INCIDENCE AND CHARACTERISTICS OF CORONARY ARTERY ECTASIA IN PATIENTS UNDERGOING CORONARY ANGIOGRAPHY AT ARMY CARDIAC CENTER LAHORE

Faisal Shafiq, Ahmad Usman, Mehboob Sultan, Khurram Shehzad, Namra Haroon*, Osama

Army Cardiac Center Lahore/National University of Medical Sciences (NUMS) Rawalpindi Pakistan, *Shalamar Hospital Lahore Pakistan

ABSTRACT

Objective: To estimate the incidence and characteristics of coronary artery ectasia in patients undergoing coronary angiography.

Study Design: A prospective analysis.

Place and Duration of Study: This study was conducted at Army Cardiac Center, Lahore over a period of two years from Jan 2018 to Dec 2019.

Methodology: Its prospective analysis of all coronary angiograms performed in our catheterization laboratory during study period. Markis classification was the basis to define and classify coronary artery ectasia. Demographical, clinical, and laboratory data were collected for each patient in this study.

Results: A total of 172 (3.9%) out of 4,372 coronary angiograms showed coronary artery ectasia. Among coronary artery ectasia group, mean age 58 ± 10 years, 90% were male, 47% were current smokers, 32% were hypertensive, 15% had diabetes Mellitus and 37% had dyslipidemia. The most common clinical presentation was Non ST-segment elevation myocardial infarction (31%), followed by Stable ischemic heart disease in 28%. Right coronary artery was the most frequent coronary artery involved (57%) while Markis Class 3 pattern was seen as most common type of coronary artery ectasia.

Conclusion: The frequency of coronary artery ectasia among our patients undergoing coronary angiography was about 4%. Right coronary artery remained the most common affected artery.

Keywords: Angiography, Coronary artery ectasia, Myocardial infarction.

INTRODUCTION

Coronary artery ectasia (CAE) is a relatively common entity and isdefined as dilatation of a part or whole of the coronary artery ≥1.5 the diameter of an adjacent normal segment[1]. It is mostly linked with atherosclerotic coronary artery disease (CAD). Occasionally, it has been associated with other conditions such as Kawasaki disease, syphilitic aortitis, connective tissue disorders such as Ehlers–Danlos syndrome, Polyarteritis Nodosa, Scleroderma and infections. CAE can be congenital in origin in a small percentage of population[1]. The exact etiologystill remains unclear. Clinicians are always skeptical regarding its cause, clinical outcome, and optimal therapy. Data across the world from coronary angiogram registries showed its prevalence varying from 2% to 5%. Also, a few studies conducted in our region produced similar results 2-5 but mostly in retrospective methodology. The incidence of CAE in subcontinent patient cohort with CAD was seen around 10%, as reported in a posthoc study[2]. The frequency of CAE without underlying CAD was shown to be 2.1% in a recent study done in Pakistan. Another study conducted in India revealed its incidence around 4.5%, with 85% of these having underlying CAD[3]. The prevalence of CAE in general population may be overestimated by these studies as it is much less likely to be discovered in patients without symptomatic CAD[4]. This study was done in a prospective method to evaluate the incidence of CAE in our patients undergoing coronary angiogram for various reasons since no large-scale study on this available in our country.

Correspondence: Dr Faisal Shafiq, Cardiology Department, Army Cardiac Center Lahore Pakistan

METHODOLOGY

A prospective analysis was done on all coronary angiograms performed at Army Cardiac

Center/CMH Lahore between January 2018 and December 2019. A total of 4,372 coronary angiograms were performed in patients with suspected or previously known CAD and also to rule out CAD in patients with structural heart disease including adult congenital heart disease. Visual and quantitative methods remained the basis of angiographic evaluation of these patients. Coronary Artery Surgery Study (CASS) was used to define the CAE, where a vessel was observed as ectatic when its luminal diameter was more than 1.5-fold the adjacent normal segment of the vesse l.5 CAE was classified according to the Markis classification 6-Type I: diffuse ectasia in two or three vessels, Type II: diffuse ectasia in one vessel and localized disease (i.e., aneurysm) in another, Type III: diffuse ectasia in only one vessel, and Type IV: coronary aneurysm in one vessel.

Epidemiological, clinical parameters and appropriate investigations such as 12 lead electrocardiogram, 2-D echocardiography, and lipid profiles were obtained for each patient in this study. Dyslipidemia was considered as the presence of any of the four points: (1) total cholesterol more than 200 mg/dl, (2) High-density lipoprotein (HDL) cholesterol more than 40 mg/dl in males and <50 mg/dl in females, and (3) Low-density lipoprotein (LDL) cholesterol >130 mg/dl (4) Triglyceride >150 mg/dl. This study does not evaluate patients further for various cardiovascular and otherassociated complications in the long term follow-up.

