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Balancing Haemorrhagic and Thromboembolic Risk in Spontaneous Coronary Artery Dissection: A Case Report

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Abstract

Spontaneous coronary artery dissection (SCAD) represents a significant non-atherosclerotic aetiology of acute coronary syndrome. In stable individuals, conservative treatment is often favoured; yet, making antithrombotic choices becomes challenging when SCAD is associated with bleeding and a simultaneous compelling indication for anticoagulation. A 69-year-old woman with longstanding atrial fibrillation, hypertension, chronic kidney disease, and previously impaired left ventricular systolic function presented with an inferior ST-segment elevation myocardial infarction (STEMI). She was receiving apixaban for atrial fibrillation and had taken her usual dose on the morning of presentation. An invasive coronary angiogram demonstrated spontaneous dissection of the right coronary artery. This was managed conservatively because she remained haemodynamically stable and pain-free. Serial echocardiograms demonstrated a mild initial increase in the pericardial effusion, without tamponade physiology, followed by stability on subsequent imaging. CT coronary angiogram performed three days after presentation revealed global haemopericardium measuring up to 20 mm and a large left atrial appendage (LAA) thrombus. Apixaban was withheld because of the SCAD and concern that anticoagulation could exacerbate the haemopericardium. Conversely, her longstanding atrial fibrillation and persistent LAA thrombus posed a substantial systemic thromboembolic risk. Serial echocardiograms and repeat cardiac CT demonstrated a stable pericardial collection without tamponade physiology or evidence of ongoing haemorrhage. After multidisciplinary team discussion, we restarted anticoagulation with renal-adjusted rivaroxaban 15 mg once daily. The patient remained clinically stable, and subsequent echocardiography demonstrated progressive regression of the pericardial effusion. However, repeat cardiac CT three months later suggested persistence of the LAA thrombus. This case illustrates how serial multimodal imaging can support individualised anticoagulant decisions when haemorrhagic and thromboembolic risks directly conflict. A negative CT coronary angiogram examination for a residual dissection flap should not supersede a compelling invasive angiographic diagnosis.

Categories: Radiology, Cardiology, Internal Medicine
Keywords: anticoagulation, atrial fibrillation, haemopericardium, left atrial appendage thrombus, multimodality imaging, spontaneous coronary artery dissection

Introduction

Spontaneous coronary artery dissection (SCAD) is caused by sudden separation within the coronary arterial wall unrelated to atherosclerotic disease or trauma. The dissection results in the formation of a false lumen or intramural haematoma that compresses the true lumen and can cause acute coronary syndrome (ACS). This differs from ACS due to atherosclerotic plaque rupture, in which plaque disruption results in superimposed intraluminal thrombus formation [1,2].

In stable patients with preserved coronary flow and no ongoing ischaemia, conservative management is preferred, as spontaneous healing is common. In contrast, percutaneous coronary intervention (PCI) carries specific complication risks, including entry into the false lumen, dissection extension, and intramural haematoma propagation [1-3]. Revascularisation is therefore generally reserved for patients with ongoing myocardial ischaemia or haemodynamic instability [3,4].

There is limited evidence to support routine dual antiplatelet therapy (DAPT) in conservatively managed SCAD, and its potential benefit must be balanced against the risk of bleeding. Anticoagulation is generally discontinued following a diagnosis of SCAD because of concern that it may exacerbate intramural haemorrhage, unless there is a separate compelling indication for its use [1,2]. Management becomes particularly challenging when a haemorrhagic complication coexists with a compelling indication for anticoagulation, such as an established intracardiac thrombus. We report a case of inferior ST-segment elevation myocardial infarction (STEMI) resulting from right coronary artery SCAD, complicated by haemopericardium and the presence of a large left atrial appendage (LAA) thrombus.

