CORONARY ARTERY ECTASIA: A REVIEW

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ABSTRACT

"*Coronary artery ectasia* (CAE)" is an uncommon kind of coronary heart disease that some people experience. It stands out due to its width being almost twice as larger than the healthy coronary artery next to it and its length being dilated by more than one-third. Positive stress tests, Angina pectoris, and acute coronary syndrome can all be signs of CAE in the absence of severe coronary constrictions. In an ectatic artery, thrombus development may be brought on by a vasospasm, distal embolization, or vascular rupture. Aspirin and other antiplatelet medications are the foundation of management for CAE. On the basis of occurrence of associated obstructive coronary artery condition and a patient's potential for bleeding, anticoagulants are utilized to prevent thrombus development. Hence those affected are encouraged to take statins as a primary preventative measure because the frequent cause of CAE is atherosclerosis. "*Angiotensin-converting enzyme* (ACE)" inhibitors may be administered to people with elevated blood pressure due to their anti-inflammatory characteristics. Calcium channel blockers and Beta-blockers may be helpful if hypertension and coronary vasospasm co-occur. Nitrates are typically not advised because they could make symptoms worse. The avoidance of thromboembolic consequences and surgical or percutaneous revascularization are additional CAE therapeutic options. The linked coronary artery's severity affects the CAE prognosis.

Keywords: Covered Stent, Ectasia, Coronary Aneurysm, Anticoagulation, Vasospasm

INTRODUCTION

Morgagni first identified "*Coronary Artery Ectasia* (CAE)" in 1761. CAE is a very uncommon coronary angiographic discovery, with regional variations in occurrence ranging from 1.5%- 5%. [1-3] Whereas aneurysm defines a more focused dilatation, ectasia describes a broad dilatation of a coronary artery. Coronary ectasia is a condition where the diameter of a coronary artery is greater than one and half times that of the maximum diameter of a nearby normal artery. We still don't fully comprehend CAE's pathogenesis, despite the many explanations that have been suggested. Similarly, there is disagreement over the natural

history and treatment of this condition due to a dearth of evidence. Along with being a clinical collection of "*Coronary Artery Disease* (CAD)", which includes myocardial ischemia and acute coronary syndromes, CAE is a clinical variation of "*Coronary Artery Disease* (CAD)".[4,5] This assessment covers CAE management and status.

CLASSIFICATION

Established on the level of participation, Markis et al.[6] divided CAE into 4 kinds. The shape and degree of the coronary arteries' involvement are frequently used to categorize it. **Box 1**

Box 1. Classification based on level of involvement. (adapted from Devabhaktuni et al[2])

Type 1

Diffuse ectasia with aneurysmal lesions in two vessels.

Type 2

Diffuse ectasia in one vessel and discrete ectasia in another.

Type 3

Diffuse ectasia in one vessel.

Type 4

Discrete ectasia in one vessel.

EPIDEMIOLOGY

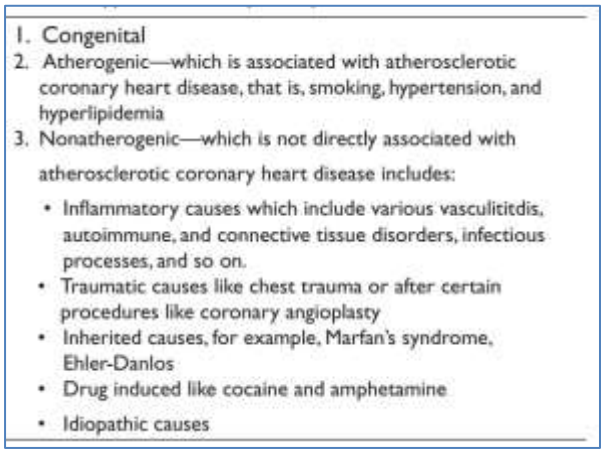
Several studies have examined the prevalence of CAE on a global level. CAE has been discovered in datasets from 0.22 to 1.4% of autopsy datasets and 3.0% to 8% of angiographic datasets, respectively. Whether the condition is widespread or isolated, the entire length of a coronary artery may be impacted. [2,6] South Indian studies[7], showed the prevalence of pure CAE without CAD is 1.9%, while the incidence of angiographic CAE is almost 4.5%.[8] In a North Indian study, it was discovered that the incidence of CAE was approximately 10% among those with ischemic heart disease. The occurrence of isolated coronary ectasia was reported to be 1.05% in an Indian study, but the prevalence of CAE was assessed to be around 5.45%.[9] In another Indian research, 4.1% of 3,014 consecutive coronary angiographies exposed the occurrence of CAE in the angiography.[2]

