Ventricular septal defect complicating delayed presentation of acute myocardial infarction during COVID-19 lockdown: a case report

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Background
Post-myocardial infarction ventricular septal defects (VSDs) have become rare in the reperfusion era but remain associated with very high morbidity and mortality. As patients defer prompt evaluation and management of acute coronary syndromes during the COVID-19 global pandemic, the incidence of these and other post-infarction mechanical complications is expected to increase.

Case summary
A 37-year-old gentleman with multiple coronary artery disease risk factors presented with intermittent chest discomfort and 1 week of heart failure symptoms. An echocardiogram demonstrated a large muscular VSD and coronary angiography confirmed the presence of an anterior wall infarction. He was subsequently referred for transcatheter VSD repair and showed rapid clinical improvement in his symptoms.

Discussion
Post-infarction VSDs remain associated with a high degree of morbidity and mortality. Surgical repair of acutely ruptured myocardium can be technically challenging, and transcatheter repair has emerged as a safe and effective alternative.

Keywords
Ventricular septal defect • Acute MI • COVID-19 • Case report

Learning points
• To recognize ventricular septal defect as a potentially devastating complication of delayed presentation of acute coronary syndrome.
• To understand the risks and benefits of both early and delayed VSD closure.
• To illustrate transcatheter therapy as a reasonable alternative to surgical repair.

Introduction
Post-myocardial infarction (MI) ventricular septal defects (VSDs) have become relatively rare in the era of thrombolytic therapy and even rarer since the widespread adoption of percutaneous coronary intervention, estimated to occur in ~0.2% of all MIs. They remain associated with very high morbidity and mortality, with an estimated survival rate of 8% at 30 days and <3% at 1 year. As patients may defer evaluation and treatment of acute coronary syndromes in the
setting of the COVID-19 global pandemic, the incidence of post-infarction mechanical complications is expected to increase.

**Timeline**

| Date       | Event                                                                                           |
|------------|-------------------------------------------------------------------------------------------------|
| 21 May 2020 | Start of intermittent chest discomfort                                                         |
| 26 May 2020 | Progressively worsening dyspnoea and lower extremity oedema prompts presentation to referral hospital |
| 27 May 2020 | Patient transferred, echocardiogram demonstrates large muscular ventricular septal defect (VSD) and akinetic anterior wall and apex |
| 1 June 2020 | Left heart catheterization demonstrates occluded left anterior descending artery; right heart catheterization demonstrates severely elevated filling pressures with large shunt (Qp:Qs 2:1) |
| 8 June 2020 | Patient undergoes transcatheter VSD repair                                                      |
| 11 June 2020| Patient discharged home                                                                          |
| 18 June 2020| Seen in follow-up clinic feeling well and appearing euvoletic with improved functional status    |

**Case presentation**

A 37-year-old Caucasian gentleman with past medical history of hypertension, hyperlipidaemia, and severe hypertriglyceridaemia as well as family history of extensive premature coronary artery disease presented with progressively worsening dyspnoea that began 1 week prior to admission. Prior to the onset of dyspnoea, he had developed acute onset left-sided chest discomfort radiating to the left arm. He had deferred seeking medical care due to the COVID-19 global pandemic, and only presented to an outside hospital when his dyspnoea progressed to the point where he was experiencing symptoms at rest with profound orthopnoea. His medications prior to admission included atorvastatin and lisinopril. Examination was notable for jugular venous pressure at the angle of the mandible, 4/6 holosystolic murmur with associated thrill, and bilateral lower extremity oedema. Initial electrocardiogram demonstrated poor R-wave progression in the precordial leads concerning for anterior infarction (Figure 1). Transthoracic echocardiography demonstrated a severely dilated left ventricle with aneurysmal anterior, anteroseptal, inferoseptal, and apical segments (Video 1). The left ventricular ejection fraction (LVEF) was severely reduced at 21%. A large VSD was noted in the mid to distal anteroseptum measuring 1.3 cm in diameter with peak pressure gradient of \( \sim 40 \text{ mmHg} \) (Video 2). Cardiac magnetic resonance imaging noted a transmural infarct and pseudoaneurysm in the left anterior descending (LAD) territory with large associated muscular VSD, with an aperture measuring 1.3 cm (Figures 2 and 3). The anterior wall demonstrated severe hypoperfusion suggestive of non-viable myocardium. Subsequent cardiac catheterization demonstrated elevated right- and left-sided filling pressures (right atrial pressure 21 mmHg and pulmonary artery wedge pressure 28 mmHg), elevated pulmonary artery pressure (52/30 mmHg with mean 40 mmHg), and a significant left-to-right shunt (Qp:Qs 2:1). His systemic cardiac output was estimated at 3.1 L/min by indirect Fick. Subsequent coronary angiography revealed a completely occluded LAD just beyond a large diagonal branch and right coronary artery with moderate proximal disease; the circumflex was free of significant disease.

The patient was diuresed aggressively, losing a total of 30 lbs. Despite his low systemic cardiac output, he had no clinical evidence of impaired end-organ perfusion and thus inotropes and temporary mechanical circulatory support were deemed unnecessary prior to intervention. Throughout his hospitalization, he was started on heart failure guideline-directed medical therapy including sacubitril–valsartan 26-26 mg twice daily, spironolactone 25 mg daily, and dapagliflozin 10 mg daily.

