

Type E coronary artery dissection caused by intravascular lithotripsy balloon rupture; vessel anatomy and characteristics in a lithoplasty complication case as detailed by optical coherence tomography: a case report

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Background

Intravascular lithotripsy is a new method used to treat calcified coronary lesions (CCLs). Percutaneous coronary intervention of CCLs has conventionally been classified as a complex procedure. In the majority of calcified cases, atherectomy is required for sufficient plaque modification prior to stent implantation. Intravascular lithotripsy has been shown to be safe and effective in clinical trials, but as worldwide usage increases, cases of complications are beginning to emerge.

Case summary

We describe a 71-year-old woman, who after an episode of non-ST-elevation acute coronary syndrome underwent coronary angiography. The culprit vessel was identified to be a severely stenosed left anterior descending artery which was also heavily calcified and tortuous. Intravascular lithotripsy (IVL) was employed for calcium modification prior to stent implantation, but the IVL balloon ruptured during shockwave lithotripsy, resulting in coronary artery dissection. Subsequent management steps and stent deployment resulted in favourable angiographic results. Our findings are further detailed on optical coherence tomography, demonstrating certain features which might predispose to IVL balloon rupture.

Discussion

We discuss the mechanism of action during intravascular lithotripsy, and how the shockwaves from the lithotripter modify calcified lesions, whilst keeping soft tissue unharmed. Results from clinical trials and multiple real-world studies have shown that complication rates are low. This case report aims to illustrate how the rupture of an intravascular lithotripsy balloon can result in coronary artery dissection. Optical coherence tomography can help in identifying potential anatomical features which may precede such complications.

Keywords

Intravascular lithotripsy • Complications • Balloon rupture • Coronary artery dissection • Optical coherence tomography • Case report

ESC Curriculum

2.1 Imaging modalities • 2.2 Echocardiography • 9.1 Aortic disease

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Learning points

- Intravascular lithotripsy can cause coronary artery dissection when the balloon ruptures.
- We highlight the immediate clinical management of coronary artery dissection due to intravascular lithotripsy balloon rupture.
- There are specific anatomical characteristics of the calcified coronary lesions which may predispose to intravascular balloon rupture which can be identified on intravascular imaging.
- Precise intravascular imaging prior to choice of calcium modification is vital to avoid risk of complications. Not all calcified plaques are the same.

Introduction

The global rise in life expectancy and increasing prevalence of diabetes has resulted in ever-increasing incidence of calcified coronary lesions (CCLs).¹ Treatment of CCLs is notoriously complex due to their higher rates of complications, longer procedural times, and greater degree of technical prowess required.

The recently introduced Shockwave Coronary Lithoplasty System (Shockwave Medical, USA) delivers acoustic pressure waves to modify calcium and has been shown to be safe in clinical trials, with no instances of coronary artery perforation and very few cases of vessel dissection.^{2,3} However, with the ever-increasing adoption of this technology, more and more complications are being reported. We aim to highlight the factors which predispose to such complications by presenting a case report where a ruptured intravascular lithotripsy (IVL) balloon resulted in a spiral coronary artery dissection.

Timeline

Day 0	A 71-year-old woman with background diabetes, dyslipidaemia, and essential thrombocytosis is referred from her local health services following an episode of non-ST-segment elevation acute coronary syndrome, with persistent chest pain and breathlessness on exertion. Electrocardiogram showed dynamic ST-segment changes in the anterior leads. Echocardiogram showed an ejection fraction of 53%
Day 1	Coronary angiogram followed by heart team discussion and family discussion
Day 2	Percutaneous coronary intervention to the left anterior descending (LAD) artery with intravascular lithotripsy and optical coherence tomography Intravascular lithotripsy balloon rupture occurred with resulting type E spiral dissection in the LAD artery which was successfully treated
Day 3	Recovery in coronary care unit with significant rise from 7 pg/mL (normal values <14 pg/mL) before angioplasty to 89 pg/mL post-angioplasty
Day 6	Well, discharged home on dual antiplatelet therapy, prescribed with Cardiprin 100 mg OD (Aspirin 75 mg plus Glycerin 25 mg) and Ticagrelor 90 mg BD, planned for at least 1-year duration before switching to single antiplatelet therapy
Day 21	Well and asymptomatic during outpatient clinic review

Case presentation

We report a 71-year-old woman, with background diabetes, dyslipidaemia, and essential thrombocytosis. She had an episode of non-ST elevation acute coronary syndrome, presenting with intermittent chest pain at rest. There were no remarkable physical findings on clinical examination.

