Ain Shams University
Faculty of Medicine
Department of Anesthesia & Intensive Care

CURRENT CONCEPTS IN DIAGNOSIS AND MANGEMENT OF ACUTE CORONARY SYNDROMES

An Essay Submitted For Partial Fulfillment of Master Degree in Intensive care

By

Noha Yosri Mahmoud Atia

M.B.,B.Ch.(2006)

Supervised By

Prof.Dr./ Samir Abd El Rahman El Sebaie Talkhan

Professor of Anesthesia and Intensive Care
Faculty of Medicine -Ain Shams University

Dr. / Ayman Ahmed Al-Sayed Abdellatif

Assistant Professor of Anesthesia and Intensive Care
Faculty of Medicine-Ain Shams University

Dr. / George Michael Khalil

Lecturer of Anesthesiology and Intensive Care
Faculty of Medicine-Ain Shams University

Faculty of Medicine
Ain Shams University
2013

Acknowledgment

*To **Allah**, goes all my deepest gratitude and thanks for achieving any work in my life.*

*I would like to express my profound gratitude & deepest appreciation to Dr. **Samir Abd El Rahman El Sebaie Talkhan** Professor of Anesthesiology and Intensive care, Faculty of Medicine, Ain Shams University, for his valuable instructions & for the time & effort he devoted throughout the entire course of the study.*

*I am deeply thankful to **Dr. Ayman Ahmed Al-Sayed Abdellatif** Assistant Professor of Anesthesiology and Intensive care, Ain Shams University, for his willing assistance& enlightening comments.*

*I would like to extend cordial appreciation to **Dr. / George Michael Khalil** Lecturer of Anesthesiology and Intensive care, Ain Shams University, for his constant advice , and creative thoughts .*

*Last but not least, my deep appreciation is expressed to **all members of my family** for their cooperation and encouragement.*

Contents

LIST OF FIGURES

LIST OF TABLES

List Of Tables

LIST OF ABBREVIATIONS

Abbreviation	Meaning
ACC	American College of Cardiology
ACE	Angiotensin-converting enzyme
ACS	Acute coronary syndrome
ACT	Activated clotting time
ADP	Adenosine diphosphate
AF	Atrial Fibirilation
AHA	American Heart Association
aPTT	Activated partial thromboplastin time
AMI	Acute Myocardial Infarction
ARB	Angiotensin receptor blocker
ASA	Acetyle Salysilic Acid
AV	Atrioventricular
BMS	Bear Metal Stent
BNP	Brain natriuretic peptide
CABG	Coronary bypass graft surgery
CCBs	Calcium Channel Blockers
CHD	Coronary heart disease
CHF	congestive heart failure
CK	Creatinine kinase
CKD	Chronic Kidney Disease
CK-MB	Creatinine kinase myocardial band
CMR	Cardiac Magnetic Resonance
COX	Cyclo-oxygenase
CPR	Cardio pulmonary ressussitation
CRF	Chronic renal failure
CRP	C-reactive protein
CrCl	Creatinine clearance
CRT	cardiac resynchronization therapy
CT	Computed tomography
CS	Coronary sinus
cTnT	Cardiac troponin T
cTnI	cardiac troponin I
CX	Circumflex
D	Diagonal branch
DAPT	Dual antiplatelet therapy
DES	Drug-eluting stent
Dl	decilitre
DPTI	Diastolic pressuer time index
DTI	Direct thrombin inhibitor
ED	Emergency depa
e.g.	for example
ECG	Electrocardiogram