RESULTS

Total 4,372 coronary angiograms were performed during study period and 172 coronary angiograms (3.9%) showed CAE (fig-1&2). Table-I shows demographic distribution of the patients. Mean age was 58.2 ± 10 years. Among them 153 (89.6%) were male and 81 (47.2%) were current smokers.

Clinical presentation of CAE patients (table-II) was non-ST segment elevation myocardial infarction (NSTEMI) in 54 (31.2%), Stable ischemic heart disease 49 (28.3%), anterior wall ST-segment elevation myocardial infarction (AWSTEMI) in 37 (21.9%), inferior wall ST-segment elevation myocardial infarction (IWSTEMI) 22 (12.8%), atypical angina in 6 (3.5%), and Valvular heart disease in 4 (2.3%) of cases. Twenty one (16.9%) angiograms showed isolated CAE without obstructive CAD.

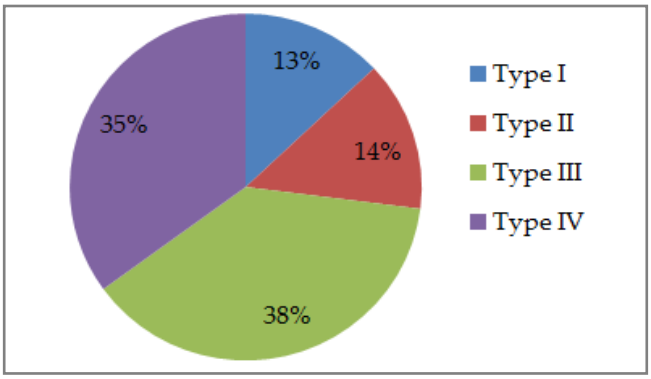

Figure-1: Distribution of diffuse ectasia (n=111) versus aneurysm (n=61) in three epicardial coronary arteries

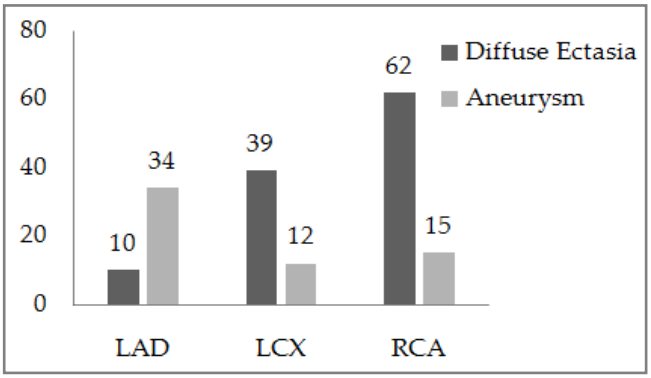

Figure-2: Percentage of CAE patient's distribution according to markis classification.

Table-I: Demographic profile of the patients.

Variables	n (%)
Age (mean ± years)	58 ± 10
Male	153 (89.6)
Smokers	81 (47.2)
Systemic Hypertension	54 (31.6)
Dyslipidemia	64 (37.2)
Diabetes Mellitus	25 (14.8)
Family history of CAD	15 (12.1)

The right coronary artery (RCA) was the most commonly affected coronary artery (57.3%), followed by the left circumflex coronary artery (LCX) in 39.2% and the left anterior descending

coronary artery (LAD) in 35.2% of the patients. Aneurysm was seen predominantly in LAD artery (36.3%), whereas ectasia was commonly seen in RCA (58.6%), followed by LCX (39.2%) and LAD (9.8%). The most frequent pattern observed in this study was Markis Class III (38%).

Table-II: Clinical profile of the patient's clinical presentation.

Variables	n (%)
NSTEMI/UA	54 (31.2)
SIHD	49 (28.3)
AWSTEMI	37 (21..9)
IWSTEMI	22 (12..8)
Atypical angina	6 (3.5)
Valvular heart disease	4 (2.3)

DISCUSSION

CAE isdefined as local or generalized enlargement of the coronary artery by 1.5 times or more than its normal diameter due to an exaggerated form of expansive vascular remodeling. The majority of CAEs commonly found in patients of atherosclerosis and CAD, and likely to be seen in response to atherosclerotic plaque growth[1,7]. The inflammatory response results in an over expression of matrix metalloproteinases, cysteine proteinases and serine proteinases that causes the partial breakdown of the vessel and weakens it resulting in ectasia[8].

The incidence of CAE has been largely analyzed in different regions of the world. Studies conducted in our region show its angiographic prevalence to be around 4 (4.5%) and incidence of isolated CAE without CAD to be 3 (1.9%). In a recent study from our region, included 4,950 coronary angiograms, 270 patients were found to have coronary ectasia (5.4%)[9]. Of them, 52 patients had isolated coronary ectasia (19.2%)[9]. Our study is the first large study in Pakistan which has prospectively tried to evaluate the incidence of CAE in patients undergoing coronary angiography. We found prevalence of CAE to be 3.9% in 4,273 consecutive coronary angiographies performed in our center.

