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CASE REPORT
Coronary heart disease

Rotational atherectomy in a dire situation: a case report

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Background Traditionally rotablation is considered as contraindicated in presence of visible thrombus or dissection. However, clinical situations may force us to undertake rotablation in presence of thrombus or dissection. We report a case of coronary rotablation done successfully in setting of acute thrombotic occlusion over an underlying non-healed dissection.

Case summary A non-dilatable lesion in proximal left anterior descending (LAD) artery after rotablation with a 1.5 mm burr resulting in non-flow limiting Type A dissection with TIMI3 flow was left on conservative management to allow it to heal. But the patient developed ST-elevation myocardial infarction on the 9th post-intervention day due to thrombotic occlusion of the LAD at the site of dissection. At this time, we were compelled to do rotablation as a lifesaving procedure in presence of both thrombus and underlying dissection with a successful outcome.

Discussion Rotablation in presence of dissection can lead to entrapment of the flap in the rotating burr leading to progression of dissection distally or sometimes there can be subintimal tracking of burr leading to perforation. In thrombotic lesions, rotablation can cause further increase in platelet activation and aggregation by the spinning burr or distal embolization of the thrombotic material promoting slow or no flow. In this unusual case with limited options for achieving successful revascularization, some out of the box steps were taken with all recommended precautions and successful outcome achieved.

Keywords Case report • Rotational atherectomy • Dissection • Thrombus

Learning points

- Rotablation is usually considered to be contraindicated in presence of dissection and thrombus.
- Clinically we are occasionally compelled to do rotablation in presence of such contraindications.
- This case report highlights the precautions that need to be taken while undertaking rotablation in presence of both dissection and thrombus.

Introduction

The occurrence of acute ST-elevation myocardial infarction (STEMI) due to thrombotic occlusion over an underlying macro dissection and undilatable calcified lesion warranting urgent rotablation as the only treatment option is a rare but difficult clinical situation. The manufacturer lists both macro dissection and thrombus as contraindications for rotablation.[1] Though, there have been case reports about rotablation in presence of dissection[2,3] or thrombotic occlusion due to STEMI[4–6] we could find only case report where both

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dissection and non-occluding thrombus in background of STEMI was treated successfully by rotablation.[7] We report a case where acute complete thrombotic occlusion of left anterior descending (LAD) artery over an underlying National Heart, Lung, and Blood Institute (NHLBI) type A dissection[8] and non-dilatable calcified lesion was treated successfully by rotablation after taking some precautions. We have discussed the technical issues that need to be considered during rotablation in such a situation.

Timeline

A 60-year-old male patient, a known case of stable angina on medical therapy for 5 months	Presented in emergency with two episodes of rest angina, with electrocardiogram showing significant ST depression in anterior leads (V1–V6) and raised troponin T
He was admitted in coronary care unit and taken up for coronary angiography next morning within 24 h after admission	A non-dilatable calcific lesion in proximal left anterior descending artery (LAD) after rotablation with a 1.5 mm burr, resulting in Type A dissection and TIMI3 flow was left on conservative management to allow it to heal
On the 9th day after angioplasty	Patient developed complete thrombotic occlusion of proximal LAD at the site of dissection. Rotablation, in presence of both thrombus and dissection, was successfully done adopting some precautions as an emergency bailout procedure
72 h following procedure	Patient was discharged in stable condition and is asymptomatic at 6 months of follow-up

