Team Medicine

Acute Coronary Syndrome

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Introduction:

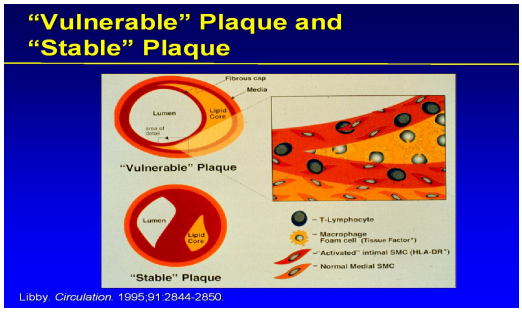
Number one killer. Almost 30% of hospital admission is cardiac related.

Clinical scenario:

History	▣ Saleh, 60yr old man ▣ Owns a businessman ▣ Smoker ▣ Not on medications ▣ No significant past medical history
Physical examination	▣ BP of 150/90 ▣ (repeated 3 times) ▣ Weight 90kg,Height 170 (BMI=31) ▣ Waist Circumference 115 cm
Lab investigation	▣ FBS=9mmol/l ▣ LDL= 4.5 mmol/l ▣ HDL=0.7 mmol/l
Information about the patient	▣ Told that he had DM ▣ Obese ▣ Dyslipidemic ▣ BP was high (likely hypertensive if repeated on future occasions) ▣ Strongly advised to change life style ▣ Undergo another check up in 3 months times after dietary changes
After period of time	▣ Saleh, forgot or was careless ! ▣ 3 yrs later, he started feeling occasional chest heaviness while rushing on the stairs. Radiated to both shoulders, and relived with rest. He decided to go to his physician again….but after finishing off some business deals
	▣ Delayed medical checkup for 3 months! ▣ Woke up at 5 AM with similar chest pain, however now at rest and severe. ▣ Perfuse sweating and nausea ▣ Called his son to take him to the ER
In ER	▣ HR 110bpm ▣ BP 180/100 ▣ O2 Saturation 95% on RA ▣ PE was normal ▣ His situation got worse
Management	Was given chewable ASA ,3 Sublingual Nitro tablets ,He had no contraindications to fibrinolytic therapy ,Just prior to receiving fibrinolytics , he lost consciousness >>>>Defibrillator paddles immediately applied and 200J delivered managed to convert him to normal sinus rhythm

What is Acute coronary syndrome(ACS)?

It is a term that encompasses both unstable angina and MI(STEMI & NSTEMI).Unstable angina is characterized by new onset (less than one month), rapidly worsening angina or angina at restwith the absence of myocardial damage. In contrast, MI occurs when cardiac myocytes die due to myocardial ischemia (there is evidence of myocardial necrosis).ACS may present as a new phenomena or against a background of chronic stable angina. The common mechanism to all ACS is rupture or erosion of the fibrous cap of a coronary artery plaque. This leads to platelet aggregation and adhesion, localized thrombosis, vasoconstriction, and distal thrombus embolism.



Type of plaques:

- Vulnerable.
- Stable.

symptoms:

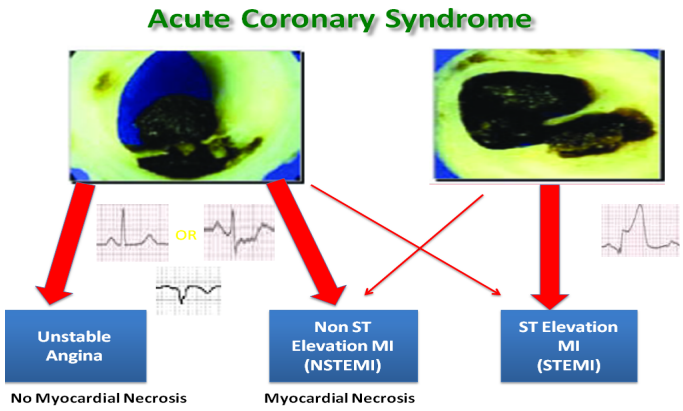
- Typical:
  New onset of chest pain, chest pain at rest or deterioration of preexisting angina
- Atypical:
  -Indigestion.
  -Nausea and vomiting.
  -Pleuritic chest pain.
  -Dyspnoea.
  -Collapse/ syncope.

Differential diagnosis:

depends on the analysis of the character of thepainand its associated features(clinical presentation), evaluation of the ECG, and measurement of biochemical markers.

Investigations:

- ECG.
- Measurement of cardiac enzymes levels(CK-MB – troponin – Myoglobin).
- Complete blood count.
- Chest x-ray.
- Echocardiography.
- Cardiac angiography.



