# Coronary Artery Aneurysm and Ectasia: A Brief Review

Yi-Pan Li[1], MD, Cheng-Yu Ko[2], MD

[1]*Division of Cardiology, Department of Internal Medicine; Kaohsiung Show Chwan Memorial Hospital; Kaohsiung, Taiwan*

[2]*Division of Cardiology, Department of Internal Medicine; National Cheng Kung University Hospital; College of Medicine, National Cheng Kung University*

## Abstract

Coronary artery aneurysm (CAA) is characterized by focal coronary dilation exceeding 1.5 times the normal adjacent vessel diameter. Predominantly caused by atherosclerosis in adults and Kawasaki disease in children, CAAs are often asymptomatic but may present with thrombosis, embolization, myocardial ischemia, mass compression or rupture. Diagnostic assessment with coronary angiography is the gold standard, but consensus on standardized management strategies is lacking due to limited randomized trials. Medical therapies target cardiovascular risk reduction and aneurysm progression, and recent studies and case series have demonstrated the efficacy of oral anticoagulants with warfarin or novel oral anticoagulants. Percutaneous interventions, such as covered stents, coil embolization, and mechanical thrombectomy, have been utilized for symptomatic cases. However, these approaches carry risks, including re-stenosis, stent thrombosis, and procedural complications. Surgical options remain reserved for selected cases. Individualized treatment and further research are essential for achieving better outcomes in CAA patients.

**Keywords:** coronary artery aneurysm, atherosclerosis, percutaneous coronary intervention

## Introduction

Coronary artery aneurysm (CAA) is a challenging clinical manifestation of coronary artery disease with the reported prevalence ranging from 0.3% to 5.3% in different case series and registries.[1-3] The definition of CAA is a focal (less than 50% of vessel length) dilation with diameter exceeding 1.5 times that of the surrounding normal artery. If the dilation involves more than 50% of the vessel length, it is called coronary artery ectasia (CAE).[2,4] Patients with CAE or CAA are thought to be at higher risk of future major adverse cardiac events (MACE). Hence, devising an optimal treatment for these patients frequently presents a clinical dilemma for physicians, despite there being several approaches available, including medical management, percutaneous coronary intervention (PCI), coronary artery bypass graft (CABG) or surgical incision. The

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**Address for correspondence:** Cheng-Yu Ko, MD

Division of Cardiology, Department of Internal Medicine, National Cheng Kung University Hospital, College of Medicine, National Cheng Kung University; 138 Sheng-Li Road, Tainan 704, Taiwan

Tel: +886-6-2353535 ext. 2383; Fax: +886-6-2753834; E-mail: iznay0908@gmail.com

aim of this article is to provide a brief review of etiology, pathogenesis, classification, clinical presentations and treatment of CAA.

## Etiology and pathogenesis

CAA and CAE are more predominant in men and usually found more proximal than distal in the coronary bed. The right coronary artery is the most commonly affected vessel, accounting for 40-61% of cases, followed by the left anterior descending (LAD) artery at 15-32% and the left circumflex coronary artery at 15-23%. Involvement of the left main coronary artery is rare, occurring in only 0.1-3.5% of cases, and is typically associated with extensive two- or three-vessel coronary artery disease.[5] Although the pathophysiological mechanism is not well understood, the most common etiology is atherosclerosis in adults and Kawasaki disease in children and adolescents.[6]

Several risk factors have been identified in association with the development of CAA and CAE, including atherosclerosis, connective tissue disease, inflammatory disorder or vasculitis. Atherosclerosis, the most common and strongly associated factor, plays a significant role in CAA formation. Genetic predisposition has also been implicated, suggesting heritable susceptibility to aneurysm development. Kawasaki disease is the most common cause of CAAs in childhood, particularly in Japan, with spontaneous resolution occurring in approximately 50% of cases. Connective tissue disorders, such as Ehlers-Danlos syndrome and Marfan syndrome, are also associated with CAA formation. Additionally, various types of vasculitis linked to inflammatory disorders, including systemic lupus erythematosus, Takayasu arteritis, rheumatoid arthritis, Wegener granulomatosis, giant cell arteritis, Churg-Strauss syndrome, and microscopic polyangiitis, have been identified as contributing factors.[4]

Histopathological analysis reveals extensive coronary artery wall destruction accompanied by diffuse vasculitis, indicating a potential role of matrix metalloproteinases (MMPs) in CAA formation. MMPs are enzymes capable of degrading connective tissue proteins, resulting in the weakening of the vascular wall.[7] Iatrogenic factors, such as intracoronary interventions (e.g., angioplasty or stenting), can cause local arterial wall injury, leading to CAA formation. Furthermore, infectious etiologies, including mycotic CAAs and post-infection complications, have been linked to direct wall invasion or immune complex deposition. Atherosclerotic and vasculitis-related CAAs tend to involve multiple coronary arteries, whereas congenital and iatrogenic CAAs are typically confined to a single artery. These distinctions emphasize the diverse etiology and clinical presentation of CAAs, necessitating tailored diagnostic and therapeutic approaches.

