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**CASE REPORT**
*Heart failure*

# Spontaneous coronary artery dissection: a difficult journey from emergency coronary artery bypass grafting to left ventricular assist device

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**Background** Excluding spontaneous coronary artery dissection (SCAD) as an aetiology of acute coronary syndrome in young adults is imperative.

**Case summary** A previously healthy 39-year-old woman experienced sudden severe chest pain, ST-segment elevation on electrocardiogram, necessitating high-dose aspirin and urgent transfer to a revascularization centre. Suffering ventricular tachycardia (VT) and ventricular fibrillation (VF), she underwent two rounds of advanced life support and venoarterial extracorporeal membrane oxygenation. Diagnosed with left main coronary artery (LMCA) SCAD, she was initially started on conservative therapy for declining left ventricular ejection fraction. However, she continued to experience an escalating anginal symptoms, worsening biomarkers, and LMCA SCAD progression, which urged the need for surgical intervention with coronary artery bypass graft surgery (CABG). Following her CABG, she experienced a worsening of her functional mitral regurgitating, which she underwent transcatheter edge-to-edge repair of her severe mitral regurgitation. Despite being listed for orthotopic heart transplantation (OHTx), her low body mass index and elevated antibodies necessitated the HeartMate III left ventricular assist device (LVAD) for bridge to transplant. After treating frequent VT episodes with medications, she eventually received a LVAD as a bridge to cardiac transplantation. Within 1 year of her receiving LVAD, she underwent a successful OHTx.

**Discussion** The pathogenesis of SCAD involves intramural haematoma formation through intimal tears or vasa vasorum haemorrhage. Adverse outcomes that could occur in SCAD patients include cardiac arrest, cardiogenic shock, reduced left ventricle systolic function, and occasionally serious cardiac arrhythmia—such as VF—which can lead to sudden cardiac death. Although most SCAD cases heal spontaneously, revascularization can be considered in case of worsening SCAD progression. Advanced therapeutic intervention including mechanical circulatory support and OHTx should be considered in refractory cases.

**Keywords** Coronary artery bypass grafting • Spontaneous coronary artery dissection • Acute coronary syndrome • Cardiogenic shock • Left ventricular assist device • Bridge to candidacy • Bridge to recovery • Bridge to transplantation

**ESC curriculum** 2.1 Imaging modalities • 3.2 Acute coronary syndrome • 6.2 Heart failure with reduced ejection fraction

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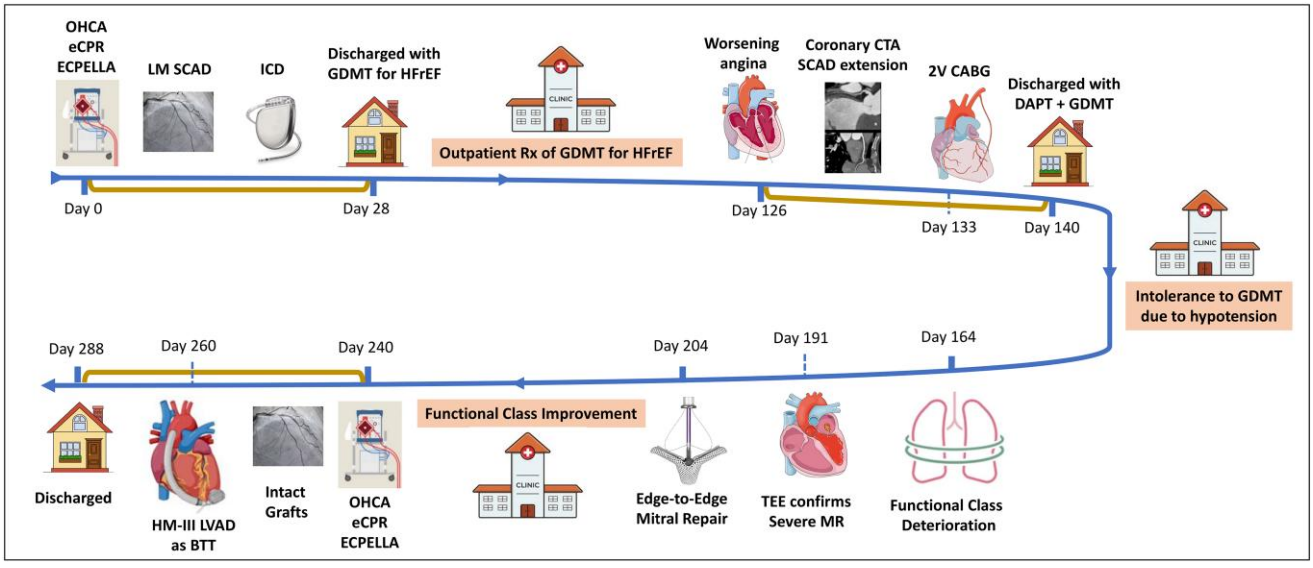
## Learning points

