

Aortic cusp perforation during rotational atherectomy: a case report

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Background

Rotational atherectomy has become increasingly utilised over the past decade. Although a relatively safe procedure in appropriately trained physicians' hands, there are a number of recognised complications.

Case summary

We describe the case of a 64-year-old female who presented with chest pain and was diagnosed with non-ST-segment elevation acute coronary syndrome. A transthoracic echocardiogram (TTE) showed normal biventricular function and no valve disease. Invasive coronary angiogram was performed which revealed a severely calcified ostial right coronary artery (RCA) disease which was felt to be the culprit of the presentation. Balloon dilatation was unsuccessful, therefore, rotational atherectomy with an Amplatz left 0.75 guide and a 1.5 mm rota-burr was utilised and improved calcium burden. This was complicated by ostial dissection, treated with stenting. A TTE following the procedure revealed moderate aortic regurgitation (AR). The patient was discharged as she remained asymptomatic. An outpatient transoesophageal echocardiogram performed eight months later showed evidence of severe eccentric AR. Cardiac magnetic resonance imaging confirmed severe AR with left ventricular dilatation. Repeat angiogram 10 months after index procedure revealed in-stent restenosis, and the patient was accepted by heart multidisciplinary team for aortic valve replacement and grafting of RCA.

Discussion

As the field of rotational atherectomy continues to expand, we propose that novel complications such as reported in this case may become recognised. Finally, we stress the importance of multi-modality imaging in the investigation and timely planning of interventions in the management of these patients.

Keywords

Case report • Rotational atherectomy • Primary coronary intervention • Aortic regurgitation • Transoesophageal echocardiography • Cardiovascular magnetic resonance imaging

ESC Curriculum

2.1 Imaging modalities • 2.2 Echocardiography • 2.3 Cardiac magnetic resonance • 3.4 Coronary angiography • 4.1 Aortic regurgitation

Learning points

- Have a high index of suspicion of aortic valve laceration if complicated procedure or clinical examination reveals a new aortic regurgitant murmur.
- We stress the importance of multi-modality imaging in the investigation and timely planning of interventions in the management of these patients.

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Introduction

Over the last decade, rotational atherectomy (RA) has become more common as a consequence of more complex and calcified coronary stenosis being treated with percutaneous coronary intervention (PCI).¹ The rate of RA as a function of total PCI numbers reached 3.1% in the UK in the year 2015.¹ For experienced users, contemporary RA offers an effective means of percutaneous intervention of highly calcified coronary lesions. There are several recognised complications including those common to all PCI but other more specific to RA (burr entrapment, RotaWire fracture, and vessel perforation).^{1–3} We present a rare case of iatrogenic aortic cusp laceration causing severe aortic regurgitation (AR) requiring valve replacement surgery, secondary to complex percutaneous coronary intervention where RA was utilised. To our knowledge, there are no published case reports describing this complication.

Timeline

Day 0: Admission under the medical team with chest pain.
 Day 1: Diagnosis of non-ST-segment elevation acute coronary syndrome was made following review of patient, electrocardiogram, and serial troponins. Patient was started on dual antiplatelet (DAPT), high dose statin (atorvastatin 80 mg), and low molecular weight heparin.
 Day 2: Admitted to coronary care unit and transthoracic echocardiogram (TTE) performed.
 Day 2: Primary coronary intervention + rotational atherectomy performed following invasive coronary angiogram.
 Day 3: Repeat TTE demonstrates new aortic regurgitation.
 Day 5: Patient discharged from hospital for outpatient follow-up on dual antiplatelet therapy, atorvastatin 80 mg, beta blocker (bisoprolol 2.5 mg), and angiotensin-converting enzyme inhibitors (ramipril 2.5 mg).
 Day 11: Patient attended outpatient cardiac rehabilitation.
 6 weeks: Review in outpatient cardiology clinic—patient reports mild dyspnoea.
 8 months: Transoesophageal echocardiography demonstrating eccentric severe aortic regurgitation with cusp perforation.
 9 months: Cardiac magnetic resonance imaging confirming severe aortic regurgitation.
 10 months: Repeat coronary angiogram demonstrating focal areas of in-stent restenosis. Discussed in heart multidisciplinary team meeting and accepted for aortic valve replacement and coronary bypass grafting.
 11 months: Surgery was performed and was uncomplicated.
 13 months: Patient seen in cardiothoracic clinic and remained asymptomatic. Current medications included DAPT, atorvastatin 80 mg, ramipril 2.5 mg, and omeprazole 40 mg.