## Clinical manifestations

Most patients with CAAs are asymptomatic and detected incidentally during computed tomography, coronary imaging or autopsy.[8] The stagnation of blood flow in CAAs promotes the formation of thrombi. The complications of CAA include local thrombosis, distal embolization and coronary spasm leading to clinical presentations of angina, dyspnea, myocardial ischemia or even sudden cardiac death. Aneurysm rupture with subsequent hemopericardium and cardiac tamponade has been reported.[9,10] Compression by giant CAAs of adjacent structures such as the right atrium, right ventricle and tricuspid valve have also been observed.[11,12] CAAs have been reported to be associated with abdominal aortic aneurysm or hypertension. Given the variety of clinical presentations, utilizing different diagnostic approaches is important.

## Classification

Aneurysmal coronary artery dilation can be either focal or diffuse, depending on whether it affects more or less than 50% of the vessel. Focal dilation, or aneurysm, can be classified

based on wall composition. A true aneurysm has a wall composed of all three vascular layers, whereas a pseudoaneurysm consists of only the adventitia (Figure 1).[5] Pseudoaneurysms are most often found in patients with previous coronary intervention, with a reported incidence of 0.3-6.0%.[13] CAAs are further classified as fusiform (transverse dimension < longitudinal dimension) or saccular (transverse dimension > longitudinal dimension) (Figures 2 and 3). Saccular aneurysms are more common in the LAD.[2,14] If the diameter of the aneurysmal artery is more than 8 mm or 4 times larger than the reference vessel it is characterized as a giant CAA.6 Diffuse dilation exceeding 50% of the vessel length is defined as coronary artery ectasia (CAE) (Figure 4). It can be categorized into four types based on its distribution. Type I involves diffuse ectasia in two or three vessels, while Type II presents as diffuse ectasia in one vessel accompanied by an aneurysm in another. Type III is characterized by diffuse ectasia in a single vessel, and Type IV refers to localized and segmental ectasia.

## Diagnosis

Non-invasive methods for diagnosing CAA include transthoracic and transesophageal echocardiography, computed tomography (CT), and magnetic resonance angiography. Radiation-free tools are recommended for follow-up in children with Kawasaki disease. Effective

