Acute coronary syndrome

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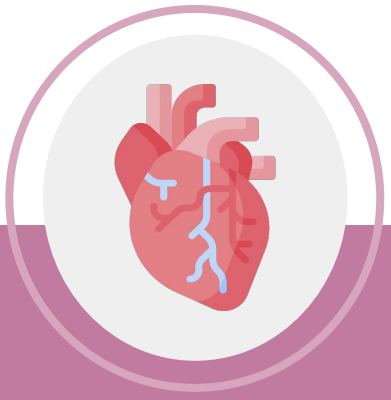

Objectives :

- ★ Distinguish STE and NSTE acute coronary syndromes by diagnosis, prognosis and treatment strategy.
- ★ Understand how ACS differs pathophysiologically from chronic coronary Syndromes.
- ★ Devise a diagnostic approach for establishing ACS.
- ★ Outline treatment strategies for ACS
- ★ Assess the short- and long-term prognosis of different types of ACS
- ★ Devise a pharmacotherapy treatment plan for a patient undergoing primary PCI for STEMI.
- ★ Devise a pharmacotherapy treatment plan for a patient undergoing fibrinolytic for STEMI.
- ★ Devise a pharmacotherapy treatment plan for a patient with NSTE-ACS.
- ★ Design a therapeutic regimen for a patient with ACS prior to discharge from hospital.
- ★ Discover online, electronic and app resources to assist clinicians with implementation of practice guidelines.

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Introduction

Anatomy (Blood supply of the heart)

The left main and right coronary arteries arise from the left and right sinuses of the aortic root, distal to the aortic valve:

➔ **Left main coronary artery:**

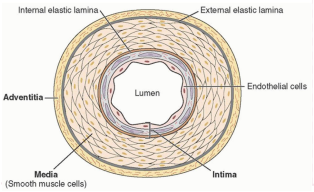
- **Left Anterior Descending (LAD):** Supply anterior 2/3 of interventricular septum, anterolateral papillary muscle, and anterior surface of LV.
- **Left Circumflex (LCX):** Supply the lateral, posterior and inferior segments of the LV.

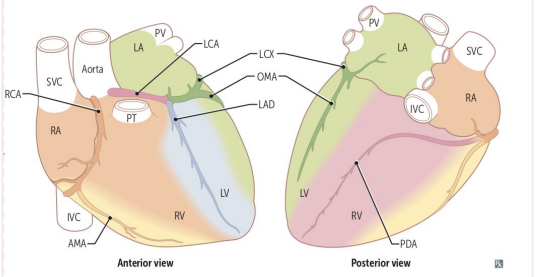
➔ **Right main coronary artery (RCA):** supplies SA node

- **Posterior Descending Artery (PDA):** Supplies AV node (dependent on dominance),
- posterior 1/3 of interventricular septum, posterior 2/3 walls of ventricles, and posteromedial papillary muscle.
- **Acute Marginal Artery (AMA):** Supplies RV.

Dominance circulation of the heart:

Right (85%) —> If PDA arises from RCA
Left —> If PDA arises from LCX





Key:
AMA= Acute marginal artery
LAD= Left anterior descending artery
LCX= Left circumflex artery
OMA= Obtuse marginal artery
PDA= Posterior descending artery
PT= pulmonary trunk
PV= pulmonary vein
RCA= Right coronary artery

Most commonly occluded arteries:
LAD - RCA - LCX

Introduction to ACS

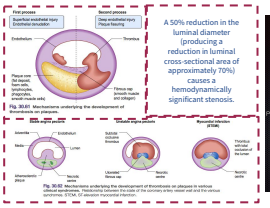
- Acute coronary syndrome is a term that encompasses **both unstable angina and myocardial infarction (NSTEMI & STEMI)**. (But not stable angina) Coronary artery disease (CAD) is divided into stable angina and ACS (UA,MI)
- Almost always occurs in patients who have **atherosclerosis**.

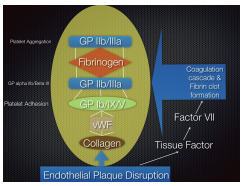
The atherosclerosis process starts from birth, when we start eating and drinking and as we evolve and start eating lipid containing diet; the process of deposits of cholesterol in the walls of the vessels happens, Multiple factors (e.g. being a male, eating unhealthy diet, smoking, inflammatory diseases like rheumatoid arthritis and others mentioned in next page) determine the rapidity of forming atheroma that eventually lead to the occlusion of the coronaries. It's inevitable that humans will have atherosclerosis it's only the rate of progression that differs. And as you can see in KSA the population of MI are younger because atherosclerosis risk factors are prevalent (¼ of the population are diabetics). The pathophysiology of ACS will be discussed in the next page

Pathogenesis of ACS — The common mechanism to all ACS is:

1- Rupture[1] or erosion of the fibrous cap of an atherosclerotic plaque, expose collagen and vWF

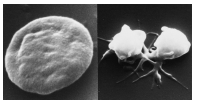
In a fixed stenosis (NO rupture), a 75% stenosis of the epicardial arteries is required to eliminate the flow in the coronaries by 90% which will eventually lead to stable angina with exertional symptoms (symptoms that come with activity or emotional stress). In contrast, plaques that only occlude 20% may be more aggressive if rupture occurs, once it ruptures a clot will be formed and it will lead to ACS. The presence of a **rich lipid pool** within the plaque and a **thin fibrous cap** are **associated with an increased risk of rupture**. Bottom line: Ischemia due to fixed stenosis (no rupture) → Stable angina, Plaque rupture with superimposed thrombus → ACS





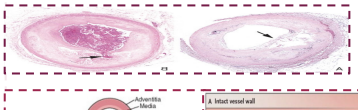
2- Platelet aggregation and adhesion, Platelets will release:

- **Thromboxane A2 (aspirin works here):** Vasoconstriction & Activate nearby platelets
- **ADP (clopidogrel and Ticagrelor work here):** Activate nearby platelets
- **Serotonin:** Vasoconstriction

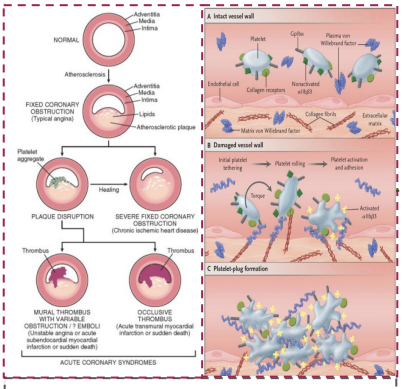


3- These signals will attract more platelets which will attach to each other by fibrinogen to **form thrombus** which will close the vessel; distal thrombus embolization is possible.

★ 4- During evolution of an atherosclerotic plaque, monocytes and other inflammatory cells bind to receptors expressed by endothelial cells. Subsequently, they migrate into the intima, and take up oxidised low-density lipoprotein (LDL) particles by phagocytosis to become lipid-laden macrophages or foam cells.



Dr: Very Imp to understand



Complete occlusion: STEMI
Incomplete occlusion: UA & NSTEMI

Acute coronary syndrome cont.