- Spontaneous coronary artery dissection (SCAD) is not uncommon aetiology of acute coronary syndrome in young women and subsequently can result in significant morbidity and mortality.
- The surgical intervention for progressive SCAD may represent an efficacious strategy for mitigating the measures aimed at ameliorating adverse clinical outcomes.
- Advanced heart failure option in the form of left ventricular assist device or heart transplantation can be considered in refractory case of SCAD.

# Introduction

Primary spontaneous coronary artery dissection (SCAD) has a complex pathophysiology, and it comprises 1–4% of all acute coronary syndromes (ACS) and more than one-third of ACS in women younger than 50 years of age.[1] The widely accepted hypothesis on the pathophysiology of SCAD is the occurrence of intramural haematoma by one of the two mechanisms: the occurrence of intimal tear or spontaneous haemorrhage in the vasa vasorum.[1,2] Multiple clinical features have been associated with an increase in the risk of adverse outcomes including cardiac arrest, cardiogenic shock, left ventricular ejection fraction (LVEF) < 35%, and the presence of left main coronary artery (LMCA) SCAD.[1–3] Here, we present a young patient who developed LMCA SCAD and had a prolonged course of multiple interventions and revascularization and eventually underwent durable mechanical circulatory support (MCS).

# Summary figure




# Case presentation

A 39-year-old female—previously healthy—developed sudden onset crushing retrosternal chest pain. Her initial electrocardiogram showed ST-segment elevation in the anteroseptal leads. She received high-dose aspirin and was emergently transferred to the primary revascularization centre. During her transfer to the hospital, she developed sustained episodes of ventricular tachycardia (VT) and ventricular fibrillation (VF) and she required two cycles of advanced cardiac life support till she had a return of spontaneous circulation. On arrival at the emergency department, she developed another episode of monomorphic VT (*Figure 1*), and after a prolonged resuscitation, an extracorporeal cardiopulmonary resuscitation (eCPR) was initiated with venoarterial extracorporeal membrane oxygenation (VA-ECMO) and she was taken emergently to the cardiac catheterization laboratory.

She had no cardiac history, and she is known to have congenital solitary right kidney and migraine. She had two uncomplicated pregnancies delivered at term and one spontaneous miscarriage with two caesarean sections; her last pregnancy was 6 years before this presentation. Following her last delivery, she underwent a placement of a levonorgestrel intrauterine device for contraception. She takes no medication and has no reported history of cigarette smoking, excessive alcohol drinking, or any form of recreational drug use. The differential diagnosis was ACS, acute fulminant myocarditis, or SCAD.

Following her eCPR with VA-ECMO cannulation, she underwent coronary angiography, which demonstrated ulcerated 80% stenosis of the LMCA, angiographically suspicious for SCAD (*Figure 2A*). Intravascular imaging ultrasound was performed using intravascular ultrasound and it showed no flow-limiting lesion, and thus, the decision was made to treat conservatively. Initial transthoracic echocardiogram

