Acute Coronary Syndrome

Objectives:

- Pathophysiology of ACS.
- Classification of ACS.
- Diagnostic workup.
- Initial management.
- Common complications of ACS.

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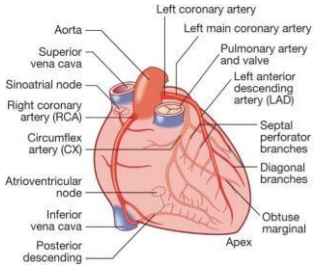
- Editing file
- Feedback

Anatomy and Histology

The coronary circulation:

The coronary arterial system consists of the right and left coronary arteries. These arteries branch from the aorta, arising immediately above two cusps of the aortic valve. The right coronary artery giving off vessels that supply the right atrium, the right ventricle, SA Node and AV Node. The vessel usually continues as the posterior descending coronary artery and supplies the posterior part of the interventricular septum and the posterior left ventricular wall. The left main coronary artery supplies the anterior septum and the anterior left ventricular wall and gives off branches to the left atrium and the left ventricle. Abrupt occlusion of the left main (Stem) coronary artery causes significant infarction of the LV and it is usually fatal because left coronary artery supplies anterior wall of LV which has significant function in cardiac contractility.



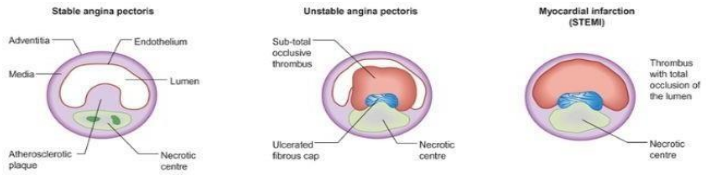
Histology: the blood vessel is composed of Tunica intima (inner), tunica media, Tunica Adventitia.

Pathophysiology and risk factors of Atherosclerosis

- **Introduction:**

Myocardial ischemia occurs when the oxygen demand exceeds the supply. Imbalance between coronary blood supply and O2 demand. The most common of that is obstructed coronary artery in the form of coronary atherosclerosis. CAD gives rise to a wide variety of clinical presentations, ranging from relatively stable angina through to the acute coronary syndromes of unstable angina and myocardial infarction.

[It is also known as coronary artery disease (CAD), Ischemic heart disease and atherosclerotic heart disease].



❖ Why do we concern about acute coronary syndrome (ACS) ?

1. Statistically ACS is number 1 killer.
2. 30% of ER admissions are cardiac related.
3. Increase in mortality rate. In Middle East, the projected increase in mortality caused by CAD from 1990 - 2020 is 171% in men, while in women the rate is 148%. (it's a burden)

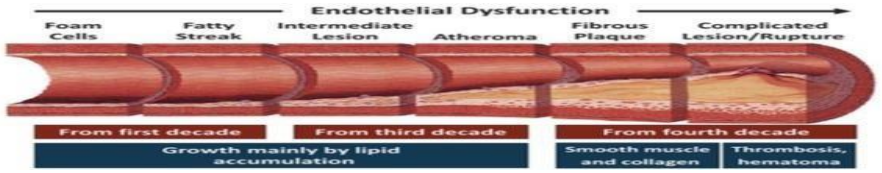
Pathophysiology:

- It's a complex inflammatory process characterized by the accumulation of lipid, macrophages and smooth muscle cells in subintimal plaques that obstructs the blood flow. It consists of : 1) Necrotic lipid core. 2) Fibromuscular cap.

1) **Lipid core formation:** It starts with endothelial damage (starts as early as 20's) (Mechanical stresses e.g. morbid hypertension or biochemical abnormalities e.g. elevated LDL, DM , free radicals from smoking) then increased permeability that allows leakage and accumulation of oxidized lipids into the subintima. Resulting in formation of flat yellow dots or lines on the endothelium called "fatty streaks". Those lipids are then taken up by the macrophages resulting in formation of "foam cells".

2) **Fibromuscular cap formation**: Cytokines are released from the macrophages and damaged endothelial cells. Resulting in further accumulation of macrophages + migration and proliferation of smooth muscle cells. Then collagen is produced with large amounts from the smooth muscle cells.Therefore, the blood vessel is narrow because of this plaque.

- Increased phagocyte and inflammatory cells around plaque is associated with Coronary Artery Disease.



3) Eventually, if the plaque is stable it will not occlude the artery, but if the plaque is vulnerable it will undergo many changes (the plaque either ruptures superficially or with a deep endothelial fissuring) that will lead to the occlusion of the artery. (70% is the cutoff point for ischemia).

