## 1. Introduction and Who Guideline applies to

Cardiac troponin I (cTnI) and T (cTnT) are components of the contractile apparatus of myocardial cells and expressed almost exclusively in the heart. UHL uses the 'Siemens Healthineers high-sensitivity troponin I (hs-cTnI) assay' on ADVIA Centaur and Atellica IM analysers from 01Jun21.

Myocardial infarction (MI) is defined as the presence of acute myocardial injury as detected by **a rise and/or fall of cardiac troponin (cTn) values** with at least one value above the 99th percentile upper reference limit (URL) **AND** with at least one of the following:

- Symptoms of acute myocardial ischaemia
- New ischaemic ECG changes
- Development of pathological Q waves
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology [1]

Sandoval et al showed that a hs-cTnI value of 120ng/L has a higher initial positive predictive value for MI than the 99$^{th}$ percentile URL (7 out of 10 patients were found to have an MI). [2] Based on a study involving over 800 patients treated in UHL, we have chosen the slightly lower threshold of 100ng/L to define patients with a high probability of MI locally.

Although cTn is very sensitive for myocardial injury, with a single hs-cTnI <5ng/L shown to exclude MI in 99.5% of patients presenting more than two hours since the onset of chest pain who do not have ongoing symptoms, [3] the clinical context must be considered to distinguish between

- Type I MI - due to atherosclerotic plaque disruption with thrombosis (also known as non-ST segment elevation myocardial infarction [NSTEMI] or STEMI, differentiated by ECG features)
- Type II MI - oxygen supply/demand imbalance, e.g. due to shock, respiratory failure or sustained tachyarrhythmia
- Acute non-ischaemic myocardial injury - e.g. due to sepsis, myocarditis or acute heart failure
- Chronic myocardial injury - stable troponin level above the 99$^{th}$ percentile without rise or fall, e.g. due to chronic kidney disease or structural heart disease

**Evidence-based treatments such as anticoagulation and angioplasty have only been shown to benefit patients with Type 1 MI** whereas patients with Type 2 MI or acute non-ischaemic myocardial injury require optimal treatment of their underlying illness. It is therefore important to think more widely about the range of possible causes of cTn elevations rather than automatically assuming a diagnosis of NSTEMI, particularly in patients without chest pain.

All myocardial injuries as defined by an elevated cTn value, irrespective of the cause, are however associated with an adverse prognosis over the long term. Optimal management strategies for patients other than Type I MI have not yet been firmly established, but

- Patients with Type II MI may benefit from an evaluation of their coronary artery disease (CAD) likelihood and consideration of CT or even invasive coronary angiography
- Patients who have a stable elevated cTn without known structural heart disease or clear alternative pathology may benefit from echocardiography or cardiac MRI [4]

This guideline describes how cTn is used to diagnose NSTEMI in the investigation of non-specific chest pain in adults aged 25 years and older. It applies to all UHL areas and staff involved in the care of such patients – including the ED, AMU, AFU, CDU and all other inpatient areas.

STEMI falls outside the scope of the guideline. Further specific exclusion criteria are listed within the algorithm shown in Appendix A.

## 2. Guideline Standards and Procedures

2.1 Assessment should follow the algorithm shown in Appendix A (page 3)
2.2 Emergency Department (ED) clinicians should use the ED acute chest pain: NSTEMI rule in/out guideline (proforma), [5] which includes the same algorithm but also covers additional aspects of NSTEMI management (**NB**: Proforma may also be useful elsewhere)
2.3 Clinicians should use the Acute MI and Cardiology Decision Aid to help differentiate between Type I MI and other causes of acute myocardial injury [6]

## 3. Education and Training

No additional skills are required to follow this guideline. Awareness of the algorithm changes within their respective teams will be raised by ED, AMU, AFU, CDU and cardiology leadership.

## 4. Monitoring Compliance

| What will be measured to monitor compliance | How will compliance be monitored | Monitoring Lead | Frequency | Reporting arrangements |
|---|---|---|---|---|
| Patients transferred from ED to CDU / CCU with assumed NSTEMI who had readily identifiable features of Type 2 MI or acute non-ischaemic MI | Audit | Ian Hudson | Annually | ESM quality and safety board |

