

A case report of a giant coronary artery aneurysm masquerading as a ventricular mass

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Background

A coronary artery aneurysm is a dilation exceeding 1.5 times the diameter of the patient's largest coronary vessel. They are rare, varying in prevalence between 1.4 and 4.9%. Additionally, they carry a high risk of potential complications, including thrombosis and myocardial infarction, with a risk of rupture. We present an interesting case of a patient with initial imaging suggesting a mass in the right ventricle.

Case summary

This patient initially presented with acute hypoxic respiratory failure related to pulmonary oedema. His course was complicated by symptomatic ventricular tachycardia and an inferoposterior myocardial infarction. Further investigation revealed a left anterior descending artery and circumflex artery thrombosed aneurysm projecting into the right ventricle. Multimodal imaging was used to arrive at his diagnosis. He continues to do well on medical therapy for coronary artery disease and heart failure.

Discussion

Clinicians should be vigilant for this rare pathology, which may be easily missed yet poses a high mortality risk. Our case demonstrates the benefit of multimodal imaging, as this patient's aneurysm was initially mistaken for a ventricular mass.

Keywords

Coronary artery aneurysm • Ventricular tachycardia • Multimodal imaging • Computed tomography • Thrombosed aneurysm • Case report

ESC curriculum

2.3 Cardiac magnetic resonance • 2.4 Cardiac computed tomography • 2.1 Imaging modalities • 2.2 Echocardiography • 3.1 Coronary artery disease

Learning points

- Coronary aneurysms may present a wide variety of symptoms, such as myocardial infarction with secondary-related ventricular arrhythmias. These aneurysms may also rupture, spasm, or fistulize with other arteries. Giant coronary artery aneurysms are rare pathologies with no current guidelines in diagnosis or management. While catheterization may be helpful, multimodal imaging may be required to detect this disease.
- Evaluation of cardiac masses is usually done initially with echocardiography and cardiac magnetic resonance imaging. We present a case of giant coronary aneurysm masquerading as a mass where a reconstructed gated computed tomography revealed aneurysmal formation, leading to a change in management.

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Introduction

A coronary artery aneurysm (CAA) is a dilation exceeding 1.5 times the diameter of the patient's largest coronary vessel.¹⁻³ The term 'giant' CAA is defined as >8 mm or more than four times the standard diameter.⁴⁻⁶ Giant CAAs have a high risk of thrombosis and myocardial infarction (MI) along with a risk of rupture.⁴⁻⁶ We present an interesting case of a patient initially presenting with respiratory failure due to pulmonary edema, developing symptomatic ventricular tachycardia (VT) and inferoposterior MI, and found to have a left anterior descending artery (LAD) and left circumflex (LCX) artery giant aneurysm. This aneurysm was initially mistaken for a right ventricular (RV) mass.

Summary figure

Timeline

Day	Events
Day 1	Presented with acute hypoxic respiratory failure Transthoracic echocardiography (TTE) suggested a mass imposing on the RV septum Computed tomography angiography (CTA) protocol for a pulmonary embolism did not suggest aneurysmal formation Catheterization suggested a left main aneurysm and obstructive disease in the LCX Transferred hospitals The patient developed VT storm shortly after arrival; amiodarone started Re-catheterization demonstrated complete stenosis in the LCX, embolic from the aneurysmal coronary arteries
Day 3	Transthoracic echocardiography showed a large thrombus in the left atrial appendage (LAA), basal inferior and posterolateral wall akinesis, and a 'large mass' is noted in the RV mid-septum
Day 4	Cardiac magnetic resonance imaging (MRI) reported an 'interventricular septal mass' involving the mid-antero-septal wall size 3.4 × 2.9 cm
Day 7	Repeat TTE showed a 3.1 × 4.1 cm 'septated mass' in the mid-ventricular septum
Day 8	Computed tomography angiography of the heart later confirmed that the mass was an aneurysm involving the proximal left anterior descending wrapped around the apex with the distal part of the left anterior descending thrombosed; this was projected towards the RV side of the interventricular septum (IVS)
~ 1 month after	Had an implantable cardioverter-defibrillator placed and followed by the cardiomyopathy team

