CORONARY ECTASIA ANEURYSM (CEA)

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CORONARY ARTERY ECTASIA/ANEURYSM (CAE)

- Relatively common entity
  - inappropriate dilatation of the coronary vasculature
- Etiology unknown
  - Multifactorial:
    - genetic predisposition
    - risk factors for coronary artery disease
    - abnormal vessel wall metabolism.

INTRODUCTION

- Coronary artery ectasia (CAE) represents a form of atherosclerotic coronary artery disease seen in 1.5- 8% of patients undergoing coronary angiography.
- The presence of ectatic segments produces sluggish blood flow, with exercise- induced angina and myocardial infarction, regardless of the severity of coexisting stenosis

Coronary Artery Aneurysm

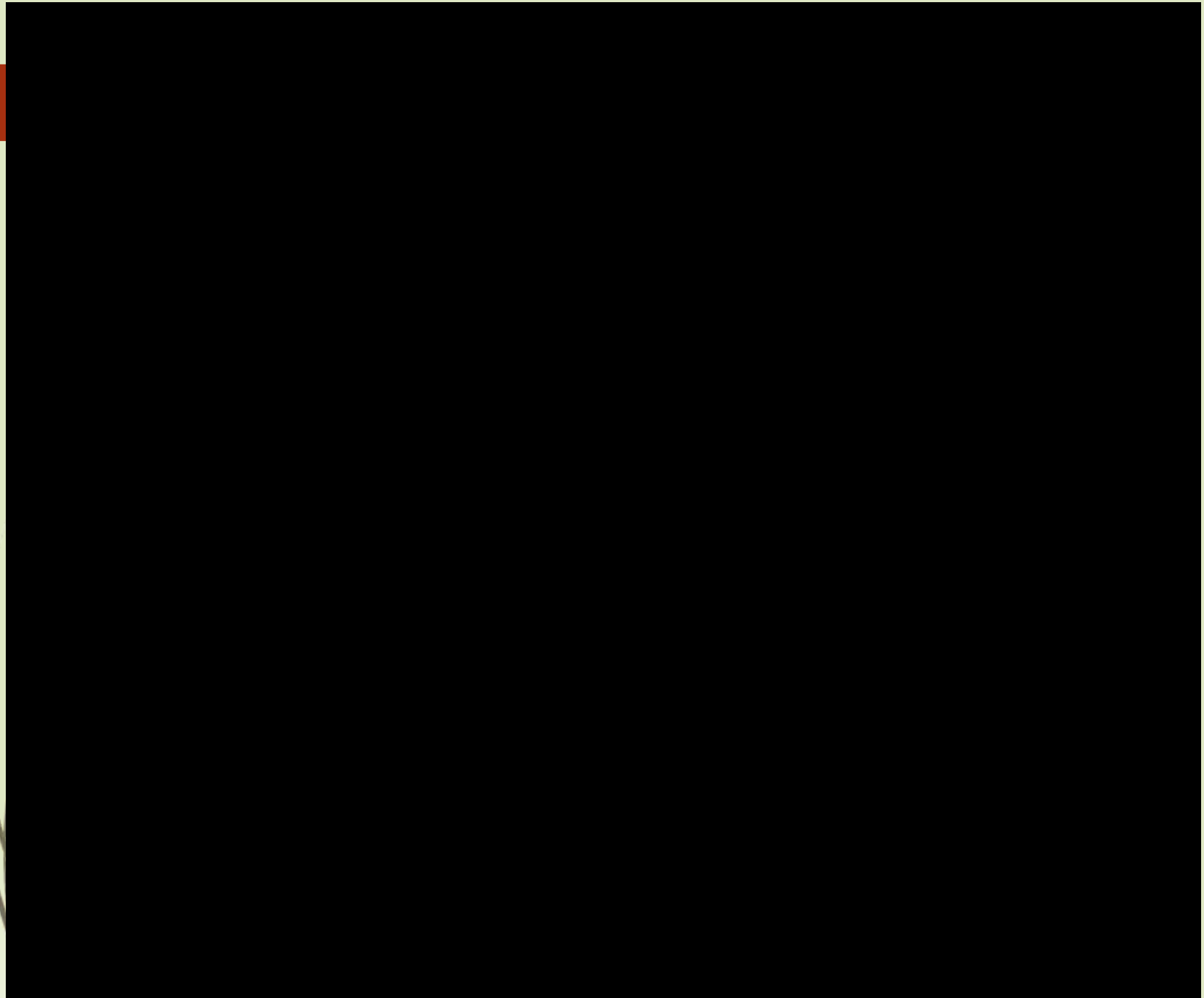
- defined as a localized, irreversible dilatation of the blood vessel lumen that exceeds the diameter of the adjacent normal segment by more than 1.5-fold (Falsett & Carrol, 1978; Swaye et al. 1983; Syed & Lesch, 1997).
- In contrast, ectasia is used to describe **a diffuse dilatation of coronary arteries that involves 50% or more of the length of the artery**; this classification is made according to the appearance and number of vessels involved (Markis et al, 1976).

