Clinical protocols for high-sensitivity troponin testing at the Emory Healthcare DOU sites (Emory Decatur Hospital, Emory Hillandale Hospital, and Emory Long-Term Acute Care) * — go-live date Sept. 22, 2021

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* Disclaimer: The high-sensitivity troponin I protocols in these slides have been developed *only* for hospitals that use the Siemens Vista Analyzer, including Emory Decatur Hospital, Emory Hillandale Hospital, and Emory Long-Term Acute Care hospital. The troponin cut points in these slides do *not* pertain 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).

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. Beckman Coulter UniCel DxI Access analyzer package insert (2020).
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

Version: September 7, 2021

Background

- Europe has been using high-sensitivity troponin testing (hs-Tn) for >5 years; U.S. hospitals in various stages of adopting hs-Tn testing
- High sensitivity troponin test is more sensitive, & more precise at low concentrations, than standard troponin
- High-sensitivity troponin testing allows for faster MI “rule outs” in chest pain patients presenting to the ED
  - This leads to more efficient ED throughput
- Tradeoff: hs-Tn less specific for treatable heart attacks (e.g. Type 1 NSTEMI), and instead detects all types of heart injury (including nonischemic myocardial injuries and Type 2 MI), that don’t necessarily warrant treatment or change management

Equivalency of values: TnI vs. hs-TnI (EHC DOU sites: EDH, EHH, ELTAC)*

Note the following differences between troponin I and hs-troponin I:

1. Units of measurement are different. Hs-TnI is reported in ng/L (whereas TnI was reported in ng/mL)
2. Hs-TnI has different "abnormal" cut point, (or 99th percentile value) in women and men.
3. To convert from hs-TnI to standard TnI (for clinical context), **divide by 1000**. Example: hs-TnI value of 100 ng/mL corresponds to a standard TnI value of 0.1 ng/mL. See table below.

TnI (ng/mL)	**hs-TnI** (ng/L)	**Notes**
< 0.003	< 3.0	LOQ** for Siemens hs-TnI
0.04	40	LOQ* for Siemens standard TnI
0.045	45	99th percentile standard TnI value
0.05	50	
0.054	54	99th percentile hs-TnI value, women
0.079	79	99th percentile hs-TnI value, men
0.1	100	
0.5	500	EHC lab "critical" value for standard TnI §
1	1000	
10	10000	
25	> 25,000	Highest calibrator value for hs-TnI
>40		Highest calibrator value for standard TnI

* EDH, EHH, and ELTAC use a Siemens Vista analyzer with the following "abnormal" (>99th percentile) cut points: >53.7 ng/L in women; >78.5 ng/L in men