Case presentation

A 60-year-old male patient (hypertensive, dyslipidaemic, and ex-smoker) on optimal medical treatment for stable angina for 5 months (aspirin, beta blocker, isosorbide-5-mononitrate, ramipril + hydrochlorothiazide combination, and high dose statin) presented to our outpatient department with the history of crescendo angina for 1 week associated with two episodes of rest angina in prior 12 h. Clinical examination was unremarkable with pulse rate of 64/min and blood pressure of 130/80 mmHg. Electrocardiogram (ECG) showed normal sinus rhythm, normal QRS axis, normal progression of R waves in precordial leads associated with 0.5–1 mm downsloping ST depression in anterior leads (V1–V6) with biphasic T waves. He was admitted in coronary care unit. Echocardiography showed normal left ventricular systolic function with ejection fraction of around 50% but hypokinesia in the mid and apical segments supplied by the LAD. The quantitative test for high sensitive cardiac troponin T (Elecsys, Roche) assessed on admission was elevated and was 84 ng/L (>5 times of normal). After reloading with 150 mg of non-enteric coated aspirin, a continuous infusion of unfractionated heparin was started (bolus of 5000 units with maintenance dose of 12 units/kg/h) with plan for coronary angiography within 24 h. Coronary angiogram done next morning revealed predominantly single vessel disease with a long 90% lesion from proximal to mid-LAD resulting in TIMI2 flow (*Figure 1A and B* and *Video 1A and B*). After loading with 60 mg of prasugrel, during angioplasty of LAD, serial dilatations with 2.5 mm non-compliant (NC) and cutting balloon failed to dilate a point in proximal LAD completely. Hence, rotablation [Rotational Atherectomy System (Boston Scientific, Natick, MA, USA)] with 1.5 mm burr (burr artery ratio of 0.5:1) at 1 50 000 rpm was undertaken (*Figure 1C* and *Video 2*) to modify the lesion. An angiogram after rotablation showed good distal flow in LAD with non-flow limiting Type B dissection[8] in mid-LAD and significant lesion from mid to distal LAD (*Figure 1D*). Following rotablation, the same point in proximal LAD could not be dilated completely with a 3 mm cutting balloon (*Figure 2*) necessitating repeat rotablation with an upsized 1.75 mm burr. Unfortunately, 1.75 mm burr was not available on shelf due to use on the same day in the adjacent Cath lab and high-pressure OPN balloon/lithotripsy balloon or laser atherectomy is not available in our hospital. At this point, an option for bailout emergency bypass surgery was given to the patient but he refused with request to try other options. Hence after consultation with the cardiothoracic surgeon, it was decided that if we failed in addressing the lesions in mid and distal LAD along with the Type B dissection with stents, then the patient would be sent for bailout bypass surgery to which the patient also agreed. Following this, stenting of the lesions in distal and mid-LAD were attempted and successfully done with 2.5 mm × 20 mm and 2.75 mm × 16 mm drug-eluting stents which expanded completely at 12 and 14 atm pressure, respectively. A check angiogram done by withdrawing the wire from LAD showed TIMI3 flow with non-flow limiting type A dissection and significant residual stenosis in proximal LAD (*Video 3*). As the risk of occlusion of Type A dissection is negligible and patient remained asymptomatic, he was shifted to our post-intervention coronary care unit where subsequently heparin infusion was started to maintain APTT to two times above normal. The 1.75 mm rota burr became available next day but by this time 24 h had passed and the patient was totally asymptomatic. Hence, a decision was taken to defer rotablation to allow the dissection to heal over next 4 weeks before reintervention (as per recommendation of the company and European expert consensus on rotational atherectomy).[1,9] He was switched to low molecular weight heparin (injection enoxaparin 1 mg/kg body weight twice daily) for next 7 days with plan to discharge thereafter. As patient had dissection in proximal

