Impact of Coronary Artery Ectasia on Myocardial Perfusion Scan

Thesis

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List of Abbreviations

Abbreviation	Description
201Tl	Thallium 201
2D	Two-Dimensional
99mTc	Technitium
CA	Coronary Angiogram
CAD	Coronary Artery Disease
CAE	Coronary Artery Ectasia
CE	Coronary Ectasia
DM	Diabetes Mellitus
e.g.	For Example
ECG	Eclectrocardiogram
ECM	Extracellular Matrix
F.H.	Family History of premature coronary artery disease
HR	Heart Rate
HTN	Hypertension
I.V.	Intravenous Injection
INR	International Normalized Ratio
LAD	Left Anterior Descending (artery)
LCX	Left Circumflex (artery)
LV	Left Ventricle
mCi	Millicurie
mg	Milligram
MMP	Matrix metalloproteinase

Abbreviation	Description
MMPs	Matrix Metalloproteinases
MR	Magnetic Resonance
MRI	Magnetic Resonance Imaging
NO	Nitrous Oxide
NO	Nitric Oxide
RCA	Right Coronary Artery
RV	Right Ventricle
SD	Standard Deviation
SDS	Summed Difference Score
SPECT	Single Photon Emission Computed Tomography
SRS	Summed Rest Score
SSS	Summed Stress Score

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INTRODUCTION

Coronary artery ectasia (CAE) is a relatively common entity characterized by inappropriate dilatation of the coronary vasculature. The most commonly used angiographic definition of CAE is the diameter of the ectatic segment being more than 1.5 times larger compared with an adjacent healthy reference segment **(Yilmaz et al., 2008)**. CAE is mainly encountered in males in most of the patients it seems to be related to coronary atherosclerosis **(Pagel et al., 2002)**.

More than half of CAE are due to coronary atherosclerosis, but occasionally they are related to other pathological entities. As the first report of coronary dilatation in a patient with syphilitic aortitis, CAE has been observed in association with connective tissue disorders such as scleroderma, Ehler-Danlos syndrome and polyarteritis nodosa but also with bacterial infections and the Kawasaki disease. In a small percentage of patients CAE can be congenital in origin **(Díaz-Zamudio et al., 2009)**. Coronary artery ectasias are attributed to atherosclerosis in 50%, whereas 20% to 30% have been considered congenital in origin. Only 10% to 20% of CEA have been described in association with inflammatory or connective tissue diseases **(Mavrogeni et al., 2010)**.

The specific causative mechanisms of abnormal luminar dilatation in CAE are essentially unknown. The hypotheses for

the origin of CAE revolve around the vascular endothelium and the biological properties of the arterial wall. CAE occurs due to two different mechanisms in two distinct patient groups, first group, in patient without coronary atherosclerosis as a result of exogenous interstitial NO vascular over stimulation. The second group, in patients with concomitant coronary artery disease due to severe and chronic arterial inflammation **(Virmani et al., 1986).**

Ectasia was classified according to the extent of involvement of the coronary vessels, with type I representing diffuse ectasia of two or more major vessels, type II diffuse ectasia in one vessel and localized disease in another, type III diffuse ectasia of one vessel only and type IV localized involvement only. In addition, CAE was classified according to the anatomical shape of the ecstatic segment in fusiform or saccular types **(Markis et al., 1976)**.

The clinical symptomatology consists of a typical exercise-induced angina pectoris. As a complication, myocardial infarction may occur, caused by repeated dissemination of micro emboli to segments distal to the ectasia or aneurysm or by a thrombotic occlusion of the dilated vessel **(Al-Harthi et al., 1991; Rab et al., 1990; Rath et al., 1985)**. Pure coronary ectasia can be implicated in angina or myocardial infarction, worse prognosis depends on the association of stenotic coronary artery disease **(Varol et al., 2009; Kühl et al., 2008)**.

AIM OF THE WORK

The purpose of our study is to investigate the prevalence and the pattern of myocardial ischemia in patients with pure coronary arteries ectasia as assessed by stress – rest gated Tc sestamibi.

Chapter (1)

CORONARY ARTERY ECTASIA

Coronary artery ectasia (CAE) is a well recognized, relatively common abnormality of the coronary anatomy that has gained a lot of attention over the past few decades as a separate entity of coronary artery disease (CAD) **(Akyurek et al., 2003)**. Its importance comes from the fact that patients suffering from CAE may present with acute coronary syndrome (ACS) just like those having stenotic atherosclerotic CAD.

The condition was first described by **Bourgon** in 1812 as a postmortem finding, while the term "ectasia" was first coined by **Bjork** in 1966 **(Figure 1).**

The advent of cardiac catheterization allowed the diagnosis to be made in life and helped more with determination of associations with the disease **(Hartnell et al., 1985)**. Yet, its etiology, pathophysiology, treatment and prognosis are all still questionable and need further research and studies.

