

Unconventional treatment of a giant coronary aneurysm presenting as ST-elevation myocardial infarction: a case report

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Background

The optimal treatment of aneurysmal or ectatic culprit vessels in the setting of acute myocardial infarction is still matter of debate, as revascularization with either percutaneous intervention or surgery is associated with low procedural success and poor outcomes.

Case summary

We report the case of a 55-year-old male patient, admitted for inferior ST-elevation myocardial infarction, who underwent successful percutaneous implantation of a micro-mesh self-expanding nitinol carotid stent in a right coronary aneurysm with intravascular ultrasonography measured diameter of 9 mm and massive thrombus apposition.

Discussion

The technical characteristics of the micro-mesh self-expanding nitinol carotid stent allow for adequate plaque coverage and good apposition even in large vessels, making this device particularly suitable for the treatment of coronary lesions with high thrombus burden, when severe coronary ectasia or aneurysms are present.

Keywords

Acute myocardial infarction/STEMI • Coronary aneurysm • Percutaneous coronary intervention (PCI) • Case report • Interventional devices/innovation

Learning points

- The optimal treatment of aneurysmal or ectatic culprit vessels in the setting of acute myocardial infarction is still matter of debate.
- We report the first case of successful percutaneous implantation of a micro-mesh self-expanding nitinol carotid stent in a thrombosed coronary aneurysm.
- The specific design of this carotid stent allows the treatment of most coronary artery aneurysms or ectasia and prevents embolic events by trapping and excluding thrombus and plaque debris.

Introduction

Coronary artery aneurysms (CAAs) or ectasia (CAE) are defined as a focal or diffuse dilation of the coronary artery of at least 1.5 times the adjacent normal segments, respectively.¹ The incidence of the disease ranges between 0.3% and 5%, and clinical presentation varies from asymptomatic condition to acute coronary syndromes.² The pathogenesis of CAA is not yet fully understood. However, a number of conditions have been suggested as potential risk factors. Among those: genetic susceptibility, specific vasculitic and connective tissue diseases (e.g. Kawasaki disease, Marfan disease), local wall injury following intracoronary manipulation and post-infectious conditions. A common underlying aetiology with coronary artery disease (CAD)

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has also been advocated.^{3,4} Treatment options include medical management, percutaneous coronary intervention (PCI), and cardiac surgery.

Herein, we report the first case of successful percutaneous implantation of a micro-mesh self-expanding nitinol carotid stent in a thrombosed right coronary aneurysm.

Timeline

Day 0	Presentation for acute inferior ST-elevation myocardial infarction. Urgent coronary angiography revealed severe coronary artery ectasia and thrombotic occlusion of the proximal right coronary artery (RCA). Successful reperfusion was achieved by means of manual thrombus aspiration and balloon angioplasty.
Day 1	Recurrence of chest pain and dynamic ST-wave changes. Coronary angioplasty of the RCA with implantation of two overlapping self-expandable stents was performed. A nitinol CGuard carotid stent was chosen to treat the severely ectatic proximal segment of the RCA.
Day 2	Echocardiography revealed inferior hypokinesia and preserved ejection fraction.
Day 6	Discharge from hospital after an uneventful perioperative period in the cardiac intensive care unit with continuous electrocardiogram monitoring.