ETIOLOGY

The cause of CAE is not entirely clear. More than half of occurrences in adults are attributed to atherosclerosis, whereas Kawasaki illness is the most frequent etiologic reason in children and adolescents. Teenage male children are more prone to get CAE. The emergence of CAE has also been linked to genetic factors. ACE's DD genotype variation appears to be a substantial risk factor for the onset of CAE. Homocysteine levels may be a factor in the breakdown of the medial arterial layer by triggering "*Matrix Metalloproteinase* (MMP)" 2 and increasing serine proteinase activity in arterial smooth muscle cells.[10-14] CAE and high

blood pressure are related. A Meta analytic research concluded that the risk of CAE was low in those with DM. This may be connected to the downregulation of MMP and the detrimental remodelling brought on by atherosclerosis. Those with CAE appear to smoke more frequently than those with CAD. Cocaine use, regardless of smoking, also predicted CAE. Low "*Endothelial Shear Stress* (ESS)" in particular makes coronary arteries more susceptible to atherosclerosis, the creation of fragile plaque, and aneurysm formation.[15-17] **Box 2**

Box 2: types of CAE (Ozcan OU et al[5])



1. Congenital
2. Atherogenic—which is associated with atherosclerotic coronary heart disease, that is, smoking, hypertension, and hyperlipidemia
3. Nonatherogenic—which is not directly associated with atherosclerotic coronary heart disease includes:
   - Inflammatory causes which include various vasculititdis, autoimmune, and connective tissue disorders, infectious processes, and so on.
   - Traumatic causes like chest trauma or after certain procedures like coronary angioplasty
   - Inherited causes, for example, Marfan's syndrome, Ehler-Danlos
   - Drug induced like cocaine and amphetamine
   - Idiopathic causes

PATHOGENESIS

The pathophysiology of ectasia has been explained by the theories listed below.[2,18-23]

1. Hemodynamic theory states that focused ectasia is caused by pressure irregularities exceeding the stenosis threshold and kinetic energy conversion to potential energy. High blood flow velocity causes endothelial damage and poststenotic vasodilation due to increased shear stress at the stenosis. Low ESS accelerates early atherosclerotic plaque to expansive remodelling (positive remodelling ectasia) in vivo.

2. Vascular remodelling: Coronary ectasia is a sort of overly positive remodelling of the artery wall that takes place during the first phases of plaque formation and following a plaque rupture event. This extension could lead to low shear stress, which would keep the cycle going.

3. Atherosclerosis causes inflammation in the arteries. Inflammatory cells from the intima damage the tunica media layer, causing ectasia. Hemodynamic variables and inflammation are the main causes of atherosclerosis and CAE. Cytokines inflame the artery wall's three layers, causing CAE. CAE patients have greater CRP and plasma interleukin-6 than persons with normal coronaries. Ectasia patients' serum had more cell adhesion molecules that help inflammatory cells transmigrate. Neutrophils are also implicated.

4. Increased NO-mediated vasodilation is particularly common in nonatherosclerotic CAE patients. NO and other pollutants weaken and ectasia artery walls via changing the extracellular matrix. Ectasia risk increases in those exposed to herbicide sprays with

acetylcholine esterase inhibitors. This medication boosts acetyl choline-mediated NO generation.

5. *Genetic*: Ectasia is linked to ACE polymorphism MMP-3 5A allele disruption. HLA linkage was established using "HLA-DR B1 13, DR16, DQ2, and DQ5" genes..