Case presentation

A 64-year-old female presented with central chest pain on a background of hypertension, and previous lung malignancy (adenocarcinoma staged as T2a, N1, and M0) for which she had a video-assisted thoracic surgery (VATS) lower lobectomy in 2014 and adjuvant chemotherapy followed by radiotherapy. On presentation, there were no positive cardiovascular examination findings, and no significant electrocardiogram (ECG) changes (Figure 1). However, serial high sensitivity troponin I (hs-TnI)

were elevated at 10 ng/L and then 84 ng/L (normal range <16 ng/L), and a diagnosis of non-ST-segment elevation acute coronary syndrome was made. As per our hospital protocol, the patient was started on dual antiplatelet therapy (aspirin—loading dose of 300 mg followed by 75 mg once a day and ticagrelor—loading dose of 180 mg followed by 90 mg twice daily), high dose statin (atorvastatin 80 mg once daily), and low molecular weight heparin (fondaparinux 2.5 mg subcutaneously once daily). The patient was admitted to the cardiology coronary care unit and a transthoracic echocardiogram (TTE) was performed while awaiting a coronary angiogram. Transthoracic echocardiogram revealed normal left ventricular (LV) systolic function and no valvular pathology. Specifically, there was no AR (see [Supplementary material online, Video S1](#)).

The following day, the patient was taken to the catheter laboratory and underwent invasive coronary angiography via the right radial approach using 6F JR4 and JL3.5 catheters. This revealed an unobstructed left coronary artery system with a severely calcified ostial right coronary artery (RCA) disease which was felt to be the culprit of the presentation (see [Supplementary material online, Video S2](#)). Initial balloon dilatation required a 2.0 × 15 mm NC TREK balloon (Abbott, USA) but this showed incomplete expansion. Therefore, rotational atherectomy with an Amplatz left (AL) 0.75 guide (Boston Scientific, USA) and a 1.5 mm rota-burr was utilised. Initial wiring through the lesion was challenging but successful using a Fielder FC wire (Asahi Intecc Medical, Japan). Subsequent rotablation required four ‘passes’ of the burr to ablate the calcium. This was challenging and despite the use of a relatively supportive guide, there was ‘backing out’ guide. Repeat angiographic images after rotablation raised the suspicion of perforation in ostium of the RCA due to dye extravasation. Balloon tamponade of the area was successful with subsequent PCI being performed successfully from the ostium to mid vessel using two overlapping 3.5 × 29 mm BIOMATRIX drug eluting stent (Biosensors International, Singapore) with post-dilation using a 3.5 × 20 mm NC TREK balloon—Abbott, USA (see [Supplementary material online, Video S3](#)). The final angiographic result appeared satisfactory. A TTE following the procedure revealed moderate AR with preserved LV size and function (see [Supplementary material online, Video S4](#)), and a computed tomography (CT) angiogram performed ruled out aortic root dissection.