Routine blood investigations revealed normal biochemical parameters. There were dynamic electrocardiographic changes with deep T-inversions from leads V2–V6, as well as the inferior leads II, III, and AVF. Two-dimensional (2D) echocardiography showed hypokinesia at the basal to mid-anteroseptal and anterior walls, with an ejection fraction of 53%. High-sensitive cardiac troponin on admission was 28 pg/mL (normal values < 14 pg/mL).

Coronary angiography was performed the next day, which showed a normal left main artery and a normal dominant right coronary artery. The left circumflex artery had a chronic total occlusion at its mid-segment, and the left anterior descending (LAD) artery showed severe stenosis at the mid-segment, which was tortuous and calcified (*Figure 1A–D*).

Given the severity of her coronary artery disease, we stopped the procedure and had a heart team discussion about revascularization options. The patient made a personal choice to proceed with percutaneous coronary intervention over bypass surgery, and we planned the procedure the next day.

On the day of the procedure, she was well. Via a transradial approach, we engaged the coronary artery and sent a workhorse coronary wire down to the distal LAD, and another workhorse wire into the large diagonal branch. A conventional non-compliant (NC) 2.5 mm × 15 mm balloon failed to fully expand despite inflation at 18 atmospheres (atm), owing to heavy calcium burden (*Figure 2A and B*).

Optical coherence tomographic (OCT) images confirmed the presence of superficial and concentric calcification throughout the lesion. Of note, there was a protruding calcium nodule along the inner curve of the tortuous bend at the mid-LAD (*Figure 3A and D*).

Given the eccentricity of calcium and tortuosity of the lumen, we decided to proceed with calcium modification by means of IVL, as there was concern about wire bias if rotablation were to be performed. We used a 3.0 mm × 12 mm balloon, deployed at 4 atmospheric pressures (atm). We delivered 3 sets of 10 IVL pulses at the distal, mid, and proximal parts of the lesion. The IVL balloon did not open well at the distal segment, thus we planned a further 10 pulses here. During delivery of the 38th pulse, the patient experienced excruciating chest pain and had a transient drop in her blood pressure.

