Late Diagnosis of a Post-Infarction Ventricular Septal Defect: An Unusual Presentation

Abstract :

Ventricular septal defect (VSD) is a rare and serious complication of myocardial infarction, usually occurring within the first few days after the acute event. We report the case of a 78-year-old woman who sustained an anterior myocardial infarction in June 2024 and underwent percutaneous coronary intervention of the mid left anterior descending artery with drug-eluting stent implantation. In May 2025, nearly eleven months later, she was admitted with recurrent heart failure decompensation. The appearance of a previously absent systolic murmur prompted repeat echocardiography, which revealed a 17-mm muscular septo-apical VSD with a left-to-right shunt. Transesophageal echocardiography showed a pseudoaneurysm bounded by a membrane and a tortuous course of the defect, an anatomy that may explain the relatively good tolerance of this large defect. Myocardial scintigraphy confirmed septo-apical necrosis. The patient ultimately underwent surgical exclusion of the apical aneurysm and VSD closure with a reinforced pericardial patch. This case shows that a post-infarction VSD may present very late and highlights the importance of considering a mechanical complication when a new murmur or otherwise unexplained heart failure decompensation occurs, even several months after myocardialinfarction.

Introduction

Post-infarction ventricular septal defect (VSD) has become rare since the widespread use of early coronary revascularization. Its current incidence is estimated at 0.1–0.4%, yet it remains a severe complication associated with high mortality despite advances in management [1]. It most often occurs within the first few days after myocardial infarction and typically presents with clinical deterioration or a new systolic murmur. Echocardiography then allows rapid confirmation of the diagnosis. In contrast, a VSD that remains unrecognized for several months is far more unusual. We report the case of a patient who sustained an anterior myocardial infarction treated by angioplasty and subsequently experienced several episodes of heart failure. The post-infarction apical VSD was ultimately diagnosed approximately eleven months after the initial event, during a further episode of heart failure decompensation.

Case Presentation

A 78-year-old woman with hypertension, obesity, and dyslipidemia was being followed for ischemic heart disease. In June 2024, she sustained an anterior acute coronary syndrome due to a subtotal occlusion of the mid left anterior descending artery (LAD), treated by percutaneous coronary intervention with implantation of a drug-eluting stent. Her initial course was uncomplicated apart from mild congestion requiring diuretic therapy. In November 2024, she was readmitted with heart failure decompensation, precipitated by dietary indiscretion in the setting of COVID-19 infection. Remote monitoring wasinitiated after discharge.

In May 2025, weight gain detected through remote monitoring prompted a new cardiology assessment. The patient had clinical and echocardiographic signs of congestion. The left ventricle was non-dilated and hypertrophied, with septo-apical akinesia and preserved global systolic function; the left ventricular ejection fraction was 58%. The left atrium was at the upper limit of normal, with an indexed volume of 33 mL/m², while an E/E′ ratio of 14 suggested elevated filling pressures. The right-sided chambers were not dilated. Peak tricuspid regurgitation velocity was 3.75 m/s, corresponding to an estimated pulmonary artery systolic pressure of 66 mmHg (a right ventricular–right atrial gradient of 56 mmHg plus an estimated right atrial pressure of 10 mmHg based on a dilated, poorly collapsible inferior vena cava). The inferior vena cava measured 27 mm and showed reduced respiratory variation. There was no significant mitral or aortic valve disease.

