

Fact Sheet

Point of Care Testing for cardiac Troponin I (Abbott i-STAT)

Your local facility has introduced Point of Care Testing (PoCT) for cardiac Troponin-I (cTnI). The local laboratory will continue to provide cTroponin testing. While there are similarities between the two forms of testing, there are some important differences to be aware of and that may impact the interpretation of test results:

- The laboratory based cardiac Troponin I assay is a highly sensitive (hs-cTnI) method and meets “NSW Health Chest Pain Pathway” criteria for reduced interval between sampling.
- The PoCT cTnI assay has a slightly inferior analytical sensitivity than the laboratory-based hs-cTnI assay.
- Therefore, to differentiate the two types of tests, PoCT cTnI results are expressed in ug/L and the laboratory hs-cTnI results in ng/L. This, in effect, gives an order of magnitude difference, i.e. three decimal places or one thousand times; e.g. 0.10 ug/L from a PoCT cTnI is equivalent to 100 ng/L.
- Due to the different analytical sensitivity and diagnostic accuracy, the laboratory hs-cTnI and PoCT cTnI assays have different clinical decision points.
- The clinical decision point for the i-STAT PoCT cTnI assays is **0.08 ug/L**.
- **In many cases the PoCT cTnI and laboratory based hs-cTnI tests are not directly comparable and therefore results measured on the PoCT device should not be converted to ng/L and interpreted against the laboratory based hs-cTnI assay.**
- It is also important that when the baseline PoCT cTnI test is followed up with the second cTnI test to meet the Universal Definition of Myocardial Infarction (see below) ***the retesting is performed using the same assay*** and the interval is in accordance with the NSW Health Chest Pain Pathway.
- When patient is going to be transferred in the follow-up period to a hospital where a different cTnI or Troponin T assay is available, the PoCT cTnI data may not help assessing the magnitude of change of Troponin I (delta). In such cases suggest take a baseline venous sample for a laboratory Troponin at the same time the PoCT cTnI test is done and send this sample together with the patient.

Interpretation of cardiac Troponin results:

- Elevation of cTroponin I is a highly specific marker of myocardial damage.
- According to an Expert Consensus, the Universal Definition of Acute Myocardial Infarction is: “In a clinical setting consistent with acute myocardial ischaemia.....detection of a rise and/or fall of cardiac Troponin.....with at least one value above the 99th percentile of a reference population” (*Thygesen et al. 2012;JACC 60(16):1581–98*).
- Also consider, an increased c-TnI above the 99th percentile may be caused by non-ischaemic or non-acute events and must be interpreted in the clinical context of the patient (for details see Table 1).

