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COMPETITION WINNER

Cardiovascular imaging

An epicardial problem case report: multimodality imaging assessment of a patient presenting with symptomatic ventricular tachycardia, secondary to a large, epicardial, lipomatous mass with myocardial infiltration

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Background Cardiac lipomas are considered benign and slow-growing adipose tissue tumours; however, the size and location can cause significant haemodynamic compromise and therefore can be malignant in clinical presentation.

Case summary The clinical case describes a rare presentation of a 50-year-old patient, presenting with recurrent haemodynamically compromising ventricular arrhythmia; secondary to an incredibly large, epicardial lipomatous mass, with associated combination of myocardial infiltration, left ventricular systolic impairment, and complex cardiac circulatory involvement.

Discussion The case illustrates the ESC guidelines on the benefits of complimentary multimodality cardiac imaging to assess complex pericardial disease, to improve both clinical understanding and management.

Keywords Case Report • Multimodality imaging • Pericardial disease • Cardiac tumour • Echocardiography • Cardiac MRI • Ventricular tachycardia

ESC curriculum 2.1 Imaging modalities • 2.2 Echocardiography • 2.3 Cardiac magnetic resonance • 6.6 Pericardial disease • 5.6 Ventricular arrhythmia

Learning points

- The case illustrates the ESC guidelines on the benefits of complimentary multimodality cardiac imaging to assess complex pericardial disease, to improve both clinical understanding and management.
- Furthermore, the case highlights the multifactorial precipitants of ventricular tachycardia substrate that can be associated with large invasive cardiac tumours. ESC guidelines support offering secondary prevention ICD if unable to reverse cause of ventricular tachycardia.

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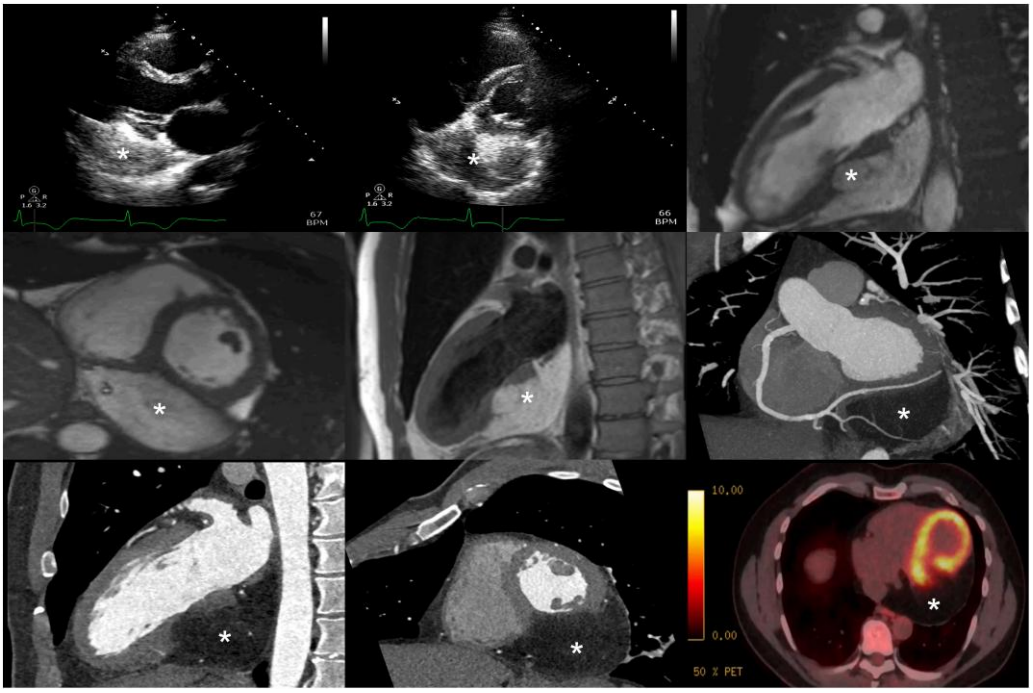
Introduction

Epicardial lipomas are typically considered benign. They are a rare cause of haemodynamic collapse.[1,2] This case illustrates how complimentary multimodality imaging improves lesion characterization and influences clinical management.

Summary figure



Patient presentation

A 50-year-old, Caucasian male presented to Emergency Department (ED) with a 7-day history of palpitations, epigastric discomfort, and presyncope. He had no history of angina, heart failure symptoms, febrile illness, or B-symptoms.