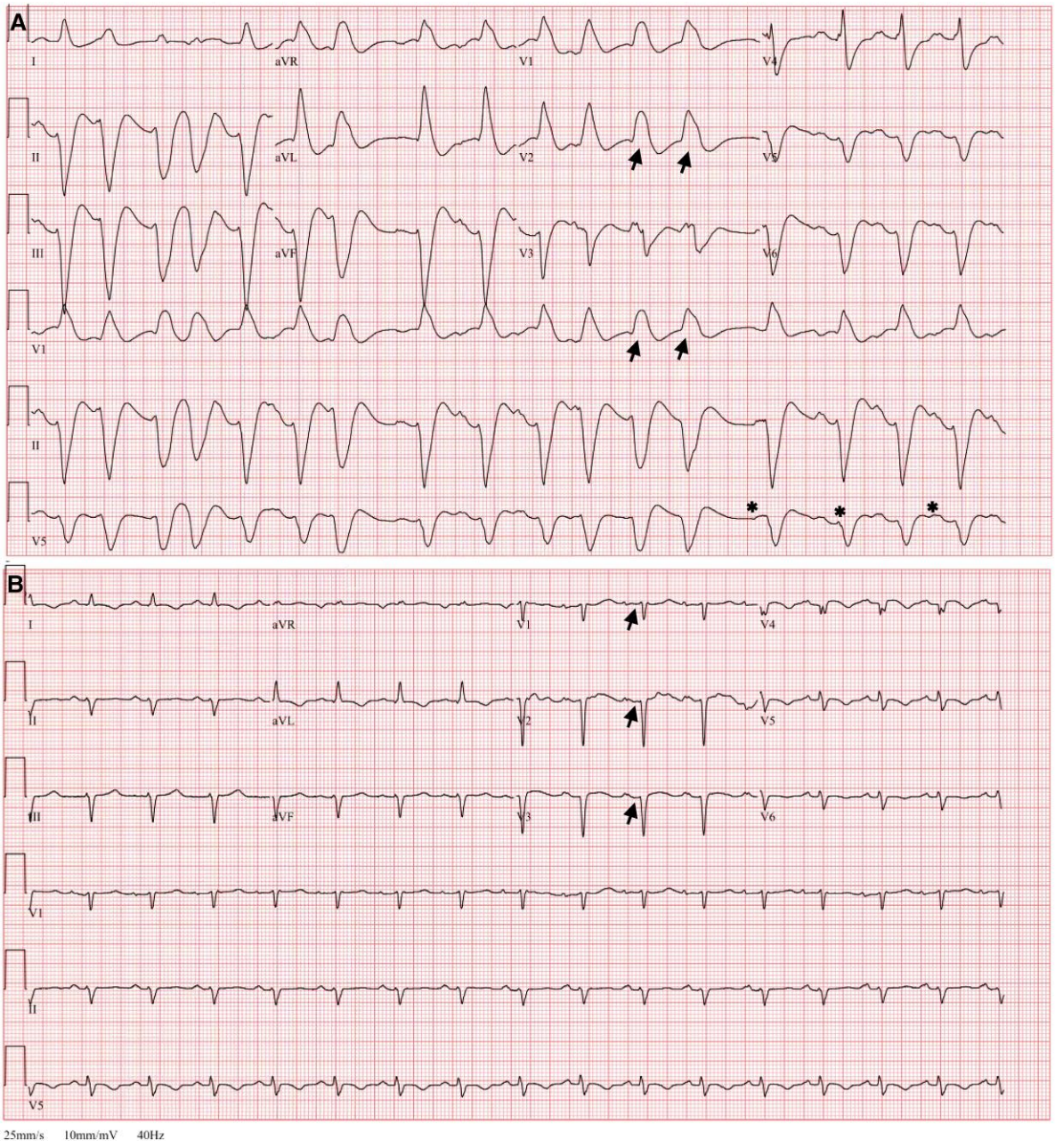



**Figure 1** (*A*) Initial presentation electrocardiogram showing monomorphic ventricular tachycardia with R wave to S wave interval > 100 ms (arrows) and presences of positive R wave and atrioventricular dissociation (asterisk). (*B*) Electrocardiogram on follow-up showing anteroseptal Q wave (arrows).

