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Review

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Diagnostic workup and treatment options for aneurysmal coronary artery disease

Alberto Barioli, Emanuele Visco, Nicola Pellizzari, Filippo Marzot, Davide Lanzellotti, Luca Favero, Carlo Cernetti

Cardio-Neuro-Vascular Department, Cà Foncello and San Giacomo Hospital, Treviso 31100, Italy.

Correspondence to: Dr. Alberto Barioli, Cardio-Neuro-Vascular Department, Cà Foncello and San Giacomo Hospital, Azienda No. 2 Marca Trevigiana, Treviso 31100, Italy. E-mail: albertobarioli@gmail.com

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Abstract

Coronary artery aneurysms and coronary ectasia are defined as focal or diffuse dilation of the coronary arteries, respectively. Although frequently silent and detected as incidental findings at coronary angiography or computed tomography, coronary aneurysms have been associated with different clinical conditions, including silent ischemia and acute coronary syndromes, and with poor clinical outcomes. The optimal management still remains unsettled, as randomized data are lacking and treatment with either surgical or percutaneous procedures faces significant challenges. This review aims to provide an update on the classification, etiopathogenesis, diagnostic workup, and treatment of aneurysmal coronary disease.

Keywords: Coronary artery aneurysm, coronary artery ectasia, coronary artery disease, percutaneous coronary intervention

INTRODUCTION

Coronary artery aneurysm (CAA) and ectasia (CAE), usually collectively referred to as aneurysmal coronary artery disease, are defined as focal or diffuse coronary dilations that exceed 1.5 times the diameter of the adjacent normal segment. Although frequently used interchangeably, the two terms reflect a different extent of the disease: the term aneurysm should be used to describe those dilations involving less than 50% of the

vessel length, while the term ectasia refers to more diffuse dilations[1]. Aneurysms are defined as giant if their diameter is > 8 mm or when it is > 4 times the reference vessel diameter[2].

The incidence of the disease ranges between 0.3% and 5%[3]. This wide range reflects the different reporting methods, where the highest incidences are found when CAA and CAE are considered together. In recent studies, the incidence of true CAA appears to be less than 1%[4-6].

All three coronary arteries may be affected by the disease, but in most cases, only one major coronary artery is involved. The right coronary artery is the most frequently affected (40%), followed by the left anterior descending (32%) and the circumflex artery (23%). Aneurysms of the left main coronary artery have rarely been reported[1]. The disease mainly involves the proximal segments of the coronary tree and is often associated with coronary artery disease (CAD). Men appear to have higher incidence rates of CAAs than women (2.2% *vs.* 0.5%, respectively)[7].

Classifications

Aneurysmal coronary artery disease can be classified in different ways. The etiopathogenetic classification identifies atherosclerotic, inflammatory, and non-inflammatory mechanisms leading to CAA formation[8]. From an anatomical perspective, aneurysms can be classified according to the integrity of the vascular wall. We can therefore distinguish true aneurysms when the three layers (intima, media, and adventitia) of the vessel wall are preserved, and false aneurysms or pseudoaneurysms, which are characterized by loss of one or two layers. According to morphology, aneurysms can also be classified into saccular, when the transverse diameter exceeds the longitudinal diameter, and fusiform in the opposite case. The former most frequently follow proximal stenosis, are often multifocal, and are more likely to present with thrombosis or rupture. In contrast, fusiform aneurysms tend to be bilateral, are associated with aneurysms of the abdominal aorta and cerebral circulation and less frequently with obstructive CAD[9,10]. The classification proposed by Markis *et al.* distinguishes four types of coronary aneurysms based on the coronary distribution of dilations: Type 1, when ectasia is present in two or three vessels; Type 2, when ectasia involves only one vessel and is associated with localized disease in a second vessel; Type 3, when dilation affects only one vessel; and Type 4, when the ectasia is localized or segmental[11]. Classifications of aneurysmal dilatations and ectasia are shown in Figure 1.

Pathogenesis, etiology and natural history of the disease

The etiopathogenesis of coronary aneurysms is poorly understood and remains a matter of debate. A strong association between aneurysmal disease and CAD has been observed in adults, suggesting a possible common underlying etiology[10,12]. In addition, a partly overlapping genetic predisposition to both aneurysmal disease and atherosclerosis has been observed, as they share an altered proliferative phenotype that promotes adverse vascular remodeling associated with variants on chromosome 9p21.3[9].

Kawasaki disease (KD), a rare inflammatory condition that can result in vasculitis of the coronary arteries, is the leading cause of CAAs in childhood[9,13]. Ten to fifteen percent of KD patients develop CAAs during the acute phase of the disease. Inflammatory response mediated by cytokines such as TNF-alpha leads to increased levels of matrix metalloproteinase (MMP) and decreased levels of their tissue-specific inhibitors (TIMP) and thus drives the degradation of the vessel wall-originating CAAs. CAAs have also been associated with other inflammatory diseases (e.g., Takayasu arteritis), collagenosis (e.g., scleroderma heart disease, polyarteritis nodosa, systemic lupus erythematosus), and connective tissue disorders (e.g., Marfan syndrome, Ehlers-Danlos disease).