(Large lipid core is pathologic change twill lead to unstable atherosclerotic plaque in CAD and subsequent rupture)

More explanation:

superficial endothelial injury (caused by mechanical or chemical stress) as well as metalloproteinases action (released from inflammatory cells in the plaque) → exposed endothelial covering of the plaque (rupture) → platelet adhesion as a result of reaction with collagen → thrombus formation on the surface of the plaque → **Occlusion of the artery.**

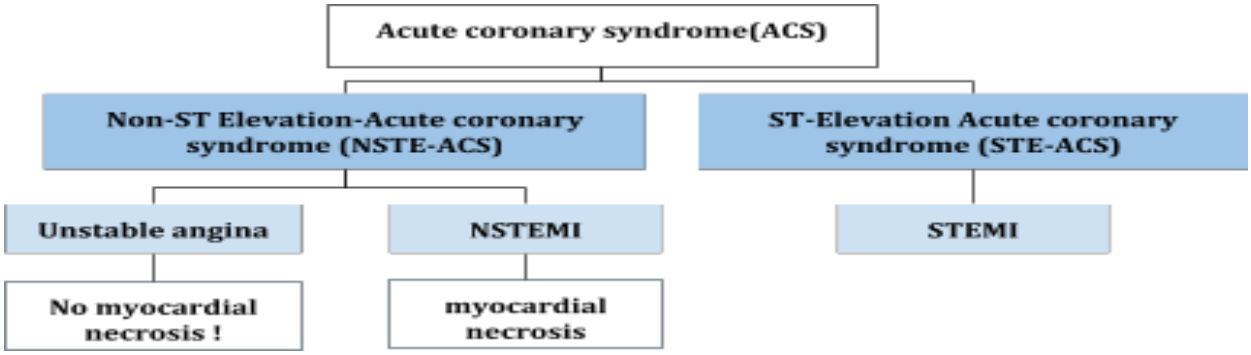
Risk factors:

CAD is an atherosclerotic disease that is multifactorial in origin, giving rise to the risk factor concept. Certain living habits promote atherogenic traits in genetically susceptible people. A number of 'risk' factors are known to predispose to the condition. Some of these, such as age, gender, race and family history, cannot be changed, whereas other major risk factors, such as serum cholesterol, smoking habits, diabetes and hypertension, can be modified. Known modifiable risk factors explain >90% of the occurrence of MI in populations around the world.

Obstructive Sleep Apnea Syndrome (OSA) is a risk factor to Cardiovascular diseases and premature death. The symptoms are: snoring, chocking attacks during sleep, dry mouth on awakening, headache, nocturia and excessive daytime sleepiness.

Most important risk factors:		Less reliable risk factors:
Modifiable	**Non-modifiable**	hard to measure or determine their role in the disease
1. Diabetes mellitus: the worst risk factor, and 1 out of 4 saudis has DM. **2. Smoking.** (smokers usually have low HDL levels because of smoking) **3. Hypertension.** **4. Hyperlipidemia** (**high** LDL and **low** HDL) "patients with familial hyperlipidemia can have fatty streaks when they were young" (High HDL is cardio protective feature) **5. Obesity**	Patient's age is above 45 in **men** and above 55 in **women**. "In our population we saw some cases in patients who were late twenties or thirties" Family history of premature CAD, it should be: - In first-degree relatives. - in males under 55; females under 65. (premature).	- Physical inactivity - Poor diet - Emotional stress (indirect, it comes in association with other factors like sedentary lifestyle) - Excess alcohol ingestion

Classification of Acute Coronary Syndrome



When a patient comes with chest pain and you are suspecting ACS,the first thing you should ask for is ECG. Based on ECG you classify the condition either ST-Elevation or Non-ST-Elevation. So, it is all based on the ECG but further investigations should be done. The next step is to investigate his enzymes.

What are the differences between USA and NSTEMI?

Unstable angina is a tissue ischemia with Highly suspicious symptoms. NSTEMI is a tissue necrosis. Also, in NSTEMI cardiac enzymes levels are elevated with associated symptoms, while in USA cardiac enzymes levels are normal WHY? Because cells are not dead (no necrosis).