Case presentation

A 55-year-old male patient with hypertension presented to our hospital with a complaint of chest pain that began 90 min earlier. The electrocardiogram (ECG) at admission showed sinus rhythm and ST-elevation in the inferior leads. All laboratory values were within normal ranges. Physical examination revealed regular cardiac rhythm without murmurs, and bibasilar pulmonary rales. The patient was administered acetylsalicylic acid 250 mg i.v. and ticagrelor 180 mg before urgent cardiac catheterization. Coronary angiography revealed a critical stenotic lesion of the left anterior descending artery (LAD) and a severe CAA and thrombotic occlusion of the proximal right coronary artery (RCA) ([Figure 1A and B](#); [Videos 1 and 2](#)). Reperfusion was achieved by means of manual thrombus aspiration and balloon dilation with 2.5 × 20 mm non-compliant balloon [NC Emerge (Boston Scientific, Marlborough, MA, USA)] ([Figure 1C–E](#)). Examination with intravascular ultrasonography showed severe CAA measuring 9 mm in diameter and a residual large thrombus burden in the proximal segment of the RCA, as well as a critical stenosis with massive thrombus apposition in the mid-segment of the vessel. Distal reference vessel diameter (RVD) was approximately 6.5 mm ([Figure 2](#); [Supplementary material online, Figure S1](#); and [Video 3](#)). Final coronary angiography showed Thrombolysis In Myocardial Infarction (TIMI) 2–3 blood flow ([Figure 1F](#) and [Supplementary material online, Video S1](#)). At this stage, the patient was asymptomatic and

haemodynamically stable, and the ECG showed ST-segment resolution. No further PCI was performed and adjunctive pharmacotherapy with low molecular weight heparin and tirofiban infusion was administered, aiming to reduce thrombus burden. An angiographic control was scheduled—not as per standard practice—few days later to evaluate the efficacy of anticoagulant and glycoprotein IIb/IIIa inhibitors therapy.

Twenty-four hours after the procedure, the patient suddenly experienced recurrence of chest pain and dynamic ST-wave changes. Repeat urgent coronary angiography revealed nearly complete occlusion of the mid-RCA ([Figure 3A](#) and [Supplementary material online, Video S2](#)). Percutaneous coronary intervention was performed from right femoral artery using an 8-Fr AL 1.0 guiding catheter (Cordis, Miami Lakes, FL, USA). After the lesion was easily crossed with a workhorse coronary guidewire, a nitinol self-apposing, sirolimus-eluting Xposition S 3.5–4.5 × 27 mm stent (Stentys, Paris, France, UE) was deployed distally first at the mid-RCA segment ([Figure 3B and C](#) and [Supplementary material online, Video S3](#)). Then, a self-expanding nitinol CGuard 8.0 × 40 mm carotid stent (InspireMD, Tel Aviv, Israel) was positioned with its distal marker inside of the proximal stent edge of the already deployed distal Xposition S ([Figure 3D](#) and [Supplementary material online, Videos S4 and S5](#)). The CGuard stent was implanted after the release of the Stentys, in order to avoid late CGuard overexpansion and subsequent Stentys malapposition. Stents were both post-dilated with a Boston NC Emerge 6.0 × 12 mm balloon inflated at high pressures. Final angiography showed no residual stenosis and TIMI 3 flow ([Figure 3E](#) and [Supplementary material online, Videos S6 and S7](#)). Intravascular ultrasonography examination confirmed good stent expansion as well as complete apposition to the vessel wall ([Figure 4](#)). Complete revascularization was achieved in the same procedural session, by drug-eluting stent implantation [Resolute Onyx 3.0 × 26 mm (Medtronic Inc., Minneapolis, MN, USA)] in the mid-LAD ([Figure 3F](#) and [Supplementary material online, Video S8](#)). The post-operative course in the cardiac intensive care unit was uneventful, and ECG continuous monitoring showed no significant arrhythmias during the hospital stay. The patient was discharged in good physical condition, with an oral treatment including aspirin 100 mg/day and ticagrelor 90 mg b.i.d., and with indication to consider transition to long-term dual antiplatelet therapy after clinical re-evaluation at 12 months. At 6-month follow-up, the patient was asymptomatic. Cycle ergometer (25 Watts more every 3 minutes) stress test, terminated due to fatigue at a heart rate of 142 b.p.m. (86% of the age-adjusted expected maximum heart rate), showed no ECG changes suggestive of ischaemia and no significant exercise-induced arrhythmias. Transthoracic echocardiogram showed preserved left ventricular ejection fraction and inferior hypokinesia.

