Acute Coronary Syndrome

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PGY-6 Cardiology Fellow

AHD – Acute Coronary Syndrome Objectives

- 1. Describe the pathophysiology of STEMI and NSTEMI. Specifically describe the difference between type 1 and type 2 MI
- 2. Describe the TIMI and GRACE score for NSTEMI
- 3. Know the appropriate management of STEMI and NSTEMI based on ACC/AHA guidelines
- 4. Describe the abnormal values for high-sensitivity troponin assays and how to use the new assay in the evaluation of a patient with chest pain who rules out, rules in and is in the indeterminant range for this biomarker

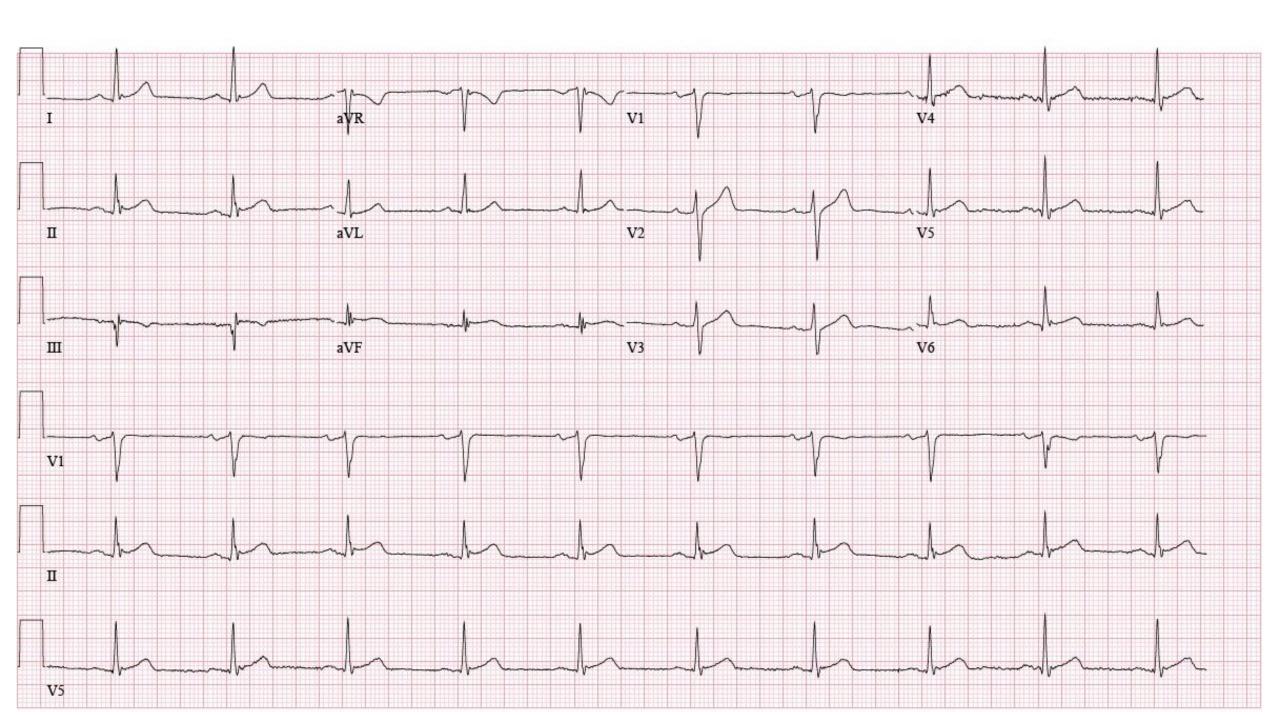
Case #1 – July 9, 2019 @ 17:00

65 yo male with PMHx HTN, HLP and hx Tobacco abuse who presented 60 minutes after onset of chest pain. 8/10 substernal chest heaviness with radiation to neck while mowing the lawn. Symptoms spontaneously resolved with rest. Associated dizziness, nausea and diaphoresis. Chest pain free in ER.

VS: BP 144/88, HR 60, 97% RA

EKG: to follow

Labs: HS Troponin 44, BNP <10, Cr 0.98, Hb 15.3, Plts 209, INR 1.0



ER First Call:

- 65 year old male PMHx HTN, HLP, Hx Tobacco use presented after chest pain with mowing. Chest pain free in ER. -VS stable -EKG no ischemia -Labs: HS Troponin 44
- A. NSTE-ACS Start ACS protocol, await repeat HS troponin
- B. STEMI Activate cardiac cath lab
- C. Recommend Observation admission, no ACS protocol
- D. DC home Chest pain free, troponin indeterminate, EKG normal

Case #1 – July 9, 2019 @ 19:00

Web page –

Dr. Intern, repeat HS Troponin 330. What orders?

BP 130/84, HR 64

Patient remains chest pain free, asking for dinner, wants to know when he can go home to let his dog out?

Case #1

65 year old male PMHx HTN, HLP, Hx Tobacco use presented after chest pain with mowing. Chest pain free in ER. -VS stable -EKG no ischemia -Labs: HS Troponin 44 -> 330

- A. NSTE-ACS ACS protocol with immediate invasive strategy
- B. NSTE-ACS ACS protocol with early invasive vs ischemia-guided strategy
- C. STEMI Activate cardiac cath lab
- D. Continue Observation admission, consult cardiology in AM
- E. DC home Patient has to let his dog out

Appropriately started ACS protocol...what is initial ACS protocol?

- A. ASA 81mg , GpIIb/IIIa inhibitor, full dose AC, pravastatin, amlodipine
- B. ASA 324mg, clopidogrel 600mg, full dose AC, HI statin, metoprolol
- C. ASA 324mg, clopidogrel 75mg, DVT prophy dose AC, No statin
- D. ASA 324mg, clopidogrel 600mg, GPIIb/IIIa inhibitor, full dose AC, type and cross with 2u pRBC on hold

NSTE-ACS Therapy

- Initial treatment MONA**
- ABCs:
 - Aspirin, Anti-platelet, Anti-thrombotic, Anti-anginal, ACEi/ARB
 - Beta-blocker
 - Cholesterol (statin)

Anti-thrombotic therapy

Agent	Mechanism	Pro	Con
UFH	Inhibits Xa and thrombin (via ATIII)	Easy to assess effect, quick on/quick off	Variable response, HIT, lab draws
LMWH	Inhibits XA and thrombin (via ATIII)	Ease of use, less platelet activation	Measuring effect, HIT
Bivalirudin	Direct thrombin inhibitor	Easy to assess affect, short half life, no HIT	ONLY FOR INVASIVE APPROACH
Fondaparinaux	Indirect Xa inhibition	Once daily	Once daily, only for conservative tx

Anti-platelet

- Aspirin: 325 mg load, 81 mg daily
- P2Y₁₂ Inhibitors:
 - Thienopyridines (indirect inhibitors):
 - *Clopidogrel (Plavix)*: 300 or 600 mg load, 75 mg daily
 - Prasugrel (Effient): 60 mg load, 10 mg daily
 - Direct inhibitors:
 - Ticagrelor (Brilinta): 180 mg load, 90 mg BID
 - Cangrelor (Kengreal)

Anti-anginal

- Nitroglycerin
 - No mortality benefit
 - Mechanism: selective coronary vasodilation
 - CAUTION: Decrease pre-load
 - Do not use in pre-load dependent RV infarct
 - Careful if severe AS

Medical Therapy

Therapy	Indications	Dose/Administration	Avoid/Caution
Beta-Receptor Antagonists	 Oral: All patients without contraindication IV: Patients with refractory hypertension or ongoing ischemia without contraindication 	 Individualize: Metoprolol tartrate 25 to 50 mg every 6 to 12 h orally, then transition over next 2 to 3 d to twice-daily dosing of metoprolol tartrate or to daily metoprolol succinate; titrate to daily dose of 200 mg as tolerated Carvedilol 6.25 mg twice daily, titrate to 25 mg twice daily as tolerated Metoprolol tartrate IV 5 mg every 5 min as tolerated up to 3 doses; titrate to heart rate and BP 	 Signs of HF Low output state Increased risk of cardiogenic shock Prolonged first-degree or high-grade AV block Reactive airways disease

