**HS troponin (hs-cTnT) FAQ Sheet for clinicians**

January 31, 2022

**Why change to hs-cTnT?**

The 2021 American College of Cardiology/American Heart Association Chest Pain guidelines endorse hs-cTnT as the preferred biomarker for assessment of patients with acute chest pain because it allows rapid detection of myocardial injury and has increased diagnostic accuracy. Hs-cTnT is rapidly becoming the standard of care in hospitals across the US.

**What is hs-cTnT?**

Hs-cTnT is a cardiac biomarker highly sensitive and specific for the detection of myocardial injury. When used in combination with clinical findings (symptoms, ECG findings, etc), it is possible to rule out ischemic myocardial injury rapidly and accurately in most patients.

**What do I need to know?**

Please refer to Care Signature pathways for using hs-cTnT for more detail and guidance.

- Low risk patients (defined by negative hs-cTnT AND non-suggestive clinical evaluation) can now be discharged from ED without additional imaging (i.e. stress testing, coronary CTA). **Note that the algorithm for emergency patients is slightly different than the algorithm for inpatients.**

- What if the troponin is not drawn on time?
  - Nursing/phlebotomy is aware to **not** draw prior to the 1 hour mark, as premature results are uninterpretable
  - Troponin drawn between 1-3 hours should be treated as a “1hr troponin”
  - Troponin drawn 3-6 hours should be treated as a “3hr troponin”

- What if it hemolyzes?
  - No need to reorder – the lab will send a “redraw” task to the RN

- Patients with CKD/ESRD:
  - Compare hs-troponin to prior hs-troponin values if available
    - use rough conversion chart to compare to 4rth generation troponin
  - Noting a change from 0 to 1 hour and 3 hour results is critical for excluding acute injury/ischemia
    - Refer to ED or inpatient chest pain pathway for details
High Sensitivity cTnT result values and interpreting results:

<table>
<thead>
<tr>
<th>Normal</th>
<th>Abnormal but indeterminate for ischemia</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 12 ng/L</td>
<td>12 – 51 ng/L at 0 hour</td>
<td>&gt;= 52 ng/L is at 0 hour</td>
</tr>
</tbody>
</table>

Depending on clinical evaluation, may require further evaluation, or possibility of being discharged without further imaging. Will require further work-up, including a repeat hs-cTnT at 1 hour. Will require further work-up, including a repeat hs-cTnT at 1 hour, and potential admission to Medicine/Cardiology or Cardiology consult.

INPATIENT: Any change of ≥ 10ng/L at 1 hour OR 3 hour from baseline may indicate myocardial Injury.

EMERGENCY DEPARTMENT: Any change of ≥ 5ng/L at 1 hour OR ≥ 10ng/L at 3 hour from baseline may indicate myocardial Injury.

Comparison chart of troponin assay:

<table>
<thead>
<tr>
<th>4th Generation Troponin T</th>
<th>5th Generation hs-cTnT</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.01 ng/ml</td>
<td>30 ng/L</td>
</tr>
<tr>
<td>0.03 ng/ml</td>
<td>53 ng/L</td>
</tr>
<tr>
<td>0.1 ng/ml</td>
<td>100 ng/L</td>
</tr>
<tr>
<td>1 ng/ml</td>
<td>1000 ng/L</td>
</tr>
</tbody>
</table>

Transition away from the concept of “rule in” or “rule out” to ischemic vs non-ischemic myocardial injury:

No clinical evidence of myocardial ischemia

Nonischemic myocardial injury

Clinical evidence of overt myocardial ischemia

ACUTE MI

Type 1 MI - Caused by acute atherothrombotic mechanisms

Type 2 MI - Myocardial necrosis in which a condition other than coronary plaque instability causes increased oxygen demand or decreased supply (hypotension, hypoxia, anemia, etc.)