

# Clinical protocols for high-sensitivity troponin I testing at Emory Hillandale Hospital (EHH), Emory Decatur Hospital (EDH), and Emory Long-Term Acute Care (ELTAC) \*

**Go-live dates for Siemens Atellica assay: December 2, 2025 (EHH); January 20, 2026 (EDH and ELTAC)**

## Emory HS troponin clinical protocols working group

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\* Disclaimer: The high-sensitivity troponin I protocols in these slides have been developed *only* for hospitals that use the Siemens Atellica Analyzer, including Emory Decatur Hospital, Emory Hillandale Hospital, and Emory Long-Term Acute Care hospital. The troponin cut points in these slides do *not* apply to Emory University Hospital, Emory University Hospital Midtown, Emory Saint Josephs Hospital, Emory Johns Creek Hospital, Emory University Orthopedics and Spine Hospital, or Grady Memorial Hospital, which use different lab analyzers (refer to separate protocols). Once the Siemens Atellica assay goes live at EHH, EDH, and ELTAC, this clinical protocol replaces the prior clinical protocols for these same hospitals which used the Siemens Vista assay.

## References:

1. Thygesen K et al. Fourth Universal Definition of MI (2018). J Am Card Cardiol 2018
2. Collet JP et al. 2020 ESC Guidelines for management of ACS in patients without persistent ST-segment elevation. Eur Heart J 2020. doi: 10.1093/eurheartj/ehaa575
3. Siemens Atellica IM Analyzer High-Sensitivity Troponin I insert (11200498\_EN Rev. 06, 2019-06)
4. Apple FS et al. Getting cardiac troponin right. Clin Chem 2021. doi: 10.1093/clinchem/hvaa337
5. Boeddinghaus J et al. HS cardiac troponin I assay for early diagnosis of AMI. Clin Chem 2019. doi: 10.1373/clinchem.2018.300061
6. Januzzi JL et al. Recommendations for institutions transitioning to HS troponin testing. J Am Card Cardiol 2019. doi: 10.1016/j.jacc.2018.12.046

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For all Emory HS troponin clinical protocols and videos, visit:

<https://med.emory.edu/departments/medicine/divisions/cardiology/hs-troponin-protocols/index.html>

For Emory HS troponin educational video, visit:

<https://youtu.be/v0muP7bveYM>

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# New versus old hs-TnI assay: EHC DOU sites: EDH, EHH, ELTAC\*

Note the following regarding hs-troponin I:

