



Coronary Aneurysmal and Ectatic Disease: Cath-Lab Diagnosis and Contemporary Interventional Management

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Coronary aneurysmal and ectatic disease (CAA/CAE) is an increasingly recognized condition in contemporary catheterization laboratory practice and is predominantly associated with atherosclerotic remodeling. It is characterized by excessive positive remodeling, vessel wall degeneration, altered coronary hemodynamics, and a prothrombotic milieu, all of which contribute to an increased risk of ischemic events. Invasive angiography remains the primary diagnostic tool, while intravascular imaging is essential for accurate morphological characterization, detection of intraluminal thrombus, and guidance of percutaneous interventions. Revascularization strategies should be individualized, with percutaneous intervention reserved for flow-limiting lesions and surgery considered for giant or complex aneurysms. Evidence guiding antithrombotic therapy is limited and largely observational. A recent network meta-analysis suggests that dual antiplatelet therapy is associated with the greatest reduction in major adverse cardiovascular events, whereas routine oral anticoagulation has not demonstrated consistent benefit. Current ACC/AHA and ESC guidelines do not provide specific recommendations for aneurysmal disease; therefore, antithrombotic management is generally extrapolated from standard recommendations according to clinical presentation. In conclusion, pending the results of ongoing trials, a phenotype-driven and individualized approach remains the most appropriate management strategy.

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Definition and clinical relevance of coronary aneurysmal disease

Coronary aneurysmal disease encompasses a spectrum of pathological coronary artery dilatations, including coronary artery aneurysms (CAA) and coronary artery ectasia (CAE). A coronary artery aneurysm is conventionally defined as a focal dilatation of a coronary segment exceeding 1.5 times the diameter of the adjacent normal reference vessel, whereas coronary ectasia refers to diffuse dilatation involving a substantial portion of the vessel length [1,2]. Although historically considered a rare or incidental angiographic finding, coronary aneurysmal disease is increasingly recognized in contemporary practice, particularly in the setting of diagnostic coronary angiography and invasive evaluation of acute coronary syndromes. From a clinical perspective, aneurysmal and ectatic coronary segments are not benign anatomical variants. They are associated with altered coronary hemodynamics, local blood flow stasis, increased

thrombogenicity, and frequent coexistence with obstructive atherosclerotic disease, all of which may contribute to myocardial ischemia and adverse cardiovascular events [3–5]. Despite growing recognition, coronary aneurysmal disease remains a poorly standardized clinical entity. There is substantial heterogeneity in definitions, imaging assessment, reported prevalence, and therapeutic approaches across studies. As a result, management is often extrapolated from conventional coronary artery disease paradigms, despite important pathophysiological differences between aneurysmal and nonaneurysmal vessels. In particular, the optimal antithrombotic strategy in patients with CAE or CAA is uncertain. While antiplatelet therapy represents the cornerstone of treatment in atherosclerotic coronary disease, the presence of aneurysmal dilatation raises theoretical and practical concerns regarding the adequacy of antiplatelet therapy alone in preventing thrombus formation and distal embolization [6]. Conversely, routine oral anticoagulation is not supported by robust randomized evidence and may expose patients to an increased risk of bleeding. This uncertainty results in marked variability in real-world practice and highlights a significant gap between pathophysiological rationale and evidence-based recommendations.

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Rationale and aims of the present review

The absence of dedicated randomized trials, coupled with the heterogeneity of available observational data, has limited the development of clear guideline-directed recommendations for the diagnosis and management of coronary aneurysmal disease. Invasive cardiologists are therefore frequently required to make complex decisions regarding imaging, revascularization strategies, and long-term antithrombotic therapy in the absence of high-level evidence. The present review was designed to provide a state-of-the-art overview of coronary aneurysmal and ectatic disease, with a specific focus on aspects most relevant to contemporary catheterization laboratory practice. Particular emphasis is placed on the pathophysiological substrate, the role of intracoronary imaging in diagnosis and procedural planning, and the spectrum of available medical, percutaneous, and surgical treatment options. In addition to a comprehensive narrative synthesis, we performed a systematic appraisal of the available evidence on antithrombotic therapy in adult patients with coronary artery ectasia or aneurysmal coronary disease of predominantly atherosclerotic origin. Studies exclusively involving Kawasaki disease were intentionally excluded from the systematic appraisal because of their distinct pathophysiology, natural history, and antithrombotic management. By integrating pathophysiological concepts, invasive imaging insights, and a structured evaluation of antithrombotic strategies, this review aims to offer a pragmatic, phenotype-based framework to support clinical decision-making in patients with coronary aneurysmal disease.

Definitions and Classification

Coronary artery aneurysm versus coronary artery ectasia

Coronary aneurysmal disease includes 2 main entities: coronary artery aneurysm (CAA) and coronary artery ectasia (CAE). CAA is defined as a focal dilatation of a coronary artery segment exceeding 1.5 times the diameter of an adjacent angiographically normal reference vessel, whereas CAE refers to a diffuse dilatation involving a longer vessel segment, typically affecting more than one third of the vessel length [1,2]. Although these definitions are widely accepted, the distinction between focal aneurysm and diffuse ectasia is not always straightforward in clinical practice, particularly when angiographic assessment is limited by vessel overlap, foreshortening, or concomitant thrombus. Importantly, CAA and CAE are often grouped together in clinical studies, despite potential differences in underlying mechanisms and clinical implications. For this reason, the term coronary aneurysmal disease is frequently used as an umbrella definition encompassing both focal and diffuse dilatative phenotypes.

Morphological classification

From a morphological standpoint, aneurysmal coronary lesions may be further classified according to their shape and longitudinal extent (Figure 1). Saccular aneurysms are characterized by a transverse diameter exceeding the longitudinal dimension and typically present as focal outpouchings of the vessel wall. In contrast, fusiform aneurysms exhibit a more uniform, elongated dilatation involving the entire vessel circumference [7]. This distinction may have practical implications, as saccular aneurysms are more often associated with localized wall disruption, pseudoaneurysm formation, or iatrogenic injury, whereas fusiform dilatations are more frequently linked to diffuse atherosclerotic remodeling or inflammatory processes. Nevertheless, robust data correlating aneurysm morphology with clinical outcomes remain limited.

