Original Article

The prevalence and clinical profile of Angiographic Coronary Ectasia

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Abstract

Objective: To determine the incidence of Coronary Artery Ectasia (CAE) at our teaching hospital to describe the patients and angiographic characteristics.

Methods: A retrospective analysis was conducted on all coronary angiograms performed at the catheterization laboratory of Karachi Institute of Heart Diseases, a tertiary care center, between the period of August 2006 and August 2008. A one year follow up was performed to look for primary and secondary endpoints. Data were collected from catheterization films, and medical records.

Results: Five thousand coronary angiograms were performed during the periof of the study. A total of 140 (2.8%) angiograms showed coronary ectasia of both mixed and pure types. Pure ectasia with no coronary obstructive lesions was seen in 75 (1.5%). The left anterior descending artery (LAD) was the most commonly affected vessel (63%) followed by the right coronary artery (RCA) 25% and 10% patients had circumflex artery involvement. The primary composite endpoint showed 6 (4.2%) patients with non-ST elevation MI, 5 (3.6%) with ST elevation inferior wall myocardial infarction, 70 (50%) with unstable angina and 2 (1.4%) deaths due to pulmonary oedema. Secondary endpoints showed 50% of the patients still complaining of chest pain.

Conclusion: Prevalence of Coronary ectasia in the population presenting to KIHD during the study period was 1.5%. Majority of patients were males, associated with dyslipidaemia, hypertension and smoking. CAE was associated with obstructive coronary artery disease in about 80% of cases. LAD was the most commonly affected vessel.

Keywords: Coronary artery disease, Ectasia, Aneurysm, coronary artery angiography (JPMA 61:372; 2011).

Introduction

Coronary artery ectasia (CAE) is defined as localized or diffuse dilation of $\geq$ 1.5 times normal adjacent segments of vessels.[1] As it was first described by Morgagni regarding its cause, clinical sequelae and treatment, it is estimated that 50% coronary artery ectasia is related to atherosclerosis, whereas 20%-30% of cases may be due to congenital anomalies.[2] CAE is mainly encountered in males in most of the patients it seems to be related to coronary atherosclerosis.[3] Aneurysms and ectasia are associated with a vast group of disorders, and the evaluation and characterization of coronary aneurysms and ectasia represent a great diagnostic task with clinical and therapeutic implications. The underlying etiology is variable and includes degenerative, congenital, inflammatory, infectious, toxic, and traumatic causes. Unlike aneurysms, ectasia is more frequently seen in association with atherosclerosis or as a compensatory mechanism in those cases in which a proximal stenosis is noted in the opposite coronary artery; ectasia is also seen in some coronary artery anomalies, such as anomalous origin from the pulmonary artery, or as a result of a high-flow state, as seen in coronary artery fistulas.[4] "Pure" coronary ectasia can be implicated in angina or myocardial infarction, worse prognosis depends on the association of stenotic coronary artery disease.[5,6] Markis classified CAE in four types: type 1 includes diffuse ectasia involving two or three vessels, type 2 includes diffuse ectasia involving one vessel and discrete ectasia in another, type 3 includes diffuse ectasia in only one vessel, and type 4 includes localized or segmental ectasia in only one vessel.[7] Coronary artery ectasia may lead to exercise induced ischaemia, especially in the diffuse form.[8] CAE is thought to be present in patients with a slow blood flow. It is usually not a benign condition, as normal smooth laminar flow is disrupted with a potential of thrombus formation. CAE is characterized by diffuse or localized inappropriate dilation of coronary arteries and is often associated with slow coronary blood flow in the presence of hypertrophic cardiomyopathy.[9] Coronary blood flow was quantified using the thrombosis in myocardial infarction (TIMI) frame-count method. TIMI frame count measurement depends on the ectasia size or ectasia ratio, and an increased ectasia ratio is markedly associated with decreased TIMI frame counts in patients with CAE.[10] In this study the data on ectectic lesion of coronary artery was analyzed.

