



Acute Coronary Syndromes: Unstable Angina and Myocardial Infarctions Part 2



**Karen Kopacek, MS, RPh
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Treatment of ACS



1. Management of ACS S/Sx
- 2. Initial Management in ED**
 - A. Patient Evaluation and Physical Exam**
 - B. Immediate General Treatment
3. Acute therapies during hospitalization
4. Chronic therapies after discharge



Initial Management in ED: Evaluation and Physical Exam

Patient Evaluation or Triage:

1. History- previous H/O CHD, chest pain or discomfort, associated symptoms, RF, **risk of bleeding**
2. Abbreviated physical exam:
 - Onset of symptoms
 - Signs and symptoms
3. **12-lead ECG (or EKG) within 10 minutes of presentation**
 - **What are we looking for on the EKG?**



Initial Management in ED: Evaluation and Physical Exam

Patient Evaluation or Triage:

4. Labs:

- Troponin (cardiac biomarkers)
- CBC: WBC, H/H, Plt count
- Chemistry panel: BUN/Scr, K+, Mg++, BS, A1c
- Fasting lipid panel
- Urine toxicology screen
- Stool guaiac

5. Vital Signs: HR, BP, RR, Temp, Pulse Oximetry



Useful Reference Labs – UW Health



- Sodium 136-145
- Potassium 3.5-5
- BUN 9-20
- Scr 0.7-1.2
- Glu (fasting) 70-99
- Mg 1.6-2.6
- Hemoglobin (M) 13.6-17.2
- Hemoglobin (F) 11.6-15.6
- HCT (M) 40-52
- HCT (F) 34-46
- Plt count 160-370 K/uL



Making a Diagnosis: Three Types of ACS

- 1. STEMI:** An elevated ST-segment with increased serum levels of cardiac biomarkers (cardiac Troponin I or T) are consistent with a diagnosis of ST-elevation myocardial infarction.
- 2. NSTEMI:** increased serum levels of cardiac biomarkers but no ST-segment elevation are said to have non-ST-elevation myocardial infarction.
- 3. UA:** Normal levels of cardiac biomarkers and an absence of ST-segment elevation are consistent with a diagnosis of unstable angina.



Pathology of UA/NSTEMI

- UA or NSTEMI commonly result from disruption of an atherosclerotic plaque and formation of a thrombus (also called the culprit lesion)
 - Clot consists of:
 - Clot produces:
- Other causes: vasospasm, post PCI restenosis



NSTE ACS Guidelines 2014



Presentation of UA/NSTEMI

- Angina at rest usually > 10 minutes or new onset of angina with minimal exertion or rapidly increasing angina
- Chest pain/pressure is more severe and prolonged than typical angina exacerbation
 - No response to SL nitroglycerin
- **EKG changes:**
- **Cardiac biomarkers required to differentiate between these two types of ACS:**
 - Little to no release =
 - Release of biomarkers =



Pathology of STEMI

- Thrombus formation over ruptured plaque:
 - Clot consists of:
 - Clot produces:
- Other causes
 - Coronary vasospasm (ex. Prinzmetal, cocaine)





Presentation of STEMI

- Similar presentation to UA/NSTEMI
- ECG changes:
- Cardiac biomarkers:
- Highest risk of mortality!



STEMI versus NSTEMI

STEMI

- Clot composed mostly of fibrin
- Total occlusion of culprit lesion in one artery common
- Complete infarction through ventricular wall
- Complete infarction that may produce characteristic Q wave on ECG