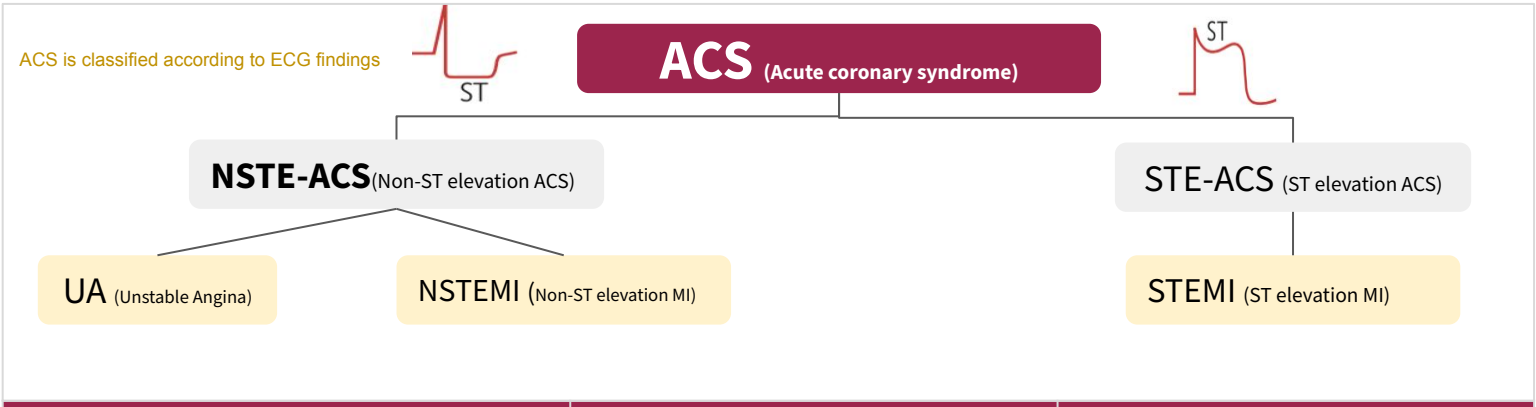
Risk factors for ACS[1]

EXTRA but important

CHOSED A Good Food

Major risk factors		Minor risk factors
Modifiable	**Non-Modifiable**	
1) **Diabete mellitus:** The worst 2) **Smoking:** Most immediate effect 3) **Hypertension:** Most common 4) **Hyperlipidemia (Cholesterol):** ↑LDL & ↓HDL (High HDL is protective)	1) Age & Gender[2] 2) **Family history & genetics:** considered a risk factor if ACS happened in **premature![3] first degree relatives** (Siblings and parents)	1) Physical inactivity (Exercise) 2) Poor diet 3) Emotional stress 4) Excess alcohol ingestion. 5) Obesity

- **Smoking cessation** among other risk factors modification shows the **most immediate effect.** Giving up smoking is the single most effective contribution a patient can make to his or her future.
- **The goal of LDL level is below 100 mg/dL**. Statins, among other lipid lowering agents, is the only one that reduces mortality rate.



Unstable angina	NSTEMI	STEMI
No myocardial necrosis	**Subendocardial infarcts**	**Transmural infarcts**
• Thrombosis with incomplete coronary artery occlusion • **Normal or nonspecific (e.g. ST depression)** on ECG but **NO cardiac biomarker elevation** • Characterised by angina on minimal exertion or angina at rest in the absence of myocardial damage. • Lasts less than 20 min.	• Subendocardium (inner 1/3) especially vulnerable to ischemia; if the blood doesn't return it progresses to STEMI. • **Normal or nonspecific (e.g. ST depression) and cardiac biomarker elevation.**	• Full thickness of myocardial wall involved. • Completer occlusion. • **ST elevation and cardiac biomarker elevation.** • Lasts more than 20 min. • The pain does not usually respond to sublingual glyceryl trinitrate.
Partial occlusion of coronary vessel → decreased blood supply → ischemic symptoms (also at rest)	Due to partial occlusion of a coronary artery	Complete occlusion of a coronary artery → ischemia→ death of myocyte (irreversible)

Q: Clinically, how would you differentiate between STEMI, NSTEMI & UA?
STEMI: Symptoms + ST elevation + Elevated Cardiac biomarkers (Troponin & CK-MB)
NSTEMI: Symptoms + (+/-) ST depression + Elevated Cardiac biomarkers (Troponin & CK-MB)
UA: Symptoms + (+/-) ST depression + Normal Cardiac biomarkers

1- If the cause of atherosclerosis is genetics (Non-modifiable) then you can't control it, you can only slow the progression with medications and treating the other reversible factors, whereas for the modifiable factors we can control them by counseling patients, advocating the community to reduce unhealthy diet, smoking and exert exercise.
2- Pre-menopausal women have lower rates of disease than men, although the gender difference disappears after the menopause.
3- **Premature:** Age<50 in men and <55 in women; family history doesn't convey a risk for the patient if the heart disease developed in elderly relatives!

Acute coronary syndrome cont.

◀ Differential diagnosis of chest pain in ER

★★ **Life threatening causes of CP:** PPPPAA

You have to know how to differentiate between them Click here for a quick summary of these conditions

Cardiac	Non Cardiac
1- Acute coronary syndrome: Substernal, radiating to arm, dyspnea on exertion, diaphoresis, worse with exertion.	**4- Acute pulmonary embolism:** Sudden onset, pleuritic, dyspnea, tachycardia, tachypnea, hypoxia, evidence of lower extremity deep venous thrombosis
2- Aortic dissection[1]: Sudden onset, severe, tearing, **radiating to the back** (associated with neurologic deficits, AR), unequal arm BP >20 mmHg[2] (take the BP in the arm with more BP), wide mediastinum	**5- Tension pneumothorax:** Sudden onset, sharp, pleuritic, decreased breath sounds and chest excursion, hyperresonant percussion, hypoxia
3- Acute pericarditis & tamponade: Sudden onset, pleuritic, better with sitting forward, radiating to the back, pericardial rub, ± tamponade (distant heart sounds, hypotension, JVD)	**6- Esophageal rupture/perforation:** Severe, increase with swallowing, fever, abdominal pain, history of endoscopy, foreign body ingestion, trauma, vomiting

◀ Can ACS present without chest pain?

- Yes, 33% of all ACS cases. They have worse prognosis, because they don't seek medical care early.
- More likely in elderly (>70y/o), diabetic individuals, women[3] (reasons are multifactorial), patients with prior HF, or Cardiac transplant
- In patients with severe diabetes, chest pain may be completely absent (silent MI) due to polyneuropathy
- Autonomic symptoms (e.g. nausea, diaphoresis (sweating, especially to an unusual degree)) are often the chief complaint

◀ Overview on the approach to ACS

1 Clinical presentation

Chest pain: Ischemic pain is described as dull or sore: Squeezing/ pressure like **NOT** sharp (knife-like) or pointlike. It's also **NOT** tender, positional or pleuritic.

2 ECG

If the patient presents with chest pain **ALWAYS begin with ECG** to determine whether it's STE-ACS or NSTE-ACS, if the ECG showed NSTE then do cardiac enzymes to determine whether it's NSTEMI or UA.

3 Cardiac Biomarkers

★ Elevated CK-MB and troponin-I indicate STEMI or NSTEMI, while Normal CK-MB and troponin-I indicate unstable angina

1- If the patient has aortic dissection every 1hr delay will increase the mortality by 3-5%, so by 10 hrs it will be ~50% (very high).
2- Patient is complaining of severe chest pain radiating to the back > check for blood pressure **in both arms** immediately (it's very important to check in both arms)
3- I have to suspect MI in elderly women with diabetes, even if they didn't show any signs or symptoms

1- ACS Clinical presentation

◀ Signs & Symptoms of ACS

Characteristic pain	• Severe, persistent, typically **Substernal**[1] aka (retrosternal or central)	
Quality	• Dull, **Squeezing**, **tightness**, **heaviness**, **pressure**, gripping aching. **NOT** sharp, knifelike, pins, stabbing.	
Radiation	**Shoulders**, **arms**, neck, lower jaw, teeth, epigastrium	
Associated symptoms	**Sympathetic**	• Diaphoresis, Cool and clammy skin
	Parasympathetic (Vagal effect) esp. RCA	• Weakness, Nausea & vomiting When heart muscle cells die. As they do so, they release biochemical markers into their surrounding environment. These biochemical markers activate the parts of the vagus nerve attached to the heart.
	Inflammatory	• Mild fever Due to the elevation of serum levels of myocardial enzymes and c-reactive protein.
Cardiac findings	• S4 (atrial contraction) (and S3 if systolic dysfunction present) gallop. • Dyskinetic bulge (in anterior wall MI) • Systolic murmur (if mitral regurgitation or VSD)	
Other	• Pulmonary rales/crackles (If heart failure present) • Jugular venous distention (IF HF or right ventricular MI)	

◀ S&S that increase/ decrease the likelihood of ACS[4]

The higher the number, the higher the likelihood of ACS.