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Case Presentation

A 69-year-old woman presented with sharp chest pain radiating to her back following a recent chest infection; the day of initial presentation was designated as Day 0. Her medical background included chronic atrial fibrillation for 10-15 years, hypertension, hypercholesterolaemia, chronic renal disease, and left ventricular (LV) systolic dysfunction. She was receiving apixaban for longstanding atrial fibrillation and had taken her usual dose on the morning of presentation. A previous echocardiogram revealed an LV ejection fraction (LVEF) of 40%, accompanied by hypokinesia in the septal, inferior, and inferolateral regions. Her creatinine clearance upon admission was 46 mL/min. The admission ECG revealed atrial fibrillation with a rapid ventricular response and ST-segment elevation in leads II, III, and aVF, along with reciprocal ST depression in leads I and aVL, consistent with an inferior STEMI. An emergency coronary angiogram (Figure *1*) revealed a spontaneous dissection of the right coronary artery (RCA); the left main stem, left anterior descending, and circumflex arteries were unobstructed. Given her haemodynamic stability and absence of ongoing ischaemia, the RCA was treated conservatively without percutaneous coronary intervention.

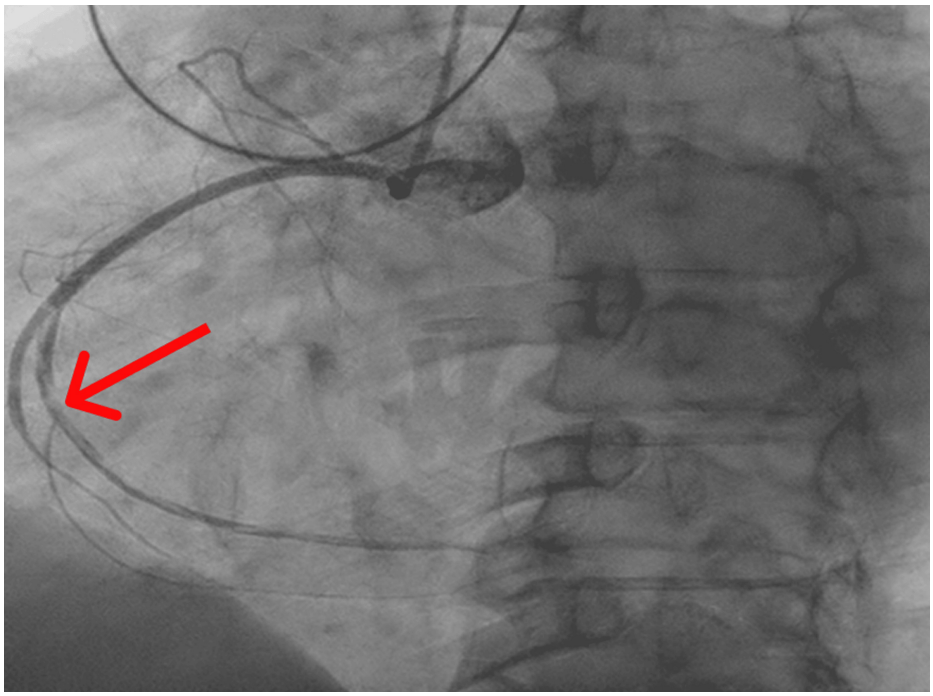


FIGURE 1: Day 0 coronary angiogram demonstrating dissection of the right coronary artery (red arrow)

A transthoracic echocardiogram was performed, revealing impaired LV systolic function, with an LVEF of 35-40%, and regional wall motion abnormalities in the inferoseptal, inferior, and anteroseptal segments. Small pockets of pericardial effusion were seen, including a pocket measuring 0.95 cm adjacent to the right atrium (Figure *2*). A CT aortogram performed on Day 0 after the invasive coronary angiogram, to exclude aortic dissection, demonstrated a high-density moderate-volume pericardial effusion concerning for haemopericardium (Figure *3*), measuring up to 17 mm. As this was a CT aortogram, the coronary arteries were not assessed in detail.