Fig. (1): Coronary arteries obtained from autopsy sample **(Sorrell et al., 1996).**

Definition:

CAE is angiographically defined as an abnormal irregular dilatation of an arterial segment at least 1.5 times more than an adjacent healthy reference segment or an adjacent normal vessel **(Hartnell et al., 1985)**.

CAE may occur alone in a condition known as "dilated coronopathy" or in association with atherosclerotic CAD.

Incidence:

CAE can be found in 0.22% to 1.4% of autopsy series **(Hartnell et al., 1985)**. In the largest series from the CASS registry, CAE was found in 4.9% of more than 20000 coronary angiograms they reviewed **(Swaye et al., 1983).**

All three coronary vessels can be affected by CAE, but in almost 75% of patients an isolated artery is ectatic **(Al-Harthi et al., 1991)**.

The proximal and mid segments of the RCA are the most commonly involved in CAE, followed by the LAD and LCX, LMCA is the least affected vessel, however, with regard to CAD, the LAD is most commonly affected, followed by the RCA and LCX **(Giannoglou et al., 2006)**. The reason for the higher RCA predisposition to CAE is not well understood.

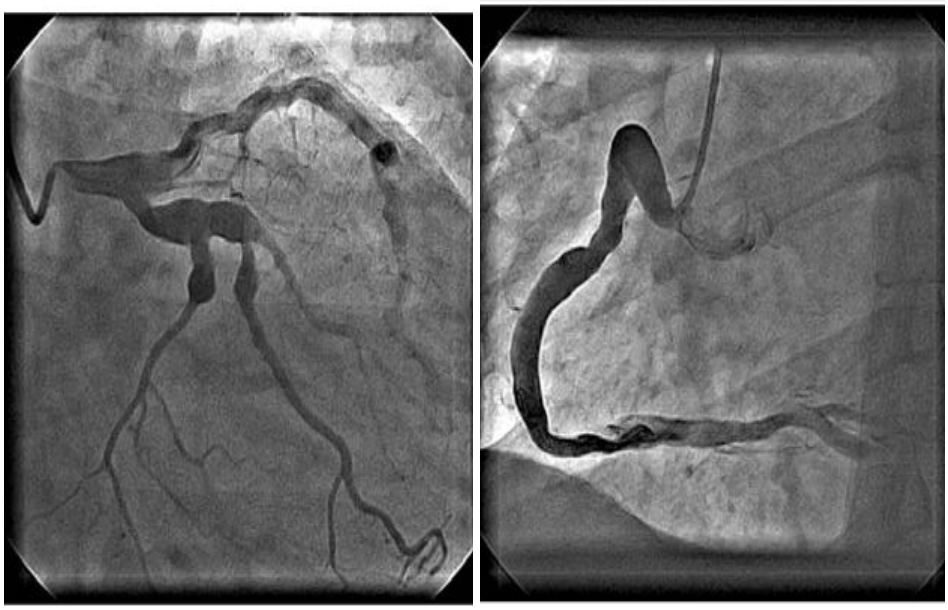
In patients with CAD coexisting with CAE, 34% of the stenotic lesions were in the vessels affected by the ectatic process, while 65% were in the nonectatic vessels. It has also been demonstrated that total CAD severity, expressed as the number of coronary stenotic lesions per patient, is found to be equivalent in patients with CAD with and without coexisting CAE **(Demopoulos et al., 1997)**.

Classification of CAE:

The first attempt for classification was proposed by **Markis et al. in 1976**, who classified CAE, based on the extent of ectatic involvement in descending order of severity into four types:

- Type I, Diffuse ectasia of two or three vessels.
- Type II, Diffuse disease in one vessel and localized disease in another vessel.
- Type III, Diffuse ectasia of one vessel only.
- Type IV, localized or segmental ectasia.

CAE has also been classified according to the anatomical shape of the ectatic segment, into fusiform or saccular types **(Befeler et al., 1977)**. Older studies preferred the term 'coronary aneurysm' for the more discrete and saccular type ectatic segments, reserving the term 'ectasia' for the fusiform diffuse vessel involvement **(Tunick et al., 1990) (Figure 2).**



(A) **(B)**

Fig. (2): Different angiographic views showing: (A) Diffuse ectasia of LMCA, proximal LAD&LCX focal ectasia. (B) Diffuse RCA ectasia **(Helmy et al., 2009).**

Etiology and Pathogenesis:

The exact causative mechanisms of abnormal luminal dilation in CAE are essentially unknown, but evidence suggests a combination of genetic predisposition, common risk factors