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Effective Medical Therapy of Coronary Artery Aneurysms and Ectasia: A Case Report

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Case Report

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Abstract

Background: Coronary artery aneurysm (CAA) and coronary artery ectasia (CAE) are uncommon but clinically significant abnormalities associated with adverse cardiovascular outcomes. This case highlights the diagnostic and therapeutic challenge of acute coronary syndrome in patients with angiographically non-obstructive disease.

Case Presentation: We present a case of a 56-year-old male who presented with retrosternal burning pain. He was found to have developed a non–ST-elevation myocardial infarction despite the absence of obstructive coronary artery disease. Coronary angiography revealed multivessel ectasia, including two mid-segment aneurysms in the left circumflex artery with an intervening slow-flow region, and a dominant, diffusely ectatic right coronary artery without significant stenosis. The presentation was attributed to impaired coronary flow dynamics and thrombus formation rather than fixed luminal obstruction. The patient was managed medically with anticoagulation, beta-blockade, statin therapy, and risk factor modification.

Conclusion: This case highlights the diagnostic and therapeutic challenge of acute coronary syndrome in patients with angiographically non-obstructive disease. It underscores the need for individualized, risk-based management strategies in patients with coronary ectasia in the absence of definitive guideline-directed therapy.

Background

Coronary artery aneurysm (CAA) and coronary artery ectasia (CAE) represent abnormal dilatation of the coronary vasculature and are generally considered related phenotypes along the same disease spectrum. A coronary artery aneurysm is defined as a focal dilatation of a coronary segment measuring at least 1.5 times the diameter of an adjacent normal reference segment, whereas coronary artery ectasia refers to diffuse dilatation involving a substantial length of the vessel. [1]

The reported angiographic prevalence of CAA and CAE is approximately 5%, with a higher frequency observed in men. These entities have been associated with a variety of etiologies, including atherosclerosis, chronic inflammatory conditions, iatrogenic causes like stent placement and substance use, although atherosclerosis remains the most common underlying cause in adults. Morphologically, coronary aneurysms are classified as saccular or fusiform based on their shape, and coronary ectasia is categorized according to the extent and distribution of vessel involvement [2]

Though they are frequently detected incidentally, increasing evidence suggests that these abnormalities are associated with adverse cardiovascular outcomes, including myocardial infarction, thrombosis, distal embolization, and major adverse cardiac events. Because randomized clinical trial data are lacking, management remains largely individualized and guided by observational evidence and expert opinion. [3]

We present a case of NSTEMI associated with multivessel coronary ectasia and aneurysmal coronary disease in the absence of obstructive coronary artery disease.

Case Presentation

Patient Information

A 56-year-old male with a history of gastroesophageal reflux disease presented to the emergency department with persistent retrosternal burning discomfort lasting approximately four hours. The discomfort was more severe than his typical reflux symptoms and did not improve with over-the-counter calcium carbonate.

The patient denied tobacco use, alcohol use, or illicit drug use. No relevant family history was reported.

Clinical Findings

On presentation, the patient's heart rate was 91 beats per minute, blood pressure was 150/90 mmHg, and oxygen saturation was 95% on room air.

Physical examination revealed no acute distress and was otherwise unremarkable.

Timeline

Time Point	Clinical Event
Day 0	Presentation with persistent chest discomfort
Day 0	Elevated high-sensitivity troponin detected
Day 0	Serial troponin increase to approximately 17,000 ng/L
Day 0	Aspirin, clopidogrel, and nitroglycerin administered
Day 0	Coronary angiography performed
Day 0	Multivessel ectasia and LCx aneurysms identified
Hospitalization	Medical therapy initiated
Discharge	Outpatient cardiology follow-up arranged

Diagnostic Assessment

Electrocardiography demonstrated sinus rhythm with left ventricular hypertrophy by voltage criteria without ischemic ST-segment or T-wave abnormalities.

Initial high-sensitivity troponin measured 1,748 ng/L and subsequently increased to approximately 17,000 ng/L, consistent with acute myocardial injury.

