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A POSSIBLE MODALITY OF INTERVENTION IN A SURGICALLY IMPOSSIBLE CASE OF GIANT CORONARY ANEURYSM: A SUCCESSFUL CASE

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ABSTRAK

Aneurisma arteri koroner besar (>20 mm atau ≥4× diameter normal) merupakan kondisi langka dengan panduan terapi terbatas dan risiko komplikasi serius seperti trombosis, ruptur, dan emboli distal. Penatalaksanaannya masih kontroversial, meliputi terapi medis, intervensi perkutan, atau pembedahan. Dilaporkan kasus laki-laki 64 tahun dengan NSTEMI berisiko tinggi yang ditemukan memiliki aneurisma sakular besar de novo pada segmen mid-LAD, disertai stenosis signifikan pada LAD dan LCX. Karena risiko operasi sangat tinggi, dipilih intervensi koroner perkutan menggunakan covered stent overlap yang berhasil menutup aneurisma (aneurysm exclusion) dan memperbaiki aliran koroner. Pasien stabil pascaprosedur dan dipulangkan dalam kondisi baik dengan rencana tindak lanjut ketat. Kasus ini menunjukkan bahwa PCI dengan covered stent dapat menjadi alternatif yang aman dan efektif pada pasien terpilih berisiko tinggi, dengan dukungan pencitraan lanjutan seperti IVUS serta pengambilan keputusan multidisipliner. Penelitian lebih lanjut masih diperlukan untuk menentukan strategi optimal dan luaran jangka panjang.

ABSTRACT

A Possible Modality of Intervention in a Surgically Impossible Case of Giant Coronary Aneurysm: A Successful Case. Large coronary artery aneurysms (>20 mm or ≥4× normal diameter) are rare conditions with limited therapeutic guidelines and a risk of serious complications such as thrombosis, rupture, and distal embolism. Management remains controversial, including medical therapy, percutaneous intervention, or surgery. We report the case of a 64-year-old man with high-risk NSTEMI who was found to have a large de novo saccular aneurysm in the mid-LAD segment, accompanied by significant stenosis of the LAD and LCX. Due to the very high surgical risk, percutaneous coronary intervention using a covered stent overlap was chosen, which successfully occluded the aneurysm (aneurysm exclusion) and restored coronary flow. The patient was stable post-procedure and discharged in good condition with a close follow-up plan. This case demonstrates that PCI with a covered stent can be a safe and effective alternative in selected high-risk patients, supported by advanced imaging such as IVUS and multidisciplinary decision-making. Further research is needed to determine the optimal strategy and long-term outcomes.

INTRODUCTION

Coronary artery aneurysms (CAA) are rarely identified in adults and are often incidentally diagnosed.[1] Additionally, a giant coronary artery aneurysm is characterized as a coronary dilation

that is fourfold greater than the normal diameter.[2] Giant coronary artery aneurysms are defined as aneurysmal dilatations exceeding 20 mm in diameter and are considered a rare manifestation in clinical practice, with a reported prevalence of approximately 0.02%.[3] Here, we presented a true CAA case of a giant saccular aneurysm in LAD, occurring de novo in a disease-naive individual. It outlines the decision-making process for intervention and follow-up, culminating in the successful percutaneous treatment of the aneurysm with a covered stent.

CASE PRESENTATION

A 64-year-old male was sent to our central hospital from our network branch hospital and admitted to our ER with symptoms of chest pain, which started 1 week before and worsened 2 days before admission, especially during strenuous exercise. The physical exam showed good vital signs and a reduction of angina symptoms, with an irregular heartbeat that corresponded with atrial fibrillation of normal rate on ECG, with several hints toward ischemia in LAD. Our initial diagnosis was high-risk NSTEMI, which was also already discovered at the previous hospital. The patient had a history of CAD, diabetes mellitus, moderate to severe aortic regurgitation (AR), and Atrial Fibrillation treated with medications.

On admission by our cardiology and internal medicine team, the patient received several medications, including parenteral administration of Nitroglycerin 20 mcg/kg/min IV on titration, NaCl 0.9% 500 ml/12 hrs IV, Furosemide 2x20 mg IV, Fondaparinux 1x2.5 mg SC before being switched to Enoxaparin 2x0.6 mg SC, and orally: Aspirin 1x100 mg, Clopidogrel 1x75 mg, Atorvastatin 1x40 mg, Digoxin 1x0.25 mg, Candesartan 1x8 mg, Spironolactone 1x25 mg, Nitroglycerin 2x2.5 mg, Xigduo XR (Dapagliflozin 10 mg/Metformin 1000 mg) 1x1, Glimepiride 1x2 mg, Vildagliptin 1x50 mg, N-Acetylcysteine 3x200 mg, and Fenofibrate 1x100 mg.

