# Ischemic heart disease

# &

# Acute coronary syndrome

لا تنسوا اخوانا في غزة من دعائكم

اللهم ارفع الظلم و البلاء عن اخواننا في غزة

Done by Abdulla Ayman Ghnaim

# Ischemic heart disease (chronic coronary syndrome)

**heart anatomy

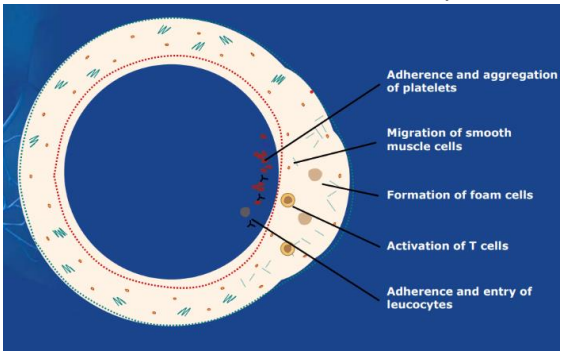
- The heart is about a fist in size measuring about 300-450 gm.
- The average beat is around 70 pumping about 6000-7500 liters of blood per day.
- The coronary supply of the heart consist mainly of the right main coronary artery (supply the inferior area /lead 2, 3, AVF) and the left main coronary artery which branch to the Left anterior descending artery (supply the anterior area / lead v1-4) and the left circumflex artery (supply the lateral area/ lead 1, AVL , V5-6) so depending on the artery affected there will be a different lead showing the changes.
- There are collateral arteries between the 2 coronary which are not active ,however they get activated by a strong stimulus usually transient heart ischemia ,that's why patients with MI on the top of previous angina are better than MI alone (activation of the collaterals).

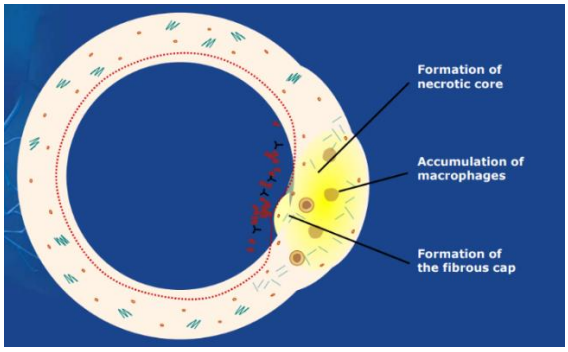
**heart physiology

- Cardiac work increase when there is increase in the work of the body , to do this the heart needs more oxygen supply which is established by increasing the coronary flow (in contrast to the increase in oxygen extraction in skeletal muscles).
- Most of the coronary flow is in the diastole (in contrast to most organs flow which is during systole).
- **Ischemic heart disease** occur due to imbalance between the oxygen demand and supply.
- The increase in **demand** occur due to increase in HR , contractility , wall tension , wall thickness and the reduced **supply** occur due to reduced coronary flow (coronary artery diseases) , Hb level , arterial oxygen saturation.

**coronary artery diseases

- Types : It could be atherosclerotic 95% or non atherosclerotic 5% like (embolism , autoimmune arteritis , coronary mural thickening , coronary luminal narrowing , congenital coronary artery anomalies).
  The atherosclerotic type have a **risk factors** including modifiable (mainly smoking , hypertension , hyperlipidemia *and obesity*, hyperglycemia *DM*) and the non-modifiable ( male gender , age M>45 F>55 , previous personal of family history of the disease *family history is valuable in a relative M>55 and F>65 of age* , mutation in the ACE)
- In a study in Jordan : it was found that 95% of patients with atherosclerosis have at least 1 risk factor ,if there is more than one risk factor it increase the risk in <u>exponential manner (2+2=10)</u>.
- Pathogenesis of the atherosclerotic plaques :
  Endothelial damage >> this will cause increase permeability of the blood vessel to LDL and increase in the adhesion molecules >> monocytes and T lymphocytes will attach to the sticky surface of the BV and inter to the sub intimal layer & there will be migration of the smooth muscle >> macrophages will take up the oxidized LDL and become foam cells that will form fatty streaks and plaques.



Note: atherosclerosis is a progressive disease that can start early in life and it progress depending on the genetics and the presence of the environmental risk factors.