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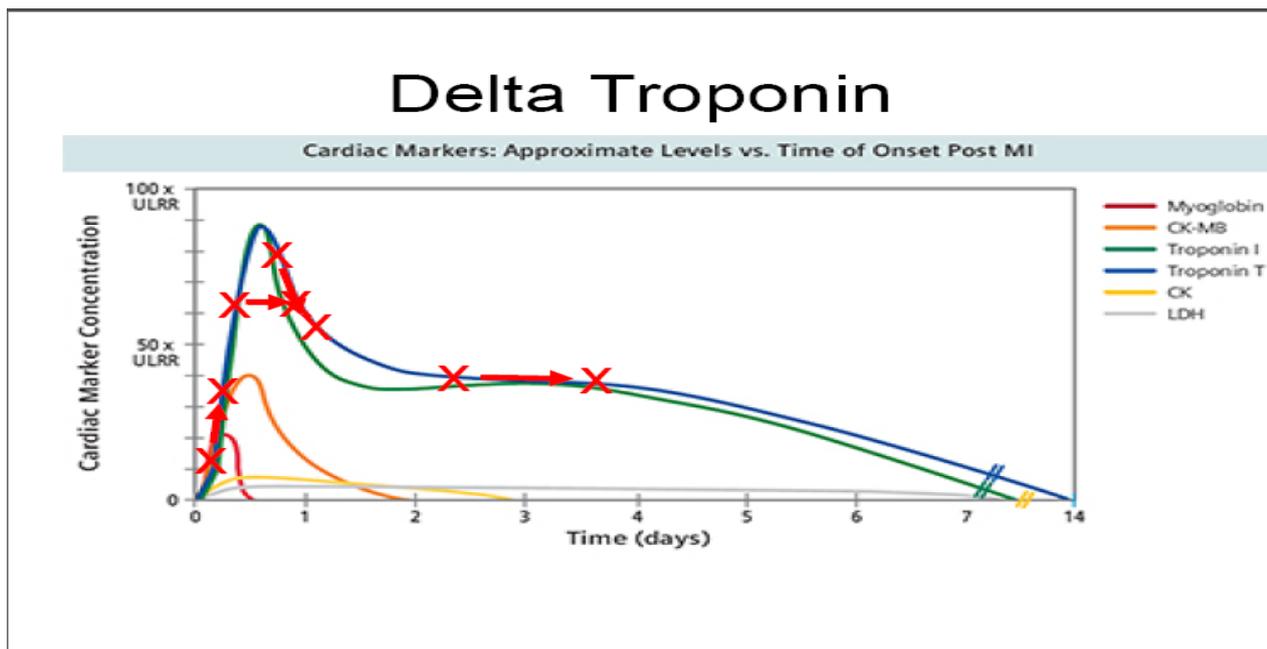
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- Clinicians must be aware that “false positive” and “false negative” results, although rare, may occur in all immunoassays, including troponin assays. This may be due to but not limited to interference from heterophilic antibodies and/or human auto-antibodies.
- In many cases the PoCT cTnI assay will have a lower diagnostic sensitivity than the laboratory-based hs-cTnI assay. The PoCT Troponin is useful to rule in the diagnosis but offers less assurance when used to rule out AMI.
- To ensure clinical diagnosis of Acute Myocardial Infarction, serial testing is essential and helps also in distinguishing an acute from a chronic or “false” elevation of cTnI.