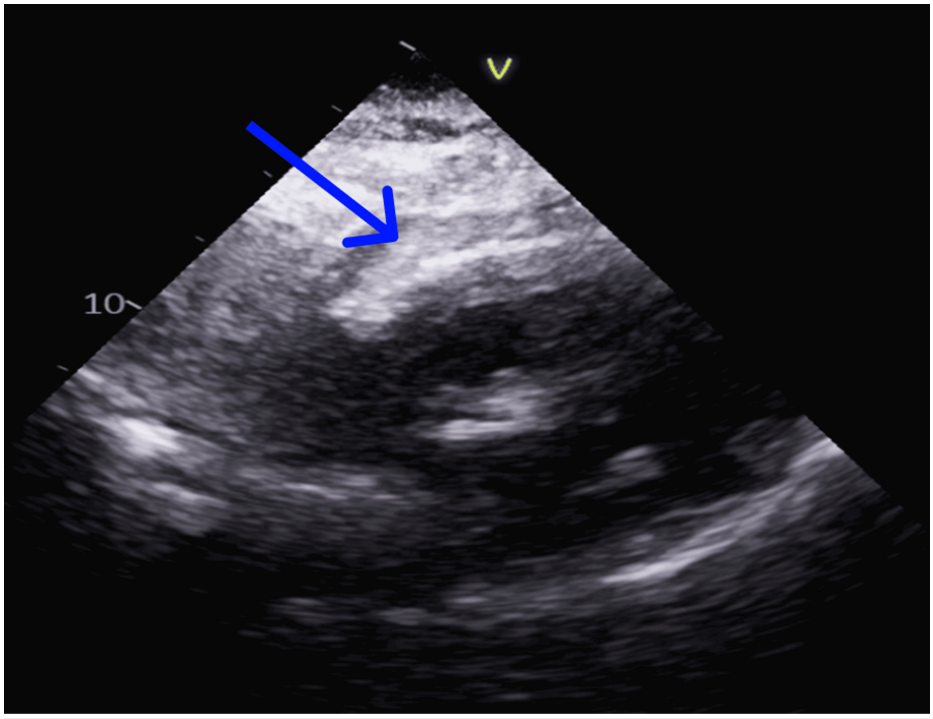


FIGURE 2: Day 0 echocardiogram demonstrating a pericardial effusion (blue arrow)

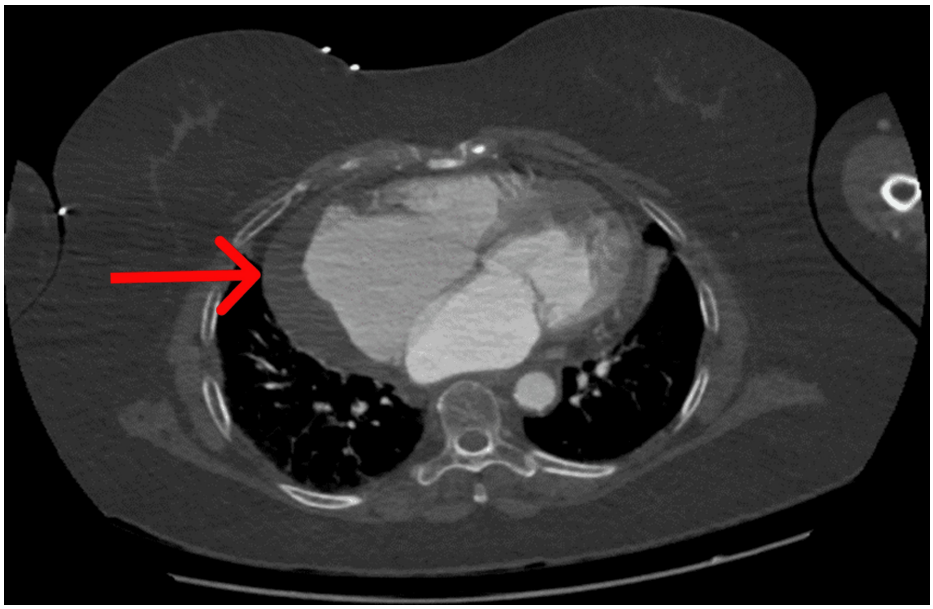


FIGURE 3: Day 0 CT aortogram demonstrating haemopericardium (red arrow)

CT coronary angiography performed on Day 3 showed a normal-calibre proximal RCA and an irregular, tapered distal RCA continuing into a small-calibre posterior descending artery. No residual dissection flap or coronary thrombus was visualised. Importantly, CT coronary angiography demonstrated global moderate haemopericardium measuring up to 20 mm and a large LAA thrombus (Figure *4*). Apixaban was then withheld because of concern that anticoagulation could exacerbate the haemopericardium. These findings created a significant therapeutic dilemma. Anticoagulation could worsen the haemopericardium and precipitate tamponade, whereas withholding it in the presence of a large established LAA thrombus places the patient at risk for systemic embolism and stroke.