ACE Inhibitors

ARB

Statins

- For patients with anterior infarction, post-MI LV systolic dysfunction (EF ≤0.40) or HF
- May be given routinely to all patients without contraindication

- For patients intolerant of ACE inhibitors
- All patients without contraindications

Individualize:

- Lisinopril 2.5 to 5 mg/d to start; titrate to 10 mg/d or higher as tolerated
- Captopril 6.25 to 12.5 mg 3 times/d to start; titrate to 25 to 50 mg 3 times/d as tolerated
- Ramipril 2.5 mg twice daily to start; titrate to 5 mg twice daily as tolerated
- Trandolapril test dose 0.5 mg; titrate up to 4 mg daily as tolerated
- Valsartan 20 mg twice daily to start; titrate to 160 mg twice daily as tolerated
- High-dose atorvastatin 80 mg daily

- Hypotension
- Renal failure
- Hyperkalemia

- Hypotension
- Renal failure
- Hyperkalemia
- Caution with drugs metabolized via *CYP3A4*, fibrates
- Monitor for myopathy, hepatic toxicity
- Combine with diet and lifestyle therapies
- Adjust dose as dictated by targets for LDL cholesterol and non–HDL cholesterol reduction

NSTEMI Summary

- Serial ECG and cardiac biomarkers
- ABCs
 - ASA 325mg then 81mg QD
 - P2Y12 Inhibitor
 - Clopidogrel 600mg or 300mg then 75mg QD
 - Ticagrelor 180mg then 90mg BID
 - Anti-thrombotic (UFH)
 - Anti-anginal (SL NTG or NTG drip)
 - Beta-blocker (PO Metoprolol)
 - Cholesterol (High intensity Rosuvastatin or Atorvastatin)

P2Y₁₂ Inhibitors

Table 1. P2Y₁₂ Inhibitors Currently in Clinical Use After Percutaneous Coronary Intervention

	Ticlopidine	Clopidogrel	Prasugrel	Ticagrelor
Class	Thienopyridine	Thienopyridine	Thienopyridine	Cyclopentyl-triazolo- pyrimidine
Pharmacology	Highly CYP- dependent conver- sion to prodrug	Highly CYP- dependent conver- sion to prodrug	Requires conversion to prodrug (less CYP dependent)	Directly acting inhibitor
Potency of platelet inhibition	+	+	++	++
Time to peak platelet inhibition ²⁴	3-4 d	4-5 h (300 mg) 2-3 h (600 mg)	2-4 h	2-4 h
Dosing, daily	Twice	Once	Once	Twice
Time required for anti- platelet effect to dissi- pate, days	5	5	7	5
Cost for 1 mo, \$	≈45.00ª	14.50 (generic) ^a 218.87 (Plavix) ^b	218.52 ^b	260.78 ^b

Case #1 Recap: July 9, 2019 @ 21:00, 12E

65 year old male PMHx HTN, HLP, Hx Tobacco use presented after chest pain with mowing. Remains chest pain free.

- BP, HR stable.
- EKG (initial and repeat) no ischemia
- Labs: HS Troponin 44 -> 330
- Started on ACS therapy including:
 - ASA 324mg
 - Clopidogrel 600mg
 - Heparin drip (bolus + infusion)

- Atorvastatin 80mg
- Metoprolol 25mg BID

What's next?

- 65 year old male PMHx HTN, HLP, Hx Tobacco use presented after chest pain with mowing. Chest pain free in ER. -VS stable -EKG no ischemia -Labs: HS Troponin 44
- A. Ischemia driven strategy Order Stress test for in the morning
- B. Early invasive strategy NPO at midnight for coronary angiogram in the morning
- C. Delayed invasive strategy Monitor clinically, coronary angiogram within 72hrs
- D. Medical management continue heparin drip for 48 hours and discharge home on NSTEMI therapy

Early Invasive and Ischemia: Guided Strategies

Recommendations	COR	LOE
An urgent/immediate invasive strategy (diagnostic angiography with intent to perform revascularization if appropriate based on coronary anatomy) is indicated in patients (men and women) with NSTE-ACS who have refractory angina or hemodynamic or electrical instability (without serious comorbidities or contraindications to such procedures).		A
An early invasive strategy (diagnostic angiography with intent to perform revascularization if appropriate based on coronary anatomy) is indicated in initially stabilized patients with NSTE-ACS (without serious comorbidities or contraindications to such procedures) who have an elevated risk for clinical events .		В

Early Invasive and Ischemia: Guided Strategies (cont'd)

Recommendations	COR	LOE
It is reasonable to choose an early invasive strategy (within 24 hours of admission) over a delayed invasive strategy (within 25 to 72 hours) for initially stabilized high-risk patients with NSTE-ACS. For those not at high/intermediate risk, a delayed invasive approach is reasonable.	lla	В
In initially stabilized patients, an ischemia-guided strategy may be considered for patients with NSTE-ACS (without serious comorbidities or contraindications to this approach) who have an elevated risk for clinical events.	llb	В
The decision to implement an ischemia-guided strategy in initially stabilized patients (without serious comorbidities or contraindications to this approach) may be reasonable after considering clinician and patient preference .	llb	С

Factors Associated With Appropriate Selection of Early Invasive Strategy or Ischemia-Guided Strategy in Patients With NSTE-ACS

Immediate	Refractory angina
invasive	Signs or symptoms of HF or new or worsening mitral regurgitation
(within 2 h)	Hemodynamic instability
	Recurrent angina or ischemia at rest or with low-level activities despite intensive medical
	therapy
	Sustained VT or VF
Ischemia-	Low-risk score (e.g., TIMI [0 or 1], GRACE [<109])
guided	Low-risk Tn-negative
strategy	Patient or clinician preference in the absence of high-risk features
Early invasive	GRACE risk score >140
(within 24 h)	Temporal change in Tn
	New or presumably new ST depression
Delayed	None of the above but diabetes mellitus
invasive	Renal insufficiency (GFR <60 mL/min/1.73 m ²)
(within 25–72	Reduced LV systolic function (EF < 0.40)
h)	Early postinfarction angina
	PCI within 6 mo
	Prior CABG
	GRACE risk score 109–140; TIMI score ≥2

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Basics...Start from the beginning:

- Clinical story
- Physical exam
- Risk factors / Risk scores
- Cardiac biomarkers
- ECG
- Imaging (Echo)

Diagnosis – Clinical Story

- Anginal chest pain
 - 1. Substernal
 - 2. Brought on by exertion or emotional stress
 - 3. Relieved by rest or nitroglycerin*



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ARTICLES | 16 DECEMBER 2003

Chest Pain Relief by Nitroglycerin Does Not Predict Active Coronary Artery Disease

Charles A. Henrikson, MD, MPH; Eric E. Howell, MD; David E. Bush, MD; J. Shawn Miles, MD; Glenn R. Meininger, MD; Tracy Friedlander; Andrew C. Bushnell, MD; Nisha Chandra-Strobos, MD

Results:

- Nitroglycerin relieved chest pain in 39% of patients (181/459) admitted through the ED who received nitro from EMS or ER staff
- 35% had chest pain relief with nitro in patients with active coronary artery disease as cause of chest pain
- 41% had chest pain relief with nitro in patients without active coronary artery disease as cause of chest pain

Conclusion: In a general population admitted for chest pain, relief of pain after nitro treatment does not predict active coronary artery disease and should not be used to guide diagnosis.

