Acute Coronary Syndrome – Part I

Evaluation

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ACS Defined

A spectrum of clinical syndromes which includes unstable angina (UA), myocardial infarction (MI) and sudden ischemic death.

Leading cause of morbidity and mortality in the US.
Clinical manifestations of plaque rupture resulting in subtotal or total occlusion of the affected artery.
ESC/ACC DEFINITION OF MI

• Pathological findings of an acute MI

  OR

• Myocardial necrosis as indicated by elevated biochemical markers such as troponin (with rise and gradual fall) or CK-MB (which rises more rapidly).

• AND at least 1 of the following:
  
  a. Ischemic symptoms (chest pain, dyspnea, weakness)
  
  b. Development of pathological Q waves on the ECG
  
  c. ECG changes indicative of ischemia (ST-segment elevation or depression.
  
  d. Coronary artery intervention history (e.g., coronary angioplasty)
The Various Syndromes

- **Stable Angina**
  - vague chest or arm discomfort (rarely described as pain), is reproducibly associated with physical exertion or stress, and is relieved within 5-10 minutes by rest and/or sublingual nitroglycerin.

- **Unstable Angina**
  - angina pectoris or equivalent ischemic discomfort with at least one of three features:
    - occurring at rest (or minimal exertion) usually lasting > 20 minutes
    - being more severe and/or of new onset (i.e., within the prior 4-6 weeks)
    - occurring with a crescendo pattern (i.e., more severe, prolonged, or frequent than previously)

- **NSTEMI**
  - Symptoms with evidence of myocardial necrosis from cardiac serum markers

- **STEMI**
  - Symptoms with evidence of myocardial necrosis from cardiac serum markers and characteristic ECG changes
Chest Pain

- Chest pain is the most common symptom associated with ACS. Pain is described as deep visceral pain "heavy" "squeezing" "crushing" typically substernal with varying radiation to neck, jaw, shoulders or arms.
Angina Type I

Atypical chest pain:

Pain, pressure, or discomfort in the chest, neck, or arms not clearly exertional.

Or not otherwise consistent with pain or discomfort of myocardial ischemic origin MI
Angina Type II - Stable

Angina without a change in frequency or pattern for a period of 6 weeks.

Angina controlled by rest and/or sublingual / oral / transcutaneous medications.

Vague chest or arm discomfort (rarely described as pain), is reproducibly associated with physical exertion or stress, and is relieved within 5-10 minutes by rest and/or sublingual nitroglycerin.
Angina Type III-Unstable

1. Angina pectoris or equivalent ischemic discomfort with at least one of three features: Occurs at rest and is prolonged, usually lasting more than 20 minutes

2. Being more severe and/or of new onset (i.e., within the prior 4-6 weeks)

3. Recent acceleration (occurring with a crescendo pattern i.e., more severe, prolonged, or frequent than previously) of angina reflected by an increase in to at least one CCS class.
Canadian Cardiovascular Society
Angina Classification

- Class 0: Asymptomatic
- Class 1: Angina with strenuous exercise
- Class 2: Angina with moderate exertion
- Class 3: Angina with mild exertion
  - Walking 1-2 level blocks at normal pace
  - Climbing 1 flight of stairs at normal pace
- Class 4: Angina at rest
Angina Type IV: MI

ST vs. non-ST segment elevation MI

a. ST-elevation MI

b. Non ST-elevation MI

c. Bundle Branch Block can cause uncertainty
American Heart Association Says...

• In 2007 1.2 million Americans will have their first or a recurrent heart attack
• 425,000 will die
• 3/4 of these deaths (~325,000 people) will occur prior to arriving to a hospital or in the emergency department
Yearly ER Visits

- Chest Pain: 6,000,000
- UA/NSTEMI: 1,400,000
- STEMI: 300,000
What Are the Numbers?

• Heart disease, stroke, and other cardiovascular diseases are the No. 1 killers of Americans, male or female

• Heart disease kills nearly 12 times as many American females as breast cancer.

• Men have a greater risk of heart attack than women do, and they have attacks earlier in life.
Risk of Dying from CAD

• One in two
• 50%
• Half
• Flip of a coin
Significance of ACS?