The patient then underwent routine labs and a chest X-ray, with daily fluid and kidney function monitoring. It is noteworthy that high-sensitivity troponin T was elevated to 119.80 ng/L from a baseline of less than 14. The patient was scheduled for Coronary Arteriography and Early PCI with IVUS Guide; however, this was postponed to the next day due to a packed schedule. Consequently, echocardiography and Coronary Computed Tomography Angiography (CCTA) were performed for screening

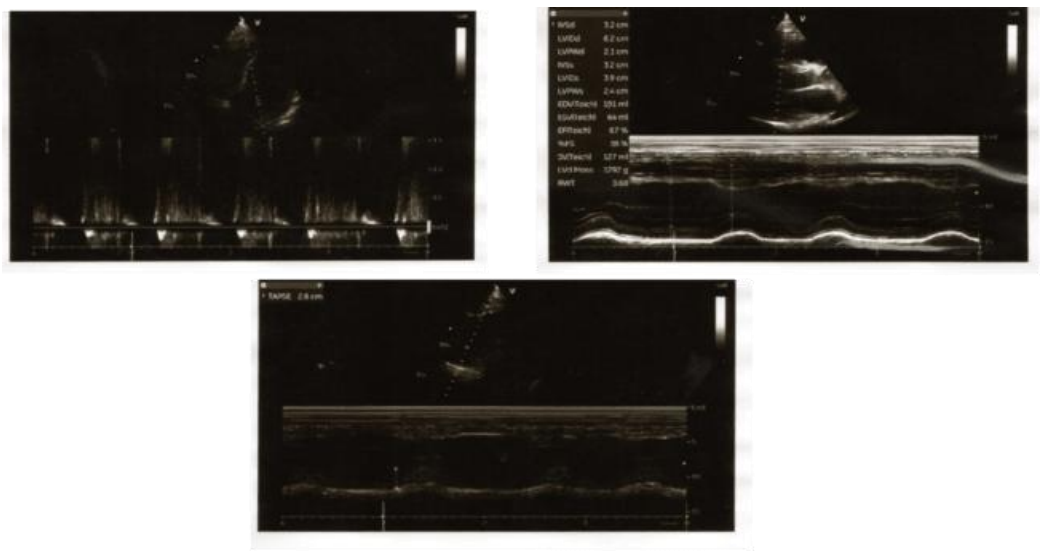

Image 1. Echocardiography Results

(a) Upper left panel: Doppler echocardiography evaluating cardiac flow patterns. **(b)** Upper right panel: M-mode echocardiography demonstrating preserved left ventricular systolic function. **(c)** Lower panel: M-mode echocardiography showing tricuspid annular plane systolic excursion (TAPSE) for right ventricular function assessment.

Upon Echocardiography, results showed normal Ejection Fraction (EF), however, our CCTA unexpectedly revealed a Giant Saccular Coronary Aneurysm in Mid LAD, with significant stenosis of LAD and Left Circumflex Artery (LCX).

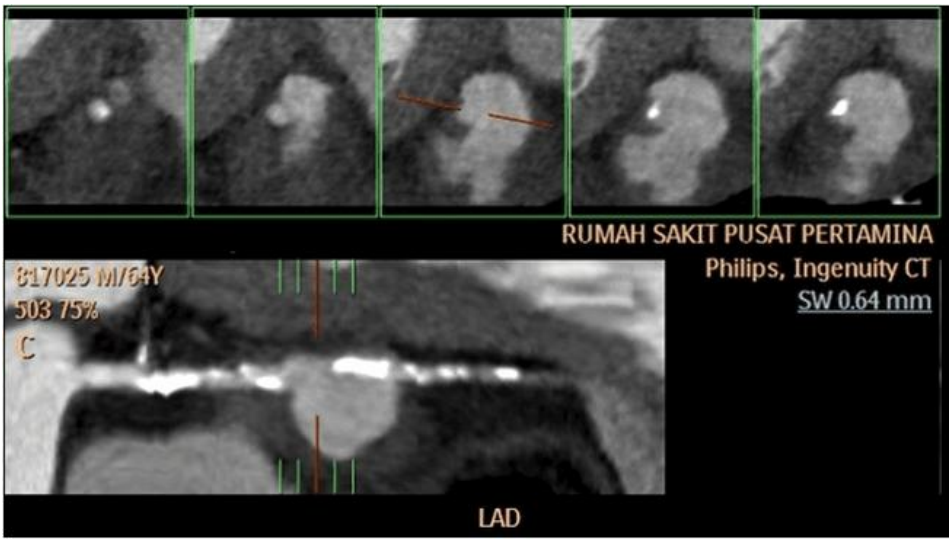

Image 2. CCTA Results

(a) Upper panel: Sequential axial CCTA images demonstrating a saccular dilatation of the mid LAD. **(b)** Lower panel: Curved multiplanar reconstruction of the LAD showing the saccular coronary aneurysm and its relationship with the adjacent coronary lumen.

