Coronary Artery Disease

Objectives:

1. Differentiate between stable and unstable angina and develop an approach plan.
2. Recognize ST-Elevation myocardial infarction (STEMI) and know the management guidelines for STEMI.
3. Recall the general guidelines in the management of non-ST-Elevation myocardial infarction.
4. Understand the importance of modification of risk factors in the prevention and management of coronary artery disease.

439 Team Leader:
Nourah Alklaib

439 Team Member:
Sarah AlQuwayz

438 Team Leaders:
Shahad Aldumkh
Rahaf Alshunaiber
Razan Alqahtani

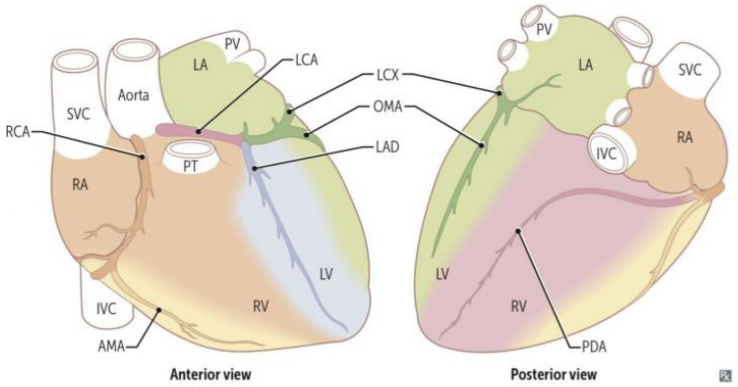
438 Team Member:
Nourah AlHassan

Editing File

Color Index

- Slides / Reference Book
- Doctor notes
- OnlineMeded / Amboss
- Important
- Extra

Anatomy (Blood supply of the heart) (Extra)



Key:

LCA: Left coronary artery
LAD: Left anterior descending artery
LCX: Left circumflex artery
OMA: Obtuse marginal artery
PDA: Posterior descending artery
PT: Pulmonary trunk
RCA: Right coronary artery
AMA: Acute marginal artery

Most commonly occluded arteries:
RCA - LAD - LCX

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.

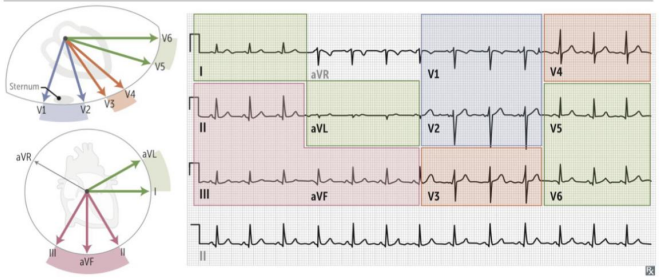
- **Right main coronary artery (RCA):** supplies SA node
  - **Posterior Descending Artery (PDA):** Supplies AV node (dependent on dominance), posterior 1/3 of interventricular septum, posteromedial papillary muscles and posterior 2/3 walls of ventricles.
  - **Acute Marginal Artery (AMA):** Supplies RV.

Dominance circulation of the heart:
- Right (85%) → If PDA arises from RCA
- Left → If PDA arises from LCX

ECG localization of STEMI (Extra)

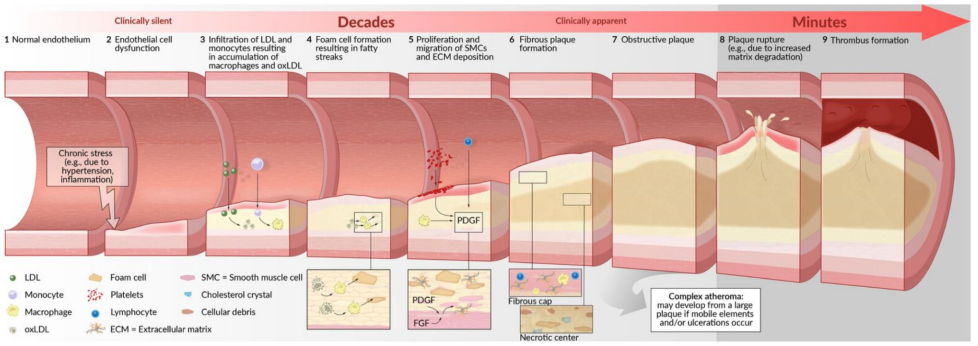
For more ECG basics click here

INFARCT LOCATION	LEADS WITH ST-SEGMENT ELEVATIONS OR Q WAVES
Anteroseptal (LAD)	V_1–V_2
Anteroapical (distal LAD)	V_3–V_4
Anterolateral (LAD or LCX)	V_5–V_6
Lateral (LCX)	I, aVL
InFerior (RCA)	II, III, aVF
Posterior (PDA)	V_7–V_9. ST depression in V_1–V_3 with tall R waves

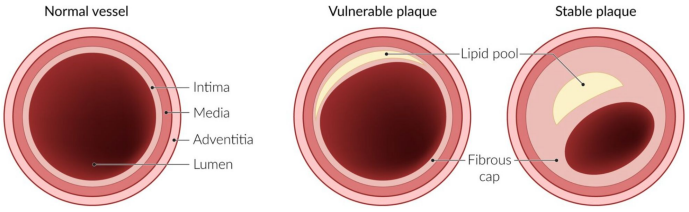


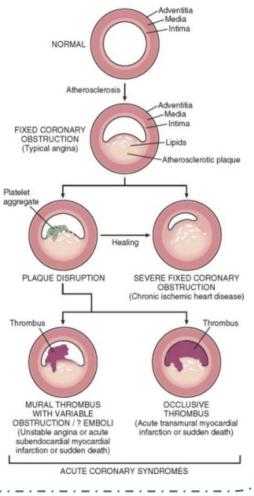
- ECG should be performed in less than 10 mins from arrival.
- These findings are seen in the **ABSENCE** of: left ventricular hypertrophy & left bundle branch block. ST elevations can be masked by a LBBB. Therefore, a LBBB with typical MI symptoms is diagnosed as STEMI, and immediate reperfusion therapy with PCI is required
- 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
- 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.

Pathogenesis



- Ischemic heart disease Also known as Coronary Artery Disease (CAD) is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium.
- Results when there is an imbalance between myocardial oxygen supply and demand.
- Disease of the coronary arteries is almost always due to atheroma and its complications particularly thrombosis.
- Most occurs because of atherosclerotic plaque within one or more coronary arteries
- Limits normal rise in coronary blood flow in response to increase in myocardial oxygen demand
- **Factors reducing oxygen supply**
    - Coronary atherosclerosis and sequelae, including:
    a. Stable atherosclerotic plaque: : lipid pool is surrounded by a thick fibrous cap and leads to extensive narrowing of the arterial lumen → vascular stenosis → increased resistance to blood flow in the coronary arteries → decreased myocardial blood flow → oxygen supply-demand mismatch → myocardial ischemia
    b. Unstable therosclerotic plaque (lipid pool is surrounded by a thin fibrous cap and minimally narrows the arterial lumen)
        - Rupture of an unstable atherosclerotic plaque (most common cause)
        - Thrombosis
        - Stenosis
    - Vasospasms
    - ↑ Heart rate: Perfusion of the coronary arteries occurs during diastole. A higher heart rate shortens diastole, thereby reducing perfusion.
    - Anemia: Anemia is a condition that not only decreases oxygen supply, but also increases oxygen demand, as it can indirectly increase the heart rate.





- **Factors increasing oxygen demand**
    - ↑ Heart rate
    - ↑ Afterload
    - Anemia

Differentiate between stable and unstable angina and develop an approach plan.

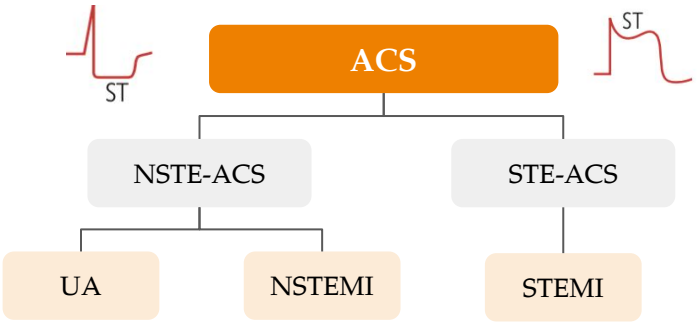
Ischemic Heart Disease (IHD)

- Is the most common form of heart disease
- Globally, ischemic heart disease remains the number one cause of mortality; it was responsible for 7 million of the 53 million deaths reported in 2010
- An estimated 17.9 million people died from Cardiovascular diseases (CVDs) in 2019
  - Representing 32% of all global deaths.
  - Of these deaths, 85% were due to heart attack and stroke.

Spectrum of IHD :

- Chronic stable angina
- Acute coronary syndrome (ACS)
  - Unstable angina (UA)
  - NSTEMI
  - STEMI

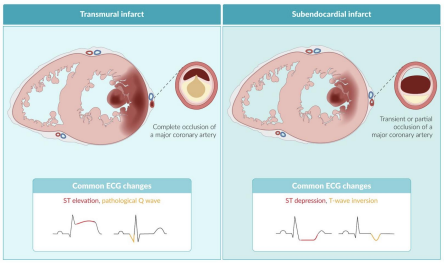
	Stable Angina	*Unstable Angina*	*NSTEMI*	*STEMI*
Pain	Exercise	@ rest	@ rest	@ rest
Relief	Rest + Nitrates	Ø	Ø	Ø
Biomarkers	Ø	Ø	↑	↑
ST Δs	Ø	Ø	Ø	↑
Pathology	70%	90%	90%	100%



ACS: Acute coronary syndromes
NSTE-ACS: Non-ST elevation ACS
STE-ACS: ST elevation ACS
UA: Unstable angina
NSTEMI: Non-ST elevation MI
STEMI: ST elevation MI

Coronary artery occlusion

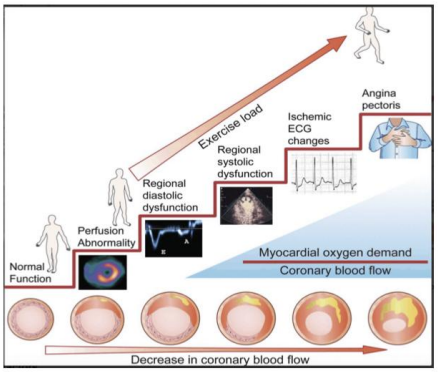
- **Partial coronary artery occlusion:** Usually affects the inner layer of the myocardium (subendocardial infarction) and manifests clinically as unstable angina and/or NSTEMI
- **Complete coronary artery occlusion:** Usually affects the full thickness of the myocardium (transmural infarction) and manifests clinically as STEMI



How Do Patients Present?

Ischemic cascade – with increasing ischemic time:

1. Blood flow changes (can be seen on myocardial perfusion)
2. Diastolic, then systolic dysfunction (wall motion abnormalities)
3. ECG changes
4. Symptoms
5. Myocardial necrosis



Ischemia cascade

Stable angina:

Characterized by:

- Constrictive retrosternal chest pain [1]
- On exertion or emotions
- Relieved by rest (5-10 min) or nitrates [2]

- **Typical chest pain:** all 3 features
- **Atypical chest pain:** 2 out of 3
- **Non-anginal chest pain:** 1 or less
  most likely non-cardiac (most likely GI in origin)

Severity of angina: Canadian Cardiovascular Society grading scale (CCS)

Its imp to document the class, because if the pt progressed to a higher class, this mean it probably progressed to an UA.

