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Clinical and Angiographic Features of Coronary Artery Aneurysm: A Case Series from Tertiary Hospital

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Abstract: ***Coronary artery aneurysm is one of the rare disorders, which occurs in 0.3%– 4.9% of patients undergoing coronary angiography. In adults, the most common cause is Atherosclerosis, which accounts for >90%, while in children Kawasaki disease is responsible for most cases. Recently, with the advent of coronary intervention with the implantation of drug-eluting stents, there are increasing reports suggesting stents cause coronary aneurysms, days months, or years after the procedure. The exact pathophysiology of coronary artery aneurysm is not completely understood till now but it is thought to be similar to that for aneurysms of larger vessels, with the destruction of arterial media, thinning of the arterial wall, increased wall stress, and progressive dilatation of the coronary artery segment. For diagnosis Coronary angiography remains the gold standard tool, providing information about the size, shape, and location and is also useful for planning the strategy of surgical resection. Computed tomography and cardiac MRI can also be useful. The natural history and prognosis of coronary artery aneurysm remain unclear. Despite the important anatomical abnormality of the coronary artery, the treatment options of coronary artery aneurysm are still poorly defined and present a therapeutic challenge. We describe clinical and angiographic features of four cases, which were managed differently followed by a review of the current literature.***

Keywords: coronary artery aneurysm, atherosclerosis, Kawasaki disease, coronary angiography, drug-eluting stents

1. Introduction

Coronary artery aneurysm (CAA) is a rare condition and is defined as dilatation of the coronary artery exceeding 50% of the reference vessel diameter.[1] Giant CAAs are defined when their diameter transcends the reference vessel diameter by greater than four times or if they are more than 8 mm in diameter.[2] the prevalence of CAA is about 0.3%–4.9% of patients undergoing coronary angiography [1] and about 1.4% of postmortem examinations.[1]

CAAs are commonly found in the epicardial coronary arteries, most common vessel involved is the proximal and middle segments of the right coronary artery (RCA) (68%), which is followed by the proximal left anterior descending (LAD) (60%) and left circumflex (LCx) (50%). CAA involving the left main coronary artery (LMCA) is very rare and occurs in only 0.1% of the patients undergoing angiography. [1,3]

Based on dilatation of the coronary artery CAA may be either focal or diffuse, and aneurysms are classified as either fusiform (longitudinal dimension > transverse dimension) or saccular (transverse dimension > longitudinal dimension) in morphology feature. Presentation of CAA is either asymptomatic or with symptoms like angina pectoris, sudden death, fistula formation, and pericardial tamponade. [4]

More recently, with the increasing use of coronary intervention with drug-eluting stents (DESs), there are increasing reports signifying that there is post-coronary procedure coronary aneurysm formation, months or even years after the intervention.[5] CAAs after coronary intervention are very rare, with a reported incidence of 0.3%–6.0% and most aneurysms are in actual fact pseudoaneurysms rather than true aneurysms.[6]

We present four cases of CAA from tertiary centers, which were managed differently followed by a review of the literature regarding etiology, pathogenesis, and the management of this rare entity.

2. Coronary Artery Aneurysm: Definition and Pathophysiology

Coronary artery aneurysm (CAA) is an extremely rare and unusual condition characterized by the abnormal expansion and dilation of the coronary arteries, which are responsible for supplying oxygen and nutrients to the heart muscles.[7] The true prevalence of CAA is not well-established, but it is estimated to affect approximately 0.3% to 5% of patients who undergo coronary angiography, a diagnostic procedure used to visualize the coronary arteries.[8] CAAs are often observed in individuals with different types of arteritis, which is an inflammation of the arterial walls. Inflammation plays a crucial role in the development and progression of CAA. The process begins with endothelial injury, which refers to damage to the inner lining of the blood vessel. This injury triggers a cascade of events, leading to the release of a variety of proinflammatory mediators. These proinflammatory mediators contribute to the weakening of the vessel wall and facilitate the growth of CAA.[9,10] In addition to inflammation, other factors such as antithrombotic therapy, which is a treatment aimed at preventing blood clots, and high levels of reactive oxygen species, which are chemically-reactive molecules that can cause damage to cells, can also contribute to the production of CAA.[11] The process of

