

***Prevalence and Morphology of
Coronary Artery Ectasia with
Dual-source CT Coronary Angiography***

Thesis

Submitted for Partial Fulfillment of Master Degree in Cardiology

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2014

Acknowledgment

*Fist thanks to **Allaht** whom I relate any success in achieving any work in my life.*

*I wish to express my deepest thanks, gratitude and appreciation to **Professor Dr. Mona Mostafa Rayan, professor of Cardiology** for her meticulous supervision, kind guidance, valuable instructions and generous help.*

*I am deeply thankful to **Dr. Sameh Sameer, Lecturer of Cardiology** for his great help, outstanding support, active participation and guidance.*

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LIST OF ABBREVIATIONS

Abb.	Meaning
AAA	Abdominal Aortic Aneurysm
ACS	Acute coronary syndrome
ALP	Apolipoprotein
APS	Antiphospholipid syndrome
AS	Ankylosing spondylitis
BMI	Body mass index
CAA	Coronary artery aneurysm
CABG	Coronary artery bypass graft
CAD	Coronary artery diseases
CAE	Coronary artery ectasia
CCA	Catheter Coronary Angiography
CREST	Calcinosis, Raynaud phenomenon, Esophageal motility disorders, Sclerodactyly, and Telangiectasia
CRP	C-reactive protein
CTCA	Computed tomographic coronary angiography
CVA	Cerebrovascular accident
CVD	Cerebrovascular diseases
DM	Diabetes mellitus

LIST OF ABBREVIATIONS (CONT.)

Abb.	Meaning
DSL	Dyslipidemia
GCA	Giant cell arteritis
HTN	Hypertension
ICAM-1	Intercellular adhesion molecule 1
IHD	Ischemic heart disease
IL-6	Interleukins-6
LAD	Left anterior descending coronary artery
LCX	Left circumflex coronary artery
LM	Left main coronary artery
MI	Myocardial infarction
MMP	Matrix metalloproteinase
MPA	Microscopic polyangiitis
MPI	Myocardial Perfusion Imaging
MRA	Magnetic resonance arteriography
PAD	Peripheral arterial diseases
PAN	Polyarteritis nodosa
PCI	Percutaneous coronary intervention
PET	Positron emission tomography
RA	Rheumatoid arthritis
RCA	Right coronary artery

LIST OF ABBREVIATIONS (CONT.)

SLE	Systemic lupus erythematosus
SPECT	Single photon emission computed tomography
SPSS	Statistical package for special science
TFC	TIMI Frame Count
TIA	Transient Ischemic Attack
VCAM-1	Vascular cell adhesion molecule 1

Introduction

Coronary artery ectasia (CAE) has been observed by pathologists and cardiologists for more than two centuries. As its first description by **Morgagni in 1761** this not so infrequent form of coronary artery disease has puzzled the clinicians regarding its cause, clinical sequel and treatment.

Coronary artery ectasia is defined as a localized or diffuse non-obstructive lesion of the epicardial coronary arteries with a luminal dilation exceeding 1.5 fold the diameter of the normal adjacent arterial segment. Falsetti and Carroll further subdivided CAE into simple ectasia (*a 1.5–2-fold segment dilation*) and an aneurysm (*a >2-fold segment dilatation when compared to a normal segment*) (**Falsetti et al., 1976**).

The main coronary angiographic characteristics of CAE are impaired coronary blood flow, delayed antegrade coronary dye filling, segmental back flow phenomenon (milking phenomenon) and stasis with local deposition of dye in dilated coronary segments (**Krueger et al., 1999**).

The clinical relevance and the pathophysiology of CAE have received increasing attention in the last decades. The exact mechanism of its development is unknown, but evidence suggests a combination of genetic predisposition, common risk

factors for coronary artery disease and abnormal vessel wall metabolism.

Functional loss of the musculoelastic components of the coronary artery media is considered the predominant aspect in the pathogenesis of CAE. (*Befeler et al., 1977*) Due to the frequent coexistence of CAE with Coronary artery disease (CAD), and as the histopathological characteristics are similar to coronary atherosclerosis, it is not surprising that the hypotheses for the origin of CAE revolve around the vascular endothelium and the biological properties of the arterial wall. However, there are some differences in the proven association between CAE and CAD.

In addition *non-atherosclerotic forms* of CEA have been described with an intact vessel intima, but with extensive media degeneration (smooth muscle cell replacement by hyalinized collagen) (*Rath et al., 1985; Mattern et al., 1972*).

The clinical course of CAE mainly depends on whether it is isolated or coexisted with CAD. Patients with coronary ectasia and coronary obstructive disease are similar in every aspect to those with similar coronary obstructive disease but without ectasia during follow-up of 5 years(*Swaye et al.,1983*).

Pure ectasia is not completely innocuous, as there is an appreciable incidence of previous myocardial infarction and angina (*Demopoulos et al., 1997*). Ectatic arteries have been shown to be more prone to spasm (*Suzuki et al., 1994*),

exercise induced myocardial ischemia (*Kruger et al., 1999*), thrombosis (*Perlman et al., 1989*), dissection (*Huikuri et al., 1991*), or rupture (*Satoda et al., 1998*). Moreover, the severity of myocardial ischemia has been significantly correlated with the degree of luminal enlargement(*Kruger et al., 1999*).

The detection of CAE is important as patients might benefit from medical treatment or surgery (*Ramappa et al., 2007*). The latter treatment is recommended in patients with CAE complications and for saccular CAE because of the higher risk of thrombosis and rupture.

With the widespread use of coronary angiography the incidence of CAE in patients undergoing this diagnostic procedure was clearly delineated. CAE has been found in 1–5% during coronary angiography (*Swaye et al., 1983; Demopoulos et al., 1997; Markis et al., 1976*). In the largest series from the CASS registry found CAE in 4.9% of more than 20 000 coronary angiograms they reviewed (*Swaye et al., 1983*). The incidence of CAE in an Indian patient cohort with ischemic heart disease has been reported to exceed 10%. It is reasonable to expect that the incidence of CAE reported in the literature overestimates the true frequency in the general population, since the standard for diagnosis of CAE is coronary angiography, and patients referred to coronary angiography are pre-selected.

In order to clarify anatomical variations, **Markis** proposed a classification of CAE based on the extent of ectatic involvement. In decreasing order of severity, diffuse ectasia of two or three vessels was classified as **Type I**, diffuse disease in one vessel and localized disease in another vessel as **Type II**, diffuse ectasia of one vessel only as **Type III**, and localized or segmental ectasia as **Type IV** (*Markis et al., 1976*).

In addition, CAE has been classified according to anatomical shape of the ectatic segment in fusiform or saccular type. (*Befeler et al., 1977*).

Older studies preferred the term ‘coronary aneurysm’ for the more discrete saccular type ectatic segments, reserving the term ‘ectasia’ for the fusiform diffuse vessel involvement(*Markis et al.,1976; Tunick et al., 1990*).

For the last four decades, since **Daoud in 1963** stated ‘*indeed no case of coronary aneurysm has yet been diagnosed antemortem*’, coronary angiography was the gold standard for the assessment of CAE.

Recent advances in multi-detector row computed tomography coronary angiography (CTCA) have continuously increased its role in non-invasive imaging of the coronary arteries. The most recent technical innovation is dual-source CT which is characterized by a high and consistent temporal resolution of 83ms through simultaneous acquisition of data with two X-ray tubes and detectors(*Leber et al., 2005*).