**Clinical Interpretation of High-sensitivity Cardiac Troponin**

**STEP 1: Increase above upper reference limit (URL)?**
- **Yes:** myocardial injury
- **No:** continue with serial testing if acute myocardial injury is suspected clinically

**STEP 2: Significant change in serial testing***?
- **Yes:** acute myocardial injury
- **No:** chronic myocardial injury

*values and sampling protocols are assay-specific (look up in Eur Heart J Acute Cardiovasc Care 2017;6:218-22)

**STEP 3: Evidence for acute myocardial ischemia?**
- **Yes:** myocardial infarction
- **No:** work-up for alternative causes of myocardial injury

**STEP 4: Troponin increase with acute myocardial ischemia**

Evidence of acute coronary atherothrombosis

- **Type 1 MI**
  - Raptured plaque with intracoronary thrombus
  - Left anterior descending

Clinical context and mechanisms making oxygen demand and supply imbalance likely

- **Type 2 MI**
  - Troponin increase with acute myocardial ischemia

Algorithm based on the locally used High-sensitivity Cardiac Troponin Assay (fill out accordingly)

<table>
<thead>
<tr>
<th>URL</th>
<th>Admission</th>
<th>2 h follow-up testing</th>
<th>Change</th>
<th>Consequence</th>
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<tbody>
<tr>
<td></td>
<td>rule out</td>
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<td>other</td>
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**Type 1 MI: triggers**
- Plaque rupture
- Plaque erosion

**Type 2 MI: examples**
- Acute heart failure
- Sustained tachycardia
- Myocarditis

**Type 2**

- Examples
  - Structural heart disease
  - Chronic kidney disease

**Type 1**

- Examples
  - Acute heart failure
  - Myocarditis

**Clinical context and mechanisms making oxygen demand and supply imbalance likely**

- Secondary to another illness or process
  - Fixed coronary atherosclerosis
  - Coronary spasm
  - Coronary microvascular dysfunction
  - Coronary embolism
  - Coronary artery dissection +/- intramural haematoma
  - Sustained tachycardia
  - Severe hypertension +/- left ventricular hypertrophy
  - Severe bradycardia
  - Respiratory failure
  - Severe anaemia
  - Hypotension/Shock

*Ischaemic thresholds vary substantially in relation to the magnitude of the stressor and the extent of underlying cardiac disease.*

**Angiographic picture from J. Mair, Medical University Innsbruck, Austria**