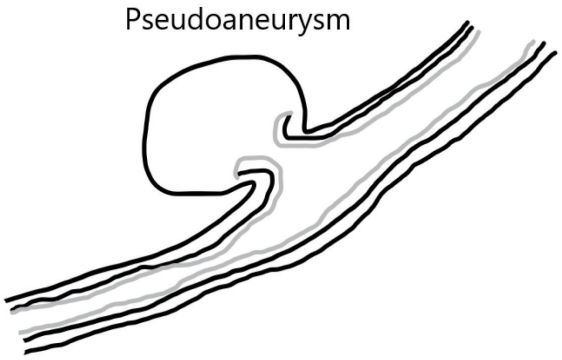


**Figure 1.** Pseudoaneurysm is characterized by loss of vessel wall integrity and is only contained by adventitia.

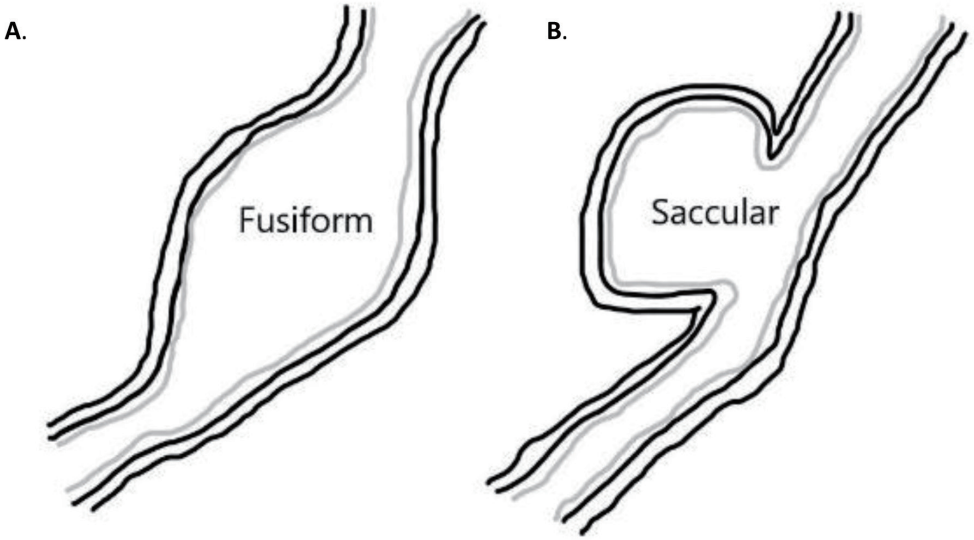


**Figure 2. (A)** In a fusiform coronary aneurysm the transverse dimension is less than the longitudinal dimension. **(B)** In a saccular coronary aneurysm the transverse dimension is greater than the longitudinal dimension.

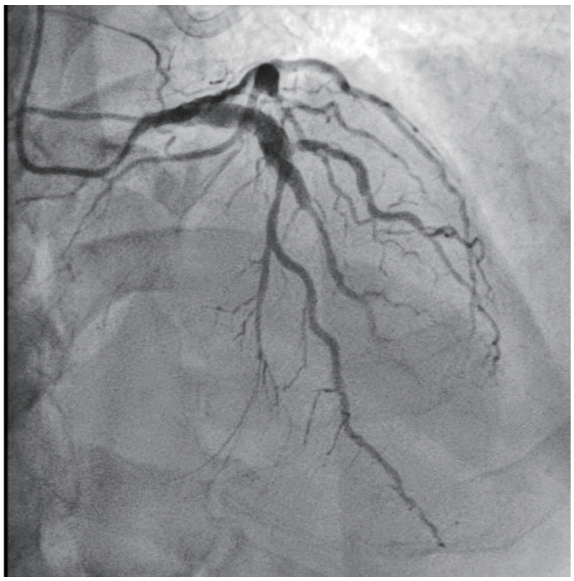

**Figure 3.** A male patient presenting with inferior STEMI was found to have coronary aneurysm and ectasia at the left coronary artery. Fusiform aneurysm was noted at the proximal left anterior descending artery.

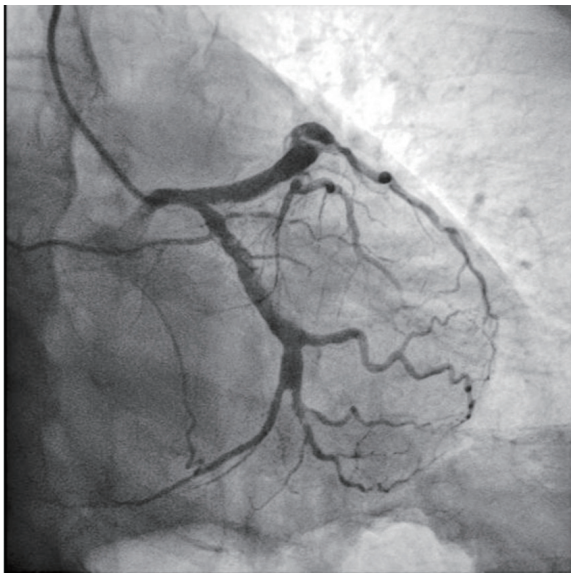

**Figure 4.** AThe coronary angiography in the patient showed ectasia of the left circumflex artery.

evaluation of CAAs requires imaging to determine their distribution, size, thrombus presence, and associated complications, such as myocardial infarction (MI).

As most patients are asymptomatic, CAAs are often incidentally detected during coronary angiography. Coronary angiography is the gold standard for invasive evaluation of the anatomic characteristics of CAA, while coronary CT angiography serves as a non-invasive alternative for follow-up assessments.6 Coronary angiography can reveal turbulent flow in aneurysmal coronary arteries, indicated by delayed dye filling, segmental backflow, and local dye stagnation.[5]

Intravascular ultrasound (IVUS) can provide anatomical information on the arterial wall structure and luminal composition, which is crucial for distinguishing different types of aneurysms. Pseudoaneurysms lack integrity, leading to a rupture-prone adventitia. IVUS can accurately measure the lumen diameter and identify any surrounding stenotic segment, allowing proper stent sizing. IVUS is invaluable during CAA treatment, providing essential guidance for adequate aneurysm coverage.[2,3]

## Treatment

Standard management of CAA remains controversial due to the lack of randomized trials. The treatment for CAA is basically tailored individually, depending on the clinical presentations (ranging from asymptomatic through effort angina to acute coronary syndrome), patient characteristics (age, comorbidities) and physician experience. The approaches include medical treatment, PCI and surgery. The optimal therapeutic strategy for achieving the best outcomes remains undecided, as no comparative studies have been conducted.