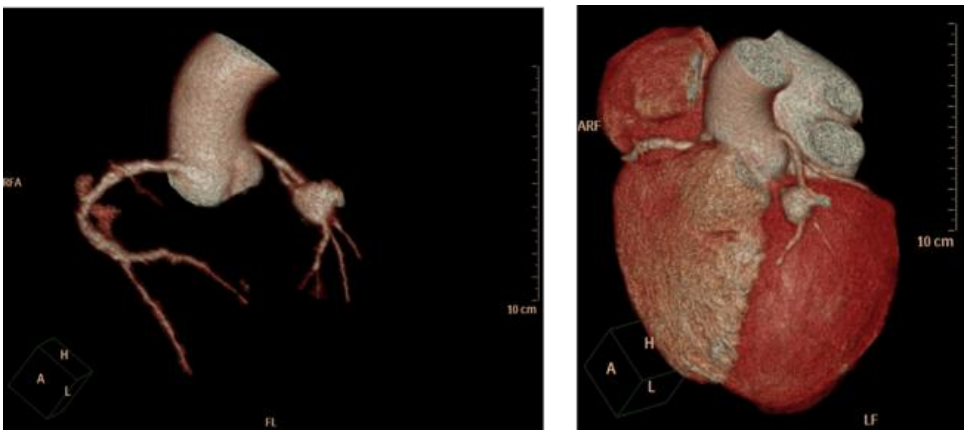

Image 3. CCTA Results in Reproduced 3D Image

(a) Left panel: 3D volume-rendered CCTA image illustrating the coronary arterial anatomy, with a saccular dilatation involving the mid LAD. **(b)** Right panel: 3D volume-rendered CCTA image of the heart showing the spatial relationship between the giant saccular coronary aneurysm of the mid LAD and surrounding cardiac structures.

Therefore, a coronary angiography (CAG) was scheduled and our plans included use of a cover stent to manage the aneurysm. The results showed significant stenosis of the LAD and LCX, but unfortunately, the aneurysm in the mid-LAD appeared to become larger, which made the team diagnosed it with progressing Giant Saccular Coronary Aneurysm of LAD, which then concluded it was unsuitable for PCI and scheduled to a second opinion consult with our cardiothoracic surgeon for Coronary Artery Bypass Graft (CABG) as soon as possible.

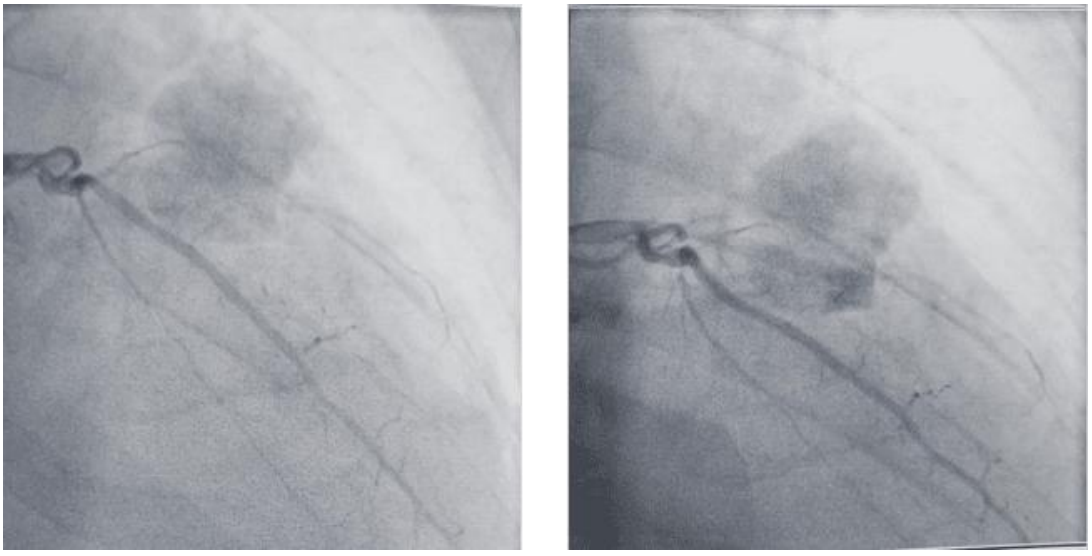

Image 4. Cardiac Catheterization Results of 21 May 2025

(a) Left panel: Coronary angiography of the left coronary system demonstrating contrast opacification of the LAD with visualization of a saccular aneurysmal segment in the mid LAD. **(b)** Right panel: Coronary angiography from a different projection confirming the presence of the mid LAD saccular aneurysm and associated luminal abnormality.

After a thorough assessment, the cardiothoracic surgeon concluded that surgical intervention is unfeasible, given the complexity of the lesion, anticipated high intraoperative risk, and potentially no better outcomes than PCI. Therefore, we were back again with our plan to deploy a covered stent to occlude the aneurysm. This time, the patient showed symptoms of dyspnea and bilateral rales on basal lung auscultation, but still with adequate work of breathing. The patient also showed signs of acute kidney injury, which was still manageable with our medication plan with tight monitoring in the ICU. We then carefully planned our options with the cardiac team, discussed, and studied the CCTA and previous IVUS results, and evaluated hemodynamic function daily and follow-up Echocardiography, which thankfully showed no cardiac tamponade and no regional wall abnormalities with good EF while the patients' symptoms subsided overnight.