Risk Factors:

- Age : males ≥45, females ≥55
- Gender (Male gender)
- DM
- Dyslipidemia
- HTN
- Smoking
- Family history of Premature CAD: males ≤55 females ≤65

Definition of MI:(Important)

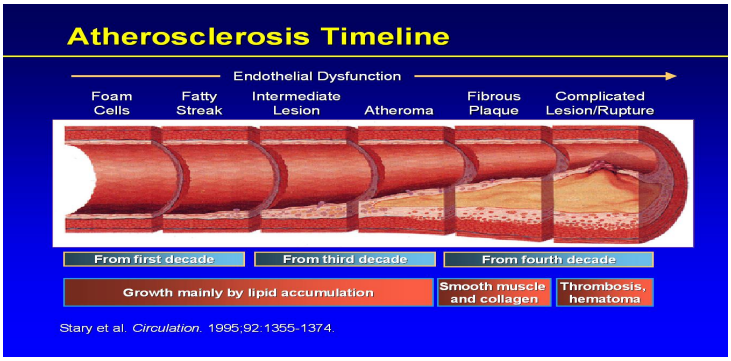
Typical rise in cardiac troponin T or I , CK-MB with at least one of the following:

1. Ischemic symptoms
2. Pathological Q wave on ECG
3. Ischemic ECG changes (e.g. STelevation or depression, new LBBB)
4. Imaging evidence of new loss of viable myocardium or a new WMA
5. Identification of an intracoronary thrombus by angiography or autopsy.

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 cardio-specific
- Starts to rise 4-6 hours after onset of ischemia, then falls within 48-72hrs.
- Cardiospecific proteins Troponin I, and T are the most sensitive & specific markers for myonecrosis.
- Released with 4-6hrs, but can last upto 2 weeks.

Unstable angina has no myocardial necrosis, that's the difference between angina and MI (STEMI & NSTEMI)



Management (immediate management):

Aims of therapy:

-Open Artery and Improve oxygen supply

1. Supplemental O2
2. Coronary vasodilators (Nitroglycerine)
3. Antiplatelet agents
4. Reperfusion therapy
   a. Fibrinolytic therapy
   b. Primary Percutanous coronary intervention (PCI)
5. Antithrombotic agents

Fibrinolytic therapy and PCI are only used in ST elevated MI

-Reduce O_2 demand

1. Beta blockers (Propranolol, Metoprolol)
2. Analgesics (Morphine)

-Other medication:

1. ACE inhibitors(Enalapril, Lisinopril)
2. Statin therapy

-Anterior Infarction (emergency case).
-Full occlusion→ ST elevation.
-Sub occlusion→ no ST elevation(could be depression or inversion)

-Anti platelet agents like Aspirin (ASA) and clopedogril:

Clopedogril is more potent than ASA, both are powerful adjuncts to reperfusion therapy.

-Reperfusion Therapy:

1- Fibrinolytics:Only used in STEMI (ST elevated myocardial infarction)

Should be given during a 12hrs window(Golden hours), ASAP

There are two types: - non fibrin specific (streptokinase)
- fibrin specific

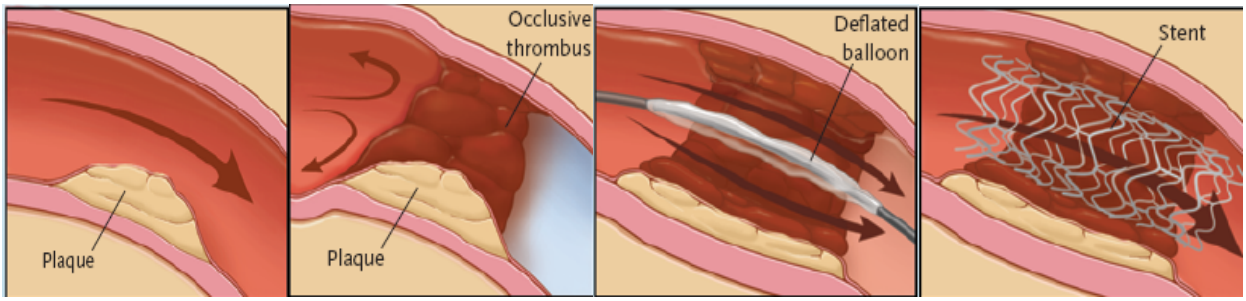
(contraindicated after 24 hours of the onset of the symptoms)

Fibrinolytics→ within 30 min.
Primary PCI→ within 90 min.
Time= muscle

Absolute contraindications
- Any prior intracranial hemorrhage — Important
- Known structural cerebral vascular lesion
- Known intracranial neoplasm
- Ischemic stroke within the past 3 months (except for acute stroke within 3 hours)
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head or facial trauma within 3 months

