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CASE REPORT

Concomitant ventricular septal rupture and interventricular septal aneurysm in neglected inferior myocardial infarction misdiagnosed with congenital ventricular septal defect: A case report

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Abstract

This study emphasizes that VSD should not be immediately diagnosed as a congenital disorder; instead, regional wall motion abnormalities in the left ventricle should also be taken into account since it may result from mechanical complications of neglected myocardial infarction.

KEYWORDS

interventricular septal aneurysm, mechanical complication, myocardial infarction, ventricular septal defect, ventricular septal rupture

1 | INTRODUCTION

This case report demonstrates the concurrence of two rare complications of neglected inferior ST-elevation myocardial infarction in a poorly controlled diabetic patient with acutely decompensated heart failure, mimicking a congenital ventricular septal defect. The existence of two concurrent MI-induced mechanical complications with a relatively non-fulminant course is rare.

Mechanical complications following acute myocardial infarction (MI) are rare, accounting for less than 1% of total cases. The prevalence of such complications is higher in patients with cardiogenic shock (3.9%).[1]Although timely reperfusion therapy can dramatically decrease the incidence of these complications, the occurrence of these complications is associated with poor clinical outcomes.[2]

In the current case report, we aimed to present an infrequently reported case of neglected inferior MI concurrently afflicted with interventricular septal aneurysm and ventricular septal rupture (VSR).

2 | CASE PRESENTATION

A 62-year-old man with congenital ventricular septal defect (VSD) and the clinical manifestations of acutely decompensated heart failure (ADCHF) was admitted to the Emergency Department. The patient had signs and symptoms of left ventricular (LV) and right ventricular (RV) failure, comprising orthopnea, paroxysmal nocturnal dyspnea, dyspnea on exertion, and symmetric edema of both lower extremities of 4 weeks duration with a relatively progressive, albeit non-fulminant, course. His symptoms had exacerbated one week before hospital admission. He had a history of uncontrolled diabetes mellitus, hypertension, dyslipidemia, and paroxysmal atrial fibrillation, together

with a family history of premature coronary artery disease in his younger brother. The patient had no complaints of chest pain, or a history of MI or coronary artery disease.

The patient had been admitted to another hospital due to COVID-19 infection three months prior to his current hospitalization. In the medical workup performed in that center, congenital VSD was diagnosed as an incidental finding during the evaluation of the cardiac complications of COVID-19.

On presentation to our hospital the patient was significantly dyspneic, NYHA class IV, tachycardia with heart rate 105 bpm, blood pressure 110/60 mmHg. Physical examination was remarkable for a holosystolic murmur pronounced at the left sternal border, elevated JVP, bilateral basilar crackles in the lungs, and symmetric edema in the lower extremities.

The laboratory tests of the patient were unremarkable except the N-terminal pro–B-type natriuretic peptide (NT-proBNP), which was highly elevated (3622 pg/ml, a pre-defined normal value based on his age and sex being <210 pg/ml) as well as a high hemoglobin A1c(HbA1c) with a percentage of 11.3%. The troponin level was within the normal range. Electrocardiography (ECG) revealed a sinus rhythm with a rightward axis deviation in the QRS vector, an incomplete right bundle branch block, a mild ST-segment elevation, a T-wave inversion in the inferior leads, and a mild ST-depression in the precordial leads (Figure 1).

In the Emergency Department, a spiral chest computed tomography (CT) scan without intravenous contrast was performed to rule out pulmonary causes of dyspnea in the patient. The modality demonstrated cardiomegaly, congested lungs, moderate right-sided pleural effusion, and ascites as an incidental finding. A cardiac CT scan and cardiovascular magnetic resonance (CMR) were prescribed for the patient, and he was asked to bring the results to the next visit. Transthoracic echocardiography was then conducted, which showed moderate LV enlargement, moderate LV systolic dysfunction (ejection fraction = 40%), significant regional wall motion abnormalities in the inferior and inferoseptal walls, and aneurysmal formation in the base and mid-inferoseptal segments of the LV, creating an intraventricular pulsatile globular structure (56 × 49 mm) at the RV cavity with paradoxical systolic expansion. This structure was connected to both the LV cavity with a wide neck (21 mm) and the RV cavity through multiple small perforations with a systolic pressure gradient of 60 mmHg (Figure 2,Videos S1-S5). The RV was moderately dilated with moderate-to-severe systolic dysfunction and moderate-to-severe tricuspid regurgitation. The estimated systolic pulmonary artery pressure was 70 mm Hg. There was a unidirectional left-to-right shunt, at a QP/QS ratio of 1.95, through small perforations in this pulsatile structure into the RV chamber. A schematic view of the interventricular septal aneurysm and VSR is depicted in Figure 3. For better evaluation of these complications and ruling out other concurrent congenital anomalies, transesophageal echocardiography was performed. The modality failed to provide complementary