endothelial repair is vital in preventing the formation of CAA. However, certain factors can interfere with this repair process. For instance, oxidized LDLs (low-density lipoproteins), also known as "bad cholesterol," and air pollution have been shown to suppress endothelial repair, making individuals more susceptible to CAA formation.[12] In conclusion, understanding the complex mechanisms underlying CAA formation is crucial in the diagnosis, management, and prevention of this rare condition. By identifying the role of inflammation, endothelial injury, proinflammatory mediators, antithrombotic therapy, reactive oxygen species, oxidized LDLs, and air pollution, healthcare professionals can develop targeted interventions to minimize the risk of CAA and its potential complications.[13]

3. Etiology

Atherosclerosis is accountable for >90% of CAAs in adults, whereas Kawasaki disease is responsible for the majority of cases in children.[14] In recent years, CAA have been described as a sequel of coronary angioplasty. This was first stated by Holmes *et al*. [15] following a balloon angioplasty. CAA have also been described after placement of DESs.[16]

Table 1: Lists the different etiologies which have been postulated for CAAs.
Causes of coronary artery aneurysm

Causes	Frequency (%)
Congenital	17
Acquired	
Atherosclerosis	52
Inflammatory disorders	
Kawasaki disease	17
Takayasu's arteritis	
Giant cell arteritis	
Behcet's disease	
Infectious	
Mycotic aneurysm	11
Septic emboli	
Bacterial	
Syphilis	
Connective tissue disorders	
Marfan's syndrome	<10
Ehlers-Danlos syndrome	
Fibromuscular dysplasia	
Drug-related	
Cocaine	Rare
Amphetamines	
Trauma	Rare
Iatrogenic (e.g., PTCA, stents, atherectomy, angioplasty, laser angioplasty)	0.3-0.6

4. Clinical Presentation

4.1 Symptoms and Signs

Coronary artery aneurysms do not have a distinct clinical presentation or pathophysiology, but patients may present with various combinations of symptoms Patients may be asymptomatic or present with symptoms such as fatigue, dyspnea, and chest discomfort.[17] Larger aneurysms can lead to heart failure and angina/myocardial infarction. Angina, palpitations, and dyspnea are specific symptoms of coronary artery aneurysm.[18]

4.2 Diagnostic Challenges

Coronary artery aneurysm (CAA) is often underdiagnosed due to overlapping symptoms with other cardiovascular disorders.[18] Delayed diagnosis can occur when specific signs and symptoms are absent during attacks. Delays in diagnosis worsen outcomes and shorten survival. Sufficient knowledge and clinical acumen are fundamental for clinicians suspecting CAA. [19,20]

5. Methods

From year 2021 - 2024, 8000 diagnostic coronary angiographies (CAG) were screened, We found 4 cases of CAA. Clinical and angiographic features are evaluated as follows

6. Case Series

6.1 Case - 1

A 60-year hypertensive male presented with Chest pain. He underwent PTCA at an outside hospital to LAD (3 × 20) and LCX (2.75 × 24). He was on Dual antiplatelets, statin, and antihypertensive therapy. After 1 year of post PTCA he developed typical angina for which he underwent a Coronary angiogram (CAG) (Fig. 1) showed aneurysm in Left main and proximal LCX, ostial left anterior descending (LAD) artery 100% occluded. There was no history of fever during the interim period and total leukocyte count (TLC) on admission was 6600/µL. his sugars and HBA1C was within normal range. LDL was 228mg/dl. Echocardiogram (Echo) showed left ventricle ejection fraction (LVEF) of 60%. Patient was treated successfully with operative aneurysmal repair and grafting. Intra operatively there was no pus in situ and cultures from the sac were negative. Currently patient is asymptomatic.

Figure 1: Fluoroscopy images of Coronary angiogram show aneurysmal dilatation of LM and proximal LAD and LCX.