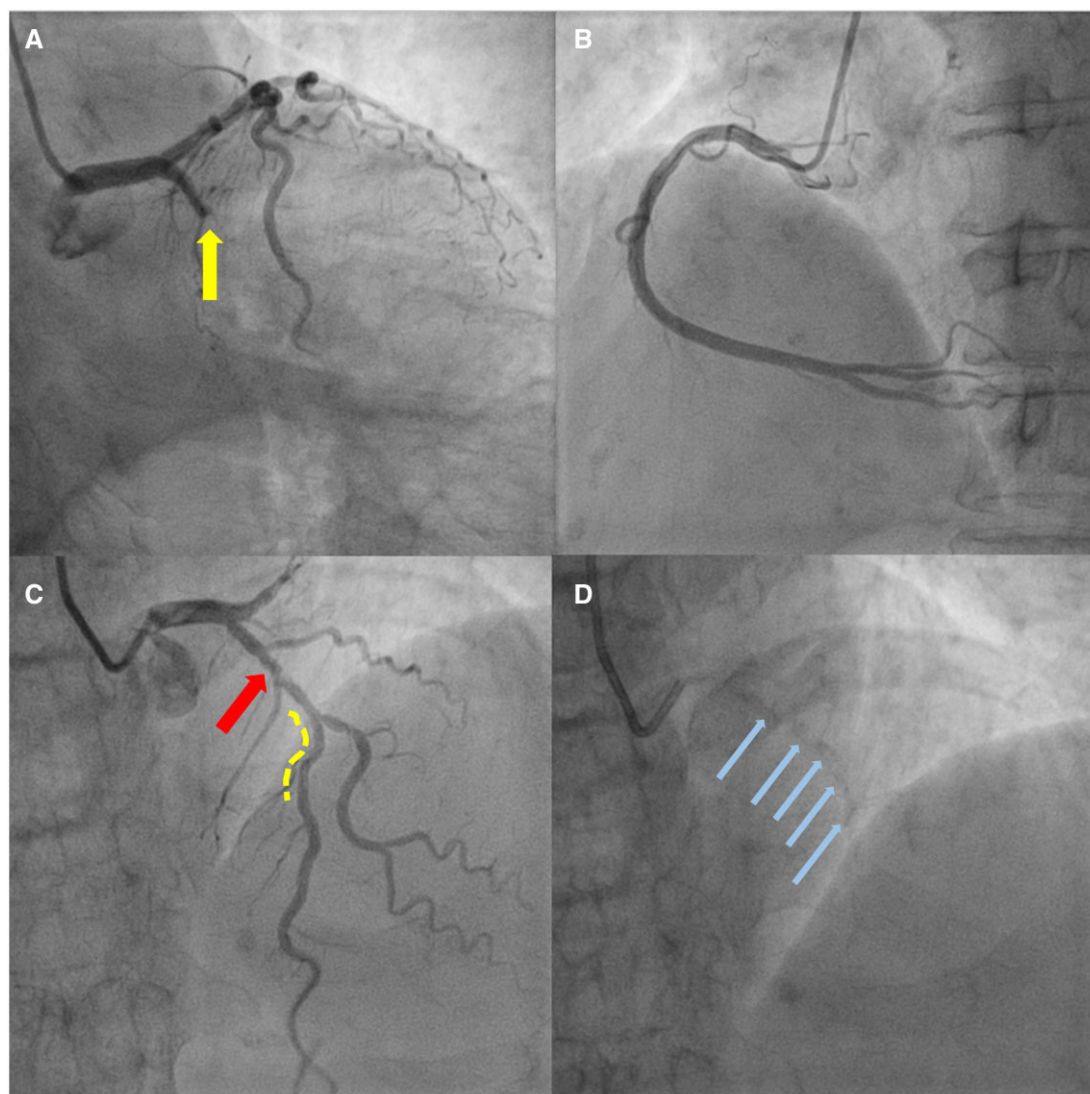


Figure 1 (A) Normal left main with totally occluded left circumflex artery (yellow arrow), (B) normal right coronary artery, (C) severe disease at the proximal to mid-left anterior descending (red arrow) with mid-segment tortuosity (yellow dashed line), and (D) heavily calcified vessel, with calcium visible without contrast opacification (blue arrows).

On fluoroscopy, there was leaking of contrast from the balloon into the subintimal space, tracking distally (*Video 1*). We immediately stopped the pulsations and deflated the balloon (*Figure 4A*).

It was clear at this point that the IVL balloon had ruptured resulting in dissection of the LAD, with visible spiralling of the contrast, persistent filling defects, and contrast staining. By definition, this is a category E coronary artery dissection (*Figure 4B and C*).

After administering pain relief medications, we performed an angiogram which ruled out coronary perforation. An immediate bedside focused 2D echocardiogram also showed no pericardial effusion. Optical coherence tomographic interrogation was considered at this

junction but deferred due to the risk of propagating the dissection during forceful contrast injection. The areas of dissection were quickly stented over with two drug-eluting stents, a 2.75 mm × 28 mm distally and a 3.0 mm × 28 mm stent proximally, with her chest pain resolving thereafter.

Post-stenting OCT (*Video 2*) evaluation was performed, showing that the stents were well expanded, with multiple fissures seen within the calcified plaque. Distally, a large break in the continuity of the vessel intima was seen, indicating a dissection. This dissection was completely sealed behind the stent struts (*Figure 5B and C*). An NC 3.0 mm × 12 mm balloon was inflated at 18 atm for stent