Diagnosis – Clinical Story

- Anginal chest pain
 - 1. Substernal
 - 2. Brought on by exertion or emotional stress
 - 3. Relieved by rest or nitroglycerin*
- Typical Angina meets all 3 criteria
- Atypical Angina meets 2 of 3 criteria
- Non-anginal CP meets 0-1 of 3 criteria

Age (year)	chest pain Atypical angina		Typical angina			
Age (year)	Men	Women	Men	Women	Men	Women
35	3-35	1-19	8-59	2-39	30-88	10-78
45	9-47	2-22	21-70	5-43	51-92	20-79
55	23-59	4-21	45-79	10-47	80-95	38-82
65	49-69	9-29	71-86	20-51	93-97	56-84

Comparing pretest likelihood of CAD in low-risk symptomatic patients with high-risk symptomatic patients (Duke Database)

Each value represents the percentage with significant CAD. The lowest (first) value of each range is the likelihood of CAD for a low-risk patient without diabetes mellitus, smoking, or hyperlipidemia. The highest (second) value of each range is the likelihood of CAD for a high-risk patient of the same age with diabetes mellitus, smoking, and hyperlipidemia. Both high- and low-risk patients have normal resting ECGs. If ST-T-wave changes or Q waves had been present, the likelihood of CAD would be higher in each entry of the table. This information was included in the 2012 ACCF/AHA/ACP/AATS/PCNA/SCAI/STS Guideline for the Diagnosis and Management of Patients With Stable Ischemic Heart Disease.^[1]

Physical Exam

- CAN BE NORMAL
- VS: BP in both arms (dissection)
- Signs of LV dysfunction: Rales, S3 gallop
- S4, Murmur, Rub
- Chest wall tenderness
- Positional pain

Risk Factors

- Hypertension
- Diabetes Mellitus
- Hyperlipidemia
- Tobacco abuse

- Obesity
- Family Hx premature CAD
- Personal Hx CAD
- Age



Must be applied to correct patient – Do not use on patient without ACS Used to predict adverse events based on observational data

- TIMI
- GRACE
- HEART

TIMI Risk Score – NSTEMI/UA

÷	TIMI Score	for UA/NSTEMI	
CALCULATOR	NEXT STEPS	EVIDENCE	CREATOF
Estimates mortality for patie	nts with unstable angina	and non-ST elevation MI.	
When to Use $oldsymbol{ u}$	P	Pearls/Pitfalls ∨	Why Use 🗸
Age ≥65		No 0	Yes +1
≥ 3 CAD risk factors Hypertension, hypercholestero family history of CAD, or currer	olemia, diabetes, ht smoker	No 0	Yes +1
Known CAD (stenosis ≥50%)		No 0	Yes +1
ASA use in past 7 days		No 0	Yes +1
Severe angina (≥2 episodes i	in 24 hrs)	No 0	Yes +1
EKG ST changes ≥0.5mm		No 0	Yes +1
Positive cardiac marker		No 0	Yes +1

Risk Category	TIMI Risk Score	All-Cause Mortality, New or Recurrent MI, or Severe Recurrent Ischemia Through 14 d After Randomization, %
Loui	0–1	4.7
Low	2	8.3
lusto uno o dio to	3	13.2
Intermediate	4	19.9
Llich	5	26.2
High	6–7	40.9

Risk Score - GRACE

In-hospital risk of death/MI

Risk Category	GRACE risk score	In-hospital death %
Low	<108	<1
Intermediate	109-140	1-3
High	>140	>3

ad 🗢	9:18 PM GRACE ACS Score		
CALCULATOR	NEXT STEPS EVIDENCE		CREATOR Why Use 🗸
When to Use 🗸	Реа	Pearls/Pittalls 🗸	
Age			0 years
Heart rate/pulse			0 beats/min
Systolic BP			0 mm Hg
Creatinine			0 mg/dL 🗲
Cardiac arrest at admission		No	Yes
ST segment deviation on EKG?		No	Yes
Abnormal cardiac enzymes		No	Yes
Killip class (signs/symptoms)	No CHF		
	Rales an	d/or JVD	
	Pulmona	ary edema	

Cardiogenic shock

iPad 🗢	9:18 PM		* 7% 💭
÷	HEART Score		*
CALCULATOR	NEXT STEPS	EVIDENCE	CREATOR
History	Slightly s	uspicious	0
	Moderate	ly suspicious	+1
	Highly su	spicious	+2

EKG

1 point: No ST depression but LBBB, LVH, repolarization changes (ex: digoxin); 2 points: ST depression/elevation not due to LBBB, LVH, or digoxin

Normal	0
Non-specific repolarization disturbance	+1
Significant ST depression	+2

Age

<45	
45-64	+1
≥65	+2

Risk factors

Risk factors: HTN, hypercholesterolemia, DM, obesity (BMI >30 kg/m²), smoking (current, or smoking cessation ≤3 mo), positive family history (parent or sibling with CVD before age 65); atherosclerotic disease: prior MI, PCI/ CABG, CVA/TIA, or peripheral arterial disease

No known risk factors	
1-2 risk factors	+1
≥3 risk factors or history of atherosclerotic disease	+2

Initial troponin

Use local assays and corresponding cutoffs

≤normal lin	nit	0
1–3× norm	al limit	+1

 \wedge

RESULT O points Low Score

Risk Score - H	HEART
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HEART Risk Score	Risk of adverse cardiac event defined as all-cause mortality, MI or coronary revascularization in 6 weeks %
0-3	0.9 – 1.7
4-6	12 – 16.6
⇒7	50 – 65

Killip Class

Classification that categorizes patients with an acute MI based upon presence of absence of physical exam findings that suggest LV dysfunction and heart failure.

Class I	No evidence of heart failure
Class II	Findings consistent with mild to moderate HF
Class III	Overt pulmonary edema
Class IV	Cardiogenic shock

Revisit Case #1

65 year old male PMHx HTN, HLP, Hx Tobacco use presented after chest pain with mowing. Chest pain free in ER. -SBP 144, HR 60, Cr 0.98 -EKG no ischemia -Labs: HS Troponin 44 -> 330

How would you risk stratify this patient?

TIMI RISK

GRACE SCORE

Revisit Case #1

65 year old male PMHx HTN, HLP, Hx Tobacco use presented after chest pain with mowing. Chest pain free in ER. -SBP 144, HR 60, Cr 0.98 -EKG no ischemia -Labs: HS Troponin 44 -> 330

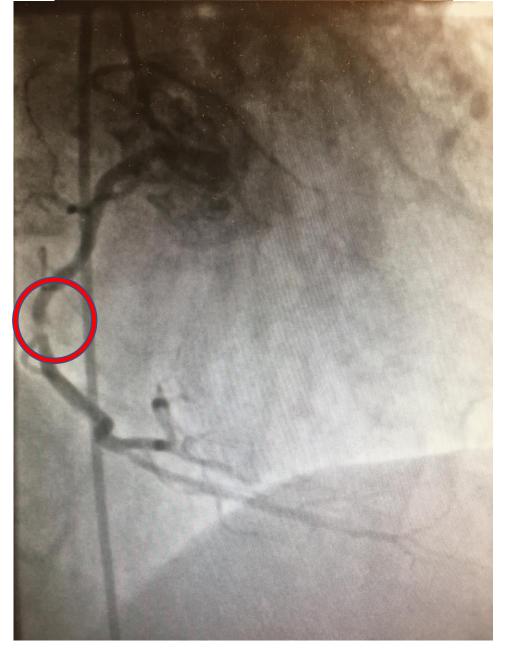
How would you risk stratify this patient? <u>TIMI RISK</u> = 4 <u>GRACE SCORE</u> = 92

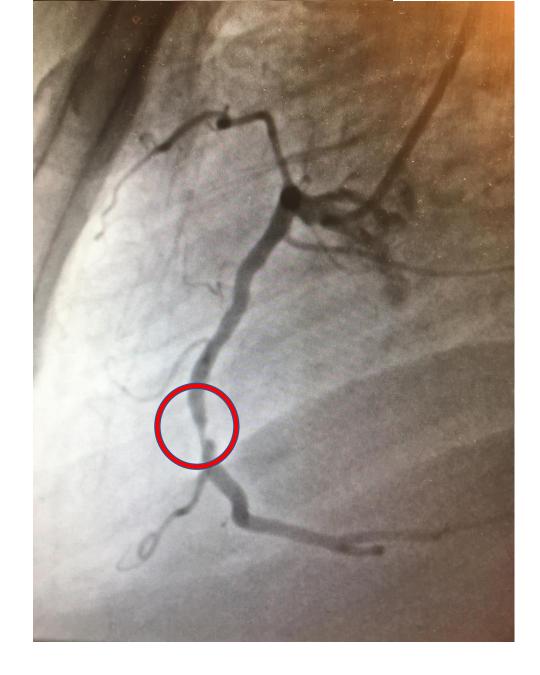
- A. Low Risk
- B. Intermediate Risk
- C. High Risk