Size-based definitions

Beyond relative vessel enlargement, several authors have proposed absolute size thresholds to define large or giant coronary aneurysms (Figure 1). Although no universally accepted cut-off exists in adults, aneurysms with a diameter exceeding 20 mm, or more than 4 times the reference vessel diameter, are commonly referred to as giant aneurysms in the literature [8]. Giant coronary aneurysms are rare but clinically relevant due to their association with marked flow disturbances, intraluminal thrombosis, distal embolization, and technical challenges during percutaneous or surgical treatment. Their natural history is poorly defined, and management strategies are largely based on small case series and expert opinion.

True aneurysm versus pseudoaneurysm

A critical distinction in the evaluation of coronary aneurysmal disease is that between true aneurysms and pseudoaneurysms (Figure 1). True aneurysms involve all 3 layers of the vessel wall (intima, media, and adventitia) and are most commonly associated with atherosclerotic degeneration or chronic inflammatory processes. Pseudoaneurysms, by contrast, result from a disruption of the vessel wall with containment of blood by the surrounding tissues, and are frequently related to iatrogenic injury following percutaneous coronary intervention, coronary dissection, or perforation [9]. This distinction has important therapeutic implications, as pseudoaneurysms may exhibit a higher risk of expansion or rupture and often warrant a more aggressive interventional or surgical approach. Intravascular imaging plays a key role in differentiating between these 2 entities.

Angiographic classifications

Several angiographic classifications have been proposed to describe the extent and distribution of coronary ectasia. Among these, the Markis classification remains the most widely used and categorizes CAE into 4 types based on the number of vessels involved and the presence of diffuse versus localized dilatation [2]. While such classifications facilitate descriptive reporting and comparison across studies, their prognostic and therapeutic value remains uncertain, and they are not routinely incorporated into contemporary management algorithms.

Epidemiology and Etiology

The reported prevalence of coronary aneurysmal disease varies considerably among angiographic series, largely due to differences in definitions, study populations, and imaging techniques. Coronary artery ectasia and aneurysms have been described in approximately 0.3% to 5% of patients undergoing coronary angiography, with higher prevalence observed in older series and in cohorts enriched for obstructive coronary artery disease [10,11]. In contemporary practice, coronary aneurysmal disease is most often identified incidentally during invasive coronary angiography performed for the evaluation of acute or chronic coronary syndromes. Diffuse ectasia appears to be more common than focal aneurysms, and the right coronary artery is the vessel most frequently involved, followed by the left anterior descending and left circumflex arteries [2,10]. Multivessel involvement is frequently observed, particularly in diffuse ectatic phenotypes.

Atherosclerotic coronary aneurysmal disease

In adult populations, atherosclerosis represents the most common underlying cause of coronary aneurysmal disease. Autopsy and

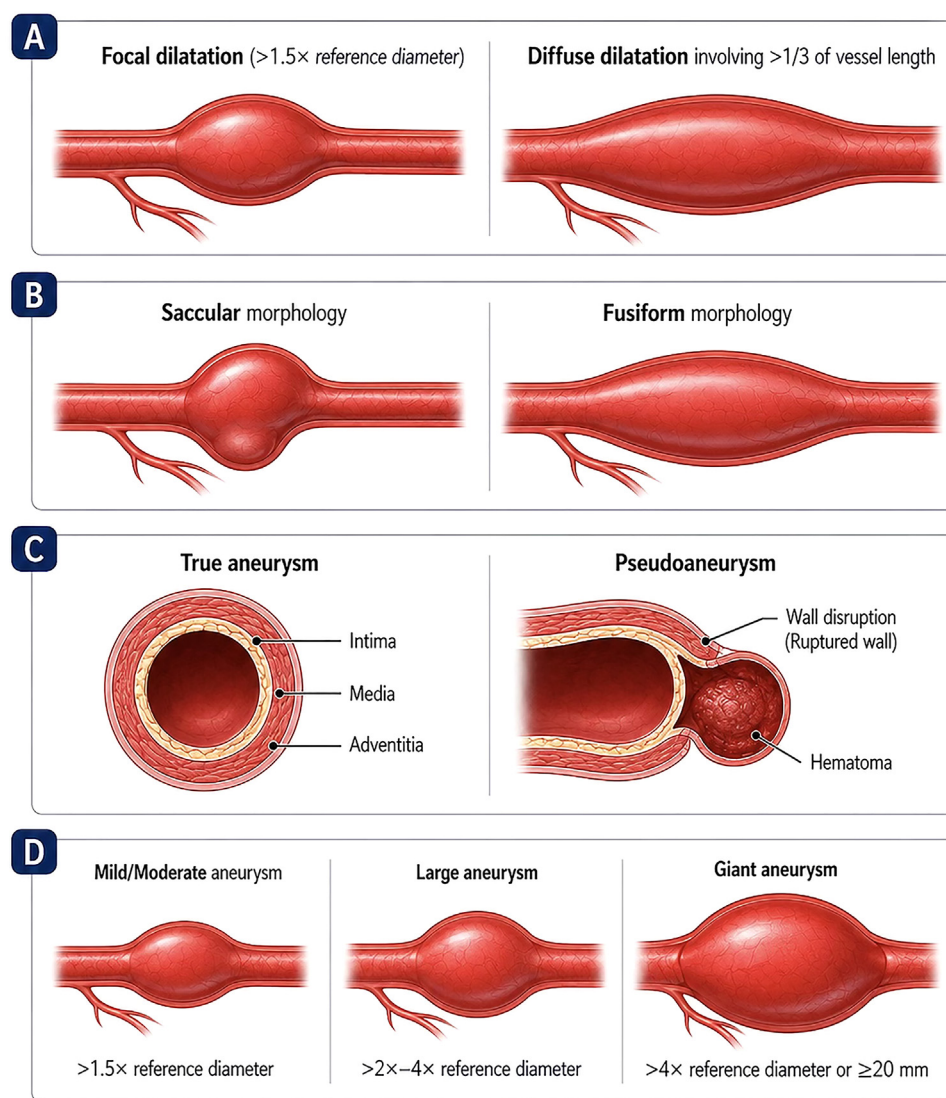


Figure 1. Morphological classification of coronary aneurysmal disease. Schematic representation of the main morphological patterns of coronary aneurysmal disease. Panel A: distinction between focal coronary artery aneurysm, defined as a localized dilatation exceeding 1.5 times the diameter of the adjacent reference vessel, and diffuse coronary artery ectasia, characterized by uniform dilatation involving more than one third of the vessel length. Panel B: 2 principal morphological configurations of aneurysmal segments: saccular aneurysms, presenting as asymmetric focal outpouchings, and fusiform aneurysms, showing circumferential and longitudinal vessel enlargement. Panel C: true coronary aneurysms, involving all layers of the vessel wall, and pseudoaneurysms resulting from focal wall disruption and extravascular blood containment, a distinction with important diagnostic and therapeutic implications. Panel D: size-based classification, highlighting the concept of giant coronary aneurysms, commonly defined as a diameter greater than 4 times the reference vessel diameter or exceeding 20 mm.