### Medical therapy

The management of cardiovascular (CV) risk factors in patients with CAA, irrespective of the presence of concurrent coronary artery disease, is advocated.[15] Previous studies have suggested

a correlation between the activity of angiotensin-converting enzyme (ACE) and aneurysm formation. It has been hypothesized that the use of ACE inhibitors or angiotensin II receptor antagonists may slow down the progression of CAA.[16] Statins have also been found to inhibit the expression of matrix metalloproteinases, enzymes that play a role in the formation of aneurysms, which may help reduce the risk of CAA progression.[17]

The choice of antithrombotic therapy in CAA also remains controversial due to the lack of randomized controlled trials, but there are some promising observational studies showing benefits from oral anticoagulant therapy in treating patients with CAA/CAE. A study of 1,698 patients with acute MI suggested a potential benefit of anticoagulation therapy in patients with CAE and acute coronary syndrome (ACS). Among CAE patients treated with oral anticoagulation who maintained a time-in-therapeutic range (TTR) > 60%, the incidence of MACE was 0%, compared to 33% in those not receiving therapeutic anticoagulation ($p$ = 0.03) after 49 months of follow-up.[18] A propensity-matched study including 1331 patients recorded in the Coronary Artery Aneurysm Registry (CAAR) demonstrated that oral anticoagulation therapy with warfarin significantly reduces the composite endpoint of unstable angina, myocardial infarction, and aneurysm thrombosis in patients with CAA (8.7 vs. 17.2%, respectively; $p$ = .01), with a notable reduction in unstable angina (4.6% vs. 10%, $p$ < .01) and aneurysm thrombosis (0% vs. 3.1%, $p$ = .03).[19] Some case reports have described the use of oral anticoagulants (OACs) in patients with ACS and CAA. These include combinations such as dual antiplatelet therapy (DAPT) with non-vitamin K antagonist oral anticoagulants (NOACs),[20] single antiplatelet therapy (SAPT) with NOACs,[21,22] and SAPT or DAPT with warfarin, later switching to NOACs.[23,24] In some cases, NOACs alone have been used to treat thrombus associated with CAA.[25] These findings emphasize the need for personalized treatment plans and further research to determine the best antithrombotic strategies for treating CAA. Figure 5 shows a case with coronary ectasia complicated by inferior ST-elevation myocardial infarction (STEMI), who was treated by PCI followed by anticoagulation and antiplatelets.

### Percutaneous coronary intervention

Data on asymptomatic patients with CAAs remain limited, with most available studies focusing on PCI outcomes in the context of acute MI.[26] In this setting, PCI in aneurysmal culprit vessels is associated with lower success rates and increased risks of no-reflow and distal embolization.[27] Furthermore, patients who survive STEMI after undergoing PCI for CAAs face higher medium-term mortality rates, as well as elevated risks of definite stent thrombosis, target vessel revascularization, and non-fatal MI.[28]

### Covered stents for CAAs

Covered stents are primarily used for treating saccular CAAs not involving side branches. Currently, no covered stents are specifically designed for CAAs. The GRAFTMASTER coronary stent graft (Abbott Vascular, Santa Clara, California) is the most commonly used, with sizes ranging from 2.75-5 mm. Its structure incorporates a sandwich technique, placing an ultra-thin layer of expandable polytetrafluoroethylene between two GRAFTMASTER stents, pre-mounted on a balloon catheter delivery system. However, this stent has a larger profile, requiring a 6-7 French guide catheter, and poor deliverability, especially in tortuous vessels. The PK Papyrus covered stent (Biotronik, Berlin, Germany) offers better bending flexibility and a smaller crossing profile due to its advanced single-stent design. It comes in sizes ranging from 2.5-5.0 mm and is deliverable with a 5-6 French guide catheter.[2] Challenges in using covered stents include poor deliverability, the need for large sheaths, increased vascular and procedural complications, difficulties in tortuous or calcified vessels, and potential side branch loss. It is recommended not to use covered stents for

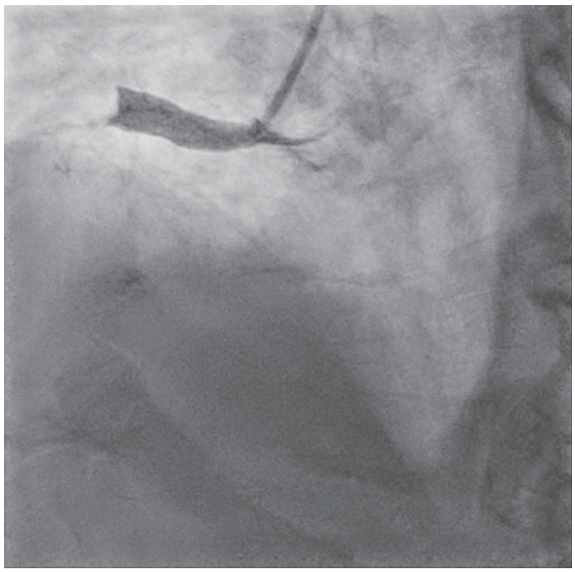

**Figure 5A.** A 56-year-old male smoker with type 2 diabetes mellitus, hypertension and dyslipidemia presented with inferior STEMI. The angiography revealed large thrombus burden causing occlusion of the proximal right coronary artery. The vessel was abnormally dilated with the presentation of ectasia.