Relative contraindications
- History of chronic, sever, poorly controlled hypertension
- Systolic pressure >180 mm Hg or diastolic >110 mm Hg
- History of prior ischemic stroke >3 months previously, dementia, or known intracranial pathology not covered in absolute contraindications
- Recent (within 2–4 weeks) internal bleeding
- Noncompressible vascular punctures
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding
- For streptokinase/anistreplase: prior exposure (more than 5 days previously) or prior allergic reaction to these agents

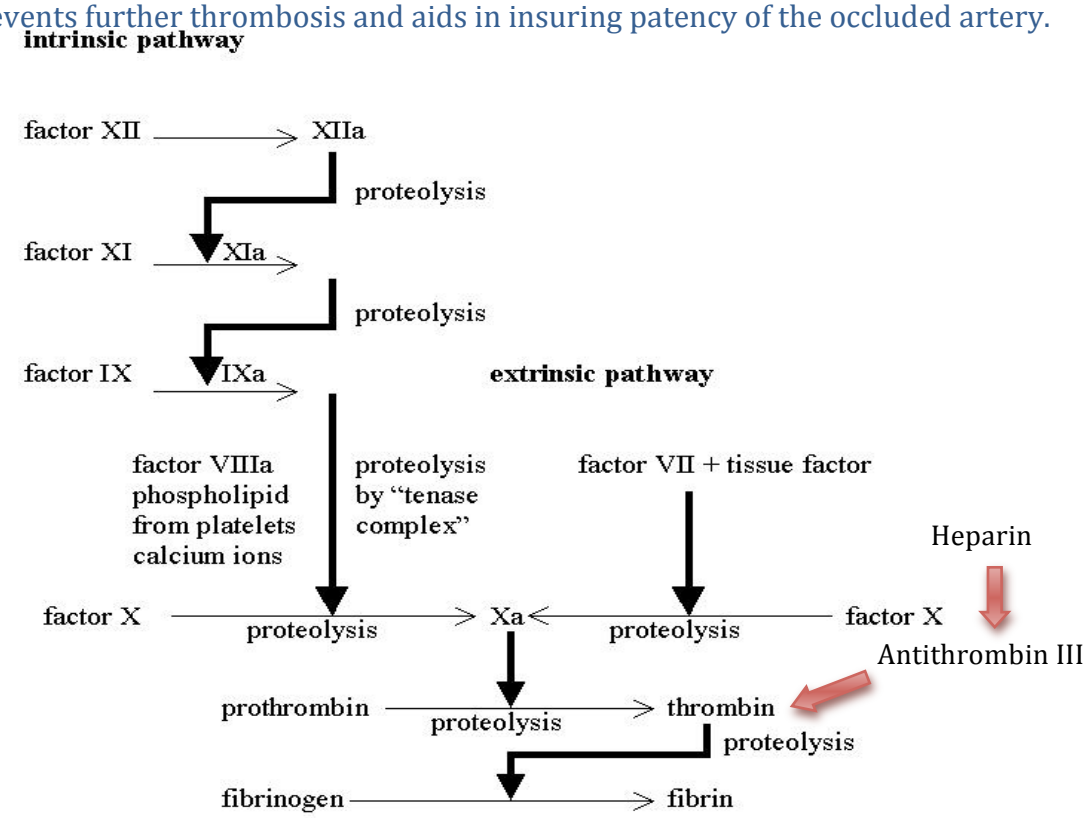
2-Primary Percutaneous Coronary Intervention (only in STEMI)



-Antithrombotics:

Heparin:
- Unfractionated
- Low molecular

Prevents further thrombosis and aids in insuring patency of the occluded artery.



Complications of MI:

- **Electrical complications (arrhythmia):**
  1. Tachyarrhythmias:
     a. Ventricular:
        - Ventricular Tachycardia
        - Ventricular Fibrillation (most common effect of MI)
     b. Supraventricular:
        - Atrial Fibrillation

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

- **Pump failure:**
  1-Heart failure (Pulmonary edema):
  - Bad prognostic sign
    - Reflects the size of the MI
    - ACE inhibitors and diuretics is cornerstonetherapy.

  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.

  3-Acute circulatory failure

- **Pericarditis**
  Post MI syndrome (Dressler's Syndrome) is characterized by persistent fever, pericarditis and pleurisy.

- **Mechanical complications (occurs late):**
  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

- **Impaired ventricular function, remodeling and ventricular aneurysm.**

Summary:

- Plaque vulnerability is affected by an inflammatory process.
- Acute coronary syndromes is a spectrum and is classified according to markers of ST changes and Myocardial necrosis.
- In STEMI , time to reperfusion is critical in myocardial salvage (time is muscle).