After the final decision by the cardiac team, the next day we prepared IVUS Guide Opticross before our second trial of covered stent, prepared graft selection and execution plan, this time we used Biometrix Alpha 3.0 36mm as a basis stent in proximal and distal entry aneurysm, then we put BeGraft 3.0 16mm overlapping with BeGraft 3.5 12mm, which then IVUS showed no flow to the aneurysm and finally concluded successful cover stent occlusion of the aneurysm. Patient was monitored closely for hemodynamic changes, showing good response and reduced cardiac symptoms. The patient was getting better and was allowed to a normal ward after 24 hours, and after 5 more days of stable monitoring, the patient was dismissed home.

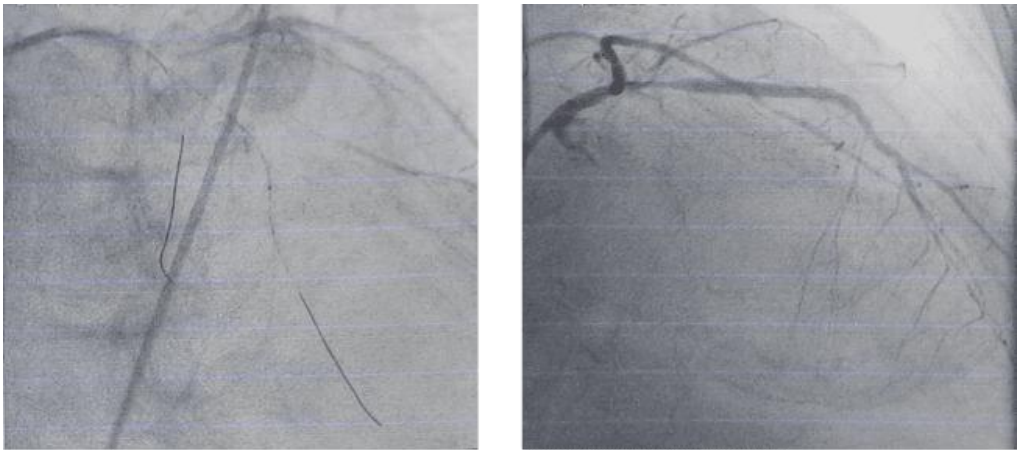

Image 5. Cardiac Catheterization Results of 24 May 2025

(a) Left panel: Coronary angiography of the LAD demonstrating luminal narrowing in the mid segment, visualized prior to further percutaneous intervention planning. **(b)** Right panel: Coronary angiography from an alternative projection confirming the extent of LAD stenosis and delineating the distal coronary flow.

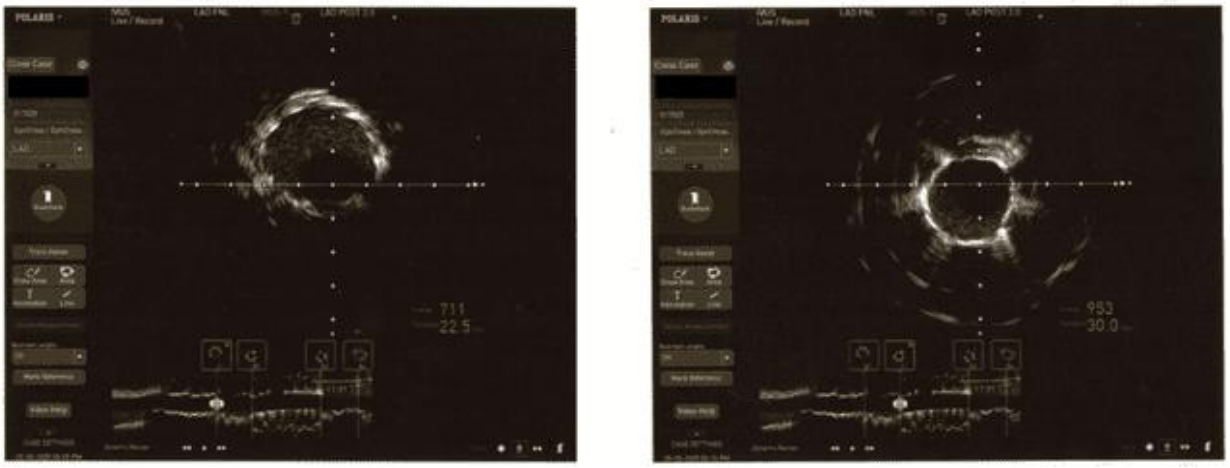

Image 6. Pre-Treatment IVUS Results of 24 May 2025

(a) Left panel: Cross-sectional IVUS image of the left anterior descending artery (LAD) demonstrating an enlarged vascular lumen prior to intervention. **(b)** Right panel: Cross-sectional IVUS image from an adjacent segment of the LAD showing luminal enlargement and vessel wall appearance assessed for procedural planning.