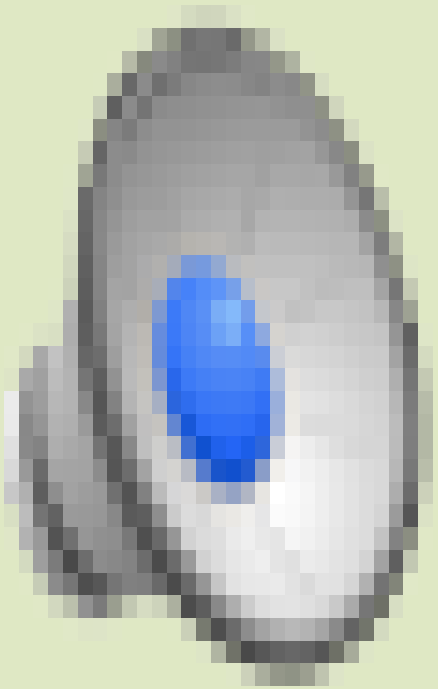
CEA, or aneurismal coronary artery disease, is defined as

Suzuki *et al.* recommended expanding Hartnell's definition to include that those arteries that are are 1.5 or more times greater than the the mean diameter of the proximal and distal portions of the ectatic area.

HISTORY

> *The first case report of a coronary artery aneurysm was by Bourgon (1812) who described the postmortem finding of a right coronary artery dilatation in a patient who died suddenly.'*





Classification

TABLE I Coronary artery ectasia classification

I.	Diffuse ectasia involving two or more vessels
II.	Diffuse ectasia involving one vessel and localized ectasia involving another
III.	Diffuse ectasia involving one vessel only
IV.	Localized or segmental ectasia only

Types I to IV are in order of decreasing frequency.

Source: Ref. No. 4.

Table-2: Percentage of individual coronary vessels affected by ectasia Modified Markis Classification.

Vessel	Focal (%)	Diffuse (%)	Total involvement (%)
LAD	7	8	15%
RCA	48	40	88%
LCX	16	19	35%
L.M	5	3	8%

Classification of CAAs		
Characteristics	**Categories**	**Luminal diameter of the aneurysm**
Shape	Saccular Fusiform	Maximum transverse diameter > longitudinal dimension Longitudinal dimension > maximum transverse diameter
Vascular wall integrity	True aneurysm Pseudoaneurysm	All vascular layers present Loss of the vascular wall integrity
Topographical extent	Type I Type II Type III Type IV	Diffuse dilatation of two or three vessels Diffuse dilatation in one vessel and localized in another Diffuse dilatation of one vessel only Localized or segmental dilatation

Modified from Antoniadis et al., 2008; Díaz-Zamudio et al., 2009.

Table 1. Morphologic and topographical classification of aneurysms and ectasias observed in coronary arteries.

ETIOLOGY

MOST COMMON >50%

Atherosclerosis
Kawasaki disease
Congenital
Arteritis (polyarteriritis nodosa, syphilis, systemic lupus erythematous, Takayasu arteritis disease, Behçet´s disease)
Mycotic
Dissection
Chest trauma
Drugs
Connective tissue disorders (SLE, rheumatoid arthritis, ankylosing spondylitis, scleroderma)
Hereditary collagen defects (Marfan and Ehlers-Danlos syndromes)
Metastatic tumor
Coronary angioplasty (balloon, laser atherectomy, stent implantation, directional coronary atherectomy, pulsed laser coronary angioplasty and brachytherapy)*

Modified from Syed & Lesch, 1997. SLE: systemic lupus erythematosus. *Mostly pseudoaneurysms.

Table 3. Etiology of Coronary Artery Aneurysms.

Atherosclerosis

Atherosclerosis is the most common cause of CEA causing morbidity and mortality worldwide.

characterized by chronic inflammatory and intimal lesions, called atheromas or

fibrofatty plaques, which protrude into the lumen, weaken the underlying media and

undergo a series of complications affecting primarily elastic arteries and larger and medium sized muscular arteries, such as coronary arteries (Libby, 2002)

Histopathological Findings

an intimal proliferation with spreading of plaque material into the vessel media leading to destruction.

As a patho mechanism for the development of pre- and poststenotic CEA, →

→ an increase in wall stress to which the artery is exposed

with the thinning and atherosclerotic destruction of the vessel media resulting in progressive vessel dilation has been proposed.