Case #1 – Next Day

Patient remained chest pain free overnight BP and HR remain within normal limits Peak HS Troponin 1,200

Cardiology consulted, taken to cath lab (early invasive)





Case #2

72 year old male with PMHx tobacco abuse and DM2 admitted to Phoenix VA with stuttering, substernal chest pressure. Denies radiation of pain or associated dyspnea, palpitations, nausea or diaphoresis. VS: BP 134/84, HR 74, 98% RA Initial EKG: nonspecific inferior T wave changes Conventional troponin normal in ER

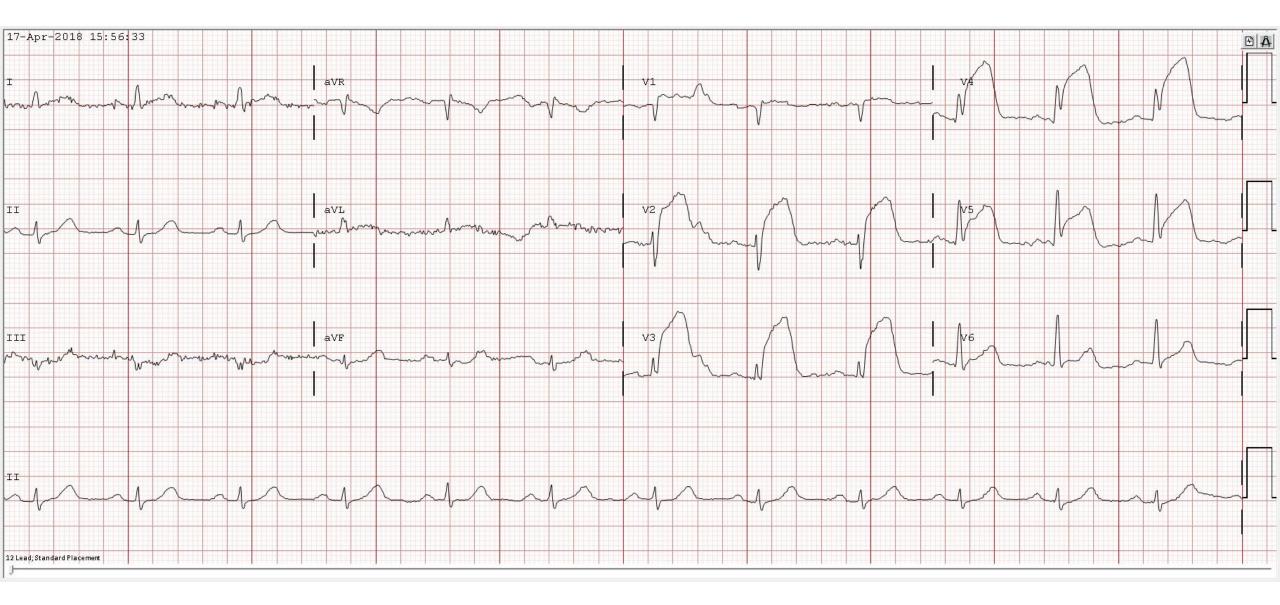
Admit to 4C telemetry ward for ACS rule out



Receive page from RN Gloria on 4C that patient has developed crushing substernal chest pain and diaphoretic.

VS: BP 98/56, HR 80, 100% 2L NC

Repeat EKG to follow



Case #2: What do you do?

- A. ACS protocol This represents NSTE-ACS
- B. STEMI Activate cardiac cath lab (No STEMI coverage at VA)
- C. STEMI Transfer to PCI capable facility
- D. STEMI Initiate thrombolytics
- E. Hope your senior resident was awake during this lecture last year

Case #2

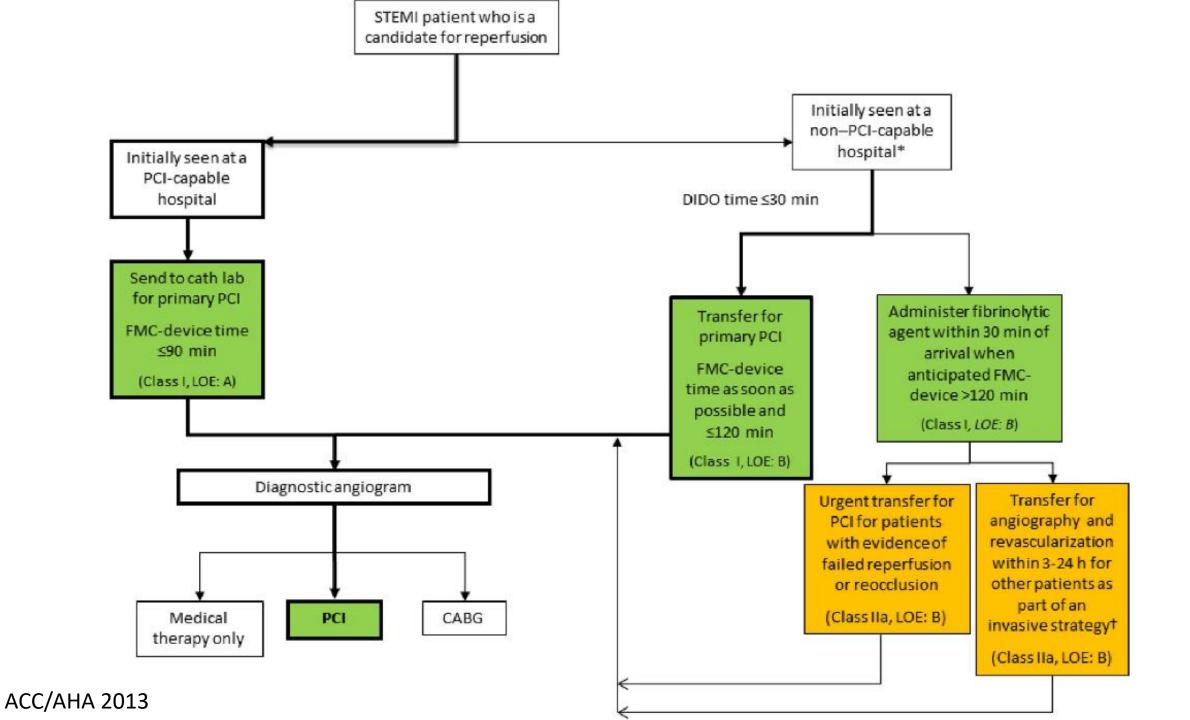
Which of the following combinations represents correct guideline recommended times for STEMI reperfusion therapy?

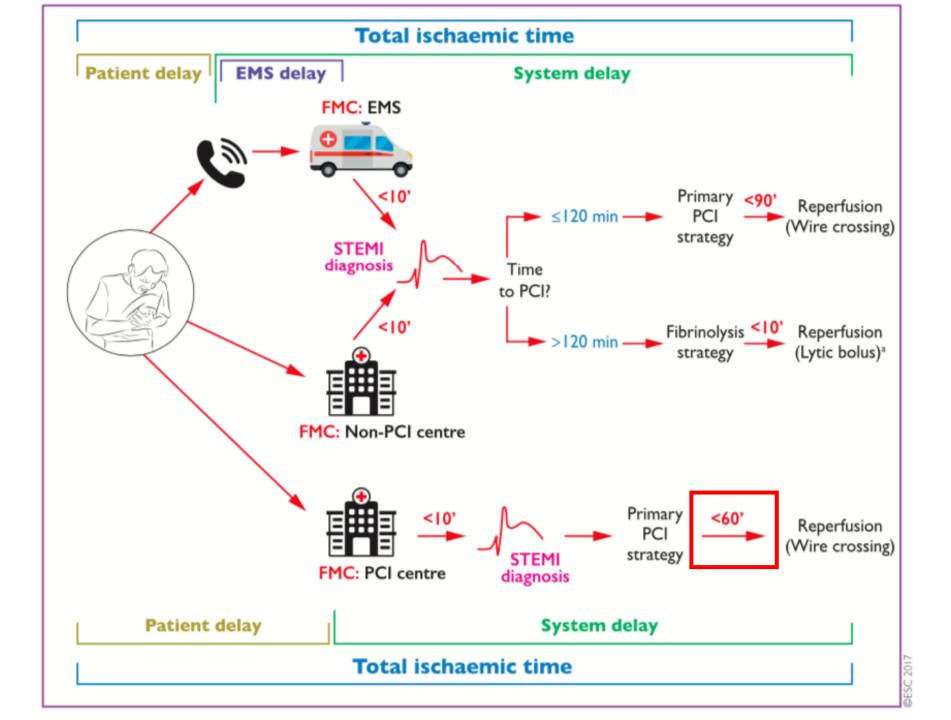
	PCI Capable Center FMC to Device (Banner)	PCI Non-Capable Center FMC to Device	PCI Non-Capable Center FMC to Lytic
		(Phoenix VA)	(Prescott VA)
A	90 min	180 min	30 min
В	60 min	120 min	30 min
С	90 min	120 min	30 min
D	60 min	90 min	30 min