- One third of patients who experience STEMI will die within 24 hours of the onset of ischemia.
- Of those who survive many will have significant morbidity.
- For many patients, the first manifestation of coronary artery disease is sudden death likely from malignant ventricular dysrhythmia.
Mortality

In-hospital fatalities account for 10% of all deaths. An additional 10% of deaths occur in the first year post-infarction.

- A steady decline has occurred in the mortality rate from STEMI over the last several decades.
  - ↓ incidence of MI
  - ↑ incidence of angina
  - ↓ fatality rate when an MI occurs
Risk Factors That Cannot Be Modified:

- Age. As you get older, your risk for CAD increases.
  - In men, risk increases after age 45.
  - In women, risk increases after age 55.

- Family history of early heart disease.
  - Heart disease diagnosed before age 45 in father or brother.
  - Heart disease diagnosed before age 55 in mother or sister.
Risk Factors That Can Be Modified:

- Hyperlipidemia
- Hypertension
- Cigarette Smoking
- Diabetes
- Overweight or obesity
- Sedentary lifestyle
- Cocaine users
- Elevated hsCRP? (High sensitivity C-reactive protein)
Lower Cholesterol-Lower Risk

- Studies of men have found that for every 1% decrease in blood cholesterol

- There was a 2% decrease in the risk of heart attack.
According to recent estimates, nearly one in three U.S. adults has high blood pressure. But because there are no symptoms, nearly one-third of these people don't know they have it.
Stop Smoking-Lower Risk

• Cigarette smoking is the most important preventable cause of premature death in the United States

• About 37,000 to 40,000 people die from heart and blood vessel disease caused by other people's smoke each year.
Effects of Smoking

• Cigarette smokers have a higher risk of fatty buildups in arteries

• Atherosclerosis (buildup of fatty substances in the arteries) is a chief contributor to the high number of deaths from smoking.

• Many studies detail the evidence that cigarette smoking is a major cause of coronary heart disease, which leads to heart attack.
Control Diabetes-Reduce Risk

• People with diabetes are 2-4 times more likely to develop heart disease

• Diabetics are more likely to have cholesterol abnormalities

• Diabetics are also more likely to have hypertension and develop renal failure

• All of which increase your cardiovascular risk
Control Weight - Reduce Risk

• Obesity, even as little as being 5 to 14 percent overweight, raises the risk of heart attack.
Manage Stress - Reduce Risk

- More and more evidence suggests a relationship between the risk of heart disease and environmental and psychosocial factors.

- These factors include job strain, social isolation and personality traits.
The Impact

• These risk factors are not additive

• Two risk factors such as diabetes and hypertension do NOT make you twice as likely to develop heart disease

• Multiple risk factors multiply your risk
What If It’s Too Late?

- Get aggressive in managing your risk factors
- Research shows that those individuals who make a total overhaul of their lifestyle are the ones most like to significantly reduce their risk
- One risk factor at a time patients do not seem to have the same level of success
Pathogenesis

- The process central to the initiation of an acute coronary syndrome is disruption of an athermanous plaque.

- Fissuring or rupture of these plaques and consequent exposure of core constituents such as lipid, smooth muscle, and foam cells leads to the local generation of thrombin and deposition of fibrin.

- This in turn promotes platelet aggregation and adhesion and the formation of intra-coronary vessel thrombus.
Multiple Triggers

- Plaque rupture with superimposed non-occlusive thrombus
- Dynamic obstruction (e.g., coronary spasm)
- Progressive mechanical obstruction (e.g., re-stenosis)
- Inflammation and/or infection
- Secondary unstable angina, due to increased myocardial oxygen demand (e.g., fever) or decreased supply (e.g., anemia)
- An individual patient will frequently have more than one of these processes as the cause of his or her episode of unstable angina
ACS is an Important Manifestation of Atherothrombosis

- **Plaque rupture**
  - **Old term**: Stable angina, UA
  - **New term**: Atherothrombosis, UA/NSTEMI, STEMI

