Coronary Artery Ectasia

A comprehensive review of literature, etiology, diagnosis, clinical features, treatment and prognosis

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Coronary artery ectasia (CAE) is a rare clinical condition which represents a form of atherosclerotic coronary artery disease. It is defined as dilatation of the coronary artery 1.5 times greater than that of an adjacent normal segment of coronary artery (1). This entity has been reported in patients undergoing coronary angiography from 3 % to 8 %, alone or in combination with stenotic lesions. The rate of recognition may increase with the use of new non-invasive imaging methods like computed tomography (CT) and magnetic resonance (MR) coronary angiography. The clinical significance of CAE is not very well defined and the results have been mixed and conflicting (2,3). As the main etiologic factor responsible for CAE is atherosclerosis, there are other factors involved in different groups of patients. The objective of this article is to review and examine the clinical characteristics of CAE, diagnosis, treatment and its prognosis. Also the purpose of this review is to update and to summarize the clinical features of CAE.

Key words: coronary artery, aneurysm, ectasia, atherosclerosis.

Koronární arteriální ektazie
Souhrnný přehled literatury, etiologie, diagnostika, klinické rysy, léčba a prognóza

Koronární arteriální ektazie (CAE) je vzácná klinická jednotka, která představuje jeden druh aterosklerotického postižení koronárních tepen. Je definována jako rozšíření lumen koronární tepny o více než 1,5násobek průměru největší přilehlé nepostižené tepny (1). U pacientů podstupujících koronarografické vyšetření se četnost CAE uvádí v rozmezí od 3 % do 8 %, a to buď samostatně, nebo v kombinaci se stenotickými lézemi. Míra rozpoznávání jedinců s tímto postižením se může zvýšit za použití nových neinvazivních zobrazovacích metod, jako jsou např. výpočetní tomografie (CT) a MR angiografie. Klinická významnost CAE není dostatečně stanovena a dosavadní výsledky byly smíšené a protichůdné (2,3). Hlavním etiologickým faktorem zodpovědným za CAE je ateroskleróza; v různých skupinách pacientů se na ní ale podílejí i jiné faktory. Článek si klade za cíl přezkoumat klinické charakteristiky CAE, její diagnostiku, léčbu a prognózu. Cílem přehledu je rovněž aktualizace a shrnutí klinických rysů CAE.

Klíčová slova: koronární tepna, aneurysma, ektazie, ateroskleróza.

Introduction

Coronary Artery Ectasia (CAE) is an uncommon but well defined entity which can be observed during diagnostic coronary angiography (4, 5, 6). It has been defined as inappropriate dilation of the coronary arteries exceeding the largest diameter of an adjacent normal vessel more than 1.5 fold (4, 5, 6, 7) (Figure 1).

The term aneurysm is used to describe focal dilatation while the term 'ectasia' refers to diffuse dilation of a vessel like aorta or coronary (8) (Figure 2).

There have been suggested different mechanism but precise etiology and pathophysiology mechanism of CAE is not recognized and there has not been a real consensus about its natural history and management of this condition because of the relative mixed and scarcity of clinical data. CAE represents not only an anatomical variant but also a clinical constellation of coronary artery disease (CAD) like association with myocardial ischemia and acute coronary syndrome.

Coronary dilatation is isolated ectasia, in association with connective tissue disorders such as Marfan syndrome, Ehlers–Danlos syndrome, sc-

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Figure 1. *Coronary artery ectasia*