CLASS	Characteristic
Class I	No angina with ordinary activity. Angina with **strenuous activity**
Class II	Angina during ordinary activity, e.g. walking up hills, walking rapidly upstairs, with **mild limitation** of activities
Class III	Angina with low levels of activity, e.g. walking 50–100 yards on the flat, walking up one flight of stairs, with **marked restriction** of activities
Class IV	Angina at **rest** or with any level of exercise

History	• Most important factor in making the diagnosis.
	• **Radiates to:** Neck, lower jaw, teeth, arms or shoulders
	• **Atypical presentations:** more likely in elderly, diabetic individuals, and women
	  ○ Stabbing, sharp chest pain
	  ○ No or minimal chest pain "Silent MI" without chest pain is more common in patients with diabetes, as a result of polyneuropathy.
	  ○ Autonomic symptoms (e.g., nausea, diaphoresis)
	• **More common in inferior wall infarction**
	  ○ Epigastric pain
	  ○ Bradycardia

Physical Examination	• It is frequently unremarkable • **Levine's sign:** Clutching fist over sternum when describing chest pain.

DDx	**Careful search for evidence of DDx :**
	Click here for a comparison table of chest pain DDx
	• **Cardiac:** Valvular disease (particularly aortic)[3], HCM, aortic dissection[4], pericarditis, myocarditis, Syndrome X (microvascular disease)
	• **Noncardiac:**
	  ○ **Pulm:** PE, pneumothorax[5] , pulmonary HTN
	  ○ **GI:** esophagitis, esophageal spasm, biliary colic, etc
	  ○ **Chest wall**[6]**:** costochondritis, fibrositis, rib fracture
	• Condition that may **exacerbate angina** (anemia, thyrotoxicosis, arrhythmias)

Risk factors	Important assessment of risk factors e.g. HTN, DM

1. Heavy, tight or gripping central or retrosternal pain may radiate to the jaw and/or arms. **NOT pluritic, positional or tender.** Associated with diaphoresis, dyspnea, N/V.
2. More than 10 min goes more with UA
3. Most similar presentation: Aortic stenosis/regurgitation.
4. Positional pain at rest, sudden
5. PE & pneumothorax → pleuritic chest pain
6. Tender pain

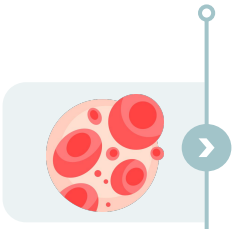
Pre-test probability of significant CAD:

- **Low pretest risk of CAD <15% (light green):** No testing recommended
- **Intermediate >15% (dark green)**
- **High risk > 50% (Orange):** Needs non-invasive stratification for diagnosis and prognosis

Pretest Probabilities of Obstructive CAD in Symptomatic Patients
(A) according to age, sex, and symptoms;
(B) according to age, sex, symptoms, and CAC

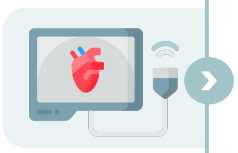
Age, y	Chest Pain		Dyspnea	
	Men	Women	Men	Women
30–39	≤4	≤5	0	3
40–49	≤22	≤10	12	3
50–59	≤32	≤13	20	9
60–69	≤44	≤16	27	14
70+	≤52	≤27	32	12

Investigations



- **Blood tests:** (anemias and hyperthyroidism[1] can exacerbate myocardial ischemia)
  - Hemoglobin
  - Renal panel
  - Serum lipids
  - Fasting blood sugar
  - Thyroid function test

- **Resting ECG:**
  - Often normal
  - Pathologic Q wave = previous infarct.
  - T-wave flattening or inversion in some leads (non-specific evidence of MI)
  - Reversible ST segment depression or elevation, with or without T-wave inversion, at the time the patient is experiencing symptoms (the most convincing evidence of MI)



- **Transthoracic Echocardiography[2]:**
  - Exclusion of alternative causes of angina (Aortic stenosis and HCM)
  - Measurement of LVEF for risk stratification
  - Identification of regional wall motion abnormalities suggestive of CAD
  - Evaluation of diastolic function

1. Can cause chest pain similar to angina
2. Goal: Atypical chest pain: confirm CAD. Typical: look for high risk features and prognosis.

Diagnostic Tests for CAD

Functional

Non-invasive Stress Tests

The next step to determine if the chest pain is coronary in nature, if its positive go to elective catheterization.

- Exercise
- Drugs (Inotropes, vasodilators) picked if patients were unable to exercise

Pick exercise whenever possible!
- Provides prognostic info: e.g. duration of exercise, METs
- Not possible if physical limitations or contraindications (e.g. critical aortic stenosis)

Test

- ECG
- ECG + Echo (Echo is not a choice if there was LBBB or a pacemaker)
- ECG + Nuclear

The goal is to detect stress induced ischemia, confirm CAD and to know if the lesion is high risk or not.
Heart rate is monitored throughout the study:
- **Estimated maximum heart rate =** 220 – age (in years)
- **Target heart rate =** 85% of the maximum heart rate

Consider functional imaging (e.g. nuclear) if:
- Need specific anatomic correlation (e.g. prior revascularization)
- Cannot accurately assess for ischemia on ECG. ECG is not interpretable in:
  - LBBB (RBBB is interpretable generally)
  - Paced rhythm
  - Preexcitation
  - Significant ST changes at rest

Positive stress test : chest pain during the stress test or positive imaging modality
- **ECG** – ST segment change "T-wave inversion or ST segment elevation"
- **ECHO** – Dyskinesia "present during stress but absent in rest this is at risk not dead tissue"
- **Nuclear** – if reversible "normal perfusion at rest, compromised with stress, identifies salvageable tissue "

Structural

- CT coronary angiography it's disadvantage is contrast so some patients if they have bad Cr levels they have to do MPI (Myocardial Perfusion Image)
- Coronary angiography
  Indications for invasive coronary angiography: Suspecting ACS
  - High risk features on non-invasive test
  - Refractory symptoms despite medical therapy

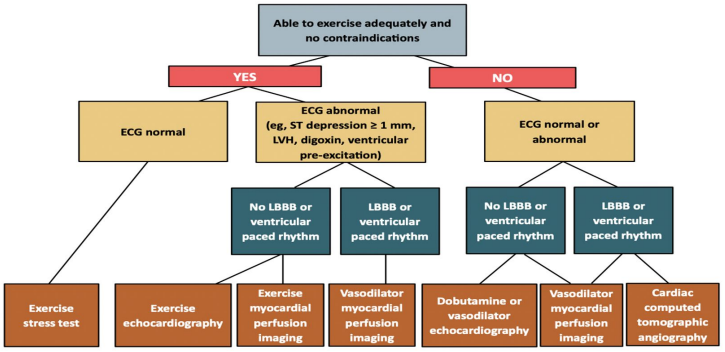


Table 5. High-risk features of noninvasive test results associated with > 3% annual rate of death or MI

Exercise treadmill
- ≥ 2 mm of ST-segment depression at low (< 5 metabolic equivalents) workload or persisting into recovery
- Exercise-induced ST segment elevation
- Exercise-induced VT/VF
- Failure to increase systolic blood pressure to > 120 mm Hg or sustained decrease > 10 mm Hg during exercise

Myocardial perfusion imaging
- Severe resting LV dysfunction (LVEF ≤ 35%) not readily explained by noncoronary causes
- Resting perfusion abnormalities ≥ 10% of the myocardium in patients without previous history or evidence of MI
- Severe stress-induced LV dysfunction (peak exercise LVEF < 45% or decrease in LVEF with stress ≥ 10%)
- Stress-induced perfusion abnormalities encumbering ≥ 10% myocardium or stress segmental scores indicating multiple vascular territories with abnormalities
- Stress-induced LV dilation
- Increased lung uptake

Stress echocardiography
- Inducible wall motion abnormality involving > 2 segments or 2 coronary beds
- Wall motion abnormality developing at low dose of dobutamine (≤ 10 μg/kg/min) or at a low heart rate (< 120 beats per minute)

Coronary computed tomographic angiography
- Multivessel obstructive CAD or left main stenosis on CCTA

Management of stable angina

- Medical management is the cornerstone of therapy and should be started in all patients with CAD.
- Invasive coronary angiography is reserved for a select group of patients.

Goals of Therapy

- Improve symptoms and quality of life
- Improve prognosis, survival
- Prevent non-fatal complications (MI, VT/VF, HF)

Cardioprotective Treatment (Quantity of life: prevent progression of disease)	• ASA (Plavix can be used as SAPT if ASA intolerant) • ACEI/ARB • Statin high potency statins (Atorvastatin, Rosuvastatin) Decreases LDL >50% (goal is LDL <70%) • Treatment of risk factors (HTN, DM, DLP, smoking, exercise program)
Antianginal Treatment (Quality of life: improve symptoms) Click here to view the MOA of each drug (Source: Doctor's slides)	• B-Blocker targeting HR 50-60 bpm (1st line) • Long-acting Nitrates (isosorbide dinitrate) (we don't use the spray) (2nd line) • CCB (3rd line) • If triple therapy (BB, nitrates, CCB) and persistent symptoms → consider revascularization (angiogram) (4th line) • Ivabridine; Ranolazine; Nicorandil; Trimetazidine small vessels that can't be treated by angio. Because some pt even after angio they have chest pain because of these vessels. • Neuromodulation / external counterpulsation • If refractory, consider: transplantation Note: Always ask about PDE5 Inhibitors (Cialis, Viagra etc) before prescribing nitrates as it causes synergistic drop in BP
Revascularization (PCI or CABG)	**Two main indications for revascularization (PCI or CABG) :** ○ Refractory symptoms despite medical management ○ Anatomy with proven prognostic benefit for revascularization (high risk: left main or proximal left anterior descending artery) (Do not intervene if the stenosis is less than 50%) • **Complications of PCI :** bleeding, hematoma dissection and pseudoaneurysm from the arterial puncture site. The use of radial artery reduces these risks. • **After PCI :** Dual Antiplatelet (ASA and P2Y12 inhibitor) should continue for 6-12 months. **Consider CABG (Imp):** ○ Left Main Disease >50% (involving proximal left anterior descending (LAD) or left main stem (LMS) vessel.) ○ Multivessel disease and diabetes. Two-vessel ○ Multivessel disease with LV dysfunction or valvular dysfunction. ○ Three vessel disease • If CABG not an option (elderly, renal problems, very frail and sick in which they are at risk for dying during surgery) , can consider **PCI**

Vasospastic (Prinzmetal's /Variant) Angina

(Not in the obj but part of the doctor's slides)

- Angina caused by transient coronary spasms (usually due to spasms occurring close to areas of coronary stenosis)
- Severe angina at rest with transient ST elevation.
- Worse between midnight and 8:00 am.
- It's more common in females.

Risk factors

5-fluorouracil and triptans

Alcohol

Smoking

Stimulants (e.g. cocaine)

Hyperventilation

Other vasospastic phenomenon: Migraine and Raynaud

Cold

Stress

Diagnosis

ECG	Coronary angiogram
ST segment elevation or depression ≥ 0.1 mV in ≥2 contiguous leads during pain	Coronary spasm on coronary angiogram shows diffuse narrowing gets better when NTG is given.

Complications

MI	AV block	VT/VF	Sudden death

Management

Vasodilators: CCB (1st line), Nitrate, Prazocin (alpha-blocking agent)

Avoid non-selective BB (risk of unopposed alpha stimulation). Worsen the vasospasm.

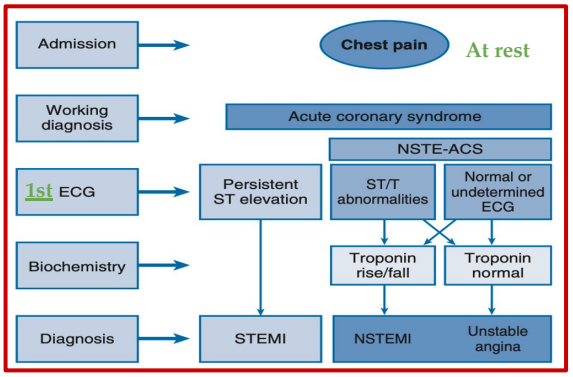
Differentiate between stable and unstable angina and develop an approach plan.