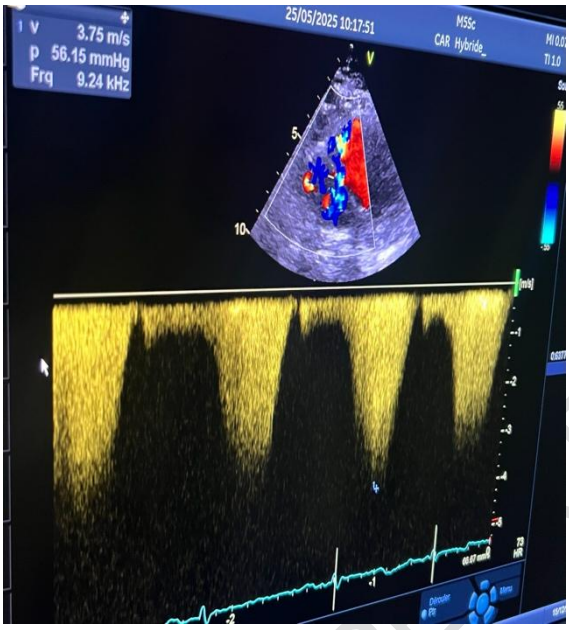

Figure 1: Transthoracic echocardiography with continuous-wave Doppler of tricuspid regurgitation showing a peak velocity of 3.75 m/s

Laboratory testing showed a hemoglobin level of 12.5 g/dL, a white blood cell count of 6.7 G/L, and a C-reactive protein level of 2 mg/L. NT-proBNP was elevated at 1,711 ng/L. Serum creatinine was 85 µmol/L, corresponding to an estimated glomerular filtration rate of 57 mL/min/1.73 m². Serum sodium was 139 mmol/L and potassium 4.1 mmol/L. Iron studies showed a ferritin level of 49 ng/mL and a transferrin saturation of 12%.

These findings led to admission for global heart failure decompensation. During the hospital stay, auscultation revealed a new systolic murmur that had not been present on admission. Given her history of anterior myocardial infarction, this late finding prompted investigation for a mechanical complication.

Repeat transthoracic echocardiography provided the key diagnostic finding. It showed a 17-mm muscular septo-apical ventricular septal defect with a left-to-right shunt and a peak gradient of 68 mmHg.

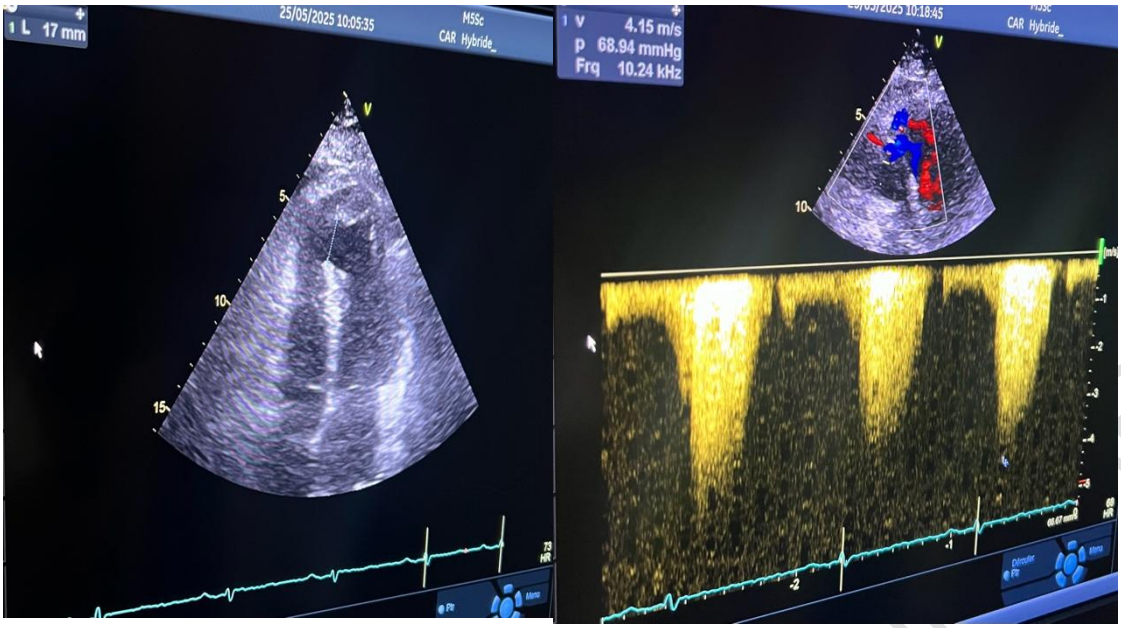

Figure 2: Transthoracic echocardiography showing a 17-mm muscular septo-apical ventricular septal defect with a left-to-right shunt and a peak continuous-wave Doppler gradient of 68 mmHg