The patient had no past medical history or prescribed medications. He was independent in daily activities and drives. He had a smoking history of 15–20 cigarettes a day and history of recreational Cannabis use. There was no family history of cardiovascular disease.

On clinical examination he was tachycardic with normal blood pressure. Heart sounds were normal with no murmur once the tachycardia was managed. His chest was clear, and there was no abdominal organomegaly or palpable lymphadenopathy.

12 lead ECG (*Figure 1A*) demonstrated sustained ventricular tachycardia (VT) at 200 bpm with right bundle branch morphology. The ED team made an initial diagnosis of supraventricular tachycardia with aberrant conduction. Adenosine and subsequent Amiodarone challenge had no effect.

The patient was reviewed by the cardiology team and a diagnosis of VT was made. The patient subsequently deteriorated with progressive hypotension and rising venous lactate of 3.5 mmol/L. Urgent direct current cardioversion (DCCV) was performed. The VT was terminated by a 150J shock with improvement in patient symptoms and haemodynamics.

ECG post-cardioversion (*Figure 1B*) showed sinus rhythm with narrow QRS morphology and prolonged QTc interval at 517 ms. There was widespread ST depression and deep T-Wave inversion in the inferolateral leads.

Blood tests demonstrated normal haemoglobin level (Hb 153 g/L) and elevated inflammatory markers (WBC 17×10^9/L, CRP 71 mg/L). There was multiorgan perfusion injury with both liver and renal insult (ALT 814 U/L, Urea 22, Creatinine 309, eGFR 19). Potassium and magnesium levels were normal. High-sensitivity Troponin I levels were dynamically elevated from 541 ng/L to 726 ng/L.

Initial differential cause of VT included that of an acute coronary syndrome or an underlying cardiomyopathy. Initial differentials of the cause of VT included acute coronary syndrome, underlying cardiomyopathy or myocarditis.

A transthoracic echocardiogram (*Figure 2*) demonstrated a large, circumferential echobright, mass, adjacent to the basal-mid inferior and inferolateral segments of the left ventricle (LV). The mass appeared to extend to the inferior portion of the right ventricle. Concerningly, it also appeared to have focal infiltration into the adjacent LV myocardium, with associated regional hypokinesia, and LV systolic dysfunction (LVEF 45–50%).

Multimodality imaging was used for lesion characterisation. A Cardiac MRI (*Figure 3*) complimented the echocardiographic findings showing an epicardial mass, measuring 111 × 86 × 54 mm, encasing the inferior and inferolateral LV wall and confirmed myocardial infiltration of the basal-to-mid segments, with associated myocardial thinning. T1-weighted dark blood imaging revealed a homogenous lipomatous mass (*Figure 3C*). There was subtle late-gadolinium enhancement at the mass interface with the myocardium (*Figure 3D*).

