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CASE REPORT

INTERMEDIATE

HEART CARE TEAM/MULTIDISCIPLINARY TEAM LIVE

Progressive Dilation of Coronary Artery Ectasia Causing Recurrent Myocardial Infarction

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ABSTRACT

We present a case of recurrent myocardial infarction with coronary artery ectasia that had progressive dilation. Both implanting drug-eluting stent and antithrombotic therapy with warfarin plus P2Y12 inhibitor were feasible. The careful follow-up including morphologic evaluation may be needed for this specific lesion. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2023;24:102044) © 2023 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

CASE PRESENTATION

A 64-year-old woman with background of hypertension, diabetes mellitus, and dyslipidemia initially presented with ST-segment elevation myocardial infarction (MI). Coronary angiography (CAG) revealed an ectatic right coronary artery (RCA) (**Figure 1A**, Video 1) with occlusion of the distal vessel. Of note, the left coronary artery (**Figure 1B**, Video 2) was also ectatic with associated slow flow, but there was no evidence of flow-limiting stenosis or thrombus. Owing to concerns that the presence of massive thrombus would predispose the patient to a high risk of stent thrombosis, the occluded RCA lesion was treated medically with antithrombotic therapy. This was initiated with continuous infusion of heparin (activated clotting time 160-180 seconds) as an inpatient, followed by both warfarin and aspirin.

One month after commencing antithrombotic therapy, the follow-up CAG showed no residual thrombus and improved coronary flow with TIMI flow grade 3 (**Figure 1C**, Video 3). Thereafter she was maintained on antithrombotic therapy with long-term warfarin monotherapy, and then 10 years later, the regimen was changed to 60 mg edoxaban monotherapy.

Fifteen years after the initial MI presentation, she was readmitted with sudden-onset anterior chest pain to our emergency department.

LEARNING OBJECTIVES

- To consider the implantation of a drug-eluting stent or covered stent to achieve coronary patency in a patient who presented with acute myocardial infarction involving coronary artery ectasia.
- To examine the possible mechanism of dilation of coronary artery and consider following the patient by morphologic evaluation.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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ABBREVIATIONS AND ACRONYMS

CAE = coronary artery ectasia

CAG = coronary angiography

MI = myocardial infarction

PCI = percutaneous coronary intervention

RCA = right coronary artery

PAST MEDICAL HISTORY

Other medical history included depression that improved with antidepressant treatment.

INVESTIGATIONS

At readmission, electrocardiography revealed ST-segment elevation in the inferior leads with sinus rhythm (**Figure 2**), and echocardiography detected hypokinesis of the left ventricular inferior wall. Owing to the patient's prior episode of inferior MI, CAG was immediately performed, which showed occlusion of the mid-RCA along with a substantial thrombus burden, which was in a different segment than the culprit lesion responsible for the previous MI (**Figures 3A and 3B**, Videos 4 and 5). Moreover, the ectatic morphology of RCA was significantly enlarged throughout the entire vessel compared with the previous examination (**Figure 4**).

MANAGEMENT

Emergency primary percutaneous coronary intervention (PCI) was carried out via transfemoral approach. After successful guidewire crossing with assistance of a microcatheter, the target lesion was dilated with the use of 2.5 mm and 3.0 mm semi-compliant balloons followed by implanting a zotarolimus-eluting stent (4.0/18 mm) in the focal stenosis where coronary flow was most restricted (**Figure 3C**). Despite significant thrombi remaining in the surrounding segments of the stented lesion, the ectatic morphology of the lesion rendered further intervention with additional stenting unsuitable. The final angiogram demonstrated TIMI flow grade 2 (**Figure 3D**, Video 6), and consequently intra-aortic balloon pumping was implemented with unfractionated heparin to optimize coronary flow while maintaining activated partial thromboplastin time within the 180- to 220-second range.

