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# Recurrent Angina and Coronary Artery Aneurysm: A Case Report

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**Abstract** Coronary artery ectasia (CAE) or aneurismal coronary artery disease is a rare dilatation of a segment of the coronary artery to more than 1.5 times of the normal adjacent segments. CAE is mainly caused by atherosclerotic disease and is usually asymptomatic. The authors present an 88 year-old-male with underlying coronary artery disease and repaired abdominal aortic aneurysm (AAA) who presents with recurrent angina and was diagnosed with CAE by coronary angiography. Patient was managed medically with aspirin, statin, beta blocker and anticoagulant.

***Keywords:*** *coronary artery ectasia, aneurismal coronary artery disease, CAE, angina, aortic aneurysm*

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## 1. Introduction

Coronary artery ectasia (CAE) or aneurismal coronary artery disease is defined as the dilatation of a segment of the coronary artery to more than 1.5 times of the normal adjacent segments [1,2,3]. This represents a rare subset of coronary artery disease which is only seen in 3-8% of patients referred for coronary angiography. [2]. Coronary aneurysms can present in two main forms. They can be either diffuse, or localized. When dilatation in artery involves the entire vessel, it is referred to as coronary artery ectasia (CAE) rather than aneurismal disease of the coronary artery. The etiology of CAE is multifaceted but is usually attributed to atherosclerosis (50%) or congenital anomaly (20-30%). While it is mainly associated with coronary artery disease, CAE has also been associated with connective tissue disease including scleroderma, Ehlers-Danlos, syphilis aortitis, Kawasaki disease, and various ANCA- related vasculitis in 10-20% of the cases [2,4]. While a subset of patients with pure CAE might have a benign course, about 40% of the patients have a higher risk of developing repeated myocardial infarction. While the pathophysiology is not well understood, a few different mechanisms have been suggested including repeated dissemination of micro emboli, or significant thrombotic occlusion of the dilated part of the vessel. This is likely from the turbulent blood flow that occurs in these parts of the vessel [5,6]. As there is no general consensus in terms of treatment, these patients must be monitored closely as the degree of their CAE can lead to serious cardiovascular adverse effects. We present an 88-year-old male with a history of repaired abdominal aortic aneurysm who presents with recurrent chest pain and was diagnosed with CAE on coronary angiogram.

## 2. Case Report

88-year-old male with underlying end stage renal disease (on hemodialysis), hypothyroidism, chronic anemia, history of ST elevated myocardial infarction (STEMI), ischemic cardiomyopathy, hypertension, and history of repaired abdominal aortic aneurysm (AAA) who presented to the emergency department (ED) with recurrent chest pain and shortness of breath. Chest pain was worse on exertion and relieved by rest. Patient had a troponin of 0.23 which trended up to 0.39 and 0.51. EKG showed ST segment depression in lead V5 and T wave inversions in lead AVL. Patient was admitted to rule out acute coronary syndrome.

On admission, blood pressure was 104/58, heart rate was 81, respiratory rate was 18 and temperature was 97.8. Sodium was 140meq/l, potassium 4.7 meq/l, chloride 100, bicarbonate 27; glucose 94mg/dl, blood urea nitrogen (BUN) 44mg/dl, creatinine 5.41mg/dl, GFR 11, WBC 9200/ml, Hgb 7.8g/dl, platelet 160,000/ml, and liver parameters were normal. Chest x-ray was unremarkable. Due to a low hemoglobin of 7.8, the patient was transfused 1 unit of packed red blood cells.

The patient had abnormal lexiscan nuclear stress scan. Cardiac catheterization and angiogram showed large multiple aneurysms of the proximal right coronary artery and huge saccular aneurysm at the mid segment of the right coronary artery and distal segment was markedly ectatic and posterior descending artery (PDA) posterolateral branches were partially visualized as the patient had a very slow flow in the right coronary artery. The patient had markedly tortuous right external iliac and common iliac arteries and moderate calcifications noted in the left coronary tree. Left heart pressures were normal.