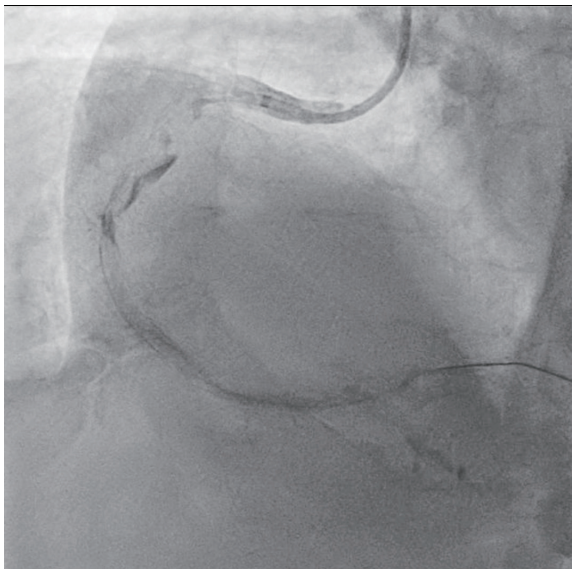

**Figure 5B.** TIMI 2-3 flow with large thrombus burden was noted after balloon angioplasty and thrombus aspiration.

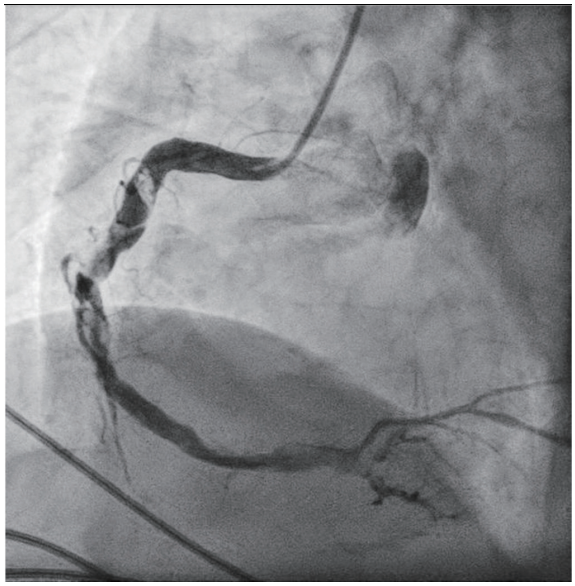

**Figure 5C.** Two days after dual antiplatelets, heparin and the support of intra-aortic balloon pump, the flow became TIMI 3 with residual thrombi.

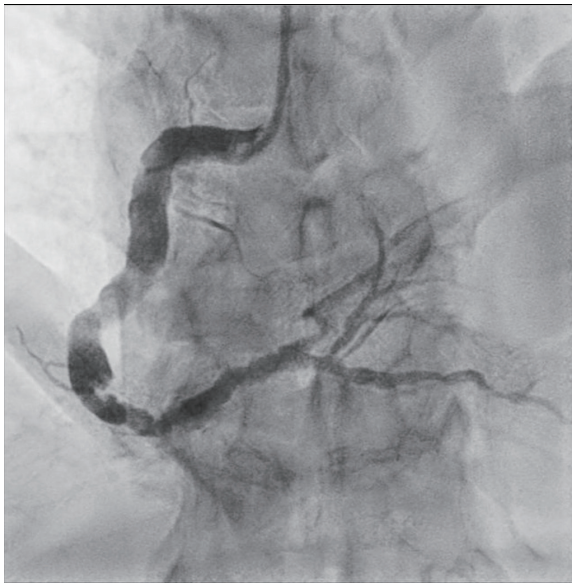

**Figure 5D.** Two months later with dual anti-thrombotic therapy (Clopidogrel plus NOAC), the thrombus burden decreased substantially.

