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CASE SERIES
Cardiac imaging

A case series of ventricular cystic masses

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Background Ventricular cystic masses are uncommon. Elucidating the cause is essential for early directed therapy and prevention of complications. We present two cases of ventricular cystic masses, one in each ventricle, after myocardial infarction (MI) and ventricular septal rupture (VSR), respectively.

Case summary Patient 1 is a 58-year-old male with left brachio-facial stroke and evolved anterior MI. A left ventricular (LV) cystic thrombus was seen on transthoracic echocardiogram (TTE) and cardiac magnetic resonance (CMR) imaging. He was started on anticoagulation with reduction in thrombus size 11 days later. Patient 2 is a 67-year-old male with evolved anterior MI, severe LV systolic dysfunction, and normal right ventricular (RV) function. He was readmitted two weeks later with fever, heart failure, *Streptococcus agalactiae* bacteraemia, and septic pulmonary emboli. Transthoracic echocardiogram showed biventricular systolic dysfunction and a RV cystic mass associated with a partial VSR. He was treated with anticoagulation and antibiotics. Repeat TTE 5 weeks later revealed near resolution of the cystic mass and complete VSR. Cardiac magnetic resonance confirmed these findings and also showed a localized mid-septal transmural infarction at the VSR site. He underwent percutaneous coronary intervention to the left anterior descending and circumflex arteries, and percutaneous VSR closure with a muscular ventricular septal defect device later.

Discussion Our two cases demonstrate that ventricular thrombi can present as cystic masses after MI and VSRs. Infectious, vascular, or oncogenic causes should be considered in the appropriate clinical context. Early diagnosis and treatment is essential to prevent embolic complications, and secondary infection.

Keywords Case series • Cystic mass • Ventricular cystic thrombus

Learning points

- Ventricular thrombi may present as cystic masses, and may be associated with mechanical complications of myocardial infarction.
- Echocardiography and cardiac magnetic resonance are useful techniques for the detection, evaluation and follow-up of ventricular cystic masses.
- Early diagnosis and treatment is a key to preventing complications.

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Introduction

Ventricular cystic masses are uncommon and embolic complications can develop.[1] We report two patients with ventricular cystic masses, one left ventricular (LV) and one right ventricular (RV), with different outcomes and complications.

Timeline

Timeline	Event
Patient 1	
2 days prior to admission	Had an episode of chest pain
Admission	No chest pain but developed left facial and upper limb weakness
(Day 0)	Magnetic resonance brain imaging revealed a right pre-central gyrus infarct.
	Electrocardiography (ECG) showed sinus rhythm with anterior ST-segment elevation and pathologic Q waves
Day 4	Transthoracic echocardiogram (TTE) showed left ventricular (LV) anteroapical akinesia with left ventricular ejection fraction (LVEF) of 45%, and cystic, mobile mass at the LV apex
	Started on anticoagulation
Day 10	Cardiac magnetic resonance (CMR) imaging showed ischaemic cardiomyopathy and an irregular mass attached to hypokinetic LV apex consistent with thrombus
Day 11	Repeat TTE showed reduction in size of the cystic mass
	Discharged
Patient 2	
4 days prior to 1st admission	Had an episode of chest pain after which he developed worsening exercise tolerance
1st admission	No chest pain. ECG showed sinus rhythm with new Q waves in V1 to V3
(Day 0)	Chest radiograph was normal
Day 4	TTE showed LVEF 30% with LAD territory akinesia and normal right ventricular (RV) function
	Started medical therapy for evolved anterior myocardial infarction.
Day 7	Discharged
2nd admission	Presented with shortness of breath, pedal oedema, and feve
(Day 15)	Chest radiograph showed right upper and lower zone consolidation
Day 17	Blood cultures grew *Streptococcus agalactiae*
Day 19	TTE revealed biventricular systolic dysfunction and a cystic RV mass overlying a possible new, mid-septal, partial ventricular septal rupture (VSR)
	Antibiotics were continued for 6 weeks and anticoagulation commenced for infected thrombus with septic pulmonary emboli
Day 55	TTE showed near resolution of RV cystic mass and a complete ventricular septal rupture
	Coronary angiogram showed significant in-stent restenoses in the left anterior and circumflex arteries
Day 62	CMR imaging showed resolution of the RV cystic mass, a haemodynamically significant VSR, and localized mid-septal transmural infarction at the VSR site. The rest of the LV myocardium was viable
Day 68	Percutaneous coronary intervention (PCI) to the LAD and LCx in-stent restenoses.
Day 95	VSR was closed percutaneously with a muscular ventricular septal defect (VSD) device

