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GRAND ROUND
Cardiac imaging

Left ventricular pseudoaneurysm: an inadvertent consequence of COVID-19—a case report

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Background Left ventricular pseudoaneurysm (LVP) is an uncommon but serious mechanical complication of acute myocardial infarction (AMI). The immediate medical complications of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) are well recognized, but its indirect effect on patients and healthcare systems is potentially less perceivable.

Case summary In this report, a 72-year-old man who was anxious about attending hospital during the SARS-CoV-2 pandemic was eventually found to have a total right coronary artery occlusion after a delayed emergency department presentation. He ultimately developed severe symptomatic heart failure and cardiac magnetic resonance imaging (CMR) revealed that a large LVP with concomitant severe ischaemic mitral regurgitation had evolved from his infarct. The patient was successfully discharged home after the surgical replacement of his mitral valve and repair of his LVP.

Discussion This case highlights a salient downstream effect of Coronavirus disease 2019 (COVID-19): the delay in presentation, diagnosis, and management of common treatable conditions such as AMI. It also underscores the importance of non-invasive multimodal imaging on the timely identification of the mechanical complications of AMI. In particular, CMR can play a crucial role in the characterization and management of LVP.

Keywords Pseudoaneurysm • Case report • COVID-19 • Myocardial infarction • Mitral regurgitation • Cardiac magnetic resonance imaging • Mechanical complications

Learning points

- Infectious disease outbreaks can lead to changes in behaviour that necessitate corrective action through large scale public campaigns.
- There is an urgent need for the availability of rapid infectious disease testing strategies and streamlined management pathways during times of crisis.
- Cardiac magnetic resonance imaging plays a crucial role in the early diagnosis, complete characterization and management of left ventricular pseudoaneurysm.

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Introduction

Cardiovascular diseases are the leading cause of mortality in Europe accounting for 45% of all deaths with ischaemic heart disease (IHD) alone accounting for 1.8 million deaths annually.[1] Acute myocardial infarction (AMI) is the most serious manifestation of IHD but advances in its prevention and acute medical management has led to a decline in event rates and improved survival over the past two decades.[2]

The mechanical complications of AMI have dropped considerably since the adoption of early percutaneous coronary intervention as the standard of care, with incidence falling from 6% between 1977 and 1982, to 0.27% between 2003 and 2015.[3] When these complications do occur, they carry a poor prognosis, with in-hospital mortality rates as high as 44%.[3]

Many international reports have shown a reduced incidence of hospitalization for AMI since the Coronavirus disease 2019 (COVID-19) pandemic has begun.[4–7] Avoidance behaviours are a well-recognized sequelae of pandemics[8] and the fear of serious illness has been shown to be a key determinant in the delayed utilization of healthcare systems.[9]

In AMI, early diagnosis and reperfusion therapy are crucial in preserving the viability of ischaemic myocardium and the prevention of downstream complications.[10] Any cause for a time delay in either of these factors has a great effect on mortality in patients with myocardial infarction (MI).[11]

Timeline

Day 0	Delayed presentation after 2 weeks of feeling generally unwell
Day 2	Basic investigations pointed towards a first presentation of heart failure (HF) with a potential ischaemic aetiology
Day 3	Initiation of disease-modifying HF therapies
Day 6	Coronary angiogram showed a total occlusion of the right coronary artery
Day 9	Discharged home on intensive medical management
Day 25	Re-presentation to hospital with acute decompensation
Day 27	Cardiac magnetic resonance imaging showed the presence of a left ventricular pseudoaneurysm (LVP) and severe ischaemic mitral regurgitation
Day 34	Surgical replacement of mitral valve and repair of LVP
Day 103	Discharge home from hospital after a lengthy period of intensive rehabilitation

Case presentation

A 72-year-old man presented to the emergency department with a 2-week history of weakness and fatigue. The patient engaged in virtual consultation multiple times with his general practitioner for these symptoms, which failed to resolve with conservative management. He was apprehensive about attending a busy tertiary hospital during the early stages of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic.

