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CASE REPORT
Coronary intervention

Juxtaposition of urgent angioplasty results in spontaneous coronary artery dissection: a case report of fresh vs. organized intramural haematoma

Hannah Z. R. McConkey and Giovanni Luigi De Maria *

The Heart Centre, John Radcliffe Hospital, Headley Way, Headington, Oxford OX3 9DU, UK

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Background Coronary intramural haematoma from spontaneous coronary artery dissection (SCAD) presents as an acute coronary syndrome, usually in young or middle-aged female patients. Conservative management in the absence of ongoing symptoms is best practice, and the artery eventually heals fully.

Case summary A 49-year-old female presented with a non-ST elevation myocardial infarction. Initial angiography and intravascular ultrasound (IVUS) demonstrated typical intramural haematoma of the ostial to mid left circumflex artery. Initial conservative management was selected, but the patient developed further chest pain 5 days later and with worsening electrocardiogram changes. Further angiography was carried out demonstrating near-occlusive disease with organized thrombus in the false lumen. The angioplasty result from this is juxtaposed with another acute SCAD case on the same day with fresh intramural haematoma.

Discussion Reinfarction is a common occurrence in SCAD, and little is known about how to predict it. These cases demonstrate the appearance on IVUS of fresh vs. organized thrombus and the relative angioplasty result in each case. Follow-up IVUS due to ongoing symptoms in one patient demonstrated significant stent malapposition, not apparent at the index intervention, in all likeliness due to intramural haematoma regression.

Keywords Spontaneous coronary artery dissection • Intramural haematoma • Coronary angioplasty • Intravascular imaging • Case report

ESC Curriculum 3.2 Acute coronary syndrome • 2.1 Imaging modalities • 3.4 Coronary angiography • 3.1 Coronary artery disease

Learning points

- Conservative management is preferred in the setting of spontaneous coronary artery dissection where possible, but if there is ongoing pain and electrocardiographic evidence of ischaemia, coronary revascularisation is required.
- The result from urgent angioplasty in this setting is unpredictable, and follow-up optimization of intervention can be scheduled after healing and absorption of the intramural haematoma.
- There is no way of predicting when disease progression will occur in an affected artery. Organized thrombus formation may be compatible with a more favourable angiographic outcome in comparison with the second.

* Corresponding author. Tel: +447764488915, Email: drhzrm@gmail.com
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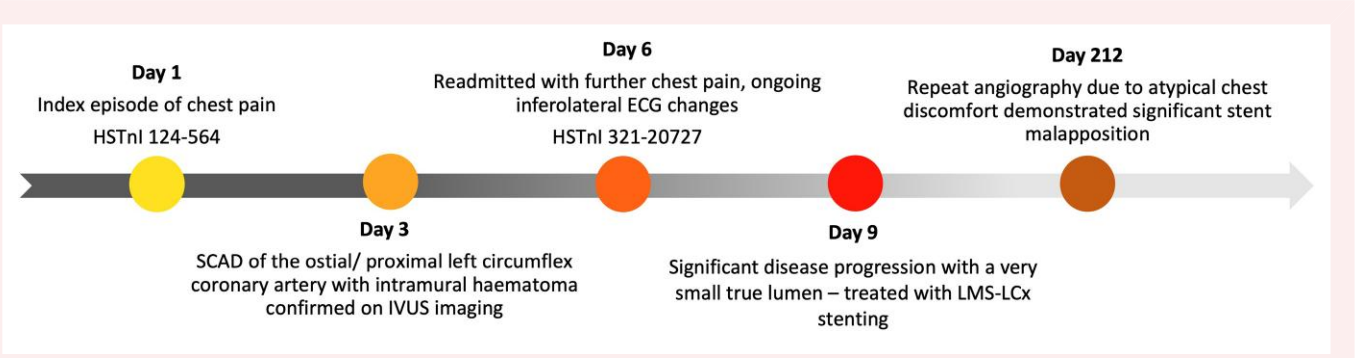
Introduction

Spontaneous coronary artery dissection (SCAD) is the acute development of a false lumen within the coronary artery wall which may compromise coronary flow by external compression of the true lumen.[1] It predominantly affects younger women without conventional risk factors for atheroma. The favoured approach is conservative where coronary flow is not impaired; however, occasionally, it is necessary to intervene due to ongoing symptoms and signs of ischaemia. The result from percutaneous coronary intervention (PCI) in these cases can be unpredictable—stent placement can cause 'tooth-pasting'–like propagation of the intramural haematoma and dissection, either distally or proximally, with occlusion of side branches and persistent suboptimal flow in main vessel. Recent analysis from three national cohort studies demonstrated PCI complications were high in SCAD patients, occurring in 38.6% (serious in 13%) of cases outside the context of ST elevation myocardial infarction, cardiac arrest, or proximal dissections.[2] This was primarily due to more extensive and proximal dissections and longer stents, and predictors of complications were more extensive and proximal dissections and a lack of angiographic contrast penetration of the false lumen. In this report, we describe the case of an acute coronary syndrome secondary to SCAD, juxtaposed with another case on the same day, with similar clinical presentation but very different angiographic results from intervention.



