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

Emory and Grady HS troponin clinical protocols working group
Laboratory Medicine: Janetta Bryksin, PhD
Cardiology: Abhinav Goyal, MD; Michael McDaniel, MD; Michael Balk, MD; Matthew Topel, MD
Emergency Medicine: Michael Ross, MD; George Hughes, MD; Matthew Wheatley, MD; Sofia Khan, MD
Hospital Medicine: Ingrid Pinzon, MD; Viniya Patidar, MD
Project Manager: Shannon Lauer, MHA, PMP

* 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:

Inquiries:
Abhinav Goyal, MD, MHS, FACC, FAHA
Professor of Medicine, Emory University School of Medicine
Prof. of Epidemiology, Emory Rollins School of Public Health
Chief Quality Officer, Emory Heart and Vascular Center
Atlanta, GA, USA Email: ABHINAV.GOYAL@EMORY.EDU

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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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.

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

* 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
**Acute CP / ischemic equivalent**

- ECG meets STEMI criteria

**Presenting ECG**

- New acute signs of ischemia

**Draw 0h hs-TnI**

- SXS ≥ 3 hrs & HEART score 0-3 & 0h Trop < 10 ng/L
- HEART score 0-6 & 0h Trop < 10 ng/L & 1h delta < 10 ng/L
- HEART score 0-6 & All Trops < 54(F)/79(M) & No delta ≥ 15 ng/L

**Draw 1h hs-TnI**

- HEART score ≥ 7; OR 0h Trop ≥ 10 ng/L and < 120 ng/L; OR 1h delta ≥ 10 and < 20
- 0h Trop ≥ 120 ng/L; OR 1h delta ≥ 20 ng/L

**Draw 3h hs-TnI**

- HEART score ≥ 7; OR Any Trop ≥ 54(F)/79(M); OR All trop < 120 & All deltas ≥ 30 ng/L

**LOW RISK**

- Consider discharge and OP follow-up

**HIGH RISK**

- Consider: Cardiology consult, Admission, Follow treatment guidelines for most likely diagnosis (Type 1 NSTEMI, Type 2 MI, or nonischemic myocardial injury)

**INTERMEDIATE RISK**

- Consider: Shared decision making
- OP follow-up for HEART score 0-3
- Observation and stress testing / CTA

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

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### HEART Score (used only in ED)

<table>
<thead>
<tr>
<th>HEART Score</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>Slightly suspicious</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Moderately suspicious</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Highly suspicious</td>
<td>2</td>
</tr>
<tr>
<td>EKG</td>
<td>Normal</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Non-specific repolarization disturbance</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Significant ST deviation</td>
<td>2</td>
</tr>
<tr>
<td>Age</td>
<td>&lt; 45</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>45-64</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>&gt; 65</td>
<td>2</td>
</tr>
<tr>
<td>Risk Factors</td>
<td>No known risk factors</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>1-2 risk factors</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>&gt; 3 risk factors OR atherosclerotic disease</td>
<td>2</td>
</tr>
<tr>
<td>Initial troponin</td>
<td>Less than upper limit of normal</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>1 to 3x normal limit</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>&gt; 3x normal limit</td>
<td>2</td>
</tr>
</tbody>
</table>

**TOTAL:**
Acute chest pain

- Chest pain sounds ischemic?
  - Yes / maybe
    - Obtain ECG
  - No
    - Obtain ECG

- ECG with acute ischemia?
  - Yes / maybe
    - Order hs-TnI and ECGs at 0h & 3h
  - No
    - Do NOT order hs-TnI

- All Trops < 54(F)/79(M), & delta ≤ 15 ng/L
  - Clinical evidence of myocardial ischemia without injury
    - Work up symptoms for other causes
    - Consider cardiac imaging
- Any Trop ≥ 54(F)/79(M), & delta > 15 ng/L
  - MYOCARDIAL INJURY
  - Consider clinical context (see following slide)

- Any Trop ≥ 54(F)/79(M)
  - “TYPE 2 MI”
    - Document and treat underlying cause
    - Heparin not indicated
  - “NSTEMI” (“TYPE 1 NSTEMI”)
    - Consider:
      - Cardiology consult
      - Treat per NSTEMI guidelines

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

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

Identifiable precipitant causing supply-demand mismatch
- Suspect acute coronary artery plaque erosion/rupture

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

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

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

Document “Type 1 NSTEMI”

References:
- 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.