- Types of the atheromatous plaques :
  **Stable** (thick fibrous cap , abundant smooth muscle , few inflammatory cells , small lipid pool //this what make it stiff and not easy to rapture) and **unstable** (thin fibrous cap , abundant lymphocytes , few smooth muscle , large lipid pool // this make it easy to rupture causing acute coronary syndrome).
- Presentation :
  - Atherosclerosis could occur in any artery in the body (cerebrovascular/coronary artery/peripheral vascular) and depending on this it cause different symptoms.
  - In coronary arteries disease it range from being asymptomatic to heart failure and sudden cardiac death with some disease in between this range like the stable angina, unstable angina , STEMI , non STEMI.
  - When the patient is present with symptoms it is too late because the disease has started a way before (we see the tip of the iceberg).
- **Chronic coronary artery syndrome**

1- Stable angina

- it is mostly caused by atherosclerosis that obstruct the coronary artery more than 70% leading to symptoms and chest pain with exertion.
- Other causes of it could be anemia (reduced O2 supply) or tachycardia / left ventricle hypertrophy (increased demand).
- History : of the pain *SOCRATES* S (retrosternal central chest pain) O (duration must be at least for 8 weeks ) C (pressure , heaviness) R (to the left shoulder , arm , forearm , ulnar part of the hand , neck , jaw *teath* , interscapulur region) A (it maybe associated with dyspnea , nausea) T (the episode last for 2-10 mins) E (it is exacerbated by anything that increase the heart rate like exertion ,emotional upset , heavy meal , cold weather and it is relived by nitrate and rest) S .
- Normal physical examination is not enough to exclude stable angina and we should ask about the risk factors and the peripheral arterial disease (including carotid bruits).
- Investigations :
  we can use ECG and stress ECG (ST depression) / ECHO and stress ECHO (abnormal wall motion/the ischemic segment will become hypokinetic).
  check for the lipid profile LDL / TG and for the CBC (anemia)
  coronary CT and coronary angiography (Gold standard)
  nuclear isotope test *dipyridamole Thallium* (decrease in the uptake of the isotope during exercise)
- <u>However diagnosis of stable angina is mainly clinical diagnosis (depending on the history).</u>
- NYHA grading of stable angina :
  Grade 1: Cardiac disease without resulting limitation of physical activity. Ordinary physical activity does not cause chest pain (dyspnea).
  Grade 2: Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity result in chest pain (dyspnea).
  Grade 3: moderate limitation in physical activity. Comfortable at rest. Less than ordinary activity causes symptoms .
  Grade 4: sever limitation: symptoms at rest.
- Management :
  Our goal here is to restore the balance between the O2 demand and supply to reduce angina symptoms and improve the prognosis (reduce mortality).
  1- Correction of the risk factors (weight reduction and aerobic exercise/ control of DM or HTN/treating anemia, thyrotoxicosis)
  2- Aspirin / statins (even if no dyslipidemia) / ACEI (especially if there is HF/HTN/DM)
  3- Symptomatic – nitrate (sublingual) , BB, CCB
  4- Angio – CABG (coronary artery bypass graft) /PTCA (percutaneous coronary intervention)

- Prognostic indications of CAD
- Left ventricle function <50% (normally it should be 50-75%) – this is associated with increased mortality
- The vessels involved (left main stem involvement is associated with increased mortality) / the severity and the extent of the ischemia
- Deferential diagnosis of angina

  1- Neuromuscular disorder 2- Respiratory disorders 3-Upper GI disorder 4- Psychological 5- Syndrome X
- Cardiac syndrome X (reduced vasodilation in the microvasculature / typical exertional angina with a positive exercise stress test *normal coronary arteries*/ more common in females , young /rarely respond to antianginal therapy but it has a very good long term prognosis).