angiographic studies consistently indicate that the majority of coronary aneurysms and ectatic segments are associated with advanced atherosclerotic changes of the vessel wall [11,12]. In this setting, aneurysmal dilatation is thought to result from excessive positive remodeling in response to plaque development, combined with degradation of the extracellular matrix and weakening of the medial layer. Importantly, atherosclerotic coronary aneurysmal disease frequently coexists with significant obstructive lesions, either within the aneurysmal segment or in adjacent vessel segments. This anatomical combination contributes to complex flow disturbances and may partly explain the increased incidence of acute coronary syndromes and adverse cardiovascular events reported in patients with coronary ectasia [4,6].

Iatrogenic aneurysms and pseudoaneurysms

The increasing use of percutaneous coronary intervention has led to growing recognition of iatrogenic coronary aneurysms and

pseudoaneurysms. These lesions may arise as a consequence of vessel wall injury related to balloon overexpansion, stent malapposition, coronary dissection, or perforation, and have been described following both bare-metal and drug-eluting stent implantation [9,13]. Iatrogenic aneurysms are often detected during follow-up angiography or intravascular imaging and may demonstrate variable clinical behavior. While some remain stable over time, others may progress or become complicated by thrombosis, highlighting the importance of careful imaging-based characterization and individualized management strategies.

Inflammatory and other nonatherosclerotic causes

Less commonly, coronary aneurysmal disease may occur in association with nonatherosclerotic conditions, including systemic inflammatory or autoimmune disorders and vasculitides [14]. In these settings, immune-mediated injury of the vessel wall leads to structural weakening and aneurysm formation. Infectious coronary

aneurysms represent a rare entity and have been described mainly in association with systemic infection or endocarditis. Given their rarity and heterogeneous presentation, available evidence is largely limited to isolated case reports and small case series.

Differential diagnosis and etiology-oriented considerations

A systematic etiological assessment is essential because coronary aneurysmal disease encompasses clinically distinct entities with different natural histories and therapeutic implications. In adult patients, atherosclerotic aneurysmal disease is usually associated with conventional cardiovascular risk factors, concomitant obstructive coronary artery disease, calcified or lipid-rich plaque, and diffuse positive remodeling. Management is primarily based on aggressive secondary prevention, antiplatelet therapy, and selective revascularization of flow-limiting lesions [8,11,12]. Iatrogenic aneurysms and pseudoaneurysms should be suspected when aneurysmal dilatation develop at the site of prior balloon angioplasty, stent implantation, coronary dissection, perforation, or other percutaneous coronary intervention. These lesions require careful anatomical characterization, preferably with intravascular imaging and/or CCTA, because pseudoaneurysms reflect focal wall disruption rather than true dilatation of all vessel wall layers. Compared with stable true aneurysms, pseudoaneurysms may warrant earlier interventional or surgical exclusion when progressive enlargement, persistent filling, or high-risk anatomy is present [9,13]. Inflammatory, autoimmune, infectious, or connective tissue-related aneurysms are less common in adult cath-lab practice but should be considered in younger patients, in those without conventional atherosclerotic risk factors, or in the presence of systemic features such as fever, elevated inflammatory markers, vasculitis, endocarditis, or multisite aneurysmal disease. In these settings, treatment should not be limited to coronary anatomy alone but should include etiology-directed therapy, such as immunosuppression for active vasculitis or antimicrobial therapy for infectious aneurysms, in collaboration with appropriate specialists [8,14]. This etiology-oriented approach helps distinguish lesions suitable for conservative management from those requiring intensified surveillance, percutaneous exclusion, surgery, or systemic disease-specific therapy.

Pathophysiological Substrate

The pathophysiology of coronary aneurysmal disease is characterized by progressive structural weakening of the vessel wall, resulting in abnormal positive remodeling and luminal dilatation. In atherosclerotic coronary aneurysms, this process reflects an imbalance between extracellular matrix synthesis and degradation, with loss of medial smooth muscle cells and fragmentation of elastic fibers [15,16]. Histopathological studies have demonstrated thinning of the medial layer, disruption of the internal elastic lamina, and replacement of normal vessel wall architecture by fibrotic tissue. These changes reduce the tensile strength of the arterial wall and predispose to progressive dilatation under physiological hemodynamic stress. Inflammation plays a central role in the development of coronary aneurysmal disease. Increased infiltration of inflammatory cells within the vessel wall is associated with up-regulation of matrix metalloproteinases (MMPs), particularly MMP-2 and MMP-9, which promote degradation of collagen and elastin within the extracellular matrix [5,16]. Clinical studies have shown elevated circulating inflammatory markers and increased MMP activity in patients with coronary ectasia compared with patients with nonectatic coronary artery disease [5]. This inflammatory milieu contributes not only to aneurysm formation but also to ongoing vascular instability and progression over time. Beyond structural abnormalities, aneurysmal and ectatic coronary segments are associated with profound alterations

in coronary blood flow. Luminal dilatation leads to reduced flow velocity, disturbed laminar flow, and areas of low shear stress within the aneurysmal segment [3,6]. These hemodynamic changes impair distal coronary perfusion and may result in exercise-induced ischemia even in the absence of significant epicardial stenosis. Furthermore, low shear stress promotes endothelial dysfunction and local prothrombotic signaling, reinforcing the link between aneurysmal morphology and adverse clinical events. Blood flow stasis within aneurysmal segments favors intraluminal thrombus formation, which may remain mural or become unstable and embolize distally. Angiographic studies may demonstrate slow flow and contrast stasis, whereas intravascular imaging may reveal layered or organized thrombus within ectatic coronary segments, particularly in the setting of acute coronary syndromes [4,6]. Distal embolization from aneurysmal segments represents a plausible mechanism for myocardial infarction with nonobstructive coronary arteries or for recurrent ischemic events disproportionate to the severity of angiographic stenosis. This thrombogenic milieu provides a strong pathophysiological rationale for intensified antithrombotic strategies in selected patients, while simultaneously raising concerns regarding bleeding risk. A key feature of atherosclerotic coronary aneurysmal disease is the frequent coexistence of aneurysmal dilatation and obstructive lesions within the same vessel. Stenoses may be located proximally, within, or distal to the aneurysmal segment, creating complex pressure and flow gradients that further exacerbate turbulence and stasis [6,12]. This interaction complicates both diagnostic interpretation and therapeutic decision-making. In particular, angiographic assessment alone may underestimate the functional significance of lesions within ectatic vessels, underscoring the importance of intravascular imaging for accurate characterization and management planning. These pathophysiological alterations translate into distinct clinical consequences, as summarized in Table 1.