Acute coronary syndrome (ACS)	
ST-elevation myocardial infarction (STEMI)	**Severe** ACS presentation. Usually lasts for more than 20 mins. ECG shows ST elevation due to **transmural infarction,** which is an infarction that involves the whole myocardium thickness. Although it might be asymptomatic in one-third of patients, and that is typically seen in postoperative patients, elderly, diabetic, and women. **The pain does not usually respond to sublingual glyceryl trinitrate.** (ST Elevation occurs mostly when the vessel is totally occluded. But always there are exceptions !)

non-ST-elevation myocardial infarction (NSTEMI)	Usually **more** than 20 minutes. **ECG shows ST depression** due to **subendocardial infarction** (tissue necrosis). **Subendocardial infarction indicates** that only the first third of the myocardium is infarcted, if the blood doesn't return it progresses to STEMI. ❖ In NSTEMI, the occlusion size is between USA and STEMI. ❖ Non-ST Elevation occurs mostly when the vessel is not completely occluded (for example: 80%). ❖ Remember : always there are exceptions !
Unstable angina (UA)	Usually **less** than 20 minutes. ECG shows ST depression due to tissue ischemia. **Pain is relieved by nitroglycerin.**

- We see ST-segment depression with unstable angina and NSTEMI. We can differentiate between them with the biochemical markers test.
- ST-segment elevation is an early indication of MI.

Fourth Universal Definition of Myocardial Infarction: (Cardiologists agreed that this is the criteria to define MI)

Typical rise and/or fall in cardiac troponin T or I with at least one of the following:

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

General approach to the diagnostic work up of ACS.

1. Clinical features: (See Talley page 46)

Patients commonly present with new onset ischemic chest pain. Physical examination is usually normal, but it is done to determine any precipitating causes of myocardial infarction (eg. aortic dissection). Remember asking about risk factors is very important in the diagnosis.

Ischemic pain Characteristics: (SOCRATES)

- **Location and Radiation: Substernal pain** that may radiate to shoulders, arms, and jaws.
- **Character: dull, heavy** and **pressure-like** pain.
- **Associated Symptoms:** Breathlessness, Diaphoresis, Nausea & Vomiting, Palpitation and Syncope.
- **Remember:** you have to ask the patient about the **onset** (sudden or gradual), the **duration**, aggravators **& relievers and severity** (using scale from 1 - 10 or other methods).

We have to differentiate between stable and unstable angina by asking about if the chest pain is even at rest or only with exertion

-It's usually not ischemic if it's pleuritic (changes with respiration), positional (changes with position) or tender to touch.
-If the pain lasts for only a few seconds, it's usually not ischemic.
-If the pain is sharp (knifelike or pointlike) **it's NOT ischemic !**

Chest pain in Typical Angina :

1. Substernal chest discomfort. 2. Provoked by exertion or emotional stress. 3. Relieved by rest or Nitroglycerine.

Chest pain in Atypical Angina :

meets 2 of the above characteristics.

Non-Cardiac chest pain : meet 1 or none of the above characteristics.

2. Electrocardiogram: (Usually happened in ER)

It usually done to monitor the ST-segment.

- **ST-segment elevation** is seen with STEMI due to transmural ischemia. ST segment elevation must be **higher than 1 mm** and seen in at least two leads. Otherwise, it's not considered elevation.
- **A T-wave inversion and Q wave** (not present normally) are highly suggestive of ACS. They may not appear during the first day of onset, so your diagnosis can't be based upon them.
- **Note that normal ECG does not exclude the possibility of ACS**.
- ECG must be performed as soon as the patient presents to the ER. In fact, ECG must be performed upon anyone who presents with chest pain of any cause.

3. Biochemical markers (For myocardial necrosis):

MI causes release of certain enzymes and proteins into the bloodstream**.**

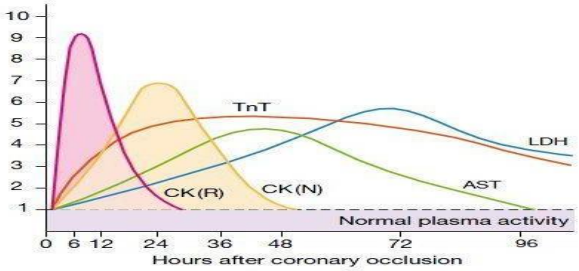
Creatine Kinase (CK)	- Creatine Kinase (CK) is released from multiple organs such as the myocardium , skeletal muscles, and the brain. - The Iso-form **CK-MB,** is cardio-specific**.** (Not that much specific) - **Starts to rise 4-6 hrs after onset of ischemia, then falls within 48-72hrs.**
Troponin	- **Cardio-specific proteins.** - **Troponin I, and T are the most sensitive & specific markers for myonecrosis.** - **Released with 4-6hrs, but can last upto 2 week**

➔ Elevated **CK-MB** and **troponin-I** indicate STEMI or NSTEMI.