  2- Variant angina – prinzmetal angina
    - It is caused by vasospasm in the large epicardia coronary arteries leading to transmural ischemia and it can lead to arrhythmia and death.
    - Symptoms : chest pain that occur at rest or night / vasospastic symptoms in other organs can occur (Reynaud , migraine) / >females
    - Investigations : negative troponin / ST elevation
    - Treatment : nitrate / CCB (decrease the spasm) / BB is contraindicated in this disease (have a risk of spasm).
    - Good prognosis

# Ischemic heart disease 2 (acute coronary syndrome)

****Acute coronary artery syndrome (Acute myocardial infarction)**

- they include unstable angina , STMI , non STMI

****myocardia infarction MI**

- Pathogenesis :
  This disease is caused by plaque (thrombus) rapture which will initiate the primary hemostasis (platelet) including
  1- platelet adhesions (using von will brand factor / GP 1b)
  2- Platelet activation (using many cytokines like thrombin / TX2 / ADP / 5HT – it will cause the platelet to form a polyposis shape).
  3- Platelet aggregation forming the platelet plug (this will expose and activation of GP 2b/3a)
  Then the secondary hemostasis will occur in which there is activation of the coagulation system (fibrinogen cross linking through the activated GP 2b/3a) and formation of the fibrin clot that will obstruct the artery.

  Note : the systems are intractable in which (platelets provide a surface for the coagulation enzymes / thrombin is a potent activator of the platelets).

  Note : the acute MI is the most common cause of death.

- The evolution of infarction :
  The most vulnerable part of the heart to ischemia/necrosis is the sub endocardium within about 20-30 mins
  Then it can reach to the epicardium within 2-6hrs
  Finally if it lift untreated the full thickness of the myocardium will be affected within 6-12hrs.
- Clinical manifestations :

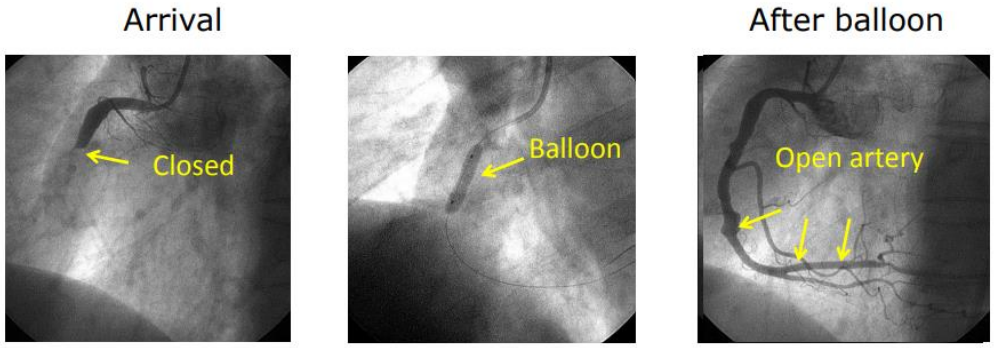
- History > chest pain *SOCRATES* in which S (mostly retrosternal) O (sudden) C (pressure/heaviness) R (it could radiate to the left shoulder , arm , forearm , and to the jaw or neck ) A T (*usually at rest or early morning occur for more than 30 mins* ) E S (it is the most severe pain a person could have *feels like he will die*) //so it is similar to angina pain but it is more severe and persistent.
- Physical exam >
  in vital signs we should look for BP (could be high or low or even normal the worst thing is to be low), Pulse , Temperature (after 24hrs it could be raised).
  In inspection anxious , sweaty , stressed.
  In auscultation S3 (more severe) , S4 (more common) , murmurs (usually after 72 hrs) , rub (after 48 hrs due to pericarditis).
- Painless MI : it is a MI that as name indicate doesn't cause pain instead it present as hypotension, arrhythmia, heart failure /it mostly occur in elderly ,DM.

- Diagnosis
  1- History & PEx
  2- ECG

- types
  STMI > begin as hyper acute wide based T wave ,then **ST elevation** , Q wave
  NSTMI > T inversion , ST segment depression
  Note: some patient could have MI with normal ECG initially (so it not always exclude MI) and in this case we repeat it again after about 40 mins.
- when to consider the ST elevation to be severely elevated
  firstly 2 adjacent leads have to show it then if it is in the
  limb leads it have to be >1mm in male or female
  precordial leads in **males** if >40 years (>2mm) and if <40 years ( >2.5mm) but in **females** >1.5mm.