LR: Likelihood rate
CI: Confidence interval

Dr: exertion is the most important feature
Dr: Important to know the likelihood (MCQ)

INCREASE THE LIKELIHOOD	LR (95 % CI)	DECREASE THE LIKELIHOOD	LR (95 % CI)
Radiates to the right arm or shoulder	4.7 (1.9–12)	Pleuritic Dr: Almost rule out ACS	0.2 (0.1–0.3)
Radiates to both arms or shoulders	4.1 (2.5–6.5)	Sharp	0.3 (0.2–0.5)
Precipitated by exertion (specifically by stairs)	2.4 (1.5–3.8)	Positional	0.3 (0.2–0.5)
Radiates to the left arm	2.3 (1.7–3.1)	Reproducible with palpation	0.3 (0.2–0.4)
Associated with diaphoresis	2.0 (1.9–2.2)	-Dr: If the number is near to 10 = Related to ACS.	

Dr: Try to memorize it

Box. Risk Stratification for Acute Myocardial Infarction and Acute Coronary Syndrome According to Components of the Chest Pain History

Low Risk

Pain that is pleuritic, positional, or reproducible with palpation or is described as stabbing[2,3,24,25,29]

Probable Low Risk

Pain not related to exertion or that occurs in a small inframammary area of the chest wall[14,31,42]

Probable High Risk

Pain described as pressure, is similar to that of prior myocardial infarction or worse than prior anginal pain, or is accompanied by nausea, vomiting, or diaphoresis[3,14,24,25,27-29]

High Risk

Pain that radiates to one or both shoulders or arms or is related to exertion[3,14,24,25,27,29]

Three features of chest pain tell whether or not the pain is ischemic in nature:
1- changes with respiration **(pleuritic)**
2- Changes with **position of the body**
3- Changes with touch of the chest wall **(tenderness)**
These three features exclude ischemia as a cause of CP with about a 95% negative predictive value meaning that (1 out of 20 pts presenting with CP will be misdiagnosed)

1- Non substernal pain doesn't exclude ACS, but will decrease the likelihood of it.
2- It's **IMPORTANT** to know what symptoms increase/decrease the likelihood of ACS the most. The Q in the exam may list the symptoms for you and ask what symptom has the highest/lowest likelihood of ACS (Check the table and LR rate). If the pain changes with respiration (pleuritic), changes with body position or if there is tenderness of chest wall (indicates a nonurgent musculoskeletal type of pain), cardiac cause of pain is highly unlikely.

1- ACS Clinical presentation (*cont.*)

Levine sign (Dr: everybody has their own different way to express their pain,you have to know what they really mean with their hand gestures)

- Levine's sign is a clenched fist held over the chest to describe ischemic chest pain.

- The Levine Sign has a **poor sensitivity** for chest pain related to myocardial ischemia or infarction.
- A patient pointing to a specific point on the chest likely does not have discomfort due to cardiac ischemia or myocardial infarction.
- **Larger areas of chest discomfort correlate with a greater likelihood of cardiac ischemia or myocardial infarction.**

High likelihood of ACS

Low likelihood of ACS

Stable angina VS Unstable angina

All Angina's start as unstable. They either progress into NSTEMI or become stable angina by treatment.

Unstable angina	Stable angina		Chest Pain
	Typical (Definite)	**Atypical** (Probable)	**Non** Cardiac
• **New onset**[1] with normal activities. • Crescendo, increase in severity. • **NOT** relieved by rest or nitroglycerin. • **Duration:** Less than 20 min.	1. **Substernal** chest pain or discomfort. 2. Provoked by **exertion** or emotional stress[4] 3. Relieved by **rest** or nitroglycerin	Meets **only 2** of the 3 typical anginal characteristics.	Meets **0-1** of the 3 typical anginal characteristics.

ECG Changes

Click here for a quick review of ECG basics

- These findings are seen in the **ABSENCE** of: left ventricular hypertrophy & left bundle branch block[2]
- **ECG should be performed immediately once ACS is suspected (Best initial)**[3], followed by measurement of cardiac biomarkers. (most accurate: angiography)

ECG should be performed < 10 min of arrival (ECG time), You should still do it even if > 1o min passes

Which artery occlusion may presents with silent ECG? Circumflex

Extra (but keep in mind):

- ECG findings can change within minutes and ST elevations can appear or disappear.
- ST depression in leads V1 and V2 would be like ST elevation elsewhere
- It is normal to have an inverted T-wave in V1 and aVR
- ST elevation is more important than ST depression. When you see both in the ECG, the most important will be ST elevation

EXTRA

ECG lead	V2-V5	V1-V3	V3-V4	V4-V6, aVL, I	I,aVL	II,III, aVF
Infarct location	Extensive anterior	(Antero) septal	(Antero) apical	(Antero) **lateral**	Lateral	**Inferior**
Vessel involved	Proximal LAD	LAD	Distal LAD	Distal & Diagonal branch of LAD or **LCX**	Proximal **LCX**	**RCA (PDA)**

Anterior wall MI is much worse than inferior wall MI due to its increased mortality. Infarctions of the posterior wall (V1 and V2) are associated with a very low mortality.

1- New onset: Angina of less than 3 months duration. In general all angina will start as unstable then either progress to NSTEMI or stabilize and become stable angina.

2- ST elevations can be **masked** by a LBBB. Therefore, a LBBB with typical MI symptoms is diagnosed as STEMI, and immediate reperfusion therapy with PCI is required.

3- What's the most **accurate** test for ACS? Coronary angiography, it should be considered with a view to revascularisation in all patients at moderate or high risk of a further events.

4- Due to sudden increase in Ach

2- ACS: ECG

STEMI

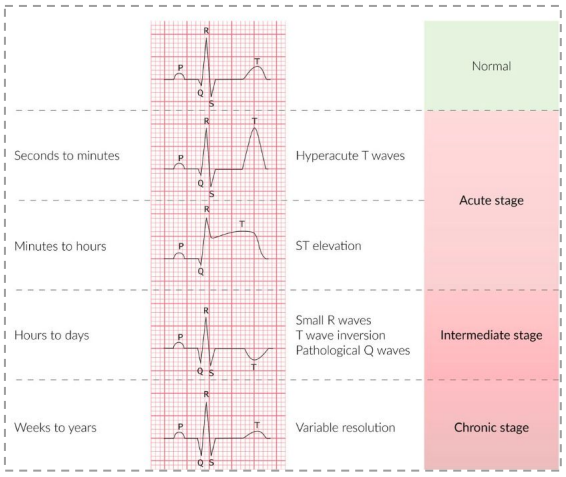
MCQ ★ Within hours there is **ST segment elevation** at the J-point with the cut point: **≥ 1 mm in two or more contiguous leads** (This cut point applies to all leads EXCEPT V2-V3) In the absence of LVH and LBBB

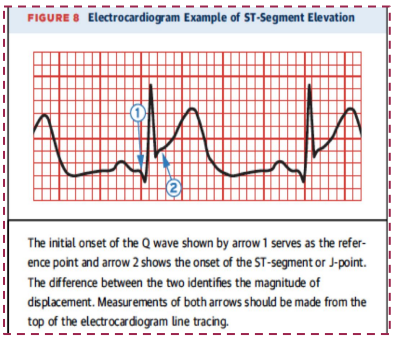
MCQ ★ **The cut point for V2-V3:**

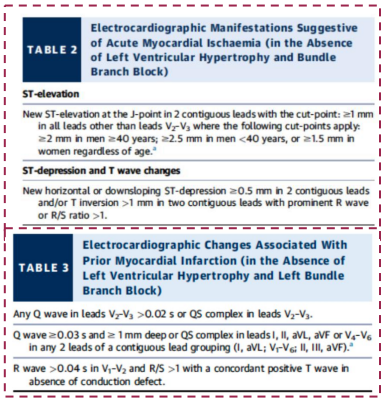
- Men >40 years: ≥2mm
- Men <40 years: ≥2.5 mm
- Women regardless of age: ≥1.5mm

- **What ECG changes are indicative of a prior Myocardial infarction?** In the absence of LVH and LBBB
  - Any **Q waves** in leads V2-V3 >0.02s or QS complex in leads V2-V3.
  - Q wave ≥ 0.03s and ≥1mm deep or QS complex in leads I, II, aVL, aVF or V4-V6 in any 2 leads of a contiguous lead grouping (I, aVL; V1-V6; II, III, aVF).
  - R wave >0.04s in V1-V2 and R/S >1 with a concordant positive T wave in absence of conduction defect.

