography can help determine the size of the hematoma and its relationship with surrounding tissues, thereby improving diagnostic accuracy. However, because echocardiography is limited by acoustic windows and operator expertise, cardiac magnetic resonance, surgical exploration, or autopsy remain the gold standard for diagnosing IDH. Multimodality imaging contributes to the diagnosis and differential diagnosis of IDH [11]. In addition, some IDH patients may exhibit conduction blocks, persistent ST-segment elevation, or new ST-segment elevation on electrocardiography [12]; careful repeated ECG monitoring combined with the patient's symptoms and signs helps in early detection of IDH. The ECG of our patient also showed persistent ST-segment elevation with left anterior fascicular block, which is consistent with the above electrocardiographic changes.

Treatment of IDH is influenced by multiple factors, including IDH anatomy, cardiac function, presence of other mechanical complications, and hemodynamic status. Current evidence on the treatment and prognosis of post-infarction IDH is limited, and mortality remains high regardless of conservative or surgical management. In clinical practice, a multidisciplinary team approach is usually required to formulate individualized treatment plans. For patients with localized IDH and stable hemodynamics, conservative treatment is a feasible option, but it should be conducted under strict imaging follow-up. Critically ill IDH patients often present with cardiogenic shock, refractory acute heart failure, or even multiple organ failure; such circumstances often force the clinical team to perform urgent surgical repair under mechanical circulatory support to restore cardiac hemodynamic stability as soon as possible. The

particularity of our patient is that, despite having a septal perforation, his clinical symptoms did not worsen, and hemodynamics remained relatively stable.

The optimal timing of surgery for such patients remains controversial. In the early phase after myocardial infarction, inflammatory oedema is prominent, and proteolytic enzyme activity peaks at 3–4 days after infarction, at which time the myocardial tissue is more fragile; at about 1 week after infarction, myofibroblasts proliferate extensively and collagen deposition reaches a peak; complete scar maturation and mechanical remodeling require several months [13]. Therefore, the choice of surgical timing presents a dilemma: early repair may relieve the shunt in time, but the risk of residual shunt and perioperative mortality is higher; delayed surgery, on the other hand, carries the risk of progressive hemodynamic deterioration and organ failure [14]. Data from the Society of Thoracic Surgeons National Database show that the mortality rate for patients undergoing surgical repair of post-infarction ventricular septal perforation within 7 days is 54.1%, compared with 18.4% after 7 days [15]. Di Bella et al. reported two patients with septal IDH and multiple small perforations with left-to-right shunts, who underwent surgical repair on days 28 and 32 after infarction, respectively, and both were discharged uneventfully. However, unlike our case, neither of those patients had clinical symptoms of heart failure [16]. In our case, elective surgery was planned at 4 weeks after onset, based on two main considerations: first, to allow resolution of myocardial inflammation and oedema; second, the patient's hemodynamics remained relatively stable and his dyspnea did not progressively worsen. Nevertheless, it is worth reflecting that although the patient's vital signs were maintained under medical support, his dyspnea persisted, and follow-up echocardiography revealed progressive global cardiac enlargement and worsening tricuspid regurgitation. This suggests that earlier application of MCS might have helped to delay heart failure progression and gain time for surgery.

Commonly used MCS modalities include IABP, extracorporeal membrane oxygenation (ECMO), and ventricular assist devices (VAD). Although the IABP-SHOCK II trial showed that IABP use did not reduce mortality in patients with acute myocardial infarction complicated by cardiogenic shock [17], IABP remains the most widely used temporary circulatory support due to its relatively low complication rate and ease of use [18]. IABP increases diastolic coronary blood flow and reduces left ventricular afterload; when combined with veno-arterial extracorporeal membrane oxygenation (VA-ECMO), it can compensate for the increased left ventricular afterload caused by VA-ECMO. Importantly, in rapidly progressing circulatory failure, the combination of peripheral ECMO and Impella (ECpella) is associated with lower mortality than ECMO alone or ECMO combined with IABP [19,20]. For patients with post-infarction ventricular septal perforation, ECpella may be especially suitable. VA-ECMO alone can provide potent circulatory support, but the increased left ventricular afterload may aggravate the left-to-right shunt and pulmonary congestion; Impella can actively reduce left ventricular pressure and volume, effectively decreasing shunt flow, but in the presence of right ventricular dysfunction or a large septal defect, it may induce a right-to-left shunt and decrease oxygen saturation [21]. The combination of the two can provide comprehensive support via VA-ECMO and left ventricular unloading via Impella, synergistically controlling the shunt and stabilizing haemodynamics.

