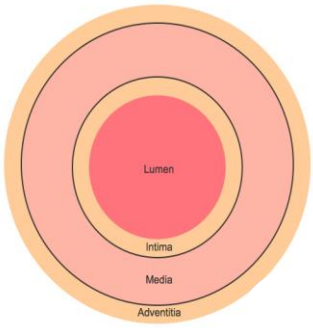
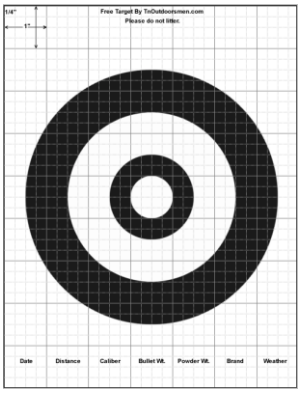
# Acute Coronary Syndrome
# "ACS"

## First of all:

You have to know that:



- Atherosclerosis is a major risk factor for ACS!
- The first layer that gets affected is the endothelium of the vessel
  **(Which vessel do you think will be affected?)**
- Fat deposition occurred in the media of the artery which increased by these

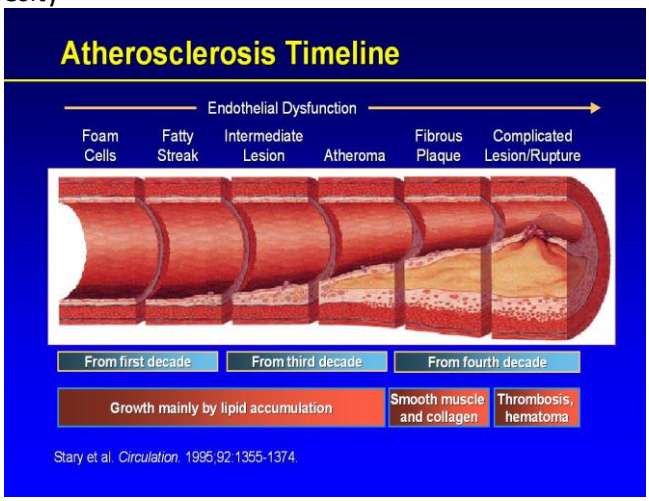
## Risk Factors:

Unmodifiable (it's not up to you to change it):

- Age
- Gender ( male > female )
- Family history of premature atherosclerosis

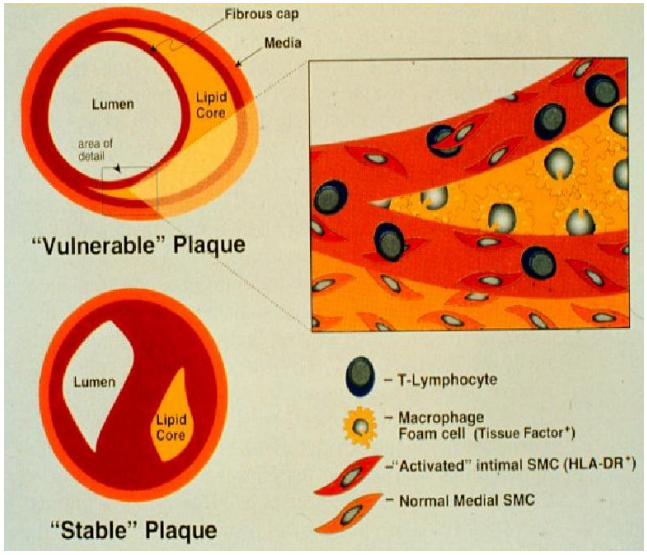
Modifiable:

- Hyperlipidemia
- Hypertension
- Diabetes
- Metabolic syndrome
- Cigarettes smocking
- Obesity




Foam ➔ streak ➔intermediate lesion ➔ atheroma ➔plaque ➔ ASC

## Types of plaques:




**Vulnerable "unstable "plaque**: collection of white blood cells (**inflammation**) and lipids in the lumen of artery = **worsening of the plaque!**
**Stable plaque:** contain thick fibrous cap (**vs. thin in the unstable**) which makes it less likely to rupture and develop an embolus**!**

| Stable plaque | Unstable plaque |
|---|---|
| **Thick fibrous cap** | Thin fibrous cap |
| Small hard lipid core | **Large soft lipid core** |

**Note:** Erosion happens gradually as collagen gets exposed

## Factors contributing Plaque vulnerability:

- Low smooth muscle cell count
- Thin Cap
- Large Lipid Core
- High Macrophage content

### What's ischemia?

It's the imbalance between body demand of oxygen and body supply of it!