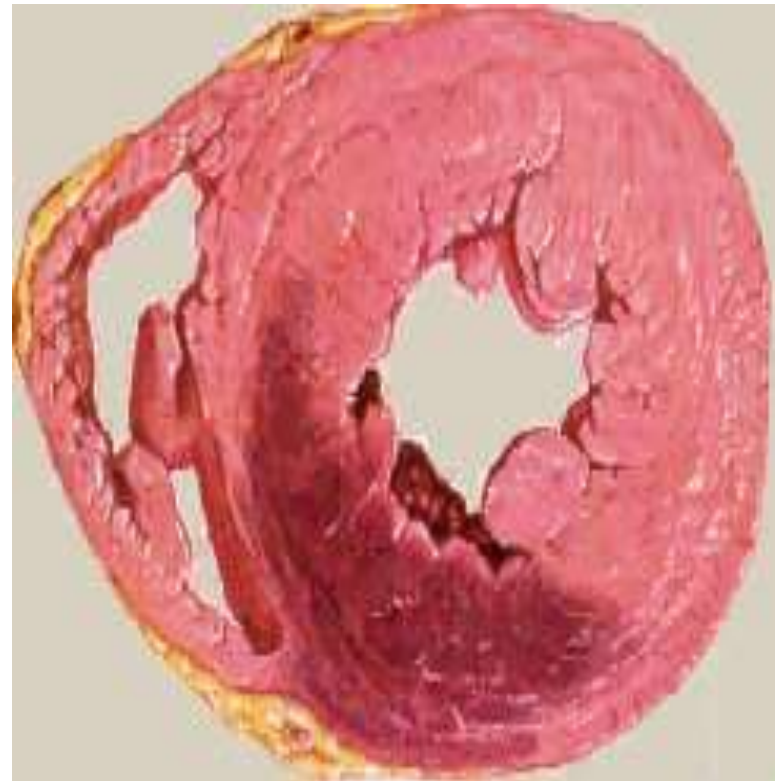
NSTEMI

- Clot composed mostly of platelets
- Incomplete occlusion of culprit lesion
- Incomplete infarction through ventricular wall
- Incomplete infarction that may not produce Q wave on ECG



Test Your Knowledge

- What type of ACS is this?
- Describe the clot that caused this damage to the myocardium.
- Were cardiac biomarkers released in this patient?
- Describe the EKG findings associated with this type of ACS.





Test Your Knowledge

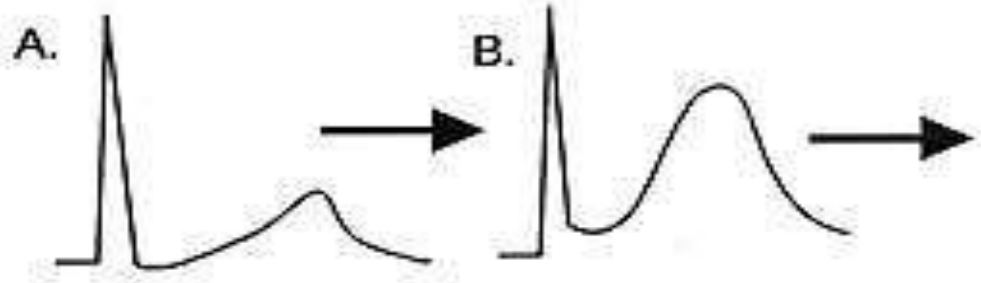
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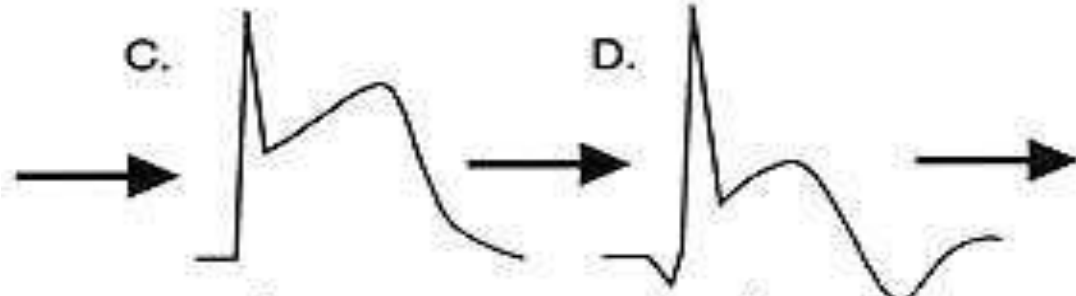


EKG: Defining the Zones of MI

- Ischemia = zone of **hypoxic myocardial tissue that may recover** if circulation and oxygen supply restored
 - ST-segment depression or no change with no release of cardiac biomarkers
- Injury = zone of **infarcting tissue where myocardial cell death is occurring** due to continued ischemia
 - ST-segment changes with release of cardiac biomarkers
- Infarction = zone of **necrotic nonviable tissue**
 - prominent Q-wave