(TTE) revealed profound LV systolic dysfunction with an estimated LVEF of 5–10% (normal > 55%) with severe diffuse global hypokinesis and with normal right ventricle and no significant valvulopathy. Initial laboratory investigations were notable for high-sensitivity troponin > 27 000 ng/L, venous pH 6.88, bicarbonate 10.3 mmol/L, and lactate 3.9 mmol/L. Her cardiac magnetic resonance imaging revealed dilated ischaemic cardiomyopathy with diffuse non-viable myocardium in the anterior, septal, and lateral myocardium (*Figure 3*).

Following her diagnosis of LMCA SCAD, she underwent temporary MCS with Impella CP (Abiomed, Danvers, MA, USA). She had an uneventful cardiac critical care stay with the ability to wean off and eventually de-cannulated VA-ECMO in the first week. Her LVEF improved to 25–30%, and she underwent an implantable cardioverter defibrillator (ICD) as a secondary prevention of sudden cardiac death (SCD). The decision was made to treat her LMCA SCAD conservatively. She was discharged from her first admission after 4 weeks of hospital stay with guideline-directed medical therapy (GDMT). Further investigation including autoimmune workup and cerebral and renal computed tomography angiography (CTA)—to rule out fibromuscular dysplasia—was unremarkable.

Four months later, she developed recurrent atypical anginal symptoms and these symptoms were associated with an increase in cardiac biomarker [i.e. high-sensitivity troponin 264 ng/L (normal range <