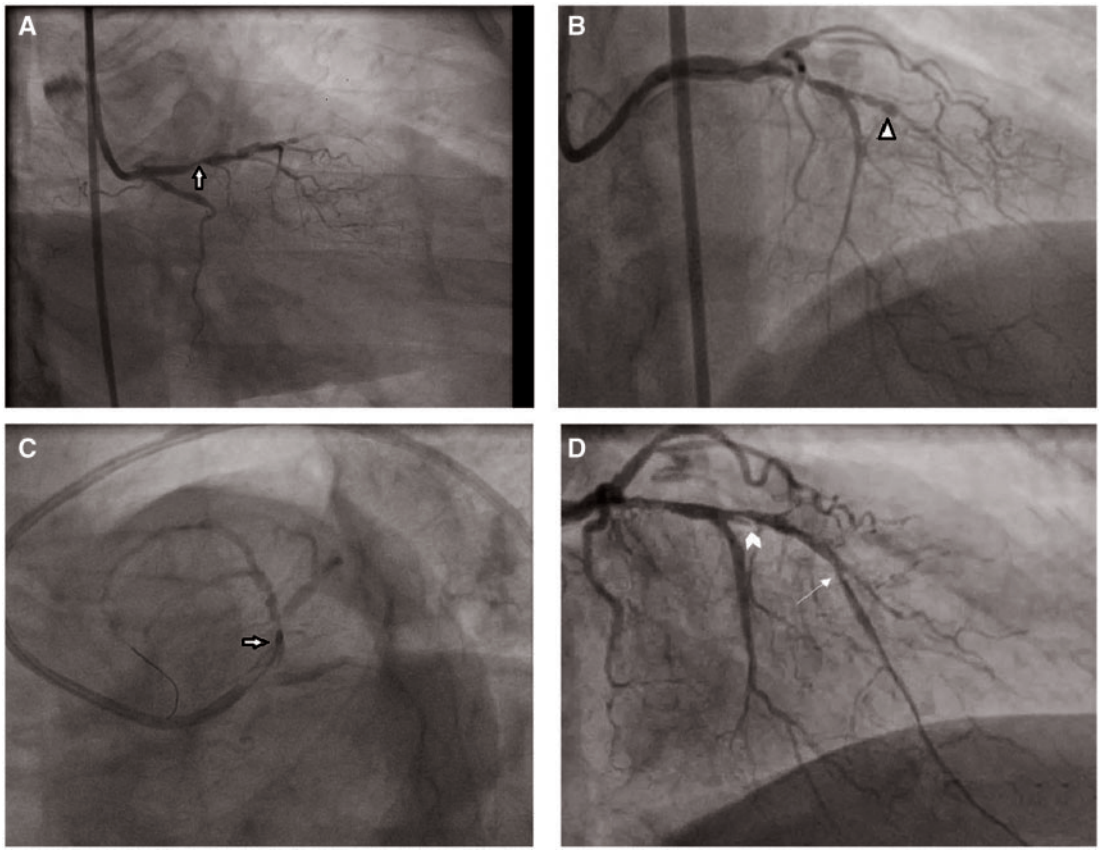
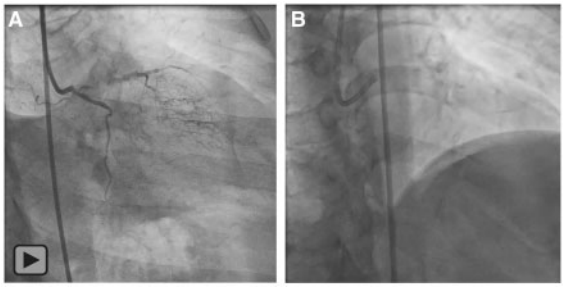
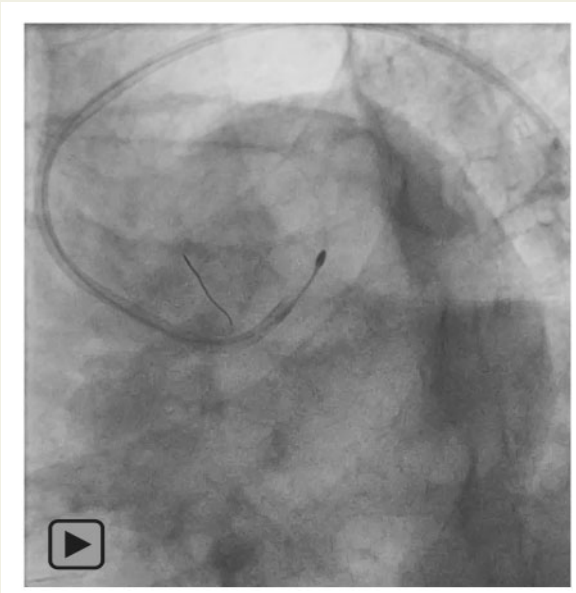


Figure 1 (*A*) Initial angiogram showing 90% eccentric lesion in proximal left anterior descending artery (white arrow). (*B*) Angiogram in PA Cranial view showing significant stenosis in mid and distal left anterior descending artery (arrowhead). (*C*) Rotablation of left anterior descending artery being done with 1.5 mm burr (arrow). (*D*) Angiogram following rotablation with 1.5 mm burr showing Type B dissection (arrowhead) in mid-left anterior descending artery and significant lesion (arrow) in distal left anterior descending artery.

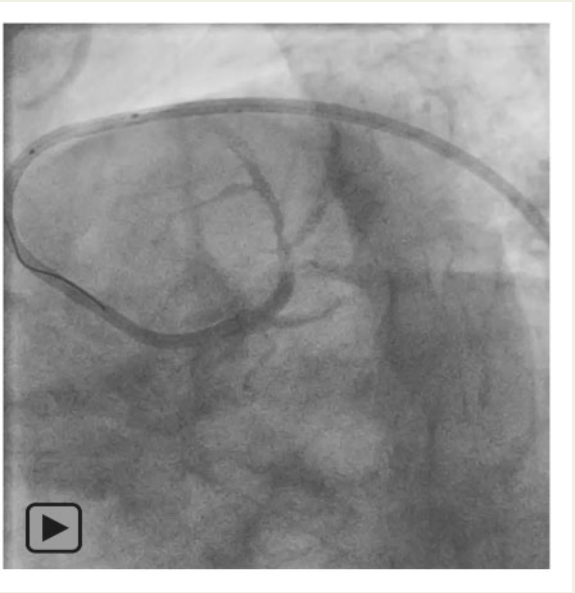


Video 1 (*A*) and (*B*) Initial angiogram showing long 90% lesion from proximal to mid-left anterior descending artery resulting in TIMI2 flow.