(because there is necrosis)

➔ Normal CK-MB and troponin-I indicates unstable Angina.

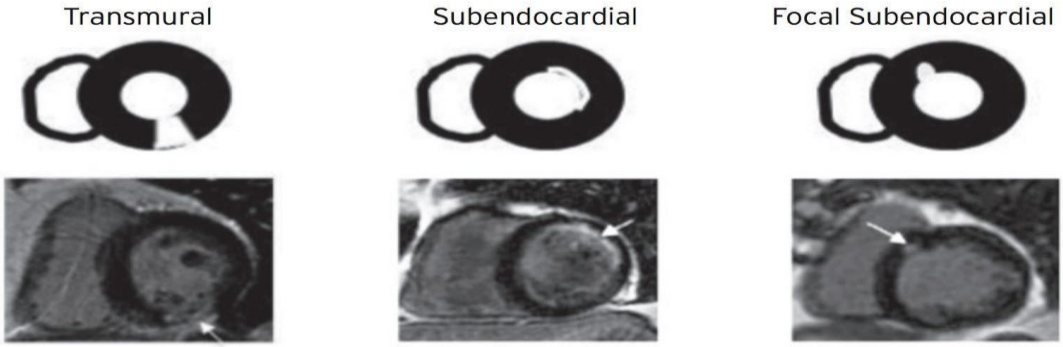
➔ Remember that CK-MB is used to detect reinfarction (because it returns to normal before troponins).



Changes in plasma cardiac marker concentrations after MI. Creatine kinase (CK) and troponin T (TnT) are the first to rise, followed by aspartate aminotransferase (AST) and then lactate (hydroxybutyrate) dehydrogenase (LDH). In patients treated with a thrombolytic agent, reperfusion is usually accompanied by a rapid rise in plasma creatine kinase (curve CK (R)) due to a washout effect; if there is no reperfusion, the rise is less rapid but the area under the curve is often greater (curve CK (N)).

You'll find **similar ECG findings in both NSTEMI and Unstable angina**, so we need biochemical markers to differentiate between the two:			
Markers	**Elevated**	**Normal**	**Indication**
Troponin-I & T + CK-MB	+++	-	STEMI or NSTEMI
Troponin-I & T + CK-MB	-	+++	Unstable Angina

Cardiac MRI Imaging:



Stress testing: (Extra)

Exercise tolerance test (ETT) is a tool used to evaluate chest pain when the etiology is not clear and ECG is not diagnostic. The idea here is that we're increasing the oxygen consumption of the heart.

Stress ECG:	ECG is recorded before, during and after exercise on a treadmill. It's very sensitive when the patient has normal ECG at rest. Findings aren't going to be different. So the result will be ST-segment depression due to subendocardial ischemia (Unstable angina, stable angina). Don't perform the test if ACS is not yet stabilized.
Stress Echocardiogram:	performed before and immediately after exercise. It detects wall motion abnormalities (dyskinesia, akinesia or hypokinesis) that are not present at rest. It is also used when we cannot read the ECG due to a baseline abnormalities. It is more sensitive than stress ECG in detecting ischemia.

Management of ACS

- **The aim of the therapy is to:**

➔ Open Artery and Improve oxygen supply :

1. Supplemental O_2 (ONLY if O2 Sat <95%).
2. Coronary vasodilators (Nitroglycerine) (increase supply and dilates systemic veins (decrease preload and thus O_2 demand)
3. Antiplatelet agents
4. **Reperfusion therapy by 2 ways :**

- Fibrinolytic therapy
- Primary Percutaneous coronary intervention (PCI)

5. Antithrombotic agents.

❖ NSTEMI and Unstable Angina are managed by all of above except fibrinolytics.

❖ STEMI are managed by all of the above including Fibrinolytics.

❖ Remember: use fibrinolytics ONLY in STEMI and within the first 12 hours after the onset of symptoms.

➔ Reduce O2 demand:

1. Beta blockers. (Block the stimulation of heart contractility and therefore reduce o2 demand)
2. Analgesics (Morphine) (analgesic as well as vasodilator)

➔ Other medications:

- ACE inhibitors. (acts as a vasodilator)
- Statin therapy. (Pleiotropic effect) (reduction in the plaque lipids which will make the plaque more stable)

Statin decreases LDL but this is not why we use it in the ER. we use it because of its anti-inflammatory property

★ Reperfusion therapy:

Reperfusion therapy is the ultimate destination,you have to open the artery either chemically(Thrombolytics = Fibrinolytics) or mechanically(PCI).