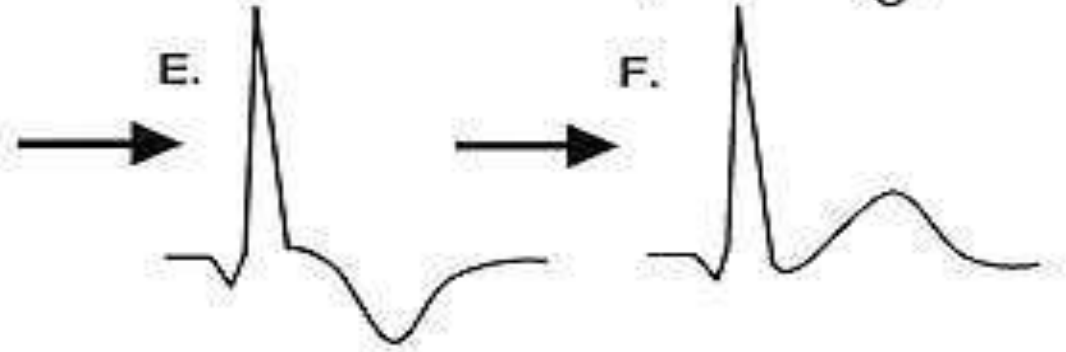


**First
several
hours**



**Within
first day**

Day 1 - 3

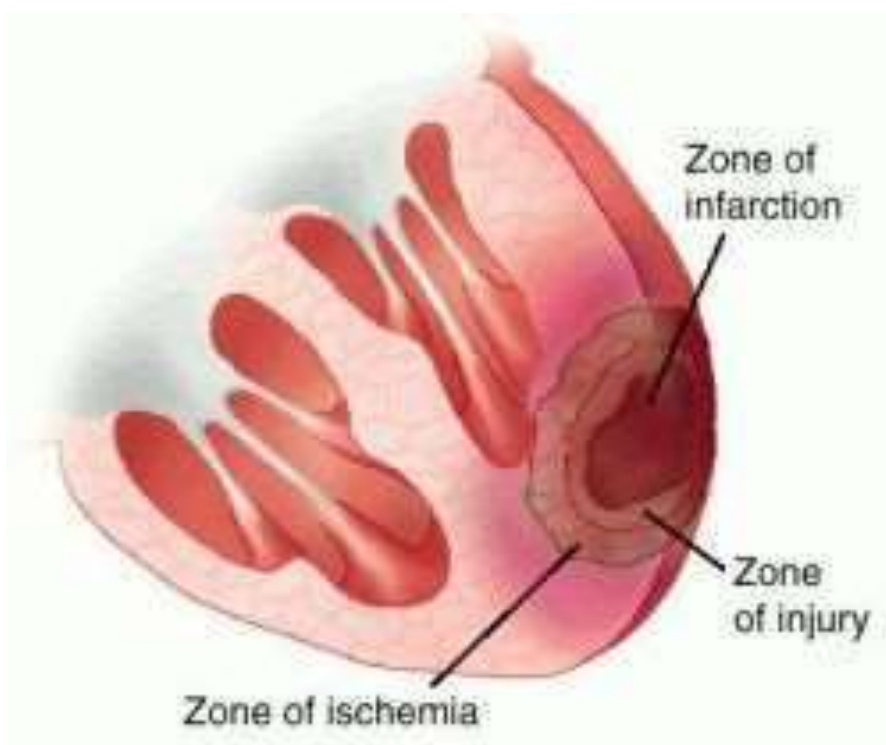


**Several
weeks or
months**

Evolution of Acute MI



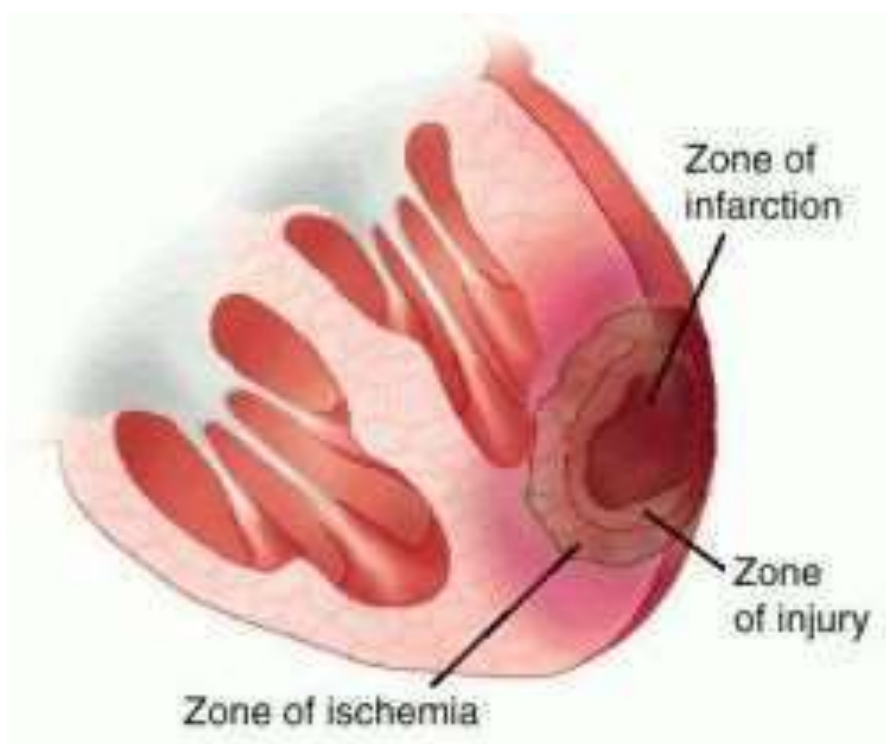
Test Your Knowledge



- Which zone represents salvageable myocardial tissue?
- Which zone represents tissue that is releasing biomarkers?
- Which zone represents necrotic tissue that may produce a Q wave on the EKG?