He also reported 4 days of progressive dyspnoea on exertion with slight limitation of physical activity, but no chest pain, palpitations, nor ankle oedema. He had increased urinary frequency, with dark urine but no dysuria. He had no dysphagia, bleeding per rectum, abdominal pain, or weight loss. He did not feel feverish and had no rigours or recent travel. Soon after admission, the patient became increasingly unwell with a spike in temperate to 39.1°C and developed a tachycardia of 133 b.p.m.

His past medical history included benign prostatic hyperplasia and his only medication at the time of admission was Dutasteride/tamsulosin 0.5/0.4 mg.

On physical examination, apart from being tachycardic, vital signs were within normal range. There were no peripheral stigmata of cardiovascular disease. The apex beat was non-displaced and auscultation was notable for a pansystolic murmur loudest at the apex that radiated into the axilla. The jugular venous pressure was not raised and no peripheral oedema was present. The patient's respiratory, gastrointestinal, and neurological examinations were unremarkable.

Notable blood tests on admission: C-reactive protein of 101 mg/L (<3 mg/L), serial high-sensitivity troponin T 500 ng/L (<14 ng/L), N-terminal pro-B-type natriuretic peptide (NT-proBNP) of 4337 pg/mL (<300 pg/mL), and ferritin of 1498 ng/mL (<250 ng/mL).

The electrocardiogram showed sinus tachycardia with diffuse non-territorial ischaemic changes as displayed in *Figure 1*, below. Ill-defined, perihilar air-space opacities were seen on a chest radiograph.

A transthoracic echocardiogram was performed. The left ventricle was dilated, with left ventricular (LV) internal diastolic dimension of 6.9 cm, and systolic function impaired, with an LV ejection fraction of 47%. The inferior and inferoseptal walls were thinned and akinetic from base to mid-ventricle. The LV filling pressure was significantly elevated and there was moderate mitral regurgitation (MR) present. Right ventricular systolic pressure was significantly elevated (>60 mmHg). This was consistent with the presenting electrocardiogram which showed a right ventricular strain pattern and S1Q3T3. This led to the decision by the supervising clinician to perform computed tomography pulmonary angiography (CTPA) to rule out a pulmonary embolism.

The CTPA was negative for pulmonary embolism; however, it showed changes consistent with pulmonary oedema with moderate bilateral pleural effusions as well as ground glass change and interlobular septal thickening in both upper lobes. There was also trace pericardial fluid with pericardial thickening raising concern for pericarditis.

Due to the patient's non-specific symptoms, raised inflammatory markers, and recorded pyrexia, the initial impression was systemic inflammatory response syndrome due to an infectious process—either viral (SARS-CoV-2) or another organism of unknown source. He