Clinical Manifestations and Natural History

A substantial proportion of patients with coronary aneurysmal disease are asymptomatic, and the condition is frequently identified as an incidental finding during coronary angiography performed for unrelated indications. This is particularly true for patients with mild or moderate diffuse ectasia without associated obstructive lesions [10,11]. In asymptomatic individuals, the clinical significance of aneurysmal dilatation is often uncertain, and the natural history may be characterized by long periods of stability. However, the absence of symptoms does not necessarily imply a benign course, as aneurysmal segments may still be associated with progressive remodeling, thrombus formation, or future ischemic events.

Chronic coronary syndromes

In patients presenting with chronic coronary syndromes, coronary aneurysmal disease may manifest as stable angina or exertional ischemia. Symptoms may arise from coexisting atherosclerotic stenoses, impaired distal perfusion related to flow disturbances within ectatic segments, or microembolization from mural thrombus [6,12]. Functional ischemia may be disproportionate to the severity of angiographic stenosis, complicating clinical assessment. In this context, coronary aneurysmal disease represents a heterogeneous substrate in which symptoms may reflect a combination of fixed obstruction and dynamic flow-related mechanisms rather than isolated epicardial narrowing.

Acute coronary syndromes

Coronary aneurysmal disease has been consistently associated with an increased incidence of acute coronary syndromes. Patients

Table 1
Pathophysiological mechanisms and clinical implications of coronary aneurysmal disease

Pathophysiological feature	Underlying mechanism	Clinical implications
Vessel wall degeneration	Medial thinning, loss of smooth muscle cells, fragmentation of elastic fibers	Progressive vessel dilatation and aneurysm formation
Excessive positive remodeling	Disproportionate outward remodeling in response to atherosclerotic plaque	Angiographic underestimation of plaque burden and lesion severity
Altered coronary hemodynamics	Reduced flow velocity, disturbed laminar flow, low shear stress	Impaired distal perfusion and exercise-induced ischemia
Flow stasis within aneurysmal segments	Lumen enlargement and turbulence	Intraluminal thrombus formation and distal embolization
Prothrombotic milieu	Endothelial dysfunction and platelet activation in low-shear environments	Increased risk of acute coronary syndromes
Inflammatory activation	Chronic vascular inflammation and matrix metalloproteinase upregulation	Disease progression and structural instability over time
Coexistence with obstructive lesions	Stenoses proximal, within, or distal to aneurysmal segments	Complex flow patterns and challenges in revascularization planning
Imaging-pathology mismatch	Limited vessel wall assessment by angiography alone	Need for intravascular imaging to guide diagnosis and treatment

with CAE or CAA may present with unstable angina, non–ST-segment elevation myocardial infarction, or ST-segment elevation myocardial infarction, even in the absence of critical epicardial stenosis [4,17]. Several mechanisms have been proposed to explain this association, including in situ thrombosis within aneurysmal segments, distal embolization of thrombotic material, plaque rupture at the margins of aneurysmal dilatation, and impaired coronary flow reserve. Angiographic and intravascular imaging findings often reveal a high thrombus burden and complex lesion morphology in this setting, underscoring the clinical relevance of aneurysmal disease during acute presentations [17,18].

Rare complications

Although uncommon, coronary aneurysmal disease may be complicated by rare but potentially serious events. Progressive aneurysm enlargement may lead to compression of adjacent cardiac structures, while fistula formation has been reported in isolated cases. Spontaneous rupture of coronary aneurysms is exceedingly rare in adults and is primarily described in case reports [11,12]. Iatrogenic pseudoaneurysms represent a distinct clinical entity and may exhibit a more aggressive course, with a higher risk of expansion or rupture compared with true aneurysms. Early recognition and appropriate management are therefore essential in this subgroup.

Prognostic implications and natural history

The long-term prognosis of patients with coronary aneurysmal disease remains incompletely defined. Observational studies suggest that the presence of coronary ectasia is associated with a higher incidence of adverse cardiovascular events compared with patients with nonectatic coronary artery disease, particularly in the setting of acute coronary syndromes [4,17]. Importantly, prognosis appears to be influenced by multiple factors, including the extent of aneurysmal involvement, coexistence of obstructive lesions, presence of intraluminal thrombus, and clinical presentation. These considerations highlight the need for individualized risk stratification and support a phenotype-based approach to management rather than a uniform treatment strategy.

Diagnostic Assessment in the Catheterization Laboratory

Coronary angiography: Strengths and limitations

Coronary angiography remains the first-line diagnostic modality for the identification of coronary aneurysmal disease. It allows visualization of luminal dilatation, assessment of aneurysm location and

longitudinal extent, and identification of associated obstructive lesions. In routine practice, angiography is often the modality through which coronary ectasia or aneurysms are first recognized, particularly in patients presenting with acute or chronic coronary syndromes [1,2]. However, angiography provides a 2-dimensional silhouette of the vessel lumen and does not allow direct visualization of the vessel wall. As a result, it may underestimate aneurysm size, fail to detect mural thrombus, or misclassify complex morphologies, especially in the presence of vessel overlap, foreshortening, or sluggish flow. In aneurysmal segments, contrast streaming and layering may further complicate interpretation and lead to inaccurate assessment of lesion severity or thrombus burden. In addition, quantitative coronary angiography relies on reference segments that may themselves be ectatic, further limiting its accuracy. These limitations are particularly relevant when procedural decisions depend on precise vessel sizing, landing zone selection, or assessment of lesion complexity, such as in the planning of percutaneous intervention or covered stent implantation.