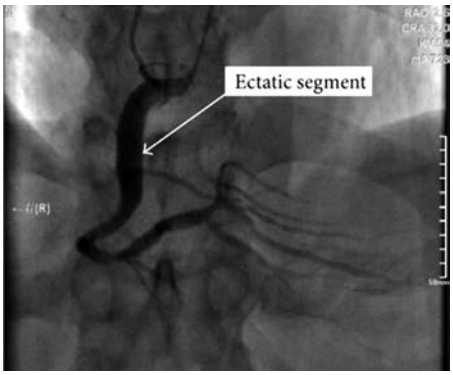
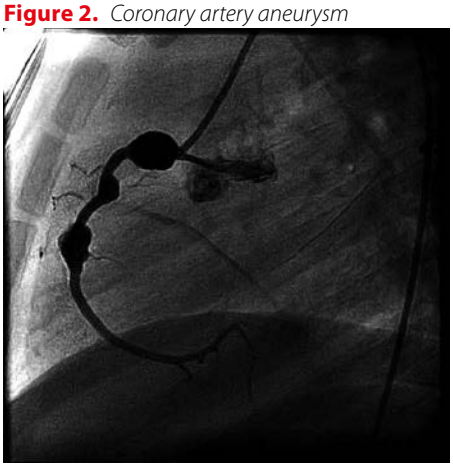


Figure 2. *Coronary artery aneurysm*



leroderma, different types of ANCA- related vasculitis (8) and also in syphilitic aortitis and Kawasaki disease (26). In a smaller percentage of patients CAE may be congenital. Acquired CAE should be differentiated from coronary aneurysms following coronary interventions. Occasionally, large ulcerated coronary plaques can be misinterpreted angiographically as coronary aneurysms. Their true cause can be revealed by intravascular ultrasound (IVUS). There is no direct correlation between ectasy at the coronary arteries level and ectasy in other arteries of the peripheral vascular system, although they may co-exist in some cases.

Etiology, risk factors and pathophysiology

Main etiologic factor causing CAE is atherosclerosis in more than half of the cases in adult patients (4, 6, 10, 12). In younger patients with Kawasaki is the most common cause (9, 10, 11, 26).

Coronary aneurysms develops in younger patients with Kawasaki disease in about 10–20 %. It remains most important manifestation of this disease which leads to premature atherosclerosis, occlusion and thrombosis with MI. After the introduction of the aspirin and intravenous gamma globulin therapy, resolution occurs in about more than half of the cases (26). In table 1, Etiology of CAE is demonstrated in more details.

Table 1. *Etiology of Coronary Artery Ectasia*

- Atherosclerosis
- Coronary artery revascularisation procedures (balloon angioplasty, stent implantation, laser angioplasty, atherectomy, brachytherapy)
- Vasculitides (Kawasaki disease, polyarteritis nodosa, syphilis, Takayasu disease, Wegener granulomatosis, Giant cell arteriitis, Churg Strauss Syndrome)
- Congenital malformations
- Chest traumas
- Connective tissue disorders (rheumatoid arthritis, Systemic lupus erythematosus, scleroderma, ankylosing spondylitis, Bechet's disease, Psoriasis)
- Collagenopathies (Marfan's syndrome, Ehlers-Danlos syndrome, hereditary hemorrhagic telangiectasia)
- Primary hyperaldosteronism

As the exact etiology and pathophysiology mechanism of CAE remains unclear there are similarities between atherosclerosis and ectasia at the histopathologic level. As a result of enzymatic degradation in the extracellular matrix which leads to exaggerated expansive remodeling, CAE may be developed (13).

The luminal dilatation is not very clear in some atherosclerotic vessels since atherosclerosis predominantly causes narrowing of the vessel lumen. The luminal size is not reduced due to dilatation at the media and external level because there is no evidence of ectasia in cases with intact and uninvolved media layer, which makes it somewhat very similar to the annuloaortic ectasia. Intravascular ultrasound has demonstrated dilatation and reduction of external membrane, positive and negative remodeling respectively (14). CAE may be a result of compensatory mechanism as atherosclerosis progresses in which luminal size and external membrane increase in size (15, 16). One of the main suggested mechanism of expansive remodeling has been overexpression of matrix metalloproteinases, the enzymatic degradation of extracellular matrix and other lytic enzymes (17). In patients with CAE the thinning of the tunica media and that is associated with severe chronic inflammation as a result of higher levels of C-reactive protein(CRP) and also vascular endothelial growth factor (18, 19).

There are other etiology and mechanisms which have been suggested like chronic stimulation of endogenous nitric oxide that leads to vascular relaxation and the occurrence of areas of ectasia is another suggested mechanism (5). Another study indicates that CAE occurs due to two different mechanisms in two distinct patient groups: (i) rarely in subjects without coronary atherosclerosis as a result of interstitial NO vascular overstimulation and (ii) commonly in patients with concomitant CAD due to severe and chronic arterial inflammation (20).