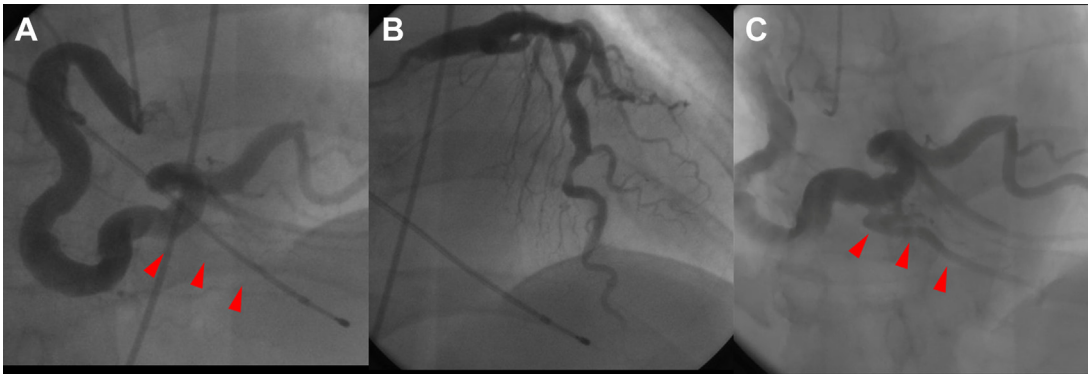
FOLLOW-UP

Seven days after the procedure, computed tomographic coronary angiography revealed that the RCA was patent, including the distal segments, with minimal residual thrombus present (**Figure 5**). Warfarin and prasugrel (3.75 mg/d) were prescribed and the patient was discharged 14 days after admission without any further adverse events. At the 12-month follow-up, the patient reported no further cardiac symptoms or events. A surveillance coronary angiogram was conducted, which revealed patent RCA, including the distal vessel, without residual thrombus or stent restenosis (**Figures 6A and 6B**, Video 7).

DISCUSSION

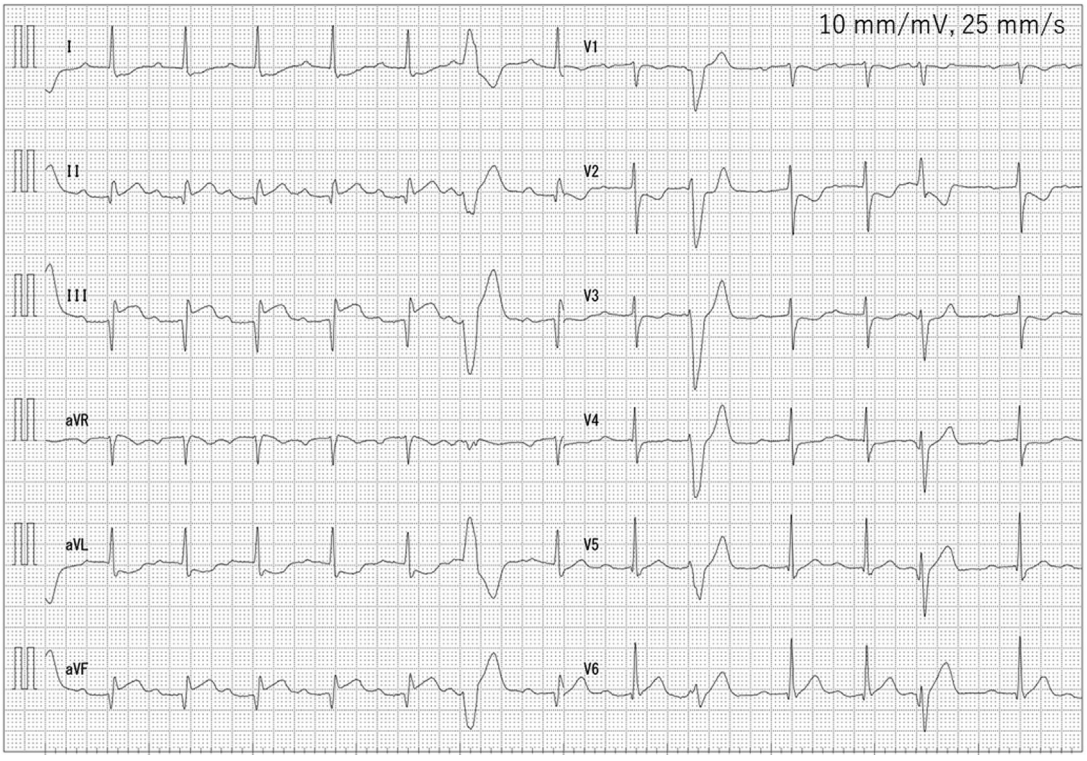
This is the first report to show progressive dilation of coronary artery ectasia resulting in recurrent MI in