Case presentation

Patient 1

A 58-year-old hypertensive, diabetic male on Nifedipine 20 mg OM, Atenolol 50 mg OM, and herbal supplementation presented with acute left brachio-facial stroke. He had an episode of central chest pain 2 days earlier. On examination, vital signs were stable. He was pain-free but had left upper limb weakness. Cardiac examination was unremarkable.

Magnetic resonance imaging of his brain showed an acute right pre-central gyrus infarct. High-sensitivity Troponin I was elevated at 6915 ng/L (normal value 0–18 ng/L). Electrocardiography showed sinus rhythm with ST-segment elevation and pathologic Q waves in leads V2 to V4 (*Figure 1*). Transthoracic echocardiogram on Day 6 of the admission showed anteroapical akinesia with left ventricular ejection fraction (LVEF) of 45% and a 2.4 cm × 2.1 cm cystic, mobile mass with soft flexible walls at the LV apex (*Figure 2*, *Video 1*). These findings were consistent with an evolved anteroapical MI with cystic LV apical thrombus.

As he was allergic to aspirin, the patient was started on clopidogrel, together with low molecular weight heparin and atorvastatin.

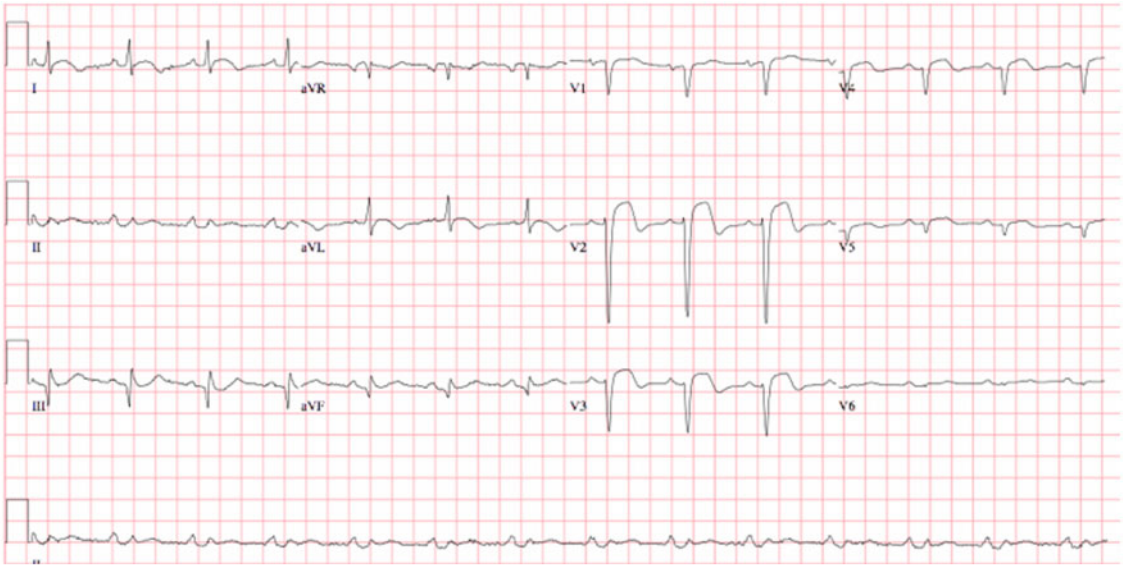


Figure 1 Electrocardiogram of the first patient showing anterior ST elevation and Q waves.