Acute coronary syndrome (ACS)

Features that help differentiate ACS from stable angina:

- Onset of symptoms at rest (or with minimal exertion)
- Lasting longer than 10 minutes unless treated promptly
- Severe, oppressive pressure or chest discomfort
- An accelerating pattern of symptoms that develop more frequently (worsening from baseline), occur with greater severity, or awaken the patient from sleep.

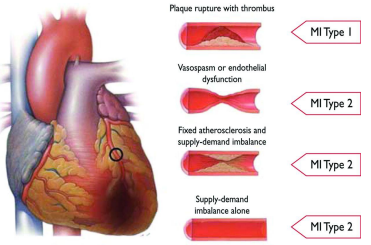
Symptoms alone do not suffice to distinguish the three types of ACS from one another.



	Unstable Angina	NSTEMI	STEMI
Infarcts	No myocardial necrosis	Subendocardial infarcts (inner ⅓)	Transmural infarcts
Occlusion	Non occlusive thrombus	Occluding thrombus sufficient to cause tissue damage & mild myocardial necrosis	Complete thrombus occlusion with more severe symptoms
ECG	Non specific ECG	ST depression +/- T wave inversion on ECG	ST elevations on ECG or new LBBB
Cardiac enzymes	Normal cardiac enzymes	Elevated cardiac enzymes	Elevated cardiac enzymes
Duration	Lasts less than 20 mins		Lasts more than 20 mins

Not All myocardial infarction is ACS

- **Type 1 MI:** MI caused by atherosclerotic plaque disruption or acute coronary thrombosis
- **Type 2 MI:** MI secondary to an oxygen supply/demand mismatch (the patient has a plaque that's not raptured presenting with elevated troponin)
- **Type 3 MI:** MI resulting in death when biomarker values are unavailable
- **Type 4 MI:** MI related to percutaneous coronary intervention
  - **Type 4a:** MI ≤ 48 hours after PCI
  - **Type 4b:** MI related to stent thrombosis
  - **Type 4c:** MI associated with restenosis after PCI
- **Type 5 MI:** MI related to coronary artery bypass grafting



Differentiate between stable and unstable angina and develop an approach plan.

Unstable Angina

Diagnosis	
Patients with typical angina	**Patients not known to have typical angina**
• Increased in severity or duration • Has onset at rest or at a low level of exertion • Unrelieved by the amount of nitroglycerin or rest that had previously relieved the pain	• First episode with usual activity or at rest within the previous two weeks (New onset of CCS3/4 angina) • Prolonged pain at rest

Recognize ST-Elevation myocardial infarction (STEMI) and know the management guidelines for STEMI.

Acute MI (STEMI / NSTEMI)

Diagnosis

Serum cardiac marker elevations and at least 1 of the following:

- Ischemic symptoms
- **Diagnostic ECG changes:**
  - ST-T wave changes, new LBBB
  - Pathological Q wave
- **Imaging:** loss of viable myocardium
- **Coronary angiogram:** Intracoronary thrombus

Acute Management

1. Initial evaluation & stabilization
2. Efficient risk stratification
3. Focused cardiac care

Chest pain suggestive of ischemia → immediate assessment within 10 minutes

Initial labs & test	Emergent care	History & physical
• 12 lead ECG • Obtain initial cardiac enzymes • Electrolytes, CBC, lipids, bun/cr, glucose, coags • CXR	• IV access • Cardiac monitoring • Oxygen • Aspirin • Nitrates	• Establish diagnosis • Read ECG • Identify complications • Assess for repercussion

Focused History

Aid in diagnosis and rule out other causes	Reperfusion questions
• Palliative / provocative factors • Quality of discomfort • Radiation • Symptoms associated with discomfort • Cardiac risk factors • Past medical history (especially cardiac)	• Timing of presentation • Contraindications to fibrinolysis • Degree of STMI risk

Physical Examination

Targeted physical examination	Recognize factors that increase the risk
• Vitals • CVS • Respiratory • Abdomen • Neurological status	• Tachycardia • Hypotension • Pulmonary rales, JVD, pulmonary edema • New murmurs/ heart sounds • Diminished peripheral pulses • Signs of stroke

ECG

UA / NSTEMI

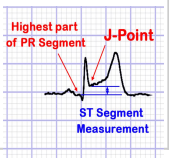
- ECG may be **normal or nondiagnostic** in more than half of patients.
- The most common abnormalities are **ST-segment depression** and **T wave inversion** (more likely to be present while the patient is symptomatic)
- Deep (>0.2 mV) T wave inversions are compatible with, but not necessarily diagnostic of NSTE-ACS.
- **Transient ST-segment elevation** lasting less than 20 minutes occurs in up to 10% of patients (suggests either coronary vasospasm or an aborted infarction)

STEMI

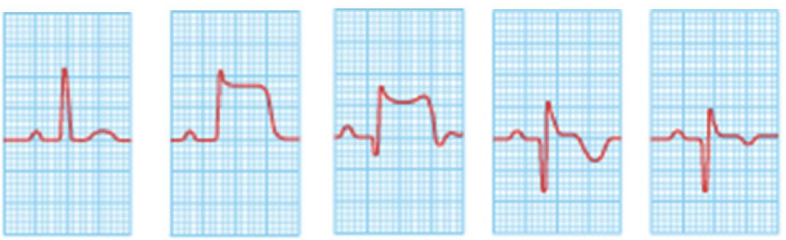
ST-segment elevation (measured at J-point)

- ≥ 1mm in all leads except V2-V3 (amplified leads) (ant. STMI)
- In V2-V3, to be significant:
  - Men < 40: ≥ 2.5 mm [accounting for early repolarization in young men]
  - Men ≥ 40: ≥ 2 mm
  - Women: ≥ 1.5 [accounting for lower amplitude ECGs in women]

In two contiguous leads



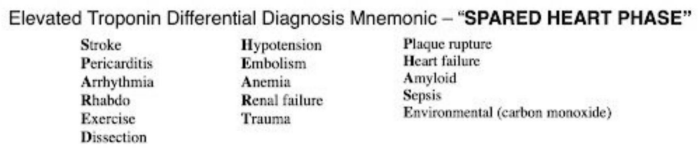
ECG changes with MI



Cardiac Markers (after ECG)

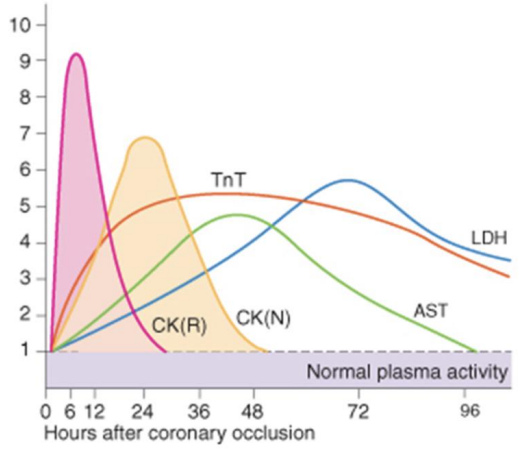
Troponin (T,I)

- Very specific and more sensitive than CK
- Rises 4-8 hours after injury
- May remain elevated for up to two weeks
- Can provide prognostic information
- Troponin T may be elevated with renal disease, poly/dermatomyositis



CK-MB isoenzyme

- Rises 4-6 hours after injury and peaks at 24 hours
- Remains elevated 36-48 hours
- Can be helpful for evaluating reinfarction because of its short half-life but is no longer commonly used
- Positive if CK/MB > 5% of total CK and 2 times normal
- Elevation can be predictive of mortality
- False positives with exercise, trauma, muscle disease, DM, PE



	Initial elevation	Peak	Return to normal
CK-MB	3 - 12 hours	24 hours	48 - 72 hours
Troponin I			5 - 10 days
Troponin T		12 - 48 hours	5 - 14 days

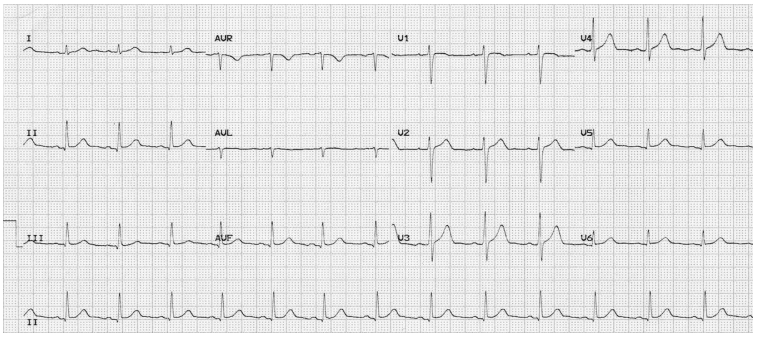
- High sensitivity troponin: first troponin assay on arrival sensitivity and specificity of 90% and NPV of 97%
- If high sensitivity troponin on arrival (0 h) and 3-6 h after arrival with no significant difference between the two sets (< 20%) → NPV of 99%
- **LDH and AST:** may be elevated due to cell necrosis

Transthoracic echocardiogram (TTE):

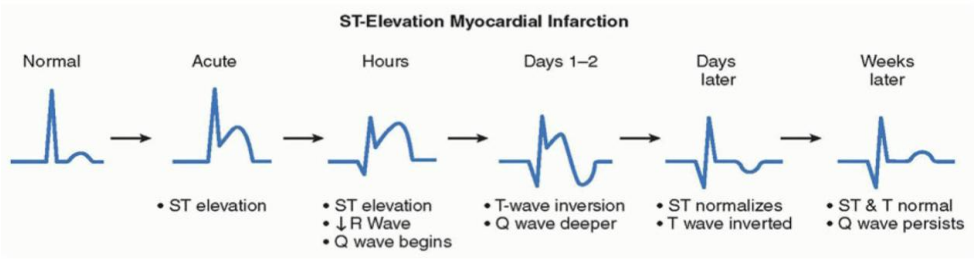
- Confirms MI
- Detetcts wall motion abnormalities
- Detects other dx: aortic dissection, pericarditis or pulmonary embolism

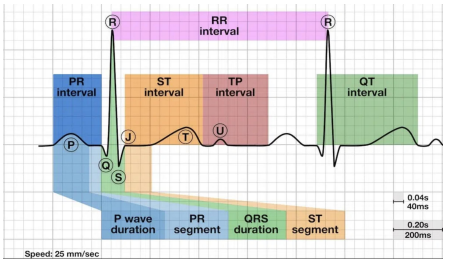
ECG Examples

Normal



STEMI

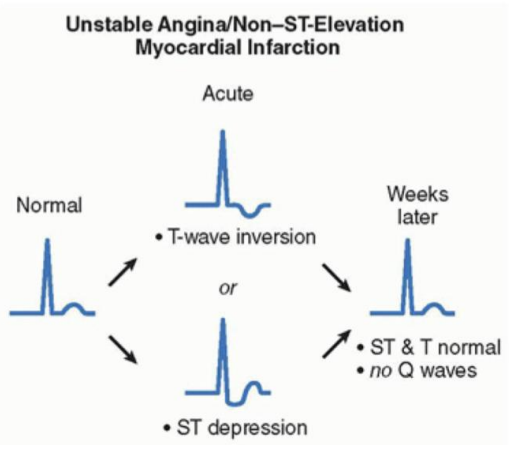




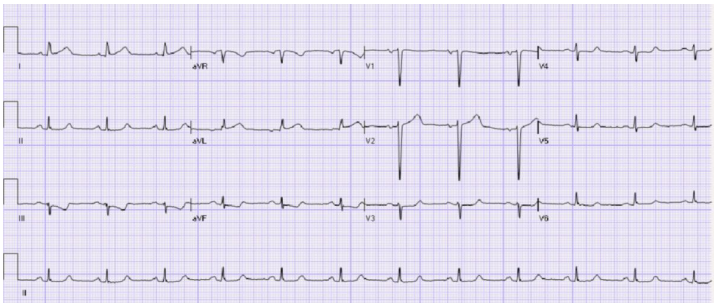
What ECG changes are indicative of a prior myocardial infarction?