CAAs with major side branches connecting to the aneurysm. In the event of major side branch loss after deployment of covered stents for aneurysm, techniques such as the use of a stiff wire under the support of a microcatheter (MC) to puncture the side branch orifice may be employed as a bailout strategy. The potential drawbacks of covered stents include a higher mid-term or long-term risk of in-stent restenosis and stent thrombosis.[2,29,30]

### Mechanical thrombectomy

In CAA patients presenting with acute MI, PCI for the culprit artery is related to higher failure rate due to high thrombus burden.[28] The AngioJet device (Boston Scientific, Marlborough, Massachusetts) has shown efficacy in patients undergoing PCI for venous grafts[31] and native coronary vessel[32] with massive thrombus, in randomized trials. There are case reports demonstrating its effectiveness in treating CAA.[33,34]

### Coil embolization for CAAs

Coil embolization can be considered when covered stent placement is not feasible, such as in tortuous or calcified vessels or when there is a high risk of major side branch loss. The procedure involves first advancing a MC and a workhorse wire into the large aneurysm. A stent is deployed to cover the aneurysm neck, jailing the MC in place. Coils are then passed via the MC to wrap around the stent, followed by in-stent post-dilation after the MC is removed. Caution is needed to avoid complications such as vascular rupture during coil embolization or coil herniation into the stent strut, which can lead to stent thrombosis.[2,35,36]

### Surgery

The optimal surgical approach for CAAs remains uncertain due to the limited number of studies and a lack of robust outcome data. Common surgical techniques include aneurysm ligation, resection, marsupialization with interposition grafts, or opening the aneurysm, suturing its afferent and efferent vessels, and performing bypass grafting when necessary.[37] While success rates are not well-documented due to the rarity of these procedures and potential reporting bias, surgical intervention is often preferred for specific cases. Surgical intervention, such as CABG, is the preferred approach for large CAAs located near bifurcations. Additionally, surgical resection is considered the first-line treatment for CAAs involving the left main coronary artery, multiple or giant aneurysms, and saphenous vein graft aneurysms.[2]

## Conclusion

Coronary artery aneurysm and coronary artery ectasia are challenging manifestations with diverse etiologies, including atherosclerosis, Kawasaki disease, connective tissue disorders, and iatrogenic factors. Patients are often asymptomatic and incidentally diagnosed. There is growing evidence showing these patients have higher incidence of MACE that can lead to severe complications such as thrombosis, embolization, myocardial infarction, and sudden cardiac death. Diagnosis relies on imaging modalities like coronary angiography, IVUS and CT, which are essential for assessing CAA/CAE characteristics and complications. There are currently no guidelines for recommendation and an individualized approach is critical, based on patients' clinical risk factors, presentation and coronary anatomy. Treatment options include medical management, percutaneous interventions, and surgical options. Further large multicenter studies are warranted to understand the etiology and optimal medical or interventional treatment for coronary artery aneurism disease.

## Reference

1. Swaye PS, Fisher LD, Litwin P, et al. Aneurysmal coronary artery disease. *Circulation* 1983;67(1):134-138. doi: 10.1161/01.cir.67.1.134 [published Online First: 1983/01/01]

