Clinical protocols for high-sensitivity troponin testing at Emory University Orthopedics and Spine Hospital (EUOSH) – go-live date Sept. 22, 2021

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*Disclaimer: The high-sensitivity troponin I protocol in these slides have been developed only for Emory University Orthopedics and Spine Hospital which uses the Beckman Coulter Access 2 Immunoassay analyzer. 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 Decatur Hospital, Emory Hillandale Hospital, Emory Long-Term Acute Care hospital, and Grady Memorial Hospital, which use different lab analyzers (refer to separate protocols).

References:

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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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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 (EUOSH) **

Note the following differences between standard troponin I and high-sensitivity troponin I (hs-TnI):

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

<table>
<thead>
<tr>
<th>standard TnI (ng/mL)</th>
<th>hs-TnI (ng/L)</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 0.002</td>
<td>&lt; 2.0</td>
<td>LOQ** for hs-TnI</td>
</tr>
<tr>
<td>0.012</td>
<td>12</td>
<td>99 percentile (abnormal) hs-TnI value for women</td>
</tr>
<tr>
<td>0.02</td>
<td>20</td>
<td>99 percentile (abnormal) hs-TnI value for men</td>
</tr>
<tr>
<td>0.03</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>0.04</td>
<td>40</td>
<td>99 percentile (abnormal) standard TnI value</td>
</tr>
<tr>
<td>0.05</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>0.1</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>0.5</td>
<td>500</td>
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</tr>
<tr>
<td>1</td>
<td>1000</td>
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</tr>
<tr>
<td>10</td>
<td>10000</td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>&gt; 25000</td>
<td>Highest reportable value of analytic range for hs-TnI</td>
</tr>
<tr>
<td>&gt;70</td>
<td></td>
<td>Highest reportable value of analytic range for TnI</td>
</tr>
</tbody>
</table>

* EUOSH uses a Beckman Coulter Access 2 Immunoassay analyzer with the following “abnormal” (>99th percentile) cut points: >11.6 mg/L in women; >19.8 ng/L in men. These cut points do NOT apply for EUH, EUHM, ESJH, EJCH, EDH, EHH, ELTAC , or Grady Memorial Hospital (see separate protocols for these operating units).

** LOQ: Lowest hs-TnI concentration that is reportable as a number with specified certainty
hs-TnI: high-sensitivity troponin I
Acute chest pain

- Chest pain sounds ischemic?
  - Yes / maybe: Obtain ECG
  - No: ECG with acute ischemia?
    - Yes / maybe: Order hs-TnI and ECGs at 0h & 3h
    - No: Do NOT order hs-TnI

  - Clinical evidence of myocardial ischemia without injury
    - Work up symptoms for other causes
    - Consider cardiac imaging

  - No myocardial ischemia

  - All Trops < 12(F)/20(M)

  - Any Trop ≥ 12(F)/20(M)
    - MYOCARDIAL INJURY
      - Consider clinical context (see following slide)

  - Order repeat hs-TnI & ECG at 6h

EUOSH hs-troponin I clinical protocol INPATIENT (not ED) – Beckman Coulter Access 2 Immunoassay analyzer

Footnotes:
1. Beckman Coulter Access 2 Immunoassay analyzer “abnormal” (>99th percentile) cut points: >11.6 mg/L (women); >19.8 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.

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)
- 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” 3

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.