FIGURE 1 Coronary Angiography at the First Time of Myocardial Infarction (15 Years Ago)



(A) Angiography shows the occlusion of the distal part of the right coronary artery (red arrowheads; LAO 0, CRA 26). (B) The left coronary artery is also ectatic with associated slow flow without stenosis or thrombus (LAO 0, CRA 27). (C) Coronary angiography after the antithrombotic therapy for 1 month shows that the thrombus has diminished at the distal part and coronary flow has improved (LAO 0, CRA 30). CRA = cranial; LAO = left anterior oblique.

FIGURE 2 **Electrocardiography at the Emergency Department**



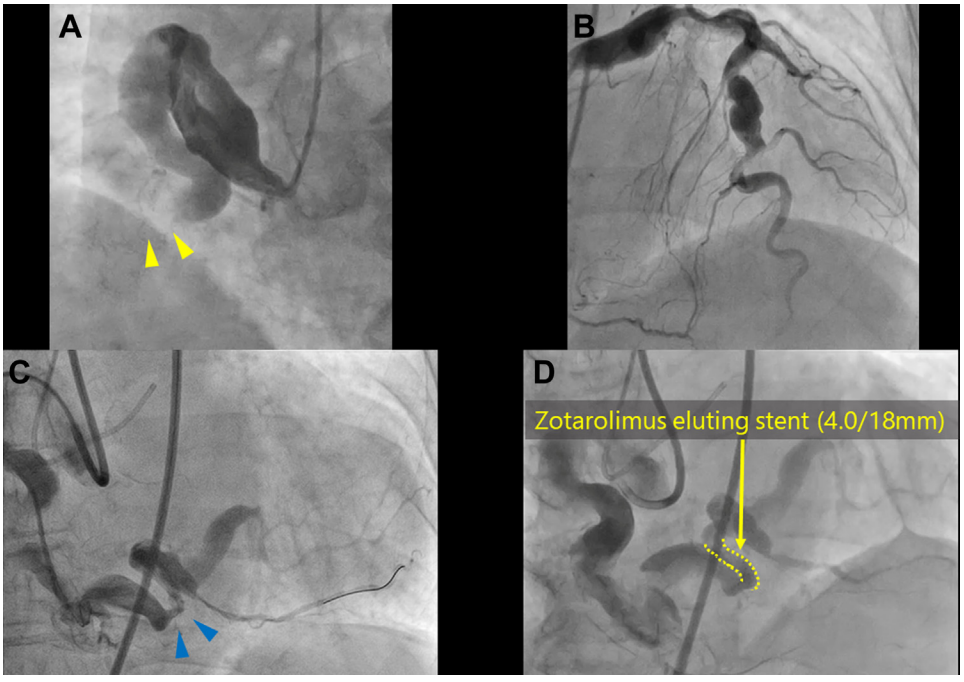
The electrocardiogram shows ST-segment elevation of the inferior leads.

the same vessel confirmed by coronary angiography. This case highlights the importance of considering the etiology of coronary artery disease to determine its underlying mechanism, to facilitate appropriate treatment, and optimal follow-up protocols.

WHY DID THIS PATIENT HAVE PROGRESSIVE ENLARGEMENT OF CORONARY ARTERY ECTASIA? It has been reported that the pathophysiology of coronary artery ectasia (CAE) is associated with systemic inflammatory diseases, infections, and connective tissue disorders.[1] Furthermore, it has been previously reported that conditions such as Kawasaki disease (acute febrile mucocutaneous lymph node syndrome), antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis, and immunoglobulin G4 (IgG4)-related diseases particularly are known to induce CAE.[2-4] In our case, a comprehensive work-up was conducted, including blood examination such as high-sensitive C-reactive protein, serum interleukin-2, antinuclear antibody, ANCA, IgG4, and other autoantibodies; however, there was no evidence of chronic inflammation detected. In addition, we conducted multimodality imaging, including arterial ultrasound examination of the lower extremities and carotid artery, as well as whole-body CT scanning. These imaging studies confirmed the absence of atherosclerotic or aneurysmal changes in the major arteries and aorta. Of note, the patient had no medical history of Kawasaki disease, no exposure to specific drugs known to be associated with coronary aneurysm,[5] and no relevant family history.