Case #2

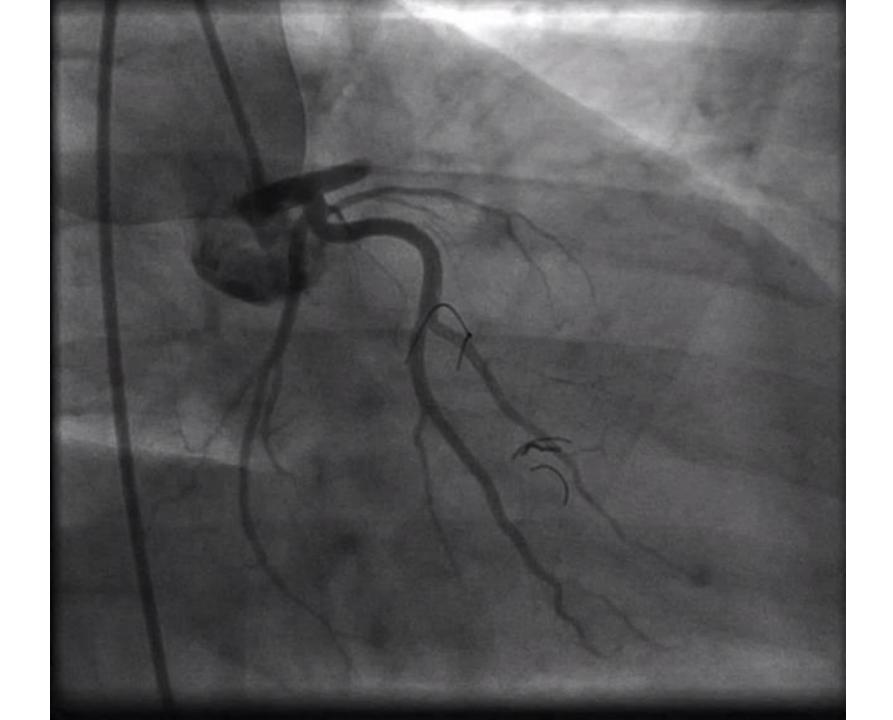
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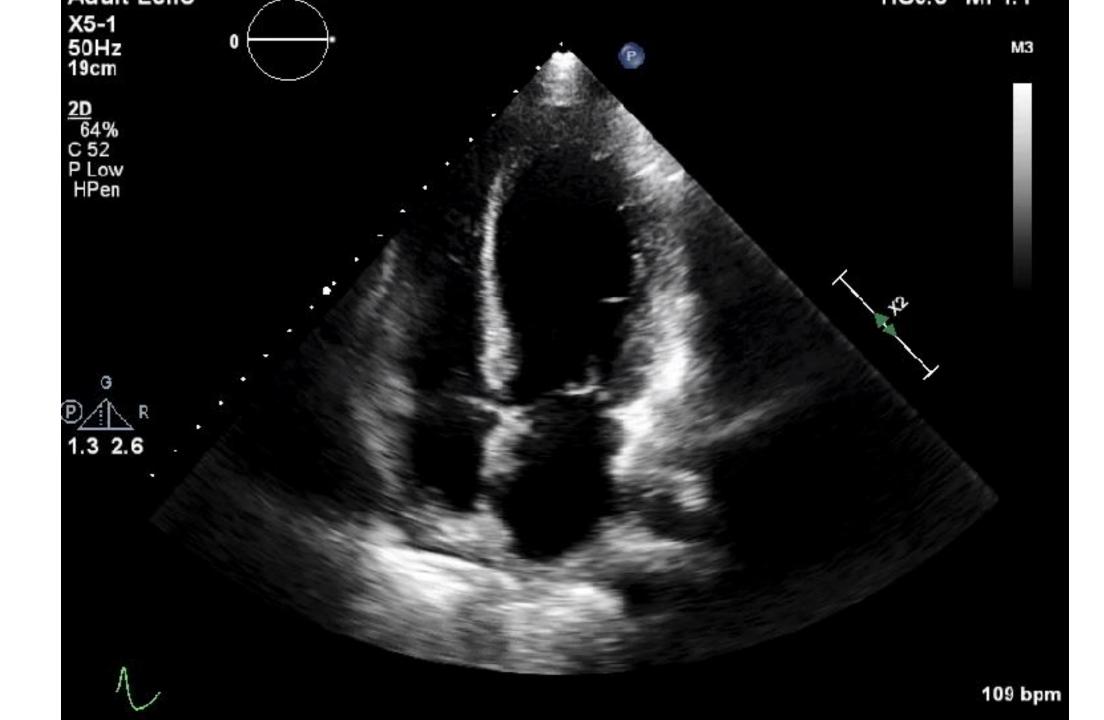
	PCI Capable Center FMC to Device (Banner)	PCI Non-Capable Center FMC to Device (Phoenix VA)	PCI Non-Capable Center FMC to Lytic (Prescott VA)
A	90 min	180 min	30 min
В	60 min	120 min	30 min
С	90 min	120 min	30 min
D	60 min	90 min	30 min





ESC 2017







82 year old female with PMHx CKD III (baseline Cr 1.6), HFrEF (EF 35%) 2/2 NICM, DM2, HTN and hx breast cancer presents to ER with weakness/fatigue, dyspnea on exertion and left sided chest pain.

VS: BP 88/50, HR 130, SpO2 86% RA

Physical Exam:

Gen: Old, frail appearing, pale, diaphoretic, asking for you to help her

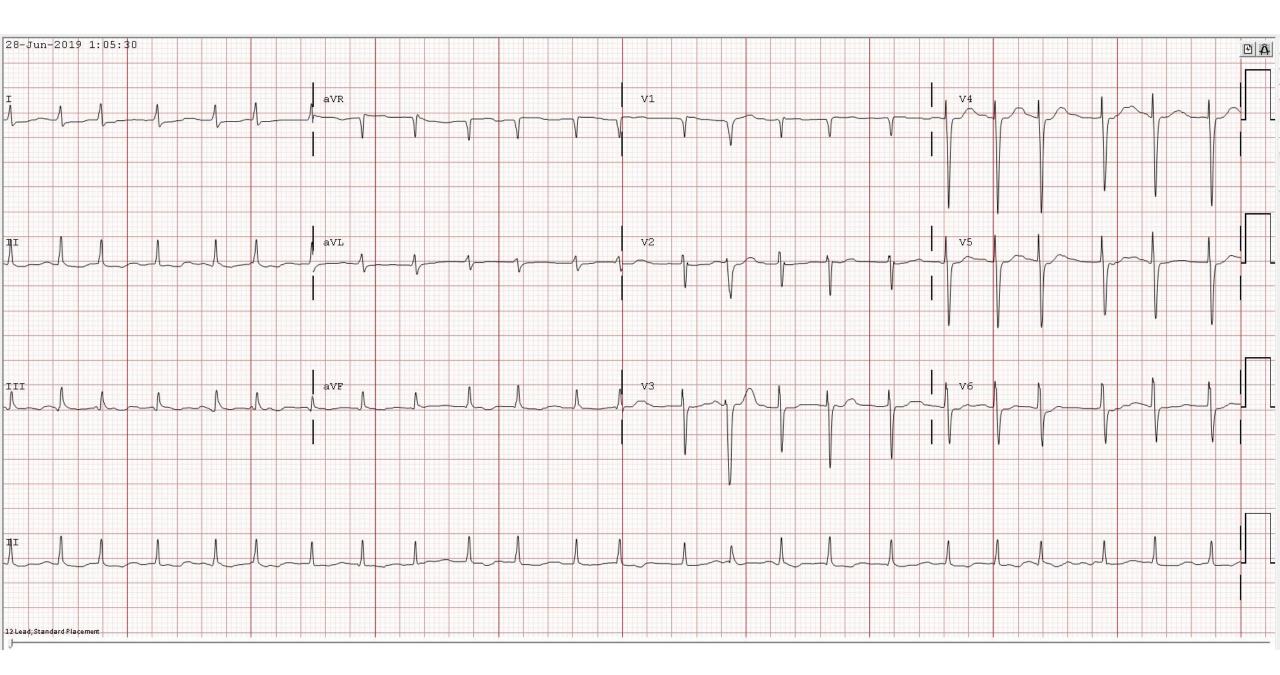
Resp: Rapid shallow breathing, poor air movement