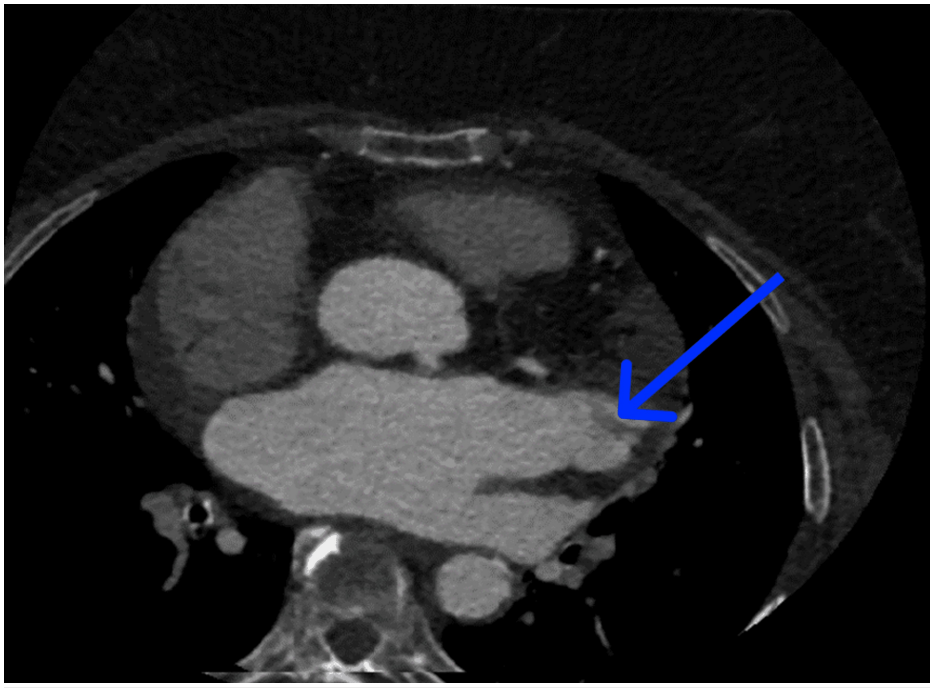


FIGURE 4: Day 3 CT coronary angiography demonstrating a large left atrial appendage thrombus (blue arrow)

On Day 4, a repeat echocardiogram showed a mild-to-moderate pericardial effusion anterior to the right atrium, measuring 0.91-1.43 cm, with no evidence of right atrial or right ventricular collapse. The LVEF was estimated at approximately 45%. A focused echocardiogram performed on Day 8 revealed a persistent pericardial effusion measuring 0.7 cm adjacent to the right ventricle and 1.5 cm adjacent to the right atrium. Repeat cardiac CT on the same day demonstrated a mild-to-moderate pericardial effusion with no interval change. The collection now appeared as simple pericardial fluid, with no CT features to suggest haemopericardium; the LAA thrombus remained present.

Following multidisciplinary team discussion, the stable size of pericardial effusion and absence of tamponade physiology favoured the decision to proceed with anticoagulation. Rivaroxaban 15 mg once daily was commenced for atrial fibrillation and the established LAA thrombus, adjusted for a Cockcroft-Gault creatinine clearance of 46 mL/min. Clopidogrel 75 mg once daily was continued as single antiplatelet therapy. Other treatments included bisoprolol, losartan, eplerenone, dapagliflozin and high-intensity atorvastatin.

The patient continued to be clinically and haemodynamically stable and was discharged on Day 11. A focused echocardiogram on Day 12 (Figure *5*) showed a small effusion measuring 0.76 cm anterior to the right atrium and 0.45 cm anterior to the right ventricle. A subsequent echocardiogram a month later revealed a small residual effusion measuring 0.67 cm anterior to the right atrium. Thus, after anticoagulation was reintroduced, the pericardial collection continued to regress without clinical or echocardiographic evidence of reaccumulation. A repeat cardiac CT performed three months later suggested persistence of the left atrial appendage thrombus.