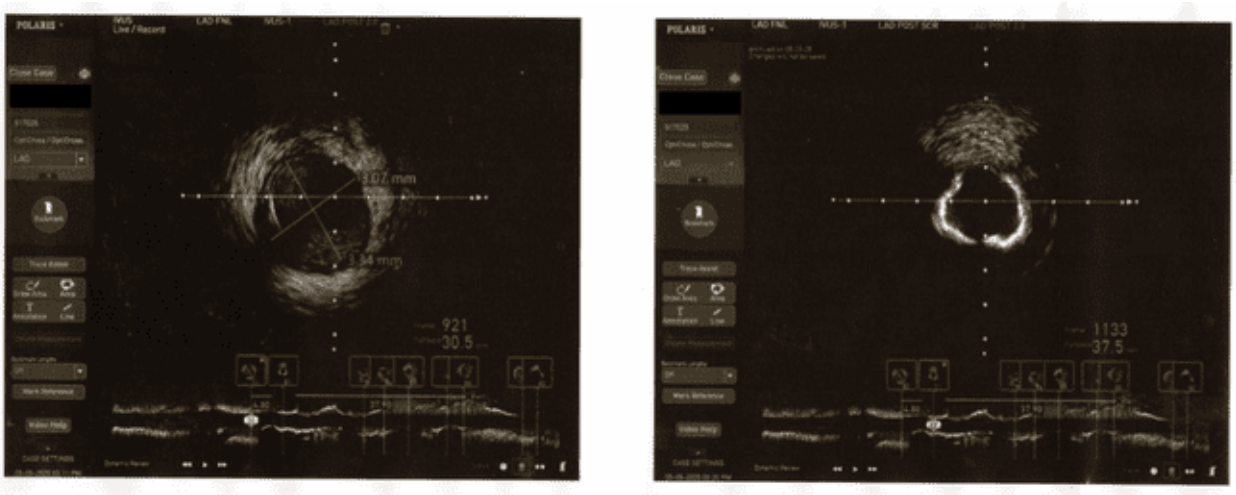

Image 6. Post-Treatment IVUS Results of 24 May 2025

(a) Left panel: Cross-sectional IVUS image of the left anterior descending artery (LAD) following intervention, demonstrating a more regular luminal contour compared with the pre-treatment assessment. **(b)** Right panel: Cross-sectional IVUS image from an adjacent LAD segment after treatment, used to confirm luminal configuration and device positioning.

Oral medications for outpatient care were: Candesartan 1x8mg, Clopidogrel 1x75mg, Dapaglifozin 1x10mg, Nitroglycerin 2x2,5mg, Rosuvastatin 1x20mg, Warfarin 1x2mg, and Spironolactone 1x25mg. Our follow-up at 1-month post-discharge was good, with no significant symptoms or reduction in quality of life; daily living activities were also unproblematic. Furthermore, the patient was scheduled by the cardiac team for LAD PCI in another month and was expected to undergo another LCX PCI later.

DISCUSSION

Coronary artery aneurysm (CAA) is an uncommon condition characterized by a localized enlargement of a coronary artery segment that exceeds 1.5 times the diameter of an adjacent normal section.[4] When the dilation affects more than half the length of the artery in a more diffuse pattern, it is referred to as coronary artery ectasia (CAE).[5] CAAs can present in focal forms, such as those associated with Kawasaki disease, or in more extensive forms resembling ectasia.[6] Morphologically, CAAs are categorized into two main types: saccular (wider than long) and fusiform (longer than wide), with fusiform aneurysms often occurring in the left anterior descending artery. Aneurysms with a diameter of 8 mm or more—or four times the size of a normal coronary segment—are termed giant CAAs.[7] Reported rates of true CAAs range between 0.3% and 5.3%, although newer studies typically observe less than 1% incidence. The right coronary artery is most frequently affected, followed by the left anterior descending, circumflex, and left main arteries. These aneurysms are more common in males and tend to occur in proximal segments.[4]

CAA is often asymptomatic and detected incidentally on imaging, such as coronary angiography or CT angiography. However, symptoms may arise if complications develop or if significant coronary artery disease is also present. These can range from chest discomfort to sudden cardiac arrest. Treatment strategies for CAA are not standardized and depend on several factors, including the patient's symptoms, the nature of the aneurysm, and the physician's judgment. Unfortunately, CAA has been linked to poorer long-term outcomes.[8]

Epidemiology and Classification

CAE is a relatively rare finding during coronary angiography, with prevalence estimates ranging from 0.3% to 5%.[9] This wide range stems from inconsistencies in how CAE is defined, operator variability in interpreting imaging, geographic factors, and differences in patient populations studied. Some studies, particularly those involving patients with ST-elevation myocardial infarction (STEMI), have reported prevalence rates as high as 9%. However, because these findings are based on patients already undergoing invasive evaluation, the actual prevalence in the general population might be overestimated.