Extra cardiac vessel dilations were reported by Daoud et al. and Stajduhar et al., who described an over proportional coincidence of coronary artery aneurysms with aneurysms of the abdominal aorta.

Daoud AS, Pankin D, Tulgan H, Florentin RA. Aneurysms of the coronary arteries, report of ten cases and review of the literature. Am J Cardiol 1963;11:228–37

Thrombogenesis in CEA

- The combination of a proximal stenosis and an immediately adjacent region of slower coronary blood flow within an aneurysm represents a powerful stimulus promoting thrombus formation.
- Additionally, turbulent poststenotic flow within the coronary aneurysm likely promotes endothelial activation.
- the presence of chronic thrombosis within an aneurysm may also promote thrombogenesis by providing clotting precursors and fibrin as nidus for new clot.
- Hence, CEA thrombosis is mediated both from platelet and endothelial derived pathophysiologic mechanisms and which is further propagated in the presence of chronic thrombus .

Clinical symptoms and pathophysiological explanations.

- The presence of aneurismal/ectatic segments due to their sluggish or turbulent blood flow, associated with increased incidence of typical exercise-induced **angina pectoris and acute coronary syndromes ,** regardless of the severity of coexisting stenotic coronary disease.

- This is due to the repeated dissemination of micro emboli to segments distal to the ectasia, or to thrombotic occlusion of the dilated vessel

- Slow blood flow in the coronary artery may also be a causative factor.

Clinical symptoms and pathophysiological explanations.

- **Micro embolisms with consecutive disturbance of coronary perfusion** may account for **ventricular arrhythmias and even sudden cardiac death**;
- The occlusion of major coronary vessels may result in acute ventricular dysfunction due to acute myocardial infarction.

Therapeutic Management

- No specific guidelines
- Customized treatment

The coronary morphology of CEA is heterogeneous; for this reason, pharmacological, interventional and surgical therapy specific to the cause is required.

In addition to the determination of the cause, therapeutic management depends on possible or manifest complications.

Medical management

- The application of **platelet inhibitors** as a prophylaxis against ischemic syndromes attributed to fibrin thrombus formation and micro emboli showering is crucial in all forms of CEA.
- **Anticoagulation with coumarin** has been propagated, although a therapeutic superiority compared with aspirin has not yet been evaluated and **not established by evidence based medicine**
- Study by **Krueger et al (1)** strongly suggest that **NTG has no therapeutic benefit in "dilated coronaropathy,"** on contrary **it may lower the ischemic threshold**. Consequently, the administration of nitrates in "dilated coronaropathy" **should be avoided**
- a reasonable therapeutic approach might be the administration of **beta-blockers due to their negative chronotropic effect and reduction of myocardial oxygen consumption** in the absence of vasodilation

1.Krueger et al. Myocardial Ischemia in Dilated CoronaropathyJACC November 1, 1999:1461–70.

- In cases of CEA where coronary ischemia persists despite medical optimization, surgical or percutaneous revascularization may be required.
- Multiple authors have reported the excellent acute and long-term results of balloon angioplasty as well as USE OF BMS/DES in lesions adjacent to coronary aneurysm ;
- Special attention should be paid to the need for adequate stent expansion and wall stabilization in these vessels.
- The implantation of covered STENTS versus BMS/DES offers a superior acute angiographic result, excluding the ectatic segment, but the long-term benefit has not been adequately proven.

?$$ Ochiai M, Yamaguchi T, Taguchi J, et al. Angioplasty of stenoses adjacent to aneurysmal coronary artery disease. Jpn Heart J. 2007; 31: 749-757

STENTYS SYSTEM : XPOSITION S (ANIMATION)

STENTYS SYSTEM : XPOSITION S (ANGIO. REP.)

CEA AND ACS

- **PCI** in the instance of thrombosis, may represent **several technical challenges.**
- Two important potential complications include **distal embolization of thrombus and stent malapposition.**
- **Yip et al** reported **no-reflow phenomenon** (defied as ≤TIMI-2 flw) in **68.2% patients** and distal embolization after primary PCI in **70%in patients with visibly thrombosed CEA.**
- **Placement of a stent** within an aneurysmal segment poses a **technical challenge**, since apposition of stent struts to a vessel of large and irregularly-variable caliber may not be feasible.
- Leaving **unopposed stent struts** — whether bare metal or drug eluting —
  may represent a nidus for thrombosis.

