Frontiers in Health Informatics www.healthinformaticsjournal.com
ISSN-Online: 2676-7104
2024; Vol 13: Issue 4 Open Access

Update in Coronary artery ectasia. A mini review

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Cite this paper as: Adnan Taan AlKhafaji, Raed Fanoukh Aboqader Al-Aouadi (2024) Update in Coronary artery ectasia. A mini review. *Frontiers in Health Informatics, 13* (4), 511-518

Abstract

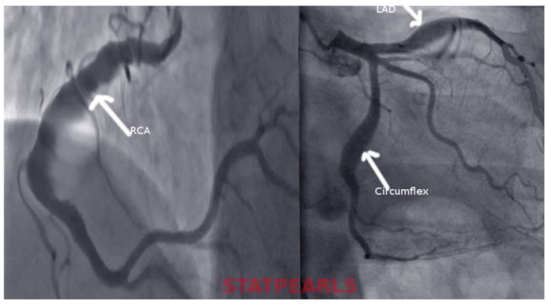
Background: Coronary artery ectasia (CAE) is characterized by the coronary artery dilatation which is abnormal, where the diameter is > one and half times that of normal part**.**
Aim of study: The aim of this paper, is to give an outline and update information on CAE and identifying the prevalence of this unique condition., reviewed disease extention and morphological changes, and investigation of clinical characteristics and complication and its management strategies.
Conclusion: CAE is a type of atherosclerotic events that might happen in 3-8% of angiographic procedures for diagnostic aims. There is currently no fixed treatment guide line. Most of the published paper in this regard has errors with a deficiency of random samples or control, and a small size of sample. Treatment involve antiplatelt, antithrombotic drugs, anti-ischemic drugs in addition to statin in addition to revascularization (surgical or percutaneous).

Keywords: coronary artery ectasia, cornaory artery disease& atherosclerosis

Introduction

Coronary artery ectasia (CAE) is mention as coronary artery dilatation where the dilation is more than 1.5 time of normal vessel.[1]. The coronary artery aneurysm mean specific segment dilatation in comparison to whole segment dilatation in CAC.[2]. Atherosclerosis is been the cause in most of cases (more than 50%)[3]. Morgagni in 1761 had describe coronary artery ectasia [4, 5 & 6]. Despite the information available that numerous reasons have been suggested, the understanding of CAE pathophysiology is quiet unwell. Correspondingly CAE is not only CAD anatomical variety but is also a clinical group of coronary disease including acute coronary syndromes and ischemia of heart[7&8].

The aim is to give an outline and updated information regarding CAE and identifying the prevalence for this unique condition and investigated clinical signs and symptoms in addition to outcomes and management.



(Lamtai M et al 2024)[9]

Coronary Ectasia Classification

CAE was classified into 4 types basing on extent of vessel segment inclusion (Table 1)[10]

Table 1. Calassification of CAE

Type 1. Diffuse ectasia with aneurysmal lesion in 2 vessels
Type 2. Diffuse ectasia in 1 vessel and discrete ectasia in another
Type 3. Diffuse ectasia in 1 vessel only
Type 4. Localized and segmental involvement

The disease CAE was estimated to range from 3-8 % in patients undergone angiography and 0.22-1.4% at autopsy finding. It might be localized or diffuse that might involve all coronary artery[11&12]
In Iraq, the frequency rate of CAE was nearly 3.3 % [13].It can be detected accidently in partients doing coronary angiography for other purposes or during MI contex (14). In MI patients, this may lead to outcome of severe sequele [15]

Etiology

CAE's causes are a little obscure. In adults ,atherosclerosis is the furthermost noticeable cause for around 50 % of CAE cases. But in children and adolescent ,the Kawasaki disease is prominent cause[16,17 &18] It was more recorded in male than female [19]. Genetic factors may also lead to the CAE development. The angiotensin converting enzyme (variation of genotype DD) carry a risk issue in development of CAE [20&21].When there was activation of boosting serine proteinase activity and matrix metalloproteinase (MMP) 2 in cells of arterial smooth muscle [22&23].343. CAE isassociated with increase in arterial blood pressure[24]. It had been recorded that diabetic patients had aCAE lower risk [25]. Patient with CAE seems to have smoking habit extra more repeatedly than those CAD persons [26]
Cocaine also been reported to relate with CAE [27].It had been reported also that the aneurysmal formation , atherosclerosis and growth of plaque may result from low shear stress inside the endothelium[28].