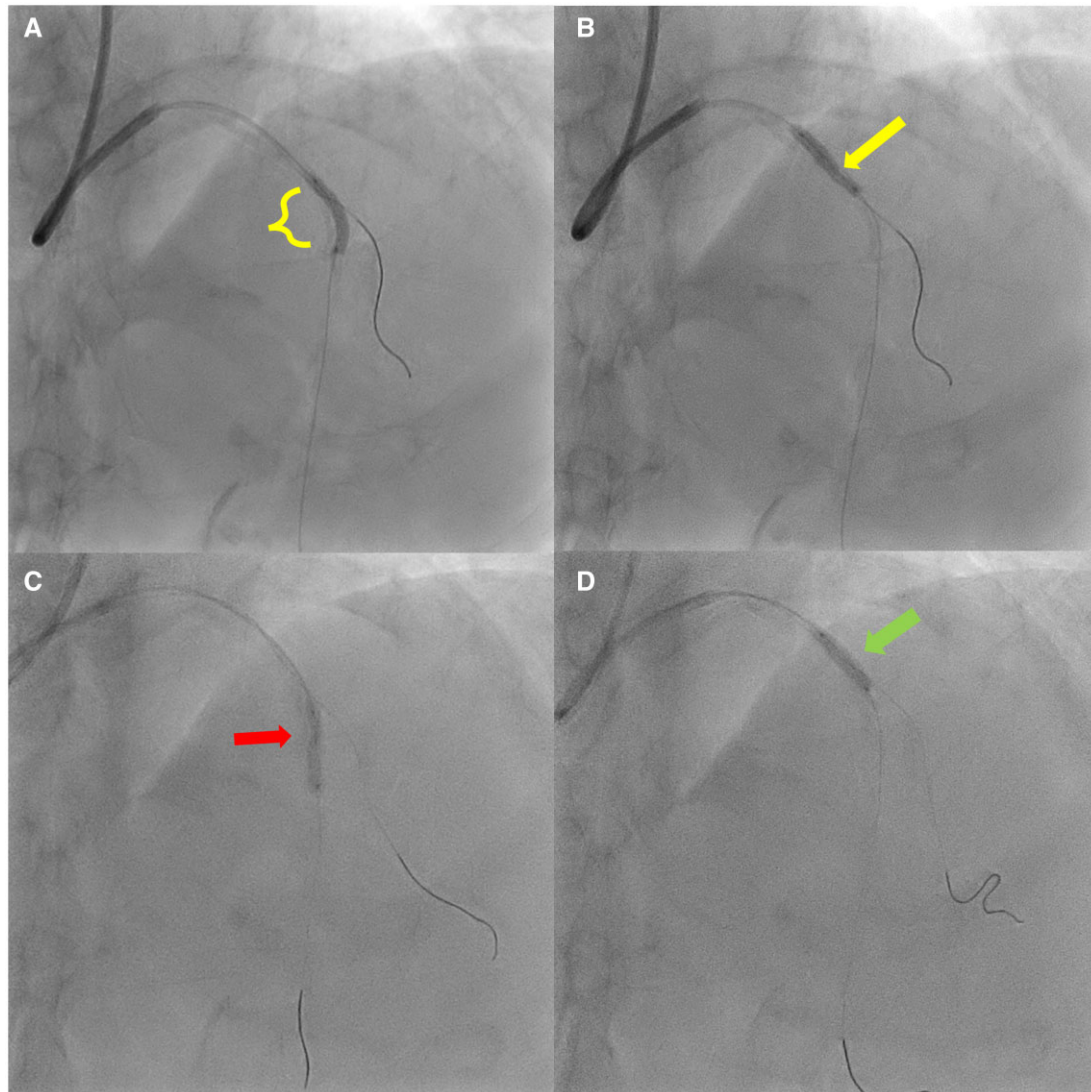


Figure 2 (A) Non-compliant 2.5 mm balloon unable to fully dilate due to the shelf of calcium along the inner curve of the left anterior descending. (B) Same non-compliant balloon with significant waisting (yellow arrow) due to heavy calcification at the proximal segment of the left anterior descending, even at high atmospheric pressures. (C) Intravascular lithotripsy balloon inflated at 4 atm and even after 10 pulses, unable to fully dilate the distal left anterior descending calcium (red arrow). (D) Intravascular lithotripsy balloon successfully opening at 4 atm, indicating successful modification of the proximal left anterior descending calcium.

optimization. Of note, the large diagonal branch appeared pinched on angiogram, but with a Thrombolysis in Myocardial Infarction 3 flow distally, we deferred further intervention. We thus ended the procedure with good angiographic results (Figure 5D). Post-procedural examination of the IVL balloon revealed a small perforation on the balloon surface with the liquid contents leaking out (Video 3).