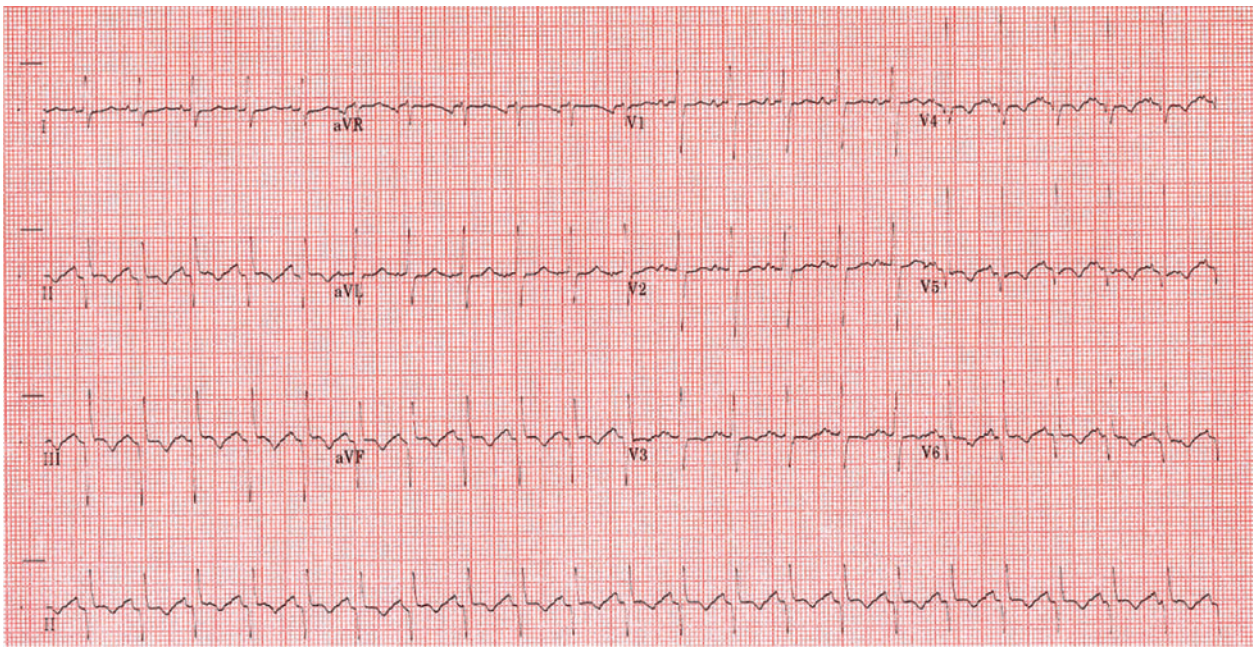


Figure 1 Electrocardiogram at presentation.

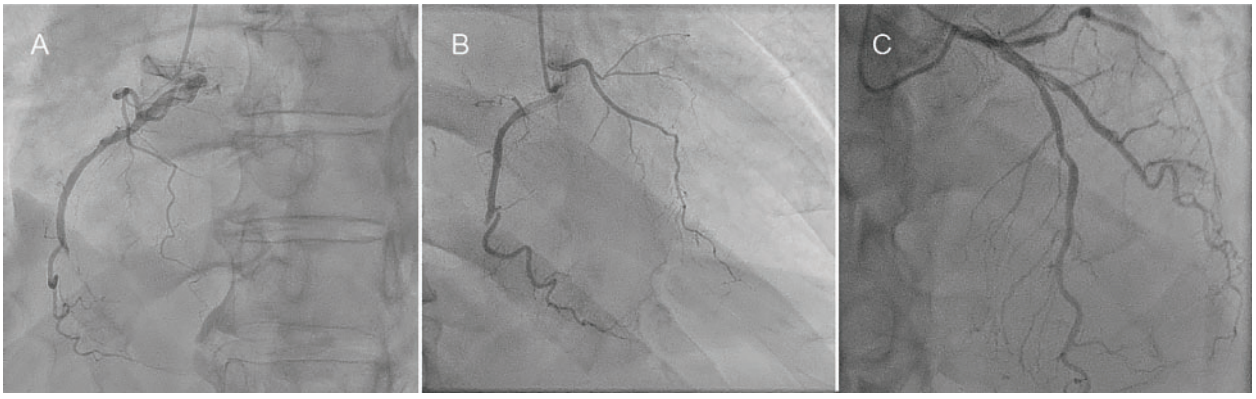


Figure 2 Coronary angiogram images. (*A*) Left anterior oblique projection view of occluded right coronary artery. (*B*) Right anterior oblique projection view of occluded right coronary artery. (*C*) Anterior–posterior cranial projection view of left coronary artery.

was investigated for COVID-19, as his presentation coincided with high community case rates, but all three nasal-pharyngeal swabs returned negative.