Role of intravascular imaging

Intravascular ultrasound (IVUS) plays a central role in the diagnostic evaluation of coronary aneurysmal disease. By providing cross-sectional imaging of the vessel wall, IVUS allows accurate measurement of true vessel dimensions, characterization of plaque burden, and differentiation between true aneurysms and pseudoaneurysms [19]. In aneurysmal segments, IVUS is particularly valuable for identifying positive remodeling, assessing the integrity of the medial layer, and detecting mural thrombus that may not be apparent on angiography. During percutaneous coronary intervention, IVUS-guided sizing and optimization are essential to avoid stent undersizing, malapposition, or incomplete aneurysm exclusion, especially when covered stents are considered [13,19]. Optical coherence tomography (OCT) provides higher spatial resolution than IVUS and enables detailed visualization of intraluminal structures, including thrombus morphology, intimal disruption, and stent–vessel interactions. OCT is particularly useful for identifying layered or organized thrombus within aneurysmal segments and for assessing stent apposition and neointimal coverage following intervention [20]. However, the need for contrast injection to clear blood from the lumen may limit OCT use in large or ectatic vessels, where complete blood clearance can be challenging. For this reason, OCT and IVUS should be regarded as complementary techniques, with modality selection guided by vessel size, clinical context, and procedural objectives. The integration of angiography with intravascular imaging is fundamental to contemporary management of coronary aneurysmal disease. Imaging findings directly influence key clinical decisions, including

the choice between conservative management and revascularization, selection of percutaneous versus surgical strategies, and optimization of antithrombotic therapy. In particular, documentation of intraluminal thrombus, severe positive remodeling, or pseudoaneurysm formation may support a more aggressive therapeutic approach, whereas stable aneurysmal segments without thrombus or significant stenosis may be managed conservatively with close follow-up. In this context, intravascular imaging represents not only a diagnostic tool but also a cornerstone of risk stratification and personalized treatment planning.

Coronary computed tomography angiography and complementary noninvasive imaging

Coronary computed tomography angiography (CCTA) represents the most useful noninvasive imaging modality for the assessment and longitudinal follow-up of coronary aneurysmal disease. Unlike invasive angiography, which provides a 2-dimensional representation of the coronary lumen, CCTA allows 3-dimensional evaluation of aneurysm size, morphology, vessel course, and relationship with adjacent cardiac and mediastinal structures. This may be particularly valuable in patients with large or giant aneurysms, left main involvement, tortuous vessels, or complex anatomy requiring Heart Team discussion [21]. CCTA can also provide complementary information on mural thrombus, calcification, aneurysm wall characteristics, and coexistence of obstructive coronary artery disease. In patients managed conservatively, CCTA may be useful for noninvasive surveillance of aneurysm stability, interval enlargement, thrombus evolution, and development of new obstructive lesions. In selected cases, it may also support procedural planning before covered stent implantation or surgical repair by defining aneurysm length, reference vessel diameter, landing zones, and side-branch relationships [21]. However, CCTA has important limitations. Image quality may be reduced by severe coronary calcification, high heart rate, motion artifacts, prior stent implantation, and very small distal vessels. In acute coronary syndromes, invasive angiography remains the preferred diagnostic and therapeutic modality because it allows immediate lesion assessment and treatment. Therefore, CCTA should be considered complementary rather than alternative to catheterization laboratory assessment, with its greatest value in stable patients, longitudinal follow-up, and preprocedural anatomical planning [19–21].

Medical Management

General principles and risk factor modification

Medical management represents the cornerstone of treatment for the majority of patients with coronary aneurysmal disease, particularly in the absence of indications for percutaneous or surgical intervention. Given the strong association between coronary aneurysmal disease and atherosclerosis in adult populations, aggressive modification of cardiovascular risk factors should be pursued in all patients, including lipid-lowering therapy, blood pressure control, glycemic optimization, and smoking cessation [22]. Statins are of particular interest in this context, not only for their lipid-lowering effects but also for their pleiotropic anti-inflammatory properties, which may theoretically mitigate ongoing vascular remodeling and aneurysm progression. Although no randomized trials have specifically addressed statin therapy in coronary aneurysmal disease, their use is generally recommended in accordance with standard secondary prevention strategies.

Anti-ischemic therapy

Anti-ischemic treatment in patients with coronary aneurysmal disease follows the general principles applied to chronic coronary syndromes. Beta-blockers, calcium channel blockers, and other anti-anginal agents may be used to control symptoms, particularly in patients with coexisting obstructive lesions or microvascular dysfunction [6]. However, the use of vasodilators in ectatic coronary arteries remains controversial. In some patients, nitrates may exacerbate flow disturbances by further reducing coronary flow velocity within aneurysmal segments, potentially worsening ischemia related to slow flow or distal embolization. For this reason, anti-ischemic therapy should be individualized and guided by clinical response rather than applied uniformly.

Antiplatelet therapy and rationale for anticoagulation

Antiplatelet therapy constitutes the foundation of medical treatment in patients with coronary aneurysmal disease, particularly when an atherosclerotic substrate is present. Aspirin is commonly prescribed for long-term secondary prevention, while dual antiplatelet therapy is indicated in the setting of acute coronary syndromes or following percutaneous coronary intervention, in accordance with established guidelines [23]. In patients with coronary ectasia without significant obstructive disease, the optimal intensity and duration of antiplatelet therapy remain uncertain. Nevertheless, given the increased risk of thrombotic events reported in observational studies, long-term antiplatelet therapy is frequently adopted in clinical practice despite the absence of dedicated randomized evidence. Conversely, aneurysmal dilatation creates a distinct hemodynamic substrate characterized by low flow velocity, stasis, and low shear stress, providing a theoretical rationale for oral anticoagulation in selected patients with large aneurysmal segments, documented mural thrombus, or recurrent ischemic events. However, this pathophysiological rationale has not been consistently translated into clinical benefit in available studies. A 2025 network meta-analysis including 5,039 patients with coronary artery ectasia demonstrated that dual antiplatelet therapy (DAPT) was associated with the greatest reduction in major adverse cardiovascular events compared with no treatment (OR 0.28, 95% CI 0.16 to 0.51), followed by aspirin monotherapy (OR 0.45, 95% CI 0.25 to 0.79), whereas anticoagulation did not confer a statistically significant benefit (OR 0.62, 95% CI 0.29 to 1.32) [24].