**UA=unstable angina; NSTEMI=non-ST-segment elevation myocardial infarction; PCI=percutaneous coronary intervention**

Types of Atherothrombotic Lesions Causing Coronary Artery Disease

Stable Angina

- Lumen
- Endothelium
- Platelets
- Thick Fibrous Cap

Non ST ↑ ACS

- Thrombus
- Inflammatory Cells
- Thin Fibrous Cap
- Lipid-Rich Core

MI = myocardial infarction.
Formation of the Platelet Plug

1. Adhesion
   - Platelets
   - vWF/GP Ib bind
   - Collagen
   - GP Ia/IIa bind

2. Activation
   - Thrombin
   - ADP
   - 5-HT
   - TXA₂
   - CD40L

3. Aggregation
   - Activated GP Iib/IIIa
   - Fibrinogen
   - 5-HT

4. Platelet Plug

vWF = von Willebrand factor; GP = glycoprotein; ADP = adenosine diphosphate; 5-HT = 5-hydroxytryptamine (serotonin); TXA₂ = thromboxane A₂.
Diagnosis of AMI

• The initial diagnosis of acute coronary syndrome (ACS) is based entirely on history, risk factors, and, to a lesser extent, ECG findings.

• Cardiac enzymes studies provide clarification on UA vs. AMI.
Patients who present with chest discomfort should undergo early risk stratification that focuses on anginal symptoms, physical findings, ECG findings, and biomarkers of cardiac injury.
History

- Symptom onset refers to the onset of cardiac ischemic symptoms related to this acute event,

- In the event of stuttering symptoms, ACS symptom onset is the time at which symptoms became constant in quality or intensity
Chest Pain

• **Pain:** deep visceral pain "heavy" "squeezing" "crushing" typically substernal with varying radiation to neck, jaw, shoulders or arms.

• Similar to angina but is:

• More severe and lasts longer (up to several hours but usually >30") and may not be completely relieved by NTG)
Signs and Symptoms

- Dyspnea
- Apprehension
- Weakness
- NV
- Diarrhea

- Pale appearance
- Cold extremities
- Diaphoresis
- Fatigue
Clinical Presentation

- The most characteristic presenting complaint is chest pain or an equivalent type of discomfort.
- Usually described as a discomfort or pressure.
- Typically located in the substernal region or in the epigastrium.
- May radiate to the neck, left shoulder, and left arm.

- The physical examination may be unremarkable.
- Physical findings can include diaphoresis, pale cool skin, sinus tachycardia, a 3rd or 4th heart sound, basilar crackles, and sometimes hypotension.
Physical Exam: Vital Signs

- Heart Rate: May be slow, normal or fast
- Sinus tachycardia, atrial fibrillation or flutter
- Sinus bradycardia most common usually due to an inferior MI (right coronary artery)
- Dysrhythmias may present as an irregular heart beat or pulse
Vital Signs-Blood Pressure

- Hypertension may precipitate MI or it may reflect elevated catecholamines due to anxiety, pain, or exogenous sympathomimetics.

- Hypotension may indicate ventricular dysfunction due to ischemia.

- Hypotension in the setting of MI usually indicates a large infarct secondary to either decreased global cardiac contractility or a right ventricular infarct.
Vital Signs

- **Respirations:** Tachypnea secondary to anxiety and/or pain
- Rales may represent congestive heart failure.
- **Temp:** Low grade fever not uncommon
Clinical Signs

- Ischemia- S4, new MR murmur, paradoxical S2
- Heart failure- increased JVP, lung crackles, S3
- Carotid or femoral bruits, decreased distal pulses
Differential Diagnosis

- Aortic dissection
- Pulmonary embolism
- Musculoskeletal
- Costrochondral pain
- Gastric ulcer
- Gastroesophageal reflux
- Esophageal: spasm / reflux / rupture
- Pleuritis
- Pneumothorax
- Pericarditis
- Herpes zoster
- Depression
- Pancreatitis
- Anxiety
EKG Infarction Pattern Includes:

- T-wave inversion, flat, depressed or inverted, usually symmetrical waves, denotes ischemia.

- S-T segment elevation, (>1-2 mm elevation in consecutive chest leads) usually elevation but can also be depressed. Indicates MI is recent (<2 week old injury) can denote injury

- Q-waves - if significant assists in the diagnosis of the MI. Significant if (a) > 25%-30% of QRS (b) > 0.04 sec or 1 mm in width more commonly observed with a transmural infarction versus non-Q wave (nontransmural) infarction.
• When there is both **ST segment elevation** and **ST-segment depression** in the same tracing, look to the leads with elevation for the primary anatomic location of injury.

• This is true even if the **ST depression** appears more obvious than the elevation (compare lead III in this ECG with V3, for example).
This ECG is 24 hours after presentation. Note the persistent injury pattern in II, III, aVF.