Case presentation

A 55-year-old male with a history of chronic heart failure with reduced ejection fraction (EF), hypertension, and diabetes mellitus presented with acute hypoxic respiratory failure. Initial vital signs showed he was afebrile, had a blood pressure of 84/45, saturating 90% on room

air, and had a heart rate of 67. Physical examination showed he had 1+ pitting oedema in his lower extremities, fine crackles on lung auscultation in the bases bilaterally, and a 3/6 holosystolic murmur best heard at the apex. His workup included a CTA due to concerns for a pulmonary embolism, which showed pulmonary oedema and severe atherosclerotic disease of the coronary arteries (see [Figure 1](#)), along with calcification in the right ventricle (RV). Cardiac troponin T measurements returned elevated at 2245 ng/L (normal ≤22 ng/L). An electrocardiogram (EKG) demonstrated ST elevations in the anterolateral leads with poor R-wave progression. Echocardiography showed a low normal left ventricular (LV) EF estimated at 45% with inferior/inferolateral wall hypokinesis and an immobile cystic mass seen in the RV apex and IVS ([Figure 2](#)). He underwent catheterization, which showed a marked aneurysmal dilation of the left main and LAD, with no apparent focal coronary artery disease (CAD). His dyspnoea, related to pulmonary oedema, worsened and required bilevel positive airway pressure (BiPAP). He later developed fascicular VT, which was controlled with amiodarone. He was transferred to our centre for further care.

On arrival, the patient continued to have VT. We considered VT due to ischaemia from a potential embolus from his aneurysmal coronary arteries vs. VT due to the mass in our differential. The patient was subsequently re-catheterized and was found to have 100% stenosis in his LCX, likely embolic from his aneurysmal coronary arteries (Supplemental Video 4). Intravascular ultrasound (IVUS) was not performed due to concerns for extreme tortuosity and angulation. Transoesophageal echo (TEE) confirmed findings on echo. A cardiac MRI (CMR) was obtained, which showed severely reduced LVEF of 26% with prominent hypokinesis of the inferior and inferolateral walls, along with associated transmural scar and superimposed thrombus. A mass was noted involving the mid-inferoseptum measuring 3.4 × 2.9 cm ([Figure 3](#) and Supplemental Figure 6) protruding into the RV. Differential diagnoses included cardiac tumour vs. sarcoidosis. A biopsy was initially planned, but a review of the non-contrast images from the CTA PE performed at the outside hospital revealed the diffuse circumferential nature of the calcium in the location of the mass ([Figure 4](#)).

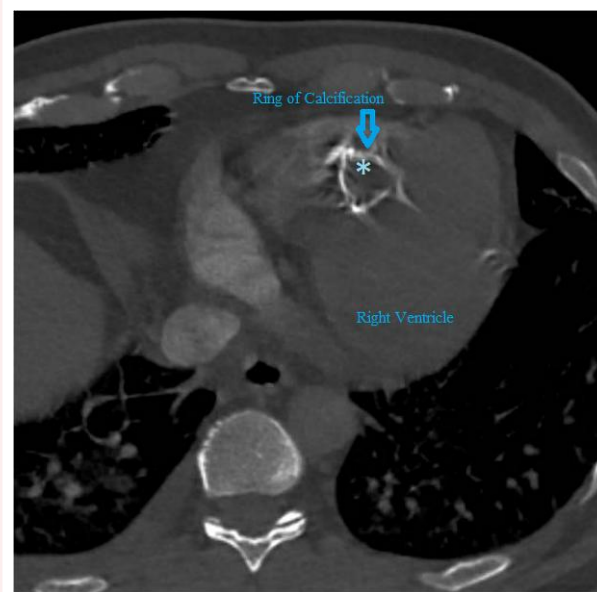


Figure 1 Computed tomography angiography with Pulmonary Embolism (PE) protocol demonstrating calcification in the right ventricle (arrow pointing to asterisk).