Systematic appraisal of available evidence

To support the narrative synthesis, we performed a systematic appraisal of published evidence on antithrombotic therapy in adult patients with CAE/CAA of predominantly atherosclerotic origin. PubMed and Embase were searched from inception to March 2026 using terms related to coronary ectasia, coronary aneurysm, antiplatelet therapy, dual antiplatelet therapy, anticoagulation, and oral anticoagulants. Eligible studies included randomized trials, observational studies, and registries reporting clinical outcomes according to antithrombotic strategy. Studies exclusively involving pediatric populations or Kawasaki disease were excluded because of their distinct pathophysiology, natural history, and antithrombotic management. Given the limited number of eligible studies, their observational design, and heterogeneity in populations and outcome definitions, a quantitative meta-analysis was not performed. The available evidence remains limited and largely observational. Yip et al reported that patients with acute myocardial infarction and angiographically documented coronary aneurysms had higher thrombus burden and worse clinical outcomes than patients without aneurysmal disease, but the study did not demonstrate a clear benefit of routine oral

anticoagulation [25]. Doi et al showed that CAE was associated with a higher incidence of long-term major adverse cardiovascular events after acute myocardial infarction, although antithrombotic regimens were not systematically compared [4]. In patients with coronary aneurysms treated with first-generation drug-eluting stents, Joo et al reported higher adverse event rates than in nonaneurysmal vessels, with therapy largely based on prolonged antiplatelet treatment rather than systematic anticoagulation [26]. A 2025 network meta-analysis including 8 studies and 5,039 patients ranked DAPT as the most effective strategy for MACE prevention, whereas anticoagulation did not show a statistically significant benefit; however, residual heterogeneity and the observational nature of most included studies limit firm conclusions [24]. The main studies informing antithrombotic decision-making are summarized in Table 2.

Ongoing trials and practical implications

Overall, current evidence does not support routine oral anticoagulation in unselected patients with adult coronary aneurysmal disease. Antiplatelet-based therapy remains the default strategy, guided by clinical presentation and revascularization status. Oral anticoagulation may be considered as an off-label option in selected high-risk scenarios, including documented intraluminal thrombus, recurrent ischemic events despite adequate antiplatelet therapy, or very large/diffuse aneurysmal disease, after careful assessment of bleeding risk. Ongoing studies, including OVER-TIME (NCT05233124), NCT05718531, and the ITACA registry (NCT05051917), are expected to clarify whether rivaroxaban-based or other intensified antithrombotic strategies provide incremental benefit over standard antiplatelet therapy [27–29]. Current ACC/AHA and ESC guidelines do not provide specific recommendations for CAE/CAA and management should therefore follow standard ACS/chronic coronary syndrome antithrombotic guidance, adapted to individual ischemic and bleeding risk [30,31].

Percutaneous Interventional Strategies

Indications for percutaneous treatment

Percutaneous coronary intervention (PCI) in the setting of coronary aneurysmal disease is technically challenging and should be reserved for selected clinical scenarios. Indications for percutaneous treatment generally include the presence of significant obstructive lesions responsible for ischemia, acute coronary syndromes related to aneurysmal segments, iatrogenic aneurysms or pseudoaneurysms following PCI, and selected cases of focal aneurysms amenable to exclusion [8,13]. In contrast, diffuse ectasia without flow-limiting stenosis is usually managed conservatively, as routine stenting of ectatic segments is associated with a high risk of suboptimal results and complications. Careful patient selection and procedural planning are therefore essential.

Drug-eluting stents in aneurysmal vessels

Drug-eluting stents (DES) may be used to treat focal stenotic lesions located within or adjacent to aneurysmal segments. However, stent implantation in ectatic vessels is associated with several technical challenges, including difficulties in accurate sizing, increased risk of malapposition, and impaired drug distribution along the vessel wall. Clinical studies have reported higher rates of stent thrombosis and restenosis when DES are deployed in aneurysmal segments compared with nonaneurysmal vessels [32]. For this reason, DES implantation should be guided by intravascular imaging whenever possible, with careful attention to landing zone selection and postdilatation to optimize stent apposition.

Covered stents and management of thrombus-containing aneurysms

Covered stents represent a specific percutaneous strategy aimed at excluding aneurysmal segments from the coronary circulation. They are most commonly considered for the treatment of focal coronary aneurysms, pseudoaneurysms, and iatrogenic vessel perforations. By sealing the aneurysmal segment, covered stents may reduce the risk of thrombosis and distal embolization [33]. Despite their theoretical advantages, covered stents are associated with important limitations. These include increased stiffness, difficulty in delivery, risk of side branch occlusion, higher restenosis rates, and a non-negligible risk of stent thrombosis. In addition, accurate sizing and complete aneurysm coverage are critical, as incomplete exclusion may negate potential benefits. Thrombus-containing aneurysmal segments represent one of the most complex scenarios in the catheterization laboratory. Strategies may include cautious thrombus aspiration, use of potent antithrombotic agents, and deferred stenting in selected cases. Routine thrombus aspiration is not recommended, but selective use may be considered in the presence of large thrombus burden and impaired flow. Stent implantation in the acute setting should be approached with caution, as residual thrombus and vessel size mismatch increase the risk of acute and late complications. In some cases, stabilization with intensive medical therapy followed by staged intervention may represent a safer strategy.

Limitations of percutaneous approaches

Despite advances in device technology and imaging guidance, percutaneous treatment of coronary aneurysmal disease remains associated with significant limitations. Long-term data on outcomes are scarce, and most available evidence derives from small observational studies and case series. As a result, percutaneous strategies should be individualized and discussed within a multidisciplinary framework, particularly when alternative surgical options are available. In many patients, PCI represents a palliative rather than definitive solution, highlighting the need for careful follow-up and adjunctive medical therapy.