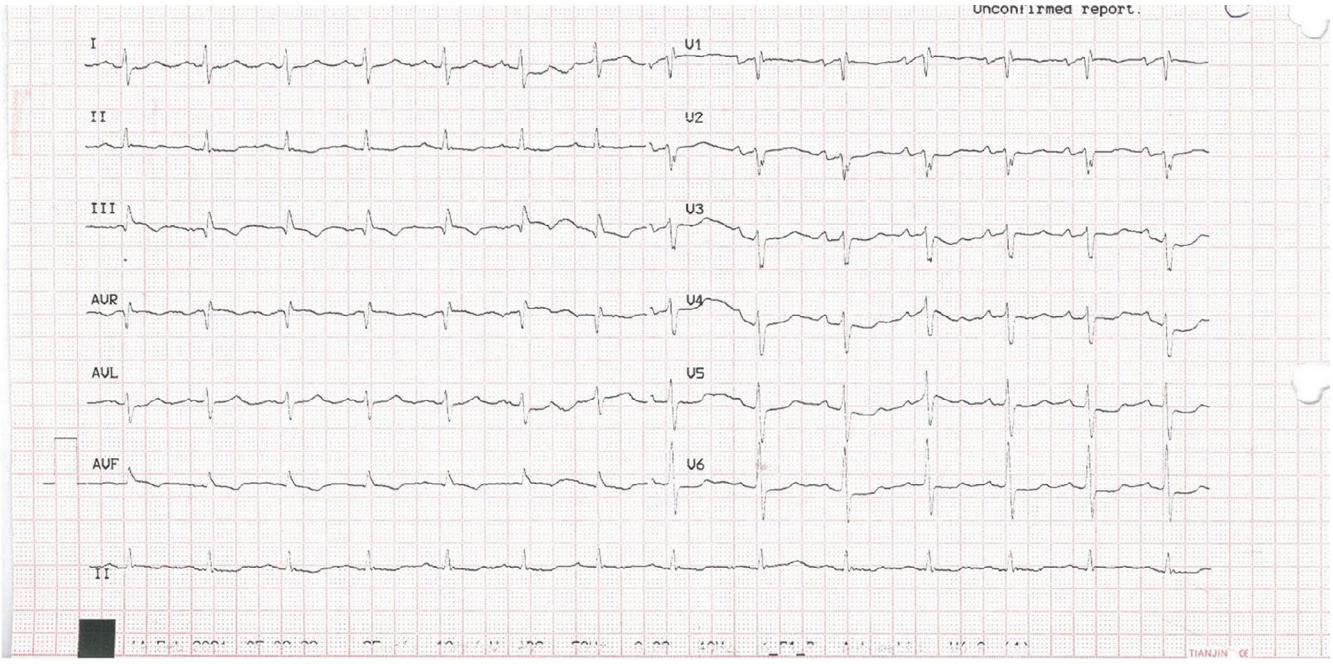

FIGURE 1 Image depicts the electrocardiography of the patient. ECG showing Sinus rhythm, RAD, Incomplete RBBB, mild ST-segment elevation and T-wave inversion in the inferior leads, mild ST-depression in the precordial leads. ECG, electrocardiogram; RAD, right axis deviation