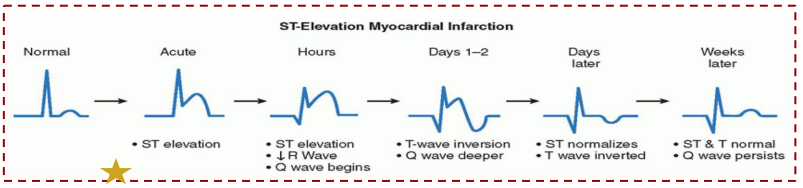
EXTRA







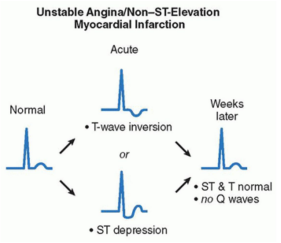
★ Dr: important



NSTEMI & Unstable angina

MCQ ★ Could be anything but **NOT** ST-elevation: New horizontal or downsloping **ST-depression**[1] ≥0.5mm **in 2 contiguous leads** and/or **T inversion** >1mm **in two contiguous leads** with prominent R wave or R/S ratio >1

- **Do all NSTE-ACS present with ST depression? No**, 50% of cases have normal ECG (½ of all NSTE-ACS have no ischemic EKG change).
- **Cardiac enzymes** are required to differentiate between NSTEMI & UA, ECG isn't sufficient.



1- There are different types of ST-depression: 1- Downsloping 2- Upsloping 3- Horizontal. Downsloping and horizontal types are the worst because they indicate ischemia, whereas upsloping is a normal variant that occurs with stress.

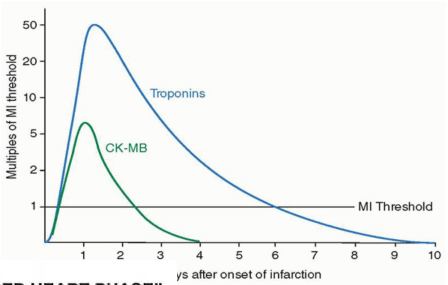
3- ACS: Cardiac biomarkers

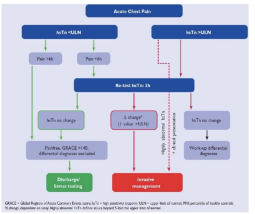
Cardiac biomarkers

Dr: actually the first biomarker elevated is not troponin it is myoglobin but due to unspecificity of myoglobin we do not count it

Troponin[1]	• **Greater sensitivity and specificity than CK-MB** for myocardial injury. (most imp marker) • Troponin I can be **falsely elevated in patients with renal failure.** • Levels of troponins T and I **increase within 3-6 hours**, **peak at about 12-24 hours** and **remain elevated for up to 14 days.**
Creatine kinase[2]	• Creatine kinase (CK), which is also produced by damaged skeletal muscle and brain, is **less sensitive** than troponin for myocardial damage. • The myocardial-bound (MB) isoenzyme fraction of CK is cardio-specific • Levels of CK-MB **Increase within 4 to 8 hours** and **returns to normal in 48 to 72 hrs**; reaches a **peak in 24 hrs.** • **CK-MB is used to detect reinfarction** (because of its short half-life)

★ **Levels of cardiac enzymes are:**
- **High in:** STEMI & NSTEMI
- **Normal in:** Unstable angina
- Troponin is also high in other conditions, check the table. (and the mnemonic below)





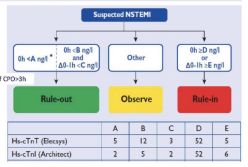


TABLE 1 Reasons for the Elevation of Cardiac Troponin Values Because of Myocardial Injury

Myocardial injury related to acute myocardial ischaemia

Atherosclerotic plaque disruption with thrombosis.

Myocardial injury related to acute myocardial ischaemia because of oxygen supply/demand imbalance

Reduced myocardial perfusion, e.g.,
- Coronary artery spasm, microvascular dysfunction
- Coronary embolism
- Coronary artery dissection
- Sustained bradyarrhythmia
- Hypotension or shock
- Respiratory failure
- Severe anaemia

Increased myocardial oxygen demand, e.g.,
- Sustained tachyarrhythmia
- Severe hypertension with or without left ventricular hypertrophy

Other causes of myocardial injury

Cardiac conditions, e.g.,
- Heart failure
- Myocarditis
- Cardiomyopathy (any type)
- Takotsubo syndrome
- Coronary revascularization procedure
- Cardiac procedure other than revascularization
- Catheter ablation
- Defibrillator shocks
- Cardiac contusion

Systemic conditions, e.g.,
- Sepsis, infectious disease
- Chronic kidney disease
- Stroke, subarachnoid haemorrhage
- Pulmonary embolism, pulmonary hypertension
- Infiltrative diseases, e.g., amyloidosis, sarcoidosis
- Chemotherapeutic agents
- Critically ill patients
- Strenuous exercise

Elevated Troponin Differential Diagnosis Mnemonic – **"SPARED HEART PHASE"**

Stroke	Hypotension	Plaque rupture
Pericarditis	Embolism	Heart failure
Arrhythmia	Anemia	Amyloid
Rhabdo	Renal failure	Sepsis
Exercise	Trauma	Environmental (carbon monoxide)
Dissection		

Dr: IMP ★ **TIMI score**

Thrombolysis In MI (TIMI) score[3]

◆ **Predict 30d and 1yr mortality in <u>UA/NSTEMI</u>:**
<u>One</u> point given <u>for each</u> of the following:

AMERICA

1. **A**ge ≥ 65 y/o
2. **M**arkers (Elevated cardiac biomarkers)
3. **E**CG (ST-segment deviation (≥0.5mm)
4. **R**isk factors (3 or more CAD risk factors) (risk factors are mentioned in page 3)
5. **I**schemic chest pain (2 or more angina events in < 24hrs)
6. **C**oronary stenosis (Prior stenosis of 50% or more)
7. **A**spirin use in the past 7 days.

MD+CALC
Calculate TIMI score with the app

0-1: Low risk[4]
2-3: Moderate risk
≥ 4: High risk

◆ **Risk of major adverse cardiac events (all-cause mortality, new or recurrent MI or severe recurrent ischemia requiring urgent revascularization) at 14 days is based on number of points:**

- **0-1 points: 5%**
- **2 points: 8%**
- **3 points: 13%**
- **4 points: 20%**
- **5 points: 26%**
- **6-7 points: 41%**

1- In cases of STEMI, time is muscle so you shouldn't wait 3-6 hrs for the cardiac biomarkers, Chest pain + ECG suggestive of STEMI are enough to diagnose STEMI. Troponins can be used later to determine the extent of the myocardial injury. Do troponin test after 6h if the pain started in less than 3h

2- CK-MB can be used in patients who have positive troponin chronically e.g. A renal dialysis patient who had surgery (e.g. CABG), post-op he became hemodynamically unstable, you can't use troponin to asses the heart in this case because it's already positive, so use CK-MB.

3- it's useful for assessing the type of treatment needed e.g. if the score is 0-1 points, the mortality rate is low in the next 14 days compared to 6-7 points, so you have time to investigate with echo and other noninvasive stuff, whereas patients with 6-7 point are at high risk and require immediate admission to CCU and may even need urgent cath. With every point there will an increase in mortality.

4- **Low-risk patients** can be managed with oral aspirin, ADP- receptor antagonists, beta-blockers and nitrates. An exercise test should be performed; a negative result has a good prognosis but an early positive test should direct the patient to an invasive strategy. If the patient is unable to exercise satisfactorily, or if the baseline ECG is abnormal (e.g. left ventricular hypertrophy or left bundle branch block), then dobutamine stress echocardiography or myocardial perfusion scintigraphy is recommended.

Dr: Management is not important for your level. Know it without the details.