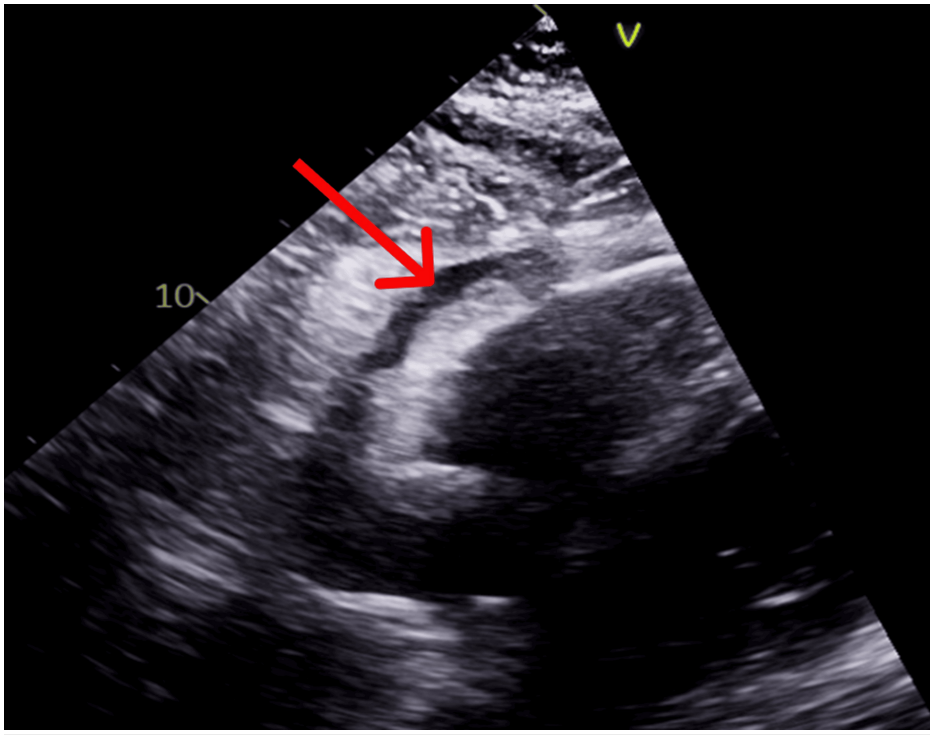


FIGURE 5: Echocardiogram on Day 12, showing small pericardial effusion (red arrow)

Discussion

This case brings together three clinically significant phenomena: inferior STEMI resulting from RCA SCAD, haemopericardium, and a large LAA thrombus necessitating anticoagulation. Its central educational significance is not in any single diagnosis but in the necessity to balance an actual bleeding complication against an immediate and substantial thromboembolic risk.

Conservative management and interpretation of CT

SCAD affects the coronary artery wall and is distinct from atherosclerotic plaque rupture. Contemporary guidelines thus support conservative approaches for stable patients and suggest interventions for ongoing ischaemia, reduced flow and haemodynamic instability, or significant jeopardised myocardial territory [1-3]. This patient's stable presentation and unobstructed left coronary system justified avoiding PCI. The absence of a visible flap on CT coronary angiogram three days after angiographic diagnosis should not be regarded as confirmation of expedited healing. The spatial resolution of coronary CT angiography is less than that of invasive angiography, particularly in small distal arteries, and acute SCAD may present as abrupt constriction of the lumen, tapering, or subtle wall changes rather than a discrete flap [3-7]. The residual irregular tapering of the distal RCA was compatible with this limitation. The invasive coronary angiogram therefore remained the definitive diagnostic study.

Haemopericardium and competing antithrombotic risks

Haemopericardium is an uncommon but potentially life-threatening complication of acute cardiac events, as accumulation of blood within the pericardial space may progress to cardiac tamponade [8]. In this patient, cardiac CT characterised the initial pericardial collection as haemorrhagic, whereas echocardiography demonstrated no evidence of haemodynamic compromise. The exact mechanism of haemopericardium could not be determined from the available imaging. Although its temporal association with RCA SCAD and STEMI suggests a possible relationship, neither frank coronary rupture nor myocardial free-wall rupture was demonstrated.