She was closely observed at our coronary care unit overnight. A repeat high-sensitive troponin-T assay showed significant rise from 7 pg/mL before angioplasty to 89 pg/mL post-angioplasty (normal values <14 pg/mL).

After 2 more days of observation, she was discharged home on dual antiplatelets (DAPT), Cardiprin, and Ticagrelor. She was also

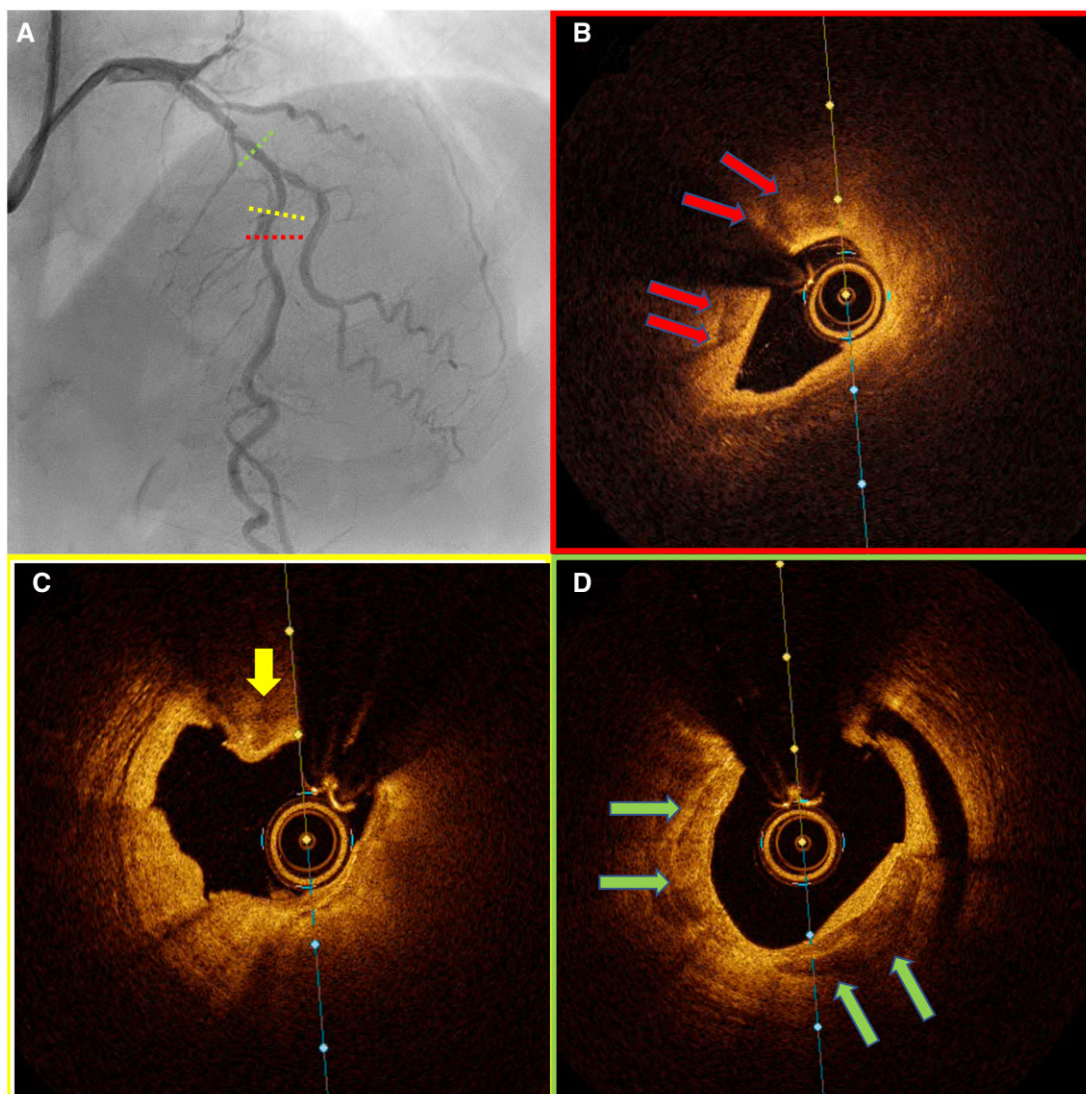


Figure 3 (A) Angiographic markers of the optical coherence tomographic images. (B) Distal tight lesion whilst heavy eccentric calcification (red arrows) seen. (C) Calcium nodule (yellow arrow) extending intra-luminally at the tortuous segment of the vessel. (D) Proximal vessel exhibiting large vessel diameter with multiple areas of superficial calcification (green arrows).

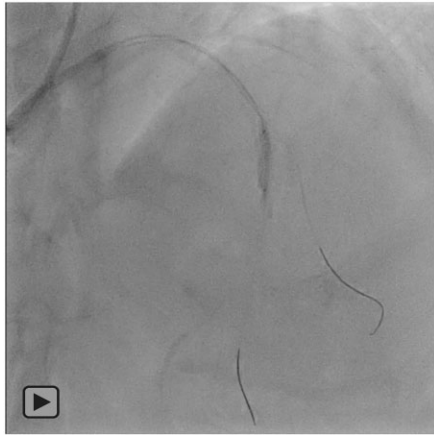
given an angiotensin receptor blocker and a statin. At 3 weeks follow-up, she reported no angina, with improving effort tolerance. Given her lack of symptoms, further intervention for the small left circumflex artery vessel occlusion has been deferred for now. We plan to provide her with DAPT for at least 12 months.

Discussion

Intravascular lithotripsy works by using expanding and collapsing vapour bubbles which create short bursts of acoustic pressure waves. These pressure waves are transmitted to the vessel and the calcium within to produce 'karate-chop' style 50 atm of pressure into the wall. In the presentation entitled 'A deep dive into IVL',⁴ Dr Ali details

the mechanism of action of IVL. The net combined effect of these energies is that the pressure waves are 'hard on hard' and 'soft on soft', thus minimizing trauma whilst cracking calcium selectively.