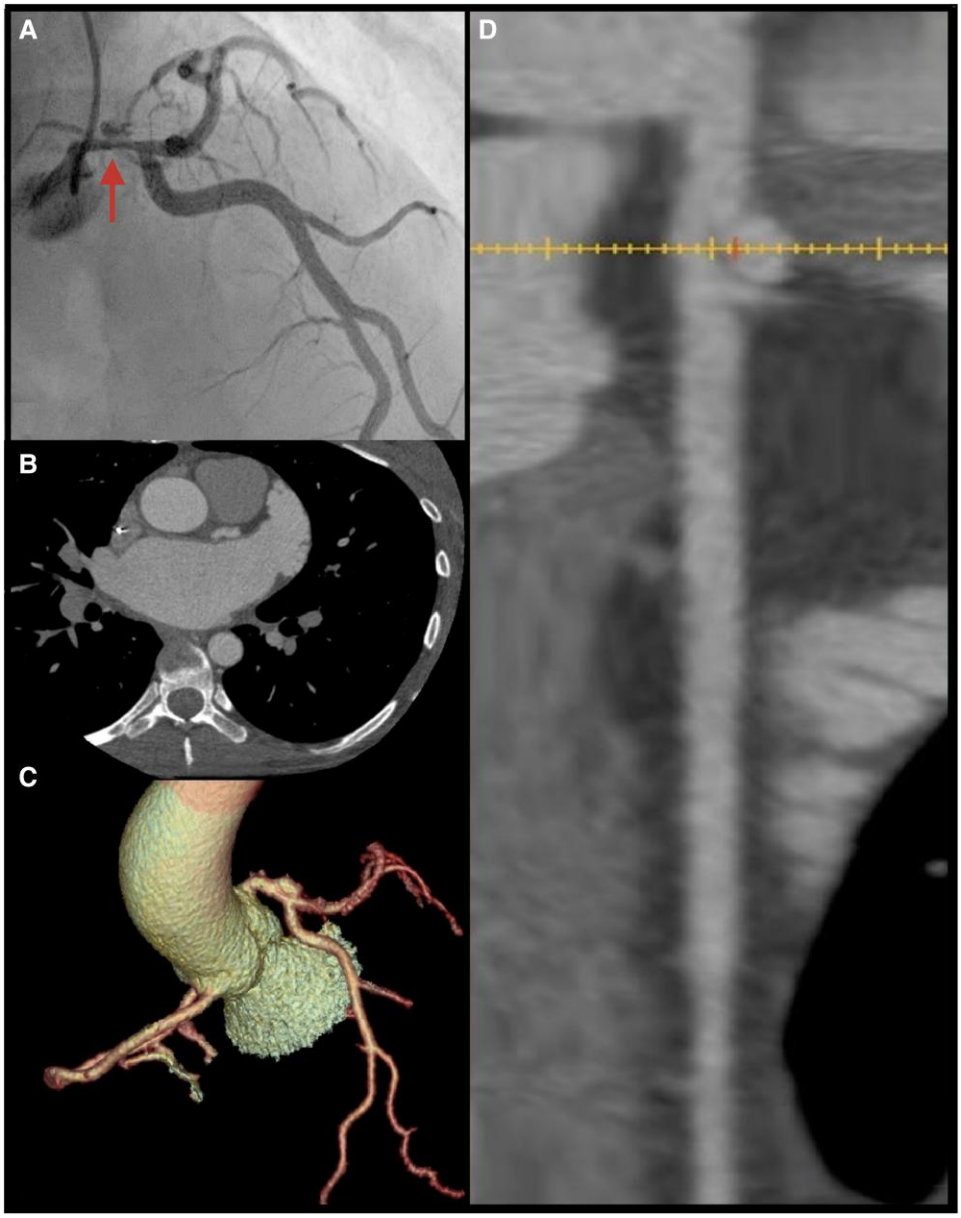



**Figure 2** (*A*) Initial coronary angiogram showing the spontaneous coronary artery dissection in still images (arrow). (*B*–*D*) Cardiac computed tomography with multi-planar reconstruction (*B*, *D*) and 3D imaging (*C*) demonstrating a focal dissection/pseudoaneurysm at the left main coronary artery.

14 ng/L) and N-terminal natriuretic peptide of almost 3000 pg/mL (normal range < 125 ng/mL)]. Implantable cardioverter defibrillator interrogation showed no sustained malignant arrhythmias. Her repeated TTE revealed stable findings with still severely reduced LVEF (15–20%). With suspicion of LMCA SCAD worsening, she underwent cardiac CTA, which showed progression of LMCA SCAD to the left anterior descending (LAD) and left circumflex arteries and the presence of LMCA pseudoaneurysm (*Figure 2*). After a multi-disciplinary team discussion, we elected to proceed with revascularization with coronary artery bypass grafting (CABG). She underwent ligation of LMCA false aneurysm and two coronary bypasses (i.e. left internal mammary artery to LAD and saphenous venous graft to the first obtuse marginal artery). She had no complication following CABG and was discharged in 2 weeks post-operatively.

One month following her CABG, she presented with worsening symptoms of heart failure, and her TTE showed worsening dilation of the left ventricular cavity and worsening her functional mitral regurgitation. Subsequent transoesophageal echocardiography revealed mitral annular dilatation, apical tethering of both leaflets resulting in decreased coaptation and severe posteriorly directed MR with systolic flow reversal in pulmonary veins, and severe LV dilatation and LVEF 15–20% (*Figure 3*). Given her recent sternotomy, she was managed with transcatheter edge-to-edge repair with improvement in her functional status and ability to tolerate GDMT.

In the following weeks, she had episodes of decline in her functional status, and her ICD interrogation showed more frequent episodes of VT, and some events were treated with ICD shocks. She was started on amiodarone (as an intravenous loading dose followed by an oral