Test Your Knowledge

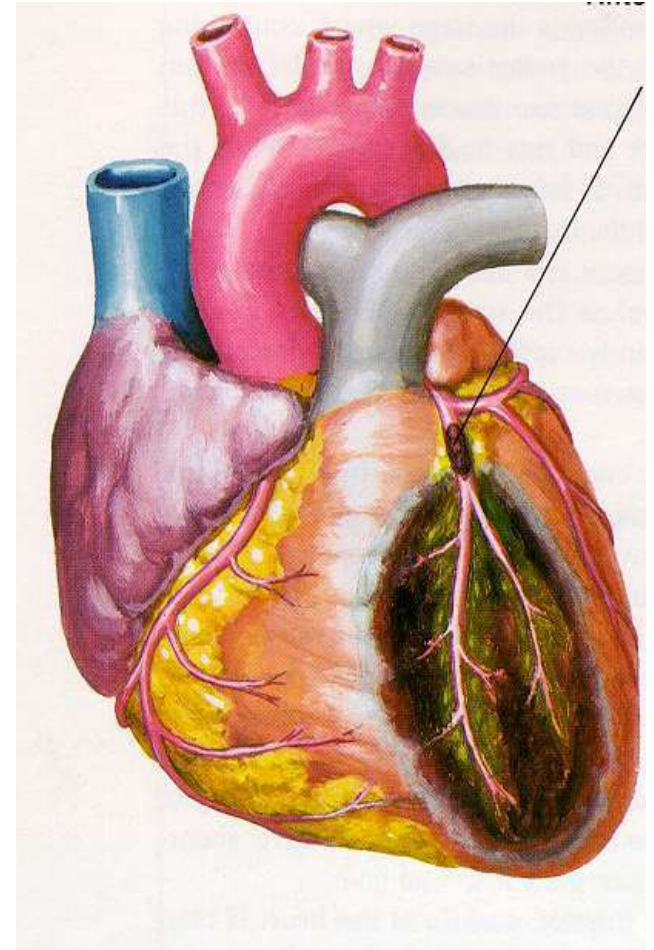


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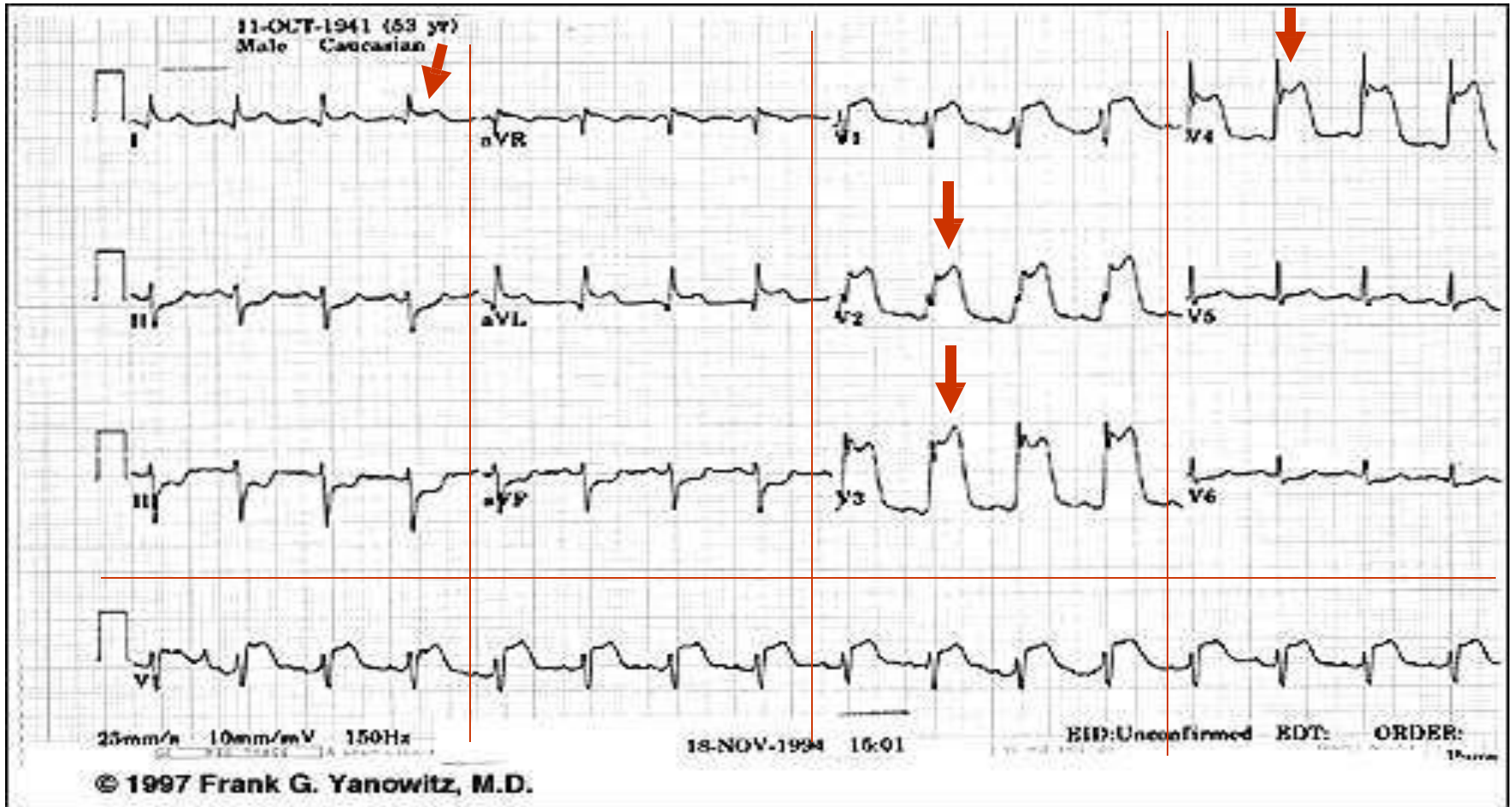
Anterior Wall MI