2. Kawsara A, Núñez Gil IJ, Alqahtani F, et al. Management of Coronary Artery Aneurysms. *JACC Cardiovascular interventions* 2018;11(13):1211-1223. doi: 10.1016/j.jcin.2018.02.041 [published Online First: 2018/07/07]
3. Abou Sherif S, Ozden Tok O, Taşköylü Ö, et al. Coronary Artery Aneurysms: A Review of the Epidemiology, Pathophysiology, Diagnosis, and Treatment. *Frontiers in Cardiovascular Medicine* 2017;4 doi: 10.3389/fcvm.2017.00024
4. Díaz-Zamudio M, Bacilio-Pérez U, Herrera-Zarza MC, et al. Coronary artery aneurysms and ectasia: role of coronary CT angiography. *Radiographics: a review publication of the Radiological Society of North America, Inc* 2009;29(7):1939-1954. doi: 10.1148/rg.297095048 [published Online First: 2009/11/21]
5. ElGuindy MS, ElGuindy AM. Aneurysmal coronary artery disease: An overview. *Global cardiology science & practice* 2017;2017(3):e201726. doi: 10.21542/gcsp.2017.26 [published Online First: 2018/03/23]
6. Matta AG, Yaacoub N, Nader V, et al. Coronary artery aneurysm: A review. *World journal of cardiology* 2021;13(9):446-455. doi: 10.4330/wjc.v13.i9.446 [published Online First: 2021/10/09]
7. Amano S, Hazama F, Hamashima Y. Pathology of Kawasaki disease: I. Pathology and morphogenesis of the vascular changes. *Japanese circulation journal* 1979;43(7):633-643. doi: 10.1253/jcj.43.633 [published Online First: 1979/07/01]
8. Syed M, Lesch M. Coronary artery aneurysm: a review. *Progress in cardiovascular diseases* 1997;40(1):77-84. doi: 10.1016/s0033-0620(97)80024-2 [published Online First: 1997/07/01]
9. Wan S, LeClerc JL, Vachiery JL, et al. Cardiac tamponade due to spontaneous rupture of right coronary artery aneurysm. *The Annals of thoracic surgery* 1996;62(2):575-576. [published Online First: 1996/08/01]
10. Channon KM, Banning AP, Davies CH, et al. Coronary artery aneurysm rupture mimicking dissection of the thoracic aorta. *International journal of cardiology* 1998;65(1):115-117. doi: 10.1016/s0167-5273(98)00086-2 [published Online First: 1998/08/12]
11. Lachmann M, Will A, Linhard M, et al. Progression of a coronary artery aneurysm with symptomatic compression of cardiac structures. *European heart journa*l 2018;39(35):3336-36. doi: 10.1093/eurheartj/ehy350
12. Khouzam MS, Khouzam N. Giant coronary artery aneurysms involving more than one coronary artery: case report. *Journal of cardiothoracic surgery* 2021;16(1):177. doi: 10.1186/s13019-021-01560-5 [published Online First: 2021/06/21]
13. Sheikh AS, Hailan A, Kinnaird T, et al. Coronary Artery Aneurysm: Evaluation, Prognosis, and Proposed Treatment Strategies. *Heart views : the official journal of the Gulf Heart Association* 2019;20(3):101-108. doi: 10.4103/heartviews.Heartviews_1_19 [published Online First: 2019/10/18]
14. Harikrishnan S, Sunder KR, Tharakan JM, et al. Saccular coronary aneurysms: angiographic and clinical profile and follow-up of 22 cases. *Indian heart journal* 2000;52(2):178-182. [published Online First: 2000/07/14]
15. Baman TS, Cole JH, Devireddy CM, et al. Risk factors and outcomes in patients with coronary artery aneurysms. *The American journal of cardiology* 2004;93(12):1549-1551. doi: 10.1016/j.amjcard.2004.03.011 [published Online First: 2004/06/15]
16. Gülec S, Aras O, Atmaca Y, et al. Deletion polymorphism of the angiotensin I converting enzyme gene is a potent risk factor for coronary artery ectasia. *Heart* 2003;89(2):213-214. doi: 10.1136/heart.89.2.213 [published Online First: 2003/01/16]
17. Tengiz I, Ercan E, Aliyev E, et al. Elevated levels of matrix metalloprotein-3 in patients with coronary aneurysm: A case control study. *Current controlled trials in cardiovascular medicine* 2004;5(1):10. doi: 10.1186/1468-6708-5-10 [published Online First: 2004/10/16]
18. Doi T, Kataoka Y, Noguchi T, et al. Coronary Artery Ectasia Predicts Future Cardiac Events in Patients With Acute Myocardial Infarction. *Arteriosclerosis, thrombosis, and vascular biology* 2017;37(12):2350-2355. doi: 10.1161/atvbaha.117.309683 [published Online First: 2017/10/21]
19. D'Ascenzo F, Saglietto A, Ramakrishna H, et al. Usefulness of oral anticoagulation in patients with coronary aneurysms: Insights from the CAAR registry. *Catheterization and cardiovascular interventions: official journal of the Society for Cardiac Angiography & Interventions* 2021;98(5):864-871. doi: 10.1002/ccd.29243 [published Online First: 2020/09/10]
20. Korosoglou G, Eisele T, Nunninger P, et al. Mechanical and pharmacologic treatment in a patient with a coronary artery aneurysm and acute coronary syndrome. *Coronary artery disease* 2017;28(4):358-360. doi: 10.1097/mca.0000000000000475 [published Online First: 2017/02/23]
21. Yan Q, Ning L, Jian Y, et al. Could the Novel Oral Anticoagulants Be Used for Coronary Artery Aneurysm? *Case reports in medicine*