MCS can effectively stabilize hemodynamics and provide critical support for a failing circulation. However, these supportive measures are also accompanied by non-negligible complications. Bleeding, infection, hemolysis, thrombosis, and limb ischemia are common complications [20].

Conclusion

In summary, ventricular septal IDH with perforation after acute myocardial infarction is a rare and devastating mechanical complication. Early recognition, appropriate timing of intervention, and adequate use of mechanical circulatory support are key to improving prognosis. Further prospective randomized clinical trial evidence is needed to define the optimal treatment strategy and surgical timing.

Abbreviations

IDH intramural dissecting hematoma

TNI troponin I

NT-proBNP N-terminal pro-B-type natriuretic peptide

ECG electrocardiogram

TTE transthoracic echocardiography

LAD left anterior descending branch

IABP intra-aortic balloon pump

BiVAD biventricular assist device

ECMO extracorporeal membrane oxygenation

VAD ventricular assist devices

VA-ECMO veno-arterial extracorporeal membrane oxygenation

ECpella ECMO and Impella

Declarations

Ethics approval and consent to participate

According to institutional policy, formal ethics approval was not required for this single case report.

Consent for publication

Written informed consent for the publication of this case report was obtained from the patient's son.

Availability of data and materials

The datasets generated and/or analyzed during the current study are not publicly available due to patient privacy and confidentiality, but de-identified data are available from the corresponding author on reasonable request.

Competing Interests

The authors declare no competing interests.

Founding

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Authors' contributions

Jie Chen: Data curation, Investigation, Visualization, Writing.

Shan Deng: Conceptualization, Supervision, review and editing.

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Not applicable.