Management of ACS

◀ 1) Concurrent treatment

Always start with MONA if you suspect ACS
Morphine Oxygen Nitrate Aspirin

Patient must leave the hospital with these 6 drugs:
1. BB **2.** ACEI
3. Aspirin **4.** Plavix
5. Heparin **6.** Statin
(3+4 = DAPT)

ACS Concurrent Treatments

Anti-ischemic therapies
- β-blockers
- Nitrates
- +/- CCB

Antithrombotic therapies

Antiplatelet agents (Use 2)
- Aspirin (Best initial) — DAPT
- P2Y12 inhibitor
- +/- GP IIb/IIIa inhibitor

Anticoagulants (use one)
- UFH
- LMWH
- Bivalirudin

Adjunctive therapies
- Statin
- ACE inhibitor

Anti-ischemic & Adjunctive therapies

1- Beta-blockers (e.g. Metoprolol, Atenolol)

- Recommended within the first 24 hours of admission
- Avoid in patients with hypotension, features of heart failure, bradycardia, RCA occlusion, cardiogenic shock (e.g., large LV infarct, low ejection fraction) or asthma.
- BB reduces long-term mortality by 25%
- COMMIT-CCS trial Day 2-15.
- Reduces HR, decrease workload on the heart
- Reduced the endpoint of death/MI/cardiac arrest.
- 1 month up to 3 year for normal LVEF

2- Statin

★ Early initiation of high-intensity statin (such as **atorvastatin"Lipitor" 80mg**) regardless of baseline cholesterol, LDL, and HDL levels.
- Stabilize plaques and lower cholesterol levels; should be part of acute and maintenance therapy
- PROVE-IT trial.
- LDL? Superior stabilization of vulnerable plaque

3- Nitroglycerin (NTG)

- Sublingual or intravenous **nitrate**
- For symptomatic relief of chest pain
- **All patients must be discharged with it**
- **Contraindications:** inferior wall infarct (due to risk for hypotension), hypotension, and/or PDE 5 inhibitor (e.g., **sildenafil**) taken within last 24 hours

Others

4) **PPI:** to prevent peptic ulcer caused by ASA
5) **Morphine**
6) **Oxygen**: in case of cyanosis, severe dyspnea or SpO2 <90%.
7) **Regular activities[1]:** 1wk if revascularized/ 1 month for sport.
8) **ACEI[2]:** ISIS- 46 weeks, PEACE no benefit

Antiplatelet agents

All patients with ACS should receive ★ **(very imp) Dual Antiplatelet Therapy (DAPT) (Aspirin + P2Y12 inhibitors)** immediately upon arrival to the ER and stay on it for 12 months.

1- Aspirin (ASA)

★ Reduces coronary reocclusion by inhibiting platelet aggregation (By inhibiting COX-1 enzyme) on top of the thrombus. **It's the best initial management (used for all acs cases), and has highest mortality decrease**
- Chewable 160 to 325 mg at presentation, then 75 to 325 mg daily
- ISIS-2: Within 24hr of STEMI, giving **aspirin reduces CV mortality** by 23% at 5 weeks f/u. Giving SK with aspirin decreases mortality by 42%.

2- P2Y12 inhibitor: Clopidogrel (Plavix), Ticagrelor[3] and Prasugrel

- More potent than ASA. Keeps stent open
- CURE trial: Effects on clopidogrel in addition aspirin in patients with ACS without ST-elevation.

1- Resumption of activity can happen as soon as a week if the Pt can do activity without CP. However, you can also do a stress test to check their functional capacity especially if they were athletes.

2- Long-term treatment with ACE inhibitors such as enalapril (10 mg twice daily) or ramipril (2.5–5 mg twice daily) can counteract ventricular remodelling, prevent the onset of heart failure, improve survival, reduce recurrent MI and avoid rehospitalisation.

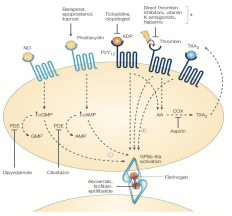
3- Nowadays **ticagrelor is prefered** over clopidogrel to be given with aspirin, bc it has a rapid onset and in a subset of diabetic patients the benefit is extending and marginally better than plavix, but if the patient can't afford it you can give clopidogrel. Clopidogrel takes 6 hrs for the onset of action, whereas Ticagrelor 2hrs.

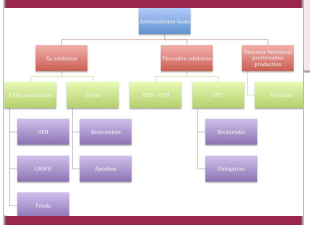
Management of ACS (*cont.*)

◄ 1) Concurrent treatment cont.

Platelet activation:

- **Activators:**
  - Collagen, vWF and Thrombin
- **Consequences of activation:**
  - AA ⟶ COX-1 ⟶ TXA2 ⟶ Release of granule content(ADP, Serotonin and Fibrinogen)





Anticoagulants[1]

TABLE 87-3 Comparison of the Features of Heparin, Low-Molecular-Weight Heparin, and Fondaparinux

FEATURE	HEPARIN	LMWH	FONDAPARINUX
Source	Biologic	Biologic	Synthetic
Molecular weight	15,000	5000	1728
Target	Xa and IIa	Xa and IIa	Xa
Bioavailability (%)	30	90	100
Half-life (hr)	1	4	17
Renal excretion	No	Yes	Yes
Antidote	Complete	Partial	No
HIT	<5%	<1%	Never

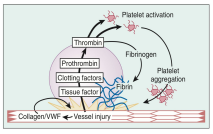
Low molecular weight heparin (LMWH), Unfractionated heparin (UFH) and, Bivalirudin, Fondaparinux

- Initiate in all patients with MI; prevents progression of thrombus; however, has not been shown to decrease mortality. Used specifically in NSTEMI/UA
- LMWH, specifically **enoxaparin, is preferred[2] over UFH**
- Anticoagulants are typically **stopped after the PCI.**
- **If PCI is not performed**, anticoagulants are typically **administered for at least 48 hours**, and preferably longer, for the duration of hospitalization (up to 8 days.)

Why is Low molecular weight heparin (LMWH) preferred over UFH?

- Greater anti-Xa activity (so greater thrombin inhibition).
- Greater release of tissue factor pathway inhibitor.
- Less thrombocytopenia.
- Higher bioavailability so s/c administration.
- Less binding to plasma protein so **more consistent** effect and **no monitoring required.**

Where All the Wars Start?



Note: If the patient has a history of Heparin Induced Thrombocytopenia (HIT) give fondaparinux.

◄ 2) Revascularization pathway

Acute coronary syndrome Revascularization pathways

STEMI

Emergent PCI available within 90 min?
(120 min if transferring to a PCI-capable hospital)

Yes

Primary PCI (First line/BEST)
(It's both Diagnostic and therapeutic tool)

No

Fibrinolytic therapy
(If no contraindication)

UA & NSTEMI

Risk Assessment
(e.g. Troponin, ECG, TIMI Score)[3]

Low

Conservative Strategy
(Proceed to cardiac cath if angina recurs or subsequent **stress test** shows substantial ischemia)

High

Invasive strategy
(Early cardiac cath with PCI or CABG as dictated by coronary anatomy)

1- A period of treatment with warfarin should be considered if there is persistent AF
2- If there were no contraindications to LMWH e.g. renal failure
3- Revascularization therapy is indicated (<24hrs) In high risk patients This includes those individuals with persistent or recurrent angina with ST changes ≥2mm or deep negative T-wave changes, clinical signs of heart failure or haemodynamic instability, or life-threatening arrhythmias (ventricular fibrillation, ventricular tachycardia).

Management of ACS (*cont.*)

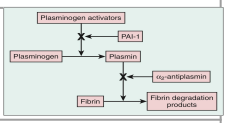
◄ 2) Revascularization pathways cont.