EF	Ejection fraction
EMS	Emergency medical service
EVR	Endo cardial viability ratioqddwv`c`2a`
Factor- Xa	Activated factor-X
FDA	Food Drug Administration
FFR	Fractional flow reserve
FMC	First medical contact
GCSF	Granulocyte -macrophage colony stimulating factor
GCV	Great cardiac vein
GFR	Glomerular filtration rate
GPIIb/IIIa inhibitors	Glypoprotein IIb/IIIa inhibitors
GRACE	Global registery of acute coronary event
HDLc	High-density lipoprotein cholesterol
HF	Heart faliuer
HIT	Heparin-induced thrombocytopenia
HMG COA	Hydroxy methyl glutaryl co enzyme A
HsCRP	High-sensitive c-reactive protein
IABP	Intra aortic ballon pump
ICD	implantable cardioverter defibrillator
ICH	Intra cranial haemorrhage
i.e.	that is
INR	Intrnational normalization ratio
IU	International units
IV	Intravenous
Kg	Kilogram
LAD	Left anterior decesending
LBBB	Left-bundle branch block
LCA	Left coronary artery
LDLc	Low-density lipoprotein cholesterol
LMWH	Low molecular weight heparin
LV	Left ventricular
LVF	Left ventricular function
MACE	Major adverse cardiac events
MBF	Myocardial biood flow
MCV	Middel cardiac vein
mg	milligram
MI	Myocardial infarction
mL	millilitre
mm	millimeter
MMPs	Matrix metallo proteinases
NO	Nitric oxide
NOS	Nitric oxide synthase
NSAID	Non-steroidal anti-inflammatory drug
NSTEMI	Non-ST elevation myocardial infarction
NTG	Nitroglycerin

NT-proBNP	N-terminal pro-hormone brain natriuretic peptide
PCI	Percutaneous coronary intervention
PDA	Posterior descending artery
PED	Phospho di esterase
PET	Positron Emission tomography
RCA	Right coronary artery
RV	Right ventricle
SA	Sinuatrial
ST	Stress test
STEMI	ST-elevation myocardial infarction
TIA	Transite ischemic attacks
TIMI	Thrombolysis in myocardial infarction
TNF	Tumor necrosis factor
TTI	Tention time index
UA	Unstable Angina
UFH	Unfractionated heparin
VCAM1	Vascular cell adhesion molecule 1
VT	Ventricular tachycardia
VWF	Von willbrand factor

Introduction

Acute coronary syndrome (ACS) describes the spectrum of clinical manifestations which follow disruption of a coronary arterial plaque, complicated by thrombosis, embolization and varying degrees of obstruction to myocardial perfusion. The clinical features depend upon extent and severity of myocardial ischemia. Complete coronary occlusion in the absence of collateral perfusion results in ST-segment elevation myocardial infarction (STEMI) or non ST-segment elevation myocardial infarction(NSTEMI).The release of sensitive markers of myocardial necrosis (e.g. troponins) is regarded as indicative of myocardial cell necrosis and fulfills the definition of myocardial infarction. If no rise in markers is detected, the term unstable angina (UA) is used and non-cardiac differential diagnosis must be considered **(Hammet al, 2001).**

Advances in our understanding of atherogenesis processes that lead to plaque rupture and the pathophysiologic hallmark of ACS allow for more precise identification, risk stratification and treatment in the future**(Faxonetal, 2004).**

Although timely and appropriate treatment reduces the risk of immediate or subsequent poor outcome, the high prevalence of risk factors for coronary Heart disease (CHD) ensures that the prevalence of future ACS will also be high. Multiple risk factors may play important role in the process of the ACS include smoking, hypertension, diabetes mellitus, dyslipideamia, metabolic syndrome and obesity. Unfortunately, the prevalence of risk factors is stratified by economics, educational level and cultural differences, among other factors, which

puts patients who may be least likely or the least able to seek skilled care at the most risk for poor outcomes **(Jeffery et al, 2007)**.