Adequate balloon size is critical to effective IVL, as good contact of the expanded balloon with the luminal wall is crucial for safe and optimal lithotripsy energy dispersion. In our index case, we pinpoint three possible causes that led to balloon rupture: (i) lumenally protruding nodular calcium (*Figure 3C*), (ii) significant tortuosity of CCL (*Figure 2C*), and (iii) possible gaps in contact between the balloon and luminal wall. The combination of these three factors might have led to compromise in the structural integrity of the IVL balloon resulting in its rupture. One point of note is that whilst the IVL balloon did rupture and cause a dissection, it managed to sufficiently modify the CCL, allowing for optimal stent implantation and expansion.



Video 1 Fluoroscopic capture of the intravascular lithotripsy balloon rupture. Notice the gradual leaking of contrast from the balloon, into the vessel wall and slowly spiralling outside the known area of vessel lumen. This alerted the operator to stop intravascular lithotripsy and deflate the balloon.

There is a paucity of published data with regards to IVL-related complications. The DISRUPT CAD III trial showed 2.6% immediate post-IVL complications, predominantly comprising of type D-F dissections, and in similar fashion were quickly stented over. There was no mention of balloon rupture in this trial.

Published real-world data from the several registries worldwide including the SING-IVL registry,⁵ a European Multicentre registry⁶ and a German multicentre registry by Aksoy *et al.*,⁷ all demonstrated similarly low complication rates. In the latter registry, there was a 9% balloon rupture rate. Overall, whilst IVL balloon rupture does occur, this rarely results in dissections or perforations. Two published case studies highlight how the IVL balloon ruptured during shockwave delivery, resulting in significant vessel dissection.^{8,9} In both cases, IVL balloon rupture occurred when therapy was delivered within tortuous vessels, however, intracoronary imaging was not used for lesion characterization.