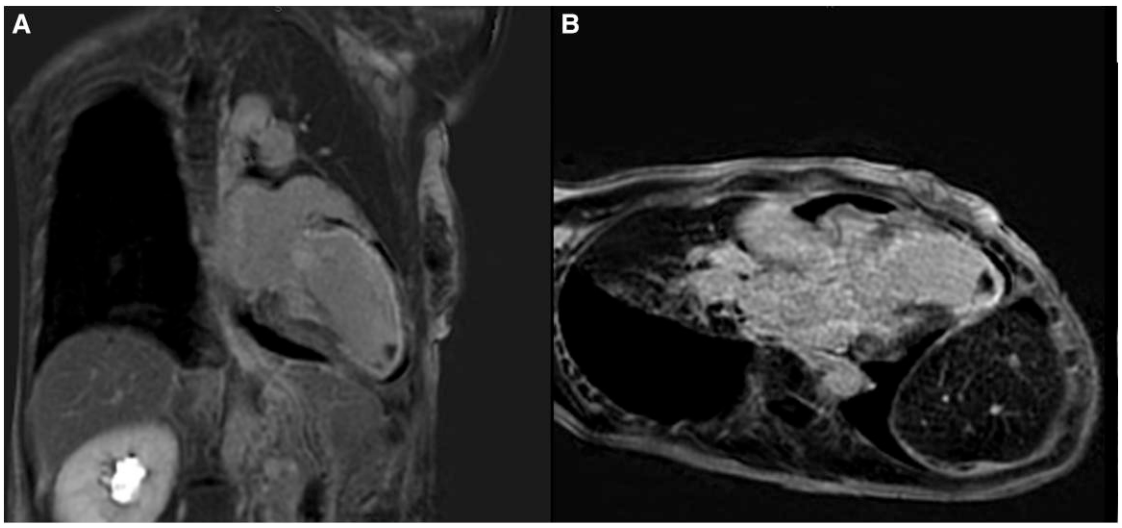



**Figure 3** (*A* and *B*) Cardiac magnetic resonance imaging revealing severely dilated cardiomyopathy with subendocardial–transmural (non-viable) late gadolinium enhancement of the anterior, anteroseptal, and anterolateral segments of the mid and basal left ventricular wall as well as left ventricular apical aneurysm with thrombus and circumferential pericardial effusion.

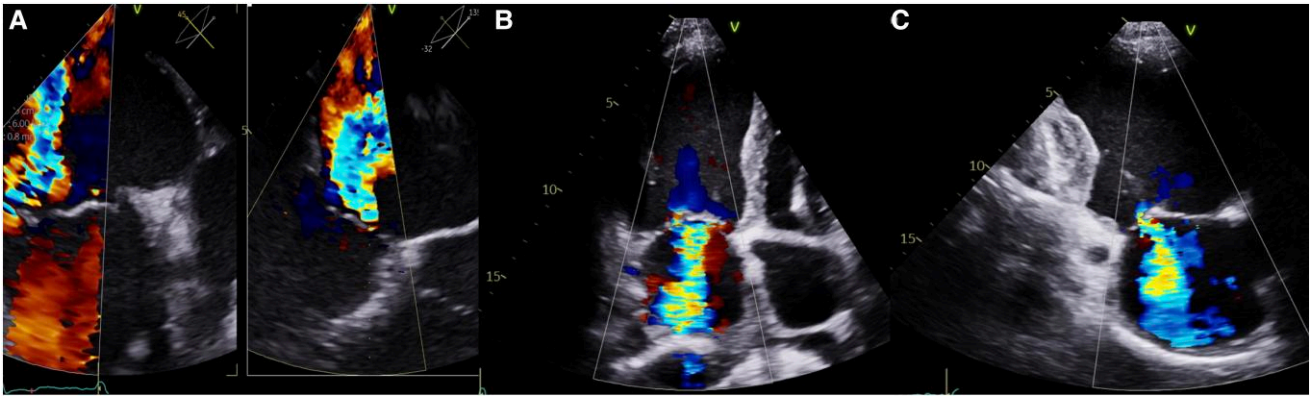



**Figure 4** (*A*) Transoesophageal and (*B*) transthoracic echocardiogram demonstrating severe mitral regurgitation directed posteriorly severe left atrial and ventricular dilatation. (*C*) Transthoracic echocardiogram 1 month after Abbott MitraClip G4 XTW (Chicago, IL, USA), residual mild-to-moderate mitral regurgitation on either side of the clip, more on the posteromedial side.

of 400 mg daily) as an antiarrhythmic agent. Her follow-up TTE showed declining in her LVEF to 15–20% and withdrawal of multiple GDMT due to intolerance. One week later, she presented to the hospital with multiple sustained events of VT that ICD failed to successfully treat with shocks. This was attributed to the failure to capture the malignant arrhythmia. On her arrival at the emergency department, she developed another VF arrest, where she underwent her second eCPR with VA-ECMO. Her repeated coronary angiography showed intact bypass grafts. At this stage, she was formally listed for orthotopic heart transplantation (OHTx).