LAD just adjacent to a significant residual calcified lesion in background of non-STEMI (a highly prothrombotic state), we decided to give enoxaparin under supervision in hospital to ensure compliance and also watch for any bleeding complication as patient was also on dual antiplatelet therapy (aspirin and prasugrel). However, on the 9th day (around 36 h after stopping anticoagulation), the patient developed acute chest pain with ST-elevation in anterior leads (*Figure 3A*) and immediate angiogram showed 100% occlusion of proximal LAD at the site of dissection with significant residual lesion (*Figure 3B* and Supplementary material online, *Video S1*). Injection abciximab was started intravenously (bolus of 0.25 mg/kg body weight with maintenance dose of 0.125 µg/kg/min for 12 h). The cardiothoracic surgeon was informed for bailout bypass surgery if required. Failing with a choice floppy wire, the LAD was crossed carefully with a Whisper wire. After failing to pass a thrombosuction catheter, a 1.5 mm balloon passed freely without any resistance to distal LAD confirming intraluminal position of wire. Dilatation with a 2.75 mm compliant balloon resulted in TIMI3 flow in LAD with no significant thrombus burden at the site of occlusion. Following this, rotablation was done with a 1.75 mm burr at the maximum recommended speed of 150 000 rpm[10] by gentle pecking motion with each run not exceeding 15 s, avoiding deceleration of more than 5000 rpm and monitoring for slow flow/perforation after each run. Every precaution was taken not to ablate the recently implanted stent just distal to the undilated calcified segment. Henceforth, three runs of focal rotablation were done (Supplementary material online, *Video S2*) and procedure



Video 2 Rotablation of lesions in proximal and mid-left anterior descending artery with 1.5 mm burr.



Video 3 Non-flow limiting type A dissection with associated significant residual lesion in proximal left anterior descending artery.

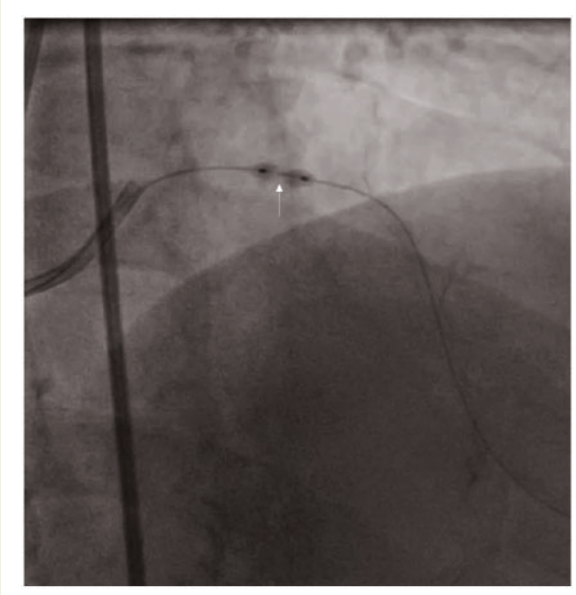


Figure 2 Dog boning effect (arrow) of 3 mm × 6 mm cutting balloon after rotablation with 1.5 mm burr.

completed without any final polishing run. Following rotablation, the lesion in proximal LAD showed complete expansion with a 3 mm NC balloon. A 3 mm × 28 mm drug-eluting stent was deployed in proximal LAD at 14 atm pressure overlapping 5 mm with the distally deployed stent resulting in TIMI3 flow (*Figure 3C* and Supplementary material online, *Video S3*) and significant resolution of ST segment in anterior leads on ECG (*Figure 3D*). The patient was discharged after 72 h with predischarge ejection fraction of around 45% [ecosprin 100 mg, prasugrel 10 mg, ramipril + hydrochlorothiazide (5 + 12.5 mg), sustained-release metoprolol 50 mg, atorvastatin 80 mg, and pantoprazole 40 mg] and he is asymptomatic at 6 months of follow-up with near-normal ejection fraction of around 50%.