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

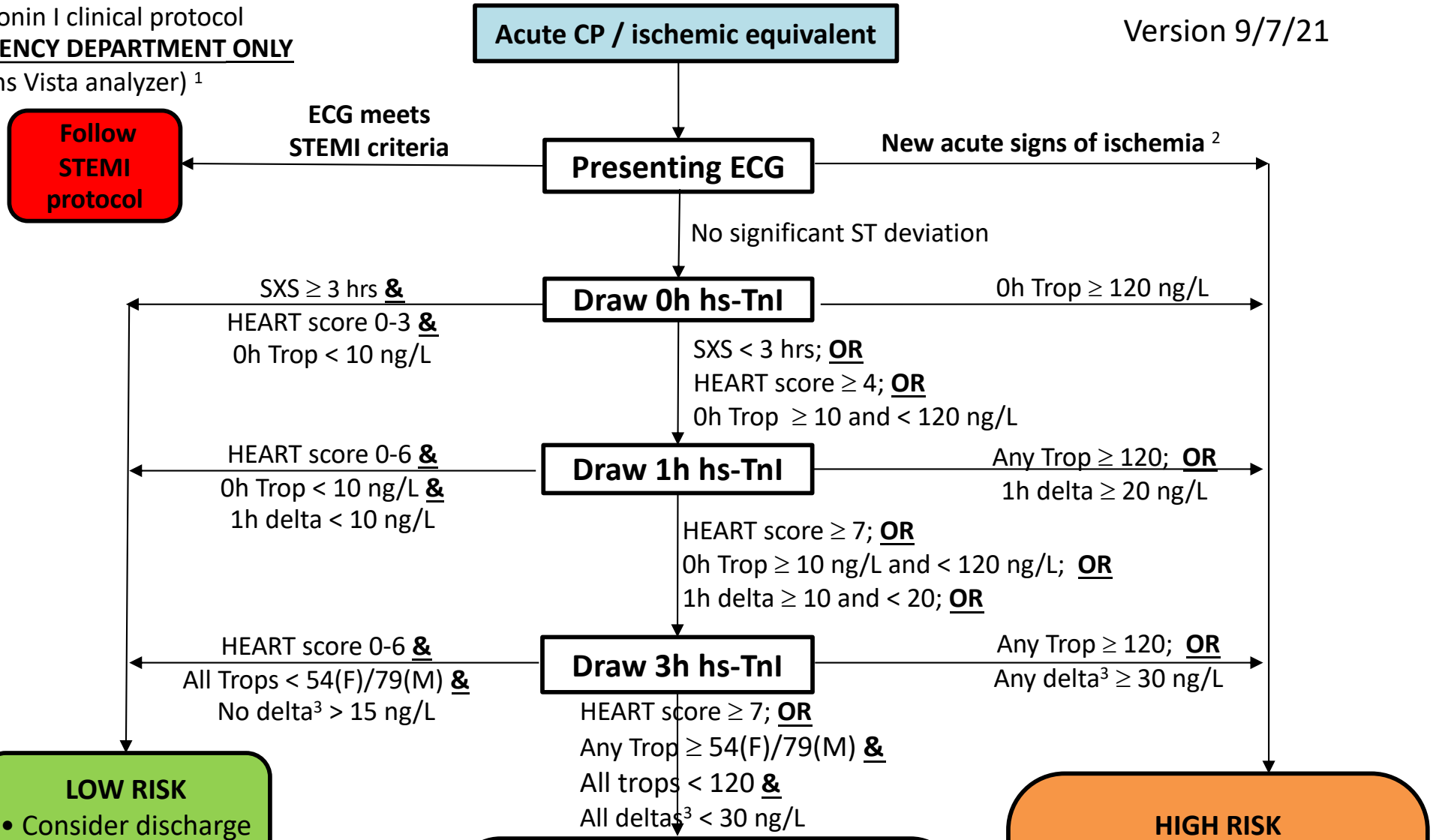
DOU: EDH, EHH, ELTAC

hs-troponin I clinical protocol

EMERGENCY DEPARTMENT ONLY

(Siemens Vista analyzer) [1]

Version 9/7/21



Footnotes:

1. Siemens Vista Analyzer "abnormal" (>99th percentile) cut points: ≥ 54 ng/L (women); ≥ 79 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

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:	

EHC DOU sites: EDH, EHH, ELTAC hs-troponin I clinical protocol
INPATIENT (not ED) – Siemens Vista analyzer [1]