Methods

A retrospective analysis was conducted on all coronary angiograms performed at the catheterization

laboratory of Karachi Institute of Heart Diseases (KIHD), a tertiary referral center, between the periods August 2006 to August 2008. A total of 5000 coronary angiograms were included. Ectasia was classified according to the Markis classification (Table-1).[7] This involved finding the major coronary vessels involved and classifying the ectasia in each vessel involved as diffuse or focal. The classification was modified to be more simple and to be able to predict prognosis and hence the choice of treatment (Table-2). The new classification defined 3 groups according to the number of the major coronary vessel involved. It was then further subdivided into focal, diffuse or mixed (i.e., focal in one vessel and diffuse in other).

Data were collected from catheterization films, and medical records. The study had primary and secondary endpoints. The primary endpoint was the major adverse cardiac event rate (MACE) — a composite end point of unstable angina with ECG changes, MI and cardiac death. Secondary endpoints were: recurrent chest pain, the need for repeat cardiac catheterization, emergency room visits for chest pain and hospital admission for chest pain.

Results

During the study period, 5000 coronary angiograms were performed. One hundred and forty angiograms (2.8%) showed coronary ectasia of both mixed and pure types. Eighty angiograms showed pure ectasia with no coronary obstructive lesions. The right coronary artery (RCA) was the commonest affected vessel by ectasia (88 %), followed by the left circumflex coronary artery (LCX) in 35 % and then left anterior descending artery (LAD) in 15 % of the patients (Table-2). Demographic data of the patients showed. The mean age as 52.1 ± 10.3 years (range 30 to 85 years). Among them 89 (64%) were younger than 65 years of age. About 80 (57%) were overweight (Body Mass Index greater than 26 kg/m^2, 50 (37%) were current smokers, 77 (55%) had hypertension, 36 (26%) had diabetes mellitus, and 58 (42%) had dyslipidaemias with elevated serum low-density lipoprotein (LDL) and triglycerides (TG) and low high-density lipoprotein (HDL) levels. The main indication for heart catheterization was anginal type of chest pain in 90% patients. The treatment modalities included: anticoagulation with warfarin alone in 20 (14%) patients, anti platelet therapy with aspirin alone in 78 (56%) patients, and a combination of aspirin and warfarin in 98 (70%) patients. Nitrate therapy was used in 48 (34%), calcium channel blockers in 35 (25%), and beta blockers in 28 (20%) patients. The average follow up period was 24 months (range 3 to 24 months). The primary composite endpoint (MACE) showed 6 (4.2%) patients with none ST elevation MI (NSTEMI), 5 (3.6%) with ST elevation inferior wall myocardial infarction, 70 (50%) with unstable angina, and 2 (1.4%) deaths reported due to pulmonary oedema. Six patients with 3 vessel diffuse ectasia had non-ST elevation MI (NSTEMI) but repeated cardiac catheterization showed no coronary obstruction. Secondary endpoints at the time of the follow up showed 65 (46%) patients still complaining of chest pain. Thirty four (24%) patients presented to the emergency room during this period. nineteen were discharged from the emergency room after being diagnosed with non-cardiac chest pain and having normal ECG and cardiac enzymes. However fifteen patients (11%) were admitted to the hospital. Repeated cardiac catheterization was needed in six patients. Two deaths were reported.

Table-1: Angiographic types according to Markis Classification.[7]

Type	Ectasia type and location	Percentage of cases in our study
Type I	Diffuse ectasia of two or three vessels	50
Type II	Diffuse disease in one vessel and disease in another vessel	17
Type III	Diffuse disease in one vessel only	5
Type IV	Localized or segmental ectasia	28

Table-2: Percentage of individual coronary vessels affected by ectasia Modified Markis Classification.