CAE is more frequently observed in men and has been associated with risk factors such as high blood pressure, smoking, and abnormal cholesterol levels—including inherited forms like familial hypercholesterolemia.[10] Cocaine use has also been linked to an increased risk. Interestingly, CAE appears to be less common in individuals with diabetes, possibly because diabetes impairs the vessel wall's ability to expand, reducing the likelihood of aneurysm formation.[11] The right coronary artery is the most commonly affected vessel, followed by the left anterior descending, circumflex, and left main arteries.

Moreover, CAE is often found in people with aneurysms in other blood vessels, such as the aorta or the pulmonary arteries. It is also frequently associated with bicuspid aortic valve disease, regardless of whether an aortic aneurysm is present.[9]

Etiologies and Pathophysiology

Atherosclerosis is the leading cause of coronary artery aneurysms, although the exact mechanisms remain unclear. It is believed that atherosclerotic damage weakens the arterial wall’s ability to withstand pressure, leading to dilation.[12] Still, not all patients with atherosclerosis develop CAAs, suggesting a possible genetic predisposition. One proposed mechanism involves matrix metalloproteinases (MMPs), enzymes that break down connective tissue proteins. A particular variant of the MMP gene, MMP3-5A, has been found more frequently in individuals with CAAs.

Besides atherosclerosis, other conditions can cause or contribute to CAAs, including congenital anomalies, Kawasaki disease, Takayasu arteritis, connective tissue disorders like Marfan and Ehlers-Danlos syndromes, and various autoimmune diseases such as lupus and Behçet’s disease. Infections (such as HIV, bacterial, or fungal causes), drug use (including cocaine and amphetamines), trauma, tumors, and even cardiac lymphomas are also implicated. Coronary interventions like stent placement can lead to aneurysm formation, with an incidence of 0.2% to 2.3% after drug-eluting stent deployment. Factors that increase this risk include long lesions, total occlusions, and implantation in infarct-related arteries. Aneurysm formation has also been reported after the use of drug-coated balloons, bioresorbable scaffolds, and rotablation.[13]

Kawasaki disease is a pediatric vasculitis that can lead to CAAs in about a quarter of untreated cases, though this risk drops significantly with proper treatment. In contrast, Takayasu arteritis, which affects young adults, involves inflammation of large vessels and may include coronary aneurysms in 10–12% of cases. Treatment for this condition includes immunosuppressive therapies, corticosteroids, and, in some cases, surgery.

A 2009 review compiled 28 reported cases of giant CAAs, finding atherosclerosis as the cause in half. Other causes included Takayasu arteritis, Kawasaki disease, lupus, and prior stent placement. In some cases, a definitive diagnosis was established by histopathological analysis following surgical procedures.[14]

Clinical Manifestation and Diagnostic Assessment

In most instances, coronary artery aneurysms (CAAs) are asymptomatic and are often discovered incidentally during coronary angiography or even post-mortem examinations. When symptoms do arise, they are influenced by factors such as the aneurysm's cause, size, progression, coexisting arterial narrowing, or complications like fistula formation, thrombosis, embolism, structural compression, rupture, or cardiac tamponade. Among these, rupture, although uncommon, is a particularly severe complication, especially in giant CAAs associated with Kawasaki disease. The underlying vascular wall damage in these cases is due to persistent inflammation driven by neutrophilic infiltration, as confirmed through histological analysis. Another distinct and clinically significant type is the saphenous vein graft aneurysm (SVGA), which frequently ruptures due to its relatively large size.

Angina during exertion is the most typical complaint in symptomatic patients, though others may report breathlessness, unstable angina, myocardial infarction, arrhythmias, sudden cardiac arrest, or signs of heart failure. Myocardial ischemia may also develop even in the absence of significant coronary narrowing, due to impaired blood flow within the dilated vessel segments.

Accurate diagnosis of CAAs involves both invasive and non-invasive imaging. Coronary angiography remains the standard diagnostic method, providing valuable details on aneurysm morphology, size, site, and associated complications, such as impaired perfusion or compression of adjacent structures. However, conventional angiography is limited to viewing blood flow within the lumen and may miss or underestimate aneurysms that are occluded or contain thrombus. Intravascular ultrasound (IVUS) can overcome this limitation by visualizing the arterial wall and differentiating true aneurysms from pseudoaneurysms or mimicking aneurysms due to plaque rupture.