Discussion

Coronary artery aneurysms and ectasia are often clinically silent conditions, and they are frequently detected by chance among patients undergoing coronary angiography or computed tomography. However, patients can develop symptoms as a result of concomitant obstructive atherosclerosis or because of a sluggish or turbulent blood flow in the ectatic segments, which lead to reduced tissue

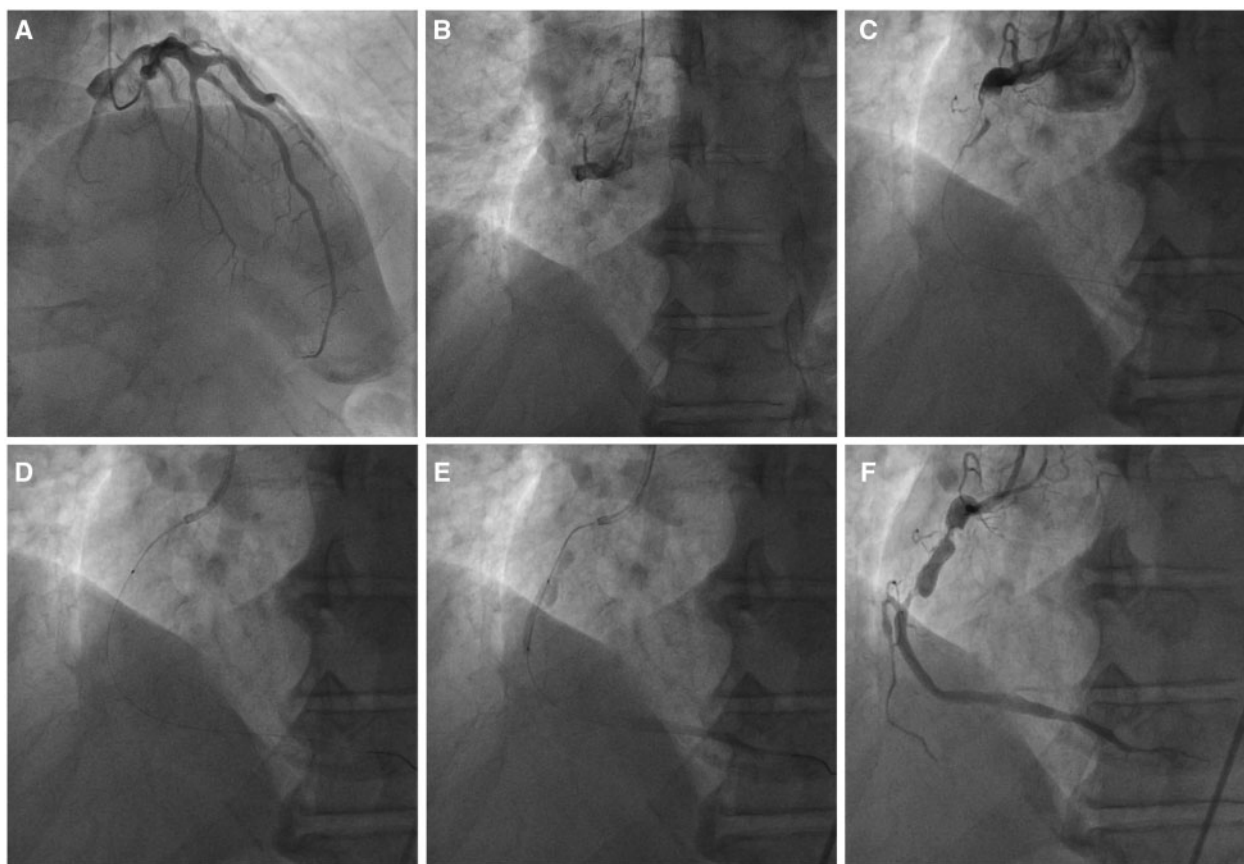
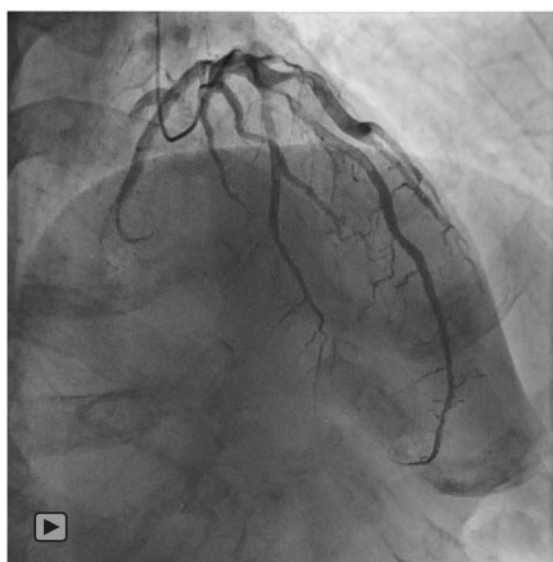
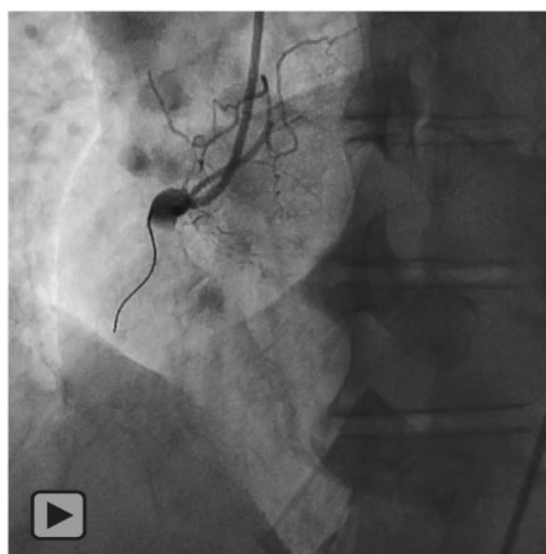