This process of "arterial remodeling" is fundamental to the pathophysiology of CAD and CAE. The incidence of typical stress-induced angina pectoris and MI is due to aneurysmal segments of coronary arteries which has sluggish and or turbulent blood flow. Patients with CAE have benign course, but there are some who still present signs of MI.

All three coronary vessels can be affected by CAE, but almost 75 % of patients will have an isolated artery that is ectatic. In patients with concomitant CAD, the proximal and mid segment of right coronary artery is the most frequently affected.

In terms of genetic predisposition, there is a link between CAE, angiotensin-converting enzyme genotyp DD and hereditary conditions like familial hypercholesterolemia (21, 22). There has not been stablished fact or link between CAE and hypertension, DM, hyperlipidemia, smoking or familial history despite of controversial reports (6, 10, 11). CAE has been associated to apical hypertrophy during systole but demonstration during left ventricular diastolic dysfunction has been controversial (23, 24). Injury of coronary artery an its media has been the main cause of coronary aneurysm following percutaneous revascularization (12) either balloon angioplasty or stent placement (20, 25). Isolated CAE is rare but it mainly co-exist with other vasculature aneurysms specially aortic aneurysm, either annuloaortic ectasia and or abdominal aortic aneurysm.

Classification, prevalence and epidemiology

CAE is classified based on the diameter of lumen, giant, medium, and small. There is another classification based on Markis et al., table 2.

Table 2. *Markis Classification*

- **Type 1**
  Diffuse ectasia of two or three vessels
- **Type 2**
  Diffuse ectasia in one vessel and localized disease in another
- **Type 3**
  Diffiuse ectasia in one vessel only
- **Type 4**
  Localized or segmental involvement

Incidence of CAE has been reported from 3% to 4,9% in coronary angiogram studies (1, 4, 6) and its prevalence may be up to 8% in the advent of using new and more advanced technologies (9, 10). CAE has a higher incidence in women than in men (6). That number is in range from 0.22% to 1.4% of autopsy series. It can be either diffuse affecting the entire length of a coronary artery, or localized. It is attributed to atherosclerosis in 50% of cases, whereas 20 to 30% are considered to be congenital in origin. In the great majority of these patients ectasia coexists with coronary artery disease (CAD). Only 10% to 20% of CAE have been described in association with inflammatory or connective tissue diseases (4).

Diagnosis

Despite of other means like intravascular ultrasound, coronary angiogram has been gold standard diagnostic tool in CAE (15, 20). For the purpose of this review we take a comprehensive look at each and every one of these methods of diagnosis of CAE.

Intravascular Ultrasound (IVUS): This method allows us to observe a coronary artery from inside out. It is a unique point of view that is generated in real time and gives valuable information in conjunction with other diagnostic tools such as coronary angiography, or CT scans. IVUS has been used and it has been very helpful for difficult cases, either to assist in the selection and sizing of a coronary artery or confirming the accurate and optimal stent placement.

X-ray coronary angiography: X-ray coronary angiography is the main diagnostic technique for the identification of coronary artery ectasia and it remains gold standard method of diagnosis.

Coronary Artery Computed Tomography (CACT): Evaluation of ectatic vessels CACT has been used and very useful in recent years. Coronary artery ectasia usually was associated with atheromatous changes, but not with significant CAD and thrombosis was a rare complication. Contrast attenuation measurements with CTCA correlated well with the flow alterations assessed with classic X-ray coronary angiography (CCA) (26). Due to high radiation, CACT use is not recommended for patients' follow-up. There are some changes and more improvements in terms of radiation doses are expected in the near future.