6.2 Case 2

A 58-year male non hypertensive non diabetic presented with Chest pain. He underwent PTCA at an outside hospital to LAD (2.75 × 24) mitigator and RCA with mitigator (3.5 × 48). He was on Dual antiplatelets, statin. therapy. After 1 month of post PTCA he developed typical angina for which he underwent a repeat Coronary angiogram (CAG) (Fig. 2) showed an aneurysm in the Left anterior descending and right coronary artery. There was no history of fever during the interim period and total leukocyte count (TLC) on admission was 6800/µL. his sugars and HBA1C was within normal range. LDL was 239mg/dl. Echocardiogram (Echo) showed left ventricle ejection fraction (LVEF) of 25%. Patient was advised operative aneurysmal repair and grafting. Intra operatively there was no pus in situ and cultures from the sac were negative. Currently patient is asymptomatic.

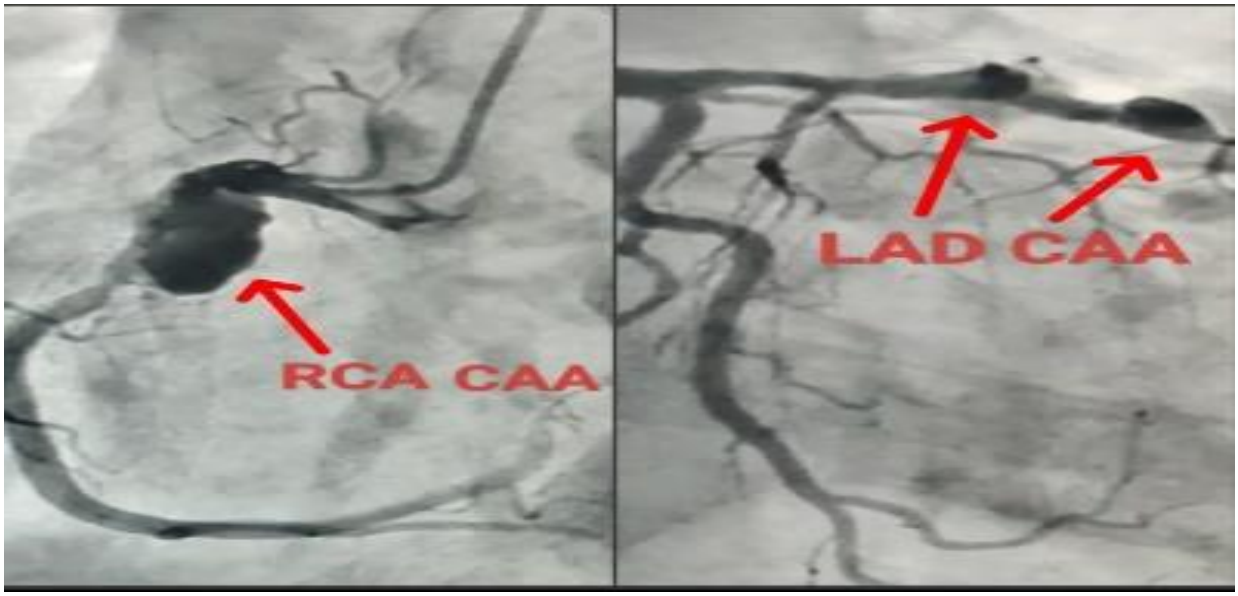

Figure 2: Fluoroscopy images of Coronary angiogram show aneurysmal dilatation of RCA and LAD

6.3 Case 3

A 43-year female hypertensive and diabetic presented with Chest pain. She underwent PTCA at an outside hospital to LAD (3.00 × 24) ultima PC, serolius eluting drug in LAD. She was on Dual antiplatelets, statin, and antihypertensive therapy. After 3 month of post PTCA she developed typical angina for which she underwent a repeat Coronary angiogram (CAG) (Fig. 3) showed giant aneurysm in Left anterior descending coronary artery. There was no history of fever during the interim period and total leukocyte count (TLC) on admission was 8200/µL. her sugars and HBA1C was within normal range. LDL was 339mg/dl. Echocardiogram (Echo) showed left ventricle ejection fraction (LVEF) of 35%. Patient was advised surgical correction of aneurysm but patient was lost to follow up and expired after 10 days due to heart failure at an outside hospital.