Table 2
Summary of studies evaluating antithrombotic therapy in adult coronary aneurysmal disease

Study	Study design	Population	Antithrombotic strategy	Main findings
Yip et al [25]	Retrospective observational study	Patients with acute myocardial infarction and angiographically documented coronary artery aneurysms	Predominantly antiplatelet therapy; oral anticoagulation used selectively	Coronary aneurysms associated with higher thrombus burden and worse outcomes; no clear benefit of routine anticoagulation
Doi et al [4]	Observational cohort study	Patients with acute myocardial infarction and coronary artery ectasia	Mainly antiplatelet-based therapy	Coronary ectasia independently associated with higher long-term MACE; excess risk not clearly modulated by antithrombotic regimen
Joo et al [26]	Retrospective cohort study	Patients with coronary artery aneurysms treated with drug-eluting stents	Prolonged antiplatelet therapy after PCI	Higher rates of adverse cardiovascular events compared with nonaneurysmal vessels; no evidence supporting routine escalation to anticoagulation

Surgical Management

Surgical management of coronary aneurysmal disease is generally reserved for selected patients in whom medical therapy and percutaneous strategies are either ineffective or technically unsuitable. Indications for surgical referral typically include giant coronary aneurysms, aneurysms involving the left main coronary artery, rapidly enlarging lesions, aneurysms associated with recurrent ischemic events despite optimal medical therapy, and cases complicated by fistula formation or suspected infection [8,11]. In addition, surgery may be considered when aneurysmal segments coexist with complex multivessel coronary artery disease not amenable to percutaneous revascularization, or when complete exclusion of the aneurysm cannot be safely achieved with covered stents. Several surgical techniques have been described for the treatment of coronary aneurysmal disease, often tailored to aneurysm size, location, and associated coronary pathology. These include aneurysm ligation or exclusion combined with coronary artery bypass grafting, aneurysm plication or resection, and, less commonly, interposition grafting [11,12]. In most cases, bypass grafting is performed distal to the aneurysmal segment to ensure adequate myocardial perfusion, while proximal ligation or exclusion prevents ongoing flow into the aneurysm sac. Complete aneurysm resection is rarely required and is typically reserved for giant or symptomatic aneurysms with compressive effects.

Available evidence on surgical management of coronary aneurysmal disease is limited and largely derived from small retrospective series and case reports. Long-term outcomes are therefore difficult to interpret, and direct comparisons with percutaneous strategies are lacking. Despite these limitations, surgical treatment has been associated with satisfactory symptom relief and acceptable long-term patency rates in selected patients, particularly those with large or complex aneurysms not suitable for percutaneous exclusion [8]. However, the invasive nature of surgery and the absence of robust comparative data underscore the importance of careful patient selection.

Role of the multidisciplinary heart team

Given the heterogeneity of coronary aneurysmal disease and the absence of standardized treatment algorithms, surgical decision-making should be integrated within a multidisciplinary Heart Team framework. Collaboration between interventional cardiologists, cardiac surgeons, and imaging specialists is essential to balance procedural risks, anticipated benefits, and long-term outcomes. In this context, surgery should be viewed as a complementary option within a broader, phenotype-driven management strategy rather than as a default approach. The choice between conservative management, percutaneous intervention, and surgical treatment should be individualized and guided by anatomical complexity, clinical presentation, and patient-specific risk profiles.

Practical Management Algorithm

Management of coronary aneurysmal disease requires an individualized and phenotype-driven approach that integrates clinical presentation, anatomical features, and thrombotic and bleeding risk. In the absence of guideline-directed recommendations supported by randomized trials, treatment decisions should be guided by a structured framework rather than by a uniform strategy, as outlined in [Figure 2](#).

Initial clinical stratification

The first step in management is clinical stratification based on presentation. Patients should be categorized as presenting with acute

coronary syndrome, chronic coronary syndrome, or incidentally detected coronary aneurysmal disease. Acute presentations warrant immediate invasive assessment and adherence to standard acute coronary syndrome protocols, with particular attention to thrombus burden and flow disturbances within aneurysmal segments.

In stable or asymptomatic patients, the clinical relevance of aneurysmal disease should be assessed in the context of symptoms, ischemia testing when appropriate, and overall cardiovascular risk profile.

Anatomical and imaging-based assessment

Anatomical characterization is central to decision-making. Key features include aneurysm size, focal versus diffuse involvement, number of vessels affected, and coexistence of obstructive coronary lesions. Intravascular imaging should be strongly considered to confirm aneurysm morphology, identify intraluminal thrombus, and distinguish true aneurysms from pseudoaneurysms. Large or giant aneurysms, diffuse multivessel ectasia, and thrombus-containing segments identify a higher-risk phenotype that may justify closer surveillance and more intensive therapy.

Antithrombotic strategy selection

Antiplatelet therapy represents the foundation of antithrombotic management in most patients with coronary aneurysmal disease. Single antiplatelet therapy is generally appropriate in stable patients without recent acute coronary syndrome or percutaneous coronary intervention, while dual antiplatelet therapy should be used according to standard indications in the setting of acute coronary syndromes or following stent implantation. Routine oral anticoagulation is not recommended in unselected patients. However, anticoagulation may be considered on an individual basis in selected high-risk scenarios, such as the presence of large or diffuse aneurysms with documented intraluminal thrombus, recurrent ischemic events despite adequate antiplatelet therapy, or evidence of distal embolization. In such cases, careful assessment of bleeding risk and periodic reassessment of the risk–benefit balance are essential.

Revascularization and escalation of care

Revascularization strategies should be tailored to the underlying anatomy and clinical context. Percutaneous intervention is appropriate for flow-limiting stenoses or culprit lesions responsible for ischemia, preferably guided by intravascular imaging. Covered stents or other aneurysm-excluding strategies should be reserved for selected focal aneurysms or pseudoaneurysms. Surgical referral should be considered in patients with giant aneurysms, left main involvement, complex multivessel disease not amenable to percutaneous treatment, or recurrent ischemic events despite optimized medical and percutaneous management. Decisions regarding escalation of care should ideally be made within a multidisciplinary Heart Team.

Follow-up and longitudinal management

Long-term management includes aggressive modification of cardiovascular risk factors, tailored antithrombotic therapy, and clinical follow-up focused on symptom recurrence and ischemic events. Repeat imaging may be considered in selected patients to monitor aneurysm evolution, particularly in those managed conservatively or with large aneurysmal segments. This pragmatic algorithm emphasizes individualized decision-making and acknowledges the current limitations of available evidence, while providing a structured approach applicable to routine clinical practice.