Magnetic Resonance Angiography (MRA): The correct follow-up of ectatic vessels is hampered by the need for repeated angiograms. Three-dimensional, non-contrast enhanced, free-breathing coronary magnetic resonance angiography facilitates visualization of the vast majority of the proximal and middle segments of the coronary arteries. MRA has already been of clinical value for the assessment of anomalous CAD, and it is in some cases superior to x-ray coronary angiography in delineating the course of anomalous vessel, but it is still considered an investigational technique for the assessment of stenotic native vessel. The correct follow-up of ectatic vessels is hampered by the need for repeated angiograms. Three-dimensional, non-contrast enhanced, free-breathing coronary magnetic resonance angiography facilitates visualization of the vast majority of the proximal and middle segments of the coronary arteries. MRA has already been of clinical value for the assessment of anomalous CAD, and it is in some cases superior to x-ray coronary angiography in delineating the course of anomalous vessel, but it is still considered an investigational technique for the assessment of stenotic native vessel. However it is proposed as a valuable tool for patients who present with severe left ventricular systolic dysfunction, where the underlying disease is either severe multi-vessel coronary artery disease or nonischemic cardiomyopathy. The fact that MRA is equal to QCA as a non-invasive technique and have some advantages has been proved (28, 29). Compared with computed tomography, MRA has the advantage of requiring no exposure to radiation or injection of a contrast agent. This makes MRA as suitable tool for follow up of CAE, since it is non-radiating and non-invasive (8). Transthoracic or transesophageal echocardiography may be option for the proximally located ectasias. Distortions in flow and wash out are common in CAE, and are clearly associated with the severity of ectasia. Delayed antegrade dye filling, is an angiographic signs of turbulent and stagnant flow in a segmental back flow phenomenon and local deposition of dye in the dilated coronary segment. This phenomenon is so called milking phenomenon.

Clinical course and symptoms

Patients with CAE do not have clear and specific symptoms and their clinical course remain unclear unless they remain associated with other concomitant disease like connective tissue disorders, Kawasaki and or CAD. Therefore many of these patients remain asymptomatic. Some may present vague chest pain others remain symptom free. Either group may present positive Stress Test or Acute Coronary Syndrome (30, 31, 32). Regardless of coexistence of CAD, any decrease of flow in coronary arteries due to stress or exercise may cause angina (8). Acute coronary syndromes may result from atherosclerotic lesions within ectatic regions of the coronary arteries which appear to be highly inflamed high-risk plaques with tendency to rupture. Ectatic vessel may be an origin of thrombus formation with distal embolization, vasospasm or vessel rupture. Due to sluggish flow in the ectatic segment of coronary arteries, thrombus and or microemboli formation may occur (10, 33). Coronary angiographic findings such as topographical extent of CAE and corrected (Thrombolysis in Myocardial Infarction) TIMI frame count are associated with severity of angina in patients with CAE (31). Another observation in patients with isolated CAE was diminished both epicardial flow and microvascular perfusion (34). Coronary microvascular perfusion is impaired even in the setting of normal epicardial flow and CAE without significant epicardial stenosis may lead to exercise induced myocardial ischemia especially if coronary tree is diffusely involved (30, 35). Measurement of the velocity of blood flow in patient with isolated CAE has shown significant higher volumetric coronary flow but reduced coronary flow reserve. This may suggest that the dysfunction of microcirculation is the underlying cause of exercise-induced myocardial ischemia in patients with CAE (36). Another cause of acute coronary syndrome and angina is vasospasm of CAE segment (10, 37). Among younger population, connective tissue disorders should be considered to make the right diagnosis. In these circumstances further investigation is necessary. For instance in young adult female, Kawasaki should be considered. In these patients treatment and management are different from the conventional atherosclerotic ectasias (26, 38).

Treatment, therapeutic approach and management

CAE is not a benign coronary anomaly and until now in contrary to atherosclerotic CAD the medical treatment has not been adequa-