Expert recommendations advise discontinuing anticoagulation following a diagnosis of SCAD unless there is a separate compelling indication [1-3]. In this patient, the haemorrhagic concern extended beyond the theoretical propagation of an intramural haematoma, as a 20-mm haemopericardium had been documented. Conversely, the large LAA thrombus represented an established thromboembolic risk in a patient with chronic atrial fibrillation. Current atrial fibrillation guidelines recommend oral anticoagulation to reduce thromboembolic risk [9]; additionally, prospective observational evidence has demonstrated thrombus resolution with rivaroxaban in patients with LA/LAA thrombus [10]. However, evidence guiding

anticoagulation in patients with concurrent haemopericardium remains limited.

The decision to reintroduce anticoagulation was guided by serial clinical and imaging assessments rather than a predetermined interval. Serial echocardiograms demonstrated no chamber collapse or other features of tamponade physiology, while subsequent CT imaging showed no progression of the pericardial effusion and no persisting CT features of haemopericardium. These findings, alongside the persistent LAA thrombus, informed multidisciplinary discussion regarding anticoagulation. Apixaban was changed to once-daily rivaroxaban to simplify the anticoagulation regimen and potentially support treatment adherence. Rivaroxaban was prescribed at a renal-adjusted dose.

Clopidogrel was continued as single antiplatelet therapy following the acute STEMI presentation, despite conservative management without PCI. This represented an individualised multidisciplinary team (MDT) decision, balancing the potential ischaemic benefits against the substantial risk of bleeding. However, the benefit of antiplatelet therapy in conservatively managed SCAD remains uncertain, particularly when combined with oral anticoagulation in patients at increased bleeding risk [2,3].

The subsequent reduction in the size of the pericardial effusion was reassuring but should not be interpreted as evidence that anticoagulation can routinely be reintroduced safely in patients with SCAD and haemopericardium. Furthermore, repeat cardiac CT imaging at three months suggested persistence of the LAA thrombus, highlighting the ongoing thromboembolic risk despite the reintroduction of anticoagulation.

Role of multimodality imaging

Echocardiography and cardiac CT provided complementary information to guide clinical decision-making. As the first-line imaging modality for evaluating pericardial disease, echocardiography facilitated serial bedside assessment of pericardial effusion size and its haemodynamic significance [8,11]. In contrast, cardiac CT provided more detailed anatomical characterisation of the pericardial collection, identified the LAA thrombus, and demonstrated an interval change from haemorrhagic to simple fluid appearance [8,11]. Together, these imaging modalities enabled a dynamic assessment of the evolving anatomical and physiological findings, informing the decision to withhold and subsequently reintroduce anticoagulation.

Conclusions

In patients with SCAD-related myocardial infarction and haemopericardium who also have a compelling indication for anticoagulation, management requires careful consideration of competing haemorrhagic and thromboembolic risks. In this case, haemodynamic stability, the absence of progressive pericardial effusion on serial echocardiography, and the resolution of haemorrhagic features on repeat CT supported the cautious reintroduction of anticoagulation in the presence of a significant LAA thrombus.

As a single-patient case report, the observed outcome cannot be generalised to other patients with SCAD and haemopericardium. Furthermore, antithrombotic management was guided by individualised multidisciplinary assessment rather than an established treatment protocol, reflecting the limited evidence available for this clinical scenario, particularly regarding the use of antiplatelet therapy alongside oral anticoagulation. Ultimately, this case highlights the value of serial multimodality imaging in informing individualised treatment decisions when haemorrhagic and thromboembolic risks coexist.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Rashaan Jackson-Wade, Ali Lordo, John R. Davies

Acquisition, analysis, or interpretation of data: Rashaan Jackson-Wade, Ali Lordo, John R. Davies

Critical review of the manuscript for important intellectual content: Rashaan Jackson-Wade, Ali Lordo, John R. Davies

Drafting of the manuscript: Ali Lordo

Supervision: John R. Davies

Disclosures

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