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Figures

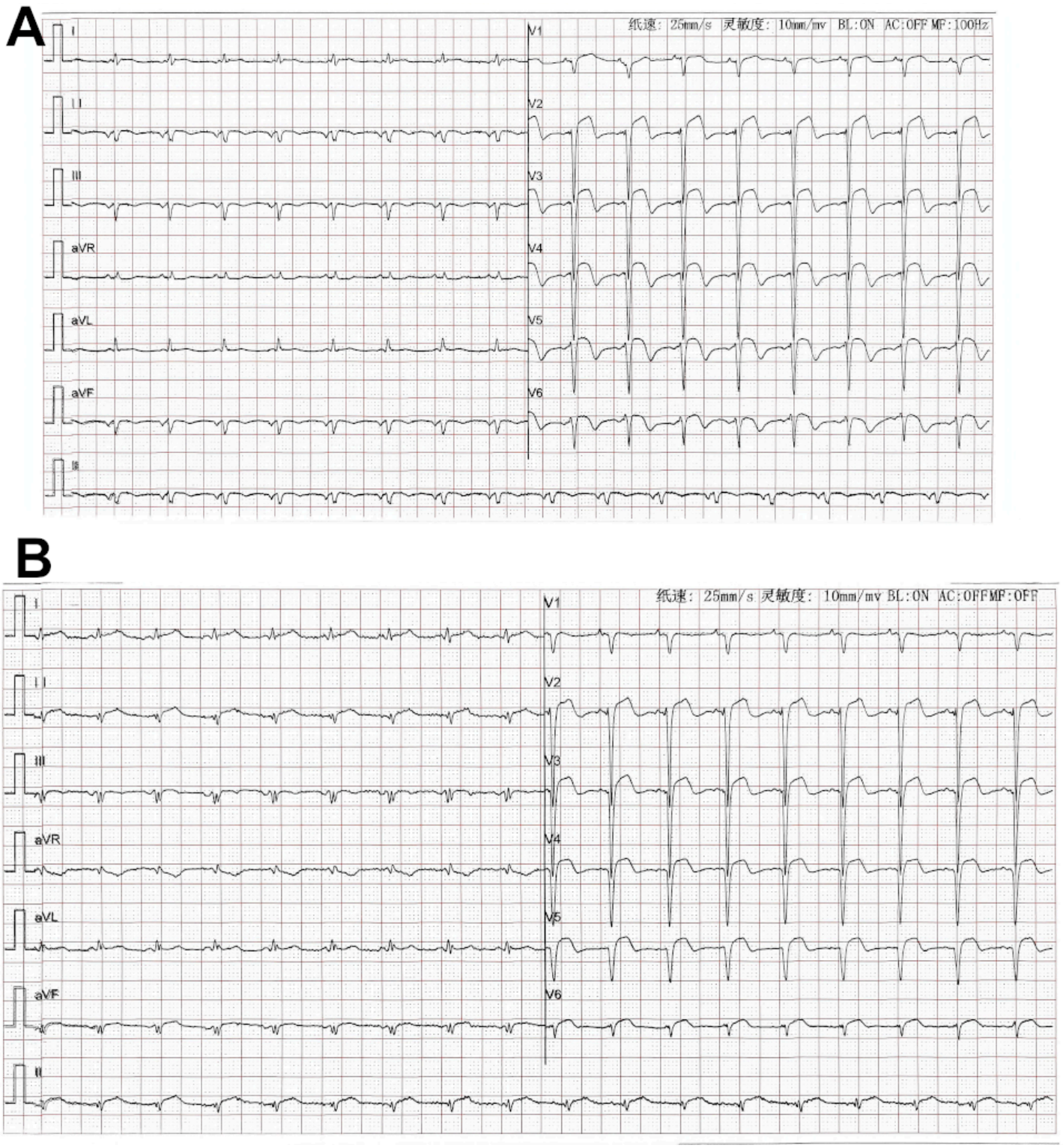


Figure 1

Electrocardiograms of the patient. **(A)** ECG on day 1 after symptom onset: extensive anterior leads (V1–V6) ST-segment elevation, pathological Q waves, new-onset left anterior fascicular block, and accelerated junctional rhythm. **(B)** Follow-up ECG on day 20 after symptom onset: the above-mentioned ST-segment elevation persisted.