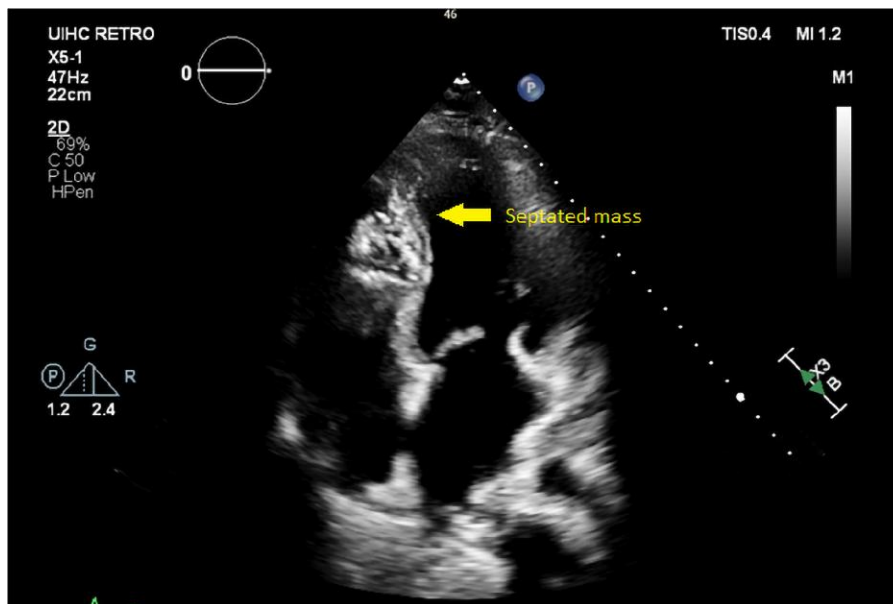


Figure 2 A transthoracic echocardiography demonstrating a 3.1×4.1 cm mass in the right ventricle abutting the mid-interventricular septum.

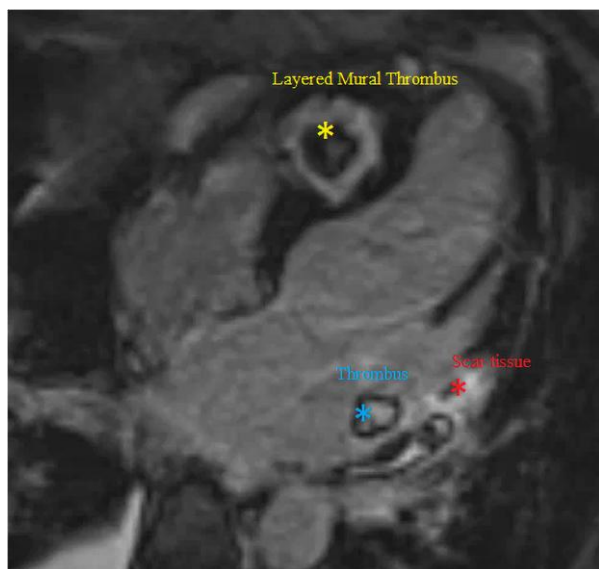


Figure 3 Cardiac magnetic resonance imaging showing a layered mural thrombus (labeled below with corresponding asterisk) involving the basal and mid-lateral wall. The interventricular septal mass was estimated to be 3.4×2.9 cm. In this image, there is a small area of scar tissue (labeled below with corresponding asterisk) with an adjacent thrombus (labeled below with corresponding asterisk) in the inferolateral wall.

Further review of CMR suggested that the mass could have a pulsatile nature. A cardiac CTA with left-sided contrast for coronary artery evaluation was then performed, which revealed a CAA. The distal

portion of the LAD and the distal portion of the LCX were connected to a sizeable aneurysmal formation, which was calcified peripherally, thrombosed, and measured 2.8×2.5 cm, consistent with a giant CAA (Figure 5 and Supplemental Videos 1–3). As this was a focal dilation, this was classified as an aneurysm and not as ectasia, which refers to diffuse dilation of a coronary artery. The aneurysm was fusiform in shape and localized to the distal portion of the LAD/LCX. Based on its shape (bulging into the ventricular wall with a broad base), this likely involved all three layers. In contrast, a pseudoaneurysm, which only involves one or two layers, typically has a narrow neck. Unfortunately, IVUS was not performed in this case to confirm luminal composition. The patient was also incidentally noted to have a LAA thrombus. The aetiology of his VT was likely multifactorial, related to ischaemia associated with the inferior/inferolateral acute MI, the occluded LCX, and the aneurysmal formation.