- Involves anterior wall of LV
- Represents occlusion in:
 - ST changes in leads 1, V_2-V_4





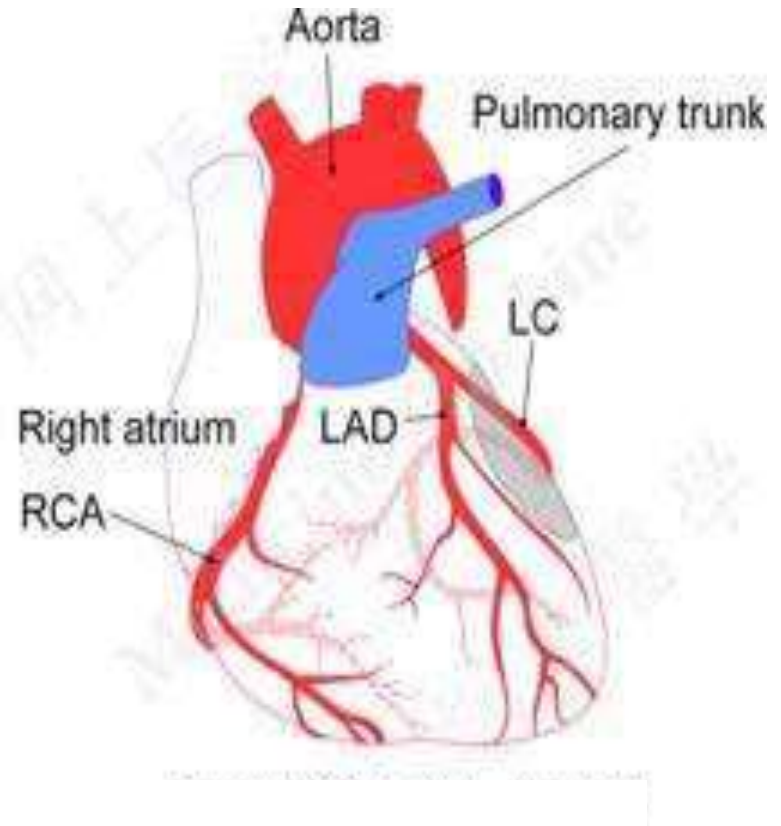
12-Lead EKG for AWTMI





Lateral Wall MI

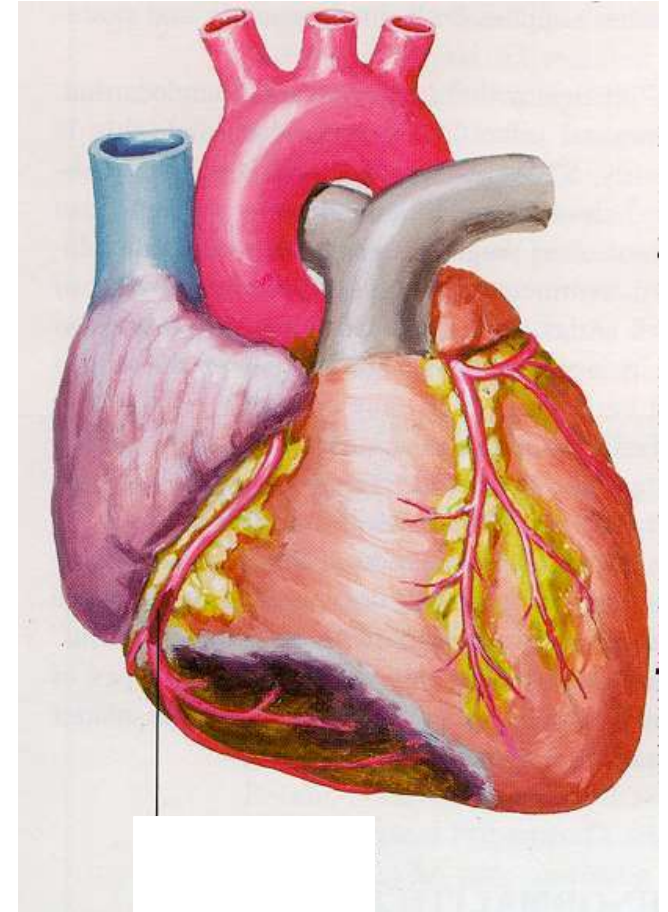
- Involves lateral wall of LV
- Represents occlusion in:
- ST changes in leads I, aVL, V5-V6.





Inferior Wall MI