- What diseases could mask the changes of ECG
  LBBB is a condition with similar ECG changes to MI ,so we Sgarbossa criteria to diagnose MI in LBBB patient.
  3- Cardiac markers
- Troponin T,I > we now use the high sensitivity troponin test (detect in 1-2hr / peak in 24hrs / last for 10-14 days).
  ~other non cardiac causes of elevated troponin include (cardiac surgery , tachycardia , myocarditis , HF , shock , sepsis , PE , renal failure) so not every person with high troponin is a MI case.
- CPK > we test the MB isoenzyme (positive if this CKMB is 5% of the total CK or it is 2 times normal) and the ratio ~MB2/MB1 have to be more than 1.5 to indicate necrosis (detected in 4-6hrs / peak in 24hrs / last for 72hrs)
  Note : no need to do both just one of them is enough.
- Myoglobin (not more used)
  4- Other tests
- CBC : high WBC , ESR , Hb
- Serum glucose : if high in non diabetic it carry poor prognosis
- Serum lipid (within 24 hrs)

- Management of STMI or low risk UA
  When the patient have persistent chest pain for more than 30 min he is suspected for MI so he should go to the nearest hospital and to not do any effort that can exacerbate symptoms.
  1- Rapid assessment (Hx & PEx)
  2- IV access (and blood sample) and ECG
  3- Medical therapy (MONA) each one of them is given if no contraindication
    - Morphine (to reduce pain / not given in case of hypotension ,hypovolemia, respiratory depression)
    - Oxygen (only given when the O2 sat is <90% )
    - Nitroglycerine sublingual or spray (not given in case of hypotension , RV infarction , recent erectile dysfunction medication )
    - Aspirin or heparin or clopidogrel or ticagrelor (antiplatelet given immediately in case of STMI / not given in hypersensitivity patient , bleeding disorders ,PUD)
  4- Perfusion strategy either PCI like balloon coronary angioplasty (door to balloon tome is <90min) or thrombolytics (only given in case of STMI or new LBBB if we need more than 90min *door to balloon time*/no contraindications)
  Notes :
    - time is very important so the sooner we open the coronary artery the better is the outcome (which is the infarction size)/ for every 30 min delay in the reperfusion the 1 year mortality increase by 8 %.




    - examples of thrombolytic therapy include Streptokinase SK , Alteplase tPA , Tenecteplase TNK-tPA (Best , given as IV bolus while the first 2 are given IV infusion in about 1 hour).
    - Limitations for thrombolytic therapy include the lower success rate compared to PCI / the possibility of reclusions and another MI.
    - Complications of thrombolytic therapy include hemorrhage , CNS bleeding , systemic embolization , allergic reaction.

➢ The contraindications for thrombolytic therapy

| Absolute contraindication | Relative contraindication |
|---|---|
| 1-Active internal bleeding | 1-Trauma or surgery > 2 weeks |
| 2-Suspected aortic dissection | 2-Active peptic ulcer disease |
| 3-Trauma or surgery < 2 weeks | 3-History CVA |
| 4-History of hemorrhagic CVA | 4-Bleedind diathesis or current use of anticoagulant |
| 5-BP> 200/120 mmHg | 5-Uncontrolled hypertension |
| 6-Prolonged CPR | 6-Previous exposure to streptokinase |
| 7-Recent head trauma or known intracranial neoplasm | 7-Pericardial friction rub |
| 8-Diabetic proliferative retinopathy | 8-Significant liver dysfunction |
| 9-Pregnency | |
| 10-Prvious allergy to the thrombolytic agent | |

5- Other routine therapies include beta blockers, ACEI , high dose of statins
6- Usually the patient is kept in the CCU for 1-2 days then discharged with medications like aspirin, ACEI, BB, statins.

- Complications of MI
  1- Electric (any type of arrhythmia can occur after MI).
  2- Mechanical including
    - Recurrent and extension of MI
    - Myocardial rapture
    - Left ventricular aneurysm
    - Heart failure (if the heart damage is >24% - heart failure but if >40% -cardiogenic shock)
    - Early pericarditis
    - Dressler syndrome (late pericarditis after 2-12 weeks)
    - Sudden death
- Differential diagnosis of MI
  In a patient with a severe sudden persistent chest pain DD include
  1- Aortic dissection (ECG and cardiac markers are normal)
  2- Pulmonary embolism (normal ECG and hypoxic patient)
  3- Acute pericarditis
- Prognosis of MI
  - The prehospital mortality rate is about 20% (mostly caused by ventricular fibrillation)
  - The hospital mortality rate is about 10% (mostly caused by cardiogenic shock)
  - The 1 year mortality rate is about 8% ,so for the MI patient the possibility of death in the first year as a total is about 40%.
  - Poor prognostic features (presence of the complications of MI / female / age>70 / DM / post infarction angina).
  - TIMI risk score for STMI