Q-waves have appeared in these leads indicating inferior necrosis or infarction.
There is also persistent ST depression in Leads V1 through V4 and look what has happened to the R-waves in those leads.

They're very tall. This development of tall R-waves in the right precordium should be interpreted as evidence of POSTERIOR myocardial infarction.
STEMI
Echocardiogram

- New wall motion abnormalities
- Caveat - this is very operator and reader dependent
How Are Nuclear Stress Images Read?

Inject sestiMIBI (radioactive tracer) during symptoms

Image later to see if reversible defect
Laboratory Testing

- Laboratory testing has become an important element of modern medicine

- Although the axiom treat the patient not the lab is still true, lab data has become a critical piece of the decision tree
Troponin will remain high for 1–2 weeks after a myocardial infarction.

Unlike Creatinine Kinase (CK), troponin is not generally affected by damage to other muscles.

Therefore muscle injections, accidents, strenuous exercise, and drugs that can damage muscle do not affect troponin levels.

Renal insufficiency can result in minor elevations of troponin and sometimes confuse the picture.
Creatinine Kinase (CK)

- Heart, skeletal muscle, brain
- Rises 6-8 hours and max within 24 hours
- Normalizes in 3-4 days
- Isoenzyme CK - MB commonly used as a screening test.
- Has given way to the use of the more sensitive troponin
Considerations in Interpreting Enzymes

- CPR, cardioversion, IM drugs, MVA can elevate CK secondary to myocardial injury/contusion

- CK-MB may be elevated in absence of elevated CK for 15% of patients with AMI –

- Reperfusion with thrombolytics may alter enzyme profile.
Timing of Release of Various Biomarkers After Acute Myocardial Infarction

[Graph showing the release of biomarkers over time after acute myocardial infarction.]
After initial evaluation a patient should be assigned to one of these four categories:

- 1. a non-cardiac diagnosis
- 2. chronic stable angina
- 3. possible ACS, and
- 4. definite ACS
Clinical features of acute coronary syndrome

Aspirin & analgesia

ST elevation or new LBBB or true posterior MI

ST depression or T inversion

ECG

Normal

Consider alternative diagnoses

STEMI

Unstable angina

NSTEMI

Raised troponin?
Chest Pain Protocol

• Registration-immediate triage for any complaint of chest pain

• Triage-Initiate facility chest pain protocol

• RN evaluation with significant history, vitals signs, ASA, prioritize to telemetry bed
Chest Pain Protocol
Emergency Department

- Telemetry
- IV
- CXR
- Oxygen
- NPO
- Bedrest (BRP)
- Neuro checks
- Medications
- Notify cath lab or cardiology

- Labs-
  - BMP (Basic metabolic panel)
  - BNP
  - CBC
  - CK/Troponin
  - PT/PTT
  - Type & Screen
BMP
Glucose
Calcium
Sodium
Potassium
CO2
Chloride
BUN
Creatinine
ED Evaluation & Management

**EMS assessment and care:**
- Monitor, support ABC
- Oxygen, ASA, NTG and MS (Morphine)
- Obtain 12 lead ECG
ED Evaluation & Management

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Immediate ER assessment:
- Check vital signs, & O₂ sat
- IV access
- Obtain a targeted history
- 12 lead ECG
- Lab studies (cardiac markers)
ED Evaluation & Management

**EMS assessment and care:**
- Monitor, support ABC
- Oxygen, ASA, NTG and MS
- Obtain 12 lead ECG

**Immediate ER treatment:**
- Oxygen
- ASA
- Nitroglycerin
- MS

**Immediate ER assessment:**
- Check vital signs, & \(O_2\) sat
- IV access
- Obtain a targeted history
- 12 lead ECG
- Lab studies (cardiac markers)
ED Evaluation & Management

EMS assessment and care:
Monitor, support ABC
Oxygen, ASA, NTG and MS
Obtain 12 lead ECG

Immediate ER assessment:
Check vital signs, & O₂ sat
IV access
Obtain a targeted history

Immediate ER treatment:
Oxygen
ASA
Nitroglycerine
MS
12 lead ECG
Lab studies (cardiac markers)

Review 12 lead ECG
Now we have diagnosed ACS let’s move on to management.