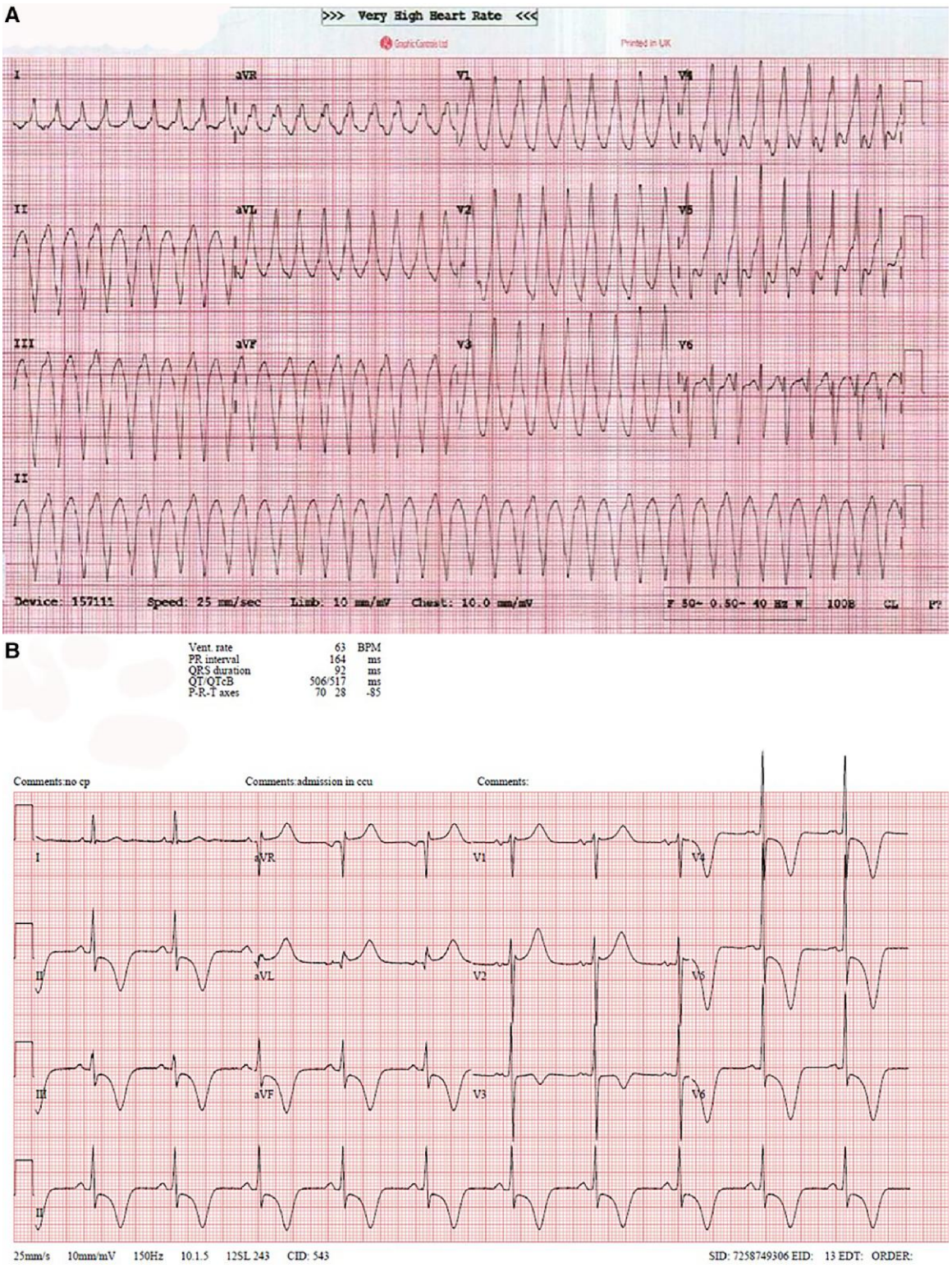


Figure 1 ECG. (*A*) ECG admission—VT with RBB morphology. (*B*) ECG post-DCCV. SR with prolonged QT interval. ST segment depression with deep T-wave inversions V4–V6, and II, III, AVF.

A gated-CT coronary angiogram (*Figure 4*) emphasized the demarcation between the epicardial mass and myocardium with associated myocardial thinning. Moreover, the study demonstrated complex involvement of the coronary circulation. The mass encapsulated both coronary arterial and venous circulation, involving the distal branches of the right coronary artery and the middle cardiac vein.

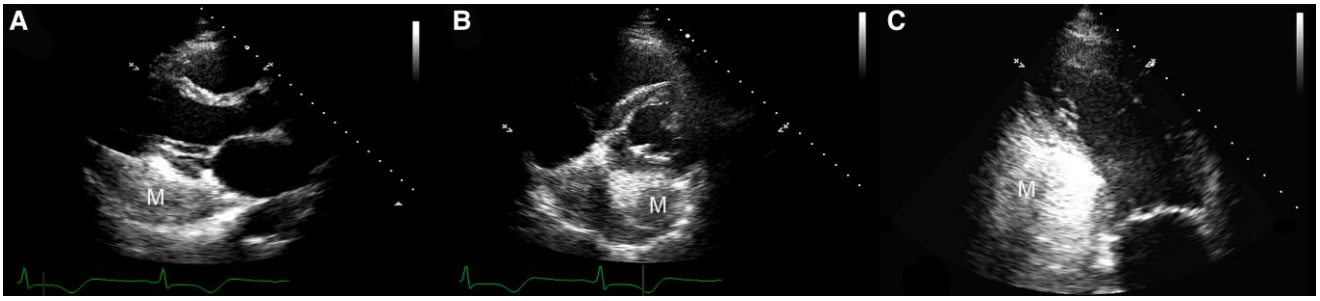


Figure 2 Transthoracic echocardiogram. (*A*) Parasternal long-axis view (*B*) Short-axis view at level of mid LV cavity (*C*) Apical 2-chamber view. Large, epicardial mass within the pericardial space adjacent to inferior and inferolateral wall with circumferential reach towards the RV cavity. The mass (M) appears to infiltrate myocardium at the basal-mid inferior and inferolateral wall. See Supplementary material online, *Video S1*.

