

Ventricular septal rupture presented with chronic heart failure symptoms: a case report

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Background

Ventricular septal rupture (VSR) is a rare mechanical complication following acute myocardial infarction, with very high mortality rate. Haemodynamic deterioration and cardiogenic shock is common in such cases. Rarely, however, patients may show only signs of chronic heart failure or be completely asymptomatic.

Case summary

We report a case of a 72-year-old male patient with VSR following a transmural myocardial infarction. He sought medical attention only after gradually experiencing symptoms of chronic heart failure, weeks after the onset of the myocardial infarction. The patient successfully underwent elective repair surgery, after optimizing the medical therapy and completing the necessary workup.

Discussion

Ventricular septal rupture repair is necessary in all cases due to the high mortality rate if left untreated. The timing of the operation, however, should be decided individually for every patient. Haemodynamically unstable patients may require early surgery, but in stable patients responding to medical treatment, delayed repair may be the best option.

Keywords

Ventricular septal rupture • Mechanical complications • Heart failure • Myocardial infarction • Case report

Learning points

- High clinical suspicion and thorough clinical examination at first medical contact can reveal rare cases of mechanical complications of myocardial infarction presented with mild symptoms.
- Although the decision of the timing for ventricular septal rupture repair is still under debate, data show that delayed intervention seems to have better outcome in patients who respond to medical therapy.

Introduction

Mechanical complications after acute myocardial infarction are very rare (<1% of cases). Those include papillary muscle rupture (0.26%), free wall rupture (0.52%), and ventricular septal rupture (VSR) (0.17%).¹ In most cases these complications, if not treated, can lead to cardiogenic shock with high mortality rates, thus they are considered situations of extreme urgency.

In this article, we present a case of a patient with late hospital arrival after myocardial infarction, complicated with VSR and symptoms of chronic heart failure.

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Timeline

August 2017	First presentation of fatigue and shortness of breath
Early October 2017	Lower limb oedema presentation
18 October 2017	Admission to the hospital—echocardiography reveals ventricular septal rupture (VSR)
22 October 2017	Right heart catheterization
25 October 2017	Coronary angiography reveals RCA occlusion
30 October 2017	New transthoracic echocardiogram shows pulmonary artery systolic pressure (PASP) improvement
4 November 2017	Transoesophageal echocardiogram reveals unfavourable anatomy for device closure
8 November 2017	Patient referred for surgical repair
14 November 2017	Successful repair of VSR
24 November 2017	Patient is discharged from the hospital
February 2018	3-month follow-up by the patient's local cardiologist—no symptoms or complaints
June 2018	6-month follow-up by the patient's local cardiologist—no symptoms or complaints

Case presentation

A 72-year-old male patient, smoker (47 pack-years), with no known medical history was presented with symptoms of fatigue, shortness of breath for the past 2 months (New York Heart Association functional Class III) and bilateral lower limb oedema since 2 weeks. His electrocardiogram (Figure 1) displayed sinus rhythm at 90 b.p.m. with left axis deviation, intraventricular conduction delay (QRS duration 118 ms) and QS morphology in leads III, aVF and V1–V5. His blood pressure and oxygen saturation were within normal range (135/85 mmHg and 96%, respectively). Lung and cardiac auscultation revealed absence of breath sounds in the right lower lobe and a harsh pansystolic murmur at the left lower sternal edge, respectively. The patient also had jugular vein distention. Chest X-ray displayed significant right-sided pleural effusion. Laboratory tests showed high proBNP (8904 pg/mL), slightly elevated troponin I (cTnI 0.083 ng/mL), mild anaemia (Hgb 12.2 g/dL, Hct 37.4%), and a normal renal function.

Transthoracic echocardiography demonstrated an aneurysm of the basal inferior and inferoseptal segments of the left ventricle (Figure 2A and B) as well as dilation and systolic impairment of the right ventricle with hypokinesia of its free wall. Colour Doppler revealed a shunt between the left and right ventricle through the aneurysmatic segment (Figure 2C) and moderate insufficiency of the atrioventricular valves. The left ventricular ejection fraction was 48% (via Simpson) and pulmonary artery systolic pressure was elevated (50 mmHg). Qp/Qs ratio was 2.4.

The patient was admitted to the coronary care unit and heart failure treatment with bisoprolol (2.5 mg q.d.), zofenopril (10 mg q.d.),

and furosemide (40 mg b.i.d.) was initiated and titrated. Delayed treatment of the septal rupture was decided due to his stable haemodynamic status. Right heart catheterization (Table 1) revealed elevated pulmonary artery and wedge pressures, whereas cardiac output was normal and Qp/Qs ratio was estimated 2.8. Coronary angiography uncovered proximal occlusion of the right coronary artery (Figure 3), while the left coronary circulation did not present severe stenosis. Right coronary artery intervention was not attempted due to the non-viability of the underlying infarcted myocardium. Acetylsalicylic acid (ASA) (100 mg q.d.) and atorvastatin (40 mg q.d.) were added.