Figure 1 Percutaneous coronary intervention of the right coronary artery (first session). (A) Severe stenotic lesion at the proximal site of the left anterior descending artery. (B) Total occlusion at the proximal site of the right coronary artery. (C) Coronary guidewire advanced distally beyond the culprit lesion. (D) Manual thrombus aspiration with Export catheter. (E) Balloon angioplasty with non-compliant balloon. (F) Final result.



Video 1 Coronary angiography (first session) showing a critical lesion of the proximal left anterior descending artery.



Video 2 Angiography showing the thrombotic occlusion of the proximal right coronary artery.

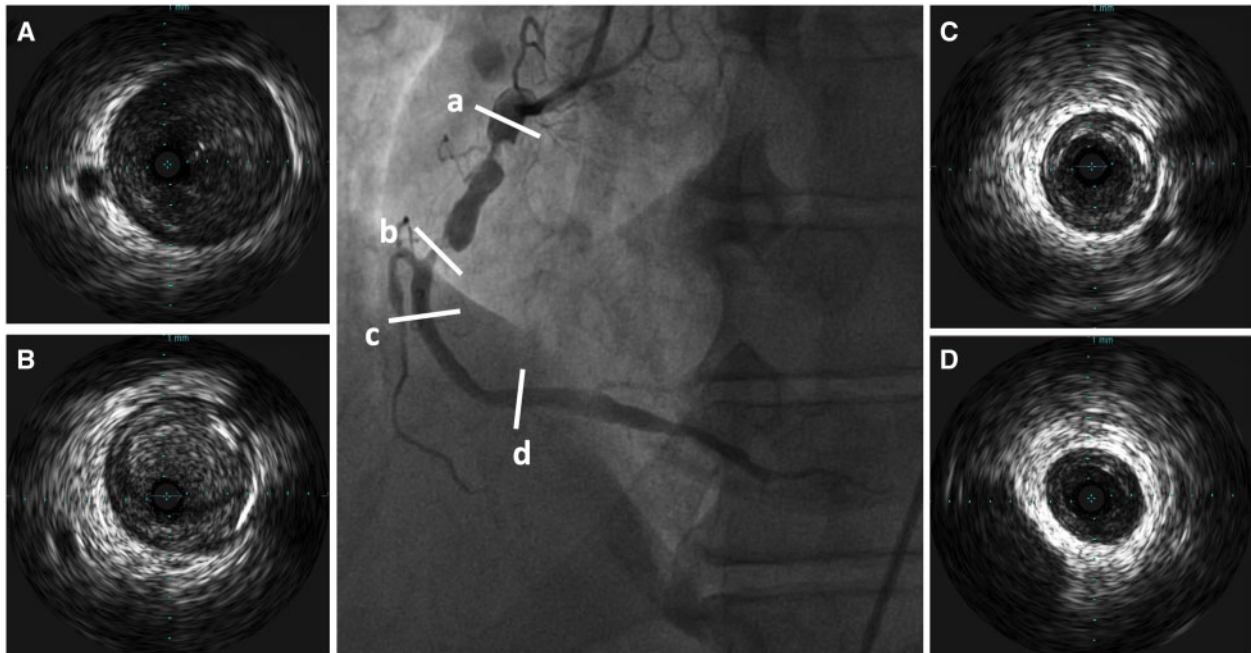
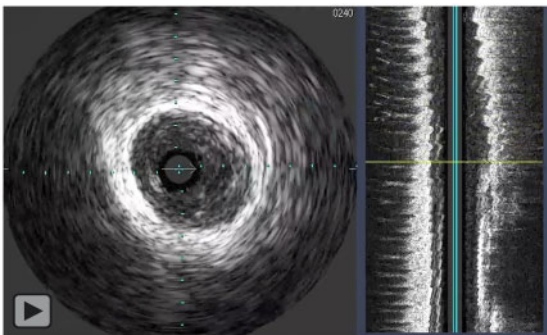


Figure 2 Intravascular ultrasonography findings of the right coronary artery (first session). (A) Marked ectasia of the proximal segment. (B) Severe stenosis with massive thrombus burden in the mid-segment. (C) Marked ectasia of the mid-segment. (D) Distal right coronary artery.



Video 3 Cross-sectional and longitudinal intravascular ultrasonography appearance of the right coronary artery.

perfusion and thrombus formation.⁵ The appropriate treatment for CAA and CAE is still matter of debate. Current recommendations are based on limited case studies and anecdotal observations, while large randomized clinical trials are missing. Moreover, revascularization with either PCI or surgery faces significant technical challenges.

In the setting of acute myocardial infarction (MI), PCI of an aneurysmal/ectatic culprit vessel is associated with lower procedural success, higher mortality and MI recurrence [mainly driven by higher stent thrombosis (ST) rates] at mid-term follow-up, compared with patients without coronary dilation.^{6–8} Stent malapposition, residual thrombus, and flow disturbances may account for the higher rates of ST observed in this population,⁸ and may be partly due to the lack of