CV: Irregular rate and rhythm, no murmur, 1+ BL LE edema

GI: Soft, distended abdomen, DRE + melena

EKG: To follow

Labs: Na 128, K 5.9, Cr 3.3, Hb 5.2, Plts 120, Lactate 4.0, Troponin 450



Case #3

82 year old female presents to ER with weakness/fatigue, dyspnea on exertion and left sided chest pain.
VS: hypotensive, tachycardic, hypoxic
EKG: Afib with RVR
Labs: HS trop 450, Cr 3.3, Hb 5.2

- A. NSTE-ACS start ACS protocol (DAPT + AC)
- B. Type I MI Activate cardiac cath lab
- C. Type II MI No ACS protocol, troponin elevation likely due to demand ischemia from rapid Afib and anemia with underlying CKD
- D. Type II MI Start ASA and Plavix, no AC given anemia
- E. Hope your senior resident is back from continuity clinic

Basics of coronary artery perfusion

Supply and Demand

- What can decrease supply?
- What can increase demand?

	Injury related to primary myocardial ischaemia		
	Plaque rupture Intraluminal coronary artery thrombus formation		
Fourth Univer	Injury related to supply/demand imbalance of myocardial ischaemia	vember 2018	
Hypoten shock	 Tachy-/brady-arrhythmias Aortic dissection or severe aortic valve disease Hypertrophic cardiomyopathy Cardiogenic, hypovolaemic, or septic shock Severe respiratory failure Severe anaemia Hypertension with or without LVH Coronary spasm Coronary embolism or vasculitis Coronary endothelial dysfunction without significant CAD Injury not related to myocardial ischaemia Cardiac contusion, surgery, ablation, pacing, or defibrillator shocks Rhabdomyolysis with cardiac involvement Myocarditis Cardiotoxic agents, e.g. anthracyclines, herceptin 	nmia	
	Multifactorial or indeterminate myocardial injury		
	Heart failure Stress (Takotsubo) cardiomyopathy Severe pulmonary embolism or pulmonary hypertension Sepsis and critically ill patients Renal failure Severe acute neurological diseases, e.g. stroke, subarachnoid haemorrhage Infiltrative diseases, e.g. amyloidosis, sarcoidosis Strenuous exercise		

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Universal definitions of myocardial injury and myocardial infarction

Criteria for myocardial injury

The term myocardial injugit should be used when there is evidence of elevated cardiac troponin values (cTo) with at least 1 value above the 99th percentile

Criteria for acute myo

upper reference limit (UF

The term acute myocard detection of a rise and/o

- Symptoms of myoca
- New ischemic ECG
- Development of pat
- Imaging evidence of
- Identification of a contract

Postmortem demonstrati between myocardial oxy suggestive of myocardial

Criteria for coronary p

Percutaneous coronary ir Coronary artery bypass g Coronary procedure-rela

for type 5 MI of the 99th cTn level are stable (≤20 addition with at least 1 c

- New ischemic ECG
- Development of nev
- Imaging evidence of
- Angiographic findin side-branch occlusic

Isolated development of and rising but less than t Other types of 4 MI inclu Postmortem demonstrat

Criteria for prior or sil

Any 1 of the following c

Criteria for Type 1 MI

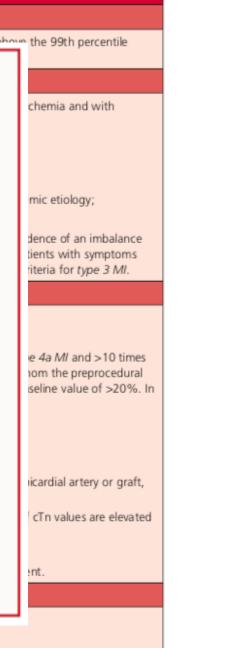
Detection of a rise and/or fall of cTn values with at least 1 value above the 99th percentile URL and with at least 1 of the following:

- Symptoms of acute myocardial ischemia;
- New ischemic ECG changes;
- Development of pathological Q waves;
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic etiology;
- Identification of a coronary thrombus by angiography including intracoronary imaging or by autopsy.*

cTn indicates cardiac troponin; ECG, electrocardiogram; URL, upper reference limit.

*Postmortem demonstration of an atherothrombus in the artery supplying the infarcted myocardium, or a macroscopically large circumscribed area of necrosis with or without intramyocardial hemorrhage, meets the type 1 MI criteria regardless of cTn values.

- Abnormal Q waves with or without symptoms in the absence of nonischemic causes.
- Imaging evidence of loss of viable myocardium in a pattern consistent with ischemic etiology.
- Patho-anatomical findings of a prior MI.



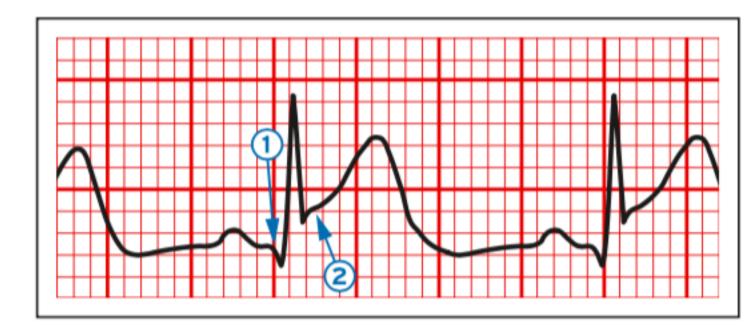
ST Elevation:

ST-elevation

New ST-elevation at the J-point in 2 contiguous leads with the cut-point: $\geq 1 \text{ mm}$ in all leads other than leads $V_2 - V_3$ where the following cutpoints apply: $\geq 2 \text{ mm}$ in men $\geq 40 \text{ years}$; $\geq 2.5 \text{ mm}$ in men <40 years, or $\geq 1.5 \text{ mm}$ in women regardless of age.*

ST-depression and T wave changes

New horizontal or downsloping ST-depression ≥ 0.5 mm in 2 contiguous leads and/or T inversion >1 mm in 2 contiguous leads with prominent R wave or R/S ratio >1.