The other main differential considered at that time was a first presentation of heart failure (HF), of potentially ischaemic aetiology. This was suspected as the patient had an audible murmur with an abnormal electrocardiogram, echocardiogram, and elevated NT-proBNP. Pulmonary embolism was also considered but was effectively ruled out.

The patient was initially admitted under the care of the on-call general medical physician. The cardiology team was consulted on day 4 of inpatient stay and took over the patient's ongoing care as an infectious process had been ruled out. Once the patient was clinically stable, a diagnostic coronary angiogram was performed which showed occlusion of the right coronary artery (RCA) at the mid-vessel, with collateralization from the left coronary system. The left coronary system was free of significant coronary disease.

He clinically improved on intravenous diuretic therapy and he was reviewed by the HF specialist team for institution of disease-modifying agents. He was discharged home on appropriate medical therapy with early planned follow-up in the HF unit. His discharge diagnosis was documented as ischaemic HF with LV involvement due to RCA occlusion. The patient's discharge prescription was composed of the following: dutasteride/tamsulosin 0.5/0.4 mg, pantoprazole 40 mg,

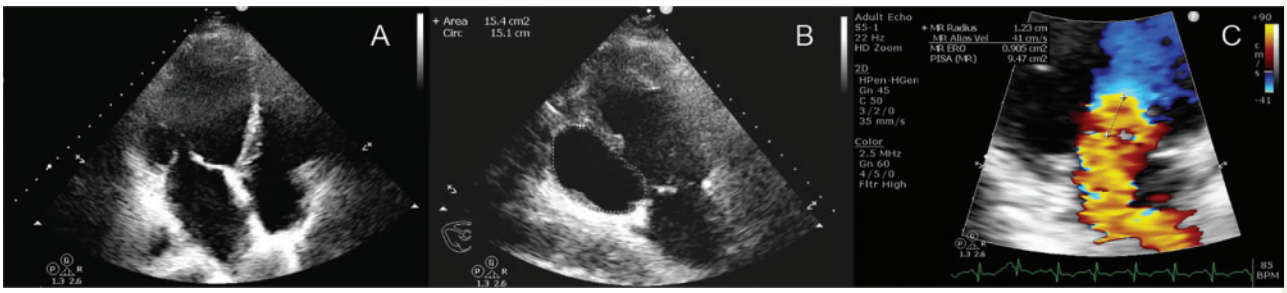


Figure 3 Repeat transthoracic echocardiogram images. (*A*) Apical four-chamber view. (*B*) Apical two-chamber view showing saccular outpouching. (*C*) Apical two-chamber view with colour Doppler quantifying the severe mitral regurgitation present.

aspirin 75 mg, clopidogrel 75 mg, furosemide 40 mg, atorvastatin 80 mg, ramipril 2.5 mg, and bisoprolol 1.25 mg.

The plan was for initial intensive medical management and discussion at a heart team meeting regardingrevascularization of his occluded RCA.

The case was discussed with the hospital heart team (including cardiologists, cardiothoracic surgeons, and radiologists) and a consensus decision was made to proceed to cardiac magnetic resonance imaging (CMR) to assess viability of the right coronary territory. This is in line with the 2018 European Society of Cardiology/European Association for Cardio-Thoracic Surgery (ESC/EACTS) guidelines on myocardial revascularisation.[12]

Two weeks after discharge, the patient represented via the emergency department with New York Heart Association class III symptoms, fatigue, night sweats, and palpitations. He was treated with intravenous diuresis and a repeat transthoracic echo showed severe MR with restricted motion of the posterior mitral valve leaflet. It also revealed a large LV saccular outpouching in the basal inferior wall.