### So, what's ACS?

It's the group of these three diseases:

- ST elevated **MI** (**STEMI**) (**complete blockage of lumen**)
- Non ST elevation **MI** (**NSTEMI**)
- Unstable angina ( **UA**)

**UA will eventually lead to MI if not properly managed!**

**Myocardial Necrosis? (Muscle cell death)**

**UA** → **MI**
**No necrosis** — **Myocardial Necrosis**

## Pathway for ASC:

There are many theories about ACS, the most common is:

- Rupture of fibrous cap of coronary artery plaque, which leads to aggregation and adhesion, localized thrombosis and distal thrombus embolism.

- Rupture ➔ thrombus formation and vasoconstriction by platelets ➔ low blood flow ➔ myocardial ischemia

## Clinical presentation:

You can detect those three by regular history, ECG and elevated biomarkers. It's important to know that these three diseases are by ECG.

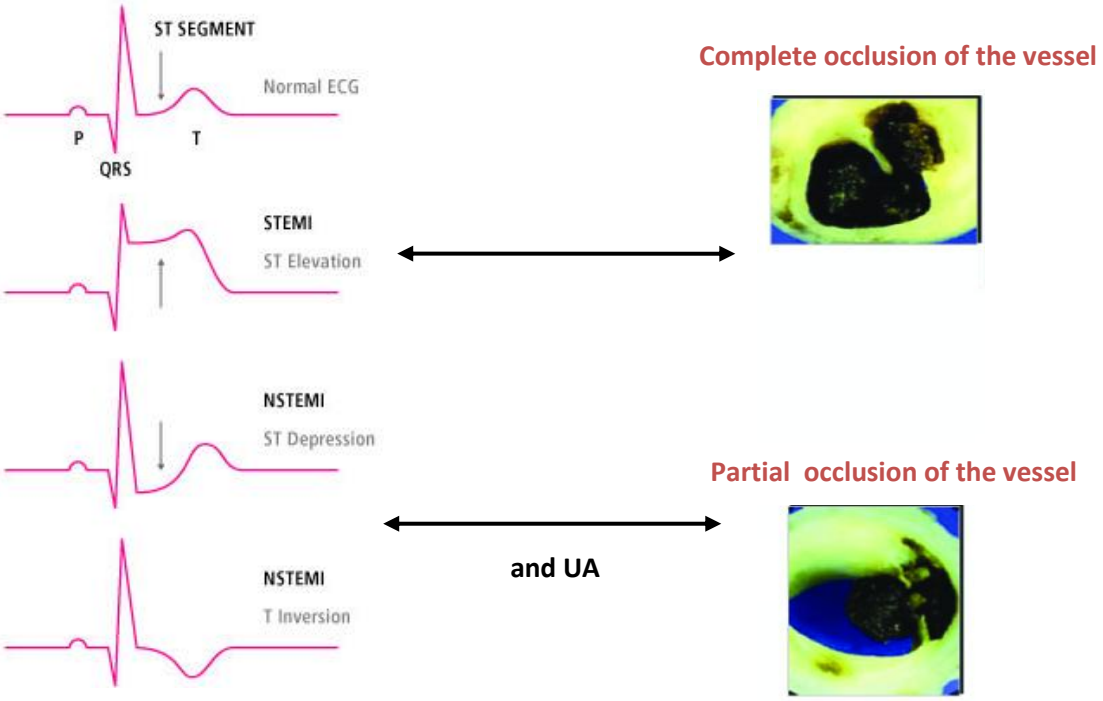
Patient in general will complain of chest pain (**even at rest**)...