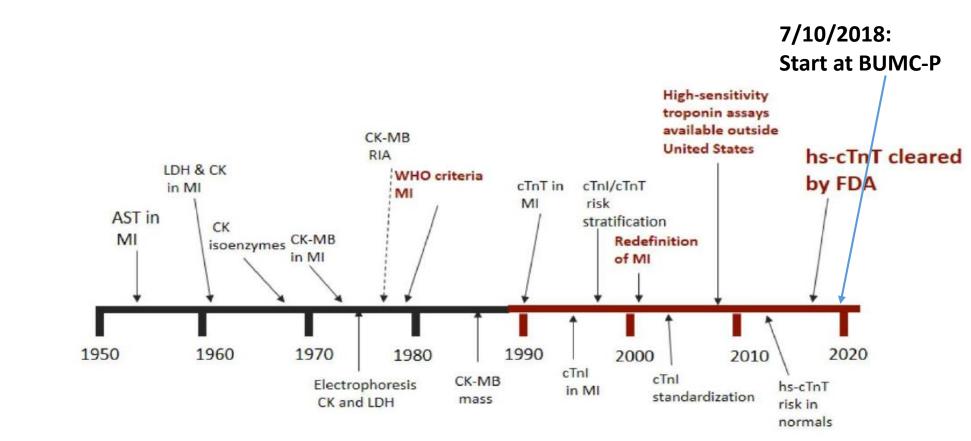


Classification of Acute MI

- Type 1:
 - Spontaneous MI related to ischemia due to primary coronary event such as plaque rupture
- Type 2:
 - MI secondary to ischemia due to either increased oxygen demand or decreased supply
- Type 3:
 - Sudden unexpected cardiac death with coronary event prior to troponin evaluation
- Type 4a:
 - MI associated with PCI
- Type 4b:
 - MI associated with stent thrombosis
- Type 5:
 - MI associated with CABG

HIGH SENSITIVITY-CARDIAC TROPONIN T hs-cTnT

Necrosis Biomarkers Timeline

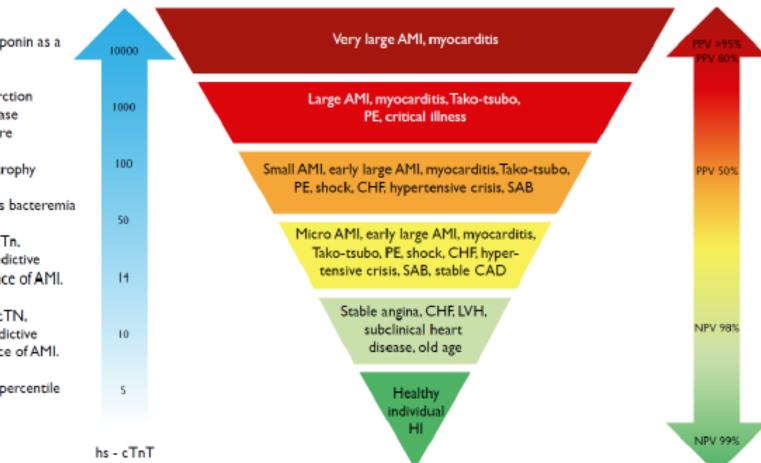


High-sensitivity cardiac troponin as a quantitative marker.

AMI acute myocardial infarction CAD coronary artery disease CHF congestive heart failure HI healthy individual LVH left ventricular hypertrophy PE pulmonary embolus SAB Staphylococcus aureus bacteremia The lower the level of hs-cTn, the higher the negative predictive value (NPV) for the presence of AMI.

The higher the level of hs-cTN, the higher the positive predictive value (PPV) for the presence of AMI.

Levels just above the 99th percentile have a low PPV for AMI



(derivative of Garg et al, Cardiac biomarkers of acute coronary syndrome: from history to high-sensitivity cardiac troponin, Intern Emerg Med. (2017) 12:147-155). This work is licensed under Creative Commons Attribution 2.0 Generic License)

Use in evaluation of suspected ACS

For STEMI patient- activate CARDIAC ALERT

Evaluation of chest pain must integrate clinical, EKG, and hs-cTnT information. Clinical care must not be based on lab values alone.

Initial hs-cTnT Lab Value

Repeat Lab at 2hr

2 hr change in lab value (increase or decrease)

Repeat Lab at 4hr

4 hr change in lab value from baseline



For STEMI patient- activate CARDIAC ALERT

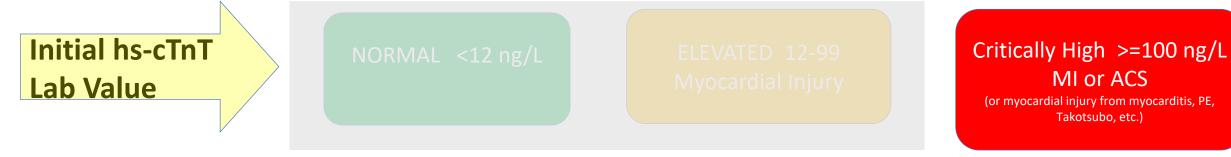
Evaluation of chest pain must integrate clinical, EKG, and hs-cTnT information. Clinical care must not be based on lab values alone.





For STEMI patient- activate CARDIAC ALERT

Evaluation of chest pain must integrate clinical, EKG, and hs-cTnT information. Clinical care must not be based on lab values alone.