Yip H-K, Chen M-C, Wu C-J, et al. Clinical features and outcome of coronary artery aneurysm in patients with acute myocardial infarction undergoing a primary percutaneous coronary intervention. *Cardiology.* 2002;98(3):132-140

Thrombogenesis/ACS in CEA

PTFE: Polytetra-fluoroethylene/Polyester (Covered) STENT AND CEA

- The use of PTFE-covered stents may also pose unique challenges:
- deployment of a covered stent in CEA may **result in occlusion of branch arteries that originate** within the subtended aneurysm;
- **Incomplete coverage of the aneurysm** may result in **persistent "leak" into the aneurysm sac**; and, PTFE-covered coronary stents pose risk for thrombosis **or in-stent restenosis**.
- **Stent length and aneurysm caliber (diameter >10mm)** have also been reported as independent risk factors for future restenosis with PTFE-covered stents

> Many authors suggests that the **exclusion of the aneurysm with a PTFE-covered stent graft would eliminate sluggish flow** through the (previously) aneurysmal segment, and would **reduce the likelihood of aneurysm thrombosis**, enlargement, or future rupture.

PCI OF GIANT ANUERYSM WITH PTFE STENT GRAFT

- Surgery may be indicated in the presence of aneurysms **three to four times the original vessel diameter (giant CEA),**

- involvement of the **left main, bifurcation lesions, or multivessel involvement**.

- Surgical treatment entails **coronary artery bypass with or without aneurysm ligation or resection**

Coronary Artery Aneurysms in Acute Coronary Syndrome: Case Series, Review, and Proposed Management Strategy

Nathan Boyer, MD[1]; Rajesh Gupta, MD[2]; Alex Shevchuck, MD[4]; Vindhya Hindnavis, MD[1]; Seth Maliske[3]; Mark Sheldon, MD[4]; Douglas Drachman, MD[2]; Yerem Yeghiazarians, MD[1]

Antiplatelet therapy should be initiated immediately upon the identification of CAA with ACS if not previously administered.

Anticoagulation with intravenous **weight-based unfractionated heparin (UFH) or subcutaneous LMWH** should be added to antiplatelet therapy.

If copious thrombus is noted within CAA during angiography, recommend additional **consideration of glycoprotein IIb/IIIa inhibitor infusion for 24-48 hours.**

Glycoprotein IIb/IIIa infusion should be accompanied by close monitoring for thrombocytopenia, anemia, or bleeding.

1. Initial Management: Antiplatelet and Anticoagulant therapy

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Conservative Versus Invasive Strategy

Most patients presenting with ACS in the context of culprit CEA identified at coronary angiography **should be managed conservatively with antiplatelet and antithrombotic therapies**.

Similar to the AHA/ACC guidelines for the management of patients with **unstable angina and non-ST segment elevation myocardial infarction ,Pts with CEA should be treated invasively**

Recommended ; that patients with the following be considered for revascularization:

(1)TIMI 0 or 1 flow in the aneurysmal vessel;
(2) Patients with recurrent angina or ischemia;
(3) Sustained ventricular tachycardia; or
(4)Hemodynamic instability including sustained hypotension

Invasive Strategy: Percutaneous Versus Surgical Revascularization

- a soft-tipped coronary guidewire should be manipulated meticulously through the CAA, taking care not to coil the wire tip in the body of the aneurysm;
- distal embolic protection could be considered, particularly in the context of copious thrombus;
- aspiration Thrombectomy is often necessary to reduce thrombus burden and improve coronary flow.
- The use of IVUS or optical coherence tomography (OCT) may further define the lesion characteristics including vessel diameter, presence of thrombus, and relationship of the CAA to branch vessels.

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From our review of the available literature and ACC/AHA ACS guidelines, we propose that patients with the following findings be referred for surgical revascularization:

- CAA involving the left main coronary artery;
- Multivessel CAD;
- Giant CAA (dilation that exceeds the reference vessel diameter by 4 times);
- CAA involving bifurcation of significant side-branch vessel; or
- Other separate indications for cardiothoracic surgery unrelated to CAA.

Discharge Stratigies :Antiplatelet and Anticoagulation

- In patients with giant CEA or with other indications for chronic systemic anticoagulation, chronic therapy with **aspirin 150 mg daily and warfarin to target an international normalized ratio (INR) of 2.0–2.5 Should be preferred**
- In the majority of other cases, however, we recommend dual-antiplatelet therapy with **aspirin 150 mg daily and clopidogrel, prasugrel, or ticagrelor,** regardless of whether conservative or invasive strategy is pursued.
- The duration of dual-antiplatelet therapy in patients presenting with ACS and CAA is unclear, and should be tailored to the patient, lesion, and treatment approach.

> The role of the **novel oral anticoagulants for treatment of CAA is unknown at this time** and is not likely to be studied given the paucity of patients with CAA and ACS.

> **Off-label use may be considered following discussion with the patient**, including careful consideration of **potential risks and benefits**.

THANK YOU!