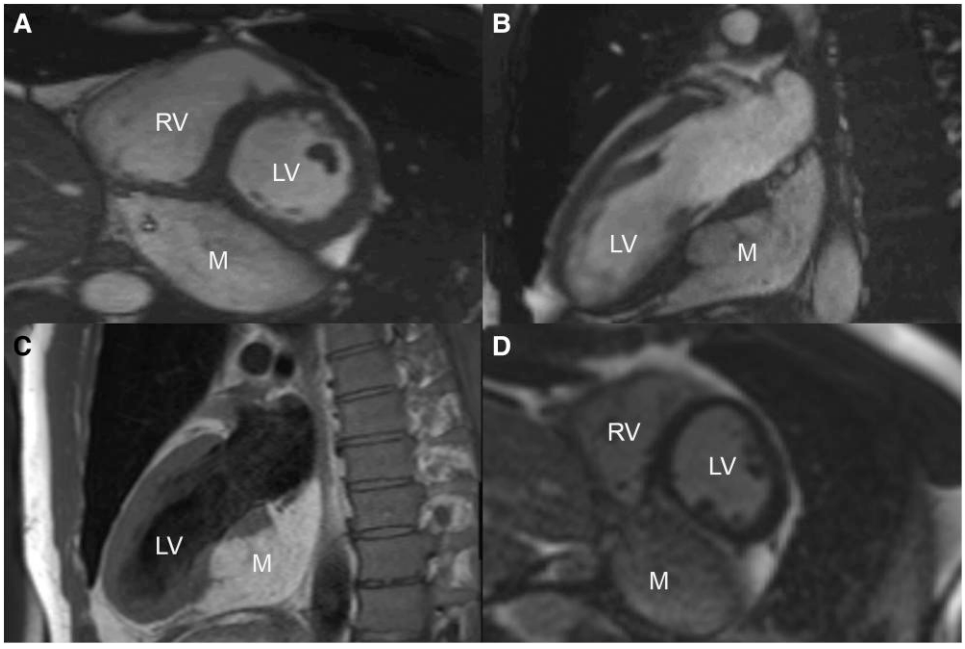


Figure 3 Cardiac structural MRI. (*A*) Short-axis at level of MV papillary muscles. (*B*) 2-chamber view. Demarcation of the epicardial mass (M) with myocardial infiltration and focal thinning of the mid-inferior wall. (*C*) T1-Weighted dark blood imaging demonstrating homogenous lipomatous tissue characterization of the epicardial mass. (*D*) LGE at epicardial tumour interface to myocardium at the inferior and inferolateral wall. See Supplementary material online, *Video S2*.

FDG-CT PET (*Figure 4D*) showed no FDG-uptake of the epicardial lesion relative to the LV myocardium and confirmed no surrounding lymphadenopathy.

This unusual case was discussed in the Oncology and Sarcoma multidisciplinary meeting (MDT). Multimodality imaging did not demonstrate features of malignancy to suggest an underlying sarcoma. The consensus was that the mass represented a large epicardial lipoma with myocardial infiltration and complex involvement of the cardiac circulatory system.

The risks of tumour resection (myocardial infarction due to circulatory involvement and myocardial rupture) were deemed prohibitive by the cardiothoracic surgeons. Consequently the patient was referred to the regional cardiac transplantation service. Unfortunately, the patient was considered unsuitable for cardiac transplantation at present due to a history of smoking and recreational drug use. Abstinence followed by reassessment was advised.

The patient was commenced on B-blockers to suppress ventricular arrhythmia. Follow-up blood tests showed normalization of liver and renal profiles. An exercise treadmill test was performed to aid risk stratification. The patient achieved Stage 4 of Bruce Protocol, with no sustained ventricular arrhythmia.

The diagnosis of a large invasive epicardial lipoma and ongoing risk of sudden cardiac death was explained to the patient who was counselled on the indication for a secondary prevention implantable cardiac defibrillator (ICD). He declined and also refused a wearable cardiac defibrillator. The patient was discharged and informed to not drive.