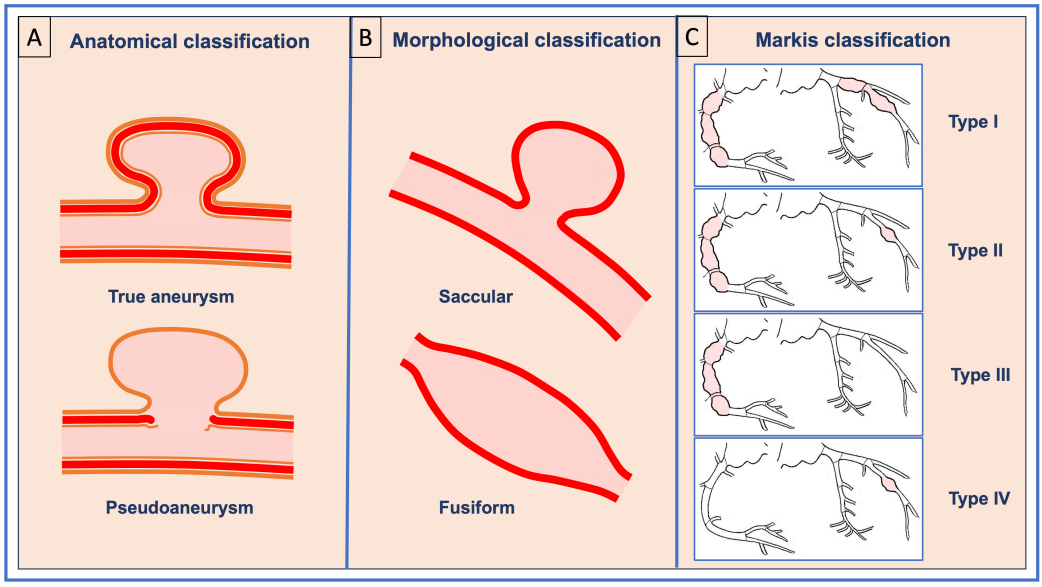

Figure 1. Classifications of aneurysmal dilatations and ectasia of coronary arteries according to (A) Anatomy (B) Morphology and (C) Number of the involved vessels.

Coronary aneurysms can be iatrogenic and due to coronary artery manipulation by percutaneous interventions. In particular, drug-eluting stent implantation has been associated with aneurysm formation. As a matter of fact, the polymer carrying the drug may determine a local hypersensitivity reaction on the vessel wall, and the antiproliferative drug eluted from the stent appears to delay neointimal healing and reendothelialization, weakening the arterial wall and ultimately leading to the development of an aneurysm[9].

Other causes of CAAs are congenital, secondary to infections (e.g., bacterial, fungal), or those resulting from drug abuse (cocaine).

A particular, although rare, type of aneurysm is an aortocoronary saphenous vein graft aneurysm, the pathogenesis of which is not yet well understood. They are generally encountered very late after bypass grafting, tend to be large in size, and not infrequently present with complications such as rupture, fistula formation, and compression of adjacent structures[14].

The natural history of coronary aneurysms has yet to be fully elucidated. Most reports in the literature include a limited number of patients with short-term follow-up, and prognosis assessment is often influenced by medical, surgical, or percutaneous treatment. Data from the Coronary Artery Surgery Study (CASS) showed that the presence of aneurysmal disease does not confer additional risk in terms of survival in patients with CAD[15]. Two other retrospective studies reported similar results, concluding that prognosis in patients with coronary ectasia seems to depend on the association with obstructive CAD[16,17]. Conversely, Baman *et al.* found an increased long-term mortality in patients with CAA/CAE, regardless of the presence of CAD[18].

Clinical presentation

The presence of CAAs and/or CAE has been associated with various clinical presentations. Patients may be asymptomatic or present with the extracardiac symptoms of connective tissue disease or associated

vasculitis. However, the most frequent clinical manifestation is ischemic heart disease, including exertional angina, acute coronary syndromes, and sudden death. These clinical manifestations may be determined by the presence of concomitant obstructive CAD. Nevertheless, exertional angina and positive exercise stress tests have been observed even in the absence of significant atherosclerosis and have been associated with flow disturbances within the aneurysm, formation of endoluminal thrombi with subsequent embolization, and microvascular dysfunction[19]. Less frequent symptoms are those resulting from compression of contiguous cardiac or extracardiac structures or from aneurysm rupture (into a low-pressure cavity or pericardium)[14,20,21]. However, spontaneous rupture is a rare occurrence[22].

Assessment

Coronary angiography is the method of choice used for the diagnosis and characterization of aneurysms. Indeed, it can assess the size, location, extent, and morphology of CAAs and CAE, as well as associated obstructive coronary artery disease, if present [Figure 2A]. Proper visualization of aneurysms with angiography is often hampered by the delayed progression of the injected contrast medium, the presence of segmental backflow, and contrast stasis in the dilated segment[3]. Intravascular ultrasonography (IVUS) has proven to be a valuable adjunctive tool for determining aneurysm size, vessel wall characteristics, presence of stenosis or thrombotic apposition[23]. In addition, it facilitates stent sizing if percutaneous coronary intervention (PCI) is planned and allows intra-procedural stent optimization. Optical coherence tomography has a limited role in assessing CAAs, but in small aneurysms, it can provide important information such as the differentiation between true and pseudoaneurysms and the state of the coronary arteries in adjacent segments[24].