Case presentation

A 49-year-old female patient presented with an unheralded episode of chest pain at rest. She was a smoker with a body mass index of 34 kg/m^2, but had no past medical history or cardiac family history. Initial high sensitivity troponin I (hsTnI) was moderately elevated at 124 and 564 ng/L with no localizing electrocardiogram changes. Clinical examination was unremarkable other than an early diastolic murmur. Echocardiography showed mild aortic root enlargement, moderate aortic regurgitation, and moderately impaired left ventricular systolic function.

Following transfer from the local hospital to our tertiary centre, coronary angiography demonstrated features consistent with type II SCAD extending from the ostium to mid left circumflex artery and confirmed on intravascular ultrasound imaging (IVUS, Supplementary material online, *Video 1*). This was managed conservatively in the absence of ongoing chest pain and electrocardiogram (ECG) changes, fully aware that intervention would require left main involvement in this young patient and that long-term patency coronary artery bypass grafting would be compromised after spontaneous resolution of dissection and intramural haematoma. Unfortunately, 3 days later, the patient developed further chest pain with further and more significant hsTnI elevation (321 to 20 727 ng/L).

Electrocardiogram demonstrated inferolateral ST changes with ongoing chest pain so repeat urgent angiography was undertaken. At diagnostic angiography, no abnormalities of the right coronary artery were noted, and a sheathless 7.5-Fr catheter (Soft Curve, Asahi, Japan) via the right radial artery demonstrated that the intramural haematoma in the left circumflex artery had progressed with higher degree of stenosis (*Figure 1I* vs. *1II*). Re-imaging with IVUS demonstrated near-occlusive disease and the presence of large thrombus within the false lumen with variable grades of IVUS appearances ranging from fresh/acute thrombus to organized/subacute thrombus (*Figure 2*, Supplementary material online, *Video 2*). A 2.5-mm Wolverine cutting balloon was sequentially inflated along the length of the haematoma, with repeated imaging demonstrating no significant interval change. An intentionally long stent was chosen (3.0 × 48 mm Synergy, Boston Scientific) and advanced well beyond the distal haematoma, back to the ostium of the left main stem, and inflated at low pressure. Post-dilatation of the stent body was carried out with a 3.5-mm non-compliant balloon and proximal optimal technique (POT) performed in the left main stem with a 5.5-mm non-compliant balloon. Kissing inflation of the left anterior descending and left circumflex bifurcation with two 4.0-mm non-compliant balloons was followed by a final POT. Intravascular ultrasound confirmed excellent stent expansion and apposition, with no evidence of intramural haematoma beyond the distal stent. The patient was subsequently discharged with plan for ongoing aortic root and valve surveillance.

By pure coincidence, on the same day, a female 60-year-old ex-smoker with no past medical history or relevant family history was brought emergently to the catheter laboratory with inferior ST elevation. Angiography demonstrated a normal left coronary system and dominant right coronary artery with pre-occlusive disease in the proximal segment compatible with intramural haematoma which was confirmed on IVUS (*Figure 3*, Supplementary material online, *Video 3*).

There was a mandate for intervention in view of ongoing chest pain and ST elevation, so the vessel was pre-dilated with a 2.5-mm Wolverine cutting balloon and stenting was deemed necessary because of significant residual stenosis with ongoing chest pain. Notably, extensive stenting back to the ostium was deemed necessary due to propagation of haematoma both distally and proximally with a small area of dye in the aortic root. Intravascular ultrasound confirmed good expansion and apposition of the stent with good coverage to the ostium and sealing the false lumen. There was resolution of symptoms and signs of ischaemia, so the case was concluded with TIMI3 flow distally despite some degree of distal propagation of the haematoma.

The first patient represented with atypical angina a few months after the initial presentations and was re-referred for angiographic follow-up. There was a good angiographic result from previous stenting; however, IVUS imaging demonstrated incidental but significant stent malapposition distally (*Figure 4*, Supplementary material online, *Video 4*), which was addressed with IVUS-guided further post-dilatation.