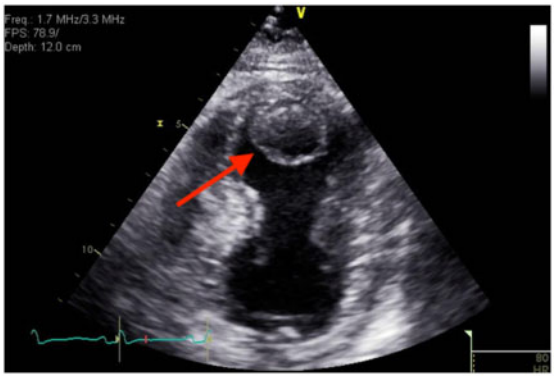
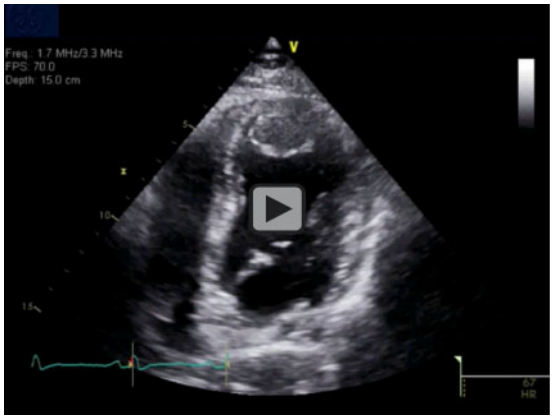


Figure 2 TTE of the first patient showing a left ventricular apical cystic and mobile mass measuring 2.4 cm × 2.1 cm (red arrow).



Video 1 Transthoracic echocardiogram of the first patient. The apical four-chamber view showed anteroapical akinesia with left ventricular ejection fraction of 45% and a 2.4 cm × 2.1 cm cystic, mobile mass with soft flexible walls at the left ventricular apex.

Cardiac magnetic resonance imaging performed on Day 10 confirmed the anterior MI. The apical LV mass was still present, irregular in shape and with limited mobility, but smaller. Cardiac magnetic resonance characteristics of hyperintensity in T1-w fast spin-echo (FSE), T2-w triple inversion recovery fast spin-echo (IR FSE) sequences, with no increase in uptake on first pass perfusion imaging (*Figure 3*) confirmed that the mass was a fresh apical thrombus.

Repeat TTE on Day 11 also showed a reduction in size of the cystic mass. The patient elected for medical therapy with plans for PCI later in his home country.

Patient 2

The second patient was a 67-year-old male smoker with non-insulin dependent diabetes, hypertension, hyperlipidaemia, and peripheral vascular disease. He was on Aspirin 100 mg OM, Rivaroxaban 20 mg OM, Omeprazole 40 mg OM, Ferrous Sulfate compound 2 tablets OM, metformin 850 mg BD, Telmisartan 40 mg OM, and sublingual glyceryl trinitrate 0.5 mg when necessary for chest pain. He had a non-ST-segment elevation myocardial infarction 6 years ago, which was treated with PCI to the left circumflex (LCx) and left anterior descending artery (LAD). He presented with worsening exercise tolerance following an episode of chest discomfort 4 days prior to admission.

His vital signs were stable. Cardiac examination was unremarkable with no evidence of heart failure. High-sensitivity Troponin I was elevated at 10426 ng/L (normal value 0 to 18 ng/L). Electrocardiography showed sinus rhythm with new Q waves in V1 to V3, ST-segment depressions in V4 to V6, I, II and aVL and less than 1 mm ST-segment

elevation in III and aVR (*Figure 4*). The chest radiograph showed no pulmonary infiltrates or consolidation. Transthoracic echocardiogram showed a severely depressed LVEF of 30% with akinesia in the LAD territory. Right ventricular function was normal.

A diagnosis of evolved anterior myocardial infarction was made. He was treated with a single antiplatelet agent as he had significant renal impairment and anaemia of uncertain cause that required blood transfusion, and discharged. He was planned for gastrointestinal endoscopic evaluation and subsequent cardiology review outpatient.

He was readmitted 2 weeks later with shortness of breath, lower limb swelling, and fever. His vital signs were stable, but he was febrile at 38.7°C. He was in sinus rhythm, had no murmurs, but had bilateral lung crepitations and bilateral pedal oedema.