2020;2020:5073814. doi: 10.1155/2020/5073814 [published Online First: 2020/05/07]
22. Choi HJS, Luong C, Fung A, et al. ST-Elevation Myocardial Infarction in Coronary Ectasia: A Case Report. *Diseases (Basel, Switzerland)* 2018;6(4) doi: 10.3390/diseases6040104 [published Online First: 2018/11/21]
23. Waqas M, Bizzocchi LL, Menegus MA, et al. Coronary Artery Ectasia: An Insight into Intraprocedural and Postprocedural Management Strategies. *Cureus* 2019;11(1):e3928. doi: 10.7759/cureus.3928 [published Online First: 2019/04/02]
24. Tomioka T, Takeuchi S, Ito Y, et al. Recurrent Acute Myocardial Infarction in a Patient with Severe Coronary Artery Ectasia: Implication of Antithrombotic Therapy. *The American journal of case reports* 2016;17:939-943. doi: 10.12659/ajcr.900474 [published Online First: 2016/12/13]
25. Yuksel M, Yildiz A, Tapan U, et al. Resolution of Extensive Coronary Thrombosis under Rivaroxaban Treatment. *Arquivos brasileiros de cardiologia* 2015;105(6):642-646. doi: 10.5935/abc.20150052 [published Online First: 2016/01/14]
26. Iannopollo G, Ferlini M, Koziński M, et al. Patient Outcomes With STEMI Caused by Aneurysmal Coronary Artery Disease and Treated With Primary PCI. *Journal of the American College of Cardiology* 2017;69(24):3006-3007. doi: 10.1016/j.jacc.2017.04.030 [published Online First: 2017/06/18]
27. Joo HJ, Woong Yu C, Choi R, et al. Clinical outcomes of patients with coronary artery aneurysm after the first generation drug-eluting stent implantation. *Catheterization and cardiovascular interventions: official journal of the Society for Cardiac Angiography & Interventions* 2018;92(3):E235-e245. doi: 10.1002/ccd.27429 [published Online First: 2017/11/23]
28. Bogana Shanmugam V, Psaltis PJ, D TLW, et al. Outcomes After Primary Percutaneous Coronary Intervention for ST-Elevation Myocardial Infarction Caused by Ectatic Infarct Related Arteries. *Heart, lung & circulation* 2017;26(10):1059-1068. doi: 10.1016/j.hlc.2016.12.006 [published Online First: 2017/02/22]
29. Cereda AF, Tiberti G, Pera IG, et al. A Giant Coronary Artery Aneurysm Treated Using Multiple Overlapping Covered Stents. *JACC Cardiovascular interventions* 2017;10(13):e127-e128. doi: 10.1016/j.jcin.2017.04.030 [published Online First: 2017/06/19]
30. Szalat A, Durst R, Cohen A, et al. Use of polytetrafluoroethylene-covered stent for treatment of coronary artery aneurysm. *Catheterization and cardiovascular interventions: official journal of the Society for Cardiac Angiography & Interventions* 2005;66(2):203-208. doi: 10.1002/ccd.20448 [published Online First: 2005/06/25]
31. Kuntz RE, Baim DS, Cohen DJ, et al. A trial comparing rheolytic thrombectomy with intracoronary urokinase for coronary and vein graft thrombus (the Vein Graft AngioJet Study [VeGAS 2]). *The American journal of cardiology* 2002;89(3):326-330. doi: 10.1016/s0002-9149(01)02235-4 [published Online First: 2002/01/26]
32. Antoniucci D, Valenti R, Migliorini A, et al. Comparison of rheolytic thrombectomy before direct infarct artery stenting versus direct stenting alone in patients undergoing percutaneous coronary intervention for acute myocardial infarction. *The American journal of cardiology* 2004;93(8):1033-1035. doi: 10.1016/j.amjcard.2004.01.011 [published Online First: 2004/04/15]
33. Giombolini C, Notaristefano S, Santucci S, et al. AngioJet thrombectomy for the treatment of coronary artery aneurysm after failed thrombolysis in acute myocardial infarction. *Heart international* 2006;2(2):94. doi: 10.4081/hi.2006.94 [published Online First: 2006/01/01]
34. Lee MS, Nero T, Makkar RR, et al. Treatment of coronary aneurysm in acute myocardial infarction with AngioJet thrombectomy and JoStent coronary stent graft. *The Journal of invasive cardiology* 2004;16(5):294-296. [published Online First: 2004/05/21]
35. Win HK, Polsani V, Chang SM, et al. Stent-assisted coil embolization of a large fusiform aneurysm of proximal anterior descending artery: novel treatment for coronary aneurysms. *Circulation Cardiovascular interventions* 2012;5(1):e3-5. doi: 10.1161/circinterventions.111.966754 [published Online First: 2012/02/18]
36. Piao ZH, Jeong MH, Jeong HC, et al. Coronary artery fistula with giant aneurysm and coronary stenosis treated by transcatheter embolization and stent. *Korean circulation journal* 2015;45(3):245-247. doi: 10.4070/kcj.2015.45.3.245 [published Online First: 2015/05/30]
37. Singh SK, Goyal T, Sethi R, et al. Surgical treatment for coronary artery aneurysm: a single-centre experience. *Interactive cardiovascular and thoracic surgery* 2013;17(4):632-636. doi: 10.1093/icvts/ivt282 [published Online First: 2013/06/28]