RCA remains the most common artery exhibiting ectatic pattern in most of the previous studies conducted[2,3,5], except asingle studywhich showeda different pattern[4]. Similar observation was also noted in anotherstudy, with 58% of CAE patients having evidence ectatic RCA though reason behind remains unclear. Atherosclerosis and plaque formation is mostly linked with CAE which can involve any of the coronary arteries with no special liking for RCA. Moreover, CAE may involve more than one coronary artery. 32% of patients in this study had multi-vessel CAE, as reported in other studies too.

CAE was most common in RCA but localized aneurysmal dilatation rather than diffuse ectasia was seen predominantly in LAD and similar finding was also observed in a recent study from the neighboring country. Yet *et al*[3], another study conducted in our country showed different results as RCA was the most common vessel with both diffuse ectatic disease as well asaneurysmal formation common in RCA[5]. Since patient population was single centered, the data on different countries could be different due to genetic and environmental differences on atherosclerosis.

Increased incidence of CAE in familial hypercholesterolemia (FH) is indicative of correlation between plasma lipoproteins and aneurysmaldilatation of coronary arteries[10]. The incidence of CAE in FH is considered due to an enzymatic degradation of the extracellular matrix and its interaction with LDL cholesterol. 64 (37.2%) patients had deranged lipid profile in this study.

Over thirty percent of patients with CAE were hypertensive and 47.2% had history of smoking which appears to be more prevalent in patients with CAE than in those with CAD. Both of these risk factors for CAD are strongly associated with CAE in previously conducted studies also[2-5]. Yet, to know about the degree of incidence of CAE associated with each etiologic factor more histophathological studies needs to be conducted. It has been proposed in the past that CAE appears to be multifactorial in many cases.

CAE has an inverse relationship with diabetes mellitus which is as an independent risk factor[11]. In this study, 25 (14.8%) patients had dia-

betes mellitus. Moreover, background frequency of diabetes in our region is almost same[12]. However, diabetes mellitus is not taken as a strong risk factor for aneurysmal dilatation of arteries.

Studies have shown that the clinical presentation of CAE remains variable, including stable ischemic heart disease, unstable angina, and myocardial infarction. It has been observed that CAEmay appear in patients without CAD which has been linked with various connective tissue disorders. The most common clinical presentation in this study was NSTEMI (31%) followed by SIHD (28%). Some studies conducted in our region have revealed SIHDas predominant disease[3,5]. STEMI is suggested to occur from the sluggish blood flow and distal embolization and complete occlusion of the aneurysmal segment by thrombus formation.

The mainstay therapy of CAE remains controversial. Most clinicians are in favor of long-term anticoagulation therapy[13], while others go for antiplatelet therapy with aspirin only in silent disease. There is controversy regarding beneficial effects of nitrates or calcium channel blockers[14]. However lipid-lowering drugs may be effective in patients with familial dyslipidemia. A few clinicians recommend IVUS for an accurate assessment of percentage vessel narrowing and to differentiate true aneurysms[15]. Percutaneous coronary intervention and coronary artery bypass grafting have similar results for CAE in a recent study[16].

Till date, a few studies are available favoring the revascularization of stenotic ectatic vessels. Hence, drug-eluting balloon-expandable stents and frequently self-expanding stents (SESs) are chosen by interventionists due to their tendency to maintain an appropriate apposition of stent struts by its constant gentle outward force. Also, some new generation Ses Stentys stents made up of nitinol and nickel–titanium is effectively self-expansible and apposes well to the vessel lumen[17]. Also, placement of a covered balloon-expandable Jostent (JOMED)appears to be an appropriate device for excluding aneurysmal coronaries[18]. Existence of thrombus may require to maintain chronic anticoagulation therapy e.g. glycoprotein IIb/IIIa inhibitors. Also thrombus aspiration during percutaneous coronary interventions should be offered. Surgical revascularization techniques like surgical excision or ligation of the aneurysm and grafting an ectatic or aneurysmal vessel by coronary artery bypass surgery is the preferred treatment in most of the cases aneurysmal dilatation is not amenable for percutaneous coronary intervention.

There are a few constraints in this studydue to a lack of follow-up patients and not knowing the nature of these aneurysmal lesions. The present study lacks detection of diagnostic markers of inflammatory response such as C reactive protein, interleukin[6], tumor necrosis factor alpha. Further, this study does not explain much about the precise incidence of CAE in our general population.

Data Analysis

Categorical variables are expressed as percentages and continuous variables are as mean ± standard deviation.

CONCLUSION

In this study, we found CAE to be presentin 3.9% of patients undergoing coronary angiograms and these results were found comparable to international literature. The prognosis and outcome of CAE is obscure and there is lack of evidence based treatment in this disorder. Hence, more prospective large-scale studies needs to be done in future.

CONFLICT OF INTEREST

This study has no conflict of interest to be declared by any author.

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