Coronary computed tomography (CT) is a noninvasive diagnostic tool that allows accurate assessment of aneurysm size, wall characteristics, anatomical relationships, and degree of thrombosis or calcifications[25-27] [Figure 2B]. Beyond the initial diagnostic characterization, coronary CT is useful for monitoring the progression of aneurysms over time; however, its use in patient follow-up raises questions about the possible risks associated with ionizing radiation.

MANAGEMENT

Appropriate management for CAA and CAE is still largely unsettled, and should be individualized according to clinical presentation, aneurysm size and characteristics, disease progression over time, and association with atherosclerotic disease[9]. Treatment modalities include medical therapy and revascularization with either PCI or surgery [Table 1]. A recent retrospective cohort study showed a relatively high MACCE rate across all treatment groups, with a non-statistically significant trend favoring surgery[28].

Medical management

Medical management of CAA, especially in the absence of concomitant atherosclerotic coronary stenosis or thrombotic complications, is still being debated. The management of CAA in the setting of Kawasaki disease falls beyond the purposes of the present paper; however, intravenous administration of immunoglobulins at the early stages of the disease has been proven to positively impact prognosis[29,30]. In adult subjects with CAA, as for non-CAA patients, search for coronary artery disease risk factors should be pursued to modify or remove them, wherever possible.

Some Authors reported on the possible interplay of the renin-angiotensin-aldosterone system (RAAS) and inflammation on aneurysm development, advocating both RAAS modulation and statin therapy as potential strategies to prevent disease progression[31-34]. However, no formal recommendations can be provided, given the lack of prospective and high-quality evidence.

Table 1. Therapeutic strategies for CAAs and CAEs

Treatment modality		Rationale	Advantages	Disadvantages
Medical therapy	ACEi/ARBs/Statins	Control CVRF/Slow CAA dilation rate?	Easy access Wide availability	Neither removal nor exclusion of CAA Risk of bleeding with long-term antithrombotic therapies
	DAPT *vs.* SAPT *vs.* OAC	Reduce thrombotic complications		
	IVIG therapy	Kawasaki disease-related CAA		
PCI	Covered Stent	Exclusion of smaller CAA Prevention of CAA rupture Coronary flow restoration in case of ongoing ischemia	Lower risk or procedural complications/quick coronary flow restoration	No removal of CAA/risk of CAA rupture/endoleaks/stent malapposition/vessel occlusion
	Double stent method			
	Self-expanding stents			
	Coil embolization ± stent-assisted			
Cardiac surgery	Resection/ligation/ marsupialization	Treatment of larger/giant CAA at high risk of rupture or involving LMCA/proximal vessel	Complete removal of CAA Treatment of CAA/CAD unsuitable for PCI	Higher risk of procedural complications
	CABG	Treatment of concomitant obstructive CAD		

ACEi: Angiotensin converting enzyme inhibitors; ARBs: angiotensin II receptor blockers; DAPT: dual antiplatelet therapy; SAPT: single antiplatelet therapy; OAC: oral anticoagulant; CAA: coronary artery aneurysm; CAE: coronary artery ectasia; CVRF: cardiovascular risk factors; PCI: percutaneous coronary intervention; CABG: coronary artery bypass graft; LMCA: left main coronary artery; CAD: coronary artery disease.

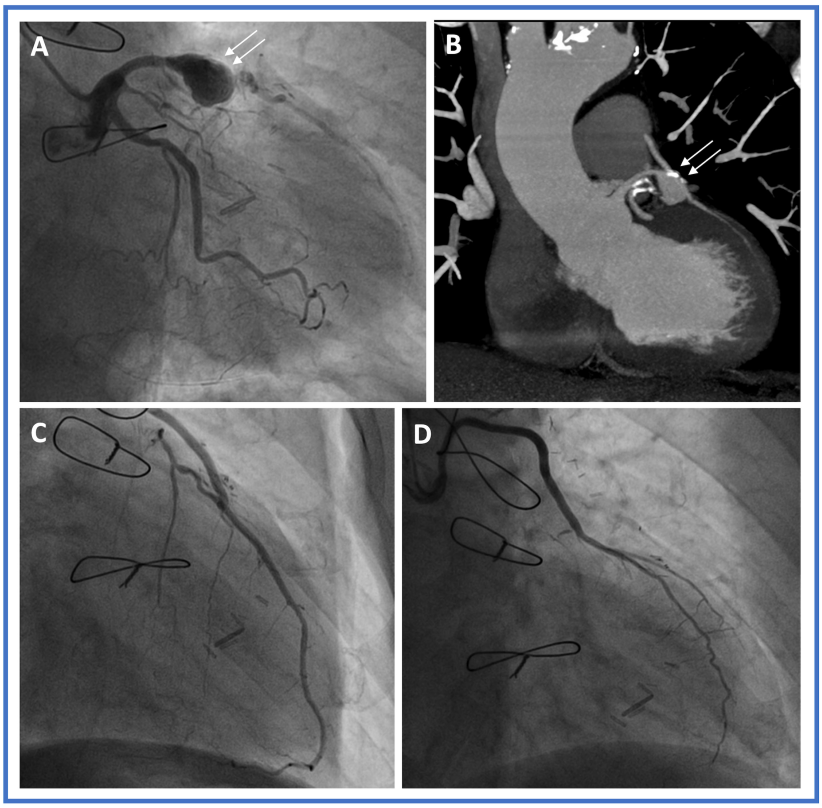

Figure 2. Angiographic (A) and CT-scan (B) appearance of a LAD saccular aneurysm (double arrows) in a patient with prior CABG. (C and D) show patency of LIMA to LAD and SVG to OM grafts, respectively. CT: Computed tomography; LAD: left anterior descending artery; CABG: coronary artery bypass grafting; LIMA: left internal mammary artery; SVG: saphenous vein graft; OM: obtuse marginal.