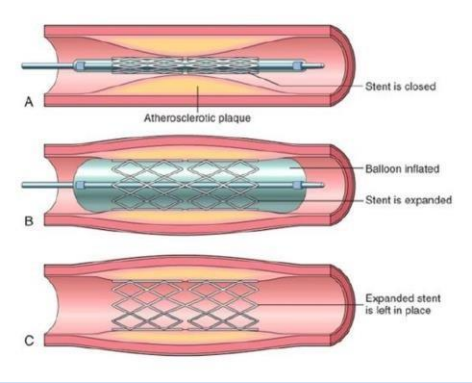
1- Fibrinolytics (Thrombolytics): (door to needle time <30 min)	
• ONLY USED FOR STEMI (NOT NSTEMI). • Reduces short and long term mortality following MI. • Should be given during a 12hr window, and given As soon as possible. There is no benefit if you give it after 12 hrs. Because after 12hrs the damage that has been done to the heart is irreversible so reperfusion by fibrinolytic won't be useful • If Fibrinolytics fails after 30-60 minutes, refer to PCI.[1] **There are 2 types of fibrinolytics:** 1. Non Fibrin specific: Streptokinase. we don't use it anymore 2. Fibrin specific: Tenecteplase (TNK) - Alteplase (first choice) - Reteplase.	
Absolute Contraindications to thrombolytic therapy	**Relative Contraindications to thrombolytic therapy**
➔ Any prior intracranial haemorrhage. ➔ Know cerebral vascular lesion. ➔ Known intracranial neoplasm. ➔ Ischaemic stroke within past 3 months. ➔ Recent major trauma/surgery/head injury (within 3 months) ➔ Active bleeding or Known bleeding disorder (excluding menses) ➔ Suspected Aortic dissection. (never use fibrinolytics in these conditions) **The worst bleeding ever is the intracranial bleeding, so DON'T GIVE Fibrinolytics if there is a risk of intracranial bleeding.**	➔ Oral anticoagulant therapy (example: Warfarin) ➔ Pregnancy or within 1 week postpartum. ➔ Noncompressible vascular punctures ➔ Traumatic resuscitation ➔ Poor controlled Refractory hypertension (systolic blood pressure >180 mmHg) ➔ Internal bleeding, e.g. active peptic ulcer ➔ Dementia. (fibrinolytics here are relatively contraindicated but still the doctors have the decision to use it according to the patient condition. For example : patient with severe MI coming to a hospital without a cath lab, the doctor can use fibrinolytics to save his life even if the patient has one of the above situations.)

[1] Remember Time is a muscle!

<table>
<tr><td colspan="2">2-Revascularization (surgical): An angiography must be done first. It’s either via CABG or PCI</td></tr>
<tr><td>CABG (Coronary Artery Bypass Graft)[2]

2 : 25 minutes</td><td>Used when the patient has:
- Three-vessels occlusion.
- Left main coronary artery occlusion.
- Left ventricular dysfunction.</td></tr>
<tr><td rowspan="3">PCI (Percutaneous coronary intervention)

1 : 42 minutes

 
</td><td>The procedure only aims to remove the clot, but a stent[3] could be placed in the artery to improve the outcome. Preferred treatment for STEMI, as long as it’s performed within 90 minutes from patient’s admission. (door to balloon time <90 minutes).</td></tr>
<tr><td>Complication of PCI:
- Rupture of coronary artery on inflation.
- Restenosis.
- Hematoma at the site of entry (e.g. femoralarea hematoma).</td></tr>
<tr><td>Used when the patient has:
- One-vessel occlusion.
- Two-vessels occlusion.
- No improvement despite maximal medical therapyof ACS.</td></tr>
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Antiplatelet Agent:

1. **Aspirin (ASA):**

Aspirin will inhibit cox-1 enzyme which lead to inhibition of platelet aggregation .

Chewable 160 to 325 mg at presentation, then 75 to 325 mg daily.

2. **P2Y12 inhibitors:** (can be used for patients with aspirin allergy)

More potent than ASA and is combined with ASA and both agents are powerful adjuncts to reperfusion therapy.

Examples: Clopidogrel, Ticagrelor and Prasugrel**.**

Antithrombotic Agent:

Unfractionated Heparin or Low Molecular Heparin.