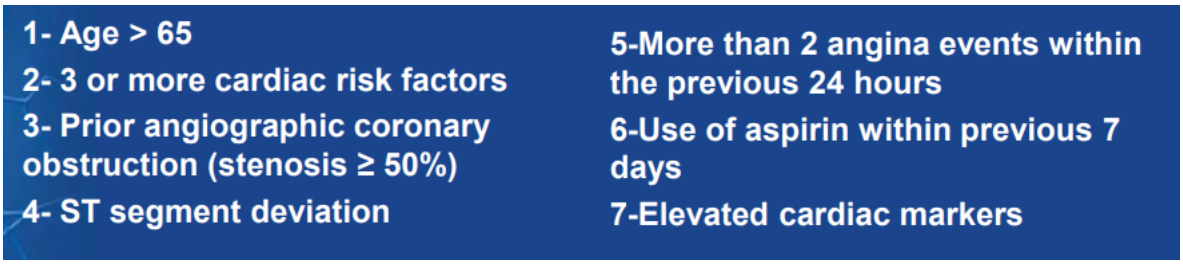
| Risk factor | Score |
|---|---|
| 1- Age>65 | 2 |
| 2- Age>75 | 3 |
| 3- Hist of angina | 1 |
| 4- Hist of hypertension | 1 |
| 5- Hist of DM | 1 |
| 6- Syst BP< 100 | 3 |
| 7- Heart rate> 100 | 2 |
| 8- Killip II-IV | 2 |
| 9- Ant M or LBBB | 1 |
| 10- Delay treat > 4 hr | 1 |

| Total Score | Risk of death at 30 days(%) |
|---|---|
| 0 | 0.8 |
| 1 | 1.6 |
| 2 | 2.2 |
| 3 | 4.4 |
| 4 | 7.3 |
| 5 | 12.4 |
| 6 | 16.1 |
| 7 | 23.4 |
| 8 | 26.8 |
| 9-16 | 35.9 |

- Management of MI
    - Risk factors modification (exercise / stop smoking / keep the BP 140-90 and HbA1C <7)
    - Aspirin / clopidogrel / ticagrelor
    - BB
    - High dose of statins
    - ACE
    - Aldosterone antagonists (especially in HF)

****unstable angina**

- Definition (it is one of 4)
    - New onset angina *acute* (<8 weeks)
    - Angina at rest or with minimal exertion
    - Chronic angina that has changed in intensity, duration, frequency of chest pain (crescendo angina).
    - Post MI angina (reperfusion angina) which occur within 2 weeks
- Pathophysiology
  Rupture or erosion of atherosclerosis / vasoconstriction / distal embolization.
- Diagnosis
  Mainly clinical / ECG (could be normal or could show T wave inversion or ST depression) / negative cardiac markers **
- Classification
    - Acute (rest pain in the last 48hrs) and subacute (no pain in the last 48hrs)
    - Primary and secondary (there is secondary causes hypotension , tachycardia , thyrotoxicosis , severe anemia)
    - High and low risk **
      ~high risk include :
      1-Rest pain > 20 minutes
      2-Accelerating ischemic symptoms in preceding 48 hr.
      3-Clinical finding of: pulmonary edema, new S3, new MR, Hypotension, Brady or Tachycardia.
      4-ECG changes: transient ST segment changes, BBB, VT
      5- DM
- TIMI risk score for unstable angina (predict risk of death/ progression to MI within 14 days) it should remain below 3




- Management of NSTMI or high risk UN

1- CCU admission and Rapid assessment (Hx & PEx)
2- IV access , blood sample , ECG
3- Medical therapy include 2 antiplatelet (aspirin , clopidogrel) and anticoagulation (LMW heparin)
4- Other treatment (BB ,statins , nitrate)
5- Perfusion therapy (invasive *preferred* or the conservative).
6- **NO THROMBOLYTICS**

بالتوفيق