Cardiothoracic surgery was consulted for the management of this patient's CAA but did not deem this patient eligible for surgery due to his acute MI and aneurysmal coronary arteries. His aneurysmal was likely chronic and managed conservatively, including serial annual TTEs to monitor its size. He started anticoagulation with apixaban due to his LAA thrombus. Current literature suggests that direct oral anticoagulants are the safest choice in treating an LAA thrombus and should be continued for at least 4–6 months with re-evaluation.^{7,8} He continued to demonstrate this clot on follow-up TEEs and thus was continued on apixaban in the long term.

A dual-chamber implantable cardioverter-defibrillator was eventually placed as secondary prevention for sudden cardiac death, and amiodarone was continued due to his history of recurrent VT. Repeat EKGs continued to show normal sinus rhythm, and he did not experience any further episodes of VT. His CAD was managed with aspirin and a statin. His heart failure, likely related to ischaemia, was managed with guideline-directed therapy, including sacubitril/valsartan, metoprolol, and spironolactone (he had a prior history of Fourier gangrene and, thus, a contraindication to empagliflozin). He remains stable on this therapy, with his EF mildly improved to 35% at his last visit.

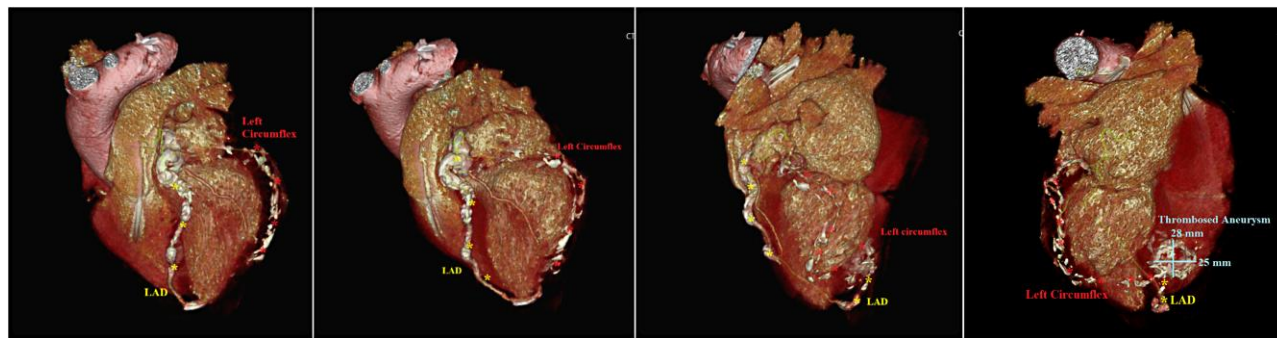


Figure 4 A panel involved reconstructed computed tomography angiography images demonstrating how the distal left anterior descending artery (labeled below as ‘LAD’ with asterisk along pathway) appears thrombosed and occluded at its distal end. This finding is consistent with an aneurysm (labeled below as ‘thrombosed aneurysm’ with lines marking dimensions) arising from an aneurysmal segment of the distal left anterior descending artery. The left circumflex is marked (labeled below as ‘left circumflex’) along its course (adjacent several asterisk marking pathway).