**Note**: UA and NSTEMI are usually differentiated from each other by Cardiac enzyme analyses! (**They have the same history, physical examination and ECG findings**)

## Diagnosis of MI:

Typical rise in cardiac troponin T or I , CK-MB with at least one of the following:

- Ischemic symptoms
- Pathological Q wave on ECG
- Ischemic ECG changes (e.g. ST elevation or depression, new LBBB)
- Imaging evidence of new loss of viable myocardium or a new WMA



## Markers of myocardial necrosis:

Note: 20% of patients with acute MI have atypical or show no symptoms (**a silent MI**) , therefore we can assess Myocardial necrosis with appearance of cardiac enzymes in the blood!

### Biochemical markers

- MI causes release of certain enzymes and proteins into the blood stream.
- Creatin Kinase (CK) is released from multiple organs such as the myocardium, skeletal muscles, and the brain.
- The Iso-form CK-MB, is cardiospecific
- Starts to rise 4-6 hrs after onset of ischemia, then falls within **48-72hrs.**

**Cardio specific proteins Troponin I, and T are the most sensitive & specific markers for myonecrosis.**

- Released with 4-6hrs, but can last up to **2 weeks**

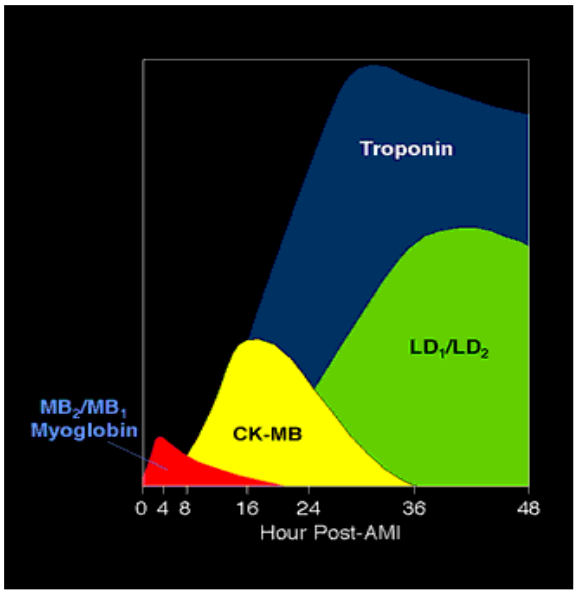



Figure shows: CK-MB and Troponins get released approximately at the same time, but Troponins last for more than 48 hours (in comparison to CK-MB that goes down after 36 hours).

## Other helpful investigations:

- CBC- Leucocytosis
- Elevated ESR
- Chest X-Ray **(** **Pulmonary Edema**)
- Echocardiography
  Wall motion abnormalities, Valvular dysfunction, r/o other causes of chest pain.

# Management

## Aim of therapy

- Improve oxygen supply by :
  1. Supplemental O2
  2. Antiplatelets drugs
  3. Antithrombotics
  4. Coronary vasodilators (**Nitroglycerine**)
  5. Reperfusion therapy: (**TIME IS MUSCLE!**)
     - Fibrinolytic therapy
     - Percutanous coronary intervention (**PCI**)

- Reduce O2 demand
  1. Beta blockers (**Propranolol**, Metoprolol)
  2. Analgesics (**Morphine**)

- Other medications
  - ACE inhibitors( **Enalapril**, **Lisinopril**)
  - Statin therapy

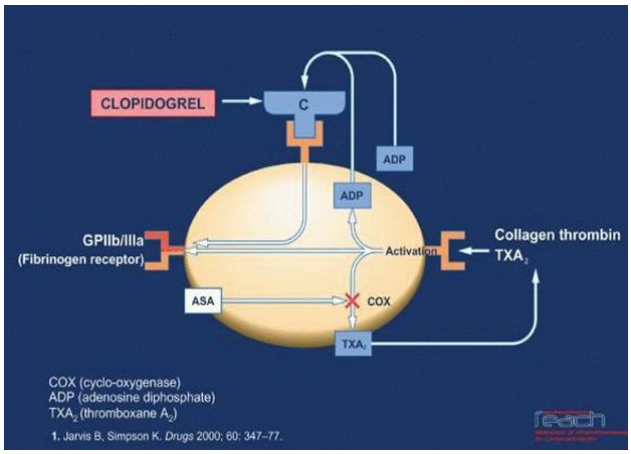
## Antiplatelet:

### Aspirin

- Decreases mortality in MI and should be administered as early as possible and continued indefinitely in patients with ACS.
- Chewable aspirin 160 to 325 mg at presentation, then 75 to 325 mg daily

### Clopidogrel

- More potent than ASA
- Irreversible ADP receptor blockers
- Adjunct to reperfusion therapy



## Antithrombotic:

### Heparin

- Unfractionated
- Low molecular

  - Used for patients with NSTEMI and STEMI
  - Prevents further thrombosis and aids in insuring patency of the occluded artery.

## Immediate! Reperfusion therapy:

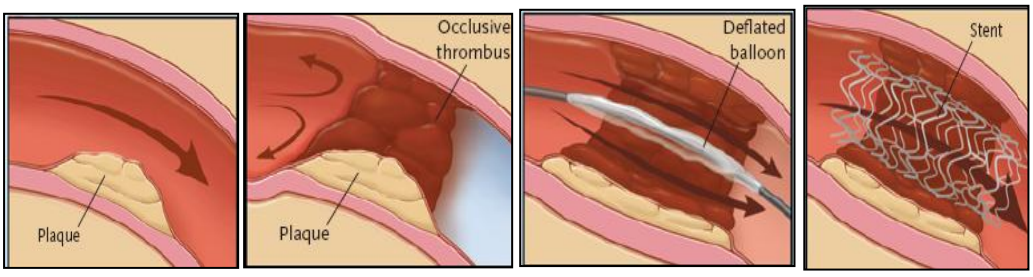
Note: early restoration of blood flow can limit necrosis, improve left ventricular function and reduce mortality rate, especially in STEMI!

- 100,000 patients.
- Should be given during a 12hr window, and given **ASAP**!
- 2 types of fibrinolytics:
  ❶Non Fibrin specific (**Streptokinase**)
  ❷Fibrin specificONLY USED FOR STEMI (NOT NSTEMI)
- Reduces short and long term mortality
- shown to be effective in numerous randomized trials involving over

### Fibrinolytics

- ACE inhibitors( Enalapril, Lisinopril)
- Statin therapy
  - Statin is not only used here for reducing cholesterol level as it works as an anti-inflammatory agent

### Primary PCI



# Complication of MI

It can be categorized into 3 major complications:

Electrical, mechanical and functional

## Electrical complications:

1. Tachyarrhythmias :

a. Ventricular:

- Ventricular Tachycardia
- Ventricular Fibrillation

b. Supraventricular:

- Atrial Fibrillation

2. Bradyarrhthmias

- 1st, 2nd, and 3ed degree AV blocks
- New LBBB, or RBBB

3. Conduction abnormalities

- Bundle branch and fascicular blocks

## Mechanical complications:

1. Mitral regurgitation

- ( 2-7 days post MI)
- Caused by papillary muscle rupture.

2. Free LV wall rupture

- Rare
- 1st 24hr upto 2 weeks

3. Ventricular septal defect

- 1-3%
- Occurs with inferior and anterior MI

## Functional complications

**Note**: the functional abnormality will be localized to the affected myocardial part

e.g. if the MI affected the left ventricle patient will have left ventricular failure

1. Heart failure

   - Bad prognostic sign

   - Reflects the size of the MI

   - ACE inhibitors and diuretics is cornerstone

     Therapy.

2. Cardiogenic Shock

   - Happens with major MI's

   - Carries high mortality ( >50% in 30 days)

   - Should be rushed for cardiac cath and either

     PCI or Coronary bypass grafting.

**That's all :)**

**SS**