Vessel	Focal (%)	Diffuse (%)	Total involvement (%)
LAD	7	8	15%
RCA	48	40	88%
LCX	16	19	35%
L.M	5	3	8%

Discussion

Coronary ectasia is defined as local or generalized aneurysmal dilatation of the coronary arteries. Coronary ectasia likely represents an exaggerated form of expansive vascular remodeling (i.e. excessive expansive remodeling) in response to atherosclerotic plaque growth. Enzymatic degradation of the extracellular matrix of the media is the major pathophysiologic process that leads to ectasia.[11] CAE is a relatively common angiographic finding with an average incidence of 1-5%. According to the angiographic definition used in the Coronary Artery Surgery Study a vessel is considered to be when its diameter is $\geq$ 1.5 times that of the adjacent normal segment in segmental ectasia.[12] The majority of CAEs are strongly associated with local and systemic atherosclerosis prevalence of ectasia and aneurysmal disease in familial hypercholesterolemia (FH) suggesting a link between plasma lipoproteins and coronary aneurysms.[13] In our study 45 (32%) patients had

dyslipidaemia seen as raised Triglycerides. Occasionally CAE associated with other pathologies such as syphilitic aortitis, connective tissue disorder (scleroderma, Ehlor-Danlos syndrome, polyarteritis-nodosa) and Kawasaki's disease. Prolonged exposure to herbicides containing acetyl cholinesterase inhibitor as well as Stimulation of nitric oxide by acetylcholine causes relaxation of vascular smooth muscle cells.[14] Diabetes mellitus was found to be independently but inversely associated with CAE.[15] Prevalence of CAE in familial hypercholesterolemia was proposed as structural weakening or active lysis of connective tissue elements in the arterial wall resulting from interaction with LDL-cholesterol. In various reports CAE has been described as isolated congenital lesion.[16] Hyperhomocysteinaemia is recognized as an independent risk factor for arterial disease including coronary artery disease, cerebrovascular disease and peripheral vascular disease.[17] Incidence of coronary artery ectasia in patients undergoing cardiac catheterization has ranged from 0.3% to 4.9% in different series, regardless of associated coronary artery stenosis.[18] In the present study 55% patients with CAE suffered from hypertension. The common features of affected individuals were middle-aged males who were smokers and obese. It is established that inflammation is affected by both smoking and cardiovascular disease.[19] There was a significant association of CAE with smoking, and obesity in our study. Involvement of coronary vessels is variable, several studies reported the RCA to be the commonest involved vessel but in some studies LAD was the main vessel.[20] Our study found that RCA was the main involved vessel. Our study showed that the clinical spectrum of CAE is variable, including stable angina pectoris, unstable angina pectoris, vasospastic angina, and myocardial infarction. The most common symptom is exertional angina.[21] Tendency to thrombosis due to diminished coronary flow and vasospasm due to structural changes in the vessel wall may cause chest pain and even myocardial infarction.[22] The treatment of CAE is controversial. Most of our patients were treated with combined use of aspirin and warfarin. Some authors recommended long-term anticoagulation therapy[23] while others recommended only antiplatelet therapy with aspirin in asymptomatic individuals. Recommended using warfarin anticoagulation therapy in these groups of patients.[24] Some studies proved that ectatic vessels are prone to vasospasm and the use of nitrates or calcium channel blockers may be useful. On the other hand, the use of nitrates as a vasodilator has been reported to worsen ischaemia.[25] Considering CAE to be a variant of atherosclerosis and the use of lipid lowering agents may therefore be beneficial to this group of patients,[26] these groups of drugs were used in our study.

Conclusion

Our current study was a retrospective analysis of angiograms reported as ectasia, with prospective follow up aimed at determining the incidence of specific cardiac events. Coronary ectasia should not be considered merely dilatation of the vessel wall, because it may lead to cardiovascular complications. It is one of the less frequent causes of myocardial infarction and management of this angiographic entity requires aggressive medical therapy.

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