A CMR viability study with contrast was performed shortly after readmission and showed a dilated LV with a focal posterior saccular outpouching arising from the basal to mid-inferior wall measuring 6.2 × 3.4 cm. The aneurysmal segment was dyskinetic with transmural late gadolinium enhancement throughout. There was also a region of endocardial low signal in the floor of the outpouching consistent with thrombus. The intact pericardium, together with the organizing thrombus at the base, contained the inferior free wall rupture. The saccular outpouching had a relatively narrow neck compared to its base and there was a sudden progression from normal thickness myocardium to an attenuated layer. Taken together all these features pointed towards the diagnosis of an LV pseudoaneurysm (LVP).[13] Overall LV systolic function was reduced with an ejection fraction of 37%. There was severe secondary MR present with tethered chordae and restricted motion of the posterior mitral leaflet.

The patient was assigned to bed rest and close monitoring in the coronary care unit. The findings and implications of the magnetic resonance imaging scan were thoroughly discussed with the patient and his family by the team involved in his care. The limited data regarding the best treatment approach for pseudoaneurysms and ischaemic MR highlight the importance of having a heart-team with wide-ranging expertise in all subspecialties of cardiology, from HF and electrophysiology to cardiac surgery and advanced imaging. This is in keeping with the 2017 ESC/EACTS Guidelines for the management of valvular heart disease.[14]

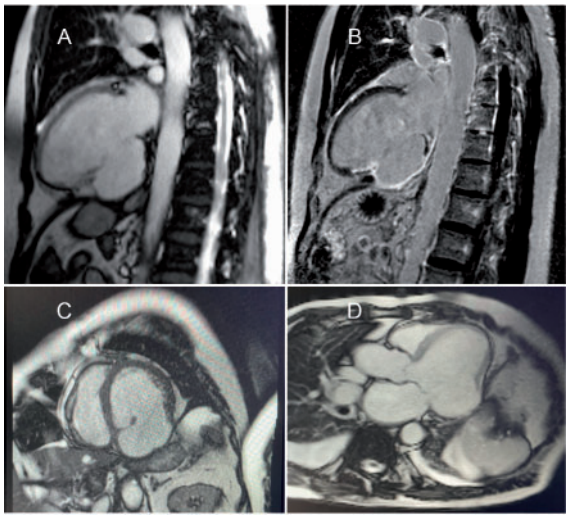
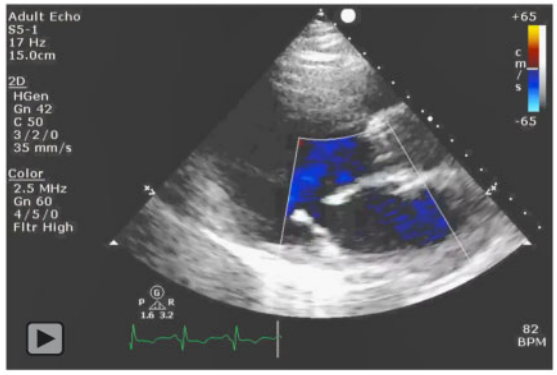


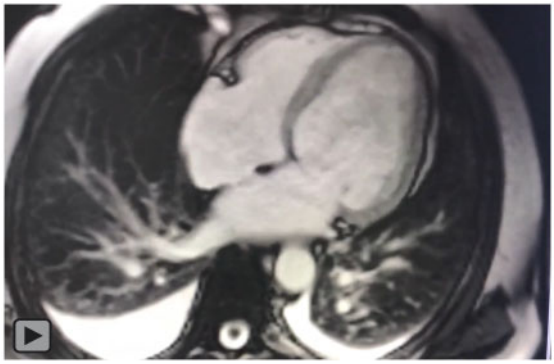
Figure 4 Cardiac magnetic resonance images. (*A*) Two-chamber steady-state free precession cine showing the pseudoaneurysm with narrower neck vs. base. (*B*) Two-chamber post-contrast phase sensitive myocardial delayed enhancement image showing transmural enhancement of myocardium and pericardium. (*C*) Short-axis steady-state free precession at basal level showing the left ventricular pseudoaneurysm and papillary muscles. (*D*) Three-chamber steady-state free precession showing transition of normal thickness myocardium to the thin wall of a pseudoaneurysm.