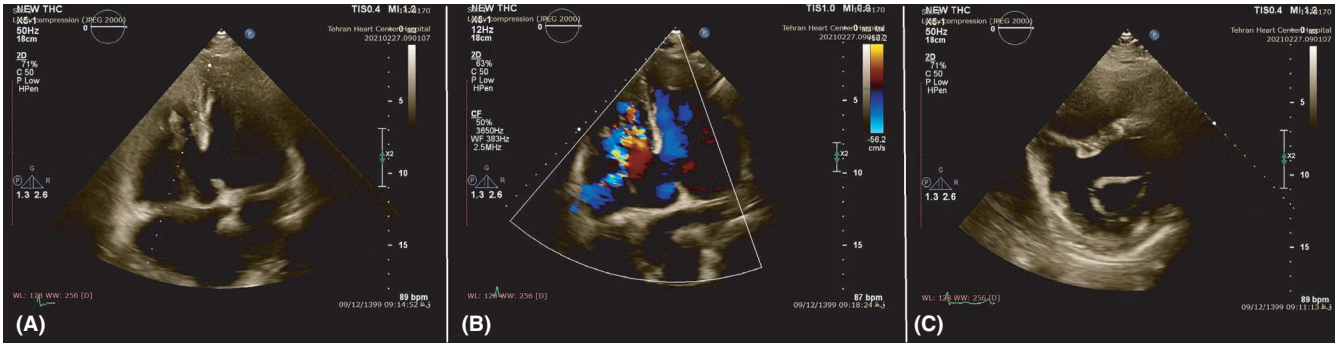

FIGURE 2 Images present echocardiographic findings. (A) Apical 4-chamber view showing the aneurysm with ventricular septal rupture (VSR). (B) Apical 4-chamber view with Color-Doppler showing flow across VSR. (C) Parasternal short-axis view showing the aneurysm

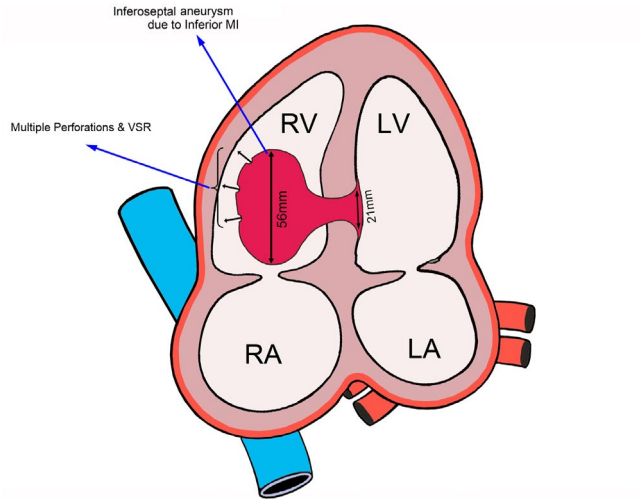

FIGURE 3 Image illustrates a schematic view of the interventricular septal aneurysm, multiple perforations (VSR). VSR, ventricular septal rupture

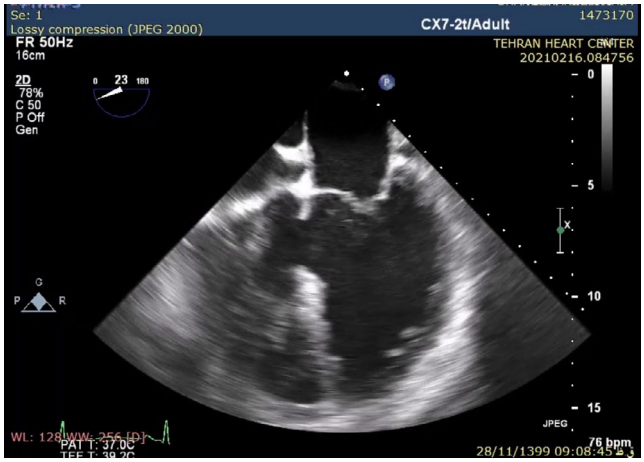

FIGURE 4 Transesophageal echocardiogram image demonstrates interventricular septal aneurysm

information and merely confirmed the results of the transthoracic echocardiography (Figure 4, Videos S6-S7).

Given the patient's diabetes mellitus, ECG abnormalities, regional wall motion abnormalities seen via echocardiography and reduced LV systolic function, coronary angiography was conducted on the patient. It revealed significant left main coronary artery stenosis and significant triple vessel disease. Additionally, the left circumflex artery was a non-dominant vessel, while the dominant right coronary artery was cut from the mid-part without antegrade and retrograde runoff (Figure 5). In addition to showing an ejection fraction of 40%, LV injection demonstrated the opacification of the LV, followed by the opacification of an aneurysm in the interventricular septum (IVS) and subsequently the RV (Figure 6, video S8). Our findings indicated the concurrence of two rare mechanical complications, namely septal aneurysm, and post-MI VSR, in a poorly controlled diabetic patient who had a

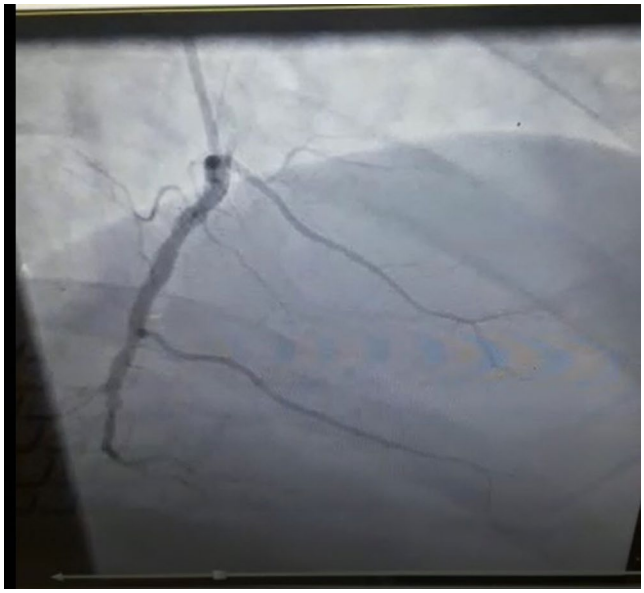

FIGURE 5 Coronary angiography of the patient demonstrates that the right coronary artery is cut off in the mid-part (arrowhead)

