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A giant left ventricular calcified pseudoaneurysm 24 years following acute myocardial infarction: a case report

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A 62-year-old male was admitted with pulmonary oedema. He had a previous history of anterior myocardial infarction in 1997 treated with intravenous fibrinolysis complicated by tamponade requiring surgical drainage. He developed left ventricular (LV) dysfunction [left ventricular ejection fraction (LVEF) 25%] and LV pseudoaneurysm. Despite oral anticoagulation with Vitamin K Antagonist (VKA) with International Normalized Ratio (INR) in the therapeutic range, and low-dose aspirin, he suffered three transient ischaemic attacks in 2009, 2010 again, and 2012 and a stroke in 2017. The LVEF was stable throughout the years, with the first documentation of LV thrombus in 2016. He had not previously shown heart failure symptoms and had no symptoms whilst taking bisoprolol and ramipril. However, the patient elected not to take Bisoprolol for 6 months. On admission, blood pressure was 135/76 mmHg, heart rate 130/min, and he required oxygen at 2 L/min with SpO2 at 97%. Physical examination showed crackles in both lung fields without any other signs of congestion. Biology sample was unremarkable and N-terminal prohormone of brain natriuretic peptide was 659 ng/mL. The electrocardiogram (*Panel A*) revealed Q waves in anterior and anterolateral leads. Chest X-ray (*Panel B*) showed a large heavily calcified LV pseudoaneurysm. LVEF was less than 10% on echocardiography with only a preserved contractility in the basal segments of the LV. Cardiac computed tomography (CT) (*Panels C* and *D*) and magnetic resonance imaging (MRI) (*Panels E* and *F*) showed a severely depressed LVEF (8%) with a 90 mm apical LV pseudoaneurysm without sign of rupture filled with a 12 mm lining thrombus. The LV end-diastolic volume dimension was of 233.86 mL/m^2 (N = 77.0 mL/m^2). The cardiac index was estimated at 1.56 L/min/m^2. Clinical condition improved under intravenous diuretics. He was discharged home under sacubitril/valsartan (24/26 mg, b.i.d.) after Ramipril wash-out, furosemide 40 mg, atorvastatin 40 mg, and increased dose of VKA for a target INR of 2.5–3.5 with the withdrawal of aspirin, without switching for direct oral anticoagulants due to unclear benefit compared to VKA in this situation. Surgical correction of LV pseudoaneurysm was not performed due to very high operative risk with few clinical benefits expected. We recommended the implantation of an internal cardiac defibrillator before discharge; however, the patient refused this intervention despite being provided with information regarding the high risk of ventricular arrhythmias, but however accepted the prescription of a LifeVest (Zoll Medical). He was included in the heart transplant program, with a complete pre-transplant assessment scheduled for a week after discharge. This case allows apprehending the natural course of untreated LV pseudoaneurysm: a low risk of rupture after the acute phase, a calcific remodelling, and a high risk of thrombo-embolic events.

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

Slide sets: A fully edited slide set detailing these cases 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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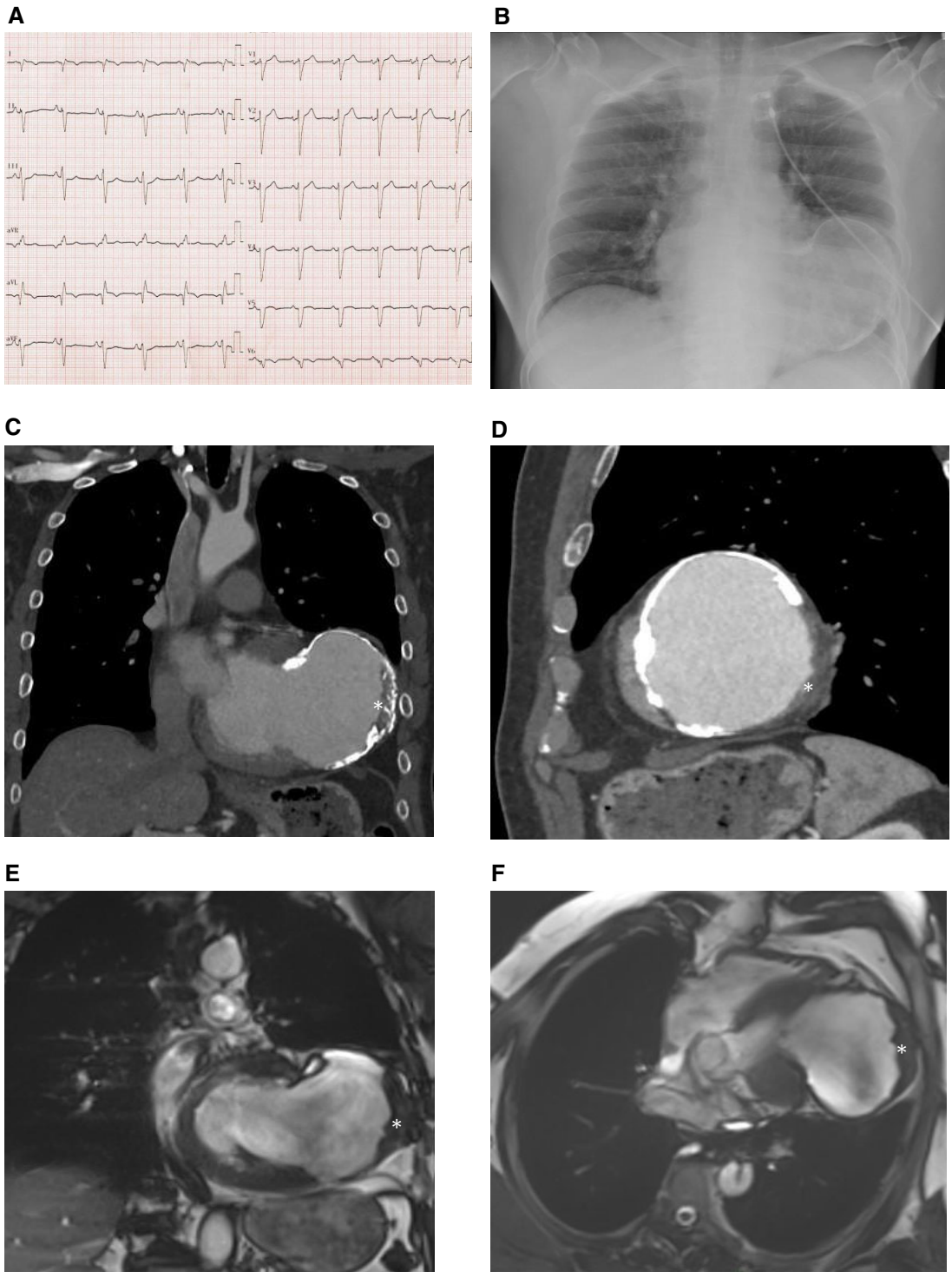

(*Panel A*) Sinus rhythm, right-axis deviation, Q wave in anterior leads (V1–V3) and anterolateral (I, aVL, and V5–V6 leads). (*Panel B*) Chest X-ray showing cardiomegaly with large LV pseudoaneurysm highly calcific. (*Panels C* and *D*) Cardiac CT showing a 90 mm LV apical pseudoaneurysm, with calcific remodelling and thrombus lining onto the apex (*). (*Panels E* and *F*) Cardiac MRI showing large LV pseudoaneurysm with very thin wall, still preserved contractility of basal segment and apical thrombus. (*).