After a multidisciplinary discussion between our heart failure, cardiothoracic surgery, interventional cardiology, and congenital heart disease teams, he was taken for transcatheter VSD closure with an Amplatzer™ post-infarction VSD occluder (Abbott, Abbott Park, IL, USA) under the auspices of a Humanitarian Device Exemption.
Right internal jugular venous and right femoral artery access was obtained, and a 0.035" J-wire was advanced through the femoral artery sheath into the ventricle and across the septal defect into the right pulmonary artery. A 6 Fr balloon-tipped 0.035" pulmonary artery catheter was advanced into the right pulmonary artery through the internal jugular sheath. A 0.035" exchange length guidewire was advanced through the pulmonary artery catheter and the catheter was withdrawn. A 6 Fr 25 cm goose neck snare was advanced over this exchange wire and exposed in the pulmonary artery. The J-wire was snared and externalized through the internal jugular sheath. An FR4 catheter was advanced over the externalized rail into the right atrium.

A 10 Fr TorqVue™ (Abbott, Abbott Park, IL, USA) system was advanced over the rail through the internal jugular sheath to the point that it touched the FR4 catheter in the right atrium. The system was then advanced across the defect back into the left ventricle across the aortic valve so that the distal end of the TorqVue™ was in the ascending aorta. The externalized rail was withdrawn through the femoral arterial sheath.

A 20 mm Amplatzer™ post-infarction VSD occluder device was prepared, advanced into the TorqVue™ system, and brought to the tip of the catheter. Under transoesophageal (TEE) ultrasound and fluoroscopic guidance, the catheter was withdrawn back into the left ventricle and the left ventricular disc was exposed. The device was then withdrawn so that contact of the left ventricular disc with the left ventricular wall was made. The neck was then exposed followed by the right ventricular disc. Once appropriate

**Figure 1** Electrocardiogram demonstrating normal sinus rhythm with poor R-wave progression seen in the precordial leads suggestive of anterior infarction.

**Figure 2** Cardiac magnetic resonance imaging with magnitude reconstruction sequence demonstrating delayed enhancement consistent with infarction (white arrows) of the anterior wall with microvascular obstruction (white stars) and with ventricular septal defect (black arrow).
positioning was confirmed, the device was deployed. Transoesophageal and ventriculography confirmed no significant leak across the defect (Figure 4).

After the successful procedure which the patient tolerated from a haemodynamic standpoint, metoprolol succinate was started. Computed tomography angiography obtained 72 h post-procedure demonstrated successful closure of the VSD (Figure 5) and the estimated Qp:Qs by volumetric analysis was 1.1:1. Two months later, his post-procedure transthoracic echocardiogram demonstrated an improvement in his LVEF to 31%. He denied any additional heart failure symptoms during outpatient follow-up.

**Discussion**

Post-MI VSDs have become relatively rare in the era of reperfusion therapy; rupture of the interventricular septum is estimated to occur in around 0.2% of Mls. Usually observed either in the first 24 h or 3 to 7 days post-MI, VSDs are associated with very high morbidity and
VSD complicating deay acute MI presentation

Conclusions

As the number of COVID-19 cases increases worldwide, patients continue to make efforts to avoid in-person contact with healthcare providers, which delays appropriate diagnosis and management of acute coronary syndromes. Efforts to educate the community on the risks associated with delayed presentation are warranted. In particular, post-MI VSD remains a devastating complication of delayed treatment and portends a very high morbidity and mortality. While there is lack of consensus regarding the optimal timing of repair, percutaneous closure remains a viable alternative to an open surgical approach.

Lead author biography

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Supplementary material

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

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Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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Figure 5 Computed tomography angiography demonstrating successful closure of the ventricular septal defect.

mortality, with an estimated survival rate of 8% at 30 days and <3% at 1 year.2 Risk factors for development of post-MI VSDs include advanced age, female gender, and occlusion of the LAD coronary artery.3,4 There is a lack of consensus regarding the optimal timing for post-MI VSD repair, with early repair (generally defined as within 1 week of presentation) associated with a 20–40% mortality rate and high risk of recurrent ventricular rupture. On the other hand, late repair balances the benefits of organization of recently infarcted tissue but portends the risk of rupture extension and death while awaiting procedural intervention.5 Prolonged closure delay may also result in the devolvement of right heart failure and pulmonary hypertension.

As surgical repair of acutely ruptured myocardium can be challenging, percutaneous post-MI VSD closure has become an attractive alternative in the absence of contraindication (defect size >35 mm, basal VSD rim near the mitral or aortic valves, and apical VSD without sufficient margins).6,7 One study of 29 consecutive patients who underwent immediate transcatheter repair of post-MI VSD demonstrated a mortality rate of 65% despite an initial procedure success of 86%—comparable to other case series of patients undergoing surgical repair.9

In the setting of the global COVID-19 pandemic, one cross-sectional observational study demonstrated longer symptom-to-first medical contact time and a higher incidence of patients presenting outside of a time window for revascularization amongst those presenting with STEMI after Hong Kong hospitals launched emergency response measures to combat COVID-19. More MI patients in that series met the primary composite outcome of in-hospital death, cardiogenic shock, sustained ventricular tachycardia or fibrillation, and use of mechanical circulatory support.10 As patients continue to avoid presenting to hospitals out of an abundance of caution for exposing themselves to COVID-19, the incidence of mechanical complications of acute MI including VSD may rise.
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