While optical coherence tomography (OCT) can help assess thrombotic features and plaque composition, its effectiveness is limited by its limited imaging depth in larger vessels. Non-invasive imaging, such as coronary CT, has recently gained popularity for its superior ability to visualize aneurysm size, thrombus, and calcification, as well as to detect complications. Echocardiography and coronary magnetic resonance angiography also play roles in diagnosis, particularly for aneurysms located in the proximal coronary arteries. Overall, a multimodal imaging approach is often necessary to thoroughly evaluate CAAs.[15]

Management and Intervention

Managing coronary artery aneurysm or ectasia remains challenging due to the limited understanding of its progression and the lack of comprehensive clinical trial data. Most available evidence is derived from symptomatic patients, especially those presenting with acute coronary syndromes (ACS), while data on asymptomatic cases remain sparse. Treatment strategies ranging from medications to percutaneous or surgical interventions are customized based on clinical presentation, anatomical complexity, and procedural feasibility.[12]

Coronary artery bypass grafting (CABG) has shown promising short- and long-term outcomes in patients with CAAs, although direct comparisons with other treatments, such as percutaneous coronary intervention (PCI), are scarce.[16] In studies comparing CABG and PCI, both approaches showed favorable results, though restenosis occurred more frequently in the PCI group.

PCI may be appropriate in selected cases with favorable anatomy, yet it presents numerous challenges, particularly due to uncertainties about its role in patients without obstructive

disease. For those with significant narrowing or ACS, procedural difficulties such as high thrombus burden, risk of distal embolization, and no-reflow phenomena complicate interventions. Strategies like using glycoprotein IIb/IIIa inhibitors and thrombus aspiration have been employed, though success rates vary. In certain cases with extensive thrombus, deferring stent implantation after aggressive antithrombotic therapy may be considered, though evidence remains limited for CAE-specific cases.

Recent studies, however, have demonstrated that PCI appears to be a longer-term, safe, and successful technique than CABG, with the use of drug-eluting stents associated with improved outcomes.[17] Covered stents may be recommended for aneurysms that are either expanding or exceedingly massive.[18] In our case, the cardiothoracic surgeon team determined that the risk of thoracotomy was high due to the development of the gigantic aneurysm and the complexity of the lesion. Consequently, the covered stent was determined to be the most suitable option, and the aneurysm was undetectable on subsequent angiography after deployment.

Patients with CAAs are at increased risk of stent thrombosis after PCI, likely due to residual thrombus, altered flow, and poor stent positioning. Accurate stent sizing is crucial yet difficult due to the irregular anatomy of aneurysmal segments, and IVUS is strongly recommended to assist with sizing and positioning. OCT can help identify thrombus and plaque features, but its use is limited in large-diameter vessels due to incomplete imaging.

Covered stents can be considered in certain scenarios, such as small saccular aneurysms not involving major branches. Devices like the GRAFTMASTER and PK Papyrus offer options with different flexibility and profile characteristics. However, limitations, including poor deliverability, vessel tortuosity, and the risk of side-branch occlusion, limit their use. Techniques like post-dilation to shorten the stent or creating fenestrations for side branch access have been explored, but are technically demanding and not widely validated.

When covered stents are unsuitable, techniques such as stent-assisted coil embolization may be used, particularly for large aneurysms with wide necks. This method, adapted from neurointerventional procedures, involves deploying coils within the aneurysm sac using a jailed microcatheter. However, this too carries risks like aneurysm rupture or coil herniation. SVGAs may be treated percutaneously in patients who are high-risk surgical candidates, using methods such as covered stents, occluder devices, or coil embolization.[9,19]