Ejection fraction was about 30-35%. There was no aortic insufficiency. Right femoral artery angiogram was unremarkable.

Patient was worked up to rule out connective tissue and autoimmune diseases as the cause of the aneurysms. The antinuclear antibodies, antimyeloperoxidase antibodies, antiproteinase 3 antibodies, C-ANCA, P-ANCA were all negative. Patient was placed on aspirin, statin, carvedilol, ranolazine and warfarin for the recurrent angina and to prevent thromboembolism. Patient was referred to a tertiary hospital for management of coronary aneurysms.

## 3. Discussion

### 3.1. Epidemiology

CAE is rare and can be found in 3-8% of patients referred for coronary angiography. [2] CAE incidence is higher in males than females. [1,7] The right coronary artery is the most commonly affected (40% of affected vessels), followed by the left anterior descending (29%) and circumflex artery (24%). [1] CAE or coronary aneurysm is mainly attributed to atherosclerotic disease (50%) while 20%-30% are considered congenital in origin and 10%-20% associated with inflammatory or connective tissue diseases such as scleroderma, Ehlers-Danlos syndrome, Kawasaki disease, syphilitic aortitis, and ANCA- related vasculitis. Kawasaki disease is the commonest cause of CAE in children and young adults. [8,9]

### 3.2. Clinical Presentation and Diagnosis

There are no classic symptoms associated with CAE and patients may present with symptoms associated with the underlying coronary artery disease (CAD), Kawasaki disease or connective tissue disorder. [10] Most CAE patients are asymptomatic although CAE may present with exercise induced angina, myocardial infarction without significant coronary artery stenosis or sudden death. CAE and coronary aneurysms cause sluggish and turbulent blood flow which increases the incidence of thrombus formation and dissemination and increased risk of myocardial infarction (MI) due to thrombotic occlusion of the dilated vessels or emboli dissemination to coronary artery segments distal to the ectasia. [2,11]

Coronary angiography is the main diagnostic technique for diagnosing CEA as it provides information about the size, shape, location and number of aneurysms. [12] CAE can also be diagnosed with intravascular ultrasound (IVUS) which allows for distinguishing between true and false aneurysms caused by plaque rupture which is clinically important as false aneurysms may lead to acute coronary syndrome. [2,12,13] CEA can be detected noninvasively using coronary magnetic resonance angiography (MRA), coronary artery computed tomography (CACT), and echocardiography. [2,12]

### 3.3. Treatment

There is no consensus on management of CAE. [10] The use of aspirin is the mainstay of treatment for isolated CAE. Chronic anticoagulation is probably beneficial and has been suggested by many authors although its benefit in CAE has not been demonstrated in a randomized clinical trial. [2,10,12] Nitrates cause further coronary artery dilatation and has been shown to exacerbate myocardial ischemia and are discouraged in patients with isolated CAE. When CAE co-exists with CAD, the treatment of CAE is the same for CAD alone which includes the use of aspirin, statin and anti-ischemic medications (calcium channel blocker, beta blocker and trimetazidine) as required. [2,10,12] However, to avoid thromboembolism aggressive modification of coronary risk factors has been advocated for all CAE patients irrespective of co-existing CAD. [2,12]

Percutaneous and/or surgical revascularization has been shown to effectively restore myocardial perfusion in patients with coexisting obstructive lesions and significant ischemia despite medical therapy. [2,10,12] Coronary artery bypass grafting is also effective for significant CAD co-existing with CAE segments [2,12]. Surgical resection may be an alternative in patients with risk of rupture from an enlarging saccular coronary artery aneurysm. [10]

## 4. Conclusion

CAE is mainly caused by atherosclerotic disease and is usually asymptomatic. We present a 88 year-old-male with underlying CAD and repaired AAA who presented with recurrent angina and was diagnosed with CAE by coronary angiography. Patient was managed medically with aspirin, statin, beta blocker and anticoagulant.

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