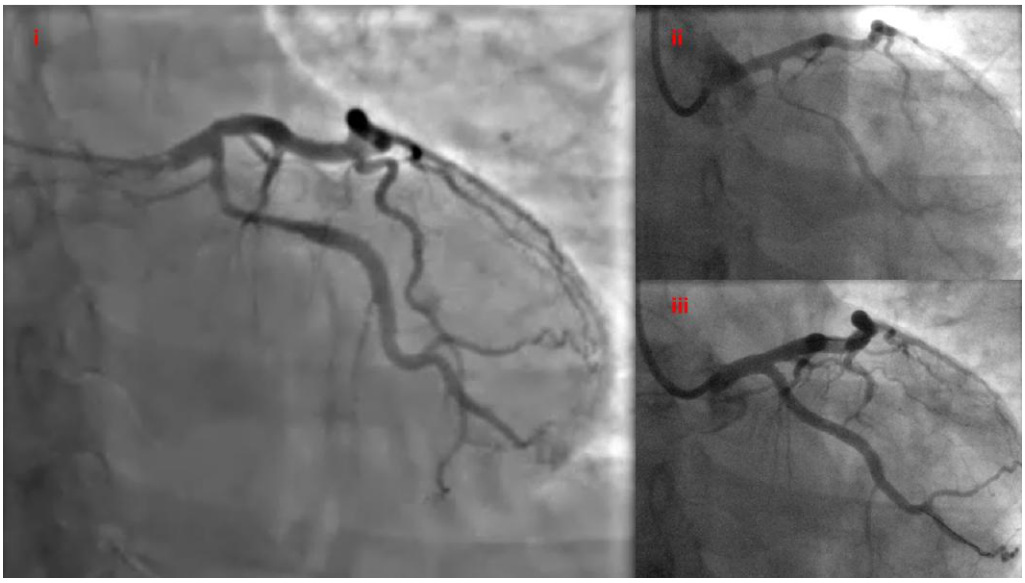


Figure 1 (*I*) Baseline angiogram of the left coronary system, (*II*) follow-up angiogram demonstrating disease progression in the context of intermittent chest pain and inferolateral ECG changes, and (*III*) post-PCI angiography.

Discussion

The first case reveals IVUS imaging of variably organized coronary intramural thrombus, demonstrates failure of cutting balloon technique to evacuate the false lumen when already partially thrombosed, and highlights the possibility for successful angioplasty in these cases, if strictly needed.

Intravascular ultrasound from case 1 demonstrates subacute intramural haematoma as initial anechogenic appearance in the false lumen (*Figure 2, I*), which evolves after 5 days into thrombus with different stages of maturation, namely fresh/acute thrombus with hyper-echogenic appearance and no signal attenuation (*Figure 2, II, D*) and more organized/subacute thrombus with mixed hypo-anaechogenic appearance (*Figure 2, II, C*).[3]

Interestingly, follow-up intravascular imaging revealed significant late stent malapposition, seemingly due to the interim healing of the vessel and regression of intramural haematoma. Recurrent symptoms are very common following SCAD and not necessarily related to stent malapposition. In this group, vasodilator therapies targeting vasospasm or hormonal therapy for cyclical chest pain has anecdotally been helpful.[1]

Up to 4% of patients presenting with acute coronary syndrome have spontaneous coronary artery dissection, and this may account for up to 35% of women aged ≤50 years.[4] Two-thirds of patients have preceding extreme physical or emotional stress,[5] and pregnancy-associated SCAD is most common in the first week post-partum. Arterial fragility from predisposing arteriopathies underlines the predicament when coronary intervention is required in these patients. Spontaneous coronary artery dissection is grouped according to the Saw classification[6] where type I represents a classical angiographic radiolucent 'flap' and linear double lumen where contrast hold-up is often observed. A type 2 pattern (the most common and observed in both cases above) is characterized by a long, smooth stenosis usually in mid-to-distal segments, and type 3 is where the appearance is indistinguishable from a focal atherosclerotic stenosis. There is now evidence to support treatment with single rather than dual antiplatelet therapy in those managed medically[7] since the latter is associated with a significantly higher major adverse cardiovascular event rate (18.9% vs. 6.0%).