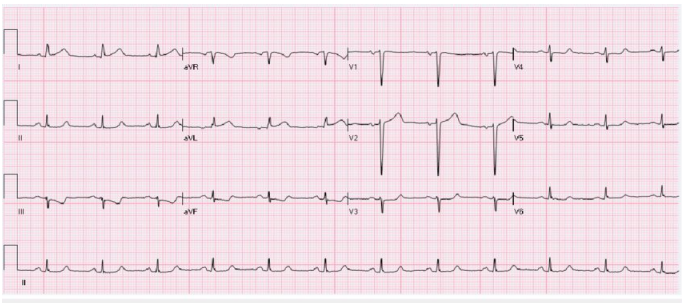
- Any Q waves in leads V2-V3 >0,02s or QS complex in leads V2-V3.
- Q wave ≥ 0.03s and ≥1mm deep or QS complex in leads l, ll, aVL, aVF or V4-V6 in any 2 leads of a contiguous lead grouping (l, aVL; V1-V6; ll, lll, aVF).
- R wave >0.04s in V1-V2 and R/S >1 with a concordant positive T wave in absence of conduction defect.

UA/NSTEMI



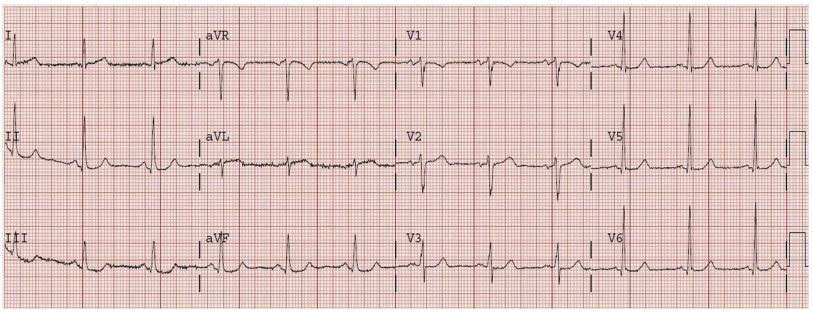
Non-specific T wave Abnormalities



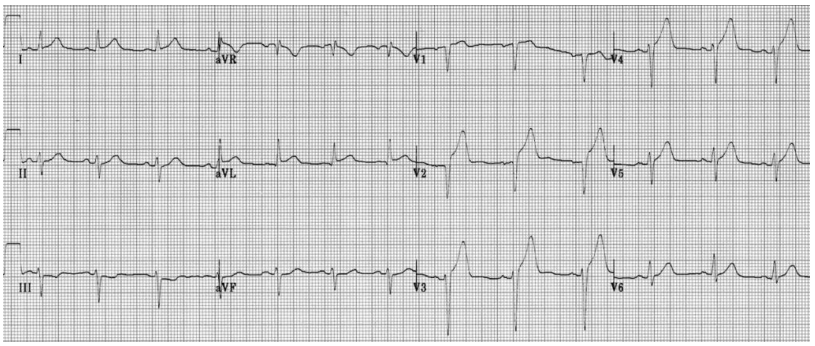


If the patient doesn't have any symptoms we do nothing for it

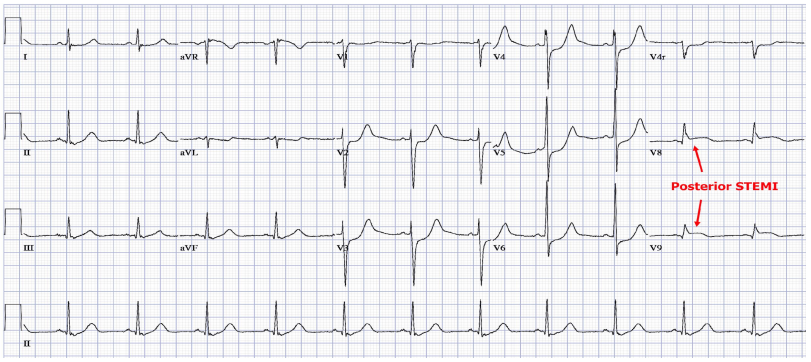
ST Depression Inferior Leads



Anterior STEMI

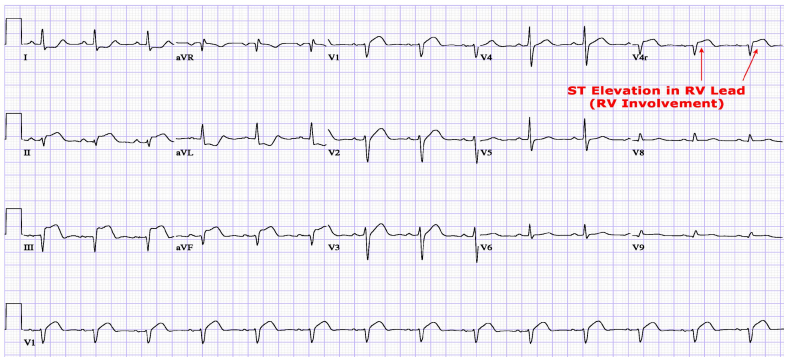


15 Lead ECG - Posterior STEMI



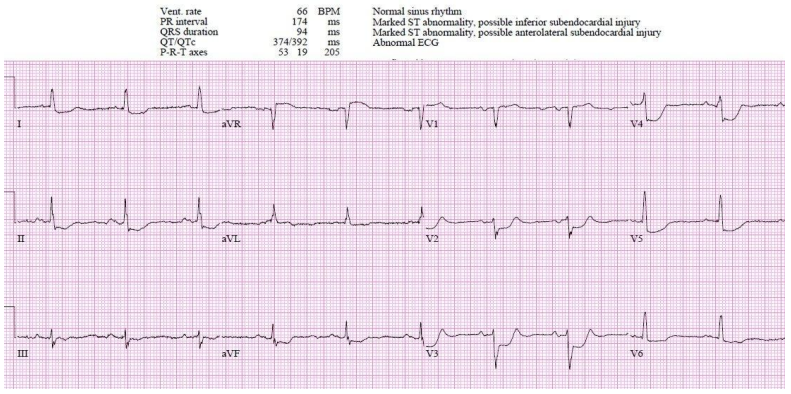
If a patient came with chest pain and the ECG showed ST depression on V1-V2 always do 15 leads ECG to look for posterior STEMI

15 Lead ECG - RV Infarct



To know if there is a RV involvement do a 15 lead ECG

STE in AVR and Diffuse ST Depression

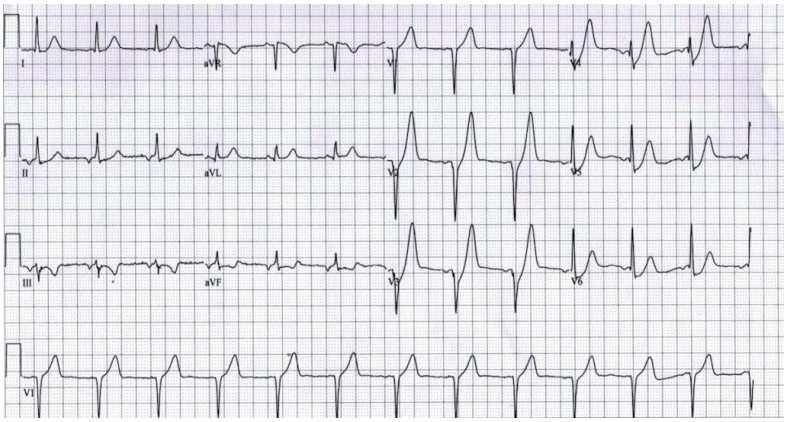


STE ≥ 1 mm in aVR with diffuse ST depression ≥ 1 mm in ≥ 6 leads

STEMI Equivalent

- Left main coronary artery stenosis
- Proximal left anterior descending artery (LAD) stenosis
- Severe triple vessel disease

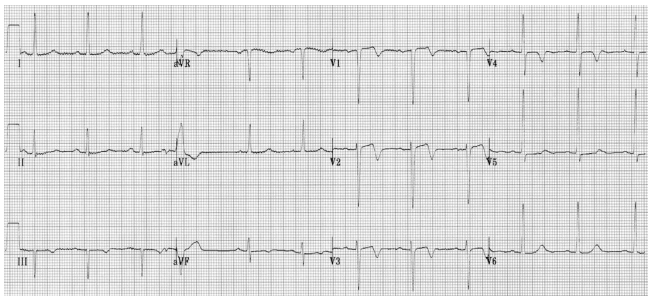
De Winter T Waves



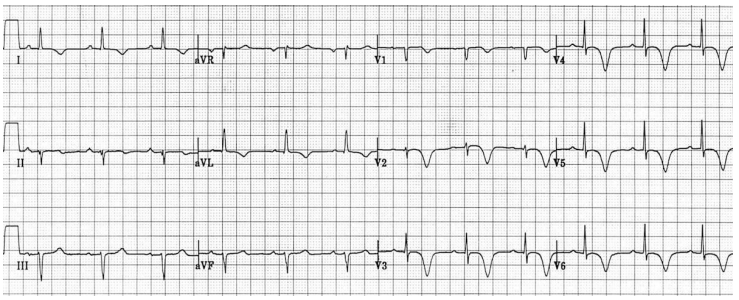
- Upsloping ST depression in the precordial leads (> 1mm at J point)
- Peaked anterior T waves (V2-6), with the ascending limb of the T wave commencing below the isoelectric baseline
- Signifies proximal LAD artery occlusion = **STEMI Equivalent**

Wellens Syndrome: Biphasic T wave

Type A pattern

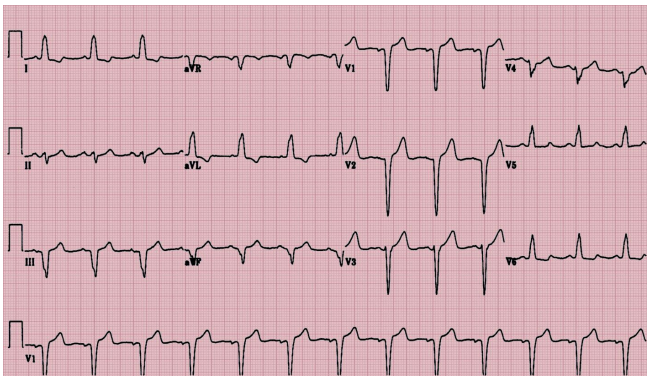


Type B pattern



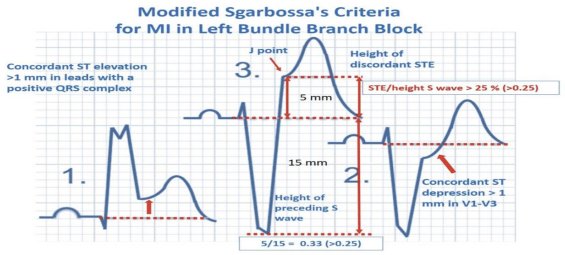
- Deep T waves in the anterior leads (V1, V2 & V3) and biphasic in type A or inverted in type B
- Indicates Critical Proximal LAD stenosis = **STEMI Equivalent**

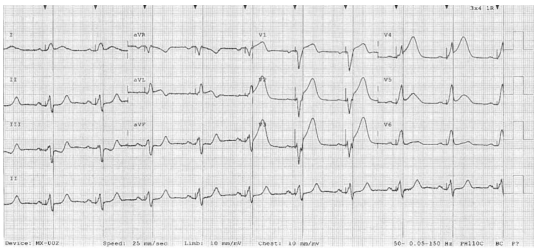
New LBBB



- QRS > 0.12 sec
- L Axis deviation
- Prominent S wave V1-V3
- Prominent R wave 1, aVL, V5-V6
- T-wave inversion

New LBBB that meets Smith-Modified Sgarbossa Criteria





Discordant ST elevation in ≥ 1 lead, as defined by
≥ 25% of the depth of the preceding S-wave

Usually new LBBB comes with ST elevation and you call it STEMI if it met the following criteria
New LBBB with Smith-Modified Sgarbossa Criteria = **STEMI Equivalent**

1. Concordant ST elevation ≥ 1 mm in at least 1 lead, or
2. Concordant ST depression ≥ 1 mm in ≥ 1 lead of V1-V3, or
3. Discordant ST elevation in ≥ 1 lead, as defined by ≥ 25% of the depth of the preceding S-wave

Concordant: ST elevation and QRS complex in the same direction
Discordant: ST elevation and QRS complex in the opposite direction

Recognize ST-Elevation myocardial infarction (STEMI) and know the management guidelines for STEMI.