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

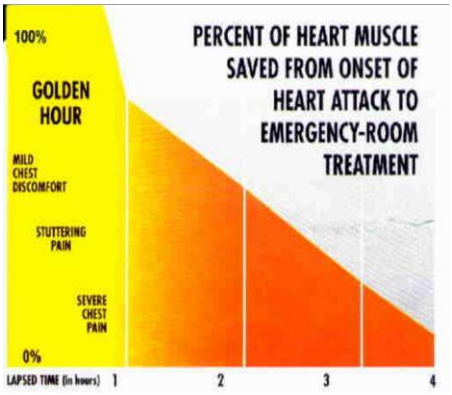
Mnemonic (discharge medications after ACS) : ABCDE
A: Aspirin and anti-anginals
B : Beta blockers and blood pressure
C : Cholesterol and cigarettes
D : Diet and diabetes
E : Education and exercise

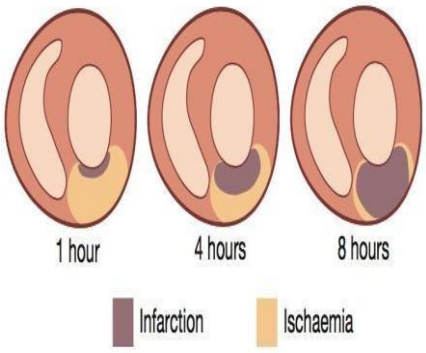
[2] Requires open heart surgery.
[3] Either a bare metal stent, or a drug-eluting stent. The latter is better.

Risk factors modification:

- Try to modify the risk factors.
- Smoking cessation among other risk factors modification shows the most immediate effect.
- The goal of LDL level is below 100 mg/dL. Statins ,among other lipid lowering agents, is the only one that reduces mortality rate.

TIME IS MUSCLE





Time is important! As we move forward damage progresses and infarction size increases; it gets harder to manage the patient and more serious complications probability increases. When it's been already 12 hours since the onset of pain there would be no point in the acute management as the injury has already been irreversible.

The first hour is the golden hour.

Complications of ACS

- **Congestive heart failure and its complication:**

Most common cause of in-hospital mortality. If severe may lead to cardiogenic shock (insufficient cardiac output).

- **Electrical Arrhythmias[4]:**

Includes:	Notes
Atrial fibrillation - Ventricular fibrillation	• **(immediately use defibrillator and CPR).**
Ventricular tachycardia	• Most common cause of death in first few days after MI is ventricular arrhythmia either VT or V-Fib
Sinus tachycardia	• May be caused by pain, anxiety or fever and it worsensischemia
Sinus bradycardia	• Commonly occurs during early stages of acute MI, especially right-sided MI. • It might be a protective mechanism[5].
Asystole and AV block	• associated with ischemia involving conduction tracts). It usually appears within first 24 hours; and as time pasts it becomes less likely to happen.

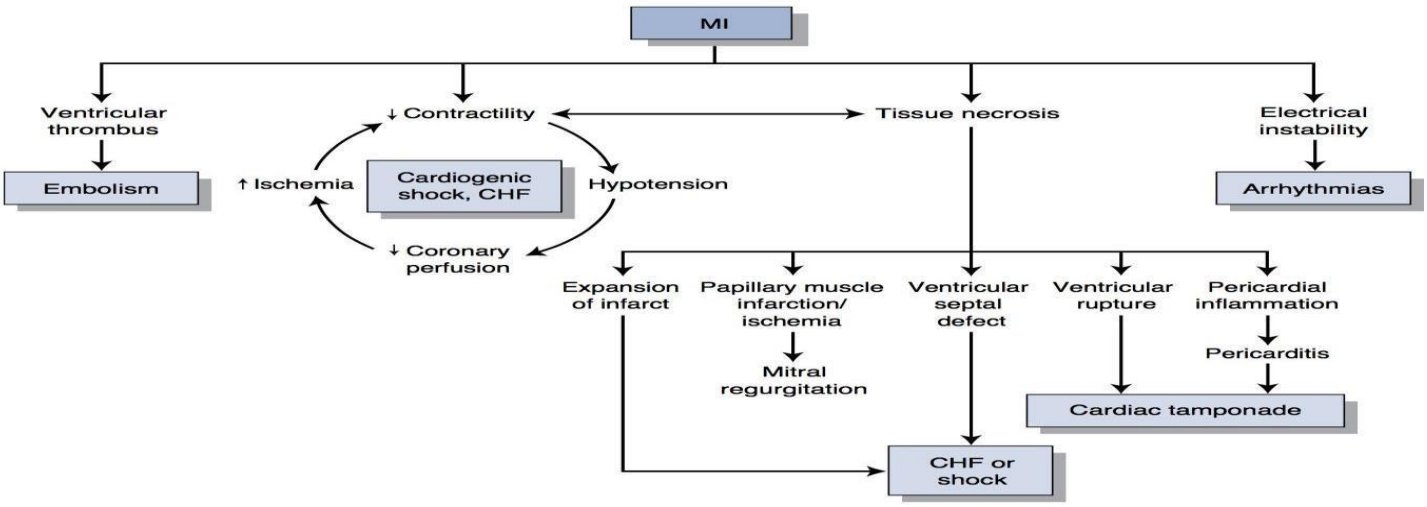
Recurrent infarction:

Usually diagnosed with heart enzymes (remember which enzyme?). Also ST-segment elevation within the first 24 hours after the first infarction may indicate reinfarction.