dedicated devices. In fact, currently available balloon-expandable and self-apposing drug-eluting stents can expand up to 6.0 mm maximum diameter. However, as in this case, the vessel diameter in the aneurysmal/ectatic coronary segment is often larger than 6 mm, carrying an intrinsic risk of stent malapposition and subsequent ST.

Several interventional techniques may help to overcome these intrinsic anatomical challenges. In general, when balloon-expandable devices are used, the implantation of stents with a length strictly limited to the stenotic tract is the strategy of choice. This helps to minimize the risk of coronary stent loss and/or malapposition and prevents landing the device in the adjacent CAA/CAE. The off-label percutaneous treatment of CAE with polytetrafluoroethylene-covered stents has also been reported, with acceptable mid-term outcomes when used for large atherosclerotic aneurysms with a diameter of 6–10 mm.⁹ However, the use of covered stents in this setting is hampered by the low flexibility of the system in tortuous vessels, the risk of occluding a side branch, incomplete coverage of the ectatic lesion, and late stent restenosis.^{10,11} An alternative strategy is the so-called ‘stent-assisted’ embolization, which involves percutaneous treatment of the CAA using membrane-covered stents and coil embolization.³ However, this technique is not useful for quite large CAA or when severe long, diffuse CAE is present, and is not practical in the setting of primary PCI.