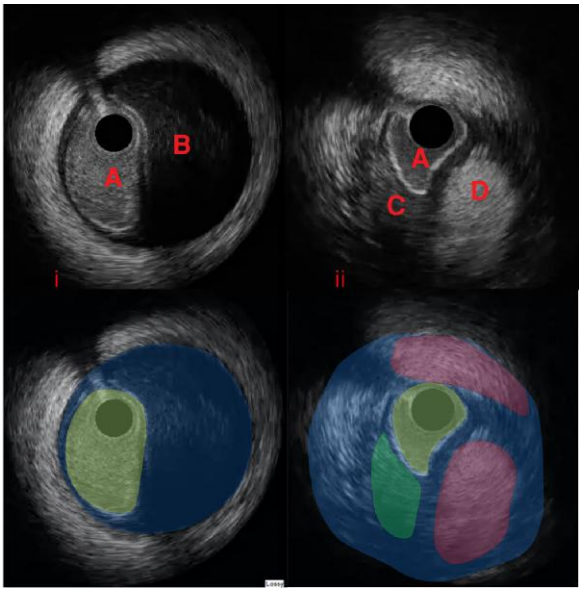


Figure 2 Intravascular IVUS in the first patient at the index procedure (*I*) and at the second presentation with reinfarction (*II*). Image (*I*) demonstrates the true lumen (*A*, yellow) and false lumen (*B*, blue). Image (*II*) demonstrates the true lumen (*A*, yellow), partially organized thrombus with mixed hypo-anechogenic appearance (*C*, green), and fresh hyper-echogenic thrombus (*D*, red).

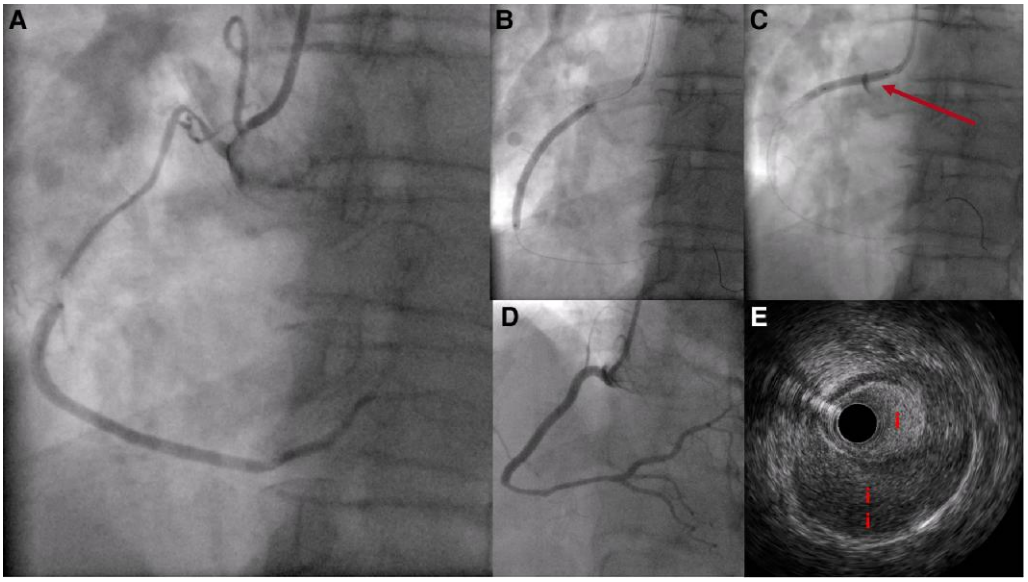


Figure 3 Images from the second case demonstrating initial angiography of the right coronary artery (*A*), placement of a stent (*B*), sealing of the aortic intramural haematoma (red arrow) (*C*), final result after extensive stenting with tooth-pasting appearance distally (*D*), and IVUS (*E*) demonstrating the true lumen (*I*) and false lumen (*II*).

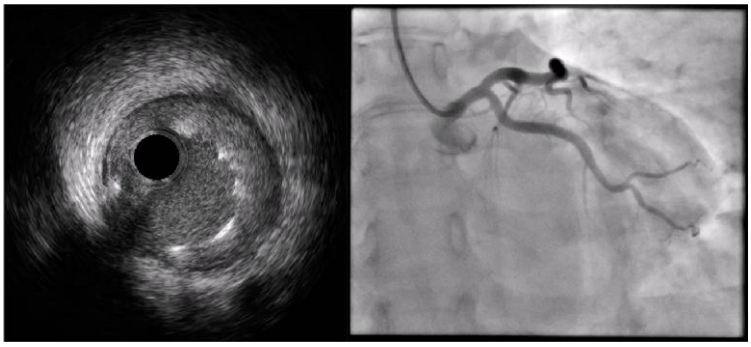


Figure 4 Comparison of false lumen appearances in Patient 1 at two different time points and in Patient 2. In Patient 1, there is clear evolution from a completely fully anechogenic false lumen at Day 3 to a mixed appearance (ranging from hyper-echogenic fresh thrombus to hypo-anechogenic organized thrombus) in line with a thrombotic evolution of the false lumen at Day 9. In Patient 2, and in a more acute phase (Day 0), the false lumen presents appearances ranging from an-echogenicity to echogenicity quite close to the one true lumen, suggesting the presence of fluid blood component, more prone to 'tooth-pasting' phenomenon.