Regarding vasodilator administration, Kruger *et al.* demonstrated that nitroglycerin worsened myocardial ischemia and its effect was related to coronary artery diameters. Taken these data into account, nitrates are not recommended in case of significant aneurysmal coronary disease[19].

In patients with acute coronary syndromes or stable CAD undergoing PCI and stenting, antithrombotic management should follow the treatment guidelines for ischemic heart disease[35-37]. In the absence of clear indications for dual antiplatelet therapy (DAPT), there is no data from randomized clinical trials or large prospective cohort studies supporting anticoagulation or DAPT in patients with CAA; moreover, the available evidence from retrospective studies is discordant.

One study investigating the outcome of STEMI patients according to the presence of CAE showed an increased risk of major adverse cardiac events (MACE) in the CAE group. Interestingly, CAE patients on Warfarin achieving a percent time in target therapeutic range (%TTR) ≥ 60% experienced significantly less MACE, with respect to those not taking anticoagulant or with a %TTR < 60% (0% *vs.* 33%; $P = 0.03$)[38].

A recent Propensity Score Matching analysis, including patients from the Coronary Artery Aneurysm Registry (CAAR), compared the incidence of primary coronary ischemic endpoint (composite of myocardial infarction, unstable angina and aneurysm thrombosis) and bleeding in patients discharged with or without anticoagulant (AC). After a 3-year median follow-up, the AC group showed a significantly lower incidence of the primary endpoint (8.7% *vs.* 17.2%, respectively; $P = 0.01$), driven by a significant reduction in unstable angina and aneurysm thrombosis, at the expense of a negligible higher risk of bleeding (mainly BARC type 1) ($P = 0.08$)[39]. However, nowadays, the only definite indication for anticoagulation in the setting of CAA refers to selected Kawasaki patients with large aneurysms or those with rapidly expanding ones[13].

Given the persistent uncertainties about the optimal antithrombotic strategy in the setting of CAA, the OVER-TIME Phase 2 trial (ClinicalTrials.gov Identifier: NCT05233124) has been designed to compare DAPT with aspirin plus a P2Y12 inhibitor versus Rivaroxaban 15 mg plus P2Y12 inhibitor for prevention of recurrent ischemic events in patients with CAE and acute coronary syndromes[40]. This study will be the first randomized controlled trial to yield safety and efficacy data regarding two different antithrombotic strategies in patients with CAE after acute coronary events.

Another clinical trial (ClinicalTrials.gov Identifier: NCT05718531) will evaluate different treatment strategies (DAPT with aspirin 75 mg and clopidogrel 75 mg, dual therapy with rivaroxaban 2.5 mg BID and aspirin 75 mg or clopidogrel 75 mg, triple therapy with rivaroxaban 2.5 mg BID, aspirin 75 mg and clopidogrel 75 mg) in acute and chronic coronary syndrome patients diagnosed with coronary artery ectasia either associated or not with obstructive CAD[41]. This study is not yet recruiting.

Percutaneous interventions

Percutaneous coronary intervention for the treatment of CAA and CAE presents significant technical challenges, and standardization of treatment is demanding due to the lack of dedicated devices. Optimal stent sizing is mandatory to avoid stent malapposition and embolization[42]. However, as already pointed out, coronary angiography has some limitations when it comes to assessing coronary aneurysms, especially in the case of endoluminal thrombosis, and may result in an underestimation of the true vascular lumen and consequently stent undersizing. In these cases, pre-procedural IVUS assessment is highly recommended. In patients presenting with acute myocardial infarction (MI), percutaneous treatment of an aneurysmal culprit lesion is associated with lower procedural success and higher rates of adverse events, including death, MI