Table 2 causes related to CAE[29].

1. Hereditary like Ehler-Danlos , Marfan's syndrome, bicuspid aortic valve, aortic root dilatation, ventricular septal defect or pulmonary stenosis
2. Atherosclerosis of coronary vessels especially if the patients is hypertensive, hyperlibidemic and smokers and it account for about 50 % of cases in adults.
3. No atherosclerosis: this might be:
a-Inflammation : like infective cause autoimmune cause connective tissue causes and vasculatitis,
Trauma e.g. coronary angioplasty trauma or trauma to chest.
b-Drug causes e.g. amphetamine and cocaine.
c-Some time it was reported as idiopathic
(Ozcan OU, Gulec S. Coronary artery ectasia. *Cor et Vasa*. 2013;55(3):e242-e247.)

CAE Pathogenesis

There were some theory that might explain CAE pathogenesis.

1.

1-Theroy of Hemodynamic: Focal CAE might be caused by irregularity of pressure above the threshold of stenosis in addition to the change of energy from kinetic to potential type. Poststenotic vasodilation and endothelial damage might results from high flow velocity as a result of greater shear stress at the stenosis level in addition to alteration of atherosclerotic plaque at an early stage to extensive remodeling (positive remodeling ectasia)[30&31].

2. theory related to remodeling of vessels: (extensive remodeling) of the arterial wall and plaque formation and also beyond rupture leading to decreasing in shear stress and . This make the shear stress to be low and vicious cycle maintance. [30&31]

3- Theory of Inflammation: Atherosclerotic process of coronary artery is considered as an inflammatory state and the spilling of cells of inflammation from the arterial intima ranging into tunica media & then result in CAE. As the inflammatory process is the link in between CAE and atherosclerosis in addition hemodynamic changes and inflammatory changes related to cytokine action like (IL-6 and CRP) [[32] In addition to that the cellular adhesion molecules like (intercellular and vascular adhesion molecules) also participate in inflammatory cells transmigration .Also the increase in neutrophil/lymphocyte ratio which is considered as inflammatory marker was reported to be high in blood of patients with CAE suggesting strong neutrophil role in inflammation.[31,33&34]

4.Nitric oxide mediated vasodilation especially in CAE which is not atherosclerotic in nature. extracellular matrix was affected by nitric oxide leading to vessel weakness and subsequent ectasia as there is high frequency rate among those using herbicide spray with inhibitors of acetyle choline that lead to augmentation of nitric oxide action by acetyl choline[35].
5.Hereditary theory: The matrix metalloproteinase -3 gene may be disrupted , [36].An angiotensin-converting enzyme (ACE) polymorphism had been associated with coronary artery ectasia. The relation of HLA link was proved by the DR16, HLA-DR B1 13, DQ2, and DQ5 genes [20].
6. Vessels disease in general : CAE might be portion of vascular disease affecting the body in general e.g. abdominal aorta and ascending aorta aneurysms or to venous illnesses e.g leg vein varicosities pampiniform plexus and coronary vein varicosities [30&37].

Clinical presentations:

CAE and CAD has similar clinical presentations [38]. Factors that might contribute risk include smoking, hypertensive disease , obesity, and stressful conditions. The clinical presentation of CAE still been unidentified. In patients with CAE with narrow coronary artery, symptoms of stable angina is the furthermost dominant presentation. Commonly atherosclerotic process was associated with CAE.**[39]**
During angiographic assessment of isolated CAE, the greatest significant predictors of ischemia were the extent of ectasia and the backflow phenomena in an ectatic artery **[40],** myocardial infarction (ST-elevation and non-ST elevation)(41&39][may be due to distal emboli of segment that is ectatic. Shunt formation, distal embolization, thrombus development and rupture are all likely complications. If there breaking through into the right atrium, right ventricle, or coronary sinus CAE can cause left-to-right shunts[11&42].

Coronary Artery Angiography

By measuring the degree of ectasia, angiography. The angiographic finding of stagnant and turbulent blood flow are delay in dye filling antegradly , reverse flow of segment, and local dye depositin in the dilated coronary artery. [2&23]

Coronary Magnetic Resonance Angiography (MRA)

It is non invasive free from radiation technique and when liked with coronary flow information it is helful in assessment of coronary obstruction in CAE. It is useful in follow up and is effect is similar to quantitative angiography of coronary artery. [2].