- Involves inferior wall and base of LV, may also involve RV
- **Represents occlusion in:**
- ST changes in leads II, III, aVF, V6.

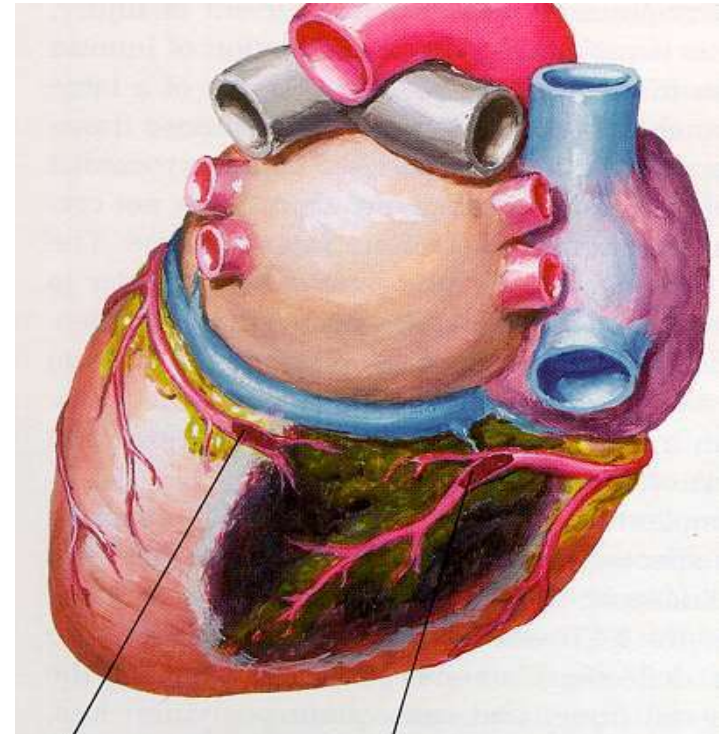




Posterior Wall MI- RARE!

(will not test on this type of ACS)

- Involves posterior wall of LV
- Represents occlusion in:
- ST changes (STEMI = ST segment depression) in V1 with large R wave.