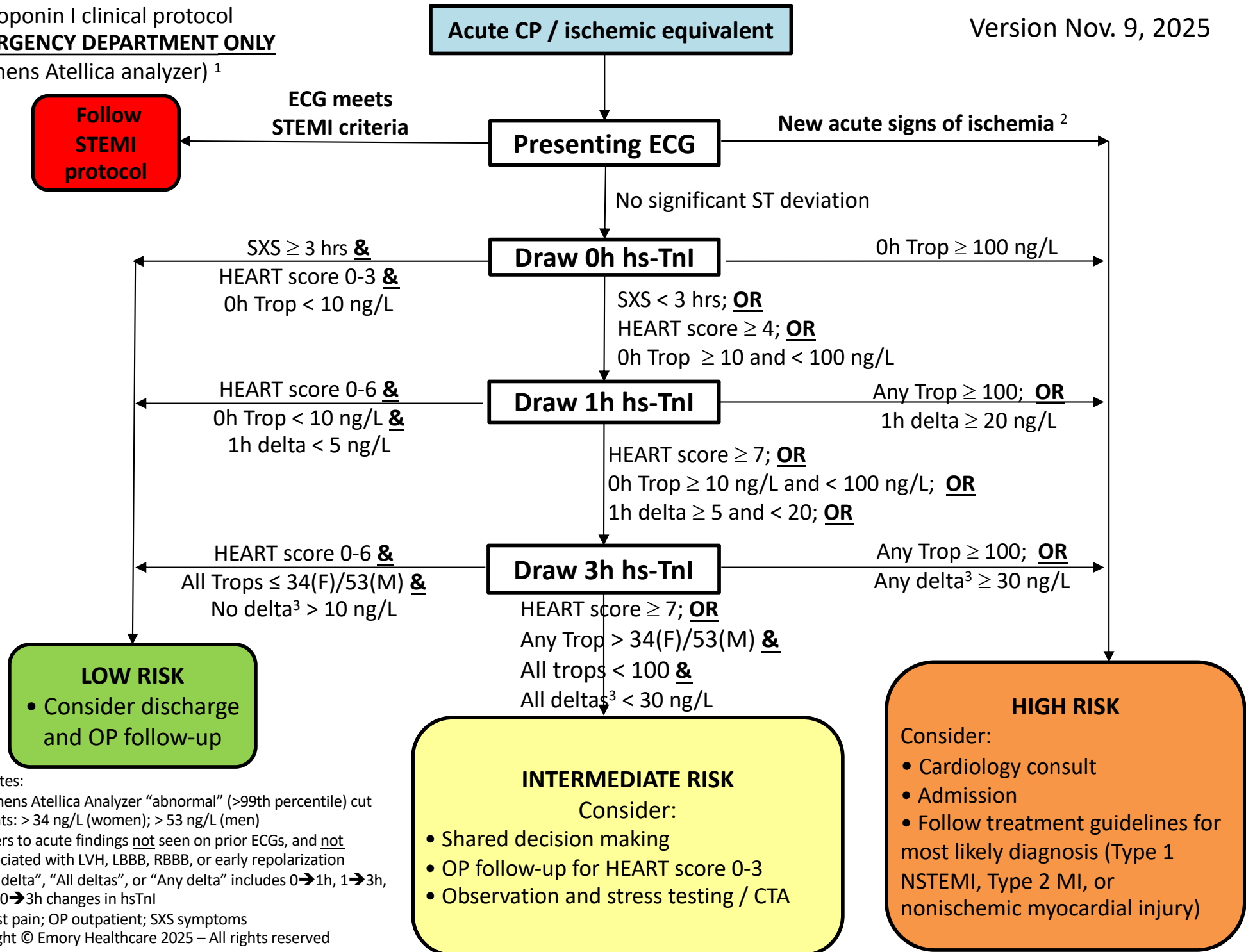
1. Units of measurement are reported in ng/L (or pg/mL)
2. Hs-TnI has different “abnormal” cut point, (or 99th percentile value) in women and men.

New assay (Siemens Atellica) hs-TnI (ng/L)	Old assay (Siemens Vista) hs-TnI (ng/L)	Notes
< 2.5	< 3.0	LOQ** for Siemens hs-TnI
50	50	
34	53	99 <sup>th</sup> percentile hs-TnI value, women
53	79	99 <sup>th</sup> percentile hs-TnI value, men
100	100	
500	500	EHC lab “critical” value for standard TnI §
1000	1000	
10000	10000	
> 25,000	> 25,000	Highest calibrator*** value for hs-TnI

\* EDH, EHH, and ELTAC now use a Siemens Atellica analyzer with the following “abnormal” (>99th percentile) cut points: >34 ng/L in women; >53 ng/L in men

\*\* LOQ: Lowest hs-TnI concentration that is reportable as a number with specified certainty

\*\*\* hs-TnI initially reported as >25,000 ng/mL will subsequently be reported in Epic as an actual value (click on value “>25,000” in Epic lab flowsheet and look under “Comments” to see actual value after sample dilution)