Coronary Artery Computed Tomography (CACT)

This technique also is a noninvasive method and assess the attenuation of contrast parallel well with flow fluctuations that was assessed by traditional X-ray angiography of coronary vessele.Its use in follow up is limited due to effect of radiation[16].

(Ultra Sound) Intravascularly (IVUS)

It is used for changes in the arterial wall and luminal size. It also recognize true aneurysms due to rupture of plaque from aneurysms that are fake. Plaque cavities that is empty may look like CAE on angiographic assessment and the differnciation between them is very important in clinical experience as false aneurysms may be complicated by acute coronary syndromes [11&43].

Inflammatory Markers

Systemic inflammatory markers (homocysteine ,CRP, neoprotein) , Cytokines like (IL-6 and TNF-alpha), [44&45].

Differential diagnoses[46]

The differential diagnoses for coronary ectasia include the following:Fibromuscular dysplasia,Bacterial syphilis, Ehler-Danlos syndrome, Atherosclerosis, Behcet disease, Mycotic aneurysm, Giant cell arteritis, Kawasaki disease, Marfan syndrome, Septic emboli,

The CAE sequel may be[46]

1. Thrombus: can lead to myocardial infarction.
2. Acute coronary syndromes.
3. Fistula formation in the cardiac chambers
4. Thrombosis
5. Embolic phenomena

6-After stenting the sequel may be :(misplacement,Embolic phenomena and thrombosis

7-Restenosis

Management

General Measures

Lifestyle modifications like
1-stopping of smoking
2- Healtthy diet
3-Practicing physical activity
4-Decrease weight
5-Control of hyperlipidemia
6-Management of other cardiovascular morbidity like hypertension and diabetes mellitus

Pharmacological Management

Antiplatelets

These drugs like aspirin are used to prevent platelet thrombotic activity to prevent ischemia due to fibrin thrombus and micro emboli as it had been shown previously that there was increase in mean platelet volume in patients with CAE in comparison to healthy control
Many studies support that there was significant platelet activation and inflammation [47]. These studies reflect strong role of antiplatelet in management of CAE. Otler study shown the dual antiplatelet treatment is less effective in management of CAE[48].

. In Kawasaki disease, both aspirin and I.V. gamma globulin are effective in CAE management[49]. 49.

Anticoagulants drugs

CAE can cause comorbities like dissection, spasm and thrombosis so the use of antigouagulant drugs in CAE has been suggested if not contraindicated especially individuals with atherosclerosis and CAD who have had myocardial infaraction with thrombotic events must be assessed for lifelong treatment and prophylaxis. If the age of patients is more than 65 year or those who use sigle or dual antiplatelet ,so it should be carefully evaluated to avoid bleeding in these groups[42&50].

New anticoagulants like apixaban, rivaroxaban and dabigatran are also used and been effective in thrombosed ectatic coronary arteries treatment[51].

ACE Inhibitors

These drugs reduce the intramural pressure, coronary dilatation and systemic hypertension and they used for CAE management. [52].

Statins

Studies proved that statin has anti-inflammatory action that decrease IL-6 and hs-CRP and been effective therapeutic option in young patients with CAE[53 &54].

Antianginal Drugs

They are avoiding ischemic destruction caused by oxygen-free radicals without changing blood fluency to coronary vasculation or consumption.of oxygen[55,56,75&58].

Interventional Management

The associated coronary artery occlusions or who have ischemic changes despite optimium medical treatment frequently advice for invasive options (coronary artery bypass graft or percutaneous coronary intervention)[55, 59,60 &61].

Prognosis

This is in a straight line associated with severity of associated coronary artery diseases. So type I and II coronary ectasias according to Markis' classification[10] have a worser prognostic events than those patients with type III or IV. The mortality rate at 2 years is shown to be around 15% while there is no correlation between the survival and ectasia width [62].Isolated CAE have a prognosis that is better and treatment with antiplatelet are been used[1].

Conclusion

CAE is considered as type of atherosclerotic events might happen in a range of 3.1 -8% of angiographic procedures. There is currently no fixed treatment guide line. Treatment involve antiplatelet, antithrombotic drugs, anti-ischemic drugs in addition to statin in addition to revascularization (surgical or percutaneous).

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