Discussion

Visible thrombus and dissections are considered contraindications of rotablation[1] but there are some unforeseen clinical situations where we may be forced to use it.

Our case developed complete thrombotic occlusion of LAD following angioplasty due to occlusion of a Type A non-flow limiting dissection in proximal LAD on the 9th post-intervention day that could not be patched with a stent during the index procedure. Our patient was completely asymptomatic for 24 h and as spontaneous occlusion of even Type B dissections (our case had Type A dissection) beyond this period is unusual,[11–13] our initial conservative approach to allow the dissection to heal over next 4 weeks before repeat rotablation was justified.[1,9] However, retrospectively we feel that there was an error of judgement on our part. A significant residual obstructive lesion just distal to the Type A dissection was also a nidus of thrombus formation along with the partially healed proximal dissection. In our case, both these factors lead to acute thrombotic occlusion of proximal LAD within 36 h after stopping anticoagulation. Non-availability of the 1.75 mm burr had forced us to leave a significant calcified lesion with accompanying Type A dissection at its proximal end without stenting during the index procedure leading to a life-threatening complication. It was a mistake on our part not to confirm availability of the 1.75 mm burr before starting rotablation. Rotablation should never be undertaken before confirming the availability of all relevant burr sizes. Following failure to modify the calcified lesion in proximal LAD with the 1.5 mm burr, imaging with intravascular ultrasound