tely addressed. Several studies has suggested chronic use of anticoagulation as therapy but this has not been proved to be the right and only management. The suggested treatment has been as follow (5): 1. anticoagulation therapy, utilizing chronic warfarin therapy to offset the risk of thrombus formation and to keep the international normalized ratio at around 2.0–2.5; 2. antiplatelet therapy, utilizing aspirin (80–360 mg/day) to minimize platelet aggregation; and 3. antispasm therapy, utilizing calcium-channel blockers. Nitrates could also be used, but added care should be emphasized to provide a nitrate-free "holiday" and prevent chronic exposure to these agents. It is worth mentioning that nitrates, by causing further dilatation of coronary arteries, have been shown to exacerbate myocardial ischemia and they are discouraged in patients with isolated CAE. Statins may have a role by inhibiting matrix metalloproteinases (39). The coexistence of CAE with obstructive coronary lesions in the great majority of patients and the observed incidence of myocardial infarction – even in patients with isolated coronary ectasias – suggested the generalized administration of aspirin in all patients with CAE. When coexisting with CAD, the prognosis and treatment of CAE are the same as for CAD alone. In isolated CAE, prognosis is better and anti-platelet drugs are the mainstay of treatment. It is absolutely crucial to make the right diagnosis of underlying cause of CAE in order to apply adequate therapy to improve patient's management and conditions. Therapy should be tailored to each individual patient because of the serious bleeding complications associated with warfarin use. There has not been any studies on NOACs for therapy of CAE. Percutaneous and/or surgical coronary revascularization can safely and effectively restore normal myocardial perfusion in patients with coexisting obstructive lesions and symptoms with signs of significant ischemia. In PCI, stent deployment has some degree of difficulties because the ectatic arteries are usually much larger than normal size (40). Therefore, selection of a stent of adequate size and its expansion are very important, and can be determined by intravascular ultrasound. Coronary artery bypass grafting has been used for the treatment of significant CAD co-existing with ectatic coronary segments 4. In Kawasaki disease, using aspirin and intravenous gamma globulin therapy reduces the occurrence CAE. In terms of overall strategy for treatment of CEA we can conclude that there is still no consensus and that is due to rarity of this condition. This may well change in the future because of new available technologies and ability to diagnose more patient with CAE and further conducting large scaled randomized trials which compares different treatment modalities.

Prognosis

The prognosis of CAE varies and the authors opinions are very different (41, 42, 43). In one study mortality has been reported of 15 % in one year with a 2 years follow-up (11) and in another study 29.1 % mortality of 5 years (41). These results and prognosis were based on the CAE classifications by Markis and colleagues, type I and type II coronary ectasias bring about the worse prognosis than type III and IV. It depends on the severity of the concomitant coronary artery disease. CAE with obstructive coronary artery disease is a very serious condition because of its potential of resulting in several adverse cardiac events. There were no relations between diameter of ectasia and survival is reported. Although no difference in mortality between patients with or without CAE was demonstrated, isolated ectasia still bears risk of myocardial ischemia and infarction. As stated earlier antiplatelet drugs underlie the therapy. Other therapeutic in management strategies of CAE involve both the prevention of thromboembolic complications and percutaneous or surgical revascularization.

With recent advances in technology in diagnosis and revascularization, treatment and treatment of CAE have significantly improved thus the prognosis of CAE seems to be much better. In one study mortality in patients with CAE was reported at rate of 2 % (42).

Conclusion

CAE is worth closer study, as different etiologies and complicated pathophysiology are still not completely understood. In recent years with improvement and use of advance technology to diagnose CAE as well as applying different modern treatment strategies, the prognosis seems to be improving significantly. But these therapeutic modalities need large-scale, randomized, controlled studies to prove their long-term effectiveness. CAE is a vascular disease mostly a form of atherosclerosis seen in 0.3–4.9 % of diagnostic coronary angiography procedures. A pattern of vascular remodeling, exaggerated expansive remodeling with enzymatic degradation of the extracellular matrix and thinning of the vessel media as a result of chronic inflammation is thought to be a major pathophysiologic process. The symptoms may be associated with the concomitant CAD, Kawasaki disease or other connective tissue disorders although most of the patients are asymptomatic. Clinical importance especially leans on its association with acute coronary events. Treatment options include, risk factor modifications for CAD, anti-ischemic therapy, antithrombotic management and percutaneous or surgical revascularization techniques. This is worth emphasizing that therapy should be tailored to each individual patient because of different underlying cause. Future trials are warranted in order to optimize the treatment and management strategies of CAE.

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