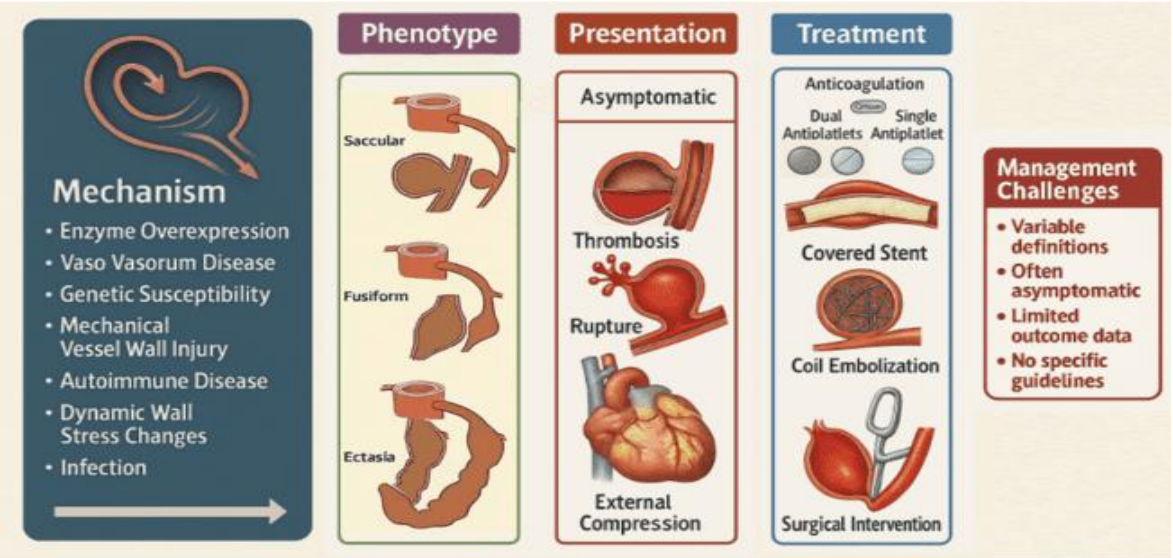

Image 7. Illustration on the spectrum of coronary aneurysm disease

Surgical Treatment

Surgical options for CAE include aneurysm resection, ligation, or marsupialization combined with bypass grafting. However, there is no consensus on when to opt for surgery. Studies comparing surgical and percutaneous interventions found no significant differences in long-term cardiovascular outcomes. Therefore, surgery is typically reserved for patients with anatomically complex diseases where PCI poses high risks, such as those with left main involvement, multiple or large aneurysms, or SVGAs with compromised flow or structural complications.[9]

Pharmacological Therapy

Atherosclerosis is believed to play a significant role in the development of coronary artery aneurysms (CAA), particularly in older individuals.[20] As such, aggressively addressing modifiable cardiovascular risk factors in this population is essential. There remains considerable uncertainty regarding the use of dual antiplatelet therapy or anticoagulation in managing patients with CAA or coronary artery ectasia (CAE), especially when these conditions are found incidentally. Currently, no strong clinical evidence exists to definitively support or refute the routine use of intensified antithrombotic treatment in such cases.

Some retrospective analyses have reported no significant difference in adverse outcomes between patients with and without CAE, suggesting that anticoagulation might not be necessary. On the other hand, other investigations have yielded conflicting findings.

For instance, one study observed a higher five-year mortality rate in patients with CAA undergoing coronary angiography compared to those without aneurysms. Another analysis of individuals undergoing coronary CT prior to non-cardiac surgery found that those with incidental CAAs experienced a notably high rate of major adverse cardiovascular events (MACE) over a follow-up period. A more recent study indicated that patients with CAE and acute coronary syndromes faced substantially increased risks of MACE, cardiac death, and nonfatal myocardial infarction. Notably, patients who received oral anticoagulation and maintained a therapeutic range for over 60% of the time had no recorded MACE, while a third of those not on effective anticoagulation experienced such events.

In the context of Kawasaki disease, some limited data suggest that anticoagulation could help lower the risk of thrombotic events. Accordingly, current clinical guidelines recommend its use only in specific Kawasaki cases involving large or rapidly enlarging aneurysms.

There have also been suggestions that angiotensin-converting enzyme inhibitors may slow the progression of CAAs, though this hypothesis lacks confirmation from long-term studies. Importantly, vasodilators like nitrates may worsen myocardial ischemia in patients with large isolated aneurysms or ectasia and are generally advised against. In Kawasaki disease, treatment with intravenous immunoglobulin has been associated with improved rates of aneurysm regression and a lower incidence of cardiovascular complications.

However, it is important to recognize the limitations of these findings. Much of the available research is based on retrospective data, often involving small sample sizes and subject to potential bias, making definitive conclusions difficult.[16]

The appropriate use of dual antiplatelet therapy (DAPT) and anticoagulation in patients with CAAs remains a subject of ongoing discussion. Data suggest that patients with CAAs, especially those presenting with ACS, are at increased risk of thrombotic events. In such high-risk scenarios, long-term DAPT may be beneficial, particularly in patients who have undergone stenting. Some experts advocate for the use of anticoagulation in cases with reduced coronary

flow, such as in giant aneurysms or those associated with Kawasaki disease, where the risk of myocardial infarction and sudden death is significant.

Conversely, in atherosclerotic CAAs, the routine use of anticoagulation is less clear due to insufficient evidence. Nonetheless, recent reports indicate a potential benefit of anticoagulants in patients with multivessel disease or ST-elevation myocardial infarction accompanied by aneurysms. Data from the CAAR registry show that most patients are prescribed aspirin, a moderate number receive DAPT, and fewer are discharged with anticoagulation, reflecting current clinical practice.[3,8]

CONCLUSIONS

This case highlights the rare presentation and successful management of a de novo giant saccular coronary artery aneurysm (CAA) in the mid-left anterior descending artery (LAD) of a disease-naïve adult patient. Through careful multimodal imaging, clinical monitoring, and collaborative decision-making, percutaneous intervention using overlapping covered stents proved to be a viable and effective alternative when surgery was deemed too high-risk. The favorable short-term outcome following intervention underscores the importance of individualized treatment strategies, particularly in anatomically complex or high-risk cases where guidelines remain ambiguous. This report provides valuable clinical insights into the procedural approach, imaging utility, and multidisciplinary considerations essential to managing giant CAAs. Further studies are warranted to refine treatment algorithms and long-term follow-up protocols, especially as incidental detection of coronary aneurysms becomes more common with advanced imaging modalities.

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