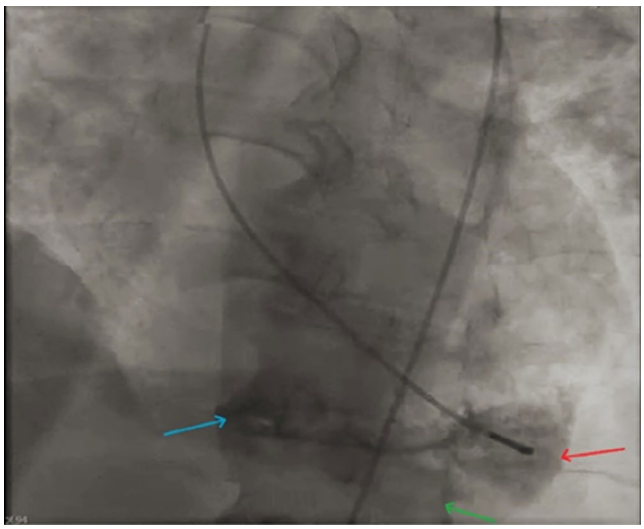

FIGURE 6 Left ventricular angiography; the LAO cranial view (20°* 20) shows the opacification of LV (red arrow), followed by the opacification of IVS aneurysm (blue arrow) and subsequently the RV (green arrow). LV, left ventricle; IVS, interventricular septum; RV, right ventricle

possible neglected inferior MI and was misdiagnosed with congenital VSD.

The patient was admitted to the coronary care unit, where he received heart failure treatments and intravenous diuretic and vasodilator therapies. We recommended coronary artery bypass graft surgery (CABG), along with aneurysmectomy and ventricular septal repair. Unfortunately, the patient refused to undergo surgery.

3 | DISCUSSION

Many patients with severe ischemic heart disease never experience angina pectoris. Episodes of silent ischemia are more prevalent in patients with diabetes mellitus than non-diabetics. Mechanical complications following an acute MI are rare, accounting for less than 1% of total cases and more prevalent in patients with cardiogenic shocks (3.9%).[1] LV aneurysms account for the most common mechanical complication in ST-segment elevation myocardial infarction (STEMI). True LV aneurysms have been reported to develop in less than 5% of all patients. Total occlusions in a poorly collateralized infarct-related artery are associated with aneurysm formation following the development of STEMI. Aneurysms occur approximately four times more often at the apex and anterior wall than the inferoposterior wall.[3]

Ventricular septal rupture is another fatal mechanical complication of acute MI. Post-infarction VSR is more common in anterior STEMI (60%), located in the anterior or apical portion of the IVS, as compared to inferoposterior STEMI (20%–40%), and located in the posterior or inferior portion of the IVS. Clinical features associated with increased risk for rupture of the IVS include lack of development of a collateral network, advanced age, female sex, and chronic kidney disease.[4]

Early reperfusion strategies have significantly reduced the rate of cardiac ruptures, including VSR, following acute MI during the recent years.[2] Although reperfusion therapy has dramatically decreased the incidence of these complications, the occurrence of these complications is still associated with poor outcomes and high mortality rates. For instance, the mortality rate of post-MI VSR, despite surgical treatments, ranges between 40% and 75%.[1] The diagnosis of these complications is vital as it significantly determines the management approach of patients regarding medical or surgical treatments. The mechanical complications of STEMI often have fulminant and catastrophic manifestations in patients, and the existence of two concurrent MI-induced mechanical complications and a relatively non-fulminant course is rare. To the best of our knowledge, the concurrence of an interventricular septal aneurysm and VSR following an acute MI has been previously described in only three case reports.

The first case, described by Zhang et al.[5] was a 65-year-old woman with a history of progressive dyspnea one month after inferior STEMI. She had not received reperfusion therapy due to the late presentation, and her echocardiographic findings revealed a large inferoposterior aneurysm connected to the LV cavity via a wide neck and a systolic shunt flow from the disrupted aperture of the aneurysm at the vicinity of the apex of the RV. The patient underwent combined CABG and LV ventriculoplasty, with good recovery.