Management of ACS

1	Immediate Medical Management
2	Reperfusion strategies
3	Chronic Medical Management/risk factor optimization
4	Driving Restrictions

<table>
<tr><th colspan="2">Immediate Medical Management</th></tr>
<tr><td colspan="2">Give NOW</td></tr>
<tr><td rowspan="2">Antiplatelets</td><td>ASA 160 mg</td></tr>
<tr><td>2nd agent:
• Ticagrelor: 180 mg once, then 90 mg BID (PLATO trial)
• Prasugrel: 60 mg once, then 10 mg daily (TRITON-TIMI-38 trial)
○ Contraindicated in pts with prior TIA/strokes
○ Less effect if age ≥ 75 or weight < 60kg
• Clopidogrel: 600 mg once, then 75 mg daily (CURRENT-OASIS 7 trial)
○ Less preferred because requires 2 activation steps (CYP 2C19 reduced-function carriers have low active drug levels, and worse outcomes)
Newer agents (Ticagrelor/Prasugrel) have NOT been evaluated in the setting of Fibrinolysis in STEMI</td></tr>
<tr><td>Anticoagulation</td><td>UHF or LMWH or Fondaparinux</td></tr>
<tr><td>Anti-anginal</td><td>PRN: Nitroglycerin, Opioids, Beta-blocker</td></tr>
<tr><td colspan="2">Start in Hospital</td></tr>
<tr><td colspan="2">• High dose statins
• ACEI/ARB
• Additional agents for LV dysfunction/arrhythmias as indicated</td></tr>
</table>

STEMI management:

Reperfusion Therapy

- STEMI is a true cardiac emergency
- You must recognize a patient with a STEMI and arrange **revascularization** ASAP.

- Options:
  - Primary PCI (also known as angioplasty)
  - Fibrinolysis/pharmaco-invasive ("drip & ship") only used in STEMI (not used in NSTEMI or UA) **Non-fibrin specific:** Streptokinase **Fibrin specific:** Tenecteplase (TNK), Alteplase and reteplase (rPA)
  - CABG if indicated

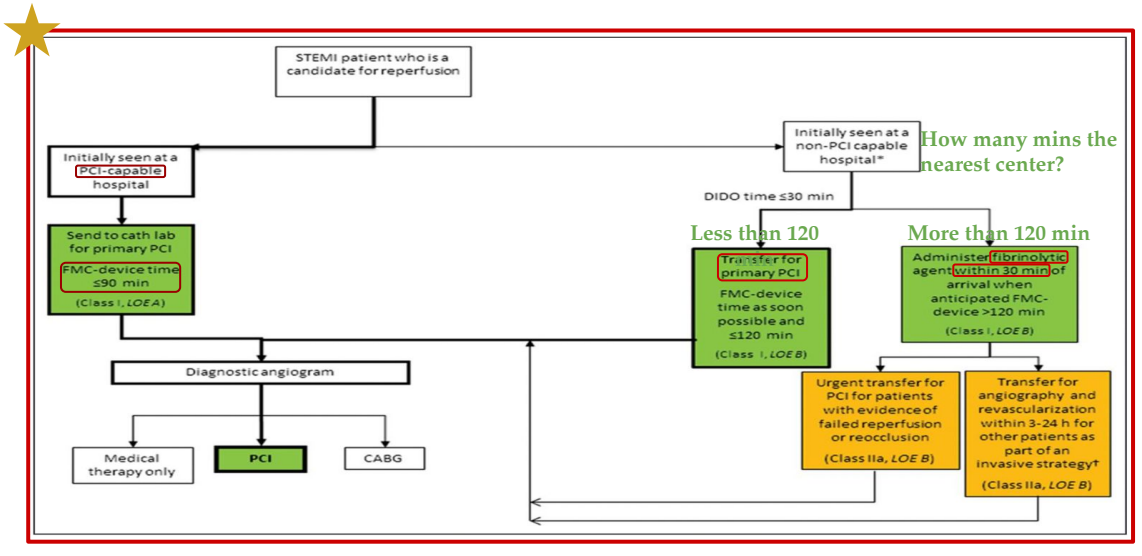
- Eligible for reperfusion therapy?
  - Symptom onset <12 hrs (Class I)
  - Symptom onset 12-24 hrs if: clinical/ECG evidence of ongoing ischemia large area of myocardium at risk or hemodynamic instability (Class IIa)

- When available, percutaneous coronary intervention (PCI) is preferred to thrombolysis
  - Thrombolysis is offered only when PCI cannot be arranged quickly enough.
  - Randomized trials have consistently shown better outcomes in terms or mortality and ischemic events with PCI.
  - Major complications such as intracranial hemorrhage are reduced with PCI.

- PCI is preferred if:
  - If timely:
    - PCI capable hospital: FMC-to-balloon time < 90 min
    - Non-PCI capable hospital: FMC-to-balloon time < 120 min
  - If later presentation (12-24 hrs of symptom onset)
  - If cardiogenic shock

- Thrombolysis is preferred if expected FMC-to-balloon time **> 120 min**
  - If fibrinolysis is selected, should be administered within 30 minutes of FMC
  - Timing of fibrinolysis the earlier, the better, but can be given up to 24 hr after onset of chest pain w STE

- **FMC =** First medical Contact, ER triage if walk-in, or EMS arrival if 911



Absolute contraindications for fibrinolysis therapy in patients with acute STEMI

- Any prior ICH
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
- Significant closed-head or facial trauma within 3 months
- Known structural cerebral vascular lesion (e.g., AVM)
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)

Box 30.41 Contraindications to thrombolysis

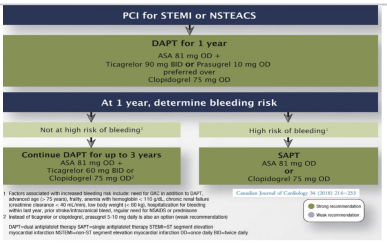
Absolute contraindications	Relative contraindications
• Haemorrhagic stroke or stroke of unknown origin at any time	• Ischaemic stroke in the preceding 3 months
• Ischaemic stroke in the preceding 3 months	• Oral anticoagulant therapy
• Central nervous system damage/neoplasm/vascular malformation	• Pregnancy or within 1 week postpartum
• Recent major trauma/surgery/ head injury (within the preceding 3 weeks)	• Non-compressible vascular punctures
• Gastrointestinal bleeding within the last month	• Traumatic resuscitation
• Known bleeding disorder	• Refractory hypertension (systolic blood pressure >180 mmHg)
• Aortic dissection	• Advanced liver disease
	• Internal bleeding, e.g. active peptic ulcer
	• Dementia

Adjunctive Therapy to Fibrinolysis

- Aspirin
- Heparin
- ACEI / ARB
- BB
- Plavix

Do not give Ticagrelor or Prasugrel when thrombolysis planned

Adjunctive Therapy in PCI (MONA BASH)

Morphine (class I, level C)	• Analgesia • Reduce pain/anxiety—decrease sympathetic tone, systemic vascular resistance and oxygen demand • Careful with hypotension, hypovolemia, respiratory depression
Oxygen (2-4 liters/minute) (class I, level C)	• Up to 70% of ACS patient demonstrate hypoxemia • May limit ischemic myocardial damage by increasing oxygen delivery/reduce ST elevation
Nitroglycerin (class I, level B)	• Analgesia—titrate infusion to keep patient pain free • Dilates coronary vessels—increase blood flow • Reduces systemic vascular resistance and preload • Careful with recent ED meds, hypotension, bradycardia, tachycardia, RV infarction
Aspirin (160-325mg chewed & swallowed) (class I, level A)	• Irreversible inhibition of platelet aggregation • Stabilize plaque and arrest thrombus • Reduce mortality in patients with STEMI • Careful with active PUD, hypersensitivity, bleeding disorders 
Beta-Blockers (class I, level A)	• 14% reduction in mortality risk at 7 days at 23% long term mortality reduction in STEMI • Approximate 13% reduction in risk of progression to MI in patients with threatening or evolving MI symptoms • Be aware of contraindications (CHF, heart block, hypotension, asthma and pulmonary edema) • Reassess for therapy as contraindications resolve
ACE-Inhibitors / ARB (class I, level A)	• Start in patients with anterior MI, pulmonary congestion, LVEF < 40% in absence of contraindication/hypotension • Start in first 24 hours • ARB as substitute for patients unable to use ACE-I 
Statins	• Early initiation of high-intensity statin (Decreases LDL >50% such as atorvastatin"Lipitor" 80 mg and Rosuvastatin) regardless of baseline cholesterol, LDL, and HDL levels. • Stabilize plaques and lower cholesterol levels; should be part of acute and maintenance therapy • Side effect: Transaminases elevation, myositis
Heparin (class I, level C to class IIa, level C)	• LMWH or UFH (max 4000u bolus, 1000u/hr) • Indirect inhibitor of thrombin • **Duration: Stop anticoagulation once patient is revascularized**
P2Y12 inhibitor before or during primary PCI	• **Ticagrelor:** 180 mg once, then 90 mg BID (PLATO trial) • **Prasugrel:** 60 mg once, then 10 mg daily (TRITON-TIMI-38 trial) ○ Contraindicated in pts with prior TIA/strokes ○ Less effect if age ≥ 75 or weight < 60kg • **Clopidogrel:** 600 mg once, then 75 mg daily (CURRENT-OASIS 7 trial) ○ Less preferred because requires 2 activation steps (CYP 2C19 reduced-function carriers have low active drug levels, and worse outcomes)

Recall the general guidelines in the management of non-ST-Elevation myocardial infarction.

Unstable angina/NSTEMI management

- Timing & risk stratification are the major differences vs STEMI
- O2 (for So2 > 90%)
- Morphine if persistent chest pain
- Sublingual nitro (if no contraindications) (Nitroglycerin contraindications: Known history of increased intracranial pressure, severe anemia, right-sided myocardial infarction, or hypersensitivity to nitroglycerin)
- **Dual antiplatelet:** ASA + Plavix/Ticagrelor
- **Anticoagulation:** Unfractionated Heparin/LMWH/Fondaparinux
- ACEI/ARB, BB and Statins

Evaluate for conservative vs. invasive therapy based upon:

- Clinical picture (normal ECG, resolution of CP, clinically stable)
- TIMI risk score more commonly used
- GRACE score

TIMI Score: Predict 30d and 1yrs mortality in UA/NSTEMI: (AMERICA)		
	Yes (1 point)	**No (0 points)**
Age ≥ 65 y/o		
Markers (Elevated cardiac biomarkers)		
ECG (ST-segment deviation (≥0.5mm)		
Risk factors (3 or more CAD risk factors) (HTN, Hyperlipidemia, DM, Smoking, Family history)		
Ischemic chest pain (2 or more angina events in < 24hrs)		
Coronary stenosis (Prior stenosis of 50% or more)		
Aspirin use in the past 7 days.		