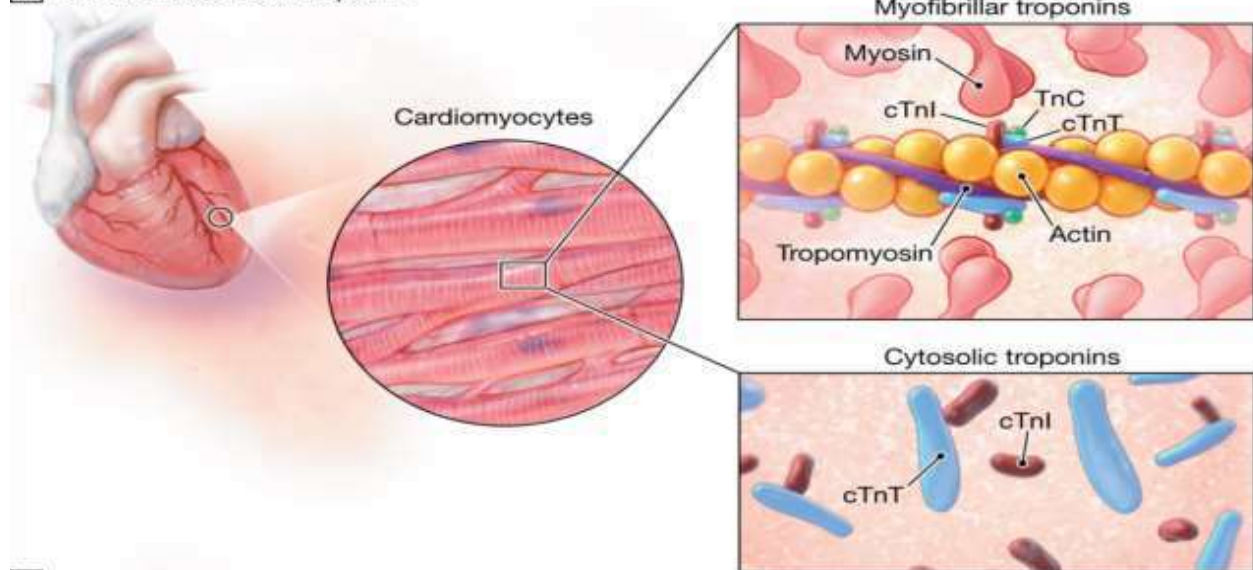




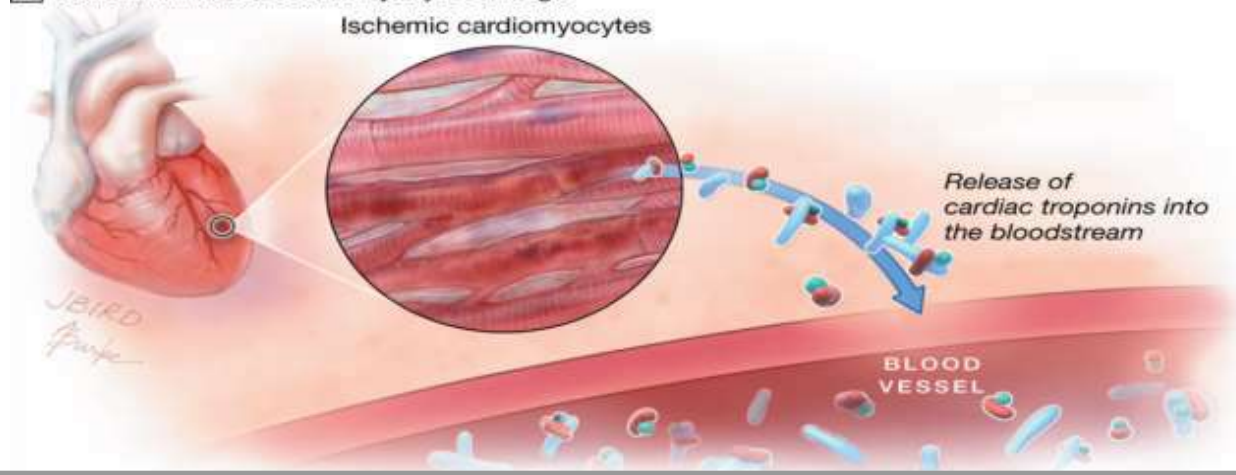
Cardiac Biomarkers

- Biomarkers are released into the bloodstream by necrotic myocardial cells
 - Myoglobin, creatine kinase (CK), CK-MB, CK-MB isoforms, and **troponin T and I**
- Levels measured at presentation and 3-6 hours after symptom onset, may be measured serially until peak level reached
- **Cardiac markers are needed to:**
 - **Confirm diagnosis of MI without ST-segment elevation**
 - **Differentiate between UA and NSTEMI**
 - **Predict prognosis of STEMI**
 - **Determine success of reperfusion therapy**