Lastly, in patients with CAA, surgical revascularization has recently been shown to be superior to medical therapy alone, whereas it appears there’s no statistically significant difference between coronary artery bypass grafting (CABG) and PCI at long-term follow-up.¹² In our case, surgical revascularization was excluded because of clinical instability, with recurrence of chest pain and new ST-segment

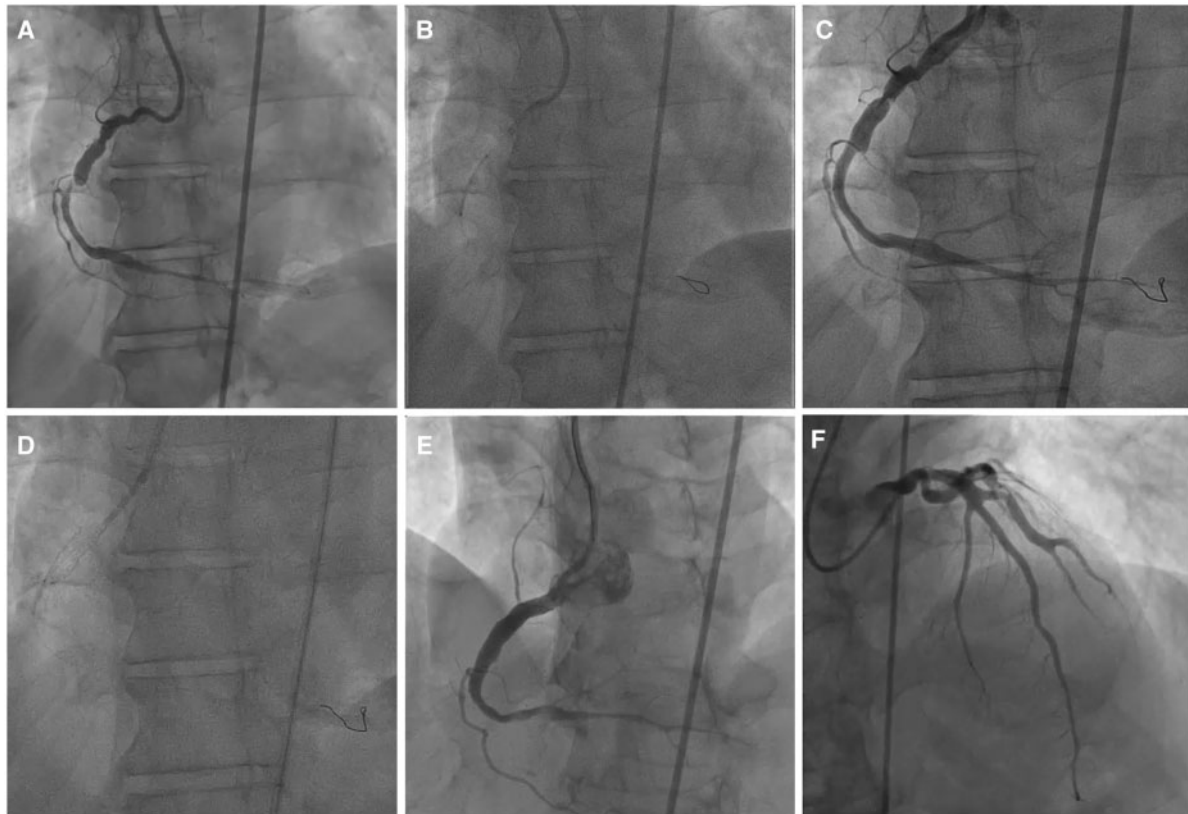


Figure 3 Percutaneous coronary intervention of the right coronary artery (second session). (A) Coronary angiography showing persistence of sub-occlusion in the context of a severe ectasia of the proximal and mid-right coronary artery. (B) Stentys Xposition S deployment in the mid-right coronary artery. (C) Result after Stentys deployment. (D) CGuard deployment in the proximal and mid-segments of the right coronary artery. (E) Final right coronary artery result. (F) Left anterior descending artery after stenting.