Given the patient remained clinically well, she was discharged on dual antiplatelet therapy, atorvastatin 80 mg, bisoprolol 2.5 mg, and ramipril 2.5 mg with plan to follow up in outpatient clinic. Upon 6-week review in clinic, the patient complained of breathlessness and a subsequent outpatient transoesophageal echocardiogram (TOE) showed evidence of severe eccentric AR some mobile leaflet tissue attached to the ventricular side of one of the cusps, likely relating to cusp perforation (see [Supplementary material online, Video S5](#)). Ejection fraction (EF) was quantified as 62 ± 5% by 3D volume method. Transoesophageal echocardiogram was delayed due to significant pressures because of the Sars-CoV2 pandemic. Cardiac magnetic resonance imaging confirmed severe AR with left ventricular dilatation (see [Supplementary material online, Video S6](#)). Repeat angiogram 6 months from the time of the index procedure showed in-stent restenosis at two focal points in the proximal and mid RCA (see [Supplementary material online, Video S7](#)). The patient was presented and accepted by heart multidisciplinary team for aortic valve replacement (AVR) and grafting of RCA. This was uncomplicated, and following discharge was reviewed in outpatient clinic where she had an improving exercise tolerance, and follow up TTE revealed a stable AVR with no AR and normal EF (65%). Medications at this stage included dual antiplatelet therapy, atorvastatin 80 mg, ramipril 2.5 mg, and omeprazole 40 mg.

Discussion

Percutaneous coronary intervention is the most used revascularisation modality for obstructive coronary artery disease. Severe coronary

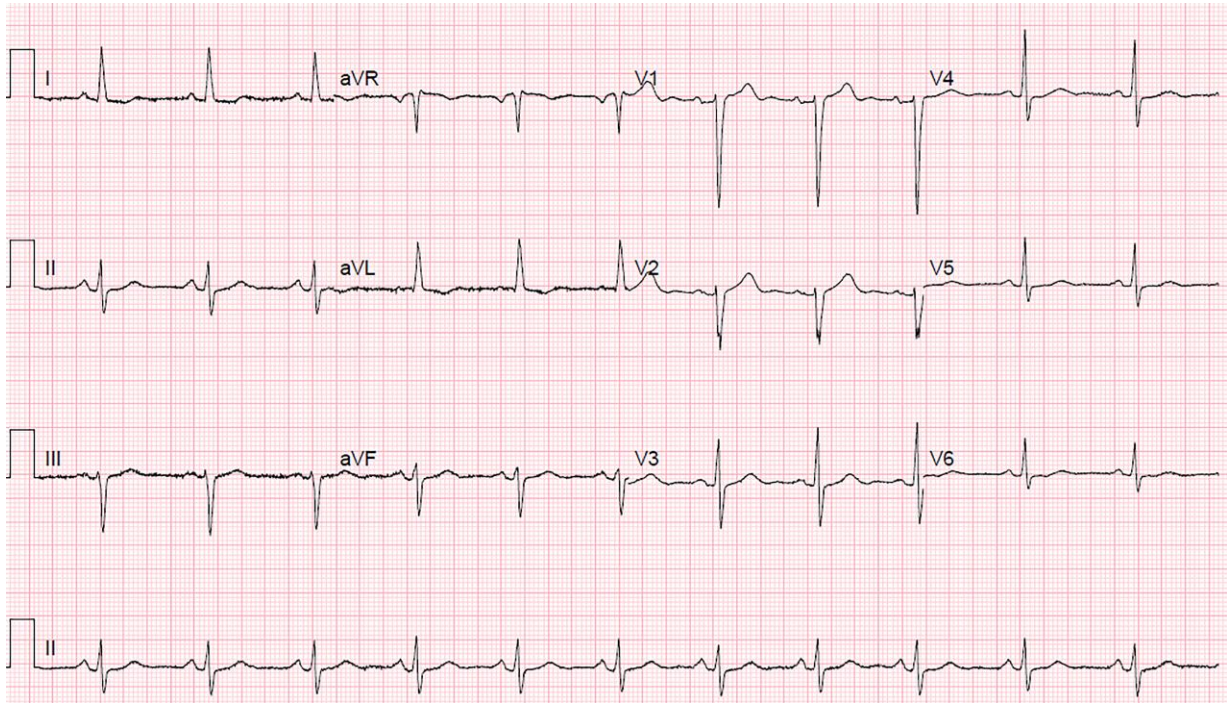


Figure 1 ECG on presentation which revealed no significant ischaemic features.