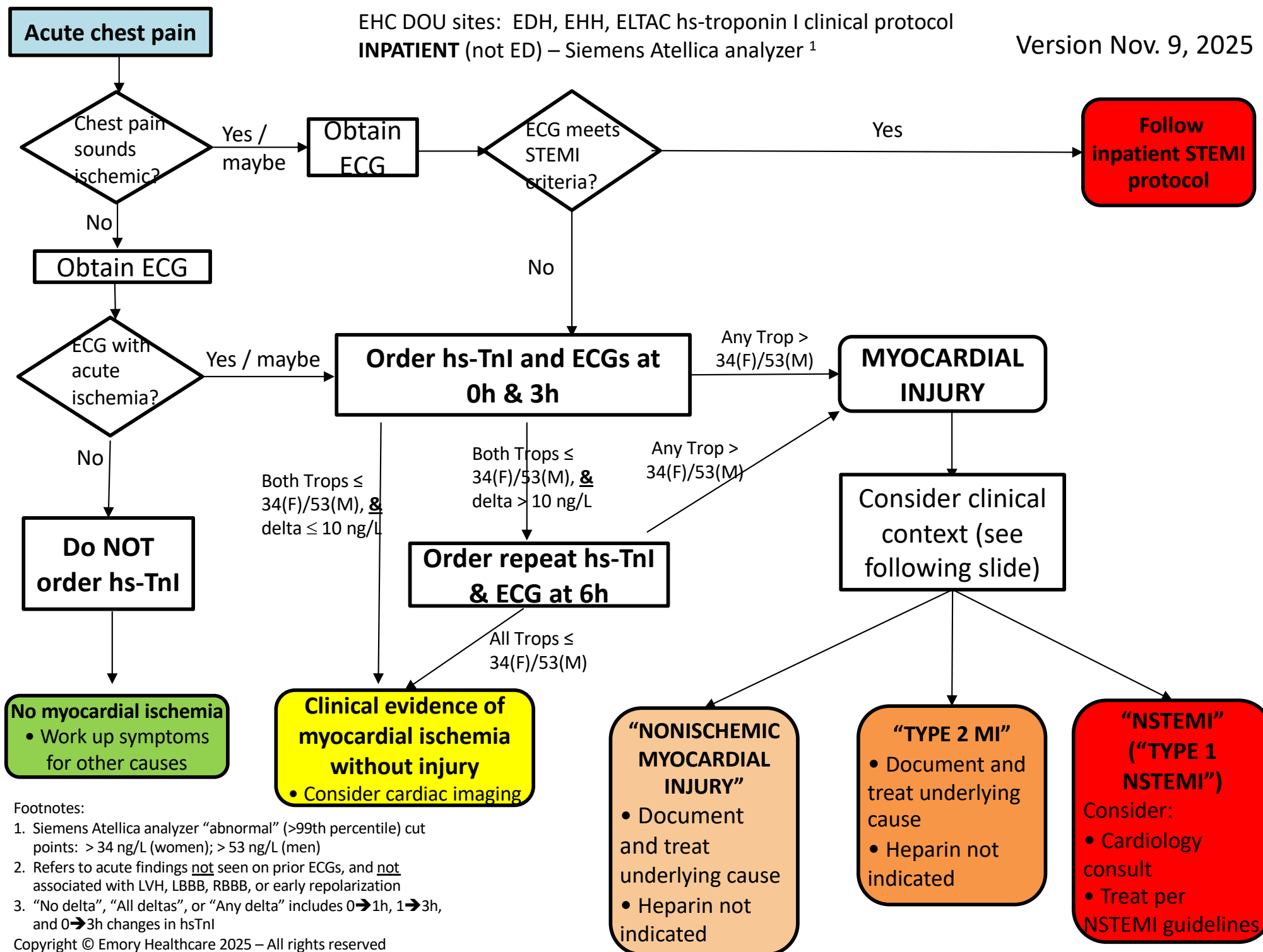
Footnotes:

1. Siemens Atellica Analyzer "abnormal" (>99th percentile) cut points: > 34 ng/L (women); > 53 ng/L (men)
2. Refers to acute findings not seen on prior ECGs, and not associated with LVH, LBBB, RBBB, or early repolarization
3. "No delta", "All deltas", or "Any delta" includes 0→1h, 1→3h, and 0→3h changes in hsTnI

CP chest pain; OP outpatient; SXS symptoms  
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# HEART Score (used only in ED)

HEART Score		
History	Slightly suspicious	0
	Moderately suspicious	1
	Highly suspicious	2
EKG	Normal	0
	Non-specific repolarization disturbance	1
	Significant ST deviation	2
Age	< 45	0
	45-64	1
	≥ 65	2
Risk Factors	No known risk factors	0
	1-2 risk factors	1
	≥ 3 risk factors OR atherosclerotic disease	2
Initial troponin	Less than upper limit of normal	0
	1 to 3x normal limit	1
	> 3x normal limit	2
TOTAL:		



Footnotes:

1. Siemens Atellica analyzer “abnormal” (>99th percentile) cut points: > 34 ng/L (women); > 53 ng/L (men)
2. Refers to acute findings not seen on prior ECGs, and not associated with LVH, LBBB, RBBB, or early repolarization
3. “No delta”, “All deltas”, or “Any delta” includes 0→1h, 1→3h, and 0→3h changes in hsTnl

# MYOCARDIAL INJURY (any hs-TnI value > 99<sup>th</sup> percentile)

## No clinical evidence of overt myocardial ischemia

- No ischemic symptoms, no ECG changes, & no abnormalities on cardiac imaging

**This is NOT an acute myocardial infarction (MI).**

**Document “NONISCHEMIC MYOCARDIAL INJURY secondary to [underlying cause]”**  
(outdated term: “non-MI troponin elevation”)

- Treat cause of nonischemic injury (if appropriate)

### Underlying causes of nonischemic myocardial injury:

#### Acute<sup>1</sup> nonischemic myocardial injury:

- Critical illness<sup>2</sup>
- Hypertensive emergency<sup>2</sup>
- Acute heart failure
- Takotsubo cardiomyopathy
- Acute pulmonary embolism (PE)
- Sepsis without shock
- Myocarditis / Pericarditis
- Acute endocarditis
- Non-cardiac surgery<sup>2</sup>
- Tachycardia (AFRVR, SVT, VT)<sup>2</sup>
- Blunt chest injury (CPR, contusion)
- Defibrillator shocks
- Cardiac ablation
- Cardiac (non-CABG) surgery
- Acute neuro event (stroke, seizure)
- Diabetic ketoacidosis
- Rhabdomyolysis
- Strenuous exercise
- Burn injuries to body

#### Chronic<sup>1</sup> nonischemic myocardial injury:

- Structural heart disease
- Severe aortic valve disease
- Hypertrophic cardiomyopathy
- Chronic pulmonary hypertension / chronic PE
- Infiltrative disease (amyloid, sarcoid, tumors, etc.)
- ESRD / advanced CKD
- Cardiotoxic agents, chemotherapy

## Clinical evidence of overt myocardial ischemia

One or more of the following:

- Symptoms of acute myocardial ischemia
- New ischemic ECG changes
- New abnormality on imaging (wall motion abnormality on echo; noninvasive stress test showing ischemia or new infarct)
- Coronary angiogram / CTA show acute “culprit” lesion

**This IS an acute MI.**  
What type of MI is it?

Identifiable precipitant causing supply-demand mismatch

Suspect acute coronary artery plaque rupture/erosion

**Document “TYPE 2 MI secondary to [underlying precipitant]”**

- Treat underlying precipitant of Type 2 MI

### Underlying precipitants of Type 2 MI:

#### Cardiac causes:

- Tachycardia (AFRVR, SVT, VT)<sup>2</sup>
- Bradyarrhythmias
- Aortic dissection
- Coronary vasospasm
- Coronary vasculitis / endothelial dysfunction / microvascular disease
- Embolism to coronary artery
- Spontaneous coronary artery dissection (SCAD)

#### Systemic causes:

- Hypertensive emergency<sup>2</sup>
- Critical illness<sup>2</sup>
- Non-cardiac surgery<sup>2</sup>
- Septic shock
- Acute hypoxic resp. failure
- Severe anemia (acute blood loss, hemolysis)

**Document “Type 1 NSTEMI”<sup>3</sup>**

Consider:

- Cardiology consult
- Treat per NSTEMI guidelines (may include antiplatelet drugs, urgent cath)

#### References:

- Thygesen K et al. Fourth Universal Definition of MI (2018). J Am Cardiol 2018.
- Goyal A et al. What’s in a name? The new ICD-10 codes and Type 2 MI. Circulation 2017;136:1180-2

1 Acute nonischemic injury is associated with a rise/fall in troponin. Chronic injury associated with “flat” troponins.

2 Some conditions may cause either a Type 2 MI or a nonischemic myocardial injury. The presence / absence of ischemic symptoms, or findings on ECG / cardiac imaging / coronary angiography may help distinguish the two.

3 The term “NSTEMI” should only be documented when referring to Type 1 NSTEMI, and not for Type 2 MI.