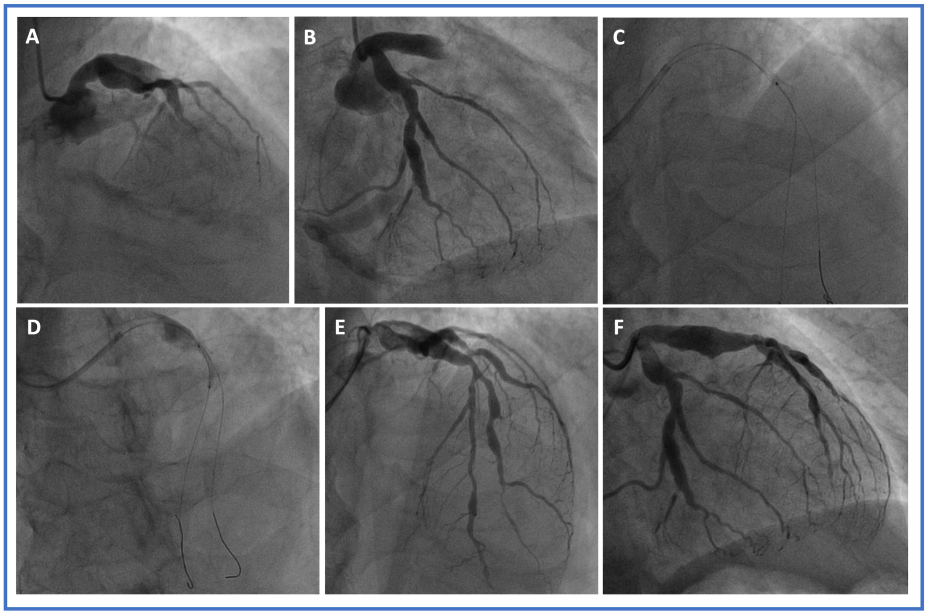

Figure 3. Cine angiography showing PCI of the LAD in a patient presenting with anterior wall STEMI. (A and B) Initial angiograms showing large fusiform aneurysm and occlusion of the proximal LAD. (C) Coronary guidewires advanced distally beyond the culprit lesion, manual thrombus aspiration with Export catheter and GPI administration through guiding catheter. (D) Balloon angioplasty with semi-compliant balloon. (E and F) Final result. PCI: Percutaneous coronary intervention; LAD: left anterior descending artery; STEMI: ST-segment elevation myocardial infarction. GPI: glycoprotein IIb/IIIa inhibitors.

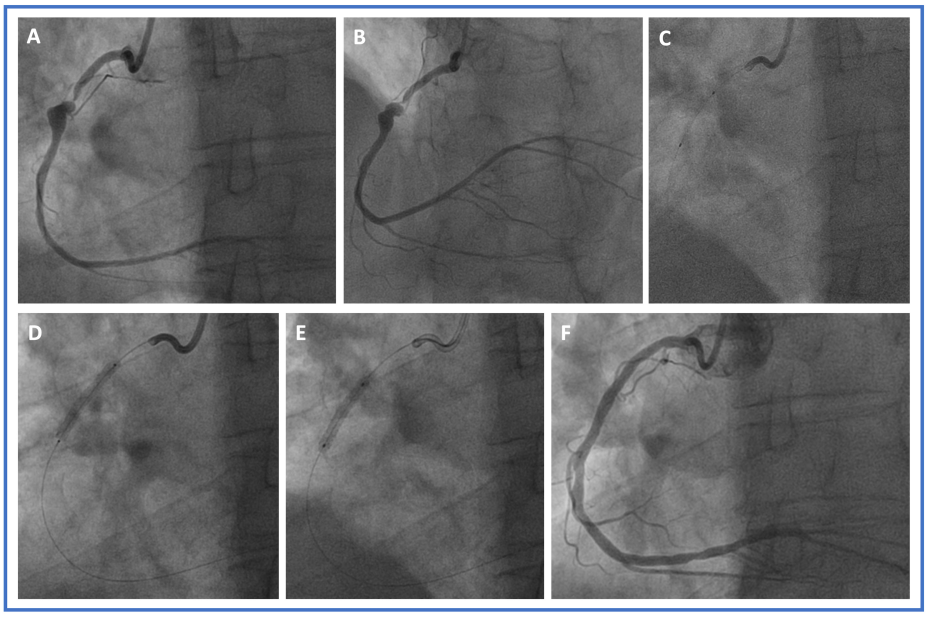

Figure 4. Cine angiography showing PCI of the RCA in a patient with stable angina. (A and B) Coronary angiography showing subocclusion and saccular mid-RCA aneurysm. (C) Pre-dilatation using semi-compliant and non-compliant balloons. (D) Bentley BeGraft covered stent deployment. (E) Post-dilation with non-compliant balloon. (F) Final result showing complete exclusion of the aneurysm. PCI: Percutaneous coronary intervention; RCA: right coronary artery.

recurrence, and stent thrombosis[5,43,44]. These higher failure rates are mainly attributed to the presence of a significant thrombus burden within the aneurysm, possible thrombus dislodgement after guidewire passage,

and stent malapposition. Combined use of thrombectomy and glycoprotein IIb/IIIa inhibitors is generally recommended as adjunctive therapy to restore blood flow in acute coronary syndromes setting[45] [Figure 3]. For all these reasons, the procedure should be carefully planned according to clinical presentation, etiology, location, and size of the aneurysm, and possible association with obstructive CAD.

When balloon-expandable stents are used to treat coronary stenoses in the proximity of an aneurysm, special care should be taken in selecting a stent with a length strictly limited to the stenotic segment, to avoid landing in the aneurysm and reduce the risk of struts malapposition. The use of self-expanding coronary stents was also reported to be safe and effective in patients with CAE or tapered segments, given the ability of self-apposing devices to adjust to varying lumen diameters[46-48]. Off-label treatment of CAAs with self-expanding carotid artery stents has also been proposed[49].

Different percutaneous strategies are available if the primary goal of the PCI is the complete exclusion of the aneurysmal sac. Covered stent implantation should be considered when treating saccular aneurysms or pseudoaneurysms that do not involve a major side branch[50] [Figure 4]. However, covered stents are bulky, not flexible, and thus may not be easy to deliver in tortuous and calcified vessels. In addition, some of them require large guiding catheters or introducer sheaths to be delivered. The most widely used covered stents are the GRAFTMASTER (Abbott Vascular, Santa Clara, California), the PK Papyrus (Biotronik, Berlin, Germany), and the 5F compatible BeGraft Coronary Stent Graft System (Bentley Innomed, Hechingen, Germany).