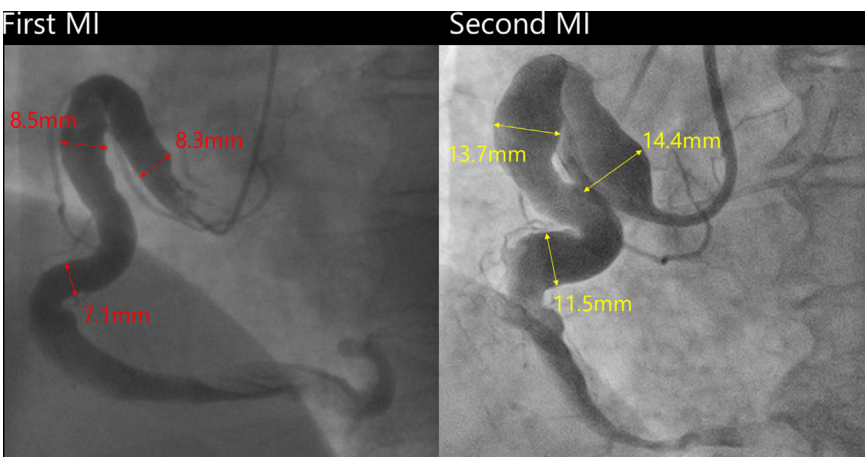
Although factors worsening CAE have not been fully elucidated, to make better decisions to manage this complex lesion it is crucial to carefully manage and follow these patients with a multidisciplinary approach.

FIGURE 3 Coronary Angiography at the Recurrent Myocardial Infarction



Angiography shows (A) total occlusion of the middle right coronary artery (LAO 40, CRA 0) and (B) ectasia of the left coronary artery (LAO 0, CRA 30) as well. (C) After the recanalization, coronary flow is restricted at the focal stenosis (blue arrowheads; LAO 0, CRA 10). (D) The final angiogram after stent implantation (yellow dotted line) remains thrombus and flow limitation at the distal part. (LAO 0, CRA 29).
CRA = cranial; LAO = left anterior oblique.

FIGURE 4 The Changes in the Diameters of Coronary Arteries Over 15 Years Measured by Quantitative Coronary Angiography Analysis



Compared with (left) the previous myocardial infarction (MI) 15 years before (LAO 40, CRA 0), angiography at (right) the second MI shows a more dilated coronary artery (LAO 41, CRA 0). CRA = cranial; LAO = left anterior oblique.

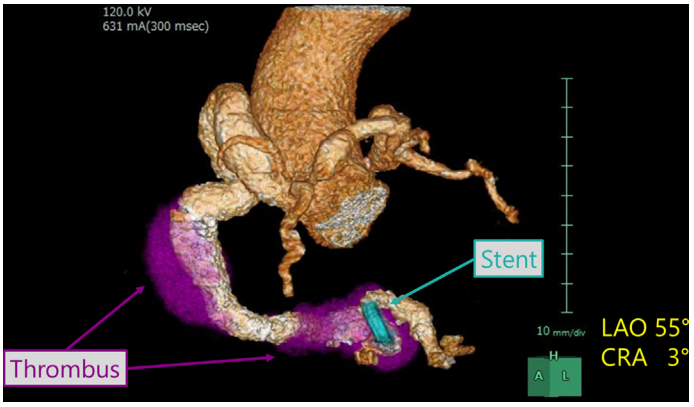
WHAT WOULD BE THE BEST POSSIBLE STRATEGY FOR THE TREATMENT OF THROMBOTIC OCCLUSION DUE TO ADVANCED CAE? PCI strategies for stenotic or thrombotic lesions associated with CAE have not been established. In our case, the presence of complex lesion morphology caused by CAE, resulting in slow flow, and concomitant flow-limiting stenosis posed a challenge in determining the optimal management strategy to improve and sustain coronary flow, even during long-term follow-up. A previous case report has shown CAE-associated acute coronary syndrome successfully treated by implanting a drug-eluting stent that resulted in a patent stent and complete sealing of the aneurysm at the 6-month follow-up.[6] In contrast to that case with relatively focal ectasia (aneurysm), our case had diffusely diseased ectasia. Therefore, the lesion morphology made us hesitate regarding implanting stents owing to the risk of significant malapposition which might result in high thrombogenicity. Finally, we selectively implanted a drug-eluting stent at a focal stenotic lesion where acceptable stent apposition was expected to be achieved.