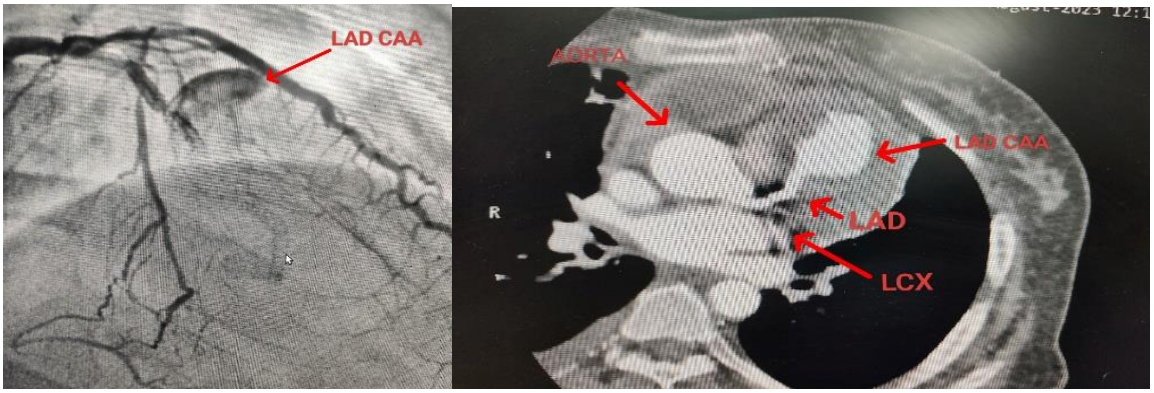

Figure 3: Fluoroscopy images of Coronary angiogram and CT angiography show aneurysmal dilatation of LAD

6.4 Case 4

A 63-year male non hypertensive diabetic presented with Chest pain. He underwent PTCA at our hospital to LAD (3.25 × 34) pronova a sirolimus eluting stent in LAD. He was on Dual antiplatelet, statin, and antidiabetic therapy. After 3 months of post PTCA he developed typical angina for which he underwent a repeat Coronary angiogram (CAG) (Fig. 4) showed giant aneurysm in Left anterior descending. There was no history of fever during the interim period and total leukocyte count (TLC) on admission was 6400/µL. his sugars and HBA1C were within normal range. LDL was 269mg/dl. Echocardiogram (Echo) showed left ventricle ejection fraction (LVEF) of 35%. Patient was advised operative aneurysmal repair and grafting, but patient refused operative procedure and currently on medical therapy.

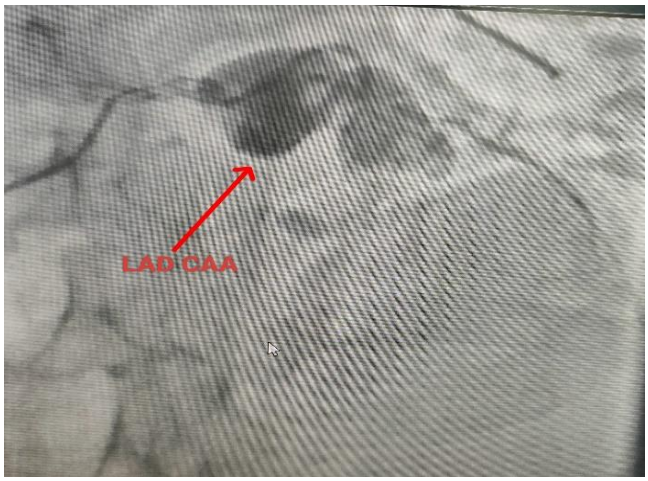

Figure 4: Fluoroscopy images of Coronary angiogram show giant aneurysm in LAD

7. Observation

7.1 Patient Demographics

In our observational case series, the incidence of coronary artery aneurysm is 0.001/ year, and it is more common in males compared to females, the mean age of presentation is 60 +- 5 years.

7.2 Clinical Characteristics

The common presentation of patients with the coronary artery aneurysm is chest discomfort after percutaneous coronary intervention, the duration from index event and presentation varies between 1 month to 1 year. The associated features with coronary artery aneurysms is diabetes, hypertension, dyslipidemia, and smoking. The risk factors that are responsible for coronary artery aneurysm. There was no evidence of infection as a cause of the aneurysm.

7.3 Angiographic Findings

The most common vessel involved is LAD followed by RCA and LCX. The proximal part of the LAD is most commonly involved. An aneurysm is associated with atherosclerotic occlusion of the coronary artery. Two patients in our case series show giant aneurysms with a diameter >8 mm. All the patients have undergone PCI with a sirolimus drug-eluting stent in the past.