Mechanical complications: (usually occurs late after MI ...days to weeks)

- Free wall rupture: leads to cardiac tamponade. Has a high mortality rate (90%).
- Rupture of interventricular septum: has a better chance with therapy than free wall rupture.
- Papillary muscle rupture: leads to mitral insufficiency and mitral regurgitation.
- Ventricular aneurysm: rarely ruptures, associated with tachyarrhythmia.
- Ventricular pseudoaneurysm: incomplete free wall rupture (pericardium is maintained), unlike the ventricular aneurysm, it tends to rupture.

[4] Will be discussed in an upcoming lecture
[5] By reducing Oxygen demand.



*It is important to notice that some of them may lead to another.

Real and typical example of how ACS patient will present?

In a regular day at the hospital, mid-august 3:00 am ER. Rashed 55 y/o accountant male, type II diabetic, heavy smoker.

presented to ER with severe central crushing chest pain started at mid night, waking from sleep, sweating profusely, having nausea, ECG was done 10 mins after his presentation to ER, the ECG showed ST elevation in leads V2, V3, V4, and V5. (anterior wall leads)
Rashed was admitted to Cath Lab around 3:55 am. Puncture and entrance to arterial system (catheterization) of coronary arteries with contrast (angiography) showed a filling defect in LADA mainly due to thrombus.

His past medical history:

- 1 year ago he came for a regular checkup, physical examination showed:

BMI: 31 (obese I) .BP: 150/90 repeated 3 time (hypertensive). Waist circumference: +115 cm

Lab Investigations showed:

FBG: 11 mmol/l (diabetic). TC: 7 mmol/l (very high). LDL: 4.5 mmol/l (very high)
HDL: <1 mmol/l (low, smoking reduces HDL)

He was advised to change his lifestyle, quit smoking, start on metformin and statins.

Rashed didn't care and didn't change his lifestyle, 6 months later he went to his GP because of occasional chest heaviness (SA) but didn't follow up .

Summery

	Unstable angina	NSTEMI	STEMI
Troponin (I & T) CK-MB	-ve	+ve	+ve
Notes	• A chest pain with no pattern lasts **less** than **20 minutes** • ST depression due to ischemia • Relieved by nitroglycerin	• Pain lasts **more** than **20 minutes** • ST depression due to subendocardial infarct that involves inner 1\3 of the wall Complications of MI: • Electrical (tachy / brady arrhythmias) • Heart failure (pulmonary edema) • Cardiogenic shock	• Sever ACS presentation • ST Elevation due to the infarct involving the myocardial thickness • **No response** to sublingual glyceryl trinitrate

"A **46 y/o man** known to have **DM** and **HTN** comes to the ER with **central crushing** chest pain"
this is a typical scenario for an ACS pt. and to differentiate between the types we need to do the following investigations

1- **ECG:** to monitor the ST elevation.
2- **CK-MB:** released in MI pt.(4-6 h) after the onset and then falls within (48-72h).
3- **Troponin:** released in MI pt. (4-6 h) and can last up to 2 weeks.

Pharmacological Therapy

1. Increase O2 supply

Vasodilators :	Antiplatelet:	Antithrombotic:
Nitroglycerine	Aspirin + P2Y12 inhibitor: clopidogrel, ticagrelor and prasugrel.	Unfractionated Heparin or Low Molecular Heparin.

2. Reduce O2 demand

Beta blockers:	Analgesics for the pain
acebutolol, atenolol, propranolol ... etc.	

3. Other medications

ACE inhibitors:	Statin therapy
captopril, benazepril … etc.	