Low risk (discharge, non-invasive tests in clinic to decide)	**0-2**
Moderate risk	**3-4**
High risk (admit, angiogram within 24-48h (invasive))	**4-7**

GRACE Score							
Age	<39	40-49	50-59	60-69	70-79	80-89	>90
Points	0	18	36	55	73	91	100
HR	<70	70-89	90-109	110-149	150-199	≥200	-
Points	0	5	10	17	26	34	-
SBP	<80	80-99	100-119	120-139	140-159	160-199	≥200
Points	40	37	30	23	17	7	0
Cr	0.0-0.39	0.4-0.79	0.8-1.19	1.2-1.59	1.6-1.90	2.0-3.99	≥4
Points	1	4	7	10	13	21	28
Killip class	I (no congestion signs)	II (S3, basal rales)	III (acute pulmonary edema)	IV (cardiogenic shock)	Cardiac arrest	Elevated cardiac markers	ST segment deviation
Points	0	15	29	44	30	13	17

Low risk	**1-88**
Moderate risk	**89-116**
High risk	**>117**

Intermediate / High risk patients

- Early invasive strategy (angiogram +/- PCI within 48hr)
- Early invasive reduce risk of rehospitalization for ACS but no mortality benefit.

Low risk patients and/or unclear diagnosis

Non-invasive stress test (often with functional imaging) to determine benefit of invasive testing

Revascularization

Ischemia Guided Strategy
• TIMI score 0-1 • GRACE <109 • Risk of complication associated with coronary angiography or revascularization **Ischemia Guided Strategy:** Medical management. Invasive intervention only if: • Failure of medical therapy (ongoing ischemic chest pain) • High-risk non-invasive stress test
Early Invasive Strategy

• Unstable patient
  ○ Hemodynamic instability
  ○ Heart failure
  ○ Ventricular arrhythmias
  ○ Recurrent and refractory angina
 	
• Positive Troponin
• Positive ECG
• Patient at high risk of recurrent events
  ○ TIMI ≥ 3
  ○ GRACE > 140
 	
• Recent PCI < months
• History of CABG

Understand the importance of modification of risk factors in the prevention and management of coronary artery disease

Risk factors of IHD (Important)		
Major risk factors		**Minor risk factors**
Modifiable	**Non-Modifiable**	• Physical inactivity (Exercise) • Depression • Obesity or metabolic syndrome

• **Diabete mellitus:** The worst
• **Smoking** Immediate improvement of CAD outcomes
• **Hypertension:** Most common
• **Hyperlipidemia (Cholesterol):**
  - High LDL (<130 normal) (Can be due to lack of LDL receptors on the liver surface) OR
  - Low HDL (>60 normal)
	• **Advanced age**
	• **Gender** (Male and postmenopausal women)
	• **Family history of premature CAD.**
	  ○ First degree relative (siblings and parents)
	  ○ Men < 55 y/o
	  ○ Female < 65 y/o	

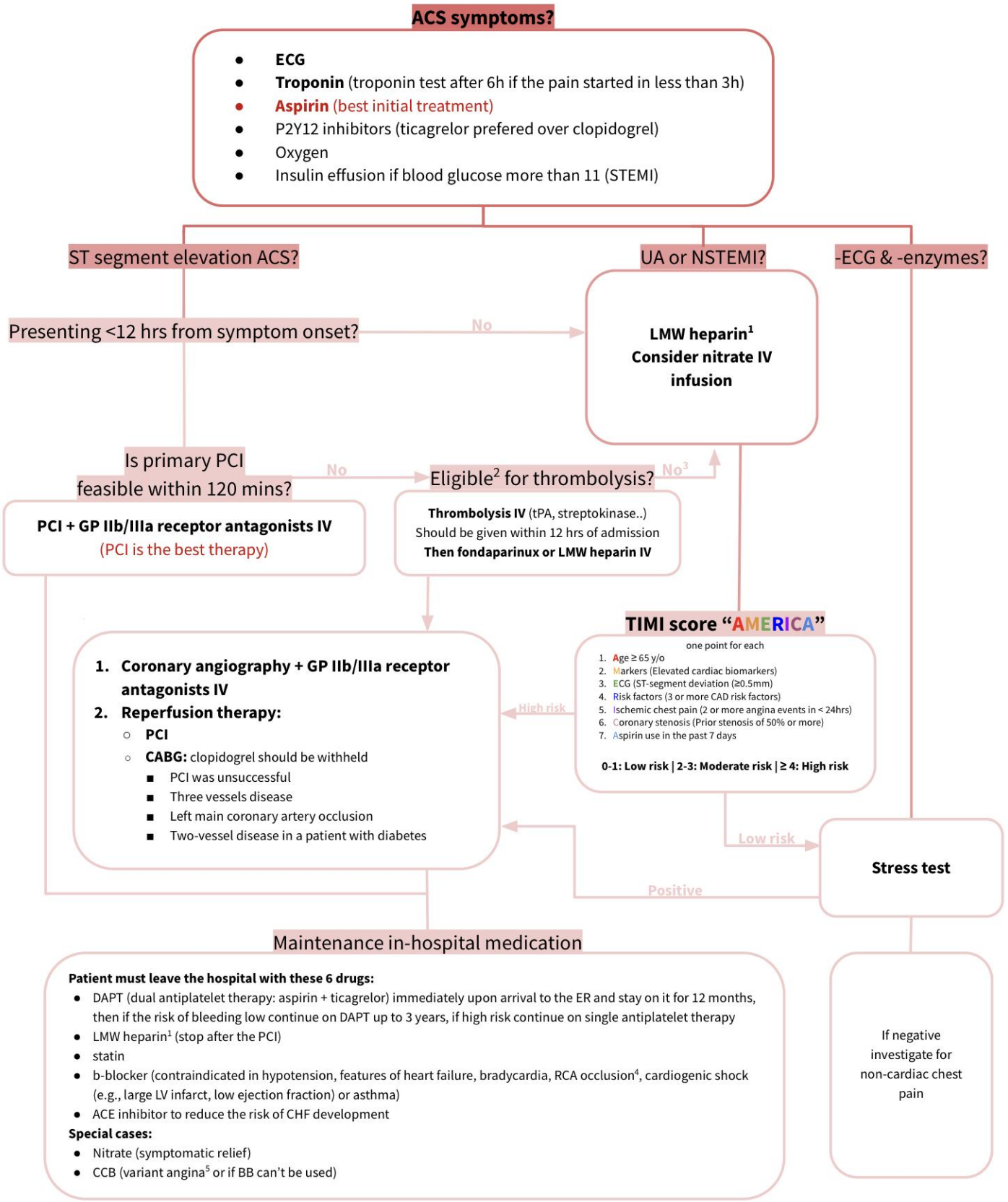
Secondary Prevention	
Disease	• HTN • DM • HLP
Behavioral	• Smoking • Diet • Physical activity • Weight
Cognitive	• Education, • Cardiac rehab program
Blood Pressure	• Goals < 140/90 or <130/80 in DM /CKD • Maximize use of beta-blockers & ACE-I
Lipids	• All patient with CAD should be treated with high intensity/dose statins • Atorvastatin 80 mg daily or Rosuvastatin 40 mg daily • Target LDL < 70 mg/dl (1.8 mmol); TG < 200 • Maximize use of statins; • Consider fibrates as first line for TG>500; consider omega-3 fatty acids
Diabetes	• Target HbA1c < 7%
Smoking cessation	• Cessation-class • Meds • Counseling
Physical Activity	• Goal 30 - 60 minutes daily • Risk assessment prior to initiation
Diet	• DASH diet • Mediterranean diet

Driving Restrictions		
Event	**Private car**	**Commercial driver (truck, bus)**
Elective PCI	48 hours	7 days
CABG	1 month post discharge	3 month post discharge
STEMI		
NSTEMI with significant wall motion abnormalities		
UA or NSTEMI with no LV damage	• PCI performed: 48 hours • PCI not performed: 7 days	• PCI performed: 7 days • PCI not performed: 30 days

Helpful ACS approach from 439's third year medicine's team

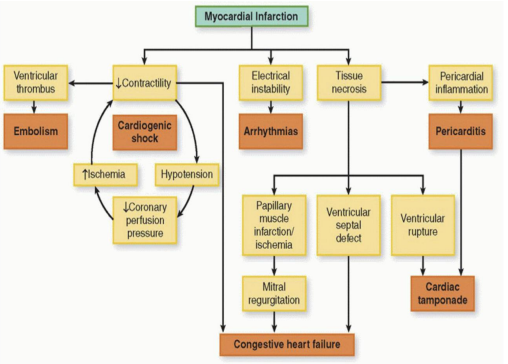
Acute coronary syndrome

Done by Naif Alsulais



1. Contraindicated in renal failure. In case of Heparin Induced Thrombocytopenia (HIT) give fondaparinux
2. Thrombolysis contraindications: Major bleeding or major trauma, recent surgery, Severe hypertension, pregnancy, peptic ulcer and nonhemorrhagic stroke within 3 months
3. If PCI cannot be performed within 120min for any reason, and thrombolysis is contraindicated, the procedure should be performed as soon as practically possible
4. cause hypotension and JVD treated by increasing Preload by IV fluid (BB, nitrate & diuretics are contraindicated)
5. Variant angina seen in young, smokers, healthy with no history cardiac disease at the night for 15 minutes

Post-MI complications

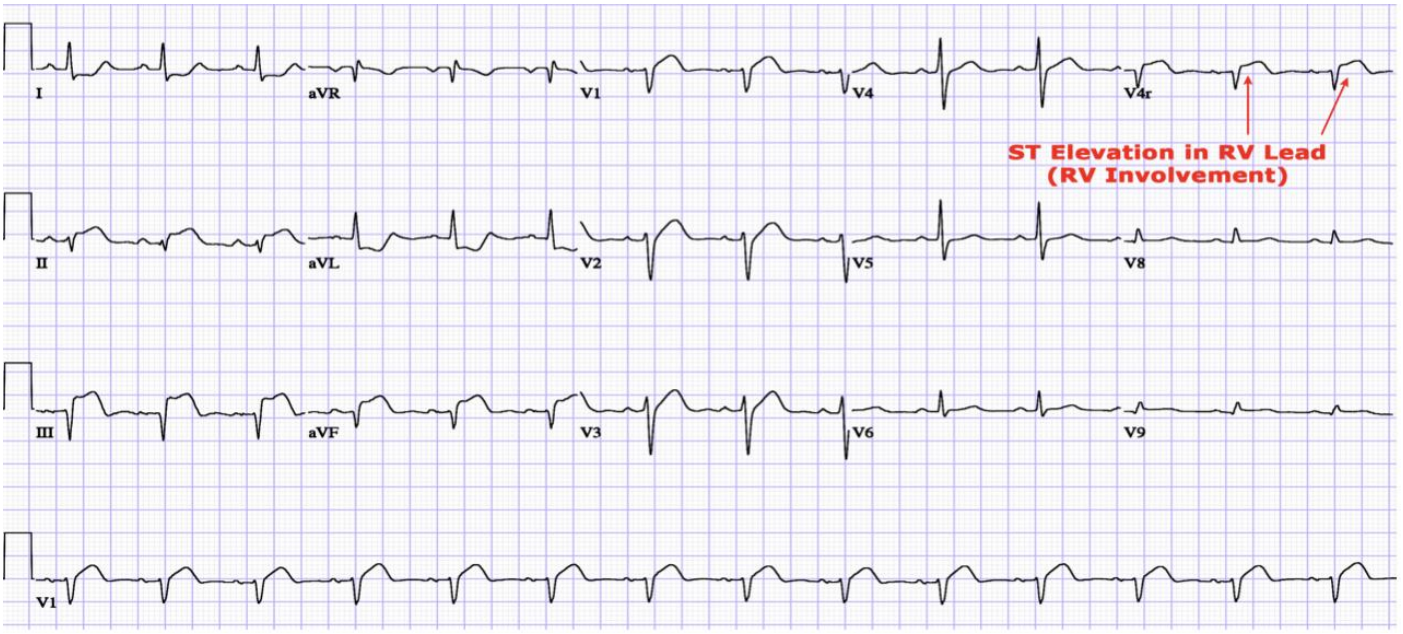


- **Heart failure** (Common)
- **Inflammatory:** pericarditis

- **Arrhythmias**
  - Tachy: atrial, ventricular (The most common cause of death in the first several days after a myocardial infarction is ventricular arrhythmia (ventricular tachycardia, ventricular fibrillation))
  - Bradycardia, heart block (esp. inferior STEMI)

- **Mechanical complications:** (Common)
  - Development of acute severe MR due to papillary muscle dysfunction or rupture
  - Interventricular septal rupture
  - LV free wall rupture

- **RV infarction:** RV involvement most frequently occurs with inferior wall STEMI.
  - **Diagnosis:**
    - **ECG:** ST elevation ≥ 1 mm in lead V1, and > 0.5 mm STE in the right precordial leads (V3R and V4R)
    - **Echocardiography** is used to confirm the diagnosis of RV involvement
  - **Clinical manifestation:** Triad of hypotension, clear lung fields, and increased jugular venous pressure.
  - **Management of RV ischemia:**
    - Early reperfusion
    - Avoidance of therapies that reduce preload (i.e. nitrates and diuretics)
    - Bolus of normal saline to maintain adequate RV preload
    - Inotrope: Dobutamine
    - Maintain AV synchrony (correction of AF, and/or AV block) with sequential pacing if needed.