Despite being listed for urgent OHTx (highest status in Canada), multiple factors including her low body mass index < 19 kg/m$^2$, blood group O, and elevated panel reactive antibodies make her waiting time for OHTx relatively long. In her third week of being on MCS, she underwent a durable left ventricular assist device (LVAD) with HeartMate III (Abbott, Chicago, IL, USA) as a bridge to transplant. Her recurrent frequent VT was managed with a combination of amiodarone and selective metoprolol. She had an uneventful course following LVAD implantation, and during her hospital stay and in subsequent follow-up in 3 and 6 months, she had no further episode of malignant arrhythmia. She was on warfarin for anticoagulation for her LVAD, amiodarone 400 mg daily, and metoprolol 50 mg twice daily. She spent around 7 months in the transplant list before she successfully underwent an OHTx (*Figure 4*).

## Discussion

Spontaneous coronary artery dissection involvement of LMCA is rare compared with other coronaries (1.5%)[3]; thus, invasive coronary angiography is a reasonable form of diagnostic workup to identify the anatomy of coronary arteries. In case of diagnostic uncertainty, non-invasive

cardiac such as CTA has been utilized in the initial diagnostic workup and in the assessment of the healing process.[2,3] In our patient with her second presentation of worsening chest discomfort, we elected to perform CTA to avoid iatrogenic/further propagation of LMCA SCAD. Similar to the rarity of LMCA SCAD, the occurrence of VF among SCAD patients is also uncommon (8.1%), and she eventually underwent ICD for secondary prevention of SCD. She developed episodes of malignant arrhythmias, which were managed with antiarrhythmic medications.

The majority of SCAD will heal spontaneously; thus, the majority of cases were treated conservatively (84.3%) compared with invasive—4.1% percutaneous coronary intervention (PCI)—or CABG in 0.7%.[3] Revascularization is a crucial management consideration in subset of SCAD patients, particularly those with proximal coronary occlusions, unstable rhythm, or haemodynamics and those progressing to occlusion after initial conservative management.[1–3] Various techniques, such as small-diameter balloon angioplasty, cutting balloon angioplasty, standard balloon angioplasty for fenestration, depressurization of the false lumen, proximal and distal edge stenting, and the use of bioresorbable scaffolds, have been suggested to minimize haematoma propagation, although there are limited comparative outcome data available.[2,3] It is imperative to appropriately select patients to undergo a revascularization approach, as PCI carries extraordinarily high rates of procedural complication (i.e. >50%) with 13% requiring an emergency CABG.[4] The decision to pursue invasive vs. conservative treatment for SCAD requires clinical evaluation and multipole cardiac imaging.

In our patient with LMCA SCAD worsening and after multidisciplinary team discussion, we elected to proceed with CABG (ligation of false aneurysm and two bypass grafts). Subsequently, her LVEF continues to worsen with the expectation to have a long wait time in the OHTx list. The potential explanation for her worsening cardiomyopathy and mitral valve regurgitation can be attributed to the presence of non-viable myocardium at the time of surgical revascularization. This non-viable myocardium could worsen the remodelling process and worsen cardiomyopathy and valvular regurgitation.

The use of inotrope in this patient was not a feasible option due to her multiple prior events of VT and VF in the past. Thus, following multi-disciplinary team discussion, she underwent LVAD as bridge to cardiac transplantation. After the implantation of the LVAD, she experienced an uneventful course, with no occurrences of malignant arrhythmias. Eventually, she underwent an uneventful OHTx and had complete recovery and was discharged home.

## Lead author biography

Dr Abdulaziz (Aziz) Joury is a distinguished medical professional with a strong background in cardiovascular medicine and clinical research. Holding an MBBS degree, he is currently a dedicated Advanced Heart Failure and Transplantation Cardiologist at McGill University, Montreal, QC, Canada. Driven by a passion for advancing patient care, he has undergone comprehensive postgraduate clinical training, including an Internal Medicine and Cardiovascular Medicine Fellowship Program at Ochsner Medical Center, New Orleans, LA, USA.

**Consent:** The authors confirm that written consent for submission and publication of the clinical case including image(s) and associated text has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** None declared.

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## Data availability

There are no new data associated with this article.

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