Over the course of hospitalization, the patient's symptoms improved significantly. A transoesophageal echocardiogram was performed (Figure 4), which showed a 14 mm serpiginous defect with irregular borders and no favourable rupture anatomy for device closure at the site aforementioned in TTE, so he was referred for surgical repair.

During surgery, scarred myocardium was found at the right ventricular wall and basal segments of the posterior and inferior walls of the left ventricle. Ventriculotomy was achieved through the posterior LV wall. The septal defect was measured 15 mm and was repaired with two Teflon patches, without any residual shunt.

The patient didn't present any complications and has been symptom-free ever since. He is followed up by his cardiologist at the local health centre.

Discussion

Ventricular septal rupture is a rare complication following myocardial infarction.¹ Clinical presentation includes acute heart failure and/or cardiogenic shock, but can rarely be completely asymptomatic.^{2,3}

The gold standard in the diagnosis of VSR is transthoracic echocardiography,⁴ which cannot only provide information about the size and location of the rupture, the ventricular function and the shunt significance, but can also help with the differential diagnosis in a patient presenting with cardiogenic shock.

Repair of the VSR should be performed in all patients even if clinically stable, because the size of the septal rupture can increase without warning.⁵ However, the timing of the repair has yet to be determined, with conflicting opinions between European and American guidelines.^{6,7} The American College of Cardiology guidelines suggest immediate surgical repair of VSR irrespective of the patient's haemodynamic status,⁷ whereas the ESC guidelines refer to delayed elective surgery as an alternative in selected patients who respond to medical therapy.⁶ Although limited studies have been conducted on the matter, literature reviews show that urgent or early surgery is associated with high mortality rate (47–60%).^{8,9} In contrast, delayed surgery in patients with VSR who respond to aggressive medical therapy seems to have better outcome, minimizing the mortality rate to less than 15%.⁹ Percutaneous closure of VSR is an emerging treatment with data supporting similar survival rate with surgical repair,¹⁰ but limitations concerning the features of the defect (size, shape, location, etc.) apply.

Our patient presented a rare case of VSR with symptoms of chronic heart failure. Although the exact time of the myocardial infarction was not reported by the patient, the onset of his symptoms



Figure 1 Electrocardiogram upon admission with sinus rhythm, left axis deviation, borderline QRS duration, QS complexes in leads V1 to V5.

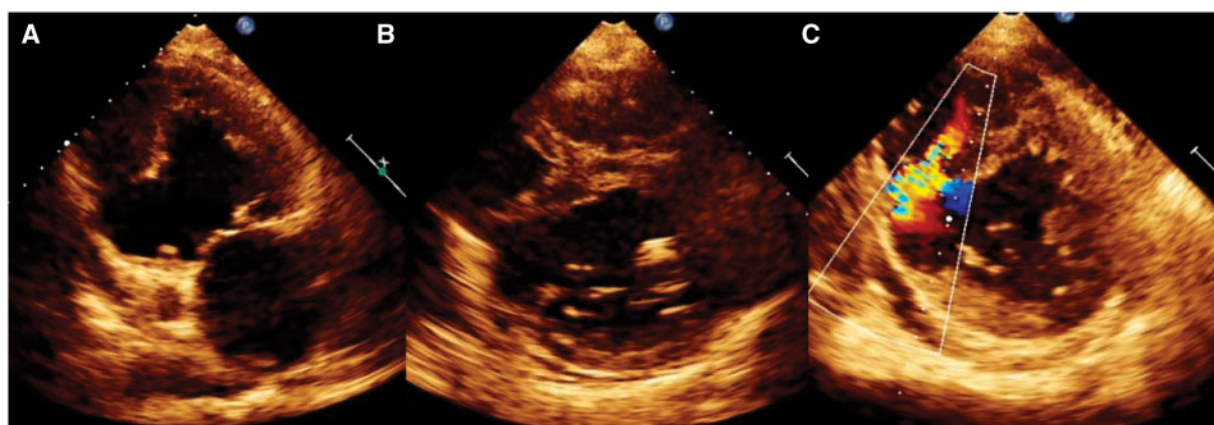


Figure 2 (A) Apical two chamber and (B) parasternal short axis views reveal wall aneurysm. (C) Colour Doppler in parasternal short axis view reveals left-to-right shunt.