Conclusion

Complications during shockwave lithotripsy remain rare. Even amongst cases of balloon rupture, angiographic vessel damage is

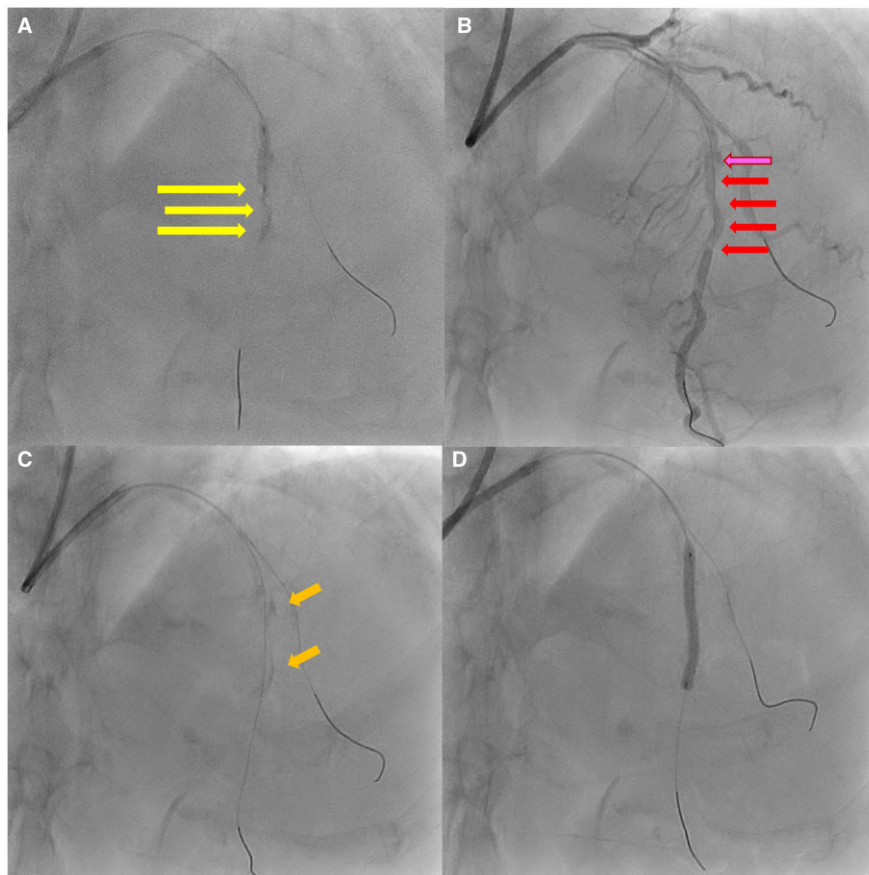
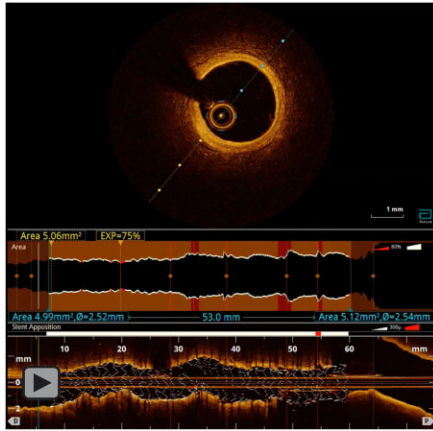


Figure 4 (A) Rupture of intravascular lithotripsy balloon with release of contrast into vascular adventitia (yellow arrows), indicating ongoing spiral dissection. (B) Repeat angiogram showing dissection entry (pink arrow) with contrast-filled dissection progressing distally. (C) Persistent staining of the dissection (orange arrows) even after contrast clearance from the main vessel, indicating a class E dissection. (D) A 28 mm × 2.75 mm drug-eluting stent implanted.



Video 2 Optical coherence tomography run-through of left anterior descending vessel after stenting, demonstrating good distal stent opposition, no distal stent edge dissection, and malapposed proximal stent struts. This optical coherence tomography run was followed by post-dilation with a 3.0 mm × 15 mm non-compliant balloon at 18 atm.