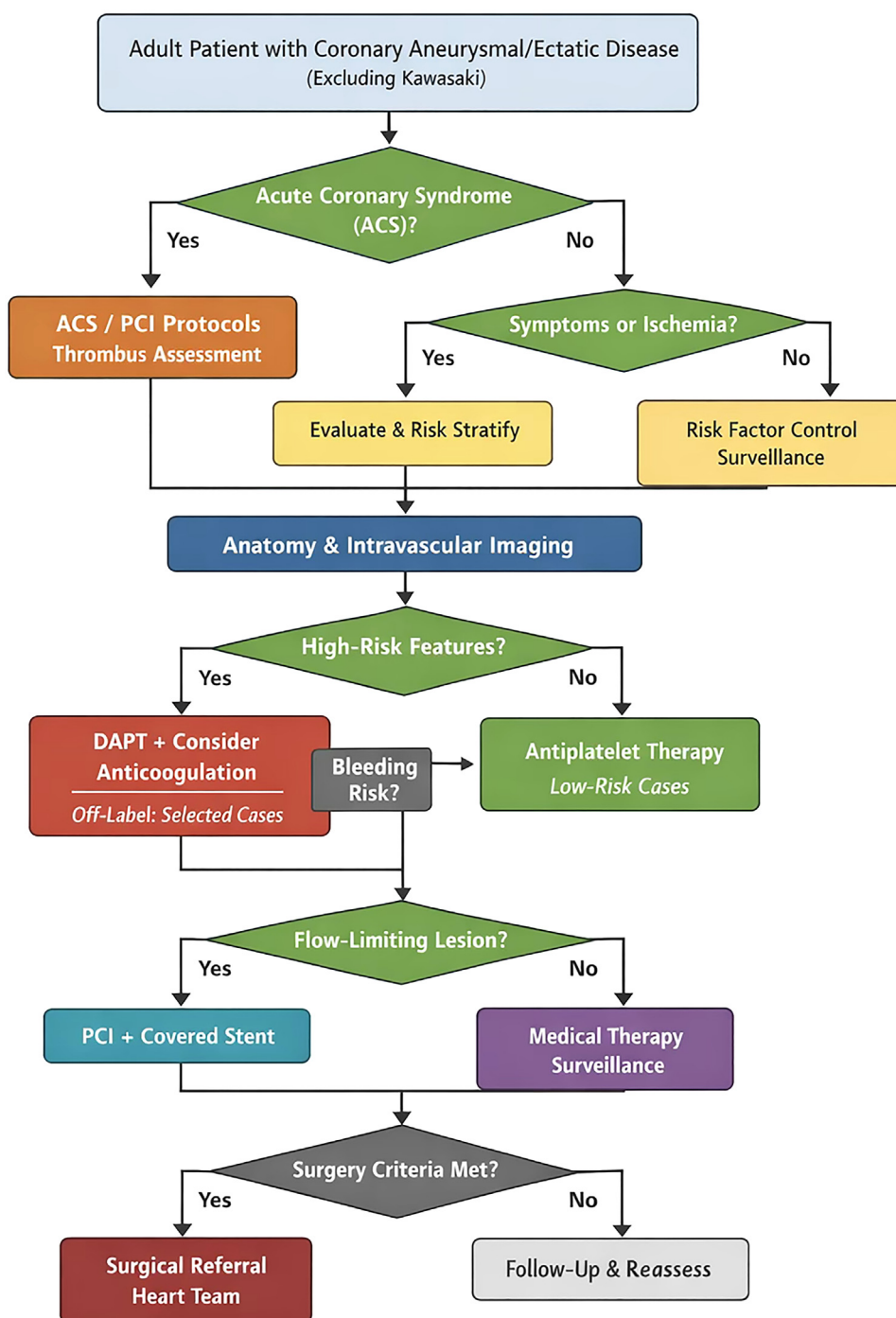


Figure 2. Practical decision-making algorithm for adult coronary aneurysmal disease. This algorithm outlines a phenotype-driven approach to the management of adult patients with CAA/CAE, excluding Kawasaki disease–related forms. Clinical presentation, anatomical features, and intravascular imaging findings are integrated to guide antithrombotic therapy, revascularization strategies, and referral for surgical management. Antiplatelet therapy represents the baseline treatment, while oral anticoagulation is considered an off-label option reserved for selected high-risk scenarios, ideally within a multidisciplinary Heart Team framework. DAPT = dual antiplatelet therapy.

Conclusions and Future Directions

Coronary aneurysmal disease represents a heterogeneous and clinically challenging condition that remains incompletely understood and poorly addressed by current guideline-directed recommendations. Once considered a rare or incidental angiographic finding, coronary artery ectasia and aneurysms are increasingly encountered in contemporary catheterization laboratory practice,

particularly in patients with acute and chronic coronary syndromes. This review highlights that adult coronary aneurysmal disease is predominantly related to atherosclerotic remodeling and is characterized by distinctive pathophysiological features, including vessel wall degeneration, altered coronary hemodynamics, and a prothrombotic milieu. These mechanisms contribute to an increased risk of adverse cardiovascular events and complicate both diagnostic assessment and therapeutic decision-making. Despite a strong

pathophysiological rationale for intensified antithrombotic therapy, the systematic appraisal of available evidence does not support routine oral anticoagulation in unselected patients with coronary aneurysmal disease. Antiplatelet-based strategies remain the cornerstone of medical management, while anticoagulation should be regarded as an off-label option reserved for carefully selected high-risk scenarios. The lack of completed randomized trials and the heterogeneity of existing observational data underscore the uncertainty surrounding optimal antithrombotic strategies in this population. From a procedural standpoint, intravascular imaging plays a central role in the accurate diagnosis and management of coronary aneurysmal disease, guiding percutaneous interventions and informing decisions regarding conservative versus invasive strategies. Surgical treatment remains an important option for selected patients with complex or high-risk anatomical features and should be integrated within a multidisciplinary Heart Team approach. Future research should focus on the development of prospective registries and dedicated clinical studies to better define the natural history of coronary aneurysmal disease and to identify patient subgroups that may benefit from tailored antithrombotic and revascularization strategies. Until such data become available, a phenotype-driven and individualized approach, as proposed in the present review, represents the most rational framework for managing this complex condition in clinical practice. The results of ongoing randomized trials, including studies evaluating rivaroxaban-based strategies in coronary artery ectasia, are eagerly awaited and may substantially refine future guideline recommendations and phenotype-based management algorithms.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRedit authorship contribution statement

Giulia Alagna: Writing – original draft, Visualization, Validation, Methodology, Data curation, Conceptualization. **Francesca Campanella:** Writing – review & editing, Visualization, Validation, Data curation. **Lucio Giuseppe Granata:** Writing – review & editing, Visualization, Validation, Methodology, Investigation, Formal analysis, Data curation. **Pierre Sabouret:** Writing – review & editing, Validation, Supervision, Project administration, Methodology, Formal analysis. **Alessandro Lupi:** Writing – review & editing, Visualization, Validation, Project administration, Methodology, Funding acquisition, Data curation. **Giuseppe Andò:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Project administration, Methodology, Funding acquisition, Data curation.

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