A) Invasive

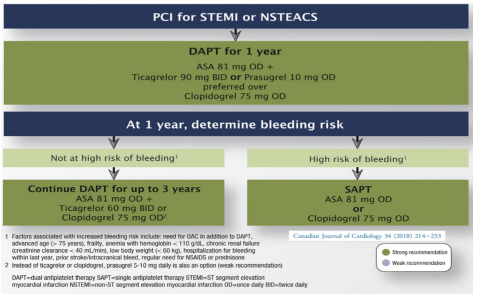
- Angiography is indispensable in evaluating a patient for the possibility of revascularization, which is either **Coronary artery Bypass Grafting (CABG)** or **Percutaneous Coronary Intervention (PCI).**

★ PCI[1]	CABG 
• PCI is commonly referred to as angioplasty. • **PCI is unquestionably the best therapy in ACS.** • **Ideally, door-to-PCI time should be < 90 minutes. It should not exceed 120 minutes.** • If PCI cannot be performed within 120min for any reason, and thrombolysis is contraindicated, the procedure should be performed as soon as practically possible.	• Not routinely recommended • CABG lowers mortality only in a few circumstances: in case PCI was unsuccessful or if it was present with **very severe disease such as:** ○ Three vessels disease ○ Left main coronary artery occlusion. ○ Two-vessel disease in a patient with diabetes

B) Fibrinolytics[2] (Thrombolytics)
(tPA, reteplase or streptokinase)

Uses	• **Only used with STEMI (NOT used in NSTEMI or UA)** • **Used If primary PCI cannot be achieved in < 120 min after onset of STEMI** (in rural area) ★ **Should be given within 12 hrs of admission.** (Has no benefit after 12hrs) • Even where thrombolysis successfully achieves reperfusion, PCI should be considered within 24hrs to prevent recurrent infarction and improve outcome.
Types	1) **Non-fibrin specific:** Streptokinase (allergenic) 2) **Fibrin specific:** Tenecteplase (TNK), Alteplase and reteplase (rPA) 
Contraindications	• **Major bleeding** into the bowel (melena) or brain (intracranial hemorrhage) • Recent surgery (within the last 2 weeks) or major trauma • Severe hypertension (>180/110 mmHg), pregnancy or peptic ulcer (within the last 21d) • **Nonhemorrhagic stroke within the last 3 months.**

Drug	Dosage	Add on
tPA Accelerated regimen **3 Doses**	**15mg** IV bolus → **0.75mg/kg (max 50)** over 30min → **0.5mg/kg (max 35)** over 1hr	Better than SK **(GUSTO-1)** 100mg over 90min
rPA (2 Doses)	**10U** over 2min then 10U at 30min	=tPA
TNK (1 Dose)	Single bolus over 10sec **<60kg=30mg, 90≥50mg** 5mg increment/10kg	=tPA **(ASSENT-2)** but less non-cerebral bleeding &Tx.
All patients get ASA load/UFH 60U/kg max 4000 then infusion 12U/kg max 1000U/hr PTT target 50-70 (UFH not beneficial with SK)		



Dual antiplatelet therapy (DAPT)should be continued for at least 12 months if PCI was performed. After 1 year you can go into Single Antiplatelet Therapy (SAPT) if the Pt has high risk of bleeding or switch to clopidogrel if the Pt is intolerant to aspirin (e.g. develops dyspepsia) or has allergy. If the patient has low bleeding risk you can continue DAPT for 36 months.

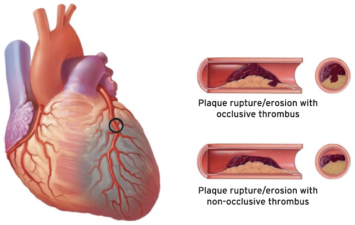
1- GP IIb/IIIa receptor antagonist (e.g., eptifibatide or tirofiban): should be considered in pre-catheterization setting.
2- Just know they exist because they are rarely given.

MI types and complications

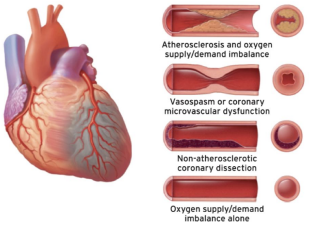
MI types[1]

MI type 1	MI type 2
Caused by CAD with atherothrombotic plaque rupture or erosion leading to either an occlusive or partially-occlusive thrombus. **(The one discussed in this lecture)**	Due to an oxygen supply-demand imbalance, either alone or in combination with atherosclerosis **without plaque rupture**, vasospasm or coronary microvascular dysfunction, or non-atherosclerotic coronary dissection.

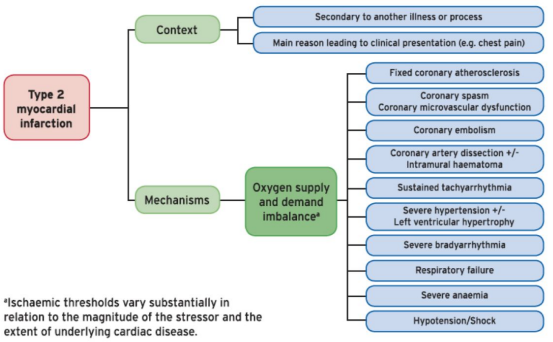
MI Type 1



MI Type 2



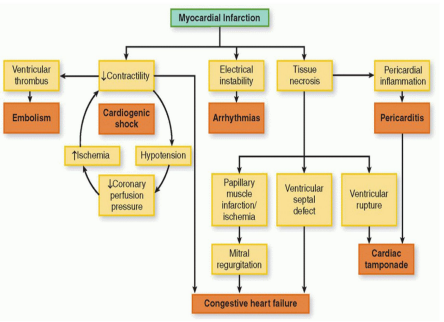
Dr: We see type 2 in ICU patients



MI Complications

DARTH VADER

1) **Death:** Early death is usually due to ventricular arrhythmia and is independent of the extent of MI.
2) **Arrhythmia:** AF and VF, especially if a scar was formed. MOST COMMON CAUSE OF DEATH, treat by B-Blockers
3) **Rupture of papillary muscles:** Usually present with pulmonary edema & shock due to MR.
4) **Tamponade**
5) **Heart failure:** ⅓ of HF presentation to ER is due to ACS
6) **Valve disease**
7) **Aneurysm of the ventricle**
8) **Dressler's syndrome**[2]
9) **Embolism:** due to ventricular thrombus[3]
10) **Recurrence/Mitral regurgitation**



ACS prognosis

OBJ.

- In almost one-quarter of all cases of MI, death occurs within a few minutes without medical care. Half the deaths occur within 24 hours of the onset of symptoms and about 40% of all affected patients die within the first month.
- The prognosis of those who survive to reach hospital is much better, with a 28-day survival of more than 85%. Patients with unstable angina have a mortality of approximately half that of patients with MI. Early death is usually due to an arrhythmia and is independent of the extent of MI. However, late outcomes are determined by the extent of myocardial damage, and unfavourable features include poor left ventricular function, AV block and persistent ventricular arrhythmias.
- The prognosis is worse for anterior than for inferior infarcts. Bundle branch block and high cardiac marker levels both indicate extensive myocardial damage. Old age, depression and social isolation are also associated with a higher mortality. Of those who survive an acute attack, more than 80% live for a further year, about 75% for 5 years, 50% for 10 years and 25% for 20 years.

1- There are 5 types of MI but you only need to know these two. We can differentiate between them by the angiogram, if you see a clot it's most likely type 1 MI, If you don't it's most likely type 2.

2- Occurring 2–10 weeks post-MI without an infective cause, characterised by persistent fever, pericarditis(pleuritic CP, dry cough,friction rub) and pleurisy, and is probably due to autoimmunity. If the symptoms are prolonged or severe, treatment with high-dose aspirin, NSAIDs or even glucocorticoids may be required.

3- That's why you have to do echo before discharging the patient, especially if the Pt had an anterior MI, and in this case you will need to prolong anticoagulation therapy

Management

	Unstable angina	NSTEMI	STEMI

Invasive approach	• Invasive strategy for NSTE-ACS (very high to intermediate risk patients): coronary angiography within 2–72 hours.
	• Ischemia-guided strategy for NSTE-ACS (in stable, low risk patients): Further testing (e.g., exercise ECG, stress echocardiography) is used to evaluate the need for coronary angiography.
	• **When all medications have been given, and the patient is not better, urgent angiography PCI (< 2 hrs) should be done, even if no ST elevations are present**
			• **First line: Emergency coronary angiography with Primary Percutaneous coronary intervention (PCI)** (Both diagnostic and therapeutic tool)
			  ○ Door to PCI time should be **< 90 mins** (should not exceed 120 mins), **if PCI cannot be performed→ start fibrinolytic therapy (thrombolytics)**
			  ○ **GP IIB/IIIa receptor antagonist** (e.g. abciximab, eptifibatide or tirofiban). **Used during PCI and stenting settings, reduces mortality**
			  ○ **Coronary artery bypass grafting (CABG):** not routinely recommended. Only in cases of failed PCI or very severe diseases e.g. TCA, cardiogenic shock