After weighing up the hypothetical risks and benefits, the patient decided to opt for attempted surgical replacement of his mitral valve and repair of his LVP.

Intraoperatively the patient was found to have severe ischaemic MR with retracted but intact chordae. An LV aneurysm was visible which roughly measured 5 × 4 cm and had a thin wall adherent to the diaphragmatic surface of the pericardium. A 29 mm Perimount Magna Ease mitral valve (Edwards Lifesciences, CA, USA) was placed



Video 1 Transthoracic echocardiogram displaying the saccular outpouching and severe mitral regurgitation.



Video 2 Cardiac magnetic resonance imaging showing the pseudoaneurysm through two- and three-chamber SSFP cine sequences.

via a left atriotomy. A cuff of pericardium was dissected and left attached to the aneurysm wall. Repair of the pseudoaneurysm was then performed by direct closure with no immediate complications.

The patient had a difficult postoperative course, spending 26 days in intensive care. His recovery was complicated by arrhythmia, cardiogenic shock, ischaemic sigmoid colitis, bowel perforation, and sepsis. He recuperated well and following intensive rehabilitation was discharged home fully independent 3 months after his initial surgery. His prescription was composed of the following: atorvastatin 80 mg, bisoprolol 7.5 mg, furosemide 40 mg, aspirin 75 mg, apixaban 5 mg twice daily, pantoprazole 40 mg, and ramipril 7.5 mg.

A repeat echocardiogram prior to discharge showed severely depressed LV systolic function with an ejection fraction of 26%. The inferior and infero-lateral walls were akinetic, but the mitral valve prosthesis was functioning well with no significant transvalvular or paravalvular regurgitation.

Discussion

Ventricular aneurysms and pseudoaneurysms as a result of AMI are rare in the Western world due to the earlier detection and appropriate management of significant coronary disease. Pseudoaneurysms occur when a blood vessel or myocardial wall ruptures and is then contained by pericardium, thrombus or adhesions.[15] The most common cause for LVP formation is MI but they also can occur after aortic valvular surgery, endocarditis and penetrating trauma.[15] Ischaemic MR occurs when the leaflets and the subvalvular apparatus are anatomically intact but functionally impaired by LV remodelling, dysfunction, and mitral annular dilation secondary to ischaemia.[16] Moderate to severe ischaemic MR occurs in 12% of patients after MI and classically follows inferior territory infarctions.[16]

This case was likely influenced by reticence to present to hospital in the setting of the COVID-19 pandemic, as well as delayed cardiology involvement in management due to concerns around a presentation potentially compatible with a communicable illness. With patients delaying presenting to healthcare services, and the effect that infection prevention and control methods can have on timely diagnostics, there may be a delay in primary percutaneous intervention in the setting of MI. Garcia *et al.*[6] showed a 38% reduction in ST-segment elevation cardiac catheterization laboratory activations in the USA in March 2020 in comparison to pre-COVID-19 times, and a large French registry study found a 30% decrease in admissions for AMI after lockdown was instigated.[7] Similar avoidance of healthcare settings was seen during the H1N1 epidemic in 2009.[8] The detrimental behavioural change provoked by a pandemic necessitates corrective action through large scale public campaigns.

The COVID-19 pandemic has caused a major impact on healthcare systems throughout the world. The delay in reaching this patient's diagnosis was caused by pathways designed to prevent the onwards transmission of the virus, but this came at the detriment of timely investigation, diagnosis and management. He would have benefitted from a physical review and an electrocardiogram as opposed to a virtual consultation with his general practitioner. Then on admission to hospital, the requirement for a number of negative SARS-CoV-2 tests impeded his transfer of care to the cardiology service and ultimately hindered his diagnosis and treatment. This case highlights the urgent need for the availability of rapid infectious disease testing strategies and streamlined management pathways during times of crisis. The shortage of hospital beds and the high rates of nosocomial transmission associated with SARS-CoV-2 has led to patients being discharged as soon as it's deemed safe to do so with investigations being deferred if unlikely to substantially change patient management. In a non-COVID-19 era, this patient's MR may have been assessed more critically prior to discharge with a transoesophageal echocardiogram (aerosol-generating procedure) or an inpatient CMR which may have identified the inherent mechanical nature of his MR earlier.