6. Generalized vascular disorders: Associating CAE with abdominal and ascending aortic aneurysms suggests a bigger arterial wall issue (deficiency). CAE is linked to "*Pampiniform Plexus, Leg Vein Varicosities, & Coronary Vein Varicosities".*

CLINICAL FEATURES

CAE is accompanied by traditional coronary risk factors such obesity, hypertension, smoking, and stress. CAE is frequently found by chance during an angiography for another cardiac condition. It is unknown what CAE looks like clinically. Stable angina is the most common symptom in those with CAE and coronary stenosis. Positive treadmill exercise tests and acute coronary syndrome are both potential diagnosis. The CAE is more usually linked to atherogenic etiology. Angiographically, the degree of ectasia and the backflow phenomenon in an ectatic left anterior descending artery were the most significant predictors of ischemia during exercise testing in patients with isolated CAE. Both non-ST elevation MI and ST-elevation MI can have changed blood flow as a result of the ectatic segment's thrombus blockage or distal embolization. The risk of problems such as thrombus development, distal embolization, shunt formation, and rupture is unknown. By piercing the right atrium, right ventricle, or coronary sinus, CAE can also result in left-to-right shunts.[1,24-26]

DIAGNOSIS

Prior to the availability of more recent diagnostic techniques like coronary "*Computed Tomography Angiography (CTA)*" and coronary "*Magnetic Resonance Angiography* (MRA)", coronary angiography was the method of choice. Angiograms reveal irregularities in blood flow filling and washout, which are related to how severe ectasia is. Angiographic indicators of turbulent and stagnant flow include local dye deposition in the dilated coronary segment, segmental reverse flow, and delayed ante grade dye filling. With the added benefit of being a noninvasive procedure, coronary MRA has been demonstrated to be comparable to quantitative coronary angiography. When combined with coronary flow data, coronary MRA may provide further insightful information about the potential for thrombotic occlusion of the aneurysmal arteries. Moreover, MRA can be utilized effectively for these patients' follow-up because it is a noninvasive, non-radiating procedure. Another noninvasive method for diagnosing CAE is coronary CTA. Because of advancements in radiation dose with current procedures, coronary CTA can be recommended as a technique of choice for patient follow-up. The "*Intra Vascular Ultra Sound* (IVUS") is a superb tool for measuring luminal size and describing alterations to the artery wall. IVUS accurately distinguishes between real and fake aneurysms brought on by plaque rupture. The distinction between empty plaque cavities and CAE is crucial from a clinical standpoint because false aneurysms can result in acute coronary syndromes.[27-30]

MANAGEMENT

General Measures

Changes in lifestyle, such as a balanced diet, giving up tobacco use, getting regular exercise, managing stress, etc. as well as the monitoring of the life style diseases.[1]

Pharmacy Management

Due to a lack of evidence-based medicine, it is a contentious subject. Due to the common coexistence of CAE and obstructive coronary lesions in patients, as well as the reported incidence of myocardial infarction, even in individuals with isolated coronary ectasia, aspirin was advised for all patients. Amplified platelet activation is also seen. Warfarin was recommended as the main chronic anticoagulant due to ectatic flow disturbances.[2,30] But, because this treatment hasn't been tried prospectively, it can't be suggested unless it's backed by more research, and until then, decisions about it should be made weighing risk versus benefit. Statins and ACE inhibitors slow disease progression. ACE inhibitors may be helpful in the prevention of CAE evolution that depends on the relation between genetic condition of the patient and CAE, however this has not yet been proved. Statins may be helpful as they reduce the MMP expressions. Nitro glycerin and nitrate derivatives should not be administered to CAE patients since they may cause angina pectoris.[31]

"Percutaneous Intervention (PCI)"

PTCA can be performed in patients where medical management is ineffective. A balloon-expandable "Polytertafluoroethylene (PTFE)" stent is beneficial for percutaneous coronary aneurysm management and exclusion.[2,31] Symptomatic patients who are not PCI candidates may benefit from surgery, which involves bypass grafting of the affected coronary arteries and CAE excision or ligation. These methods yield positive results.

Prognosis

Long-term CAE outcomes are uncertain. CASS registry patients with and without CAE had similar survival rates. After this, several investigations came to the conclusion that CAE is merely a subtype of atherosclerosis that carries no additional risk because they were unable to demonstrate a mortality variance between CAD and CAE.[2,33]

CONCLUSION

In between 3 and 8% of coronary angiography procedures performed for diagnostic purposes, CAE, a kind of atherosclerosis, manifests itself. There is presently no accepted treatment strategy for CAE. Every piece of prior research has weaknesses, such as a limited sample size, no control group, and a lack of randomization. The rarity of this ailment makes it difficult to conduct significant randomized clinical trials. Surgical/ percutaneous revascularization, antithrombotic management, and Anti-ischemic medications, are available as treatment options. To establish the most effective method for managing CAE, more testing is required.

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