Non- Invasive approach	**[Fibrinolytics (thrombolytics) should not be used]**
	• **Dual antiplatelet therapy (DAPT), includes:**
	  ○ **Aspirin, reduces mortality** and coronary reocclusion
	  ○ P2Y12 inhibitors: Clopidogrel or **ticagrelor** (prefered over clopidogrel due to its rapid onset)
	• Anticoagulants: initiated in all patients with MI, **prevents progression of thrombus** but has not been shown to decrease mortality
	  ○ Unfractionated heparin (UFH)
	  ○ Low molecular weight heparin (LMWH): **Enoxaparin (preferred over UFH).**
	  ○ Fondaparinux
	• **GP IIB/IIIa receptor antagonists:** (e.g. abciximab, eptifibatide or tirofiban)
	  ○ Considered in intermediate/high-risk patients with early-invasive strategy.
	  ○ **Used during PCI and stenting settings**
			• **Fibrinolytic therapy (thrombolytics): should be given within 12hrs of admission (has no benefit after 12hrs)**
			  ○ Non-Fibrin specific: streptokinase (should not be given repeatedly)
			  ○ Tissue plasminogen activator **(t-PA)** (fibrin specific): Alteplase, reteplase, tenecteplase
			  ○ **Contraindications: major bleeding,** recent major surgery, severe HTN, **ischemic stroke < 3 months**
			• Antiplatelet and anticoagulation therapy: should be initiated initially without delaying revascularization AND **in those undergoing PCI.**
			  ○ Dual antiplatelet therapy (DAPT), includes:
			  ■ Aspirin: reduces mortality
			  ■ P2Y12 inhibitors: Clopidogrel, **ticagrelor** or prasugrel **(it is the best option of P2Y12 used in stenting settings, keeps the stent open)**
			  ○ LMWH typically stopped after the PCI

Therapy	In what cases is effect greatest?
Aspirin	Everyone, as the best initial therapy
Clopidogrel or prasugrel or ticagrelor	• Those undergoing angioplasty or stenting, second antiplatelet drug with aspirin • 2 antiplatelet drugs in all MIs
Beta blockers	Everyone, effect is not dependent on time; started any time during admission
ACEI/ ARB	Everyone, benefit best with ejection fraction below 40%
Statins	Everyone, goal LDL <70 mg/dL
Nitrates	Everyone, no clear mortality benefit
Heparin	After thrombolytics/PCI to prevent restenosis, initial therapy with ST depression and other NON-ST elevation events (unstable angina)
Calcium channel blockers	Can't use beta blockers, cocaine-induced pain, Prinzmetal or vasospastic variant angina

Summary of Treatment Differences between Cardiac Events

	Stable angina	Unstable angina/non-ST elevation MI	ST elevation MI
Aspirin	Yes	Yes	Yes
Beta blockers	Yes	Yes	Yes
Nitrates	Yes	Yes	Yes
LMW heparin (enoxaparin)	No	Yes	Yes, but only after revascularization
GPIIb/IIIa meds	No	Yes	No
Thrombolytics	No	No	Yes, but not as good as PCI
CCBs	No	No	No
Warfarin	No	No	No
Antiplatelet drug	No	Yes	Yes

Cases

◀ Case study 1

A 70-year-old man presents to the emergency department complaining of central chest pain that has been getting worse in the past 3 days, although he reports he is not in pain currently. He reports a history of "squeezing" pain in his chest for the past 5 years that occurs occasionally with physical exertion and resolves with rest; this pain has been occurring at rest for the past 3 days, is occurring more frequently (up to 6 times per day), and lasts a few minutes longer than usual. His medical history is significant for hypertension, hypercholesterolemia, a 40-pack-year smoking history, and a family history of heart disease. Vital signs include a temperature of 37.0°C (98.6°F), blood pressure of 130/90 mm Hg, pulse rate of 80/min, respiratory rate of 12/min, and oxygen saturation of 99% on room air. His physical examination is remarkable for slightly decreased breath sounds bilaterally and a normal cardiac examination. X-ray of the chest reveals clear lungs with a normal-sized heart and mediastinum. An ECG showed ST-depression of > 1 mm in leads V4–V6.

- **What is the diagnosis?** This patient's clinical presentation and the ECG showing ST-segment depression of > 1 mm in leads V4–V6 is consistent with ischemia and should be considered acute coronary syndrome (ACS). ACS can be subdivided into ST-elevation myocardial infarction (STEMI), unstable angina and non–ST-elevation myocardial infarction (NSTEMI); an ST-elevation MI cannot be diagnosed in this case given the absence of ST elevation on ECG. Unstable angina and NSTEMI can be distinguished by the presence of positive serum biomarkers for an NSTEMI (troponin I, CK, CK-MB); regardless, their treatment is similar.
- **What are the immediate next steps in management?** ABCs (Airway, Breathing, Circulation) assessed and secured, 12-lead ECG, Cardiac monitor, Oxygen, IV access, Resuscitation equipment at hand, Chewed oral aspirin (162 or 325 mg), Nitrates and morphine, Heparin, β-blocker (if no hypotension, cardiogenic shock)
- **What's the most appropriate next step in management?** The next step, after immediate medical therapy as described above, is risk stratification using the Thrombolysis in Myocardial Infarction (TIMI) risk score to determine how aggressive a therapeutic approach is warranted. An early invasive strategy option is used for high-risk patients (i.e., patients with therapy-resistant chest pain, a TIMI score ≥ 3, ↑ troponin, or ST changes >1mm.)

◀ Case study 2

A 48-year-old man presents to the ED complaining of crushing substernal chest pain. He is diaphoretic, anxious, and dyspneic. His pulse is 110/min, blood pressure is 175/112 mm Hg, and respiratory rate is 30/min. His oxygen saturation is 94%. Aspirin, oxygen, sublingual nitroglycerin, and morphine are given, but they do not relieve his pain. ECG shows ST segment elevation in V2 to V4. The duration of symptoms is now approximately 30 minutes.

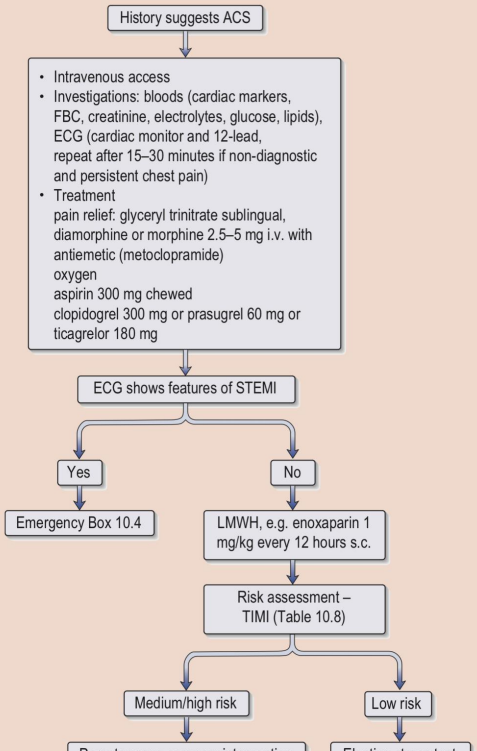
- **What is the diagnosis?** Acute STEMI
- **What are the next steps in management?** It depends on whether the hospital can provide PCI within 90 min or not, if they can then it's the treatment of choice. If not, then fibrinolytics can be given (Contraindications must be ruled out)

Summary

Acute coronary syndrome	
Definition	• Acute coronary syndrome (ACS) refers to acute myocardial ischemia and/or infarction due to partial or complete occlusion of a coronary artery. • **There are three clinical entities grouped under ACS:** unstable angina pectoris, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI).
Symptoms	• Typical cardiac **chest pain is substernal** in nature, often described as a feeling of pressure, and is relieved with rest and/or nitrate use. The pain may **radiate to the lower jaw, neck, shoulders**, epigastrium. • Additionally, autonomic symptoms such as **diaphoresis**, nausea, and vomiting are common.
Diagnosis	• **Best initial test:** ECG • **Most accurate test:** Coronary angiography **STEMI:** • ST elevation + Elevated cardiac biomarkers **NSTEMI:** • Normal or nonspecific (e.g. ST depression) **NO** ST-elevation + Elevated cardiac biomarkers **Unstable angina:** • Normal or nonspecific (e.g. ST depression) **NO** ST-elevation + Normal cardiac biomarkers

Emergency Box 10.3

Immediate management of acute coronary syndrome (ACS)



ECG, electrocardiography; FBC, full blood count; LMWH, low-molecular-weight heparin; STEMI, ST segment elevation myocardial infarction; TIMI, thrombolysis in myocardial infarction.