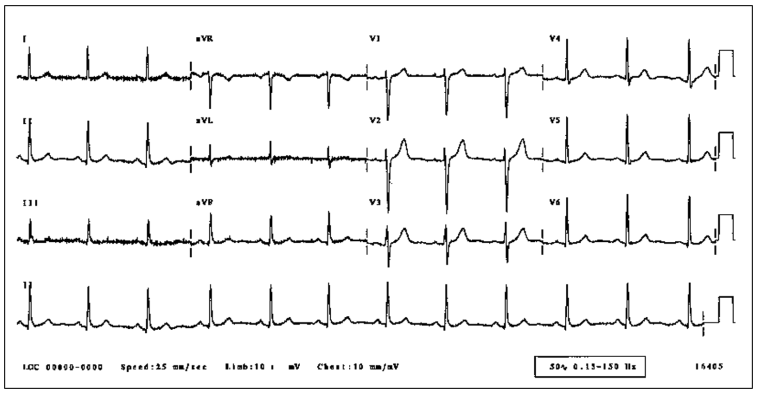
15 lead ECG – RV infarct

Case

A 58-year-old man presents for a general evaluation. He has been experiencing rare episodes of **chest discomfort, which occur with marked activity and are promptly relieved by rest.** His cardiovascular risk factors include hyperlipidemia, prior tobacco use, a family history of coronary artery disease, and a history of hypertension.

Physical examination shows a heart rate of 80/min and a blood pressure of 140/80 mm Hg bilaterally. The cardiac examination is normal. The jugular venous pressure and carotid and peripheral vascular examinations are unremarkable. concentrations are normal.

- **Can you explain his clinical presentation? What is the diagnosis?**
  Stable angina "typical chest pain"



Normal ECG
V2 no ST elevation

- **Labs:**
  - Normal Hb and TSH
  - LDL 4.2 mg/dl (High, Normal LDL <3 mg/dL)
  - HbA1C 7.5% (DM)

- **Echo:**
  - Normal LV size and systolic function
  - Normal RV size and systolic function
  - No significant valvular abnormalities

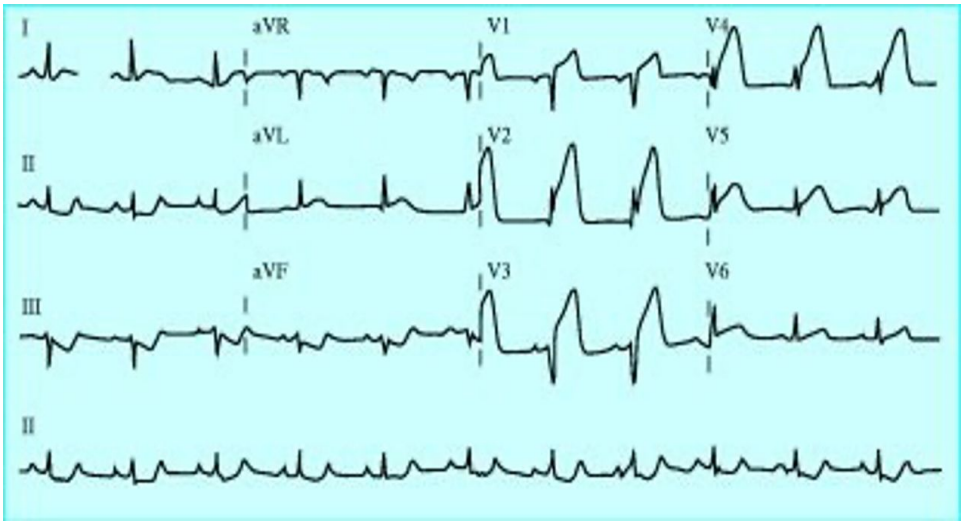
- **Pharmacological Myocardial perfusion scan** was performed as patient can not exercise due to severe knee OA.
  - Low risk anterior wall ischemia

- **How are you going to manage this patient ?**
  - Patient started on the following medications:
    - ASA
    - Lisinopril 5mg OD
    - Atorvastatin 80 mg OD
    - Bisoprolol 5mg OD (Antianginal)
    - Nitro spray PRN
    - Metformin 500 mg BID (given elevated Hba1c)
  - Seen in follow up after 3 months and her symptoms has resolved.

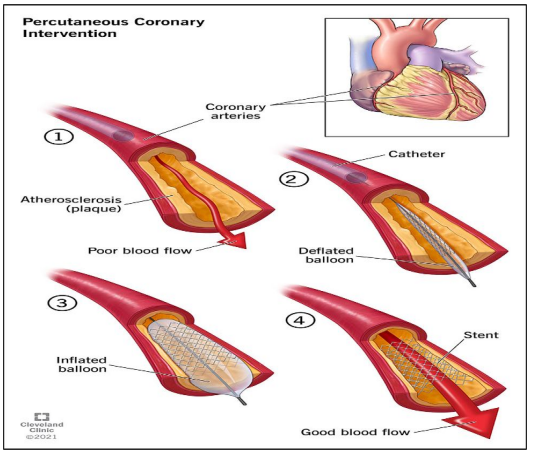
Case

- 2y later, presented with sudden onset retrosternal chest pain **at rest.**
- Gradual progressive then persistent for more than 1 hour.
- Transient jaw discomfort.
- Associated with mild shortness of breath, sweating with nausea.
- Physical exam:
  - In distress.
  - Diaphoretic and pale.
  - Hemodynamics stable.
  - Normal heart sounds, no murmur.
  - Systems review unremarkable

- **Can you explain his clinical presentation?**
- **What is the diagnosis?**
- **Management?**
  Plaque rupture causing ACS, presenting with chest pain at rest and requiring emergent management. Make sure the patient is stable, take a brief Hx & Px and do an ECG (should be within 10 mins) to know if it's STEMI or not.



- Code STEMI activated
- Coronary angiogram showed critical thrombotic stenosis (90-95%) at mid LAD
- PCI with drug eluting stent (DES) to mLAD.
- Uneventful recovery in CCU.



Introduction

Coronary artery disease is blockage in the heart arteries. It's caused by chronically progressive **atherosclerosis** (the plaque) that obstructs the lumen, decreasing the ability of the arteries to perfuse the myocardium. This produces ischemia when cardiac demand increases; there's an imbalance in the demand to supply ratio. For these conditions, both reperfusion (getting rid of the plaque) and reducing the workload of the heart will improve symptoms. When an **acute thrombus** forms from endothelial injury the lumen can quickly become occluded, resulting in a **supply ischemia**; no amount of demand reduction will save this tissue. Reperfusion is required to prevent myocardial death.

The spectrum of coronary artery disease begins with **stable angina** where the coronary artery disease is known and the patient knows how far they can go before symptoms start. **Unstable angina** is worsening of symptoms with less work, more pain with the same work, or pain refractory to nitroglycerin. **NSTEMI** is still demand ischemia, but there's elevation of the troponins. **STEMI** implies acute thrombosis and transmural infarct.

Risk Factors

CAD is just vascular disease in the heart arteries; the risk factors are the same for all vascular disease. **Diabetes**, **Smoking**, **Hypertension**, **Dyslipidemia**, and **Obesity** are **modifiable** risk factors. **Age** (M > 45, F > 55) and **family history** of early vascular disease are **non-modifiable** risk factors.

Patient Presentation

The Diamond classification identifies patients' risk of coronary artery disease based on the symptoms. There are three components. [1]**Substernal** chest pain, [2] **Worse with Exertion**, and [3]**Better with Nitroglycerin**. 3/3 is called typical, 2/3 is called atypical, and 0-1 is called non-anginal. The more positives, the higher the likelihood that this chest pain is anginal. The classic description is a **crushing**, **retrosternal chest pain** that will **radiate down the arm** and **up the jaw**.

Associated symptoms are also useful. The presence of **dyspnea, nausea/vomiting**, or **diaphoresis** with the onset of the chest pain increases the suspicion of myocardial ischemia.

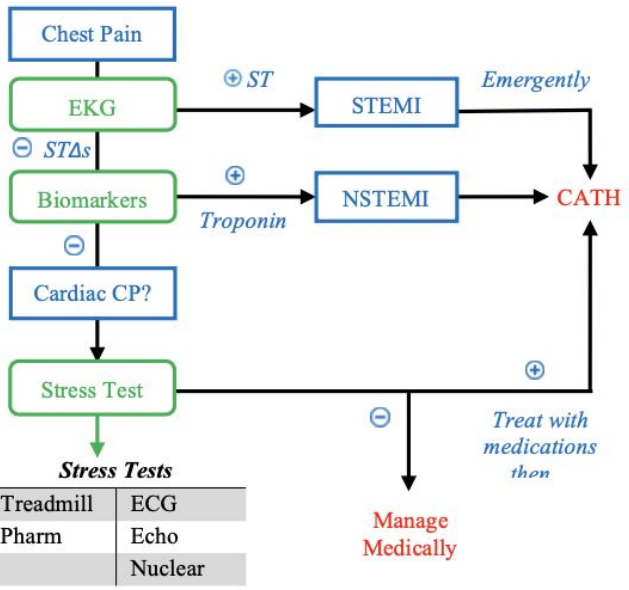
Consequences of the infarct may also be identified. **Congestive heart failure** (pulmonary edema, JVD, poor distal perfusion) and **arrhythmia** (especially heart blocks and ventricular tachycardia) can be seen, but are often absent.

Diagnosis

Rule out the most severe disease (STEMI) first with a **12-Lead ECG** looking for **ST-segment elevations** or a new **LBBB.** STEMI goes to **emergent cath.** If negative rule out NSTEMI with biomarkers (**Troponin-I**). NSTEMI goes to **urgent cath.** If both the troponins and the ECG are negative, you're left considering if this pain is coronary in nature at all. This can be determined using the stress test. If the stress test is positive, go to **elective cath.**

	Stable Angina	*Unstable Angina*	*NSTEMI*	*STEMI*
Pain	Exercise	@ rest	@ rest	@ rest
Relief	Rest + Nitrates	Ø	Ø	Ø
Biomarkers	Ø	Ø	↑	↑
ST Δs	Ø	Ø	Ø	↑
Pathology	70%	90%	90%	100%

Sxs	*Assoc Sxs*	*Risk Factors*
1. Substernal	Dyspnea	Diabetes
2. Exertional	N/V	Smoking
3. Relieved with NTG	Presyncope	HTN
		HLD
3/3 = Typical		Family Hx
2/3 = Atypical		Age > 45 M > 55 F
0-1 = Nonanginal		



Stress Tests	
Treadmill	ECG
Pharm	Echo
	Nuclear

If the person **can't walk** for any reason, use pharmacologic stress (either dobutamine or adenosine)

If the person has a **normal ECG**, use ECG
If the person has an **abnormal ECG**, use Echo
If the person has an **abnormal Echo** or CABG, use Nuclear

Diagnostic Modalities

1. The **stress test**

Regardless of the mechanism used, it's looking for the same thing: evidence of ischemia under stress. The goal is to get the patient to target heart rate (85% of their maximum) and have them sustain it. The test is positive if there's chest pain during stress or the imaging modality is positive. For **ECG** test, look for ST segment changes (T wave inversion or ST segment elevations). For the **Echo,** look for **dyskinesia** (also called akinesis) that's present on stress but absent at rest (this is at-risk but not dead tissue). **Nuclear** stress tests demonstrate perfusion with Thallium. The reversibility (normal perfusion at rest, compromised with stress) identifies salvageable tissue. Whenever the stress test is positive, the next step is catheterization.