Reperfusion Therapy

1. Fibrinolytic

For STEMI only!!
Used within the first 12 hours after the onset of the symptoms
2 types: Non-fibrin specific: streptokinase
Fibrin specific: tenecteplase (TNK) – alteplase reteplase

Absolute contraindication (never use fibrinolytics):
Prior intracranial hemorrhage # cerebral vascular lesions
intracranial neoplasm # ischemic stroke within the past 3 months
suspected aortic dissection # active bleeding or known bleeding disorder
recent close-head trauma / surgery within 3 months

Relative contraindication where it depends on the doctor's decision:
Oral anticoagulant, pregnancy\ 1 week postpartum, internal bleeding (peptic ulcer)… etc.

2. Primary PCI

- Preferred treatment for STEMI
- Best time time to open the artery is within 60 minutes

Examine Yourself!!

1. Which one of the following represent the aim of managing patient with acute coronary syndrome?
   A. Opening the occluded artery and improving oxygen supply.
   B. Reducing O2 demand.
   C. Both A and B.
   D. None of above.

2. A 55-year-old man has just arrived in accident and emergency complaining of 30 minutes of central crushing chest pain. Which feature is most indicative of myocardial infarction at this moment in time?
   A. Inverted T waves
   B. ST elevation
   C. Q waves
   D. Raised troponin

3. A 80 years old female diabetic came to the emergency 9 hours ago with central chest pain, burning, troponin was 10 mg/dl (high), vital sign is normal. ECG shows deep T wave inversion in V1-V3. Which of the following is the diagnosis?
   A. NSTEMI
   B. Pericarditis
   C. Unstable angina
   D. Pulmonary Embolism

4. Fibrinolytic are only used in which of the following conditions?
   A. NSTEMI within the first 12 hours.
   B. STEMI within the first 12 hours.
   C. Unstable angina
   D. All of above.

5. All of the following are absolute contraindication of fibrinolytic therapy except?
   A. Any prior intracranial hemorrhage.
   B. Know cerebral vascular lesion.
   C. Ischemic stroke within past 3 months.
   D. Pregnancy or within 1 week postpartum.

6. A 55-year-old man with type 2 diabetes presents with a 1-hour history of severe central chest pain. Which of the following statements is true?
    A. normal baseline troponin and elevated 6-hour troponin level is suspicious of myocardial infarction
    B. normal ECG excludes myocardial infarction
    C. normal initial troponin level excludes myocardial infarction
    D. Failure of chest pain to resolve with nitrates confirms myocardial infarction

7. Which of the following are the worst factor for ACS ?
    A. DM
    B. HTN
    C. Smoking
    D. Hyperlipidemia

8. A 43 year old man has an extensive anterior myocardial infarction and has received antiplatelet, anticoagulant and statin therapy. He is referred for an echocardiogram. What will transthoracic echocardiography most usefully assess in this setting? (Extra Question)
    A. Cardiac arrhythmia
    B. Future prognosis
    C. Left ventricular function and the presence of mural thrombus
    D. Myocardial scar formation
    E. Thrombus in the left atrium

9. A 59-year-old male smoker complains of severe substernal squeezing chest pain of 30-minute duration. The paramedics have given sublingual nitroglycerin and oxygen by nasal cannula. His blood pressure is 110/70 mmHg and heart rate 90 bpm on arrival to the emergency room. The ECG is normal. Which of the following is the best next step? (Extra Question)
    A. Echocardiography
    B. Thallium stress test
    C. Aspirin
    D. Coronary angiography
    E. Coronary artery bypass

10. A 49-year-old man is rushed to accident and emergency complaining of a 20-minute history of severe, crushing chest pain. After giving the patient glyceryl trinitrate (GTN) spray, he is able to tell you he suffers from hypertension and type 2 diabetes and is allergic to aspirin. The most appropriate management is:
    A. Aspirin
    B. Heparin.
    C. Clopidogrel.
    D. Warfarin

11. A 65-year-old female has just arrived to the emergency department complaining of 20 minutes of central crushing chest pain. Which ONE of the following features is most indicative of myocardial infarction in this patient?

A. Peaked T wave.

B. Presence of U wave.

C. ST elevation.

D. Q waves.

12. A 40 years old male presents to the ER having 1 day history of chest pain. the pain started gradually, and continued until coming to the ER. It was located in the center of the chest and did not radiate, and was somewhat worse on lying on his back. He did not notice a change of pain severity with exertion. His vital signs were normal, and ECG revealed ST elevation from V1 to V6 ,I, AVL, II, III and AVF. The most likely diagnosis of this chest pain is:

A) Acute myocardial infarction

B) Acute pulmonary embolism

C) Pericarditis

D) Gastro -esophageal reflux

1-C / 2-B / 3-A / 4-B / 5-D / 6-A / 7-A / 8-C / 9-C / 10-C / 11-C / 12-C