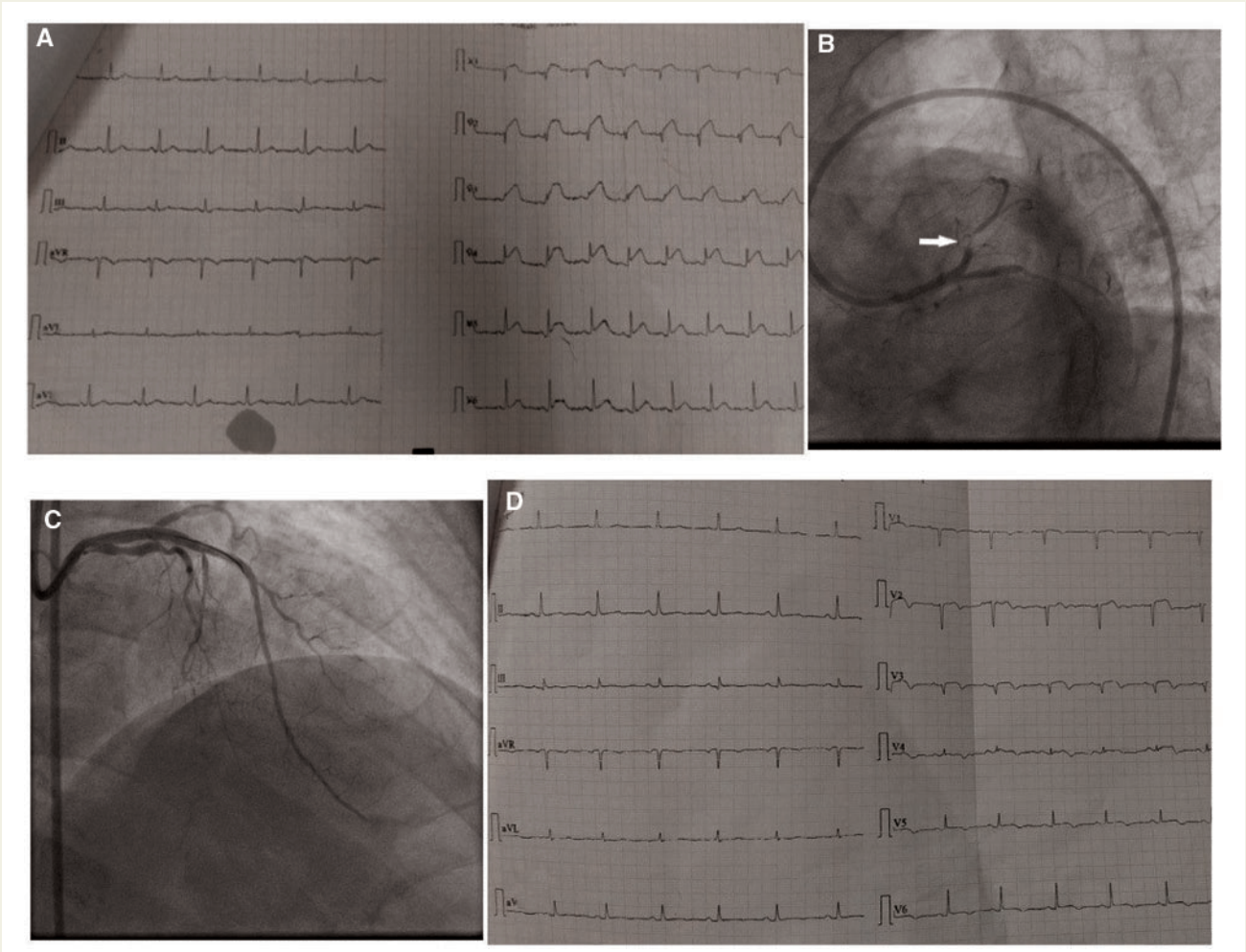


Figure 3 (*A*) Electrocardiogram showing acute ST-elevation myocardial infarction with ST-elevation from V1 to V6. (*B*) Angiogram showing complete thrombotic occlusion of proximal left anterior descending artery (arrow). (*C*) Angiogram showing TIMI3 flow in left anterior descending artery. (*D*) Post-angioplasty of left anterior descending artery, electrocardiogram showing ST segment resolution in V1–V6.

(IVUS) should have been done to find out the pattern of calcification and plan the future calcium modifying device. However, apart from rotablator, no other calcium modification device (lithotripsy balloon, high-pressure OPN balloon, or laser atherectomy) was available with us. Hence, we decided to use the 1.75 mm burr and if it failed, we would have done IVUS to document the cause of failure and send the patient for bailout surgery. We adopted this strategy to avoid imposing the financial burden of the IVUS catheter to the non-insured patient and our added apprehension that the IVUS catheter might not negotiate the non-dilated calcific segment (*Figure 2*). In our case, the use of the 1.75 mm burr was, however, successful in modifying the calcific lesion confirmed by complete expansion of a 3 mm NC balloon (balloon artery ratio 1:1) post-rotablation.

Rotablation in presence of dissection can lead to entrapment of the flap in the rotating burr leading to progression of dissection distally or sometimes there can be subintimal tracking of burr leading to perforation.[2] In our case, distal propagation was not possible as there was already a stent deployed distally. Hence, we chose directly the 1.75 mm burr required in our case. However, in situations where there is macro dissection with risk of distal propagation recommendations are (i) to start with the 1.25 mm burr and follow a step up burr approach if required to minimize atherectomy in the dissected segment,[2] (ii) brief runs not exceeding 15 s at normal recommended speed of 1 40 000–1 50 000 rpm with check for dissection propagation/abrupt closure/perforation after each run,[7,13] (iii) avoid rotating the burr at slower than recommended speed or deceleration of more than 5000 rpm as it increases the risk of entanglement of the dissection flap in the spinning burr,[2] (iv) avoidance of polishing run to minimize contact time of burr with dissection flap[7] and (v) backup availability of a covered stent in case of perforation.[7]

Rotablation is also avoided in the presence of thrombus due to the concern for further increase in platelet activation and aggregation by the spinning burr[7] or distal embolization of the thrombotic material promoting slow or no flow. The recommendations for performing rotablation safely in presence of acute thrombotic occlusions are (i) use of GPIIb/IIIa receptor antagonists to inhibit speed induced platelet activation,[14] (ii) avoidance of rotating the burr at slower than recommended speed as it is less efficient and leads to production of larger

ablated particles that can precipitate slow or no flow[15] and (iii) avoidance of final polishing run.

Rotablation can lead to stent ablation which in turn can cause subacute or late stent thrombosis as some stent struts may become irregular or damaged.[16] Hence, we tried to do a focal ablation avoiding the stented area. But for benefit of doubt, we placed our proximal stent ~5 mm within the previously deployed stent (ideally optical coherence tomography imaging, presently unavailable in our institute should be done).

Conclusion

Severe coronary dissection or acute thrombotic occlusion should remain a contraindication for rotablation. Nevertheless, in this unusual case with limited options for achieving successful revascularization acutely, some out of the box steps were taken with all recommended precautions and successful outcome achieved.

Lead author biography

Dr Saibal Mukhopadhyay obtained his MBBS degree from Calcutta Medical College in 1991, MD degree in General Medicine from Pandit BD Sharma PGIMS, Rohtak, Haryana in 1997, and his DM degree in cardiology in 2002 from Gobind Ballabh Pant Hospital, New Delhi. He is currently working as Professor and Head, Department of Cardiology, Gobind Ballabh Pant Hospital, New Delhi.

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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