8. Treatment Strategies

There are no guidelines specified for the management of coronary aneurysms and management is still ill-defined and present a therapeutic challenge to interventional cardiologists. Most of the information, we have at present, is based on the anecdotal case reports and expert consensus.

The treatment of CAA consists of medical management, percutaneous intervention and surgical resection. however, the appropriate treatment for CAAs depends on the individual clinical situation. In their study, Aoki *et al*.[21] proposed that the treatment of CAA be "individualized" considering the size of the aneurysm, expansion history, risk factors, clinical features, pathophysiology, and symptoms to decide when and if to apply therapy alternatives. We discussed four cases with CAA, which were managed surgically resection, CABG, and with conservative treatment - All management strategies were undertaken according to individual anatomical and clinical information.

8.1 Medical management

The evidence suggests that asymptomatic small CAAs can be managed conservatively.[22] Medical therapy generally consists of attempts to prevent thrombo-embolic complications in patients with aneurysmal arteries who are at increased thrombotic risk through the administration of antiplatelets and anticoagulant medications.[23] Aspirin is the preferred choice; and a second antiplatelet agent (such as clopidogrel, prasugrel, or ticagrelor) is considered unless there is clinical contraindication.[24] Lima *et al*. reported two

cases of left main CAA that were managed conservatively with warfarin and aspirin who remained well at 6 months follow-up.[25] Other studies have demonstrated resolution of angiographic thrombus and outstanding clinical outcomes with the use of intravenous heparin, eptifibatide, and aspirin, and long-term DAPT on discharge.[26]

8.2 Percutaneous coronary intervention

Percutaneous coronary intervention is new option consist of convetionla stent implantation, coil embolization, autologous saphenous vein covered stent grafting and one case has been reported when DES implantation superimposed on a polytetrafluoroethylene (PTFE)-covered stent graft.[27]

PTFE-covered stents have emerged as a new tool for the treatment of CAAs.[28] However, some multicenter randomized trials in comparing expanded PTFE-stent graft with bare metal stents have demonstrated that these stents do not improve clinical outcomes and may be linked to a higher incidence of restenosis and early thrombosis.[29]

Szalat *et al*. [28] reported one of the largest retrospective studies comparing outcomes of patients treated with surgery or with PTFE-covered stents and found that there are similar outcome in both groups i.e. no deaths only few people developed stent thrombosis.

Saccà *et al*. [30] reported a case of successful coil embolization and occlusion of CAA in the terminal LMS in a patient with prior coronary artery bypass graft surgery, including left internal mammary artery to LAD artery.

8.3 Surgery

Surgical management is indicated in symptomatic patients who have obstructive coronary artery disease or evidence of embolization leading to myocardial ischemia and in patients with coronary aneurysm with a risk of rupture.[31] severe coronary disease, CAA near the bifurcation, progressive enlragment of CAA, CAA in left main coronary artery, complication such as fistula, compression of cardiac chambers, Giant CAA. Surgical procedures include resection, coronary artery bypass surgery, aneurysm ligation, marsupialization with interposition graft. Surgical approach for treatment of CAA is considered to be safer and more reliable [32]

9. Prognosis

The prognosis of CAA depends on the size of the aneurysm where small aneurysms have a favorable prognosis with a low risk of mortality [33,34] while giant CAAs have a high risk of morbidity and mortality.[35] About 50% of such giant aneurysms may become obstructed and are associated with arrhythmia, myocardial infarction, or sudden death.

10. Discussion and Conclusion

CAA is a rare disease in coronary arteries, occurring more commonly in men. It tends to progress over time and has a high rate of coexistence with CAD. The most common presentation of CAA is asymptomatic but can lead to devastating complications leading to death. Proper management can prevent acute events. Treatments for CAA include medical management including antiplatelets, anticoagulants, percutaneous coronary intervention, and surgical resection or repair. A prospective study is needed to determine optimal therapy. There is direct correction between aneurysm size and the prognosis of the patient, hence large aneurysms including Giant aneurysms should me managed aggressively while small aneurysms without progression can be managed conservatively with medical management close follow-up.

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