Video 3 Post-procedural analysis of the intravascular lithotripsy balloon, clearly showing the ruptured balloon with contents leaking out.

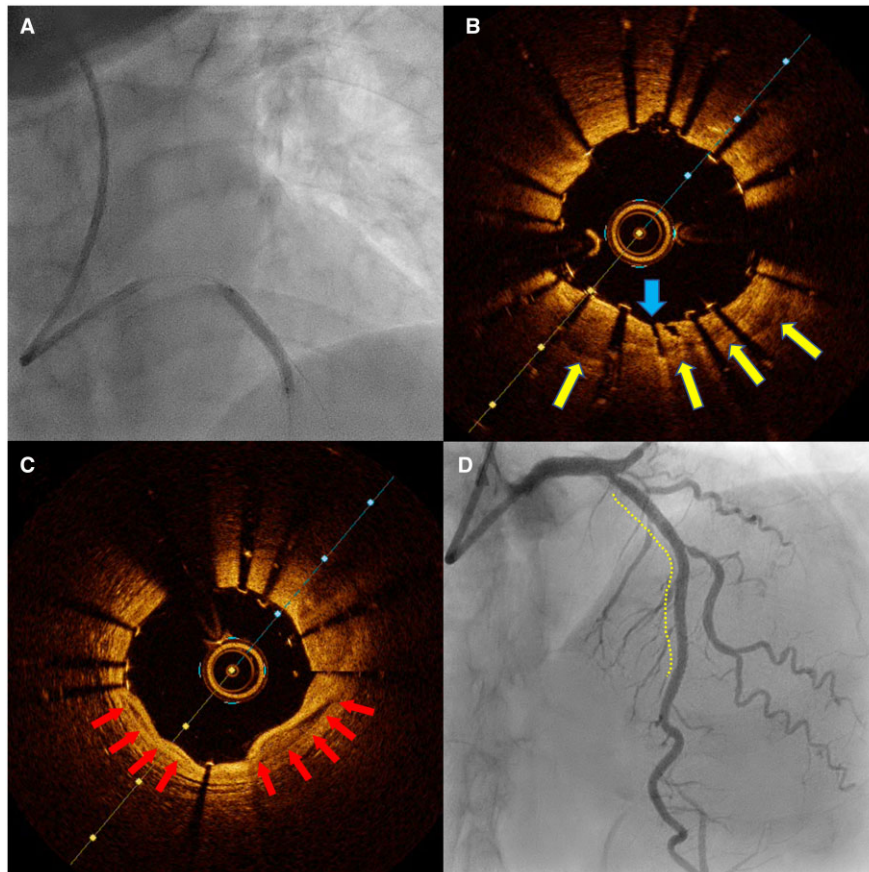


Figure 5 (A) Implantation of a second 3.0 mm × 28 mm drug-eluting stent, overlapping the distal stent. (B) Post-stent optical coherence tomographic showing a well-expanded stent, presence of cracks in the calcium layer (blue arrow), and the superficial calcium well modified behind the stent struts (yellow). (C) Optical coherence tomographic image at the level of dissection, clearly showing the dissection flap (red arrows) which has been sealed behind the stent struts. Notice the extent of the dissection flap, close to half the vessel circumference. (D) Final angiogram showing the stented segment (yellow dashes) with a well-expanded stent and good angiographic results. The pinched diagonal branch was not treated in view of excellent Thrombolysis in Myocardial Infarction 3 flow.

infrequent, with only a handful of documented cases. Specific anatomical features may predispose to such complications, but concrete evidence is lacking. These further cement the role of intravascular imaging during IVL. In conclusion, IVL is not a fail-safe tool and complications can occur, as such intravascular imaging should be used to accurately characterize the lesion prior to calcium modification.

Lead author biography



Dr Tjen Jhung Lee, MD, MRCP, is a cardiology fellow at the renowned Institut Jantung Negara (National Heart Institute). He has a keen interest in the field of interventional cardiology and intravascular imaging. Dr Lee trained at the Sechenov First Moscow State Medical University in Moscow, where he graduated with first class honours in 2011. In 2016, Dr Lee became a

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Supplementary material

[Supplementary material](#) is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case 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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