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Cardiac enzymes and markers for myocardial infarction

Cardiac biomarkers should be measured in all patients who present with chest discomfort consistent with [acute coronary syndrome \(ACS\)](#).

Elevations of cardiac enzyme levels should be interpreted in the context of clinical and [ECG findings](#)^[1].

- Cardiac troponins T and I are the preferred markers for myocardial injury as they have the highest sensitivities and specificities for the diagnosis of acute myocardial infarction^[1].
- Patients with negative cardiac biomarkers within six hours of the onset of symptoms that are consistent with ACS should have biomarkers remeasured in the timeframe of twelve hours after the onset of symptoms.
- Peak circulating enzyme levels tend to occur earlier and are often higher following successful thrombolytic therapy.

Indications for measurement of cardiac enzymes

- Any patient presenting with a possible [ACS](#).
- Routinely following [percutaneous coronary intervention \(PCI\)](#).
- Routinely following surgical revascularisation ([coronary artery bypass graft \(CABG\)](#)).

Troponins T and I

Cardiac troponin I and T have displaced myoglobin and creatine kinase-MB as the preferred markers of myocardial injury^[2]. However, uncertainties and questions remain on the value of high-sensitivity cardiac troponin assays, including their best clinical use^[3].

Troponin is a protein released from myocytes when irreversible myocardial damage occurs. It is highly specific to cardiac tissue and accurately diagnoses myocardial infarction with a history of ischaemic pain or ECG changes reflecting ischaemia. Cardiac troponin level is dependent on infarct size, thus providing an indicator for the prognosis following an infarction^[4] .

New high-sensitivity cardiac troponin assays have been developed that can measure troponin values at much lower levels. With the use of these high-sensitivity assays, more patients with unstable angina will be classified as having non-ST-elevation myocardial infarction. These assays may therefore define a high-risk patient population and may lead to more appropriate therapy and improved outcomes in these patients^[5] .

- Cardiac troponins T and I are highly sensitive and specific for cardiac damage. Troponin I and T are of equal clinical value.
- Serum levels increase within 3-12 hours from the onset of chest pain, peak at 24-48 hours, and return to baseline over 5-14 days^[1] .
- Troponin levels may not be detectable for six hours after the onset of myocardial cell injury. The most sensitive *early marker* for myocardial infarction is myoglobin.
- Troponin levels should be measured at presentation and again 10-12 hours after the onset of symptoms. When there is uncertainty regarding the time of symptom onset, troponin should be measured at twelve hours after the presentation.
- The risk of death from an ACS is directly related to troponin level and patients with no detectable troponins have a good short-term prognosis.
- Elevated troponin levels can occur in patients without an ACS and are associated with adverse outcomes in many other clinical situations, including congestive heart failure, sepsis, acute pulmonary embolism and chronic kidney disease. Other cardiac causes include myocarditis and aortic dissection.

Creatine kinase

- Myocardial muscle creatine kinase (CK-MB) is found mainly in the heart.

- CK-MB levels increase within 3-12 hours of onset of chest pain, reach peak values within 24 hours, and return to baseline after 48-72 hours.
- Sensitivity and specificity are not as high as for troponin levels.

Myoglobin levels

- Myoglobin is found in cardiac and skeletal muscle.
- It is released more rapidly from infarcted myocardium than troponin and CK-MB and may be detected as early as two hours after an acute myocardial infarction.
- Myoglobin has high sensitivity but poor specificity. It may be useful for the early detection of myocardial infarction.

Natriuretic peptides

- Studies in several types of ACS have shown that elevated levels of natriuretic peptides - eg, B-type natriuretic peptide (BNP) - are independently associated with adverse outcomes - especially mortality^[6] ^[7] .

Other findings

- Leukocytosis may be seen within several hours after an acute myocardial infarction. It peaks in 2-4 days and returns to normal levels within one week.
- Patients without biochemical evidence of myocardial necrosis but with elevated C-reactive protein (CRP) level are at increased risk of a subsequent ischaemic event.
- Erythrocyte sedimentation rate (ESR) rises above reference range values within three days and may remain elevated for weeks.

Future developments

There are a number of novel biomarkers under investigation, but none has been tested and proven to alter outcome of therapeutic intervention^[4]. Although some have improved prediction of outcome in acute myocardial infarction, none has been demonstrated to alter the outcome of a particular therapy or management strategy. Examples include:

- Heart-type fatty acid binding protein and copeptin (in combination with cardiac troponin) diagnose myocardial infarction or ACS in the early hours following symptoms.
- Mid-regional pro-atrial natriuretic peptide, ST2, C-terminal pro-endothelin 1, mid-regional pro-adrenomedullin and copeptin all provide information in predicting death and heart failure.
- Growth differentiation factor-15 and high-sensitivity CRP may predict death following an ACS.

Further reading

- [Consensus guideline for recording a standard 12-lead electrocardiogram](#); Society for Cardiological Science & Technology, September 2017.
- [TIMI Risk Scores](#); Cardiology.org

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