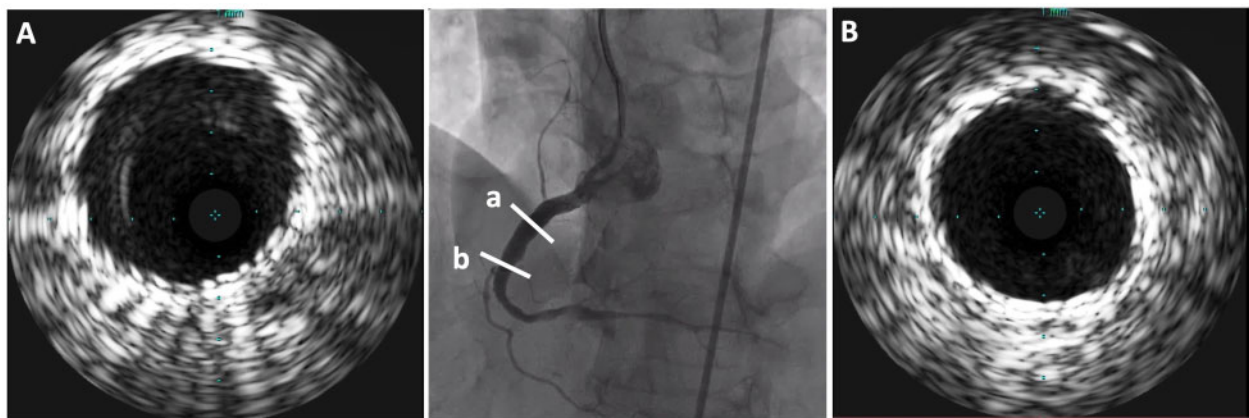


Figure 4 Intravascular ultrasonography showing good stent expansion and apposition to the vessel wall in the proximal (A) and mid (B) right coronary artery.

changes, and because of pre-treatment with ticagrelor. However, we believe that CABG is a viable alternative to PCI in cases of large CAA, and therefore should be considered as a preferred strategy in stable patients.

To our knowledge, our report is the first description of a self-expanding CGuard carotid stent implantation for the treatment of CAD in the context of CAA/CAE. This device consists of a thin strut nitinol self-expanding stent that is combined with a polyethylene

terephthalate mesh. The stent is available in an array of diameters (6–10 mm) and lengths (20–60 mm) and is CE mark approved for carotid artery stenting. We believe that its technical characteristics make the device particularly suitable for the treatment of coronary lesions with high thrombus burden, when severe CAA/CAE is present. As a matter of fact, the CGuard stent can achieve a good apposition in vessels with RVD between 4.8 and 9 mm, allowing the treatment of most CAA/CAE with lower risk of stent malapposition. Moreover, its specific design, with a free-cell area of $165 \mu\text{m}^2$, allows the stent to trap and exclude thrombus and plaque debris preventing acute and late embolic events during and after PCI procedures.¹³ Longer follow-up studies in a larger population are needed to validate these preliminary observations.

Lead author biography



Alberto Barioli graduated in Medicine and Surgery at the University of Padua in 2011; he obtained a Postgraduate Diploma in Cardiology at the same University in 2017. From 2017 to 2018, he worked as a research fellow in the Hemodynamics and Interventional Cardiology Unit of the Cardiology Clinic of Padua. Since 2019, he is a staff member at the Interventional Cardiology Unit of the Cardio-Neuro-Vascular Department of the Ca' Foncello

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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.

Conflict of interest: None declared.

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