This figure is important, it will help you understand the proper order in which you should manage patients with ACS

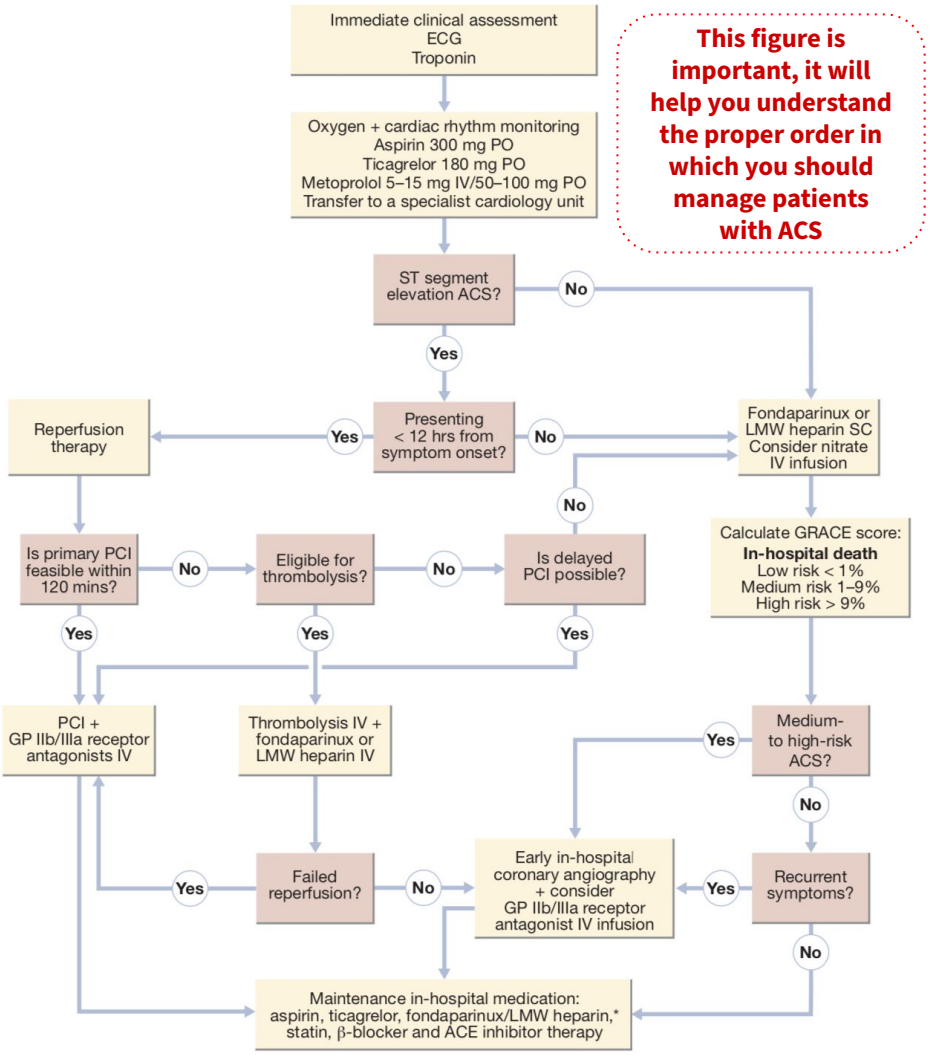


Fig. 16.70 Summary of treatment for acute coronary syndrome (ACS). *Not required following PCI. For details of the GRACE score, see Figure 16.62. (ACE = angiotensin-converting enzyme; ECG = electrocardiogram; GP = glycoprotein; IV = intravenous; LMW = low-molecular-weight; PCI = percutaneous coronary intervention; PO = by mouth; SC = subcutaneous) *Adapted from SIGN 93, Feb 2007, and updated in SIGN 148, April 2016.*

Lecture Quiz

Q1: A 57-year-old man presents to the ED with worsening substernal chest pain occurring over the past 20 minutes. He has a medical history significant for a 2-pack-per-day smoking history, gout, obesity, hypercholesterolemia, hypertension, osteoarthritis of both knees, inflammatory bowel disease, and recently diagnosed type 2 diabetes that is well controlled on oral antiglycemics (hemoglobin A1c of 7.8%). On physical examination, he is in moderate distress, diaphoretic, and nauseous, and has a temperature of 37.5°C (99.5°F), a pulse of 112/min, blood pressure of 142/85 mm Hg, and a respiratory rate of 22/min. He tests positive for myocardial infarction (MI) by serial cardiac enzymes. He is started on the appropriate therapy and is ready for discharge the following evening. What is the number one preventive measure the patient can take that will decrease his immediate risk for a second MI?

A- Decrease the amount of cholesterol in his diet
B- Exercise three times a week
C- Lower his blood pressure to the 120/80 mm Hg range
D- Quit smoking

Q2: A middle-aged man is brought to the ED by ambulance after developing acute-onset hemiparesis and aphasia while at work. Brain imaging identifies a large ischemic area in the right anterior cerebral artery/middle cerebral artery distribution. Bedside carotid duplex shows complete occlusion of the right internal carotid artery just distal to the bifurcation. Lab- oratory tests show a platelet count of 250,000/mm3, blood glucose of 110 mg/dL, and normal coagulation times. His blood pressure is 140/85 mm Hg and he is afebrile. Due to confusion and aphasia, it is almost impossible to obtain a history from the patient. The physician is planning on administering thrombolytics, but needs certain key information beforehand. Suddenly, the patient's wife arrives in the ED. Which of the following items in the patient's recent medical history is a contraindication to thrombolytic therapy?

A- Bruise on the leg
B- Documented carotid stenosis
C- Myocardial infarction 10 years ago
D- Stroke 2 months ago

Q3: 63-year-old man is brought into the ED by his wife after he complains of acute-onset chest pain, "like an elephant is sitting on my chest." After the patient is stabilized, his electrocardiogram is noted to have ST-segment elevations in leads II, III, and aVF. This patient is most likely suffering an acute myocardial infarction affecting which part of the heart?

A- Anterior
B- Inferior
C- Posterior
D- Lateral

Q4: A 65-year-old man presents with central crushing chest pain for the first time. He is transferred immediately to the closest cardiac unit to undergo a primary percutaneous coronary intervention. There is thrombosis of the left circumflex artery only. Angioplasty is carried out and a drug-eluding stent is inserted. What are the most likely changes to have occurred on ECG during admission?

A- ST elevation in leads V1–6
B- ST elevation in leads V5–6
C- ST elevation in leads II, III and AVF
D- ST depression in leads V1–4

Q5: A 60-year-old man presents to accident and emergency with a 3-day history of increasingly severe chest pain. The patient describes the pain as a sharp, tearing pain starting in the centre of his chest and radiating straight through to his back between his shoulder blades. The patient looks in pain but there is no pallor, heart rate is 95, respiratory rate is 20, temperature 37°C and blood pressure is 155/95mmHg. The most likely diagnosis is:?

A- STEMI
B- NSTEMI
C- Aortic dissection
D- Pulmonary embolism

Q6: A 61-year-old man presents with a 2-hour history of moderately severe retrosternal chest pain, which does not radiate and is not affected by respiration or posture. He complains of general malaise and nausea, but has not vomited. His ECG shows ST segment depression and T wave inversion in the inferior leads. Troponin levels are not elevated. He has already been given oxygen, aspirin and intravenous GTN; he is an occasional user of sublingual GTN and takes regular bisoprolol for stable angina. What would be the most appropriate next step in his management?

A- IV LMWH
B- Thrombolysis with alteplase
C- Angiography with stenting
D- Oral clopidogrel

Answers: Q1:D | Q2:D | Q3:B | Q4:B | Q5:C | Q6:A

Answers Explanation File!

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