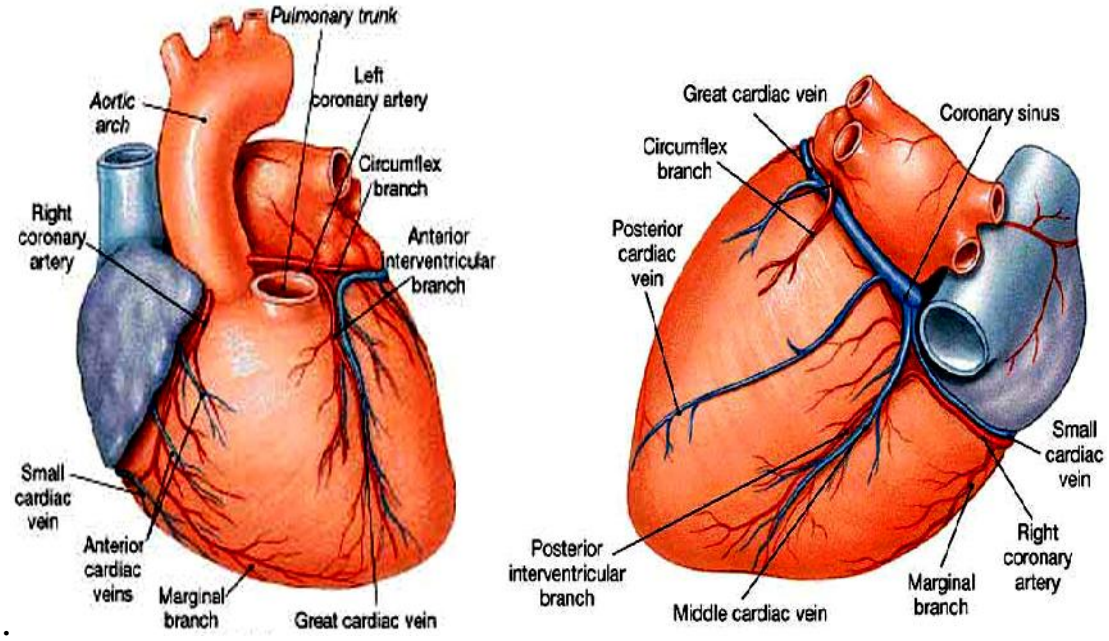
Prompt diagnosis and treatment offer the greatest potential benefits for the myocardial salvage in the first hours of STEMI and early focused management of UA and NSTEMI reduces adverse events and improve outcome. Thus, it is important that healthcare providers recognize patients with potential ACS in order to initiate the evaluation and appropriate management as early as possible. In the case of STEMI, this recognition also allows prompt notification of the receiving hospital and preparation foremergent reperfusion therapy**(Steget al, 2003)**

Prognosis in ACS is based on the extent of coronary disease and left ventricular (LV) function, overlaid with the short term risk associated with the culprit lesion and the unstable state. The short term risk is related almost entirely to myocardial infarction (MI) and its complications and to the recurrences of unstable angina. Risk is highest in the hours, days and first month after the onset of symptoms. **(Bounhoureet al, 2004)**

Applied anatomy and physiology of coronary circulation

Coronary circulation is the circulation of blood in the blood vessels of the heart muscle (the myocardium). The vessels that deliver oxygen-rich blood to the myocardium are known as coronary arteries(fig 1). The vessels that remove the deoxygenated blood from the heart muscle are known as cardiac veins**(Fusteret al, 2001).**

An overview of the coronary arteries:



(A) Anterior View **(B) Posterior View**

Fig 1;An anterior and posterior views for coronary arteries (Susan, 2004)

- Left Main or left coronary artery (LCA)
  - Left anterior descending (LAD)
    - diagonal branches (D1, D2)
    - septal branches
  - Circumflex (CX)
    - Marginal branches (M1,M2)
    -
  - Right coronary artery
  - Acute marginal branch (AM)
  - AV node branch
  - Posterior descending artery (PDA)