He had leucocytosis with white blood cell count 21.1 $\times$ 10^9/L (normal value 4 to 9.6 $\times$ 10^9/L) and neutrophil count 19.26 $\times$ 10^9/L (normal value 1.90 to 6.60 $\times$ 10^9/L). CXR showed new consolidation in the right upper and lower zones (*Figure 5*). Blood cultures grew

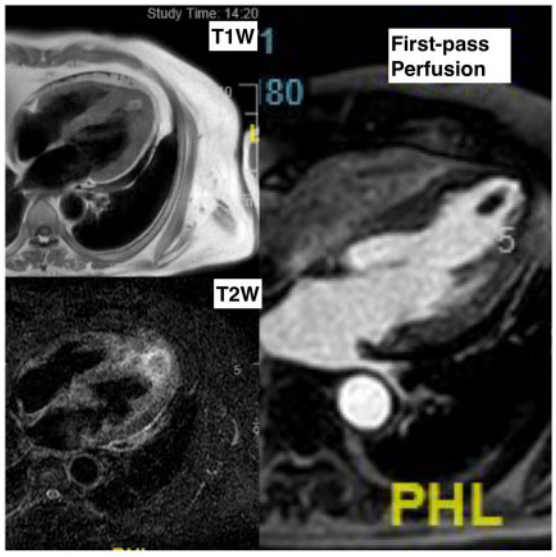


Figure 3 Cardiac magnetic resonance of the mass showing hyperintensity in T1-w FSE and T2-w triple IR FSE sequences, with no increase in uptake on first-pass perfusion.

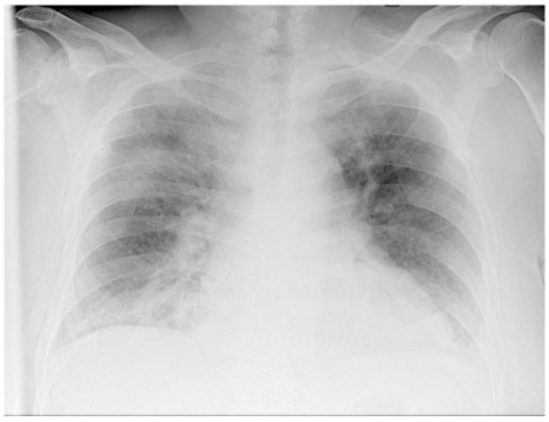


Figure 5 CXR of the second patient showing pulmonary consolidation in the right upper and lower zones.

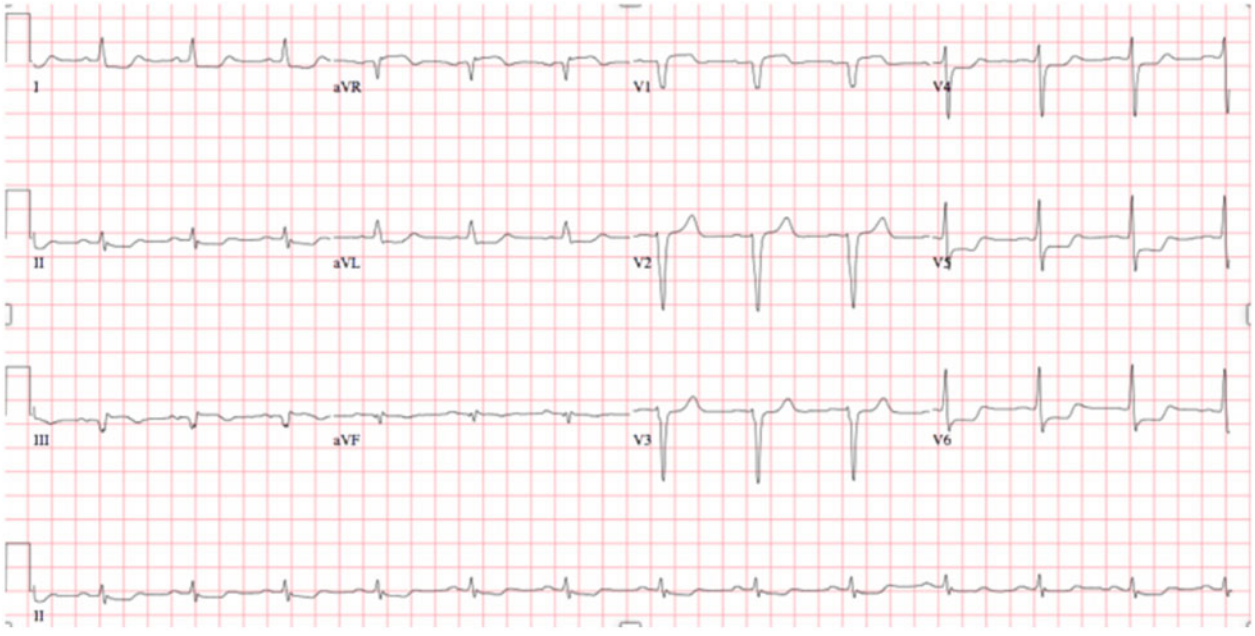


Figure 4 Electrocardiogram of the second patient with anterior Q waves.