Several reports have highlighted the fall in AMI presentations during the COVID-19 era,[6,7] and the seemingly consequential rise in the mechanical complications of AMI.[4,5] This patient suffered two severe mechanical complications of MI; LVP and the critical disruption of mitral valve architecture. The concomitant presence of ischaemic LV dysfunction and severe ischaemic MR resulted in the rapid progression to symptomatic HF despite the initiation of disease-modifying therapy. This may have actually benefitted this patient by resulting in

his early re-presentation to medical services whereas in undiagnosed ischaemic LV dysfunction, his presentation could have been delayed to the point where the patient would first have suffered terminal LVP rupture.

LVPs have very poor prognostic outcomes, with the risk of rupture as high as 45% if left untreated.[15] Recommendations on how to manage pseudoaneurysms are largely based on small retrospective case studies.[15,17,18] Due to their infrequency, no randomized controlled trials have been done to provide clarity on the best management approach, be this surgical or non-operative medical therapy for those with increased intraoperative risk and small pseudoaneurysms. Recently, the novel percutaneous closure of LVPs has been described in the literature with promising results.[19]

Large pseudoaneurysm formation is an absolute indication for surgical repair, although, this historically carried with it a high risk of mortality—as much as 23–38%.[18] Modern surgical techniques have improved perioperative mortality to less than 10% in simple pseudoaneurysms, but complex surgical repairs involving valvular replacements carry a grave prognosis.[18] While echocardiogram is generally the first imaging modality used in the search for the mechanical complications of MI, CMR provides superior quantification of ventricular size, function and degree of infarction.[20] CMR is less limited by both observer variability and the technical skill that is required for image acquisition. It also provides valuable information on the aetiology, severity, and prognosis of MR.[20] Diagnosis of LVPs can be challenging and this case highlights the important role that CMR can play in the timely identification and proper delineation of the anatomy to allow appropriate surgical approach planning.

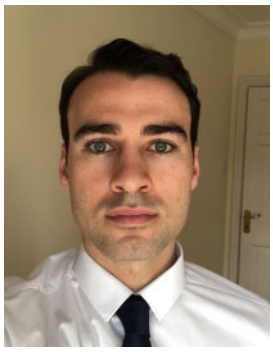
There is poor data currently available to guide clinicians on the use of anticoagulation to reduce the risk of thromboembolism associated with LVP. The theoretical advantage provided by therapeutic anticoagulation must be juxtaposed against the increased risk of rupture and haemorrhage. This decision must be made on a case by case basis, taking all available clinical factors into account and balancing the potential risks and benefits.

This case also underscores the significance of joint decision making in clinical practice, especially in situations where potential poor outcomes are anticipated. It is important that the patient is fully educated on all aspects of their clinical situation, especially when evidence is lacking, so that they can make an informed decision that conforms with their personal values and expectations.

Conclusion

The importance of maintaining access to healthcare services and the encouragement of appropriate early presentation during the ongoing COVID-19 pandemic cannot be overstated enough. The preservation of diagnostic and therapeutic capabilities during similar future settings should be an essential goal for all healthcare systems. Non-invasive multimodal imaging has revolutionized our ability to diagnose and treat LVPs. CMR allows the complete characterization of their anatomy and plays a vital role in directing the management of these rare but serious complications.

Lead author biography



Stephen Brennan is completing his internship at St. Vincent's University Hospital (Dublin). He is a graduate of the National University of Ireland Galway Medical School and is also studying a Masters in Preventive Cardiology with the National Institute for Prevention and Cardio vascular Health.

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 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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