In the case of large aneurysms complicated by coronary thrombosis, the "stove-pipe technique", which involves the delivery of multiple overlapping covered stents within the aneurysm and post-dilation at proximal and distal landing zones, has also been described[51].

An alternative strategy is the so-called "double stent method", which consists of implanting a non-covered stent within another, with the aim of reducing stent permeability and promoting aneurysm thrombosis, while preventing the delayed endothelization observed after covered stent implantation[52].

When the previous techniques cannot be used due to tortuosity, calcifications, or fear of side branch occlusion, the stent-assisted coil embolization technique can be used. This technique involves the placement of a microcatheter inside the aneurysm over a floppy 0.14" guidewire. A drug-eluting stent is advanced over a second guidewire and deployed in the main vessel, jailing the microcatheter between the stent struts and the artery. Multiple coils are then delivered through the microcatheter inside the aneurysm. After angiographic confirmation of successful coil embolization of the aneurysm, the microcatheter is retrieved and the stent can be finally post-dilated[53-55].

In patients presenting with large CAAs, such as those originating from saphenous vein grafts, percutaneous closure with Amplatzer vascular plugs seems an attractive alternative to surgery when the affected graft supplies a small myocardial territory[14,56,57].

We performed an extensive search using online literature databases to identify all the available studies reporting the outcomes of patients with CAAs and CAE undergoing PCI. In particular, MEDLINE (PubMed), Google Scholar, and ClinicalTrials.org were searched using a combination of Boolean operators ("OR", "AND") for key terms including "Coronary artery ectasia", "Coronary artery aneurysm", "Percutaneous management", and "Outcomes". We included all the studies published from 1991 to May 2023. Data are shown in Table 2.

Table 2. Most representative studies on outcomes of coronary artery aneurysms patients undergoing different percutaneous interventions

First author, year	Study sample size and population characteristics	Study design	Follow-up length	Outcomes	Measure of effect and/or *P*-values	Comments
Schram *et al.*, 2018[58]	77 no-reflow STEMI pts *vs.* 154 with TIMI flow ≥ 2 after PPCI. 32 pts with Ectatic IRA	Case-control study	N/A	No reflow post-PPCI	CAE frequency higher in no-reflow pts (33.8% *vs.* 3.9%; $P < 0.001$). CAE pts less often underwent stenting and experienced more no-reflow (25.6% *vs.* 81.3%; $P < 0.001$)	CAE remained a strong independent predictor of no-reflow at multivariate analysis
Fujii *et al.*, 2017[59]	39 STEMI pts with Ectatic IRA *vs.* 705 pts with non-Ectatic IRA	Retrospective	775 days	Post-PPCI TIMI flow. 30-day and 1-year all-cause death	Higher TIMI flow ≤ 2 in the Ectatic IRA group (46.2% *vs.* 6.5%; $P < 0.0001$) All-cause 30-day and 1-year death was lower in the Ectatic group (0% *vs.* 9.4%; $P = 0.045$ and 2.6% *vs.* 14.5%; $P = 0.036$, respectively)	
Abdelfattah *et al.*, 2021[60]	41 pts with TCE (2 CAA, 2 CAPA)	Retrospective	10 months	Successful angiographic closure; adverse events related to tce; mortality	Successful angiographic closure in 87.8% of cases (100% of CAA and CAPA patients). No TCE-related adverse events in CAA/CAPA pts. 1 CAA patient died from respiratory failure 42 days after the procedure	
Wu *et al.*, 2020[61]	33 pts with CAA and stenosis > 60%, treated with DES	Retrospective	1 year	Reduction of CAA size, MLD, and DS after PCI, and 1 year thereafter	MLD and DS improved right after PCI ($P < 0.01$). TIMI flow grade was enhanced after PCI ($P < 0.01$). At 1-year f/u, maximal CAA diameter was reduced compared with that just after PCI ($P < 0.01$)	No clinical events reported at 1-year f/u
Parikh *et al.*, 2019[62]	32 pts treated with PTFE CCS (24 CAP, 8 CAA)	Retrospective	49 months	TLR, ISR, all-cause mortality	Neither in-hospital or 30-day mortality nor evidence of TLR at f/u. All-cause mortality was 12% at 1 year and 38% at 3 years	
Hachinohe *et al.*, 2019[63]	190 pts treated with CCS for SVG (51.4%), CAP (25.0%), and CAA (11.8%)	Retrospective	6.0 years	TV-MI, TVO, TLR, ST, cardiac death	TLF lower in CAA than in SVG ($P = 0.023$). Higher TVO, TV-MI, and ST in pts with CAP ($P = 0.010$, 0.047, and 0.046, respectively) TVO rate was highest in pts with SVG (43.8% at 10 years) No differences in TLR and cardiac death between groups	
Joo *et al.*, 2018[6]	78 CAA pts *vs.* 269 non-CAA pts after DES implantation	Retrospective	16.1 months	MACE (all-cause death, nonfatal MI and TLR)	CAA patients had higher MACE (26.9% *vs.* 2.2%; $P < 0.01$), driven by nonfatal MI and TVR. ST was higher in the CAA group (12.8 *vs.* 0.74%,$P < 0.001$)	Analyses after PSM showed similar results
Yip *et al.*, 2002[64]	24 STEMI pts with aneurysmal IRA	Retrospective	19 months	In-hospital outcomes and survival at 19 ± 30 months f/u	No-flow, cardiogenic shock, and in-hospital death rates were 62%, 25%, and 8.3%, respectively. Survival at f/u was 90.9%	Post-PCI distal embolization in 70.8% of pts with aneurysmal IRA
Briguori *et al.*, 2002[65]	7 pts with CAA treated with PTFE CCS	Retrospective	35 months	In-hospital outcomes, and MACE at f/u	Angiographic success in 100% of cases 1 (14%) MACE event (TVR)	
Iannopollo *et al.*, 2017[5]	32 STEMI pts with CAA as CL *vs.* 2280 STEMI pts without CAA as CL treated with PPCI	Retrospective	1 year	Primary outcome (all-cause death and recurrent MI). Definite ST	CAA as CL in STEMI was independently associated with primary outcome (HR: 2.24; 95%CI: 1.02-5.39; $P = 0.04$) and ST (HR: 6.29; 95%CI: 2.32-17.05; $P < 0.001$)	Primary endpoint was driven by MI due to higher rates of ST
Djohan *et al.*, 2022[66]	36 STEMI pts with CAE *vs.* 1816 STEMI non-CAE	Retrospective	3 years	MACE (all-cause mortality, MI, unplanned	At 3 years, all-cause mortality was significantly more frequent in the non-CAE group (0.0% *vs.*	CAE pts had more RCA involvement, less coronary