LCA

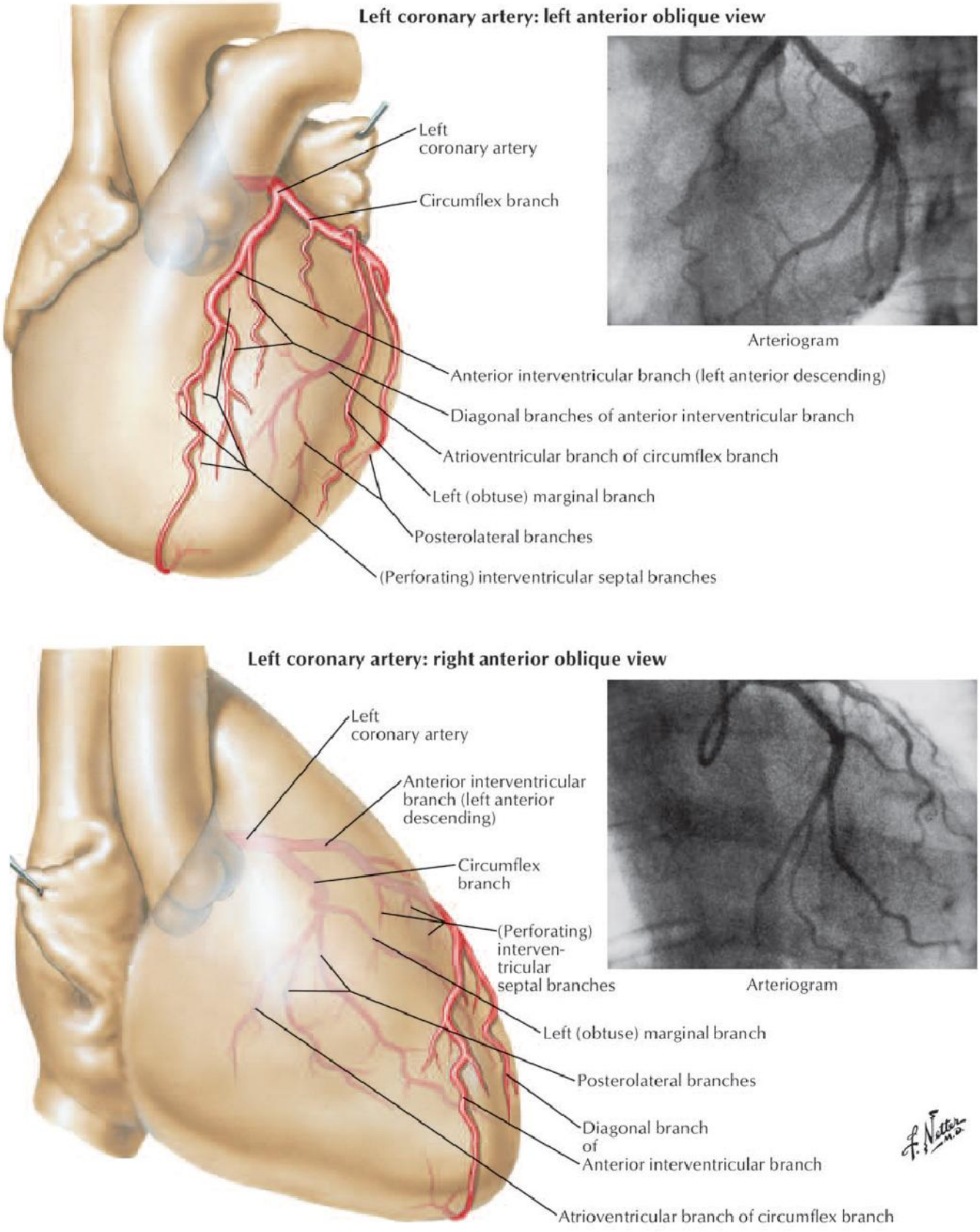


FIGURE 2. Diagram showing the left coronary artery.**(David,2007)**

The left coronary artery (LCA) is also known as the left main (fig 2).
The LCA arises from the left coronary cusp.

The aortic valve has three leaflets, each having a cusp or cusp-like configuration.
These are known as the left coronary cusp (L), the right coronary cusp (R) and the posterior non-coronary cusp (N).
Just above the aortic valves there are anatomic dilations of the ascending aorta, also known as the sinus of Valsalva. The left aortic sinus gives rise to the left coronary artery, and the right aortic sinus which lies anteriorly, gives rise to the right coronary artery. The LCA travels between the right ventricle outflow tract anteriorly and the left atrium posteriorly and divides into LAD and CX**(Robinet al, 2008).**

LAD

The LAD travels in the anterior interventricular groove and continues up to the apex of the heart. The LAD supplies the anterior part of the septum with septal branches and the anterior wall of the left ventricle with diagonal branches.
The LAD supplies most of the left ventricle and also the AV-bundle.

CX

The CX lies in the left AV groove between the left atrium and left ventricle and supplies the vessels of the lateral wall ofthe left ventricle.
These vessels are known as obtuse marginals (M1, M2…), because they supply the lateral margin of the left ventricle and branch off with an obtuse angle.
In most cases the CX ends as an obtuse marginal branch, but 10% of patients have a left dominant circulation in which the CX also supplies the PDA.

In 15% of cases a third branch arises in between the LAD and the CX, known as the ramus intermedius or intermediate branch .This intermediate branch behaves as a diagonal branch of the CX**(Robin et al, 2008).**