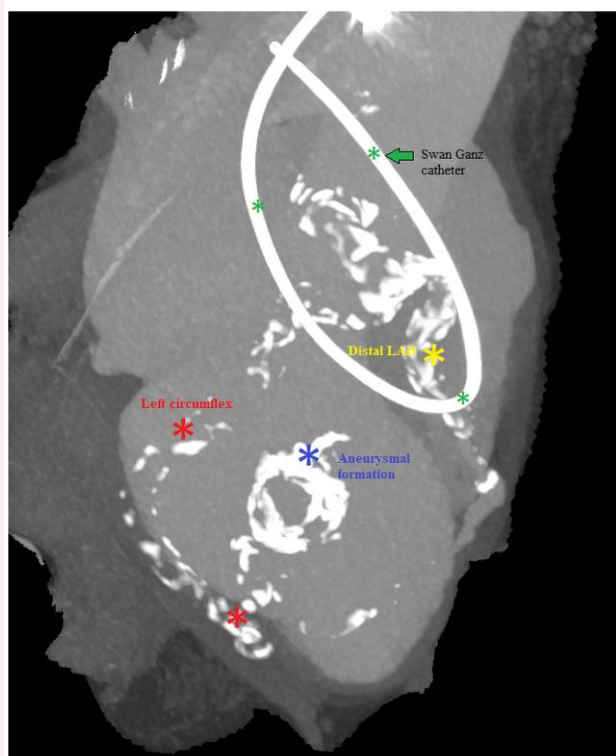


Figure 5 Contrast computed tomography showing distal left anterior descending artery (labeled as ‘distal LAD’ with adjacent asterisk) leading to a formation consistent with an aneurysm (labeled as ‘aneurysmal formation’ with adjacent asterisk). The left circumflex (labeled as ‘left circumflex’ with adjacent asterisk) is marked along its course, as is the Swan–Ganz catheter (labeled as Swan Ganz catheter with arrow pointing to catheter and asterisk along its structure).

Discussion

The incidence of CAA is unclear and varies widely, reported between 1.4 and 4.9%.^{1,3,9} Several studies show that the right coronary artery (RCA) is typically the most involved, followed by the LAD, LCX, and the left main, which is rarely affected.^{1,5,6} There are several potential complications of CAAs. They are predisposed to thrombus formation due to prolonged abnormal flow.^{1,10} In a few described reports, vasospasm and fistulation have been reported.^{5,11} These aneurysms may also rupture, leading to sudden cardiac death in some cases.^{3,12,13}

Coronary angiography is the suggested mode for diagnosis for this disease.^{1,10,14,15} In our case, the catheterization did suggest aneurysmal dilation of the LAD but did not suggest that his aneurysmal dilation included the distal LAD bulging into the septum due to poor visualization and poor distal filling. Therefore, while angiography may be the standard, clinicians should recognize its limitations in sensitivity. Moreover, while our patient had a CTA for PE evaluation, the right-sided contrast on the CT PE precluded adequate diagnosis of CAA. It is critical to have left-sided contrast to allow for proper CAA evaluation. In this patient, the CTA heart with left-sided contrast confirmed that this suspected ‘mass’ was a thrombosed giant CAA in the septum projecting towards the ventricle.

Management and treatment of CAAs are based on anecdotal experiences rather than large-scale controlled trials. For asymptomatic minor aneurysms, medical management may be appropriate; however, for most aneurysms, surgical intervention should be considered.^{1,5,15,16} Anticoagulation is based on the observations of thrombus formation, with limited data on its efficacy.^{1,14,16} In our case, our patient was deemed a poor surgical candidate and was treated conservatively with management for his heart failure, VT, and known atrial appendage clot.

We illustrate a case of a giant CAA originating from the LAD and the LCX, incidentally diagnosed when he presented with VT due to embolic MI from his aneurysmal coronary arteries. Initially, this aneurysm was mistaken for a ventricular mass. Our case demonstrates the benefit of multimodality imaging, as this patient’s diagnosis was not made after TEE, CTA for PE, CMR, or catheterization. Computed tomography angiography with left-sided contrast was needed for adequate

diagnosis. While the incidence of this pathology is rare, clinicians should be vigilant for CAA as it carries a significant mortality risk.

Lead author biography



Sruti Prathivadhi-Bhayankaram is currently a third-year internal medicine resident at the University of Iowa, Hospitals and Clinics. She plans to pursue a cardiology fellowship, with an interest in imaging, electrophysiology, and cardiology.

Supplementary material

[Supplementary material](#) is available at *European Heart Journal – Case Reports* online.

Consent: The authors 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: M.L.A. is a consultant for Tersera.

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Data availability

The data underlying this article are available in the article and its online [supplementary material](#).

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