				revascularization, CHF, and stroke)	11.5%, $P < 0.028$), and on multivariate analysis, CAE was not an independent predictor of MACE (HR: 0.62, 95%CI: 0.29-1.31; $P = 0.209$)	stenting and post-PPCI TIMI 3 flow
Amirzadegan *et al.*, 2020[67]	87 CAE pts undergoing PCI	Retrospective	1 year	MACE (mortality, nonfatal MI, repeated revascularization, and stroke)	CAE was significantly associated with urgent repeat revascularization (HR: 2.40; 95%CI: 1.13-5.86; $P = 0.013$). No differences in all-cause mortality and nonfatal MI	At multivariate analysis, CAE not associated with MACE (HR: 1.65, 95%CI: 1.08-4.78; $P = 0.391$)
Baldi *et al.*, (2022)[68]	154 CAE STEMI pts *vs.* 380 non-CAE STEMI pts	Retrospective with PS weighting	3.3 years	Recurrent MI at the longest available follow-up	After PPCI, the corrected TIMI frame count ($P < 0.001$) and myocardial blush grade ($P < 0.001$) were lower in CAE pts. At multivariate analysis, the risk for the primary outcome was significantly higher in pts with CAE (aHR: 1.84; $P = 0.017$)	All-cause mortality and cardiac death were comparable between study groups
Campanile *et al.*, 2014[69]	101 acute MI pts with IRA Ectasia	Retrospective	2 years	MACE (cardiac death, MI recurrence, and new revascularisation)	Procedural success was 70.3%. Incidence of MACE was 6.9%, 17.8%, and 38.5% at 30-day, 1-year, and 2-year f/u, respectively. 8.9% had ST	
Bogana Shanmugam *et al.*, 2017[44]	25 CAE STEMI *vs.* 80 non-CAE STEMI	Retrospective	36.6 months	Procedural success. MACE (death, emergency revasc, nonfatal MI, or unstable angina)	CAA group had less angiographic success (24% *vs.* 77%; $P < 0.01$) and higher MACE (44.0% *vs.* 16.3%, $P = 0.01$), driven by nonfatal MI, TVR, and sudden death	
Ipek *et al.*, 2016[43]	99 STEMI pts with ectatic IRA *vs.* 1,556 STEMI pts with no ectatic IRA	Retrospective	1 year	In-hospital and 1-year MACE	Higher rates of no-reflow in CAE pts (13.1% *vs.* 5.4%, $P = 0.004$), no differences in in-hospital or 1-year mortality, TVR or ST	
Erden *et al.*, 2010[70]	31 MI pts with IRA Ectasia *vs.* 612 MI pts without IRA Ectasia	Retrospective	Post-discharge	TIMI flow grade, TIMI myocardial perfusion grade < 3, thrombus burden score, and distal embolization	TIMI Flow Grade < 3, TIMI Myocardial Perfusion Grade < 3, and Distal embolization were more frequent in pts with IRA Ectasia (11.6% *vs.* 41.9%, $P < 0.001$; 24.6% *vs.* 61.2%, $P < 0.001$ and 9.1% *vs.* 38.7%, $P < 0.001$, respectively). Thrombus burden score was higher in the CAE subgroup (4.48 *vs.* 4.97, $P = 0.004$)	IRA Ectasia was an independent predictor of adverse procedural outcomes
Doi *et al.*, 2017[38]	51 MI pts with CAE *vs.* 1,647 MI pts without CAE	Retrospective with PSM	49 months	MACE (cardiac death and nonfatal MI). Individual components of the composite outcome	CAE was a predictor of MACE (HR: 3.25, 95%CI: 1.88-5.66; $P < 0.001$), cardiac death (HR: 2.7, 95%CI: 1.37-5.37; $P = 0.004$), and nonfatal MI (HR: 4.92, 95%CI: 2.20-11.0; $P < 0.001$). The association was confirmed both in multivariate and in a PS-matched cohort analysis	Higher MACE in pts on Warfarin with %TTR < 60% or in those not anticoagulated.
Araiza-Garaygordobil *et al.*, 2022[71]	539 STEMI pts treated with PPCI, 56 (10.3%) with CAE	Retrospective	1 month	MACE (cardiogenic shock, CHF, recurrent MI, death). Survival at 1 month	No statistically significant differences in MACE between groups (HR: 0.87, 95%CI: 0.34 - 2.19, Log-rank $P = 0.76$). No differences in survival at 1-month f/u, regardless of DAPT or interventional strategy used	Rates of major bleeding were also comparable
Mir *et al.*, 2022[72]	9,671 CAA pts (52.7% non-ACS and 47.3% ACS) *vs.* 6,834,239 non-CAA pts	Retrospective	30 days	All-cause in-hospital mortality and hospital readmission rate	Similar rates of in-hospital mortality between CAA and non-CAA pts (2.17% *vs.* 2.56%; $P = 0.08$). Hospital readmission rate significantly higher for non-CAA patients (non-CAA 13.8% *vs.* CAA 4.6%; $P = 0.001$)	
Eid *et al.*, 2023[73]	13,499 STEMI pts undergoing PPCI	Meta-analysis	3.16 years	Primary: all-cause mortality, MACE(s)	All-cause mortality (OR: 0.95, 95%CI: 0.58-1.56; $P = 0.79$),	Also, TVR and the need for