Three weeks later he represented to ED with palpitations and haemodynamically unstable VT requiring DCCV. Antiarrhythmics

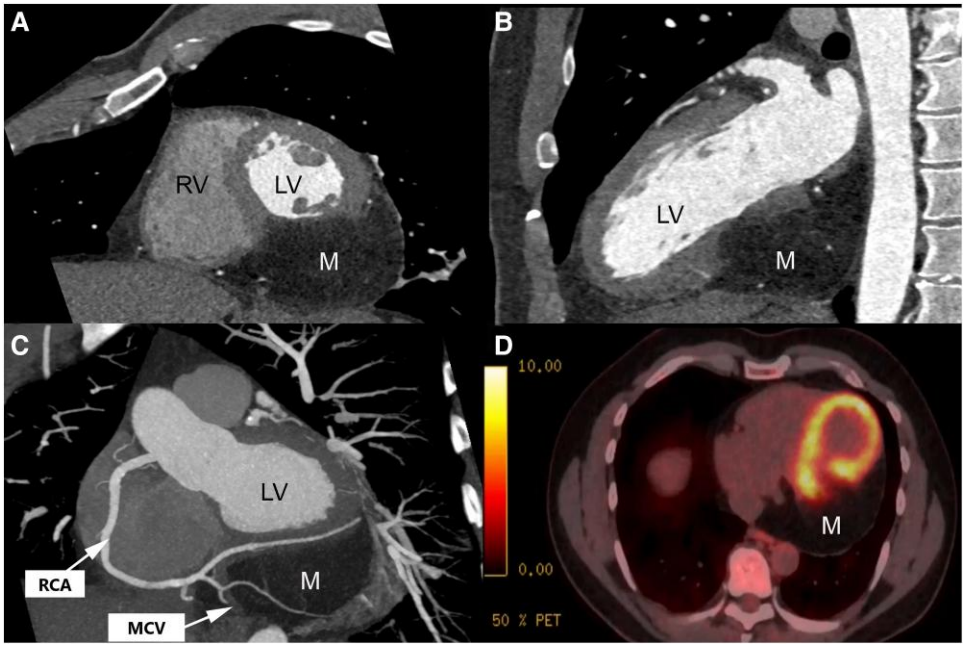


Figure 4 CT coronary angiogram and FDG-CT PET study. (*A*) Short-axis view at level of mid LV cavity, (*B*) 2-chamber view, and (*C*) Optimized view of right coronary artery. Epicardial mass (M) encapsulation of right coronary artery distal branches and middle cardiac vein. (*D*) FDG-CT PET. No FDG-avid uptake of epicardial mass (M) relative to LV myocardium. See Supplementary material online, *Video S3*.

were optimised and the patient was advised again to undergo ICD implantation which he still declined. He agreed for outpatient Cardiology follow-up.

Discussion

Primary cardiac tumours are rare (incidence of ~0.2%).[1] Cardiac lipomas account for 8.4% of primary tumours. The majority of cardiac lipomas are considered benign, slow growing adipose tissue tumours. The size and location can however cause significant haemodynamic compromise and therefore can be malignant in presentation.[2]

This is a rare case of an incredibly large epicardial lipoma precipitating recurrent haemodynamically significant VT. The ECG in VT (*Figure 1A*) has RBB morphology with late transition in V5 and negative QRS in inferior leads, suggestive of a pathological origin site towards the basal inferior myocardium. The VT substrate is likely secondary to a combination of factors. There is direct myocardial infiltration within this region with subtle LGE demonstrated on the tumour interface on CMRI. Furthermore, there is associated regional dysfunction and LV systolic impairment, increasing risk of ventricular arrhythmia. Additionally, due to the complex encapsulation of the coronary arterial circulation, myocardial ischaemia within this territory cannot be entirely excluded.

The case demonstrates how complimentary multimodality imaging can be utilized to assess complex pericardial disease to improve both clinical understanding and management (ESC Guideline 4.1.7).[3] Echocardiography defined the initial abnormal epicardial mass. Tissue characteristics and vascular involvement were subsequently defined by complimentary CMRI and CT respectively, as is advocated in cases of abnormal pericardial thickening or mass (ESC Guideline 3.5.1).[3] FDG-CT PET was useful in excluding an inflammatory lesion and surrounding lymphadenopathy to suggest a malignant process.

There are no specific treatment guidelines on cardiac lipoma management although literature suggests radical surgical resection should be considered in symptomatic patients.[2] An MDT approach was used to formulate a management plan due to case complexity. As a conservative approach was employed, ESC guidelines support offering secondary prevention ICD, where unable to reverse the culprit cause of VT (ESC Guideline 6.2.2.1).[4]

Lead author biography

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Supplementary material

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

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Data availability

The data underlying this article are available in the article and in its online supplementary material.

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