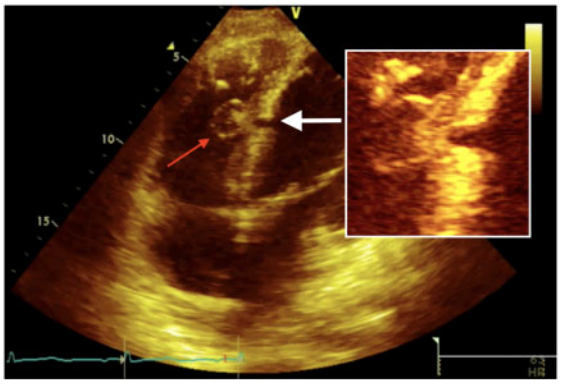


Figure 6 Transthoracic echocardiogram of the second patient showing a right ventricular cystic mass (thin red arrow) measuring 1.8 cm × 1.4 cm, overlying a partial ventricular septal rupture (thick white arrow). Close-up in image insert.

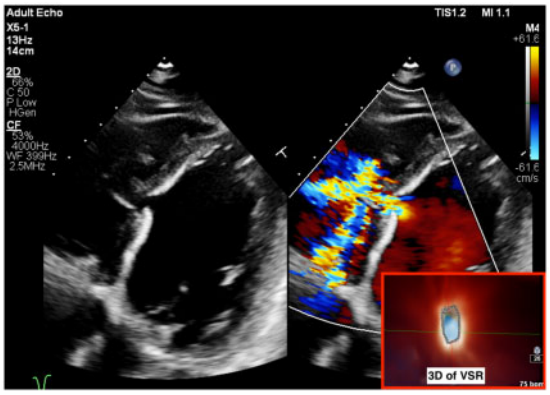
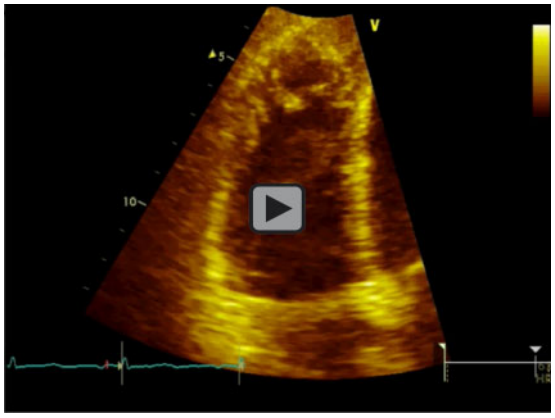
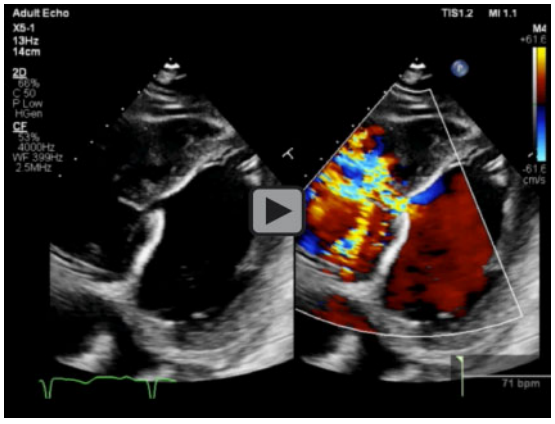


Figure 7 Transthoracic echocardiogram performed 5 weeks later showed development of a complete ventricular septal rupture with remnants of the cystic mass (thin red arrows). The insert shows a 3D reconstruction of the ventricular septal rupture.



Video 2 Transthoracic echocardiogram of the second patient showing severe left ventricular and moderate right ventricular systolic dysfunction, and also a cystic mass measuring 1.8 cm × 1.4 cm in the right ventricle, overlying a small, well-defined and localized mid-septal gap in the myocardium measuring 0.5 cm.