Coronary angiography demonstrated a patent left main coronary artery bifurcating into the LAD and LCx arteries. Mild proximal LAD ectasia with distal luminal irregularities was identified without significant stenosis. The LCx demonstrated two aneurysmal dilatations in the mid-vessel and an additional distal aneurysmal segment. (Fig. 1) The segment between the two mid-LCx aneurysms demonstrated slow coronary flow without significant obstructive stenosis. The RCA was dominant and diffusely ectatic throughout its course without focal stenosis. (Fig. 2)

Figure 2: Ectatic Right Coronary Artery. Coronary angiography demonstrating a dominant, large right coronary artery (RCA) with diffuse ectasia throughout its course, free of obstructive stenosis.

The absence of obstructive coronary artery disease together with angiographic evidence of diffuse ectasia, aneurysmal dilatation, and slow coronary flow suggested that impaired coronary hemodynamics and thrombosis-related mechanisms contributed to the patient's myocardial infarction.

Therapeutic Intervention

The patient was treated medically with:

- • Apixaban
- • Carvedilol
- • High-intensity statin therapy
- • Angiotensin receptor blocker

Aggressive cardiovascular risk-factor modification was recommended.

Follow-Up and Outcomes

The patient remained clinically stable throughout hospitalization and experienced no recurrent chest discomfort prior to discharge.

He was discharged with outpatient cardiology follow-up and instructions regarding medication adherence and cardiovascular risk-factor modification. At the time of manuscript preparation, no additional follow-up imaging or long-term outcome data were available.

Based on these findings, the non–ST-elevation myocardial infarction was attributed to coronary artery ectasia with impaired coronary flow rather than obstructive atherosclerotic disease. The patient was managed medically with apixaban, carvedilol, high-intensity statin therapy, and an angiotensin receptor blocker.

Discussion

Coronary artery aneurysm (CAA) and coronary artery ectasia (CAE) are uncommon coronary abnormalities that have increasingly been recognized as clinically significant entities rather than benign angiographic findings. Observational studies have demonstrated that the presence of CAA is associated with adverse long-term outcomes, including increased five-year mortality, higher rates of coronary and non-coronary vascular events, and an elevated risk of major adverse cardiac events. [4]

Atherosclerosis remains the most frequently identified risk factor for CAA in adults. Other reported etiologies include Kawasaki disease, Lyme disease, systemic vasculitides, connective tissue disorders, and substance use. [5] The pathophysiology of aneurysm formation is incompletely understood and is thought to be multifactorial, involving atherosclerotic plaque ulceration with compensatory positive remodeling, chronic nitric oxide–mediated vascular overstimulation, and direct vessel wall injury from inflammatory mediators in genetically predisposed individuals. [6] These mechanisms ultimately result in degradation of the arterial media, leading to progressive weakening and luminal dilatation.

Most CAAs are asymptomatic and are often detected incidentally during coronary angiography. When symptoms occur, they are typically related to complications such as in situ thrombosis, distal embolization, coexisting obstructive atherosclerotic disease, or, less commonly, aneurysmal enlargement causing compression of adjacent cardiac structures.[7] In the present case, the patient's presentation with non–ST-elevation myocardial infarction in the absence of flow-limiting stenosis supports impaired coronary flow dynamics and thrombotic risk related to diffuse ectasia as the most plausible mechanism of myocardial injury.

Management strategies for CAA vary according to the underlying etiology, aneurysm characteristics, and clinical presentation. In Kawasaki disease, treatment pathways are relatively well established. In contrast, management of atherosclerotic CAA remains controversial due to the absence of randomized controlled trials and formal guideline recommendations. In symptomatic patients with obstructive thrombotic disease—manifesting as acute coronary syndrome or stable angina involving the aneurysmal segment—treatment decisions depend on aneurysm size, thrombotic burden, and anatomical considerations. In such cases, covered stent implantation may be considered when the aneurysm is the culprit lesion. If side branches are involved, or in the presence of excessive tortuosity or heavy calcification, stent-assisted coil embolization may be required. [8]

Surgical repair is generally reserved for high-risk scenarios, including aneurysms involving the left main coronary artery, fistula formation, aneurysm diameter greater than 2.5 cm, post-percutaneous coronary intervention aneurysms, rapidly enlarging aneurysms, or compression of adjacent cardiac chambers, regardless of symptom status. [9]