On the other hand, even for this young patient, coronary artery bypass surgery would not be a better alternative therapy, owing to CAE enlargement, as demonstrated during 15 years of follow-up. Therefore, it would be difficult to decide ideal segments for multiple bypass grafting with guarantee for their long-term patency.

In this case, the patient experienced recurrent occlusion while on antithrombotic therapy with edoxaban monotherapy, therefore, even with appropriate dosage, direct oral anticoagulant may not be enough to prevent thrombotic events, especially for patients with large CAE.

Doi et al[7] reported that in patients who underwent primary PCI after MI with CAE, the use of a target therapeutic range of warfarin ≥60% in combination with antiplatelet therapy resulted in no recurrence of MI over a 49-month follow-up period. Although there was no robust evidence regarding the optimal antithrombotic therapy regimen for managing patients with CAE, in this case we prescribed both warfarin and with P2Y12 inhibitor (prasugrel). Antiplatelet considerations included possible genetic variations in the CYP2C19 isozyme, which can lead to reduced efficacy due to impaired conversion of clopidogrel to its active metabolite.[8] Of note, as of this writing the patient remained free from the recurrence of MI and any bleeding events for a period of 12 months.

FIGURE 5 **Coronary Computed Tomographic Angiography After Primary Percutaneous Coronary Intervention**



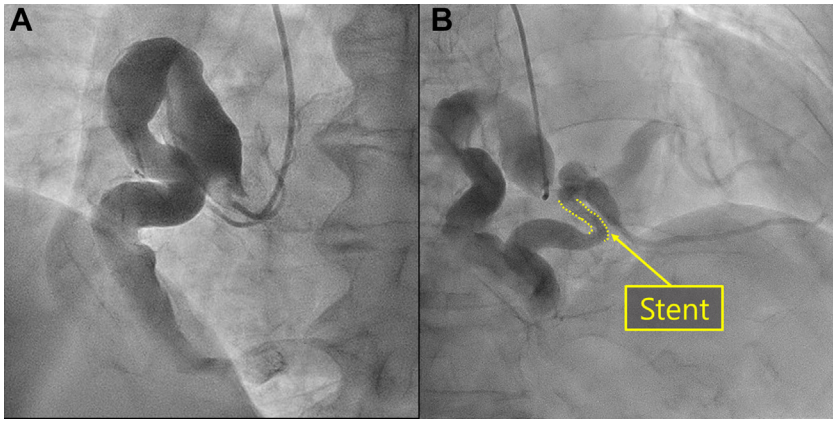
Computed tomography shows the remained thrombus (purple-colored area) but the coronary flow is preserved up to the distal part (stent: turquoise-colored area; LAO 55, CRA 3). CRA = cranial; LAO = left anterior oblique.

HOW SHOULD WE FOLLOW THE PATIENT? This case showing progressive dilation of CAE emphasizes the importance of effectively managing risk factors associated with atherosclerosis and maintaining careful follow-up. Not only clinical follow-up, but also morphologic assessment of CAE itself through multimodality imaging surveillance, such as CAG, computed tomographic coronary angiography, or magnetic resonance imaging, may be helpful with early identification of progressive disease and preventing MI recurrence.

CONCLUSIONS

CAE may exhibit considerable variance regarding its causes, distribution, and morphologic characteristics. The present case represents recurrent MI due to CAE, which progressively enlarged over a period of 15 years. It is vital that patients with CAE are comprehensively investigated, including whether there is an underlying cause, ensuring adequate antithrombotic therapy, and remain under surveillance including morphologic evaluation of the coronary arteries as needed.

FIGURE 6 Coronary Angiography 12 Months After Second Myocardial Infarction



Angiography reveals that the thrombus has disappeared without flow limitation or stent (yellow dotted line) restenosis (left: LAO 40, CRA 0; right: LAO 0, CRA 30). CRA = cranial; LAO = left anterior oblique.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS acute myocardial infarction, coronary artery ectasia, coronary dilation, thrombus

APPENDIX For supplemental videos, please see the online version of this paper.

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