artery calcification is a notable risk factor for procedural failure, complications and long-term sequelae in contemporary PCI.⁴

Balloon angioplasty—even with high-pressure inflation—can fail to adequately dilate heavily calcified lesions.⁵ This is because angiography balloons are prone to asymmetric expansion (especially semi-compliant balloons) increasing the risk of coronary dissection and perforation.^{5,6} Severely calcified plaques also hamper the delivery of angiographic balloons and stents. Rotational atherectomy was introduced three decades ago and is based on the principle of differential cutting that ablates hard inelastic atherosclerotic material while rendering the inner lumen surface smooth.⁷ Over time, RA has shifted from initially being used as a debulking strategy to plaque modification to facilitate balloon angioplasty and stent expansion.⁸ This shift in approach has been led by the results of the STRATAS and CARAT trials which showed that aggressive debulking strategy was associated with higher rates of angiographic complications.⁸

There are a number of well described and well recognised direct complications of RA.⁸ ‘No Reflow’ results from microvasculature embolization of debris and thrombi and is associated with periprocedural MI.⁹ Coronary perforation secondary to RA is rare with reported incidence of up to 2% in literature and is often associated with poor technique with the use of large burr.⁸ However, it should be noted that the rates of coronary perforation are higher when PCI is used in conjunction with RA as compared to PCI alone (which has a reported incidence of about 0.4%).⁸ Burr entrapment within the calcified lesion is a rare but serious complication of RA which usually requires surgical retrieval—this has a reported incidence of 0.4%.¹⁰ The incidence of complications of RA varies among studies—likely because the sample sizes of studies have been small, however, a recent national prospective multicentre study by Sakakura *et al.*¹¹ reported the incidence of important procedural-related complication at 1.3%. Acute AR secondary to PCI is extremely rare with a reported incidence of approximately 0.0001%.¹² Aortic regurgitation secondary to RA is an even rarer occurrence with only one previous case described in literature (abstract). In this case described by Vadgonkar *et al.*,¹³ rotational atherectomy

guided PCI of heavily calcified left anterior descending and left circumflex coronary artery resulted in acute AR secondary to tears in the non-coronary cusp which extended all the way to the free margin of the right coronary cusp.

The likely mechanism by which aortic cusp perforation occurred during this procedure is through the traction and probable disengagement of the guide during multiple ‘burr runs’ through the heavily calcified ostial right coronary artery. The resulting traction allowed the rota-burr to ‘freely hang’ in the ascending aorta close to the cusps. At some point during the procedure, the burr would have come in contact with the cusps momentarily allowing for the subsequent cuspal perforation and ensuing aortic regurgitation.

Specific to this case, the use of a more supportive guide in the form of an AL1 guide catheter may have improved support during the rotablation and thus minimised the chances of disengagement of the guide from the RCA and subsequent retropulsion of the rota-burr. However, the use of an AL 0.75 guide would be deemed more than suitable for most procedures of this complexity. Furthermore, a femoral approach to access would provide more support to the guide catheter and thus also minimise the aforementioned challenges in the case. However, femoral access does not come without its own risks and in the majority of cases of rotablation can and should be carried out through the safer radial access route.

A recent study showed that the use of RA in elderly patients with severe AS and concomitant calcific CAD is safe and well tolerated.¹⁴ This therefore suggests that the field and scope of RA will likely continue to grow. It therefore is important to recognise novel complications as reported by our case as the field grows.

Conclusion

Our case demonstrated the importance of having a high index of suspicion of AV laceration in patients who have undergone complex coronary intervention especially if it was a complicated procedure, or if

clinical examination reveals a new aortic regurgitant murmur. This should prompt urgent investigation. Finally, we stress the importance of multi-modality imaging in the investigation and timely planning of interventions in the management of these patients.

Lead author biography



Nihit Shah (BMBS, BMedSci, MRCP) is a graduate of the University of Nottingham and has currently completed Stage 1 of Internal Medical training in the United Kingdom and has been accepted into cardiology speciality training in the West Midlands Deanery, UK.

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.

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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