A Structure of cardiac troponins



B Ischemia-induced cardiomyocyte damage

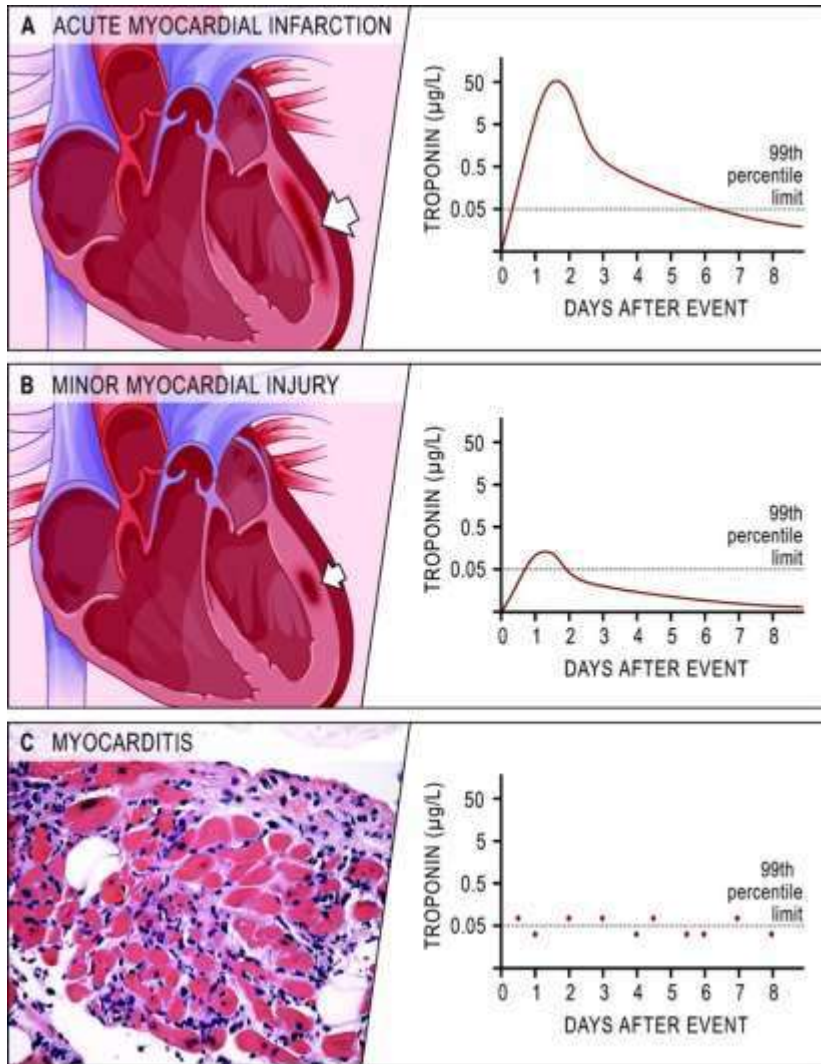


- Ischemia alters cell membrane integrity causing release of cytoplasmic troponin into the bloodstream
- Sarcolemmal membrane breaks down in necrotic cardiomyocytes to further release troponin
- Released cTnT and cTnI can be measured by commercial assays





Troponin release after myocardial injury



- cTnT and cTnI levels are considered “positive” when levels are above the 99th percentile limit of normal.
- Different commercial assays available so each institution will have its own cutoff value.
- **UWHC values for cTnI:**
Negative: 0-0.03 ng/mL
Suspicious for injury:
 ≥ 0.04 ng/mL

Melanson S E et al. Circulation 2007;116:e501-e504

Other Conditions Associated with Elevated Troponin Levels



- Acute or chronic renal dysfunction
- HF
- Tachy- and brady-arrhythmias
- PE
- Stroke
- Apical ballooning syndrome (Takotsubo cardiomyopathy)
- Post PCI or CABG
- Sepsis
- Trauma, surgery
- Myo- and peri-carditis



Treatment of ACS



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Initial Management in ED: Immediate General Treatment

Indicated for all ACS patients:

1. ASA 325mg chewed and swallowed ASAP (ASA allergy?)
2. IV NTG
3. Oxygen 2-4 L/min if patient hypoxic ($POx < 90\%$), has signs of HF, or c/o SOB (Target $POx \geq 94\%$)
4. IV Morphine for severe chest pain despite IV NTG (avoid if possible)
5. Anti-coagulant to keep thrombus from growing and prevent further platelet activation and aggregation



MONA₂



Treatment of ACS



1. Management of ACS S/Sx
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- 3. Acute therapies during hospitalization**
 - A. Non-pharmacologic Management**
 - B. Pharmacologic Management
4. Chronic therapies after discharge



Goals of Acute Therapies

- Early restoration of blood flow to minimize infarct size and salvage ischemic myocardium (MI) or prevent complete occlusion that can lead to an MI (UA)
- Prevent coronary artery re-occlusion
- Prevent or minimize complications
- Improve morbidity and mortality
- Relieve chest pain/discomfort

Non-pharmacologic Management of ACS



- Serial cardiac markers (until troponin level peaks)
- Continuous 3-lead ECG rhythm strip (telemetry)
- 12-lead ECG every 6-8hrs for 24 hours
- Bed rest 12-24 hours
- ICU care for 24 hours in patients with continued angina, hemodynamic instability, arrhythmias, or large MI
- Hospitalization for 2-3 days

Non-pharmacologic Management of ACS



- Labs
 - CBC: WBC, H/H, plt count
 - Chem panel: BUN/Scr, K+, Mg⁺⁺
 - FLP: LDL, UFH affects TG level

- ECHO prior to discharge

Thank-you!