Disease progression is unpredictable and relatively common. Reinfarction can occur in 6.1–17.5% of cases,[8] and some practitioners advocate a longer length of stay to monitor for further ischaemia. There was a striking temporal change in the angiographic and IVUS appearance of intramural haematoma and thrombus formation in the first case between the two procedures taken a few days apart (*Figure 5*). This maturation of thrombus within the false lumen could be advantageous in the setting of required coronary intervention; a thrombosed false lumen may signify resolved intramural bleeding, and therefore, stenting (if required) could be less susceptible to a 'tooth-pasting' phenomenon. Whilst speculative, this is biologically plausible not only because is there less fluid blood within the wall, but also because the vessel wall in the meantime may have started to heal, becoming more rigid due to the thrombus itself. This might be a response to stenting similar to that observed in case of large or hard plaque in the wall, with limited risk of dissection propagation.[9]

Intravascular imaging can provide clear understanding of the coronary architecture which can subsequently aid decision-making. Ongoing research in this area (including the EURObservational Research Programme SCAD registry and https://beatscad.org.uk) is necessary to help answer some of these unknowns.

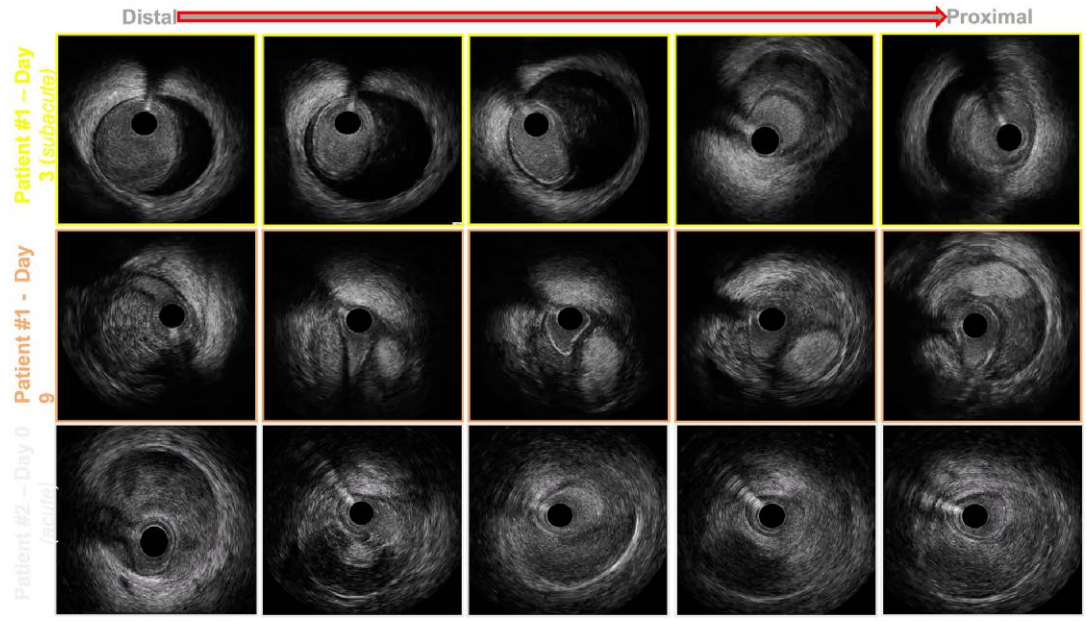


Figure 5 Follow-up IVUS (left) and angiography (right) in case 1, demonstrating significant stent malapposition which is not obvious on angiography alone.

Lead author biography

Dr Hannah McConkey is a final year interventional cardiology fellow at John Radcliffe Hospital, Oxford, UK. She graduated from Cambridge University and King's College London in 2007 and underwent junior doctor rotations in London, Surrey, Berkshire, and Oxford. She was granted a prestigious BHF Clinical Research Training Fellowship in 2016 and was awarded a PhD for her work studying paradoxical low-gradient aortic stenosis at King's College London based at St Thomas' Hospital. Her areas of interest are coronary artery disease and intervention, TAVI, calcium modification, and cardiac physiology.

Supplementary material

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

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: Informed consent was obtained from both patients in accordance with the Committee on Publication Ethics (COPE) guidelines.

Conflict of interest: None declared.

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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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