2. **Catheterization**

This is the **best test** for the diagnosis of coronary artery disease. It assesses the **severity of stenosis** AND helps rule out **Prinzmetal's angina** (clean coronary arteries producing ischemia as a product of vasospasm - treat with CCB).

Acute Treatment

Patients presenting with angina need **Aspirin**, first and foremost. **Nitrates** can be given to alleviate pain, but must be avoided in right-sided infarcts (II, III, AvF). **Beta-blockers** reduce myocardial work and prevent ventricular arrhythmias (the thing that kills patients in the first 24 hours). **ACE-inhibitors** have long term benefits. **Statins** are the mainstay of therapy for cholesterol. If it's certain this is Acute Coronary Syndrome, **therapeutic heparin** and **clopidogrel load** should be used as well. Oxygen and morphine are used prn.

Interventional management choices are **Stent** or **CABG**. The decision is made based on the severity of occlusive disease. If it's really bad (i.e. requires multiple stents) do a CABG. If the atherosclerosis is global, distal, or microvascular then **medical management only** may suffice.

For thrombolysis, either the administration of **tPA** (within **12 hours** of onset) or **heparin** is done only when catheterization is not available AND they're in an acute disease (STEMI).

Chronic Therapy

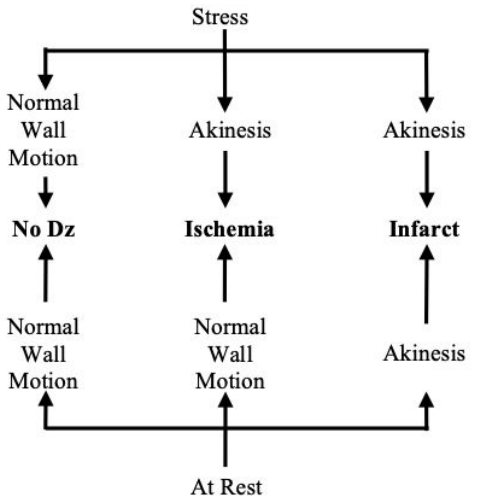
1. *Adjust risk factors*
   a. LDL – **High potency statin**. Old LDL goal < 100. Now, start statin.
   b. DM – tight glucose control to near normal values (**80-120** or **HgbA1C < 7%**) with oral medications or insulin.
   c. HTN – regular control of blood pressure to **<140 / <90** with **Beta-Blockers** (reduce arrhythmias) and **ACE-inhibitors.** Titrate heart rate to between 50-65 bpm and 75% of the heart rate that produced symptoms on stress test.
2. *Reduce Risk of Thrombosis*

Aspirin (Cox-Inhibitor) is the standard therapy. **Clopidogrel** (ADP-inhibitor) can be used if ASA allergy.

Can't Exercise: Peripheral Vascular Disease, Claudication, vasculitis, diabetic ulcers, SOB at rest, etc.

Can't Read ECG: Any BBB or old infarct

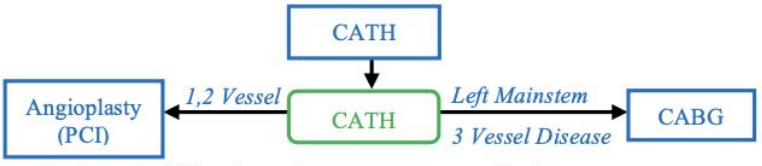
"Dead Things Don't Move"



Acute Presentation: MONA-BASH

Morphine	**B**eta-Blocker
Oxygen	**A**CE-inhibitor
Nitrates	**S**tatin
Aspirin	**H**eparin

Indication	*Duration*
Drug Eluding Stent	Clopidogrel x 12 months
Bare Metal Stent	Clopidogrel x 1 month
Angioplasty Alone	No Clopidogrel



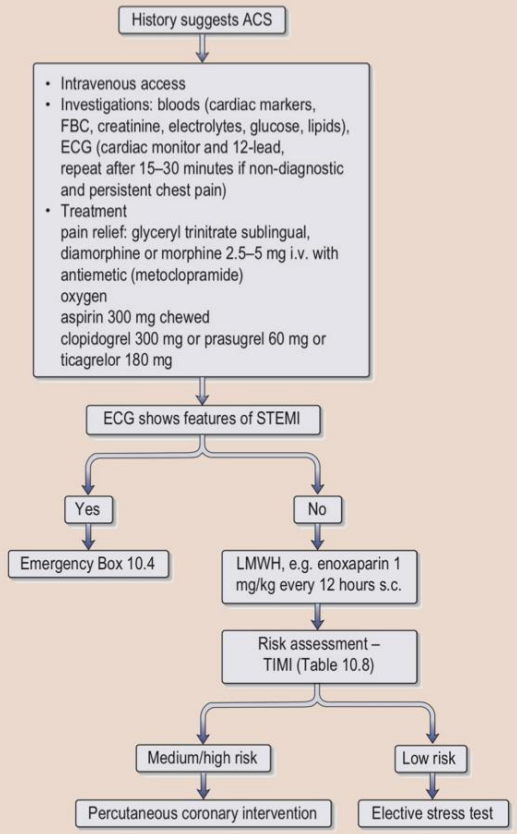
Treatment	*When to use it*	*Goals*	
Statins	Any ACS	LDL < 70	HDL > 40
β-Blockers	Any ACS	SBP < 140	DBP < 90
ACE-i	Any ACS	SBP < 140	DBP < 90
ASA	Any ACS	No goal	
Clopidogrel	ASA allergy or stents	No goal	
Angioplasty	ST↑ or + Stress; 1 or 2 vessel disease		
CABG	ST↑ or + Stress; Left-Mainstem or 3 vessel disease		
tPA	ST↑; no PCI available, no transport		
Heparin	ST↑ or + Stress; contraindication to tPA		

Surgery = Left Mainstem OR 3-vessel disease; surgery = CABG
Angioplasty = 1,2 Vessel Disease

Extra Helpful Information

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.

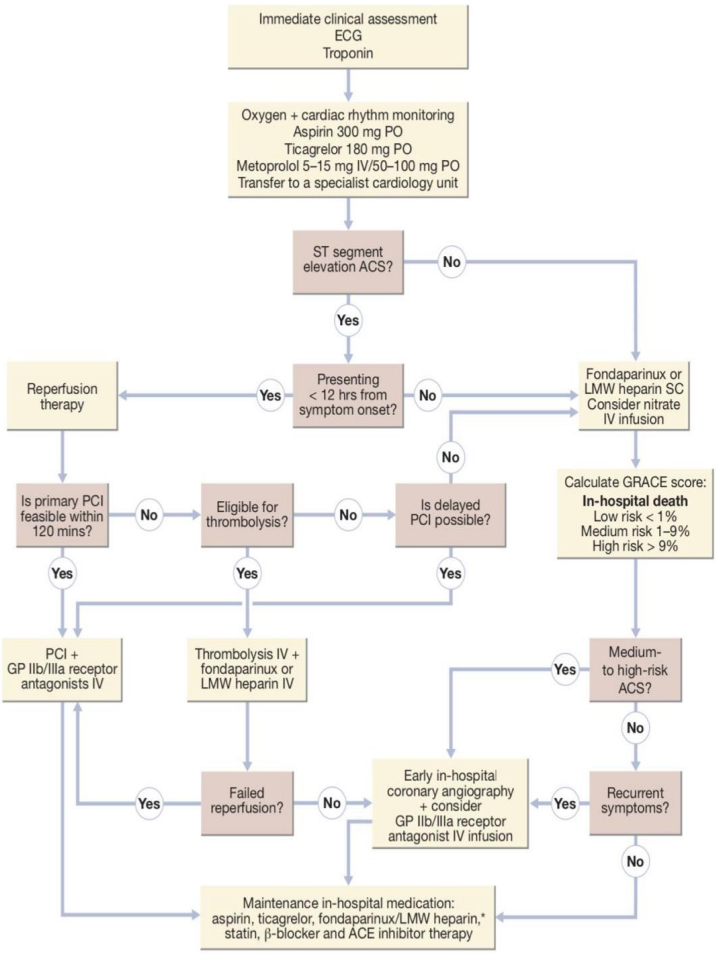


Fig. 16.70 Summary of treatment for acute coronary syndrome (ACS). *Not required following PCI. For details of the GRACE score, see Figure 16.62. (ACE = angiotensin-converting enzyme; ECG = electrocardiogram; GP = glycoprotein; IV = intravenous; LMW = low-molecular-weight; PCI = percutaneous coronary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

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

Door to:	Duration
ECG	10 minutes
Needle (fibrinolytic)	30 minutes
PCI	90 minutes
Transfer to another hospital	120 minutes

Lecture Quiz

1:E / 2:D/ 3:E

Q1: A 56 YO F, comes to the clinic complaining of chest tightness , happened 4 times this month. It occurred across her chest in a belt like distribution. It only happens with exertion, and when she is emotional. She has HTN, DM, and obesity. On examination she was vitally stable and chest pain free, heart and lung sounds were normal . a resting ECG shows no abnormalities. And the result of a treadmill ECG stress test from a year ago were negative for ischemic changes.

Which of the following is the best next step in the management ?

A. Troponin-I
B. Invasive coronary angiography
C. No further diagnostic testing is required
D. Treadmill ECG stress test
E. Coronary computed tomography angiography

Q2:A 67 YO M, comes to the clinic complaining of chest pain. happened 4 times this month while walking around the block. The pain does not radiate and doesn't have any other associated symptoms. If he pause for several min it goes away. He does not get the pain every time he walks and has not noticed whether the time of the day or foods he eat contributes. He have HTN and he is obeese and sedentary. On examination he was vitally stable and chest pain free, heart and lung sounds were normal. Resting ECG showed Q waves in V1-4 and several premature ventricular contractions.

Which of the following is the best next step in the management ?

A. Treadmill ECG stress test
B. Coronary CT angiography
C. No further diagnostic testing is required
D. Dobutamine stress echocardiogram
E. Invasive coronary angiography

Q3: A 56 YO M, complains of chest pain that is crushing and substernal, worse with exertion and better with rest. He knows how far he can go before he gets symptoms. He has HTN, DM, Dyslipidemia and obesity. On examination he was chest pain free, vitally stable. He was sent for coronary CT angiography which demonstrated obstructive CAD. invasive coronary angiography showed 70% stenosis in LAD, 40% in LC and 55% in distal right coronary artery. There were also some minor luminal irregularities in OM1, OM2 and diagonal branch of the LAD.

Which of the following is the best management for his obstructive CAD ?

A. Angioplasty with bare - metal stent (BMS)
B. Angioplasty alone
C. CABG
D. Medical management
E. Angioplasty with drug - eluting stent (DES)

Lecture Quiz

4:C

Q4: **A 75 YO M, presented to the ED with sudden onset chest pain. It is crushing and substernal in nature and present at rest. He has not has this pain before. He was given ASA and NGT in ED with mild improvement in the chest pain but it is still ongoing. He has HTN and Dyslipidemia and he is a smoker. ECG showed ST segment elevation in the lateral leads. He is brought to the cath lab where a 100% occlusion of the LCA which was reperfused by PCI. a drug eluting stent is placed. There are two other stenotic lesions, both near 50% that are not intervened upon.**
Which of the following medication regimen is prefered to treat his hypertension ?

a. Valsartan
b. Carvedilol and amlodipine
c. Metoprolol and valsartan
d. amlodipine and valsartan
e. HCTZ and amlodipine