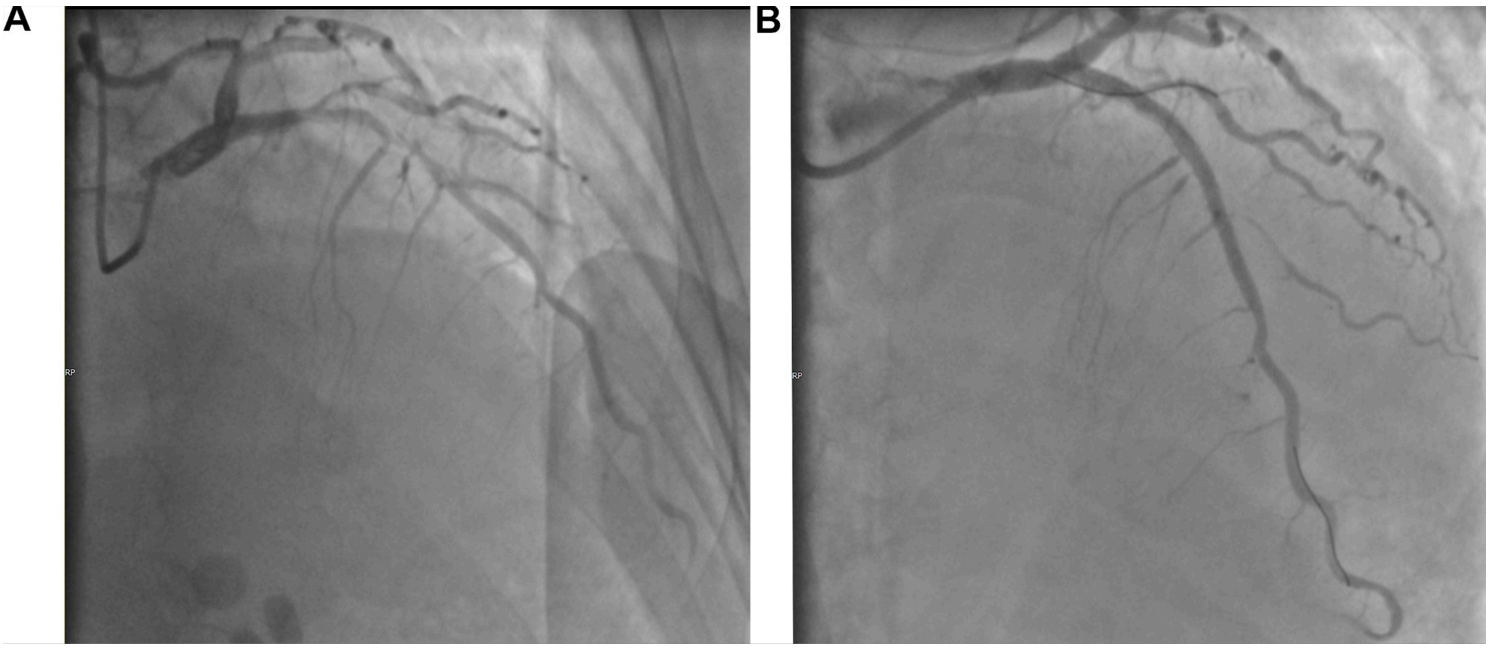


Figure 2

Follow-up coronary angiography results. **(A)** Angiography showing 95% stenosis in the LAD with suspected dissection. **(B)** After implantation of two stents in the proximal LAD, the stenosis was relieved and distal flow was restored.

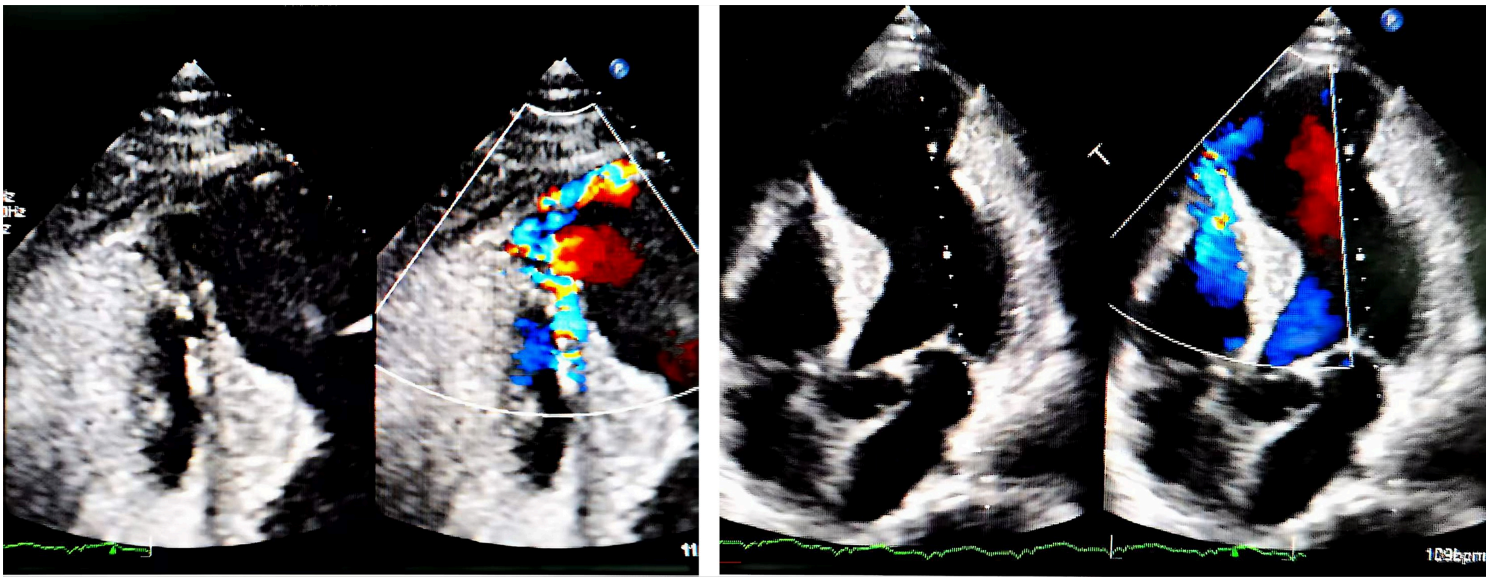


Figure 3

Transthoracic echocardiography demonstrating apical septal intramural dissection with rupture (complex septal rupture).

Supplementary Files

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