Video 3 Transthoracic echocardiogram of the second patient performed 5 weeks later showed development of a complete ventricular septal rupture with remnants of the cystic mass.

Streptococcus agalactiae. Transthoracic echocardiogram showed severe LV and moderate RV systolic dysfunction and a new cystic mass measuring 1.8 cm × 1.4 cm in the RV overlying a new, incomplete gap in the middle of the interventricular septum. This gap was small, measuring 0.5 cm, well-defined and localized (*Figure 6*).

The working diagnosis was RV cystic thrombus overlying a partial ventricular septal rupture (VSR) following a recent evolved anterior MI, complicated by *Streptococcus agalactiae* bacteraemia, secondary infection of the cystic thrombus, and septic pulmonary emboli. He was treated with 6 weeks of intravenous ceftriaxone and anticoagulation with warfarin.

Five weeks later, TTE showed almost complete resolution of the RV cystic mass. However, there was now a complete VSR (*Figure 7*). Coronary angiogram performed revealed significant LAD and LCx in-stent restenosis (ISR). Further assessment by CMR one week later showed complete resolution of the RV cystic mass and a haemodynamically significant VSR in the middle of a localized, mid-septal transmural infarct (*Figure 8*). The rest of the myocardium was viable.

He was deemed to be a high risk candidate for surgery and was treated percutaneously with drug-eluting balloon coronary angioplasty to the LAD and LCx ISR. Percutaneous VSR closure with a muscular VSD device was successfully performed later. However, the patient was readmitted with severe pneumonia, type 1 respiratory failure and septic shock 3 months later. He required intubation and intensive care management, and subsequently passed away.

Discussion

Ventricular cystic masses are uncommon.[1] Differential diagnoses include infections secondary to Echinococcus resulting in hydatid cyst,[2]

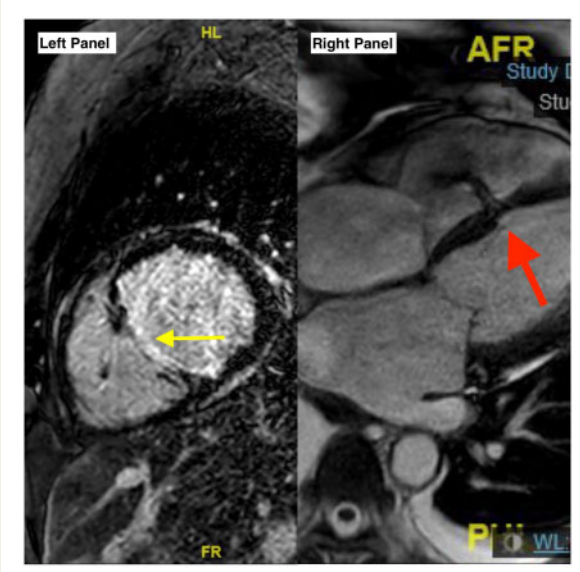


Figure 8 Cardiac magnetic resonance imaging showed resolution of the right ventricular cystic thrombus. Left panel: The late gadolinium enhancement (LGE) image showed a localized, mid-septal transmural infarct (thin yellow arrow) and viability in the rest of the left ventricular. Right panel: Steady state free precision (SSFP) sequence showed a haemodynamically significant ventricular septal rupture (thick red arrow).

or Taenia Solium causing cysticercosis,[3] vascular malformations such as capillary haemangiomas,[4] cystic thrombi,[2] congenital blood cysts[5] and intracardiac tumours. Right ventricular cystic masses are less common than LV ones.[1]

The clinical context and the characteristics of the mass help to determine the most likely diagnosis. The most commonly described cause of a ventricular cystic mass in the post-MI patient is cystic thrombus.[2,6,7]

Left ventricular thrombus has become an uncommon post-MI complication. Prior to the era of PCI, LV thrombus was present in 7–46% of patients.[8] Since the introduction of early primary PCI, its incidence has declined.[9] Risk factors for LV thrombus formation include large infarct size, anterior MI, LV aneurysm, and severe apical asynergy.[10]Virchow's triad of blood stasis, hypercoagulability and endothelial injury plays an important role in the formation of LV thrombus after MI.[10] Increased blood stasis from dyskinetic or akinetic segments, a hypercoagulable state from inflammation, and subendothelial injury, increase aggregation of fibrin, platelets and red blood cells, and subsequent thrombus formation. The thrombus may appear cystic depending on the degree of aggregation of these factors, anticoagulation treatment, as well as the passage of time.[6]