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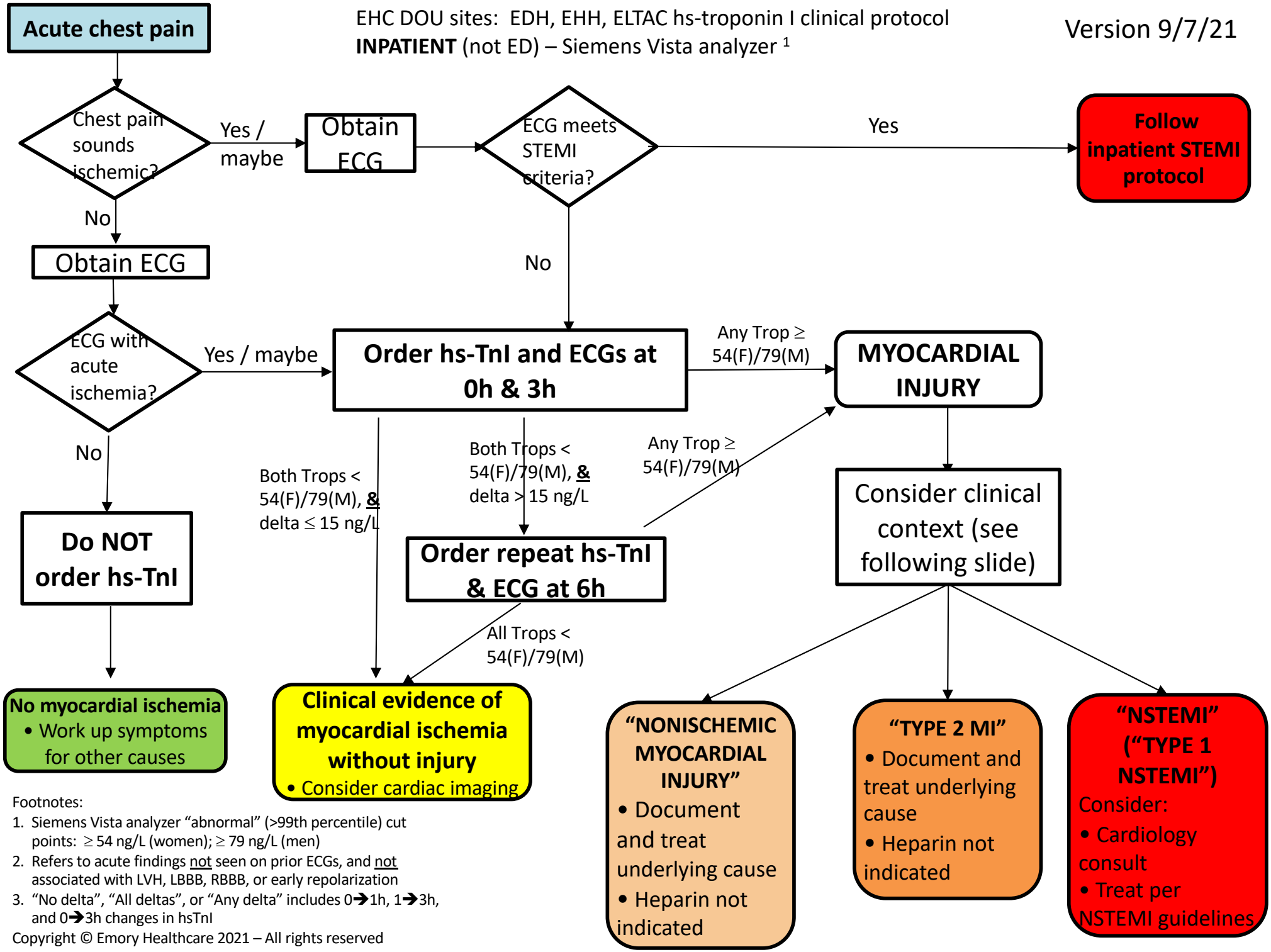

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Chart version: August 24, 2021
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MYOCARDIAL INJURY
(any hs-TnI value > 99th percentile)

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[1] nonischemic myocardial injury:
- Critical illness[2]
- Hypertensive emergency[2]
- Acute heart failure
- Takotsubo cardiomyopathy
- Acute pulmonary embolism (PE)
- Sepsis without shock
- Myocarditis / Pericarditis
- Acute endocarditis
- Non-cardiac surgery[2]
- Tachycardia (AFRVR, SVT, VT)[2]
- 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[1] 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

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)[2]
- Bradyarrhythmias
- Aortic dissection
- Coronary vasospasm
- Coronary vasculitis / endothelial dysfunction / microvascular disease
- Embolism to coronary artery
- Spontaneous coronary artery dissection (SCAD)

Systemic causes:
- Hypertensive emergency[2]
- Critical illness[2]
- Non-cardiac surgery[2]
- Septic shock
- Acute hypoxic resp. failure
- Severe anemia (acute blood loss, hemolysis)

Suspect acute coronary artery plaque rupture/erosion

Document "Type 1 NSTEMI" [3]
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 Card 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.