	(389 CAA *vs.* 3,843 non-CAA pts)			and re-MI. Secondary: TVR, need for mechanical support	MACE (OR: 4.04, 95%CI: 0.34-47.57; *P* = 0.17), re-MI (OR: 2.13, 95%CI: 0.83-5.47; *P* = 0.08) were all non-significantly associated with CAE status	mechanical supportive devices were similar between groups
Núñez-Gil *et al.*, 2018[4]	256 ACS + CAA pts *vs.* 500 ACS pts	Retrospective with PSM	52 months	MACEs (Mortality, Bleeding or MI)	CAAs were independent risk factors for both all-cause mortality (HR: 3.1, 95%CI: 1.8-5.6; *P* < 0.001) and MACEs (HR: 2.3, 95%CI: 1.4-3.8; *P* < 0.001)	

HR: Hazard ratio; OR: odds ratio; CI: confidence interval; STEMI: ST segment elevation myocardial infarction; TIMI: thrombolysis in myocardial infarction; PPCI: primary percutaneous coronary intervention; IRA: infarct related artery; CAE: coronary artery ectasia; TCE: transcatheter coil embolization; CAA: coronary artery aneurysm; CAPA: coronary artery pseudoaneurysm; DES: drug eluting stent; MLD: minimal lumen diameter; DS: diameter stenosis; PCI: percutaneous coronary intervention; PTFE: polytetrafluoroethylene; CCS: coronary covered stent; TLR: target lesion revascularization; ISR: in-stent restenosis; SVG: saphenous vein graft; CAP: coronary artery perforation; TV-MI: target vessel myocardial infarction; TVO: target vessel occlusion; ST: stent thrombosis; TLF: target lesion failure; MACE: major adverse cardiovascular event(s); TVR: target vessel revascularization; PSM: propensity score matching; CL: culprit lesion; MI: myocardial infarction; CHF: congestive heart failure; RCA: right coronary artery; TTR: time in therapeutic range; DAPT: dual antiplatelet therapy; ACS: acute coronary syndromes; N/A: not applicable; revasc: revascularization.

Surgical interventions

Surgical treatment of CAAs generally consists of resection or ligation of the aneurysm associated with aortocoronary bypass[74]. Surgery should be considered for giant aneurysms with a high likelihood of rupture or compressing adjacent structures, in the presence of complications such as fistula formation, or when coronary anatomy precludes percutaneous treatment[75,76] [Figure 2C and D].

CONCLUSION

Coronary artery aneurysms and ectasia are often asymptomatic and detected incidentally at coronary angiography or CT-scan. However, they have been associated with clinical manifestations that mimic those of ischemic heart disease, and with poor outcomes regardless of clinical presentation. Given the uncertainty about pathogenesis, natural history, and optimal treatment strategy for CAA, multicenter registries are warranted both to determine the prognosis of asymptomatic patients with CAAs/CAEs and to inform about the outcome of each therapeutic approach. For the time being, given the lack of high-quality studies and randomized trials, we suggest a patient-tailored treatment depending on the size, location, and morphology of the aneurysm, clinical presentation, and patient's comorbidities.

DECLARATIONS

Authors' contributions

Contributed to the design of the paper, the literature review, and the writing of the manuscript: Barioli A, Visco E

Supervised the project: Cernetti C, Favero L

Discussed the results and contributed to the final manuscript: Barioli A , Visco E, Pellizzari N, Marzot F, Lanzellotti D, Favero L, Cernetti C

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All authors declared that there are no conflicts of interest.

Ethical approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

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