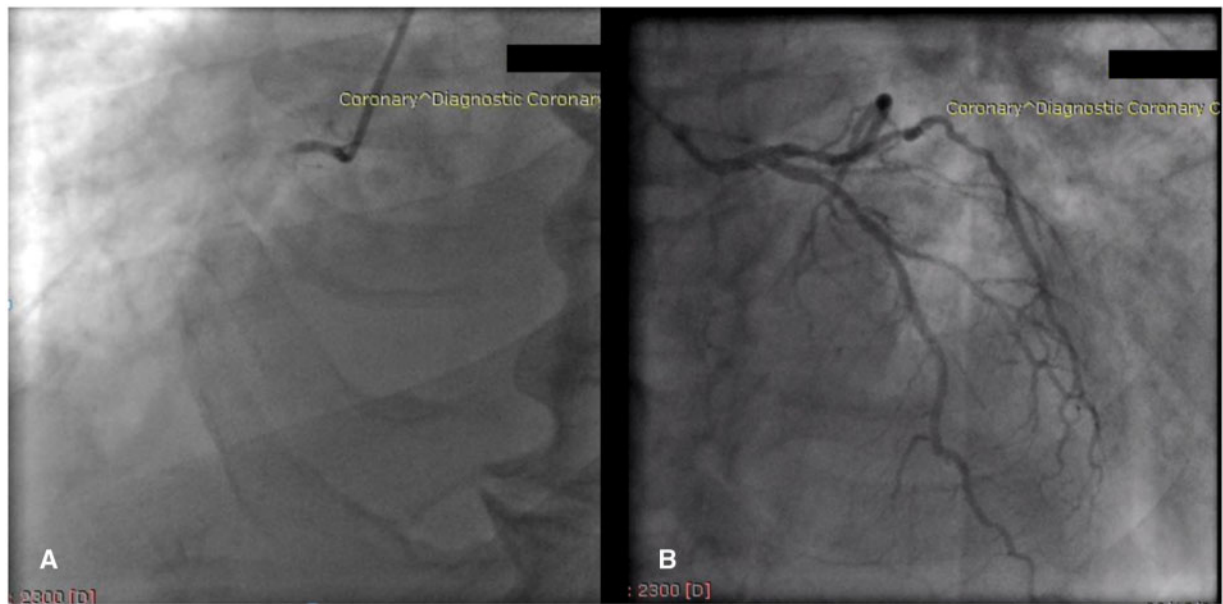


Figure 3 Coronary angiography: (A) proximal occlusion of the right coronary artery. (B) Left anterior descending and circumflex arteries with no significant stenosis.

Table 1 Right heart catheterization

Aortic pressure [systolic/diastolic (mean)]	104/71 (81) mmHg
Right ventricle pressure [systolic/diastolic (mean)]	60/18 (38) mmHg
Pulmonary artery pressure [systolic/diastolic (mean)]	60/25 (42) mmHg
Right atrium pressure [mean]	26 mmHg
Pulmonary capillary wedge pressure [mean]	33 mmHg
VO ₂	203.9 mL/min
Cardiac output	6.6 L/min
Cardiac index	3.6 L/min/m ²
Pulmonary vascular resistance	0.5 WU
Systemic vascular resistance	11.5 WU
Qp/Qs	2.8

suggests it had happened a couple of months before he visited the Emergency Department. Considering the findings of the TTE, the cause of the symptoms would be the gradual deterioration of the right ventricular function due to systolic dysfunction and volume overload through the ventricular shunt. The haemodynamic status of the patient was already stable before medical attention, despite the moderate size of the septal defect. Having this luxury in such a case, gave us the opportunity to optimize the medical treatment and perform the necessary workup before referring the patient for surgical closure. Percutaneous closure was rejected due to unfavourable anatomical characteristics of the rupture.

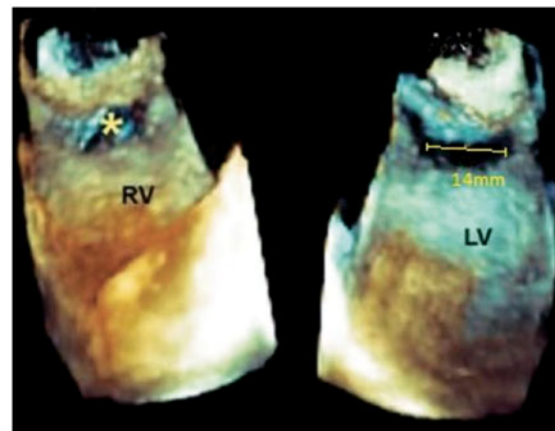


Figure 4 3D TOE visualization of the rupture.

Conclusion

Although extremely rare, there have been reported cases of asymptomatic or mild symptomatic patients with VSR, including the one in this article. High clinical suspicion and thorough physical examination at first medical contact can help identify these cases early. Rupture repair should be performed in every case, but the timing of the operation may differentiate, depending on the haemodynamic status of the patient. Literature suggests that elective surgery in patients who are stable and respond to medical treatment may have better results than early repair.

Lead author biography



Dr. Ioannis Michelis completed his medical studies at the University of Athens in 2009. He began clinical training in Cardiology at Chalkis General Hospital, which was completed at 251 Air Force General Hospital, Athens in 2018. In 2017 he was trained as an echocardiography fellow at OLV Hospital in Aalst, Belgium. He currently remains as a consultant at 251 Air Force General Hospital.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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