Table 1: Causes of increased Cardiac Troponin I

<ul style="list-style-type: none">• Acute Myocardial Infarction• Tachy or bradyarrhythmias• Aortic dissection or severe aortic valve disease• Severe hypo or hypertension, e.g. haemorrhagic shock, hypertensive emergency• Acute or chronic heart failure• Hypertrophic cardiomyopathy• Coronary vasculitis, e.g. SLE, Kawasaki synd.• Coronary artery spasm, e.g. cocaine• Severe pulmonary embolism or pulmonary hypertension• Dialysis dependent renal failure	<ul style="list-style-type: none">• Cardiac contusion or surgery. Including but not limited to CABGS & stenting.• Rhabdomyolysis with cardiac involvement• Myocarditis, severe sepsis• Cardiotoxic agents, e.g. anthracyclines, CO poisoning• Severe burns affecting > 30% body surface• Severe acute neurological conditions, e.g. stroke, trauma• Infiltrative diseases, e.g. amyloidosis, sarcoidosis• Extreme exertion, e.g. marathon running• Frequent defibrillator shocks
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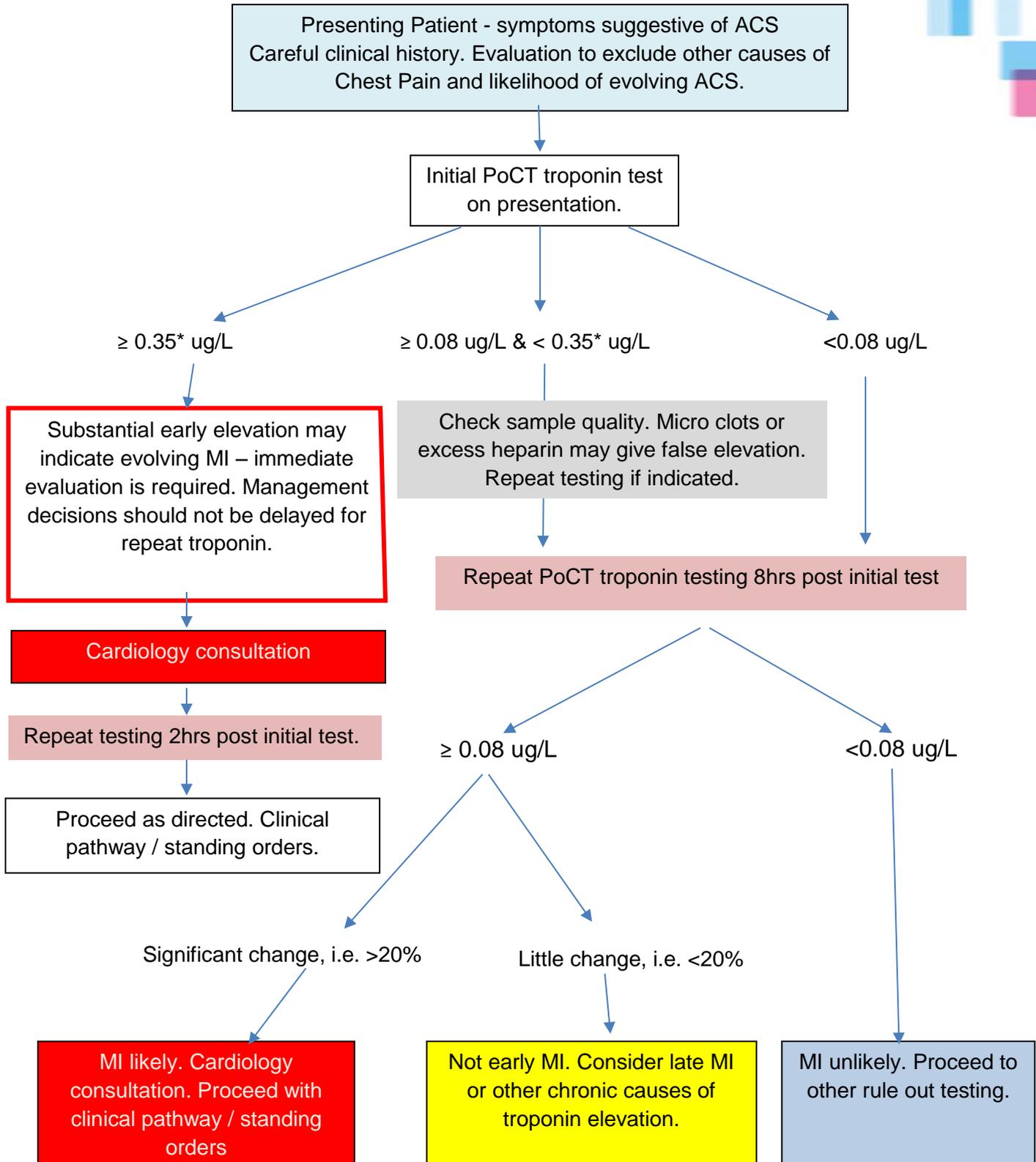
Table from: Jones GDR, Finckh A, Wilson S. Introducing Highly Sensitive Troponin into Routine Use - A Worked Example. The Clinical Biochemist Reviews Troponin Monograph 2012; pp97-102.



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PoCT cardiac Troponin flowchart. Abbott i-STAT cTnI



*0.35 ug/L is a level that will exclude most chronic elevations “false positive” of cTnI. In a setting strongly suggestive of MI and cTnI <0.35 ug/L management decisions should also not be delayed. Cardiac Troponin is only part of the evaluation of ACS. Values between 0.02 & 0.08 ug/L may represent detectable cardiac troponin or analytical noise.

Adapted from: National Heart Foundation of Australia. Acute Coronary Syndromes treatment algorithm. Updated Sept 2011.