Right ventricular thrombus on the other hand, often originates from systemic venous or tumour thrombosis.[11] It is a rare complication of anterior or inferior MI,[12] and associated with concomitant infarction of the RV apex. Our 2nd patient had a RV cystic mass together with a partial VSR, both of which were atypical. The cystic thrombus was mid-septal rather than apical,[13] and the VSR did not occur adjacent to a hypercontractile myocardium, which is an additional characteristic feature seen on echocardiography.[13]

Ventricular septal ruptures may develop after transmural infarction of the ventricular septum, a phenomenon commonly associated with evolved MI.[14] This is seen in our second patient, who had a delayed presentation of evolved anterior MI due to concerns regarding the recent Coronavirus disease 2019 (COVID-19) pandemic. Coagulation necrosis of infarcted tissue causes weakening of the septum and susceptibility to tears from the shear stress generated by adjacent hypercontractile myocardium.[13] They can occur in any anatomic location supplied by the septal branches[13] and are usually located at the border of living, contractile myocardium, and necrotic tissue. Anterior infarctions are usually associated with simple apical ruptures, and inferior infarctions with complex basal septal ruptures and oblique, serpiginous routes.[14]

Our patient's early partial septal defect was small, 0.5 cm in diameter, and highly localized. It penetrated the septum directly and its edges were clean and smooth. It was located in the middle of a motionless septum, away from any adjacent hypercontractile physical stressor. Its association with overlying cystic thrombus strongly suggested a complication of transmural infarction rather than a congenital malformation, such as a myocardial crypt or incompletely closed VSD.[15] Mild aneurysmal left-to-right bulging of myocardium over the septal gap was consistent with stretching of infarcted myocardium under LV pressure, culminating later in a blow-out rupture. We believe that this is the first reported case of RV cystic thrombus associated with VSR.

Embolic complications may result from ventricular thrombi if left untreated. Left ventricular thrombus may be associated with systemic emboli[16] causing stroke and other end-organ infarcts. Right ventricular thrombus may result in pulmonary embolism and infarction. Rarely, thrombus may act as a nidus for secondary infection.[17] This may result in mural endocarditis or myocarditis, or cause septic emboli.

As such, early detection and diagnosis is essential. Transthoracic echocardiogram can be performed quickly, at the bedside if necessary, without need for radiographic contrast, gadolinium, or radiation. Limitations of image quality and artefacts can be circumvented by use of ultrasound contrast.[18] Cardiac magnetic resonance imaging can further characterize the lesion and differentiate thrombus from other possible diagnoses.[10]

Anticoagulation for at least 3–6 months[19,20] is the treatment of choice. It greatly reduces the risk of systemic embolization.[9] The decrease in size and resolution of thrombus with anticoagulation was well demonstrated by serial imaging in our two patients. Current guidelines recommend vitamin K antagonists as first line oral anticoagulants, but direct acting oral anticoagulants are reasonable second line alternatives.[20] However, the duration of anticoagulation remains uncertain. Long-term anticoagulation may be considered in patients with persistent thrombus, even if it is well organized, spontaneous echo contrast with severe and persistent wall motion abnormalities, or with thrombi that formed in the absence of any acute provocative factor, such as myocardial infarction or heart failure.[10] Concomitant guideline-directed therapy is essential for any ischaemia and cardiomyopathy. Mechanical complications of MI will require surgical or percutaneous treatment.

In conclusion, our two cases demonstrate that cystic thrombi can occur in both left and right ventricles in the presence of a MI and VSR. Imaging allows for characterization and diagnosis. Embolic and infective complications can occur. Hence, early treatment is essential.

Lead author biography

Jieli Tong is a Cardiology Consultant at Tan Tock Seng Hospital, which is a large multidisciplinary hospital in central Singapore. Her interests lie in cardiac echocardiography and cardiac oncology.

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 patients in line with COPE guidance.

Conflict of interest: There are no conflicts of interest.

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