Transesophageal echocardiography provided a more precise understanding of this unusual anatomy. It confirmed the septo-apical VSD and demonstrated a pseudoaneurysm bounded by a membrane, resulting in a tortuous course of the defect. This configuration may have contributed to the persistence of a high gradient despite the anatomically large defect. Myocardial scintigraphy also demonstrated septo-apical necrosis, consistent with the regional akinesia and the location of the mechanical complication.

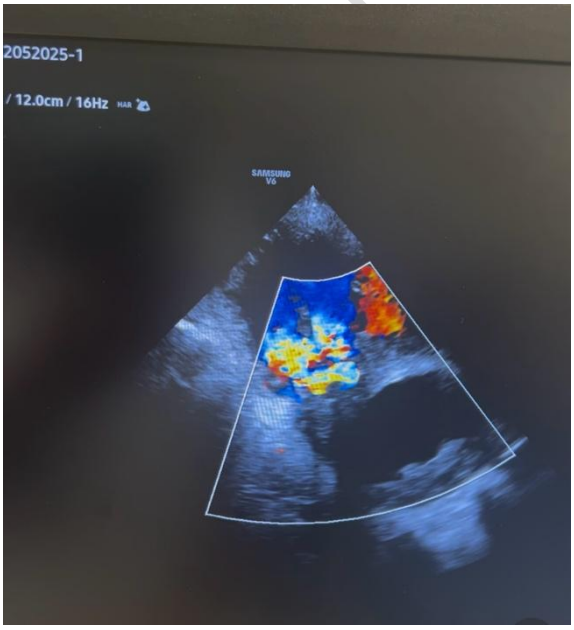

Figure 3: Transesophageal echocardiography showing the septo-apical VSD associated with a pseudoaneurysm bounded by a membrane, resulting in a tortuous course of the shunt on color Doppler

The patient was transferred to Brabois University Hospital for specialized management. Preoperative coronary angiography showed no restenosis of the mid-LAD drug-eluting stent. Following multidisciplinary discussion, surgical treatment was selected. The procedure combined surgical exclusion of the apical ventricular aneurysm with closure of the VSD using a reinforced pericardial patch. The postoperative course was uncomplicated, with complete closure of the VSD and no residual shunt.