The second case, described by Shafi et al.[6] was a 49-year-old man with a recent inferior STEMI and percutaneous coronary intervention on the right coronary artery. Two weeks after the occurrence of a MI, the patient was admitted to hospital with ADCHF (New York Heart Association [NYHA] functional class IV). Echocardiography showed a large inferoseptal wall aneurysm with VSR at the apical side of the aneurysm with a left-to-right shunt. He underwent VSR repair and left ventriculoplasty, which led to a good postoperative outcome.

The third case, described by Khanal et al.[7] was a 61-year-old man who was diagnosed with a inferior wall myocardial infarction about a month ago, did not undergo thrombolysis, and was on optimal medical therapy. One month after the acute MI episode, he was admitted to hospital with ADCHF. Echocardiography indicated a giant true posterobasal aneurysm and VSR. The patient underwent successful CABG and ventriculoplasty along with VSR patch repair and had a good clinical outcome after surgery.

Unlike the 3 cases mentioned above, where all had a clear history of MI; our patient did not have an obvious episode of MI; hence, he was misdiagnosed with congenital VSD.

The marked point in our patient, in comparison with the cases mentioned earlier, was that he had VSR that originated from multiple perforations in the wall of the IVS aneurysm, whereas there was only one perforation in the three cases.

The scintillating point in our diabetic patient who had no apparent symptoms of the acute coronary syndrome (probably neglected inferior MI) was the concurrent two rare post-MI mechanical complications, namely VSR and an IVS aneurysm, which mimicked the congenital VSD. As mentioned earlier, the patient was admitted to hospital as a result of infection with COVID-19, and he underwent echocardiography to rule out the cardiac complications caused by SARS-CoV-2 infection. The cardiologist in that hospital misdiagnosed the wide neck (21 mm) of a true aneurysm as congenital VSD. This misdiagnosis increases the importance of differentiation between infarct-related VSR and congenital VSD. In view of the patient's age (62 years) and the onset of symptoms at this age, the presence of congenital VSD in this patient was unlikely, as the onset of symptoms in congenital VSD is usually in younger ages. In addition, the abnormalities in the LV regional wall motion and angiographic findings further supported the diagnosis of infarct-related VSR.

A neglected inferior MI and lack of reperfusion therapy due to the asymptomatic nature of the infarction led to the emergence of post-MI mechanical complications in the patient.

Although CMR was the preferred non-invasive diagnostic modality for the patient to accurately diagnose the disease causality, unfortunately, our patient refused to undergo complementary diagnostic procedures; however, CMR should be considered a preferred diagnostic modality after initial diagnostic evaluation.

4 | CONCLUSIONS

Neglected MI deprives patients of reperfusion therapy and has two critical consequences. First, it increases the incidence of post-MI mechanical complications, such as VSR. Second, the absence of a clear history of MI in these patients can erroneously lead to a diagnosis of congenital disease instead of MI-related VSR.

Therefore, in adult patients with VSD, we recommend that adequate attention be paid to other clinical findings in echocardiography, ECG, and other diagnostic procedures.

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CONFLICTS OF INTEREST

The authors declare no conflict of interests. None of the authors is employed by a government agency and has any primary function other than research and/or education. None of the authors is submitting this manuscript as an official representative or on behalf of the government.

AUTHOR CONTRIBUTIONS

Tahereh Davarpasand, Seyedeh Hamideh Mortazavi, and Atefeh Lalvand: Drafting of the article. Tahereh Davarpasand, Reza Mohseni-Badalabadi, Mohammad Sadeghian, and Atefeh Lalvand: Critical revision of the article for important intellectual content. Tahereh Davarpasand, Reza Mohseni-Badalabadi, Mohammad Sadeghian, Seyedeh Hamideh Mortazavi, and Atefeh Lalvand: Final approval of the article. Reza Mohseni-Badalabadi, Mohammad Sadeghian, Seyedeh Hamideh Mortazavi, Atefeh Lalvand: Provision of study materials or patients. Reza Mohseni-Badalabadi, Mohammad Sadeghian, Seyedeh Hamideh Mortazavi, and Atefeh Lalvand: Administrative, technical, or logistic support. Tahereh Davarpasand and Atefeh Lalvand: Collection and assembly of data.

CONSENT

Informed written consent was obtained from the patient for the publication of the case report.

DATA AVAILABILITY STATEMENT

The datasets used during the study could be provided by the corresponding author on a reasonable request.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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