Medical therapy remains the cornerstone of management for non-obstructive CAA and CAE. Patients undergoing stent implantation should receive dual antiplatelet therapy (DAPT) for at least 12 months in accordance with established guidelines. [2] In symptomatic patients without obstructive disease, single

antiplatelet therapy—typically aspirin—is generally considered baseline treatment. Short-term DAPT may be considered in selected higher-risk scenarios, such as recent acute coronary syndrome with an ectatic, non-stented culprit vessel or significant thrombus burden; however, its routine use in stable, non-obstructive CAE remains debated and is not supported by randomized data. [10, 11]

Long-term oral anticoagulation is not routinely indicated for all patients with CAE but may be considered in selected high-risk individuals, including those with extensive or multivessel ectasia, prior or recurrent acute coronary syndromes, or documented intracoronary thrombus. While warfarin was historically used, recent case reports and small series suggest increasing use of non–vitamin K antagonist oral anticoagulants (NOACs). A recent systematic review of case reports suggested that combined antiplatelet and anticoagulant therapy may provide more effective protection against major adverse cardiac events in CAE patients; however, high-quality randomized trials are still lacking. [10]

Adjunctive medical therapies also play an important role. Statins and angiotensin-converting enzyme inhibitors or angiotensin receptor blockers may attenuate aneurysm progression by reducing matrix metalloproteinase activity and limiting positive vascular remodeling. Beta-blockers may provide anti-ischemic benefit, while calcium channel blockers can be useful in mitigating coronary vasospasm. Aggressive risk-factor modification remains essential, given the association between aneurysm progression and atherosclerotic burden. [10]

Conclusion

This case highlights the diagnostic and therapeutic challenges associated with atherosclerotic coronary ectasia presenting as myocardial infarction and underscores the need for individualized, risk-based medical management in the absence of definitive guideline-directed therapy.

Abbreviations

ACS
Acute Coronary Syndrome
CAA
Coronary Artery Aneurysm
CAE
Coronary Artery Ectasia
DAPT
Dual Antiplatelet Therapy
LCx
Left Circumflex Artery
LAD
Left Anterior Descending Artery
NSTEMI

Non–ST–Elevation Myocardial Infarction
NOAC
Non–Vitamin K Antagonist Oral Anticoagulant
RCA
Right Coronary Artery

Declarations

CARE Guideline Compliance

This case report has been prepared and reported in accordance with the CARE (CAse REport) Guidelines. A completed CARE checklist is available and may be provided upon request.

Ethics Approval and Consent to Participate

Institutional Review Board approval was not required for this single-patient case report in accordance with institutional policy and applicable regulations.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical information and images. A copy of the written consent is available for review by the Editor-in-Chief upon reasonable request.

Competing Interests

The authors declare that they have no competing interests.

Patient Perspective

A formal patient perspective was not obtained.

Guarantor

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Author Contribution

SG conceptualized the study, contributed to data collection, and drafted the initial manuscript. KP assisted with data interpretation, literature review, and manuscript revision. SB supervised the project, provided critical revisions for important intellectual content, and approved the final version of the manuscript. All authors read and approved the final manuscript.

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Availability of Data and Materials

All relevant data supporting the findings of this case report are contained within the article. Additional de-identified information may be available from the corresponding author upon reasonable request, subject to patient confidentiality requirements.

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Figures

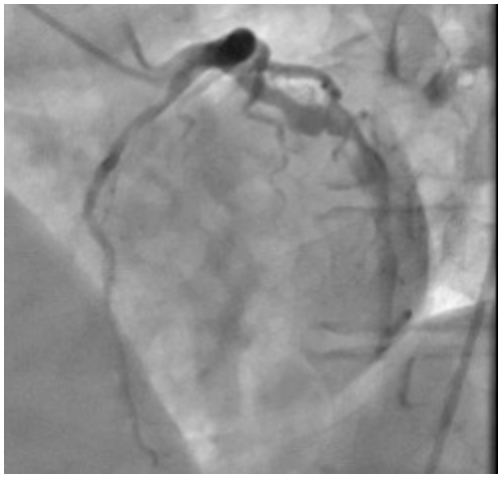


Figure 1

Angiographic evidence of slow coronary flow within the left circumflex artery (LCx)



Figure 2

Ectatic Right Coronary Artery. Coronary angiography demonstrating a dominant, large right coronary artery (RCA) with diffuse ectasia throughout its course, free of obstructive stenosis.

Supplementary Files

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