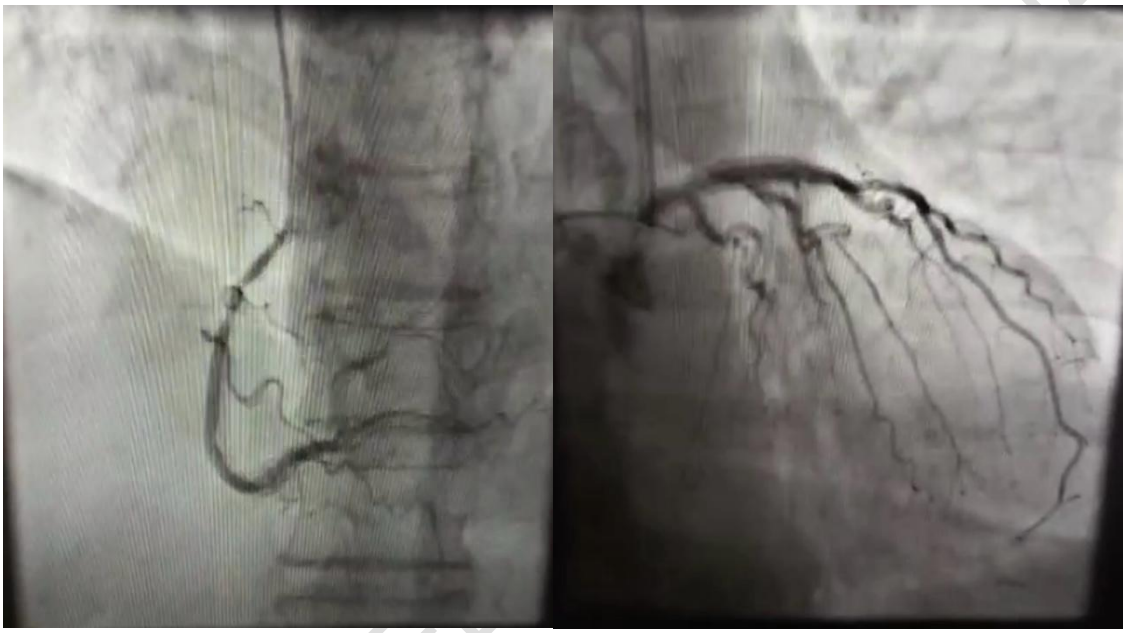

Figure 4: Preoperative coronary angiography showing patency of the left anterior descending artery stent

Discussion

Delayed presentations combining a VSD and a pseudoaneurysm have nevertheless been reported. Shimono et al. described this association in a 77-year-old woman after anterior myocardial infarction [2], while Yerekesh et al. reported an apical pseudoaneurysm associated with a 12-mm VSD discovered two months after myocardial infarction [3]. Melina et al. also reported septal rupture associated with a contained pseudoaneurysmal cavity [4].

The main distinctive feature of our case is its anatomy. Despite a large 17-mm septo-apical defect, the interventricular gradient remained high at 68 mmHg. Transesophageal echocardiography demonstrated a pseudoaneurysm bounded by a membrane and a tortuous course of the communication. This configuration probably limited the shunt and may partly explain the relative clinical tolerance and delayed

diagnosis of the VSD. Such complex, serpiginous morphologies have been described in post-infarction septal rupture. Transthoracic and transesophageal echocardiography were complementary in defining the anatomy, while myocardial scintigraphy demonstrated septo-apical necrosis and confirmed the concordance between the infarcted territory, regional akinesia, and the mechanical complication.

Despite this relative stability, definitive treatment remained necessary. In the multicenter study by Sánchez Vega et al., mortality was 91.5% with conservative treatment compared with 52.3% after surgery [5]. The optimal timing of repair remains debated and depends particularly on hemodynamic status and whether repair can be delayed to allow consolidation of infarcted tissue [6–8]. This approach is also reflected in the European survey by Ronco et al., in which surgery was preferred by 71.8% of centers and delayed repair by 87.2% when the clinical condition allowed it [6].

In our patient, the combination of a complex apical VSD and a pseudoaneurysm led to surgical exclusion of the aneurysm and closure of the VSD with a reinforced pericardial patch, an approach comparable to that reported in similar cases [2,4]. This case mainly emphasizes that a post-infarction VSD may exceptionally be diagnosed several months after the initial event, particularly when complex anatomy limits its hemodynamic impact. The appearance of a new murmur in a patient with a history of myocardial infarction should therefore prompt investigation for a mechanical complication, even long after the acute event.

Conclusion

This case illustrates an unusual presentation of a post-infarction VSD diagnosed nearly one year after the initial event. Its association with a pseudoaneurysm and the tortuous course of the defect probably contributed to its relative tolerance despite the size of the defect. A new murmur or otherwise unexplained heart failure decompensation in a patient with a history of myocardial infarction should prompt investigation for a mechanical complication, even several months later. Once the diagnosis is established, surgery remains a cornerstone of management and, as in our patient, can address both the VSD and the pseudoaneurysm during the same procedure [9].

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