## 5. Supporting References

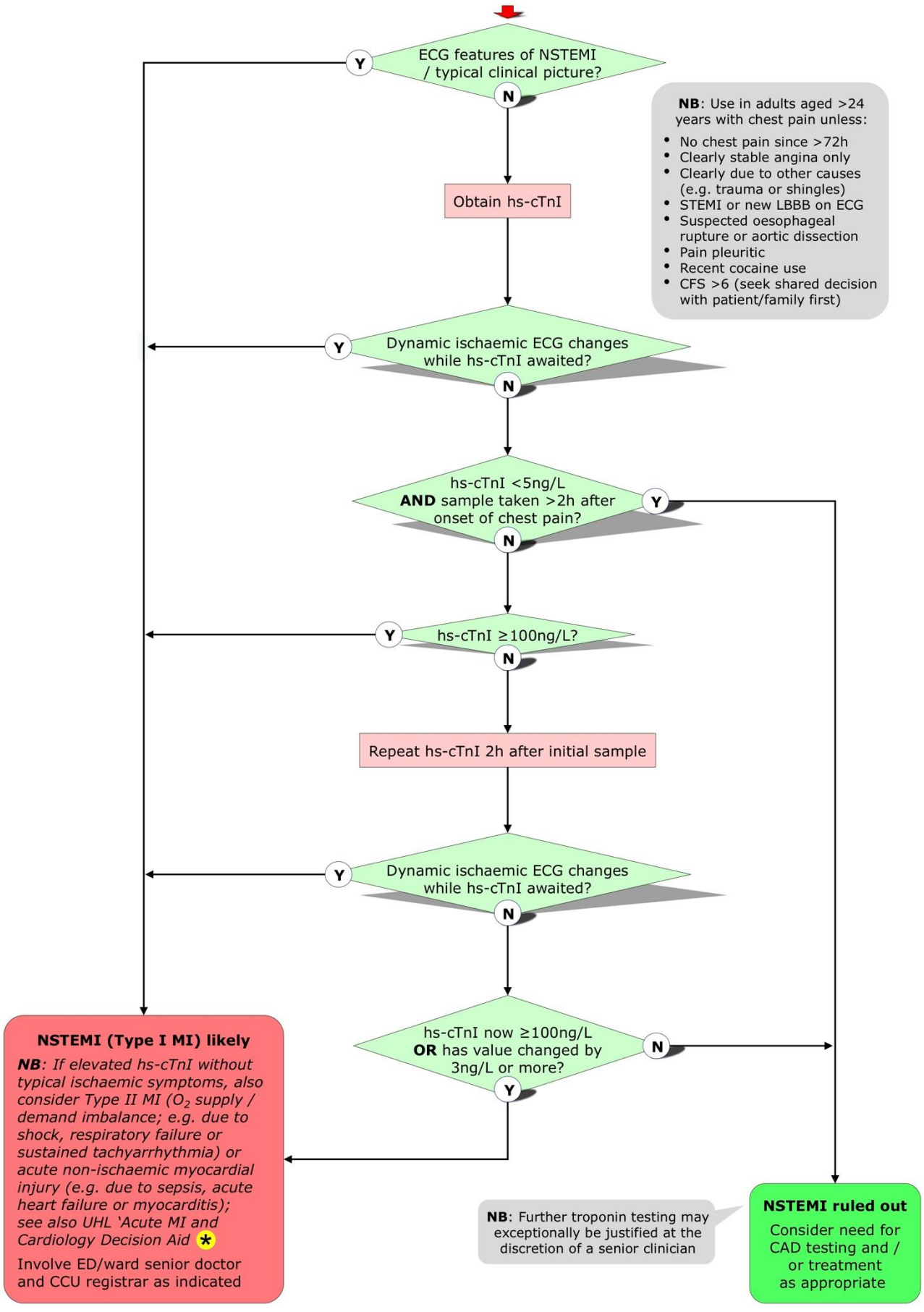

1. ThygesenK, AlpertJS, JaffeAS et al. Fourth Universal Definition of Myocardial Infarction (2018). Circulation 2018;138:e618-e651.
2. SandovalY, NowakR, deFilippiCR et al. Myocardial Infarction Risk Stratification with a single measurement of high-sensitivity troponin I. JAmCollCardiol 2019;74:271-82.
3. ShahAS, AnandA, SandovalY et al. High-sensitivity cardiac troponin I at presentation in patients with suspected acute coronary syndrome: a cohort study. Lancet 2015;386:2481-8.
4. ChapmanAR, AdamsonPD and Mills NL. Assessment and classification of patients with myocardial injury and infarction in clinical practice. Heart 2017;103:10-18.
5. WieseMF. Acute chest pain NSTEMI rule in or out UHL emergency department guideline. UHL PAGL. 2021. (Accessed 29 Apr 2021.)
6. RobertsE. Acute MI and Cardiology Decision Aid Including Troponin Interpretation. UHL 2017. (Accessed 29 Apr 2021.)


## 6. Key Words

Non-ST segment elevation myocardial infarction, NSTEMI, MI, troponin, cTn, hs-cTnI, chest pain, coronary care, CCU, emergency, ECG

| CONTACT AND REVIEW DETAILS | |
|---|---|
| **Guideline Lead (Name and Title)**<br>Pankaj Gupta, Metabolic Physician / Chemical Pathologist<br>Martin Wiese (reviewer), Emergency Physician | **Executive Lead**<br>Andrew Furlong, Medical Director |
| **Details of changes made during review:**<br>• Introduction rewritten, with added emphasis on<br>  ○ The need to distinguish between Type I MI and other types of acute myocardial ischaemia<br>  ○ The adverse prognostic relevance of elevated cTn values, irrespective of the cause<br>  ○ Potential investigation strategies for patients with non-ACS myocardial injuries added<br>• Guideline standards section rewritten, referencing the 'ED cute chest pain: NSTEMI rule in/out guideline' (proforma) and the 'Acute MI and Cardiology Decision Aid'<br>• Troponin result-based flow of patients in algorithm updated<br>  ○ Initial hs-cTnI threshold value for increased to 100ng/L based on updated evidence<br>  ○ Patients with significant cTn change at 2h all triaged to cardiology, irrespective of degree of elevation<br>• Education and training, monitoring and references sections all updated | |

Appendix A. Using troponin in the investigation of non-specific chest pain. Back to 1st page




* Acute MI and Cardiology Decision Aid Including Troponin Interpretation