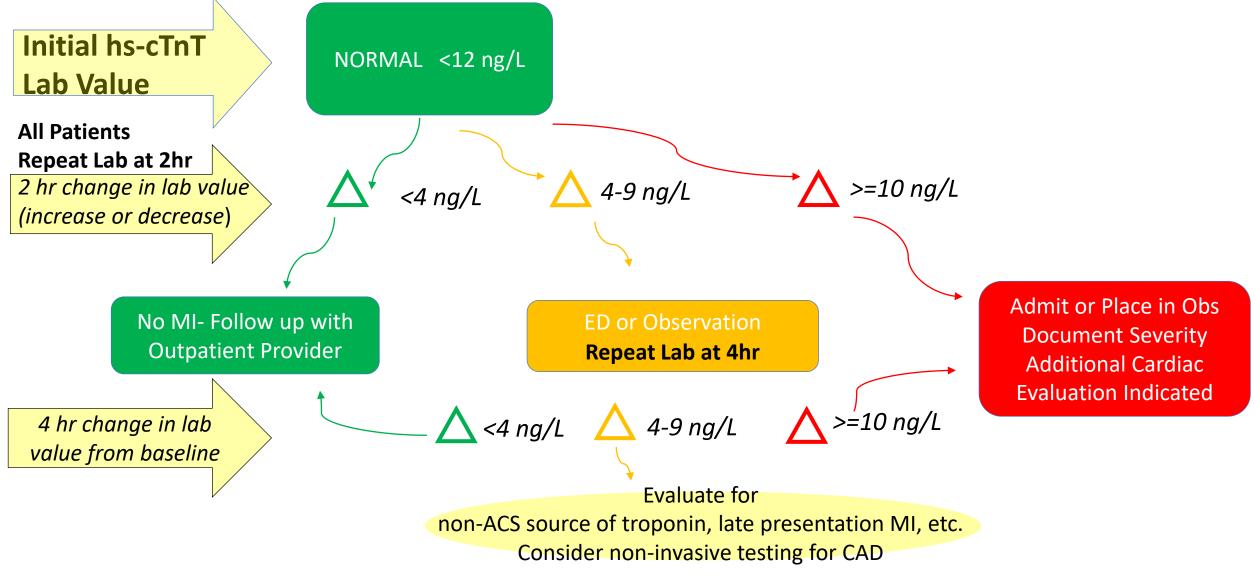


Admit or Place in Obs Document Severity Additional Cardiac Evaluation Indicated



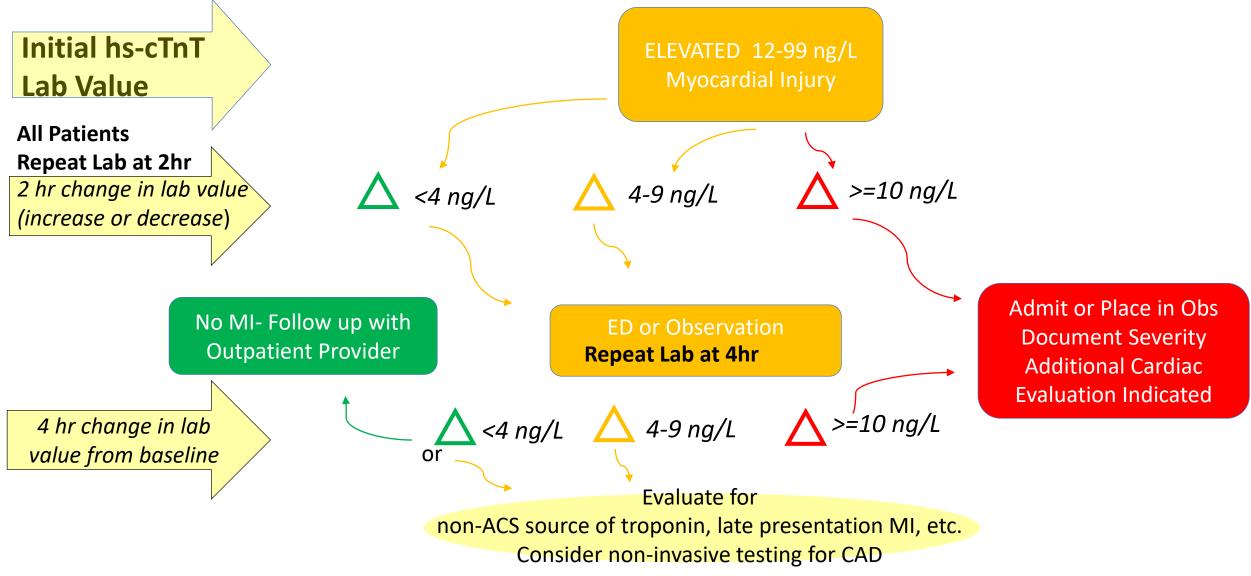
For STEMI patient– activate CARDIAC ALERT

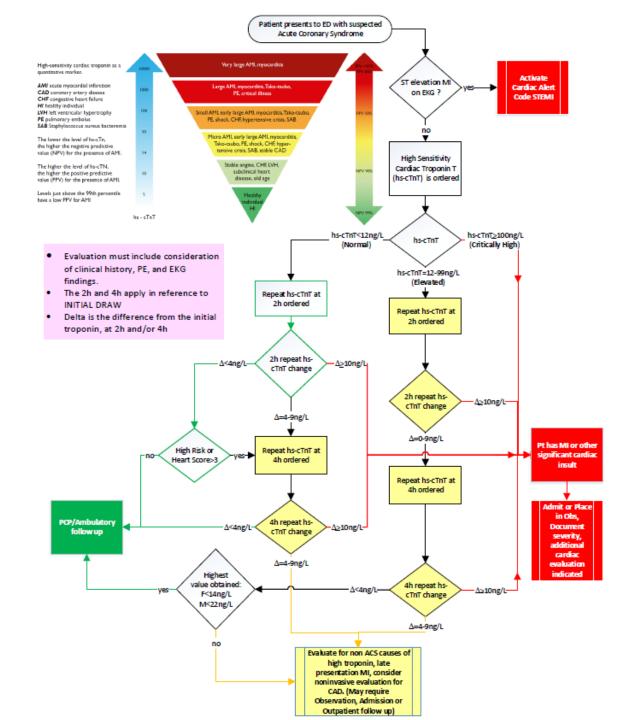
Evaluation of chest pain must integrate clinical, EKG, and hs-cTnT information. Clinical care must not be based on lab values alone.

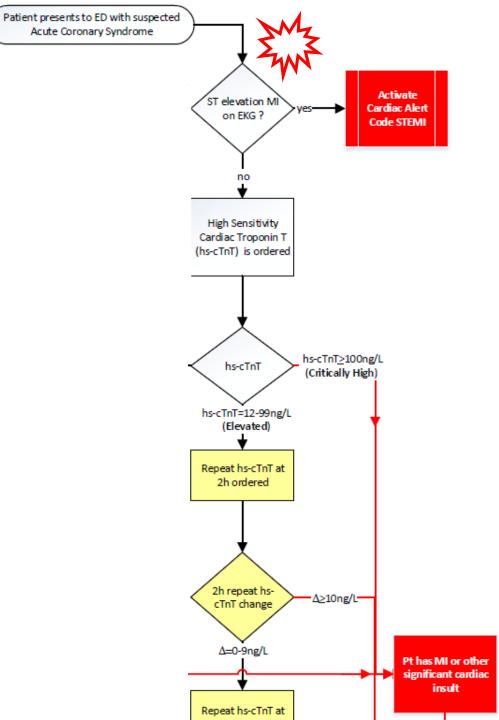


For STEMI patient- activate CARDIAC ALERT

Evaluation of chest pain must integrate clinical, EKG, and hs-cTnT information. Clinical care must not be based on lab values alone.

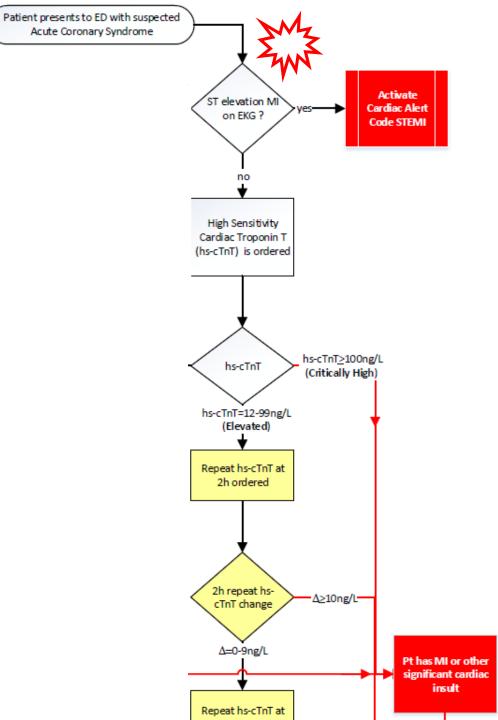






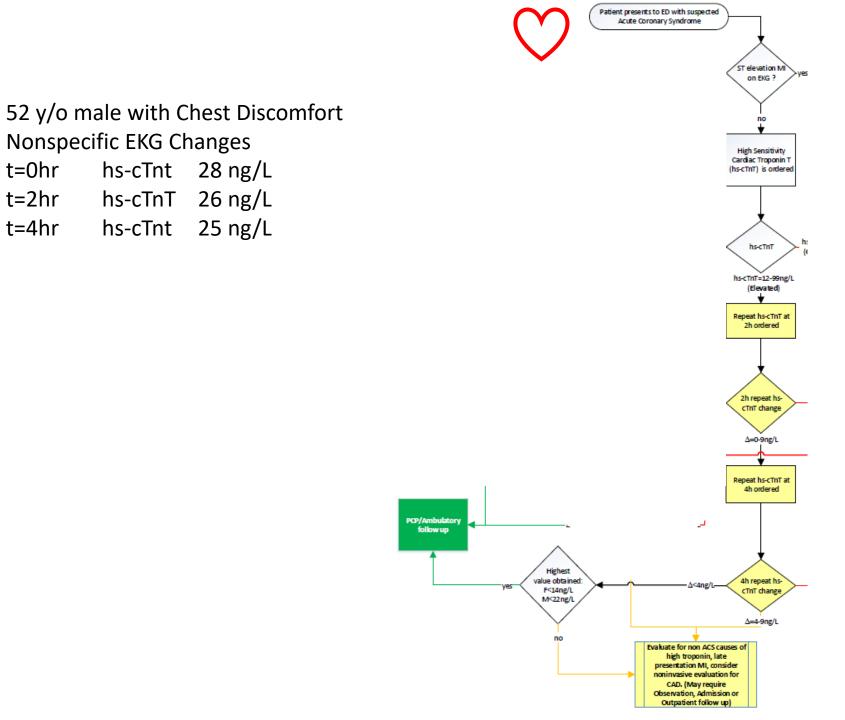
Banner Health

74 y/o female with Chest Discomfort Nonspecific EKG Changes t=0hr hs-cTnt 152 ng/L



Banner Health

62 y/o Male with Chest Discomfort Nonspecific EKG Changes t=0hr hs-cTnt 36 ng/L t=2hr hs-cTnT 49 ng/L



t=0hr

t=2hr

© OPEN ACCESS True 99th centile of high sensitivity cardiac troponin for hospital patients: prospective, observational cohort study



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HIGH SENSITIVITY TROPONIN?

OR

LOW SPECIFICITY TROPONIN?

Tips

Consults

- 1. Know the acuity
- 2. Know your question
- 3. Know your patient

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